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Integration of QTL detection and marker assisted selection for improving resistance to Fusarium head blight and important agronomic traits in wheat

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ABSTRACT

Fusarium head blight (FHB), caused by *Fusarium graminearum*, is one of the most destructive wheat (*Triticum aestivum* L.) diseases worldwide. Identification of quantitative trait loci (QTL) conferring FHB resistance followed by marker assisted selection (MAS) is an efficient approach to breed FHB-resistant varieties. In this study, 38 additive QTL and 18 pairs of epistatic QTL for FHB resistance were detected in four environments using a population of recombinant inbred lines (RILs) derived from varieties Neixiang 188 and Yanzhan 1. Six QTL clusters were located on chromosomes 2D, 4B, 4D, 5A, 5D and 7B, suggesting possible polytrophic functions. Six elite lines with good FHB resistance and agronomic traits were selected from the same population using the associated markers. Our results suggest that MAS of multiple QTL will be effective and efficient in wheat breeding.

1. Introduction

Fusarium head blight (FHB), caused by *Fusarium graminearum* Schwabe, is a common disease in wheat (*Triticum aestivum*) and barley (*Hordeum vulgare*), that causes yield losses and threatens human health [1–3]. Due to global warming and agronomic practices, such as irrigation and retained stubble that may carry the pathogen, FHB has become more frequent and more severe in recent years. The disease has gradually extended to the northern major wheat production areas of China. [4] In the Yangtze River valley and Northeast Spring Wheat Zone, FHB regularly causes 10%–15% of yield losses, and nearly 50% in epidemic years [5].

Resistant varieties play an important role in controlling FHB. However, there are relatively few resistance genes used in wheat breeding in China. FHB resistance is a quantitative trait controlled by major and minor genes [3,6–10] located on all wheat chromosomes, except 7D [9]. Chinese variety Sumai 3,

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which carries the major resistance QTL *Fhb1*, is widely recognized as the best resistance source and is extensively used in wheat breeding programs worldwide [6,11–14].

Marker assisted selection (MAS) is a promising method for breeding in the genomics era. Thousands of QTL and genes conferring traits of agronomic importance have been identified in major crops, and these can be used to accelerate MAS. At present, QTL detection and functional analysis are separate from MAS. Many molecular markers for targeting genes/loci are not useful during the selection process because of low polymorphism across different genetic backgrounds and incomplete association with target traits. In this study, we attempted to select promising breeding lines with FHB resistance and good agronomic traits by combining QTL analysis and MAS. In a recombinant inbred line (RIL) population derived from cultivars Yanzhan 1 (YZ1) and Neixiang 188 (NX188) FHB resistance and other important agronomic traits were simultaneously selected using molecular markers, and several elite lines were produced.

2. Materials and methods

2.1. Plant materials

One hundred and ninety nine $F_{7:8}$ RILs were developed by single-seed descent from the cross YZ1 × NX188. YZ1 is an early maturing cultivar released in Henan Province of China, in 2000; NX188, a high yielding cultivar with wide adaptation and released in 2000, was the fourth most widely planted cultivar in China (470,000 ha) in 2004.

2.2. Evaluation of agronomic traits

The RILs and their parents were planted in Beijing and in Luoyang, Henan province, in the 2003–2004 and 2004–2005 wheat seasons. All lines were phenotyped as single relicates in four environments. Thirty seeds of each line were sown in a two-row plot of 2 m in length. Plant height (PH) was measured in the field at maturity. Spike length (SL), spikelet number per spike (SPI), spike compactness (SC, SC = SPI/SL), grain number per spike (GNS), and thousand-grain weight (TGW) were measured after harvest.

2.3. FHB evaluation

FHB responses were assayed under natural conditions in the 2005–2006 and 2006–2007 cropping seasons in Jianyang, Fujian province. Although no wheat is commercially produced in the area extremely severe FHB infections are common. Field management was the same as that for agronomic evaluations. Sumai 3, Mianyang 26, and Yangmai 5 were used as the resistant, susceptible, and moderately susceptible controls, respectively.

