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Mapping QTL associated with resistance to Fusarium head blight in the Nanda2419 × Wangshuibai population. II: Type I resistance

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Abstract Fusarium head blight (FHB) is a serious disease in wheat and barley affecting both yield and quality. To identify genes for resistance to infection, the RIL population derived from 'Nanda2419' × 'Wangshuibai' and the parents were evaluated for percentage of infected spikes (PIS) in four different environments. Using a 2,960 cM marker framework map constructed for this population, ten chromosome regions were detected for their association with type I resistance through interval mapping with Mapmaker/QTL, among which QTLs mapped in the intervals of *Xwmc349*~*Xgwm149* on chromosome 4B, of *Xwmc96*~*Xgwm304* on chromosome 5A and of *Xgwm408*~*Xbarc140* on chromosome 5B were revealed in at least three environments and have Wangshuibai as the source of resistance alleles. *Qfhi.nau-4B* and *Qfhi.nau-5A* had larger effects and explained up to 17.5 and 27.0% of the phenotypic variance, respectively. To detect epistasis QTLs, two-locus interactions were examined by whole genome scan. Interactions of five locus pairs were found to have significant effects on type I resistance with the LOD score ranging 3.8–6.5 and four of them conferred resistance in parental phase. The one with the most significant effect was *Xcfd42*~*Xgwm469* (6D)/*Xwmc390-2*~*Xbd04* (2A) pair. No QTL × E interaction was detected for PIS. It was found that flowering time did not have significant effects on PIS in this population. Our studies indicated that Wangshuibai is useful for breeding for both type I and type II scab resistance and the markers associated with the QTLs could be used in marker-assisted selection and isolation of scab-resistance QTLs.

Keywords Fusarium head blight · QTL · Type I resistance · Wangshuibai · Epistasis

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Introduction

The yield and quality of wheat suffers seriously from Fusarium head blight (FHB) or scab disease, caused mainly by *Fusarium graminearum* Schwabe. Genetically controlled resistance is the basis of scab-resistance breeding, which is critical for combating this disease. So far, two types of scab resistance, including type I resistance against initial infection and type II resistance against fungal spread within the spike (Schroeder and Christensen 1963), were most extensively studied. Other types of resistance, for instance, type III for kernel resistance, type IV for yield tolerance and type V for decomposition or non-accumulation of mycotoxins, were also proposed (Mesterhazy 1995), but have not been widely accepted because of some conceptual weakness or methodological difficulty (Bai and Shaner 2004). Association of morphological and physiological traits with scab resistance has been reported in some germplasms; however, the results were inconsistent between different studies (Atanasoff 1920; Buerstmayr et al. 2000; Steiner et al. 2004; Gilsinger et al. 2005).

Scab resistance is controlled by multiple genes whose effects are greatly influenced by environments (Snijders 1990; Parry et al. 1995). This complexity has limited our understanding of the resistance mechanisms and made conventional scab-resistance breeding a difficult, time consuming and tedious practice. The availability of molecular markers and marker maps has fostered the progress of scab-resistance genetics. In the last few years, quantitative trait loci (QTL) for type II resistance have been mapped in some segregating populations and most of the resistance alleles are contributed by resistance

germplasms, such as 'Arina' (Paillard et al. 2004), 'CM-82036' (Buerstmayr et al. 2002), 'Frontana' (Steiner et al. 2004), 'Fundulea201R' (Shen et al. 2003), 'Renan' (Gervais et al. 2003), 'Sumai no. 3' (Anderson et al. 2001; Xu et al. 2001), Wangshuibai (Lin et al. 2004; Zhou et al. 2004) and so on. QTLs locating on chromosomes 3B and 6B were the most commonly found and had the largest effects. A few studies have detected QTLs against the initial infection in populations derived from Sumai no. 3 (Xu et al. 2001), CM-82036 (Buerstmayr et al. 2003), Frontana (Steiner et al. 2004) and 'Goldfield' (Gilsinger et al. 2005), but no common QTLs have been found. The QTL mapping information has been employed in marker-assisted improvement of scab resistance and development of near-isogenic lines (Del Blanco et al. 2003; Yang et al. 2003).

