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ORIGINAL PAPER

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

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Abstract Scab disease caused by *Fusarium* spp. has been a major concern for both wheat producers and consumers. Deployment of scab-resistant varieties is the major strategy to curb this disease. To identify the scab resistance genes in wheat cv. Wangshuibai, we produced a $F_{6:7}$ recombinant inbred line (RIL) population by crossing Wangshuibai with the scab-susceptible cultivar Nanda2419. The RILs were evaluated for scab resistance in the field by single floret inoculation in two replicates in 2002 and one replicate in 2003. The number of diseased spikelets (NDS) and the length of diseased rachides (LDR) were investigated to reflect the Type II resistance. Among 654 simple sequence repeat (SSR) markers surveyed, 326 were found to be polymorphic between the parents. A partial molecular map was constructed with these markers that covered over 2,210 cM of the wheat genome. Six chromosome regions showed association with both NDS and LDR in a one-way ANOVA analysis, even though the variation explained by them varied between the two traits. Eight intervals were detected for their association with Type II resistance through interval mapping, five of which were not identified in single-point analysis. The quantitative trait loci (QTL) with large effects were the ones in the interval of *Xgwm533-3–Xgwm533-1* on chromosome 3B and in the interval of *Xwmc539–Xbarc024* on chromosome 6B, whose alleles favoring resistance originate from Wangshuibai. In addition, a QTL whose resistance allele originated from Nanda2419 was consistently detected in the interval of *Xs1021m–Xgwm47-1* on chromosome 2B. These results suggest that Wangshuibai is the major source for Type II resistance in this population. The markers associated with these QTL would facilitate the use of scab-resistant genes of Wangshuibai in scab resistance breeding programs of wheat.

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Introduction

Scab or *Fusarium* head blight caused by the *Fusarium* spp., primarily *Fusarium graminearum*, is a destructive disease of wheat worldwide, and is particularly damaging in regions with a warm and humid climate during the flowering stage (McMullen et al. 1997). The disease can cause bleached spikes, sterility, poor seed filling, low test weight, and tombstone seeds, and the resulting loss in yield can range from 10% to 40% in epidemic years (Wang 1996). In addition, seed infections deteriorate the grain quality and contaminate the grains with mycotoxins, making the grains unsuitable for both human consumption and for use as fodder. Thus, scab disease poses a serious food security problem.

Even though chemical and biological measures have been proposed to control this disease, the employment of scab-resistant varieties is still the most economical and effective control method (Parry et al. 1995). Since Arthur (1891) reported genetic variations in scab resistance in wheat, a large number of germplasms have been screened (Snijders 1990; Liu and Wang 1991; Saur 1991). While no germplasm immune to this disease is currently available, a few cultivars highly resistant to scab have been identified, such as Sumai No. 3 and Wangshuibai from China, Nobeokabouzu from Japan, Frontana from Brazil and Praa 8 and Novokrumka from Europe (Gilbert and Tekauz 2000). Sumai No. 3 has been the most widely used resistance germplasm in breeding programs (Li et al. 2000; Del Blanco et al. 2003). However, such a narrow genetic basis makes it a high priority to identify and utilize alternative resistance resources.

Scab resistance of wheat has been classified into three types: Type I resistance against initial penetration, Type II against fungal spread within spikes (Schroeder and Christensen 1963) and Type III for toxin decomposition (Miller et al. 1985). Additional resistance types have been proposed by Mesterhazy (1995). Scab resistance is controlled by both major and minor genes whose effects are greatly influenced by environment (Snijders 1990; Ban and Suenaga 2000). Because of this genetic complexity, our understanding of the resistance mechanism is very limited, and progress in scab resistance breeding is slow and far from meeting our needs.

Recently, quantitative trait loci (QTL) for scab resistance have been reported in Sumai No. 3 and its derived lines, such as Ning7840, CM-82036 and ND2603 (Bai et al. 1999; Waldron et al. 1999; Anderson et al. 2001; Buerstmayr et al. 2002; 2003; Del Blanco et al. 2003), and in a few other resistant varieties (Gervais et al. 2003; Shen et al. 2003a). Most of these studies concluded that the major genes controlling scab resistance were on chromosomes 5A, 3B and 6B of the wheat genome.

Wangshuibai is one of the best scab-resistant germplasms that has originated from Jiangsu, China and has hardly been used in breeding programs. In the investigation reported here, a RIL population of Nanda2419 × Wangshuibai was created, and QTL for Type II resistance are reported. The results indicate that Wangshuibai contains eight QTL for Type II resistance, and the relationships of these loci to published scab resistance QTL are discussed.

Materials and methods

Plant materials

A $F_{6:7}$ recombinant inbred line (RIL) population was produced by single-seed descent from wheat cv. Nanda2419 × cv. Wangshuibai, 154 of which were used in this study. Nanda2419, a spike selection of Italian var. *Mentana*, is susceptible to scab. The RILs and the parents were grown in a field of the Nanjing Agricultural University in a randomized block design with two replicates in 2002 and a single replicate in 2003. Fifteen seeds of each line were planted in a 1-m row. A misting system was set up to provide humidity during the stage of disease development.