About 15 and 20 days after flowering, 30 spikes of each line were randomly selected. FHB severity in each spike was classified into five grades of symptoms on spikelets and spike rachi: 0 for no incidence on spikelets and spike rachis, 1 for ratio of incidence on spikelets less than 1/4 and no incidence on the rachis, 2 for ratio of incidence on spikelets between 1/4 and 1/2 and no incidence on the rachis, 3 for ratio of incidence on spikelets between 1/2 and 3/4 and incidence on spike rachis, 4 for ratio of incidence on spikelets of more than 3/4 or dead spikelets. [15], FHB disease index (DI) of each line was calculated as follows: DI = (Σ severity score of an individual spike × number of spikes)/(the highest severity score × total number of spikes).

The FHB resistance score of a genotype was graded by DI from 1 to 7 [15,16], representing R, R–MR, MR, MR–MS, MS, MS–S and S, respectively. Where R = resistant, S = susceptible, and M = moderate disease.

2.4. Construction of a linkage map and QTL analyses

A total of 328 publicly available SSR and DArT markers were mapped on 25 linkage groups (http://wheat.pw.usda.gov/GG2/index.shtml) [17] covering a total genetic distance of 3848.2 cM and providing partial linkage groups for all chromosomes. QTL for agronomic traits and FHB resistance were analyzed separately. Composite interval mapping (CIM) was performed using QTLNetwork 2.0 software [18] on the individual line means in order to detect additive QTL, epistatic QTL, and QTL × environment interaction (QE). QTL nomenclature followed the protocols of McIntosh et al. [19], in which the research institution is abbreviated as "caas" (Chinese Academy of Agricultural Sciences).

3. Results

3.1. FHB response and agronomic traits

Consistent FHB responses of both parents and RILs were observed during the 2005–2006 and 2006–2007 cropping seasons, and the correlation coefficient was 0.56 ($P < 0.01$). NX188 had a significantly lower DI and resistance score than YZ1. FHB DI and resistance scores for the RIL population showed a continuous distribution with transgressive segregation, particularly, some lines exhibiting higher resistance than the resistant parent (Table 1).

The frequency distributions for six agronomic traits were continuous with broad variation and transgressive segregation in all environments (Table 1).

3.2. QTL for FHB resistance and agronomic traits

A total of 38 additive and 18 epistatic QTL for FHB and agronomic traits were detected across all environments (Table 2 and Fig. 1). Variation at single loci explained 0.40%–34.96% of the phenotypic variation. These QTL were distributed on 17 wheat chromosomes except for 1A, 1D, 7A and 7D. Twenty QTL had negative additive values, indicating that alleles from YZ1 reduced the phenotypic effect, whereas the alleles from NX188 increased the phenotypic values. At the remaining 18 loci, alleles from NX188 had positive additive values.

Additive QTL for FHB resistance were detected on chromosomes 2D, 4B, 4D, 5B and 5D. The contribution of single QTL ranged from 1.01% to 12.86% (Table 2 and Fig. 1). *QFHB.caas-5D* and *QFHB.caas-4D* showed larger effects than others. Favorable alleles at these five additive loci were from both parents, such as *QFHB.caas-4D*, *QFHB.caas-5B*, and *QFHB.caas-5D* from NX188 and *QFHB.caas-2D* and *QFHB.caas-4B* from YZ1 (Table 2).

Table 1 – Means, ranges, standard deviations (SD) and coefficients of variation (CV) of agronomic traits and FHB response of parents and RILs in two seasons.