Type II resistance evaluation is routinely conducted by single floret inoculation with a macroconidial spore suspension and then scoring the number of infected spikelets 14–21 days post-inoculation. The percent number of infected spikelets over the total number of spikelets is often used to represent type II resistance. Lin et al. (2004) investigated the type II resistance of the recombinant inbred line (RIL) population derived from 'Nanda2419' × Wangshuibai by using the number of diseased spikelets and the length of diseased rachides as the disease indices to avoid influence of the variation in the length of spikes and the total number of spikelets among different lines. Type I resistance is evaluated in a different way, often by measuring the disease severity or percentage of infected spikes (PIS) after spray inoculation or natural inoculation in disease nurseries, with the former including both type I and type II resistance.

Type I resistance is the host's first barrier to infection by *Fusarium* species and is important for scab-resistance breeding. Here we report our findings in QTL mapping for this trait using the Nanda2419 × Wangshuibai population.

Materials and methods

Plant materials and resistance evaluation

The $F_{7:9}$ RIL population derived from the cross of Nanda2419 × Wangshuibai (Lin et al. 2004) and the parents were used in this study. The evaluation was performed with four trials in a randomized complete block design in 3 years from 2003 to 2005 at the normal planting season. The 2003 trial and two 2005 trials consisted of two replicates and the 2004 trial had four. One of the 2005 trials was placed in Jiangpu county (JP), Jiangsu and the others at different fields in Jiangsu Academy of Agricultural Sciences (JAAS), Nanjing, Jiangsu. Each plot had one row in 1 m length in 2003 or two rows spaced 25 cm apart. The sowing density was 15 seeds per row.

At 50% anthesis of each plot, mixed conidial suspension of four local virulent strains of *F. graminearum* was sprayed on the heads in the JAAS trials and repeated 2 days later in all but 2004, when the plants were sprayed once a day during anthesis. At JP, the inoculation was conducted by scattering scabby wheat grains on the soil surface about 10 days before anthesis and repeating the inoculation a week later. After inoculation for 15–20 days, the number of spikes with visible FHB symptom in at least one of their florets and the total spikes of each plot were scored. PIS was used to represent the type I resistance.

Flowering time

Flowering time, defined as the date of 50% appearance of anthesis, was recorded for each line in all trials.

Statistical analysis

Analysis of variance (ANOVA) and correlation was performed with 145 lines (2003) or 278 lines (2004, 2005) of this population using statistical software Data Desk v. 5.0 (Data Description, Inc., Ithaca, NY, USA). Each site–year combination was treated as an environment. The line-based broad-sense heritability was calculated with the formula $h^2 = \sigma_g^2/(\sigma_g^2 + \sigma_{ge}^2/e + \sigma_e^2/re)$ using variance components estimated based on ANOVA, where σ_g^2 is the genetic variance, σ_{ge}^2 is the variance from genotype × environment interaction, σ_e^2 is the experimental error, e is the number of environments and r is the number of replications.

QTL analysis

The framework map (Lin et al. 2004) used in this study included 310 loci in 40 linkage groups, covering over 2,960 cM of the wheat genome. One hundred and thirty six lines used in the map construction were analyzed for QTL mapping. QTLs were detected with Mapmaker/QTL v. 1.9 (Lander and Botstein 1989) through simple interval mapping (SIM) and composite interval mapping (CIM) as described in Lin et al. (2004), where the QTL with the highest LOD score from SIM was fixed at the given peak position using the 'sequence' command for a second round of whole map scan. The LOD score for declaring a QTL was 2.0 in SIM or 2.0 higher than that of the fixed QTL in CIM.