Resistance evaluation

A mixed conidial suspension of four local virulent strains of *Fusarium graminearum* was used for inoculation at anthesis. Approximately 15–20 spikes per line were inoculated at anthesis by single floret inoculation with about 1,000 conidiospores. Following inoculation, each spike was covered with a plastic bag for 24 h. Misting was applied every 3 h to maintain the moisture. Data were collected 15–20 days post-inoculation. We found that the visible symptoms of scab often spread faster along the rachides from the inoculated floret than among the spikelets. Consequently, the number of diseased spikelets (NDS) and the length (in centimeters) of diseased rachides (LDR) were investigated to evaluate Type II resistance.

Marker analysis

DNA was extracted according to Ma and Sorrells (1995). The parents were surveyed for polymorphism using randomly amplified polymorphic DNA (RAPD) primers and simple sequence repeat (SSR) markers, including those of the gwm series (Röder et al. 1998), the barc series (published by P. Cregan's laboratory, http://www.ba.ars.usda.gov/research/) and the wmc series designed from expressed sequence tags (Gao et al. 2003). Marker S1021m was developed by our laboratory from a SSR derived from a RAPD sequence. SSR amplification was performed following the procedure of Röder et al. (1998). The polymorphism detected was screened in the RIL population. The linkage map was constructed using MAPMAKER MACINTOSH v2.0 (Lander et al. 1987), and a LOD of 3.0 was used to construct the framework map. Additional markers were placed into the maps by the TRY command with a LOD = 1.0. The recombination fractions were converted into map distances (centiMorgans) using the Kosambi mapping function (Kosambi 1944). The linkage groups were tentatively assigned to chromosomes based on the marker locations in the maps developed by Röder et al. (1998) and by Cregan's laboratory when such marker information was available. Chinese Spring (C.S.) nulli-tetrasomics were used to confirm the chromosomal locations of markers for putative QTL.

QTL analysis

Analysis of variance (ANOVA) was conducted using statistical software DATA DESK v.5.0 (Data Description, Ithaca, N.Y.). Heritability was estimated using the formula $h^2 = \sigma_g^2/(\sigma_g^2 + \sigma_e^2)$, where σ_g^2 and σ_e^2 were estimated using the mean square value from ANOVA. One-way ANOVA of each marker was performed to detect loci associated with scab resistance with a significant level of P=0.01. Before interval mapping analysis, data showing skewed distribution were log- or square-root-transformed. Simple interval mapping (SIM) was carried out using MAPMAKER/QTL v.1.9 (Lander and Botstein 1989). The significance threshold was set at LOD=2.0 to declare a putative QTL. Once putative QTL peaks were detected through the whole map scan, the one with the highest LOD score was fixed at the given QTL position using the SEQUENCE command for a second round of whole map scan in order to identify other putative QTL given the presence of this QTL (fitting a two-QTL model). This strategy is similar to the composite interval mapping (CIM) with one background marker in model 6 of QTL CARTOGRAPHER (Basten et al. 1994). In the

second scan, a new QTL was declared when the log-likelihood of the two-QTL model (the new one plus the fixed one) was 1.5 higher than that of the single fixed QTL.

Results

Parent polymorphism and map construction

Of the 654 SSR markers surveyed, 326 (49.8%) identified polymorphism between the parents, corresponding to 402 loci (33.9% of the total loci detected by all of the SSR markers). Paillard et al. (2004) reported that 61% of the SSR markers were polymorphic between winter cultivars Arina and Frono, and up to 80% of the SSR primer pairs detected polymorphism between Opata 85 and the synthetic wheat W7984 (Röder et al. 1998). These results suggest that the polymorphism level between Wangshuibai and Nanda2419 is low, even though they have different geographic origins. With the exception of some of the wmc markers, all of the other markers used can be assigned to individual chromosomes or groups based on previous mapping information. The polymorphic levels varied greatly among different chromosomes or groups. Groups 4 and 6 were much less polymorphic than the other groups: of the 31 and 36 markers surveyed, only 11 (35.5%) and 17 (47.2%) SSR markers were polymorphic between the parents, respectively. For groups 1, 2, 3, 5 and 7, the percentage of polymorphic markers was 61.5%, 56.7%, 60.9%, 50% and 71.1%, respectively. On average, 55.4% of the genomic DNA-derived SSR markers and 44.1% of the EST-derived SSR markers detected polymorphism between the parental lines, indicating that the EST-derived markers are less polymorphic.

Of the polymorphic loci detected between the parents, 300 were mapped in the RIL population. Cases of heterozygosity were found for almost every polymorphic marker but usually only in less than 3% of the RILs. These cases were regarded as missing data in the map construction. Two hundred and sixty-six of the mapped loci were assigned to 40 linkage maps, covering 2,210 cM of the wheat genome. The remaining were unlinked. Only five adjacent intervals distributed in different linkage groups had a distance over 25 cM, and the largest one was 33.1 cM (Table 1). Six linkage groups with a total of 103 cM could not be related to a specific chromosome because of the existence of homologous loci for some SSR markers or due to the lack of previous mapping information. When linkage groups belonging to the same chromosomes were merged, we noticed that the total genetic distances mapped for the individual chromosomes varied greatly (Table 1). Maps of chromosomes 1A, 1B, 2A, 2B, 2D, 3B, 4B, 5B, 5D , 6B, 7A and 7B had the best coverage, each being over 80 cM. No marker linkage was established for chromosome 6A, and the linkage distances for chromosomes 1D and 6D were less than 10 cM. In a homoeologous group-wise framework, group 4 and group 6 were poorly represented, which is consistent with their polymorphism level in this population.