Trait	Environment	Parent		RIL			
		NX188	YZ1	Mean	Range	SD	CV
FHB repose							
Disease index	Jianyang, 2005	0.40	0.79	0.41	0.16–0.70	0.10	24.11
	Jianyang, 2006	0.42	0.76	0.64	0.26–0.90	0.12	24.34
Resistance score	Jianyang, 2005	4	7	5.31	2–7	1.11	20.97
	Jianyang, 2006	2	7	5.71	2–7	1.32	23.09
Agronomic traits							
Grain number per spike	Beijing, 2003	50.80	40.40	41.28	29.60–58.00	6.11	14.80
	Beijing, 2004	49.84	38.22	41.15	16.12–67.60	8.48	20.60
	Luoyang, 2003	59.23	58.70	57.03	43.20–78.00	6.81	11.94
	Luoyang, 2004	45.47	53.66	45.69	15.60–27.20	6.33	13.85
Plant height (cm)	Beijing, 2003	62.80	66.20	66.38	38.20–97.40	14.15	21.32
	Beijing, 2004	67.05	66.20	64.27	36.22–100.40	14.01	21.80
	Luoyang, 2003	71.27	78.30	71.72	42.33–108.40	15.31	21.35
	Luoyang, 2004	67.73	66.23	65.44	35.20–101.20	12.92	19.74
Spikelet compactness	Beijing, 2003	2.12	2.13	2.27	1.63–3.28	0.30	13.12
	Beijing, 2004	2.29	2.11	2.16	1.54–3.72	0.32	15.08
	Luoyang, 2003	2.16	2.26	2.16	1.65–2.99	0.24	11.28
	Luoyang, 2004	2.48	2.38	2.32	1.66–3.33	0.31	13.25
Spike length (cm)	Beijing, 2003	9.80	7.80	8.06	5.70–11.80	1.30	16.13
	Beijing, 2004	9.63	8.27	9.08	6.50–12.10	1.25	13.77
	Luoyang, 2003	9.78	9.75	10.12	7.10–13.70	1.22	12.07
	Luoyang, 2004	8.98	10.04	9.58	6.83–12.20	1.23	12.84
Spikelet number per spike	Beijing, 2003	20.80	16.60	18.00	14.12–24.80	2.21	12.28
	Beijing, 2004	22.03	17.47	19.47	13.23–27.10	2.63	13.50
	Luoyang, 2003	21.17	22.05	21.55	18.33–25.60	1.23	5.65
	Luoyang, 2004	22.27	23.94	21.92	15.50–27.20	2.05	9.35
Thousand-grain weight (g)	Beijing, 2003	46.84	42.15	35.02	15.03–52.47	8.15	23.27
	Beijing, 2004	37.85	39.03	40.92	17.40–59.21	7.34	17.94
	Luoyang, 2003	50.50	49.92	48.32	37.64–57.62	4.12	8.50
	Luoyang, 2004	46.83	44.97	43.08	30.78–54.40	4.56	10.58

Five additive QTL were detected for GNS on chromosomes 2B, 4B, 5A, 5B and 5D, with phenotypic contributions ranging from 3.63% to 10.13% (Table 2 and Fig. 1). Alleles increasing GNS from NX188 were at *QGNS.caas-4B*, *QGNS.caas-5B* and *QGNS.caas-5D*, and the positive alleles at other loci were from YZ1. QE interactions were detected for all five QTL and accounted for 3.57% of the phenotypic variation. One pair of additive QTL showed interaction, accounting for 6.02% of the phenotypic variation (Table 3).

Five additive QTL for PH detected in Beijing and Luoyang were located on chromosomes 2D, 4B, 4D, 5A and 5D explained phenotypic variation of 1.51% and 34.96%, respectively (Table 2 and Fig. 1). Alleles at the *QPH.caas-4D* and *QPH.caas-5D* loci reducing PH were from YZ1, and the other alleles reducing height came from NX188. *QPH.caas-4B* and *QPH.caas-4D* were located in marker intervals co-inciding with dwarfing genes *Rht-B1* and *Rht-D1*, respectively, and *QPH.caas-2D.1* was identified at the position of *Rht8*. The effects of *QPH.caas-4B* and *QPH.caas-4D* were much greater than that of *QPH.caas-2D*. This result confirmed an earlier finding that the effects of *Rht-B1* and *Rht-D1* were much larger than that of *Rht-8* [20]. *QPH.caas-5A* and *QPH.caas-5D* had minor effects on reducing PH. Four pairs of QTL showed interactions (Table 3) that explained phenotypic variation of 4.44%.