QTL scan was also performed in single-environment and multiple-environment models through SIM and simplified composite interval mapping (sCIM) with MQTL version 1.0 following the procedures as described by Tinker and Mather (1995a). In the single-environment model, the presence of QTLs was inferred based on SIM, and the QTL positions were refined according to the sCIM test statistics. In the multiple-environment model, QTLs and genome-wide QTL × E (environment) interactions for scab resistance were examined using

SIM and the QTL locations were inferred based on peaks for both main effects and QTL × E interactions (Tinker and Mather 1995a, b). The statistic significance threshold used here to declare the presence of a QTL and QTL × E interactions was determined by 1,000 random permutations with a genome-wide type I error rate of 5%. The proportion of variance that was explained by a single QTL or QTL × E to the total variance was estimated using the equation $R^2 = 1 - 1/\exp(TS/n)$ (Tinker 1996), where n is the total number of lines excluding those with missing data, the TS is the peak value of a distribution generated by SIM.

Epistasis detection

To survey the two-locus epistatic interactions, we first held one of the marker locus (the 'anchor locus' 'A') constant, and then scanned the genome with MQTL for the second locus 'B' that interacts with the 'A' locus using a walking speed of 1 cM. When the test statistic for interaction model (A + B + A×B) versus additive model (A + B) was equal to or larger than 9.1, which is equivalent to LOD = 2.0 in MAPMAKER/QTL (Tinker and Mather 1995b), the 'B' locus was chosen as the putative epistasis QTL (epQTL). To better establish the intervals of the two-locus interaction, a second round of interaction scan was then performed by scanning the genome again using the walking speed of 1 cM after holding each of the chosen putative epQTLs constant. The final significant threshold declaring the epQTL was set by 1,000 random permutations with a genome-wide type I error rate of 5%. Linked epQTLs were defined as independent when more than 30 cM genetic distance separated the interval peaks.

Chromosome assignment of QTLs

Individual QTLs were assigned to chromosomes based on the linked markers. For those assignments in doubt, the markers linked to or in the QTL intervals were chosen to analyze nulli-tetrasomic lines of 'Chinese Spring'. In this process, the DNA was extracted according to Ma and Sorrells (1995) and PCR with the chosen SSR primers was performed following the procedure described in Lin et al. (2004).

Results

Phenotypic analysis

Percentage of infected spikes

In the Nanda2419 × Wangshuibai population, PIS of the individual lines varied greatly and the population data displayed a near-normal distribution with the peaks toward the resistance tail in all the trials (Fig. 1). Wangshuibai is constantly more resistant than Nanda2419. The population means fell between the parents in all environments but 2004JAAS in which Nanda2419 had a lower PIS than the population means, which could be explained by the fact that the flowering time of Nanda2419 in 2004 just met the cold, dry weather condition that is unfavorable for the disease development. The early-heading lines in the population in this year could appear more resistant than normal. Based on ANOVA combined across the environments, the variance between the replications reached the significant level of $P = 0.05$, and those between environments, between RILs and of lines × environments were all significant at $P \leq 0.0001$. ANOVA with the single environment data indicated that 2005 JP was the only trial that had significant difference between the replications. However, the correlation coefficient between the replications of this trial was as high as 0.753. The environment-wide correlation coefficients ranged 0.439–0.679, significant at $P \leq 0.0001$. The line-based broad-sense heritability of PIS was 85.2%.

These results indicated that the line-based PIS data in each trial was suitable for further analysis and the type I resistance in these populations was determined by polygenes.

Relationship of flowering time with type I resistance

Nanda2419 flowered 3–7 days earlier than Wangshuibai in the four trials. In the RIL population, the ranges of flowering time were 19, 10, 13 and 10 days in 2003, 2004, 2005 JAAS and 2005 JP, respectively, suggesting a large variation in flowering time among the RILs. Correlation analysis showed that the 2004 trial was the only one in which the PIS was positively related to flowering time,