Phenotypic analysis

In the replicated trial in 2002, the between-line variation was highly significant (P=0.0004 for NDS and P=0.0009 for LDR), but the variation between the replicates was not. The broadsense heritability estimated for the two traits using 2002 data was 34% for NDS and 31.4% for LDR.

The 2002 NDS data displayed a distribution skewed toward Wangshuibai (Fig. 1a) with a mean of 1.84 and a difference between the maximum and the minimum (range) of 3.77. The 2003 data were similar to those of 2002 with respect to distribution, and the correlation across the years was significant at P<0.0001 but with a coefficient of only 0.39. The LDR data showed a double-peak like distribution in both years, implying that LDR should be controlled by a major gene (Fig. 1b). The turning points between the two peaks fell at 1.7 in 2002 and 1.6 in 2003. The correlation coefficient between the 2 years of data was 0.446 (P<0.0001).

The NDS and LDR were highly correlated with each other. The correlation coefficients between them were 0.707 in 2002 and 0.690 in 2003 (P<0.0001).

Table 1 Chromosome coverage of the linkage groups established. Linkage groups assigned to the same chromosomes were added together

Chromosome	Number of mapped loci	Total linkage distance (cM)	Maximum adjacent interval distance (cM)
1A	16	129	20.7
1B	22	231	27.3
1D	2	7	6.9
2A	14	85	17.1
2B	24	219	21.3
2D	15	138	33.1
3A	4	37	16.6
3B	30	273	23.1
3D	6	65	23.6
4A	2	17	17.3
4B	17	101	18.6
4D	2	12	12.1
5A	7	59	17.7
5B	16	125	16.8
5D	21	214	30.8
6A	0	0	-
6B	11	80	21.2
6D	2	2	2.1
7A	15	146	19.5
7B	16	91	20.7
7D	8	76	25.9

Table 2 Markers associated with number of diseased spiklets (NDS) and length of diseased rachides (LDR) in one-way ANOVA, the phenotypic variation explained by them and symbols assigned to corresponding QTL

Markers	Source of resistance allele[a]	NDS				LDR				QTL symbol
		2002[b]		2003[b]		2002[b]		2003[b]		
		P	R^2 (%)	*P*	R^2 (%)	*P*	R^2 (%)	*P*	R^2 (%)	
Xs1021m	N	0.0019	8.6	0.0083	5.9	0.0008	9.9	*0.0444*	*3.5*	*QFhs.nau-2B1*
Xwmc532	W	0.0053	7.3	–	–	*0.015*	*5.6*	–	–	*QFhs.nau-3A*
Xwmc054-1	W	*0.0489*	*3.5*	*0.0488*	*3.3*	0.0039	7.4	–	–	*QFhs.nau-3B1*
Xgwm533-3	W	0.005	7.1	0.0016	8.6	0.0001	13.4	0.0018	8.4	*QFhs.nau-3B2*
Xwmc539	W	0.0075	5.9	0.0006	10.9	*0.0157*	*5.9*	0.0001	13.6	*QFhs.nau-6B*
Xgwm469	N	–	–	0.0089	6.1	0.0059	6.9	–	–	*QFhs.nau-6D*

[a]W, Wangshuibai; N, Nanda2419
[b]Probability values around 0.05 in one or more cases (trait or year) but less than 0.01 in other cases are listed in italics

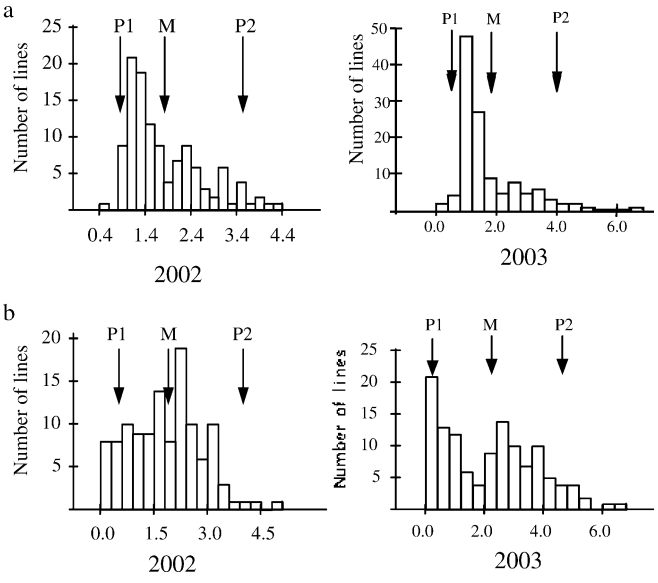

Fig. 1 Distribution of number of diseased spiklets (NDS) in 2002 and 2003 (**a**) and length of disease rachides (LDR) in 2002 and 2003 (**b**). *P1* Wangshuibai, *P2* Nanda2419, *M* average value of the trait

One-way ANOVA analysis

Number of diseased spiklets

Three chromosome regions derived from Wangshuibai and two from Nanda2419 showed significant association with NDS. Table 2 lists the markers with the highest probability of significance and the fraction of phenotypic variation explained by them. The QTL represented by *Xs1021m* on chromosome 2B, *Xwmc539* on chromosome 6B and *Xgwm533-3* were detected in both years and had the largest effects in 1 year or 2 years. *Xgwm533-3* was assigned to chromosome 3BS based on the locations of markers linked to it in other maps. This localization was further confirmed by nulli-tetrasomic analysis. The Wangshuibai alleles of *Xgwm533-3* and *Xwmc539* reduced the NDS up to 31% and 37.2%, respectively (Table 3).