Eight additive QTL for SL were detected on chromosomes 1B, 2D, 4A, 5A, 5D, 6A and 7B, and explained 4.12%–11.97% of the phenotypic variation (Table 2 and Fig. 1). Of these *QSL.caas-1B* and *QSL.caas-2D* gave the largest effects. The map position of *QSL.caas-2D* was similar to that of *QPH.caas-2D* in the *Rht8* region, suggesting that *Rht8* affected SL. Alleles increasing SL were from NX188, viz. *QSL.caas-1B*, *QSL.caas-4A.1*, *QSL.caas-5D* and *QSL.caas-6A*, whereas the other four were from YZ1. Interactions between three pairs of QTL accounted for 3.54% of the total phenotypic variation (Table 3).

Additive QTL for SPI were detected on chromosomes 1B, 5A, 5B and 5D, and each explained 0.40%–23.99% of the phenotypic variation (Table 2 and Fig. 1). All three favorable alleles with larger effects on increasing SPI were from NX188 and explained 53.6% the variation. QE interactions were detected for all QTL, accounting for 9.78% of the phenotypic variation. These data indicated that spikelet numbers were affected by environmental variation. Interaction was detected between two pairs of QTL on four chromosomes (Table 3), and together accounted for 3.43% of the phenotypic variation.

Six additive QTL for SC were detected on chromosomes 2D, 4A, 5A, 6B and 7B, and each explained between 2.83% and 17.34% of the phenotypic variation (Table 2 and Fig. 1). All except *QSC.caas-4A.1* increased SC and all were derived from NX188 and contributed for 39.31% of the phenotypic variation. QE interactions were detected for four of the QTL. The latter had a very small effect (0.22%) on phenotypic variation. Interactions between four pairs of QTL were detected (Table 3), and together accounted for 6.45% of the phenotypic variation. These results showed that spike

Table 2 – QTL with additive effects and QE for agronomic traits and FHB resistance in two years.

QTL	Marker interval	Site (cM)	A [a]	R^2_A (%) [b]	R^2_{AE} (%) [c]
Resistance to FHB					
QFHB.caas-2D	*Xwmc111–Xwmc112*	75.6	−0.3679**	4.70	0.004
QFHB.caas-4B	*Xgwm0925–Xgwm0898*	84.9	−0.4341**	5.69	0.20
QFHB.caas-4D	*Xpsp3007–DFMR2*	60.0	0.5727**	9.32	0.85
QFHB.caas-5B	*Xwmc235–Xwmc28*	18.3	0.3135**	1.01	0.03
QFHB.caas-5D	*Xgwm292–Vrn-D1*	1.1	0.6017**	12.86	0.05
Grain number per spike					
QGNS.caas-2B	*Xgwm429– Xgwm410*	20.0	1.9723**	3.63	0.05
QGNS.caas-4B	*Xcfd39a–Xgwm0925*	74.5	−1.8482**	7.08	0.05
QGNS.caas-5A	*Xgwm304–Xbarc56*	60.4	1.0243**	3.90	0.29
QGNS.caas-5B1	*Vrn-B1–Xwmc75*	0	−0.9614**	10.13	0.05
QGNS.caas-5D	*Xgwm292–Vrn-D1*	1.1	−1.3031**	5.62	3.13
Plant height					
QPH.caas-2D	*Xwmc111–Xwmc112*	89.6	4.0370**	3.96	0.03
QPH.caas-4B	*Xgwm0925–Xgwms0898*	85.9	8.0044**	31.92	0.07
QPH.caas-4D	*Xpsp3007–DFMR2*	68.0	−8.4021**	34.96	0.04
QPH.caas-5A	*Xgwm1258.1–Xwmc327*	30.3	1.9853**	3.75	0.01
QPH.caas-5D	*Xgwm292–Vrn-D1*	1.1	−1.5159**	1.51	0.73
Spike length					
QSL.caas-1B	*Xgwm11–Xcfd21b*	85.9	−0.4645**	11.67	0.01
QSL.caas-2D	*Xwmc111–Xwmc112*	73.6	0.4892**	11.97	0.04
QSL.caas-4A-1	*Xcfd71a–2–Xgwm397*	10.1	−0.3102**	4.84	0.01
QSL.caas-4A-2	*Xbarc78–Xgwm160*	25.2	0.2676**	4.28	0.01
QSL.caas-5A	*Xwmc327–Xgwm293b*	44.9	0.3625**	4.10	0.04
QSL.caas-5D	*Xgwm292– Vrn-D1*	1.1	−0.3119**	8.22	0.229
QSL.caas-6A	*Xgwm169–Xgwm617*	162.0	−0.2270**	4.12	0.03
QSL.caas-7B1	*TaCK7B–Xwmc276*	113.0	0.1831**	4.05	0.01
Spikelet number per spike					
QSPI.caas-1B	*Xbarc187–Xwmc419b*	59.6	−0.7454**	10.17	0.02
QSPI.caas-5A	*Xgwm304–Xbarc56*	60.4	0.2200**	0.40	0.92
QSPI.caas-5B	*Xgwm408–Xwmc235*	2.6	−0.8927**	23.99	2.26
QSPI.caas-5D	*Xgwm292–Vrn-D1*	1.1	−0.9349**	19.44	6.58
Spike compactness					
QSC.caas-2D	*Xwmc111–Xwmc112*	74.6	−0.1301**	17.34	0.01
QSC.caas-4A1	*Xwmc516–Xcfd71b*	6.0	0.0307**	3.46	0.
QSC.caas-4A2	*Xbarc78–Xgwm160*	26.2	−0.0634**	9.82	0.19
QSC.caas-5A	*Xgwm304–Xbarc56*	58.4	−0.0267**	2.83	0.02
QSCcaas-6B	*Xbarc79–Xbarc1008*	74.9	−0.0337**	4.40	0
QSC.caas-7B	*TaCK7B–Xwmc276*	112.0	−0.0580**	4.92	0.01
Thousand-grain weight					
QTGW.caas-2A-1	*Xpsp3039–Pm4*	209.8	0.9423**	2.90	0.57
QTGW.caas-2B	*Xcfd19–Xgwm191a*	146.2	0.8891**	4.01	0.27
QTGW.caas-3D	*C19L34.3D–Xgwm191c*	103.2	0.9315**	4.59	0
QTGW.caas-4B	*Xgwm0925–Xgwm0898*	84.9	2.1747**	15.47	3.39
QTGW.caas-4D	*Xpsp3007–DFMR2*	67.0	−2.1228**	18.30	2.66