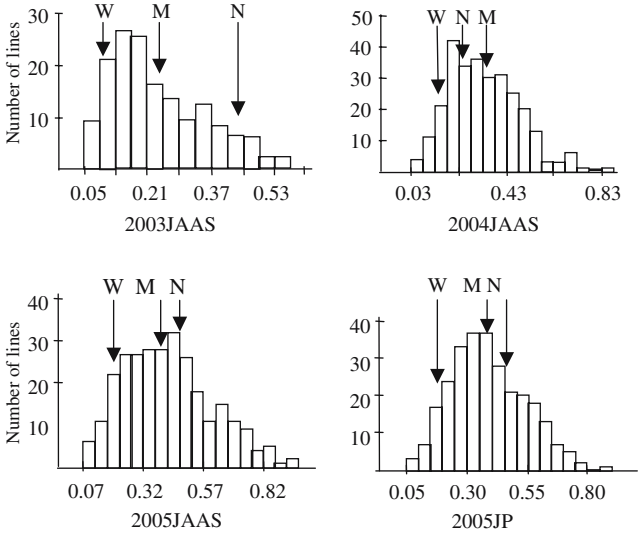

Fig. 1 Distribution of PIS in the Nanda2419 × Wangshuibai population. *W* Wangshuibai, *N* Nanda2419, *M* mean value

but with a correlation coefficient as small as 0.192. Therefore, the flowering time did not affect too much the type I resistance in these experiments.

QTL analysis

Interval mapping

Five chromosome regions showing significant association with PIS were identified through SIM (Table 1). Two of the QTLs, mapped on chromosomes 4B and 5A, respectively, were revealed in all environments and the one mapped to the interval of *Xgwm539*~*Xwmc181*~*Xaf12* on chromosome 2D was found in two environments. The QTLs found in multiple environments were also detected using MQTL. The chromosome locations of all the QTLs could be determined based on the maps of the linked markers that have been assigned to the individual chromosomes by referring to the published SSR maps or by nulli-tetrasomic analysis (Lin et al. 2004).

Qfhi.nau-5A, mapped in the 0.4 cM interval of *Xwmc96*~*Xgwm304* (Table 1, Fig. 2), had the largest effects in all environments and explained up to 27.0% of the phenotypic variance. Estimated with the closest marker *Xwmc96*, this QTL reduced up to 36.0% of the PIS in the four trials (Table 2). Through sCIM with MQTL, *Qfhi.nau-5A* was mapped in the 5.2 cM region defined by *Xwmc446*~*Xwmc705*~*Xwmc96*~*Xgwm304* with the peaks varying within 2.8 cM. Combined across the environments, this QTL was positioned 0.1 cM away from *Xwmc96* (Fig. 2).

Qfhi.nau-4B was mapped in the 9.7 cM region defined by *Xwmc349*~*Xgwm149*~*Xgwm513* with the individual sets of data and explained up to 17.5% of the phenotypic variance. Its peak position varied in a distance of 3.7 cM (Table 1, Fig. 2). Combined across the environments, this QTL was mapped right at *Xgwm513*, 1.4 cM from *Xgwm149* (Fig. 2). In one of the four trials it caused reduction of PIS to a degree similar to *Qfhi.nau-5A* (Table 2).

Combining *Qfhi.nau-4B* and *Qfhi.nau-5A* together, they explained 24.8, 24.8, 26.1 and 34.8% of the phenotypic variance in 2003, 2004, 2005 JAAS and 2005 JP, respectively, and reduced up to 47.2% of PIS (Table 2).

Qfhi.nau-2D1 was detected in 2003 and 2005 JAAS with the peaks mapped less than 10 cM from each other in the 21.2 cM region defined by *Xgwm539*~*Xwmc181*~*Xaf12*. It explained up to 12.3% of the phenotype variance.

When *Qfhi.nau-5A* was fixed for CIM analysis with Mapmaker/QTL, all the QTLs detected by SIM remained and the peak positions were basically not affected. Some putative new QTLs were found, most of which appeared only in one of the four environments