Length of diseased rachides

All five markers associated with NDS, except for *Xwmc532*, were also related to LDR in one or both years. *Xwmc532* was significant in 2002 at *P*=0.05 (Table 2). In addition, the chromosome region represented by *Xwmc054-1*, which showed association with LDR in 2002, was also significant for NDS in both 2002 and 2003 at *P*=0.05. Wangshuibai contributed the resistance allele of this QTL. The regions with the highest effects were represented by *Xgwm533-3* on chromosome 3BS and *Xwmc539* on chromosome 6B, explaining up to 13.6% of the phenotypic variation. This 3BS QTL expressed more stably in the 2-year period and caused more than 34% reduction of the LDR (Table 3).

The results show that chromosomal regions with relatively large effects on NDS and LDR were consistently detected. The QTL identified were named as shown in the

Table 3 Average effects of alternative alleles of two significant loci as evaluated in the Nanda2419 × Wangshuibai population (*W* Wangshuibai allele, *N* Nanda2419 allele)

Marker	Location	2002				2003			
		W	N	Additive value	Reduction (%)	W	N	Additive value	Reduction (%)
NDS									
Xgwm533-3	3B	1.62	2.08	-0.23	22.1	1.49	2.16	0.33	31.0
Xwmc539	6B	1.49	1.96	-0.24	24.1	1.24	1.98	0.37	37.2
LDR									
Xgwm533-3	3B	1.48	2.27	-0.40	34.8	1.81	2.78	0.48	34.7
Xwmc539	6B	1.42	1.92	-0.25	26.1	1.34	2.52	0.59	46.8

last column of Table 2. In total, four QTL were derived from resistance alleles from Wangshuibai and two from Nanda2419.

Interval mapping analysis

The QTL intervals found for both traits covered almost all regions identified in the one-way ANOVA (Table 4, Fig. 2). When the QTL with the largest effect from each set of data was fixed, additional QTL intervals were detected by the second scanning (Table 4).

Using the 2003 data, we mapped *Qfhs.nau-6B* for NDS and LDR at the position 4 cM proximal to *Xwmc539* in the interval of *Xwmc539–Xbarc024* and at *Xgwm644*, respectively, both explaining the highest fraction of phenotypic variation. The distance between the two loci was 13.7 cM (Fig. 2). Using the 2002 data, we detected a QTL for LDR by CIM and mapped it at 1 cM proximal to *Xwmc398* in the 2.1-cM interval of *Xwmc398–Xgwm644*, implying that the same QTL was detected with the two methods.

The *Qfhs.nau-3B2* for NDS was positioned at 3 cM proximal to *Xgwm533-3* in the 9.0-cM interval of *Xgwm533-3–Xgwm533-1* using the 2002 data and at 8 cM proximal to *Xgwm533-1* using the 2003 data. These two positions were 14 cM away from each other. But when the chromosome 6B QTL was fixed, *Qfhs.nau-3B2* in 2003 was re-positioned at the same position as in 2002. The *Qfhs.nau-3B2* for LDR was mapped at 7 cM proximal to *Xgwm493* in the 9.3-cM interval of *Xgwm493–Xgwm533-3* using the 2002 data and at 2 cM proximal to *Xgwm533-3* using the 2003 data. The latter QTL was re-mapped at the position corresponding to that of 2002 (Table 4). Thus, the distance of the QTL peaks for these two traits were concluded to be 5.3 cM. When both the *Qfhs.nau-3B2* and *Qfhs.nau-2B1* QTL were fixed, *Qfhs.nau-3B1* was detected using the 2002 LDR data in a three-QTL model (data not shown).

The *Qfhs.nau-2B1* identified by one-way ANOVA was detected through either SIM or CIM using 2002 data and fallen in the interval of *Xs1021m-Xgwm47-1*. The peak positions were within the distance of 2.5 cM, sharing the same one-log confidence interval. In CIM analysis, this interval also showed association with 2002 NDS when the LOD differential was set at 1.2.

Through CIM, a QTL was mapped on 1B at 8 cM proximal to *Xgwm018* in the interval of *Xgwm018-Xbarc181* for NDS and at 3 cM proximal to *Xwmc419* in the interval of *Xwmc419-Xgwm018* for LDR. They were 17.9 cM from each other (Table 4, Fig. 2), but overlapped in the one-log support confidence intervals. In addition, new QTL were mapped for NDS at *Xwmc113* on 5B, and for LDR at 10 and 11 cM proximal to *Xgwm383-2* on 3D and to *Xwmc322* on 3A, respectively. This 3A QTL was not linked to the one associated with *Xwmc532*.