[a] Additive effects. Positive values indicate that alleles were derived from YZ1; negative values indicate that the alleles were derived from NX188. [b] Percentage of phenotypic variation explained by QTL with additive effect. [c] Percentage of phenotypic variation explained by QE interaction. ** Significance at $P = 0.001$.

compactness was controlled by genes with additive and epistatic effects.

Additive QTL for TGW were detected on chromosomes 2A, 2B, 3D, 4B and 4D, and each one explained between 2.90% and 18.30% of the phenotypic variation (Table 2 and Fig. 1). *QTGW.caas-4B* and *QTGW.caas-4D*, with the largest effects explained 15.47% and 18.30% of the phenotype variation, respectively. One favorable allele came from each parent. QE interactions were detected and explained 6.89% of the phenotypic variation in total. Interactions between three pairs of the QTL were detected (Table 3), accounting for 6.76% of the phenotypic variation.

Six gene clusters were detected for the 56 additive and epistatic QTL identified in this study, and were located on chromosomes 2D, 4B, 4D, 5A, 5B, 5D and 7B (Table 4 and Fig. 1). These QTL clusters suggested polytrophic effects conferred by some loci. Four QTL (*QPH.caas-2D*, *QSC.caas-2D*, *QSL.caas-2D* and *QFHB.caas-2D*) were located in the region *Xwmc111–Xwmc112* on chromosome 2D where *Rht8* was located. The positive values for PH and SL and negative values for SC and FHB suggested that the allelic effects from YZ1 in this QTL cluster were for increasing PH, and SL, but decreasing SC and FHB (increasing FHB resistance) or alternately that the allele from NX188 decreased PH and SL but increased SC and FHB. Four QTL (*QGNS.caas-4B*, *QPH.caas-4B*, *QTGW.caas-4B* and *QFHB.caas-4B*) were located in the region *Xgwm0925–Xgwm0898* on chromosome 4B, co-locating with dwarfing gene *Rht-B1*. The positive