Table 1 QTLs for PIS detected through interval mapping with Mapmaker/QTL

Methods of QTL detection	QTL[a]	Interval	Location	Source of resistance allele	Length (cM)	Peak position (cM)[b]	LOD[c]	R^2 (%)[d]
			2003JAAS					
SIM	*Qfhi.nau-2D1*	*Xwmc181*~*Xaf12*	2D	W	5.9	*Xwmc181* + *4.0*	3.3	12.3
	Qfhi.nau-4B	*Xwmc349*~*Xgwm149*	4B	W	8.3	*Xwmc349* + *6.0*	3.2	12.1
	Qfhi.nau-5A	*Xgwm304*~*Xbarc56*	5A	W	0.4	*Xgwm304* + *0**	6.5	20.3
CIM	*Qfhi.nau-2D2*	*Xwmc445-1*~*Xgwm311-1*	2D	W	9.7	*Xwmc445-1* + *9.0*	2.6	8.6
	Qfhi.nau-5B	*Xgwm408*~*Xbarc140*	5B	W	26.0	*Xgwm408* + *0*[f]	1.7	4.6
			2004JAAS					
SIM	*Qfhi.nau-4B*	*Xwmc349*~*Xgwm149*	4B	W	8.3	*Xwmc349* + *7.0*	3.5	13.1
	Qfhi.nau-5A	*Xwmc96*~*Xgwm304*	5A	W	0.4	*Xwmc96* + *0*[e]	6.0	18.7
	Qfhi.nau-7D	*Xgwm437*~*Xwmc488*	7D	N	15.7	*Xgwm437* + *10.0*	2.2	10.7
CIM	*Qfhi.nau-3B*	*Xgwm533-2*~*Xwmc054-1*	3B	N	9.2	*Xgwm533-2* + *7.0*	2.1	6.9
	Qfhi.nau-4A	*Xwmc501-2*~*Xwmc161*	4A	N	4.3	*Xwmc501-2* + *4.0*	2.0	5.6
	Qfhi.nau-7A	*Xwmc338-2*~*Xwmc83*	7A	N	16.5	*Xwmc338-2* + *10.0*	3.4	12.5
			2005JAAS					
SIM	*Qfhi.nau-2D1*	*Xgwm539*~*Xwmc181*	2D	W	15.3	*Xgwm539* + *10.0*	2.4	10.1
	Qfhi.nau-4B	*Xwmc349*~*Xgwm149*	4B	W	8.3	*Xwmc349* + *6.0*	4.8	17.5
	Qfhi.nau-5A	*Xwmc96*~*Xgwm304*	5A	W	0.4	*Xwmc96* + *0*[e]	5.3	16.6
	Qfhi.nau-5B	*Xgwm408*~*Xbarc140*	5B	W	26.0	*Xgwm408* + *0*	2.2	7.4
CIM	*Qfhi.nau-7B*	*Xbarc126-2*~*Xwmc476*	7B	N	19.7	*Xbarc126-2* + *8.0*	2.4	10.4
			2005JP					
SIM	*Qfhi.nau-4B*	*Xgwm513*~*Xbarc20*	4B	W	3.8	*Xgwm513* + *0*	5.0	16.2
	Qfhi.nau-5A	*Xwmc96*~*Xgwm304*	5A	W	0.4	*Xwmc96* + *0*[e]	9.1	27.0
CIM	*Qfhi.nau-5B*	*Xgwm408*~*Xbarc140*	5B	W	26.0	*Xgwm408* + *8.0*[f]	1.9	8.8

[a]Quantitative trait loci that overlapped in the one-log support confidence intervals were given the same symbol
[b]The position is represented by the left boundary locus of the interval plus a genetic distance (in centiMorgan) proximal to it
[c,d]The LOD and R^2 values for the QTLs from CIM were derived from the corresponding values of CIM minus the corresponding values of the fixed QTL
[e]The QTL position fixed for CIM
[f]The QTL had a LOD value less than 2.0, but was detected in multiple environments