Table 4 QTL detected by interval mapping

Traits		Methods of QTL detection[a]	QTL[b]	Interval	Source of resistance allele	Location	Length (cM)	Peak position (cM)[c]	LOD[d]	R^2 (%)[e]
NDS	2002	SIM	*Qfhs.nau-3B2*	*Xgwm533-3–Xgwm533-1*	W	3B	9.0	*Xgwm533-3*+3¶	2.6	13.6
		CIM	*Qfhs.nau-5B*	*Xwmc113–Xgwm544*	W	5B	10.0	*Xwmc113+0*	1.9	8.1
			Qfhs.nau-2B1	*Xs1021m-Xgwm47-1*	N	2B	2.5	*Xs1021m+0*	1.9	7.0
			Qfhs.nau-1B	*Xgwm018-Xbarc181*	W	1B	11.8	*Xgwm018*+8	1.7	7.6
	2003	SIM	*Qfhs.nau-3B2*	*Xgwm533-1–Xbarc147-1*	W	3B	14.0	*Xgwm533-1*+8	2.4	13.3
			Qfhs.nau-6B	*Xwmc539–Xbarc024*	W	6B	5.7	*Xwmc539*+4¶	4.7	17.8
		CIM	*Qfhs.nau-3B2*	*Xgwm533-3–Xgwm533-1*	W	3B	9.0	*Xgwm533-3*+3	1.5	5.6
LDR	2002	SIM	*Qfhs.nau-2B1*	*Xs1021m-Xgwm47-1*	N	2B	2.5	*Xs1021m+0*	2.2	8.6
			Qfhs.nau-3B2	*Xgwm493-Xgwm533-3*	W	3B	9.3	*Xgwm493*+7¶	3.9	17.4
		CIM	*Qfhs.nau-3A*	*Xwmc322-Xwmc153*	W	3A	24.0	*Xwmc322*+11	1.5	11.3
			Qfhs.nau-2B1	*Xs1021m-Xgwm47-1*	N	2B	2.5	*Xs1021m*+1	2.4	8.0
			Qfhs.nau-6B	*Xwmc398–Xgwm644*	W	6B	2.1	*Xwmc398*+1	1.5	4.5
	2003	SIM	*Qfhs.nau-3B2*	*Xgwm533-3-Xgwm533-1*	W	3B	9.0	*Xgwm533-3*+2	2.4	10.8
			Qfhs.nau-6B	*Xgwm644–Xwmc105*	W	6B	5.2	*Xgwm644+0*¶	5.4	19.5
			Qfhs.nau-2B2	*Xwmc154–Xgwm429*	N	2B	22.4	*Xwmc154*+9	2.2	14.9
		CIM	*Qfhs.nau-3B2*	*Xgwm493–Xgwm533-3*	W	3B	9.3	*Xgwm493*+7	2.5	9.0
			Qfhs.nau-1B	*Xwmc419–Xgwm018*	W	1B	12.9	*Xwmc419*+3	1.7	7.1
			Qfhs.nau-2B2	*Xwmc154–Xgwm429*	N	2B	22.4	*Xwmc154*+7	1.5	9.5
			Qfhs.nau-3D	*Xgwm383-2–Xgwm664*	W	3D	10.3	*Xgwm383-2*+10	1.5	6.6

[a]SIM and CIM, simple and composite interval mapping, respectively with MAPMAKER/QTL
[b]Quantitative trait loci that overlap in the one-log support confidence intervals are assigned the same symbol
[c]The position is represented by the left boundary locus of the interval plus a genetic distance (in centiMorgans) proximal to it. The fixed QTL position for each CIM is indicated by¶
[d,e] The LOD and R^2 values for the QTL from CIM were derived from the corresponding values of the two-QTL model minus the corresponding values of the fixed one