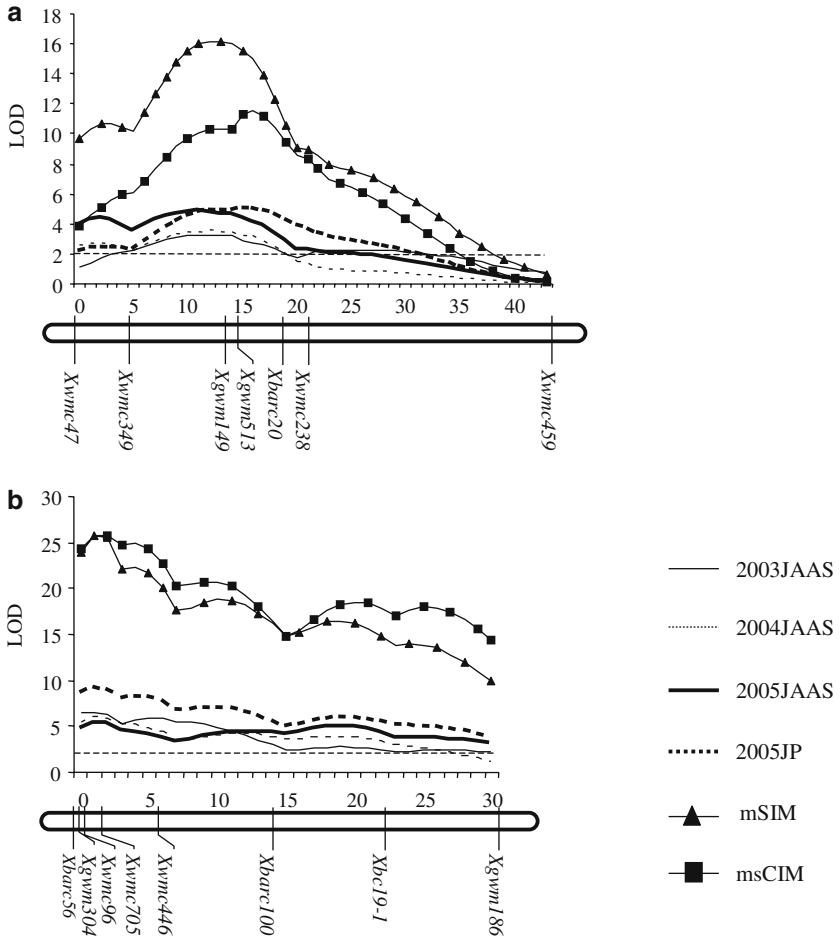

Fig. 2 Mapping QTLs on chromosomes 4B (**a**) and 5A (**b**) through SIM with MAPMAKER/QTL and through SIM and sCIM with multiple environment model of MQTL. mSIM and msCIM mean, respectively, the SIM and sCIM in the multiple environment model

Table 2 The reduction[a] of PIS (%) due to the resistance alleles at the two major QTLs independently or in combination

	Xwmc96	*Xgwm149*	*Xwmc96* + *Xgwm149*
2003	36.0	30.0	47.2
2004	30.4	25.4	40.8
2005JAAS	30.2	30.5	43.3
2005JP	33.7	27.3	43.6

[a](Wangshuibai homozygous–Nanda2419 homozygous)/Nanda2419 homozygous

(Table 1). The only exception was *Qfhi.nau-5B*, which was detected through SIM using the 2005 JAAS data, was also revealed through CIM in 2003 or 2005 JP with a LOD score of 1.7 or higher. Combining it together with *Qfhi.nau-4B* and *Qfhi.nau-5A*, the phenotypic variation explained by them varied from 29.2% in 2003 to 36.4% in 2005 JP. *Qfhi-nau-2D2*, detected by CIM using the 2003JAAS data, was not linked to *Qfhi-nau-2D1* genetically (Table 1).

In SIM analysis, *Qfhi.nau-7D*, detected using the 2004 data, was the only QTL with the resistance allele contributed by Nanda2419. However, four of the five new putative QTLs identified through CIM had the resistance alleles contributed by Nanda2419 and most of them were revealed only in 2004 (Table 1). Because of the particularity of the 2004 data as mentioned before and the low repeatability of the new QTLs across the environments, they need to be confirmed.

Using the multiple-environment model of MQTL, genome-wide examination of QTL × E interactions for scab resistance was conducted, but no single QTL × E interaction was detected with a significance level higher than the threshold set by 1,000 random permutation test.

Epistatic interaction

In the preliminary round of scanning for two-locus epistatic interactions, 354 locus pairs were found to have association with PIS with test statistics equal to or larger than 9.1. Nineteen of them, involving five chromosomal regions, were significant with a genome-wide type I error rate of 5% and the LOD score varied from 3.8 to 6.5 (Table 3). The one having the highest LOD score was the *Xcfd42*∼*Xgwm469* (6D)/*Xwmc390-2*∼*Xbd04* (2A) pair. None of the epistatic loci were detected independently in the interval mapping for their association with PIS. The phenotype means of the recombinant geno-

types in all the significant pairs but *Xwmc273-1*~*Xwmc634* (7D)/*Xgwm52*~*Xgwm341* (3D) were higher than the parental genotypes (Table 3). These results suggested that some loci are co-adapted for resistance in evolution and exist in both resistant and susceptible germplasms.

Discussion

Wangshuibai is an important and unique germplasm for scab resistance of wheat. In this study, we showed that Wangshuibai made the largest contribution to type I resistance in the Nanda2419 × Wangshuibai population. All the four QTLs with higher LOD scores have Wangshuibai as the source of resistance alleles, among which the 5A QTL had the largest effects (Table 1). Most of the QTLs with Nanda2419 as the source of resistance alleles were only detected using the 2004 data, as shown in correlation analysis between flowering time and PIS, which could be attributed to the special weather conditions in that year that favored the plants flowering earlier for resistance. No significant QTL × E interaction was found in this population with the four-environment data.

In the population from the cross of Frontana × 'Remus', QTLs associated with PIS distributed on chromosomes 1B, 2B, 3A, 4B and 6B, respectively, of which the 3A QTL near the centromere contributed largest (Steiner et al. 2004). But no common markers were available for precise comparison other than the chromosomes involved. QTLs for FHB incidence or type I resistance have been reported in the populations derived from Sumai no. 3 × 'Gamenya' (Xu et al. 2001) and from 'Patterson' × Goldfield (Gilsinger et al. 2005); however, none of them were mapped to the intervals same as those in this study. Thus, we concluded that Wangshuibai has a unique genetic basis for its type I scab resistance.

Three of the chromosomal regions associated with type I resistance in Wangshuibai have been related to scab resistance in other cultivars. For example, *Qfhi.nau-5A* seemed to be similar in position to the 5A QTL allele mainly for type I resistance in CM-82036 (Buerstmayr et al. 2003). Association of this QTL interval with type II scab resistance, although only with secondary importance, was also reported in CM-82036 (Buerstmayr et al. 2002), in Fundulea201R (Shen et al. 2003), and in Frontana (Steiner et al. 2004). However, in the Nanda2419 × Wangshuibai population no type II resistance QTL was mapped in this interval (Lin et al. 2004). Somers et al. (2003) found a QTL in the same region controlling the deoxynivalenol (DON) accumulation. Since both type I resistance and type II resistance could affect the DON level, it is not yet known if this was because of the existence of type I or type II QTL in this region. The position of *Qfhi.nau-4B* might be in agreement with that of the QTL allele linked to *Xwmc238* on chromosome 4B in 'Wuhan-1' for field resistance that includes the effects of both disease incidence and disease spread (Somers et al. 2003). In Frontana, Steiner et al. (2004) identified a minor QTL allele for FHB incidence on this chromosome. *Qfhi.nau-2D1* shared the interval with the QTL in Arina × 'Forno' RIL population for FHB AUDPC (the area under disease progress curve) (Paillard et al. 2004) and the 2D QTL allele in Wuhan-1 from single floret inoculation (Somers et al. 2003). QTLs for scab resistance on chromosome 5B reported by Bourdoncle and Ohm (2003) and by Paillard et al. (2004) were not the same as the *Qfhi.nau-5B* based on the mapping information.

Even though the QTLs with Nanda2419 as the source of resistance alleles showed less repeatability over the environments (Table 1), *Qfhi.nau-7B* was mapped to the same interval as the 7B QTL detected in the F2:3 population derived from 'Ning7840' × 'Freedom' (Gupta et al. 2000) and in the RIL population derived from 'Dream' × 'Lynx' (Schmolke et al. 2005). This QTL coincided with the QTL for heading date in the Dream × Lynx population. Similarly, *Qfhi.nau-7B* was mapped to the interval of the QTL for flowering time in the Nanda2419 × Wangshuibai population (unpublished data).