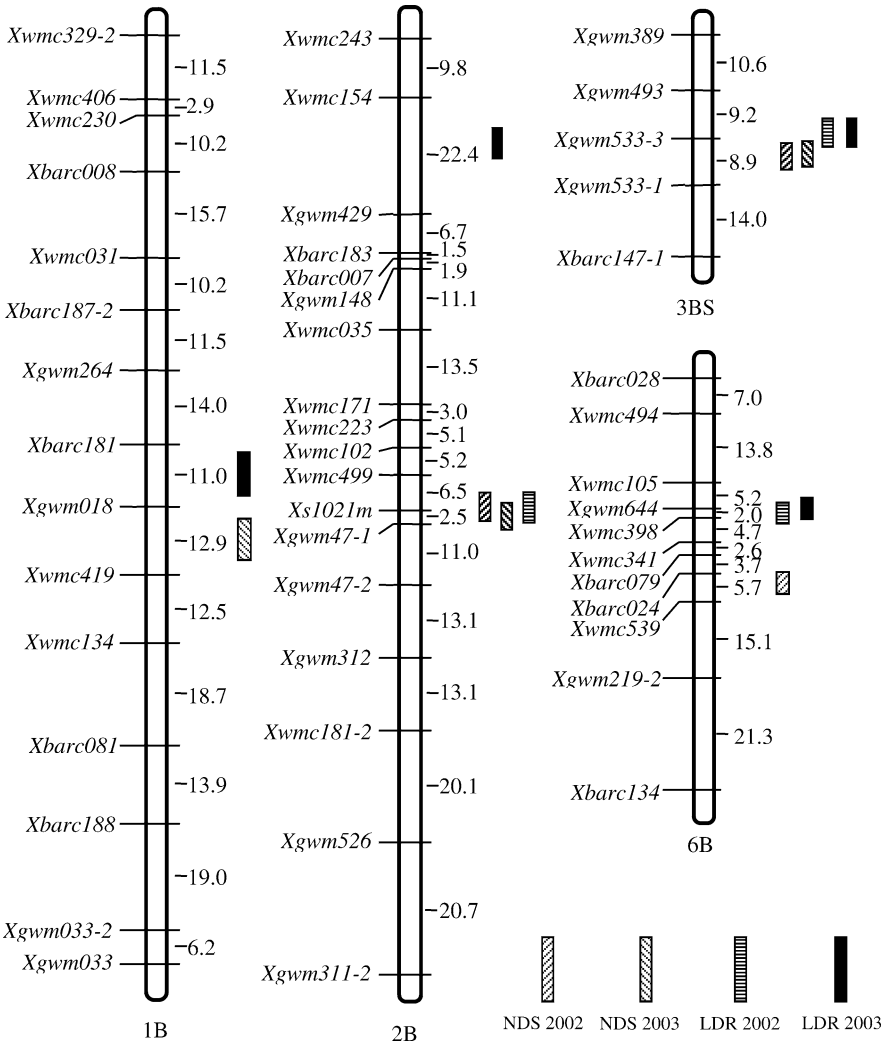

Fig. 2 The locations of QTL detected through interval mapping. The *numbers* to the right are map distances in centiMorgans. The QTL peak regions are indicated with *bars*

The multiple regression model using QTL derived from CIM explained 31.9% and 23.4% of the phenotypic variation for NDS, and 34.4% and 46.8% of the phenotypic variation for LDR in 2002 and 2003, respectively.

Discussion

The percentage of infected spikelets has often been used to evaluate the resistance to the spread of scab. Since the average length of spikes and the number of spikelets in different lines of our population varied greatly, we chose to use the absolute values of NDS and LDR as the parameters to represent Type II resistance. These two traits were highly related. We were able to demonstrate in the present investigation that both of them served our goal well.

To map QTL for Type II scab resistance, we first identified primary QTL by means of SIM. Through the second round of the whole map scan, which we performed by fixing the QTL with the highest LOD score using the SEQUENCE command, new QTL were identified, such as *Xgwm018+8* for NDS and *Xwmc419+3* for LDR. In addition, through the second round scan, the reproducibility and position accuracy of some QTL were increased. For instance, *Qfhs.nau-6B* for LDR mapped using the 2002 data was at a position within 1.1 cM of that mapped using the 2003 data, and *Qfhs.nau-3B2* for NDS mapped using the 2002 data was at the same position as that mapped using the 2003 data. These results could not be achieved simply by SIM and by using the 2-year average in mapping (data not shown). The implication was that the expressivity of some QTL varied in different years. By using the composite mapping strategy, we effectively overcame at least part of this complexity.

Among the eight QTL associated with Type II resistance identified through interval analysis, *QFhs.nau-2B1*, *QFhs.nau-3B2* and *QFhs.nau-6B* were the most consistent across the 2 years and between the traits. The peaks of the QTL intervals corresponding to the two traits—for example *QFhs.nau-3B2* and *QFhs.nau-6B*—were not always at the same position, but they were closely linked to each other and shared or overlapped the one-log confidence intervals. Further studies are needed to clarify if different genes control these two traits. On chromosome 3BS, marker gwm533 has three polymorphic

loci, two of which correspond to the SSR map of Röder et al. (1998). *Xgwm533-3* and *Xgwm533-1* were closely linked to each other and to *QFhs.nau-3B2*. *Xwmc054-1*, linked to the less prominent *QFhs.nau-3B1* (Table 2), was 10 cM from *Xgwm533-2*. In the wheat ITMI map, *Xgwm533-1* and *Xgwm533-2* are 67 cM from each other on chromosome 3B (Röder et al. 1998). We were not able to fill the gap between these two chromosome regions with the markers used, likely because of the lack of polymorphism. Except for the chromosome 6D and chromosome 2B QTL, all others have their alleles favoring resistance contributed by Wangshuibai.

Two-way interaction analysis for each pair of the QTL was performed, but no significant epistatic interaction was revealed in this study (data not shown). However, the epistatic effects could be important for scab resistance. Increasing the population size may help clarify this issue.

Quite a number of QTL, mainly in Sumai No. 3 and its derived lines, have been reported for scab resistance. These have been mapped to more than a dozen different chromosomes, including 1B, 2A, 2B, 2D, 3A, 3B, 3D, 4B, 5A, 5D, 6A, 6B and 6D. The QTL that fell in the interval of *Xgwm493–Xgwm533-1* on chromosome 3B and linked to *Xgwm644* on chromosome 6B have been detected in CM-82036 (the 3BS QTL), ND2603, Ning894037 and Sumai No. 3 and had the largest effects (Anderson et al. 2001; Buerstmayr et al. 2002; Shen et al. 2003b). However, an additional locus, *Xgwm533-3*, has been mapped between *Xgwm493* and *Xgwm533-1* in Wangshuibai (Fig. 2). Polymorphism in this region has also been reported by Bai et al. (2003) and Liu and Anderson (2003) between Sumai No. 3 and various scab-resistant germplasms, including Wangshuibai. Thus, the allelism of this 3BS QTL from different germplasms deserves confirmation. *QFhs.nau-2B1* linked to *Xs1021m–Xgwm047* was consistently identified in our population. In Ning7840, there was a QTL linked to *Xgwm120* on chromosome 2B (Zhou et al. 2002), which was about 18 cM from *Xgwm47* (Röder et al. 1998). The second chromosome 2B QTL linked to *Xgwm429* was in the nearby region of *Xgwm374*, which was associated with scab resistance in Renan (Gervais et al. 2003) and about 45 cM from *Xgwm47*. The chromosome 1B QTL detected in this study can be related to the one linked to *Xbarc8* in F201R by Shen et al. (2003a) on the basis of their common association with *Xgwm018*, but the former was less consistent in different environments. The locus *Xgwm383* linked to the chromosome 3D QTL has also been linked to a QTL on chromosome 3B in cv. Renan (Gervais et al. 2003). Two minor QTL on chromosome 3A have been detected in Wangshuibai. Even though the *Xwmc532*-linked QTL in Wangshuibai, detected by one-way ANOVA, was not verified by interval mapping and only appeared in 2002, its mapping position showed coincidence with the chromosome 3AS QTL linked to *Xgwm2* reported by Otto et al. (2002). Gervais et al. (2003) identified a QTL on chromosome 3AL linked to *Xbcd372* in Récital. In Frontana, a chromosome 3A QTL was identified in the region likely between *Xgwm2* and *Xgwm155* (Steiner et al. 2004). Gupta et al. (2000) also reported QTL on chromosomes 3AL and 3AS in Ning7840. *Xgwm469* on chromosome 6D showed an association with scab resistance in both years in the one-way ANOVA analysis, however, interval mapping was not done because no linkage map was available. Resistance QTL on chromosome 6D have been reported by Gervais et al (2003) and Paillard et al (2004). Unfortunately, the positions of these QTL could not be cross-referenced due to the lack of common markers. The coincidence in locations of QTL found in different studies could imply a narrow genetic basis for scab resistance in common wheat. Consequently, comprehensive comparative studies on the resistance in different germplasms are necessary in order to gain an understanding of the genetics of scab resistance.

Nanda2419 was selected from Italian wheat var. *Mentana*, which is one of the crossing parents of the Brazilian scab resistance germplasm Frontana. Akagomughi is also in Mentana's pedigree. Thus, Akagomughi serves a pivot to link Sumai No. 3, whose parents are Funo and Taiwain Xiaomai, Frontana and Nanda2419 together. However, the three resistance QTL found for Nanda2419 have not been reported in Sumai No. 3. Since Funo, Nanda2419 and Taiwan Xiaomai are all susceptible to scab, the identification of QTL in susceptible parents or their lines proves the importance of exploiting transgressive segregation in scab resistance breeding.

Wangshuibai is an excellent source of both Type I and Type II resistance. In this study, QTL related to Type II resistance were found. Wangshuibai seems to possess more genes conferring resistance to the spreading of scab than Sumai No. 3. We are expanding the population to up to 500 lines to more precisely characterize these QTL loci. The markers identified as showing an association with scab resistance are all SSR markers and can be easily applied in breeding programs. Therefore, their availability will facilitate the exploitation of scab resistance genes in Wangshuibai.

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References

Anderson JA, Stack RW, Liu S, Waldron BL, Field AD, Coyne C, Moreno-Sevilla B, Mitchell Fetch J, Song QJ, Cregan PB, Frohberg RC (2001) DNA markers for *Fusarium* head blight resistance QTL in two wheat populations. Theor Appl Genet 102:1164–1168

Arthur JC (1891) Wheat scab. Ind Agric Exp Stn Bull 36:129

Bai GH, Kolb FL, Shaner G, Domier LL (1999) Amplified fragment length polymorphism markers linked to a major quantitative trait locus controlling scab resistance in wheat. Phytopath 89:343–348

Bai GH, Guo PG, Kolb FL (2003) Genetic relationships among head blight resistant cultivars of wheat assessed on the basis of molecular markers. Crop Sci 43:498–507

Ban T, Suenaga K (2000) Genetic analysis of resistance to *Fusarium* head blight caused by *Fusarium graminearum* in Chinese wheat cultivar Sumai 3 and the Japanese cultivar Saikai 165. Euphytica 113:87–99

Basten CJ, Weir BS, Zeng ZB (1994) ZMAP—a QTL cartgrapher. In: Smith C, Gavora JS, Benkel B, Chesnais J, Fairfull W, Gibson JP, Kennedy BW, Burnside EB (eds) 5th World Congr Genet Applied Livestock Prod. Comput Strategies Software vol 22, pp 65–66http://statgen.ncsu.edu/qtlcart/cartographer/

Buerstmayr H, Lemmens M, Hartl L, Doldi L, Steiner B, Stierschneider M, Ruckenbauer P (2002) Molecular mapping of QTL for *Fusarium* head blight resistance in spring wheat. I. Resistance to fungal spread (Type II resistance). Theor Appl Genet 104:84–91

Buerstmayr H, Steiner B, Hartl L, Griesser M, Angerer N, Lengauer D, Miedaner T, Schneider B, Lemmens M (2003) Molecular mapping of QTL for *Fusarium* head blight resistance in spring wheat. II. Resistance to fungal penetration and spread. Theor Appl Genet 107:503–508

Del Blanco IA, Frohberg RC, Stack RW, Berzonsky WA, Kianian SF (2003) Detection of QTL linked to *Fusarium* head blight resistance in Sumai 3-derived North Dakota bread wheat lines. Theor Appl Genet 106:1027–1031