At least 19 chromosomes of wheat have been reported for their association with scab resistance in about 30 germplasms. In all of these studies, the individual QTLs acted mainly additively, even though epistatic and dominant effects have been reported (Bai et al. 2000; Buerstmayr et al. 2000). We found that epistasis could have important contribution to type I resistance through examination of two-locus interactions. It was shown that the locus involved may not

Table 3 Epistatic interactions detected for PIS across the environments

Locus A		Locus B		Phenotype means (%)[a]				LOD
Interval/location	Distance (cM)/Peak	Interval/location	Distance (cM)/peak	aabb	AAbb	aaBB	AABB	
Xcfd42~*Xgwm469*/6D	15.7/*Xcfd42* + *0*	*Xwmc390-2*~*Xbd04*/2A	25.3/*Xwmc390-2* + *14*	29	45	39	22	6.5
Xgwm67~*Xgwm219-1*/5B	16.4/*Xgwm67* + *6*	*Xgdm93-4*~*Xwmc149-1*/2B	10.2/*Xgdm93-4* + *10*	28	38	40	29	3.9
Xwmc273-1~*Xwmc634*/7D	10.8/*Xwmc273-1* + *2*	*Xgwm52*~*Xgwm341*/3D	10.3/*Xgwm52* + *8*	38	31	25	39	4.1
Xwmc311~*Xgwm611*/7B	15.0/*Xwmc311* + *10*	*Xgwm67*~*Xgwm219-1*/5B	16.4/*Xgwm67* + *6*	26	42	38	27	4.8
Xwmc517~*Xbf09*/7B	14.1/*Xwmc517* + *13*	*Xba02-1*~*Xwmc01*/3B	6.6/*Xba02-1* + *0*	26	39	40	32	3.8

[a]The capital letters mean Wangshuibai genotype

have major effects, but its interaction with another locus, either in couple phase (parental type) or in repulse phase (recombinant type) could have significant impacts. In most of the digenic interactions detected in this study, the parental genotypes favored resistance, implying a tendency of co-adaptation for resistance in evolution. The two-locus interactions without individual effects were also found in the Renan × 'Récital' population by Gervais et al. (2003). It is necessary to validate the epQTLs and take them into consideration in breeding programs.

Significant correlation of flowering time or heading date with FHB severity has been reported (Gervais et al. 2003; Somers et al. 2003; Paillard et al. 2004; Steiner et al. 2004; Schmolke et al. 2005), but co-localization of the resistance QTLs with QTLs for flowering time or heading date was not always detected (Somers et al. 2003; Steiner et al. 2004). In our study, the only correlation of flowering time with PIS was found using the 2004 data and no QTLs except *Qfhi.nau-7B* detected through CIM using the 2005JAAS data overlapped the QTL intervals for flowering time (data not shown). As suggested by Buerstmayr et al. (2000), these two traits should be independent genetically. The flowering time could affect scab resistance by allowing plants to escape infection or by slowing down spreading when the weather conditions were not optimal for disease infection or development.

Our results demonstrated that type I resistance and type II resistance in Wangshuibai are controlled by different genes. This finding is consistent with the views of Schroeder and Christensen (1963) as well as Miedaner et al. (2003). Among the ten chromosome regions associated with type I resistance and 11 regions associated with type II resistance (Lin et al. 2004) identified in the Nanda2419 × Wangshuibai population, the only overlap was in the interval of *Xgwm533-2*∼*Xwmc054-1* where *Qfhi.nau-3B* has its resistance allele from Nanda2419, while the type II QTL has its resistance allele from Wangshuibai. In the Frontana × Remus population, Steiner et al. (2004) obtained similar results. Of the five QTLs for type I resistance and six QTLs for type II resistance they found, the one mapped on chromosome 2B was the sole QTL that were associated with both types of resistance.

Mapping QTLs including epistatic loci for scab resistance in Wangshuibai paves the way for better utilization of this germplasm. To elucidate the effects of the individual QTLs, genetic stocks such as near-isogenic lines are being developed using the molecular markers and genetic interactions of the QTLs are being examined employing the immortal F_2 population created using the RILs.

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