Gao L, Tang F, Li H, Jia J (2003) Analysis of microsatellites in major crops assessed by computational and experimental approaches. Mol Breed 12:245–261

Gervais L, Dedryver F, Morlais JY, Bodusseau V, Negre S, Bilous M, Groos C, Trottet M (2003) Mapping of quantitative trait loci for field resistance to *Fusarium* head blight in an European winter wheat. Theor Appl Genet 106:961–970

Gilbert J, Tekauz A (2000) Review: recent developments in research on *Fusarium* head blight of wheat in Canada. Can J Plant Pathol 22:1–8

Gupta A, Lipps PE, Campbell KG (2000) Finding quantitative trait loci associated with *Fusarium* head blight of wheat using simple sequence repeat markers. In: Ward RW et al. (eds) Proc 2000 Natl Fusarium Head Blight Forum. Michigan State University, East Lansing, pp38–32

Kosambi DD (1944) The estimation of map distances from recombination values. Ann Eugen 12:172–175

Lander ES, Botstein D (1989) Mapping Mendelian factors underlying quantitative traits using RFLP linkage maps. Genetics 121:185–199

Lander ES, Green P, Abrahamson J, Barlow A, Daley MJ, Lincoln SE, Newburg L (1987) MAPMAKER: an interactive computer package for constructing primary genetic linkage maps of experimental and natural populations. Genomics 1:174–181

Li BQ, Liu FL, Xu RC, Huang C, Cheng FS, Liu JY, Meng J, Mou JM (2000) Sumai No. 3: its development, genetic characteristics, and applications in wheat breeding for *Fusarium* head blight. In: Raupp J, Ma Z, Chen P, Liu D (eds) Proc Int Symp Wheat Improve Scab Resistance. KSU Printing Services, Manhattan, Kan., pp 187–193

Liu SX, Anderson JA (2003) Marker assisted evaluation of *Fusarium* head blight resistant wheat germplasm. Crop Sci 43:760–766

Liu ZZ, Wang ZY (1991) Improved scab resistance in China: sources of resistance and problems. In: Saunders DA (ed) Wheat for the nontraditional warm areas. Proc Int Conf. CIMMYT, Mexico, DF, pp 178–188

Ma ZQ, Sorrells ME (1995) Genetic analysis of fertility restoration in wheat using restriction fragment length polymorphisms. Crop Sci 35:1137–1143

McMullen M, Jones R, Gallenberg D (1997) Scab of wheat and barley: a re-emerging disease of devastating impact. Plant Dis 81:1340–1348

Mesterhazy A (1995) Types and components of resistance to *Fusarium* head blight of wheat. Plant Breed 114:377–386

Miller JD, Young JC, Sampson DR (1985) Deoxynivalenol and *Fusarium* head blight resistance in spring cereals. J Phytopath 113:359–367

Otto CD, Kianian SF, Elias EM, Stack RW, Joppa LR (2002) Genetic dissection of a major *Fusarium* head blight QTL in tetraploid wheat. Plant Mol Biol 48:625–632

Paillard S, Schnurbusch T, Tiwari R, Messmer M, Winzeler M, Keller B, Schachermayr G (2004) QTL analysis of resistance to *Fusarium* head blight in Swiss winter wheat (*Triticum aestivum* L.). Theor Appl Genet (in press)

Parry DW, Jenkinson P, McLeod L (1995) *Fusarium* ear blight (scab) in small grain cereals-a review. Plant Pathol 44:207–238

Röder MS, Korzun V, Wendehake K, Plaschke J, Tixier MH, Leroy P, Ganal MW (1998) A microsatellite map of wheat. Genetics 149:2007–2023

Saur L (1991) Sources of resistance to head blight caused by *Fusarium culmorum* in bread wheat and related species. Agronomie 11:535–541

Schroeder HW, Christensen JJ (1963) Factors affecting resistance of wheat to scab caused by *Gibberella zeae*. Phytopath 53:831–838

Shen X, Ittu M, Ohm H (2003a) Quantitative trait loci conditioning resistance to *Fusarium* head blight in wheat line F201R. Crop Sci 43:850–857

Shen X, Zhou M, Lu W, Ohm H (2003b) Detection of *Fusarium* head blight resistance QTL in a wheat population using bulked segregant analysis. Theor Appl Genet 106:1041–1047

Snijders CHA (1990) Genetic variation for resistance to *Fusarium* head blight in bread wheat. Euphytica 50:171–179

Steiner B, Lemmens M, Griesser M, Scholz U, Schondelmaier J, Buerstmayr H (2004) Molecular mapping of resistance to *Fusarium* head blight in the spring wheat cultivar Frontana. Theor Appl Genet 109:215–224

Waldron BL, Moreno-Sevilla B, Anderson JA, Stack RW, Frohberg RC (1999) RFLP mapping of QTL for *Fusarium* head blight resistance in wheat. Crop Sci 39:805–811

Wang YZ (1996) Epidemiology and management of wheat scab in China. In: CIMMYT (ed) *Fusarium* head scab: global status and future prospects. CIMMYT, El Batan, pp 97–105

Zhou WC, Kolb FL, Bai GH, Shaner G, Domier LL (2002) Genetic analysis of scab resistance QTL in wheat with microsatellite and AFLP markers. Genome 45:719–727