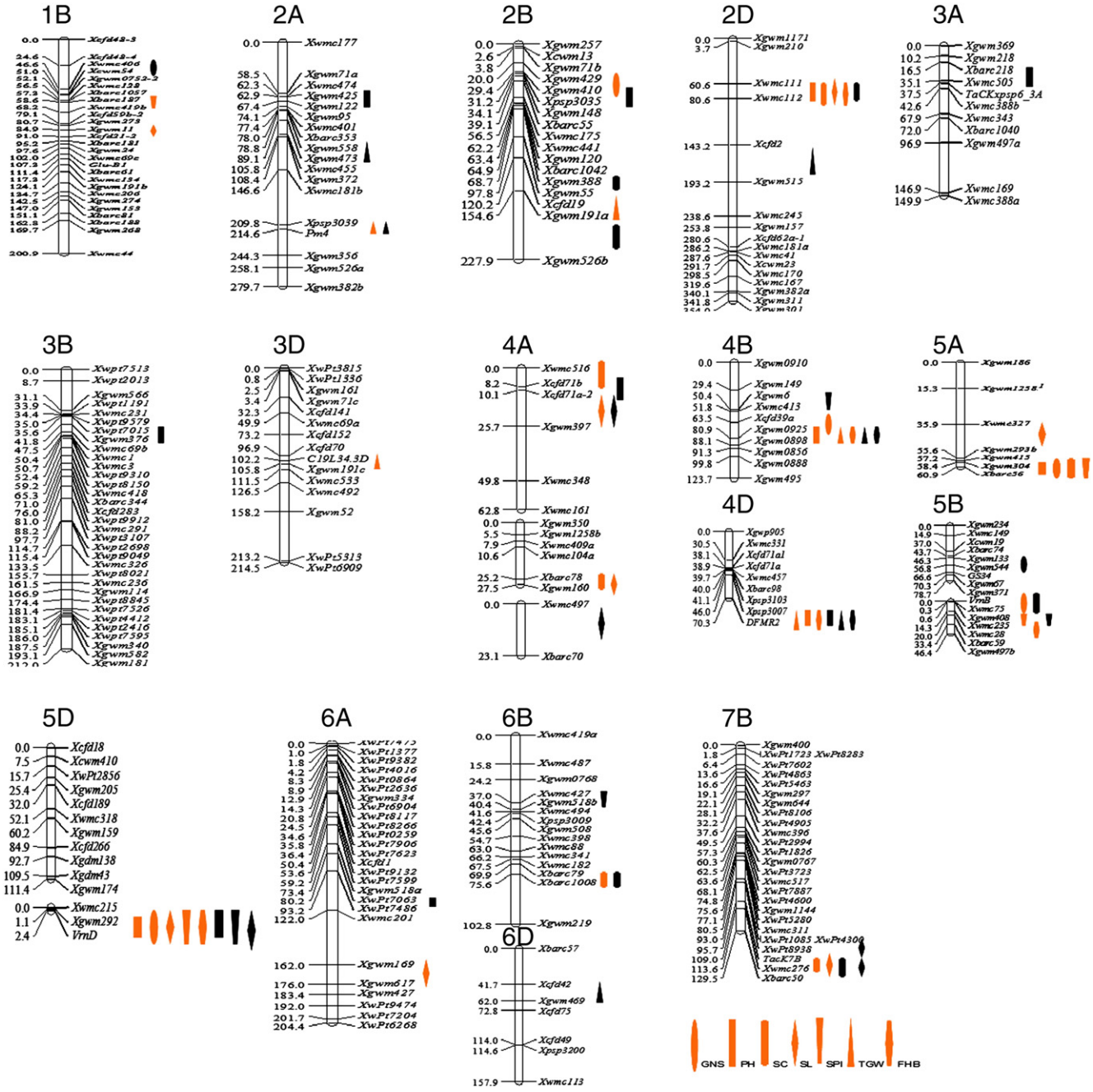

Fig. 1 – Positions of QTL associated with PH, SL, SPI, SC, GNS, TGW and FHB resistance in wheat. Red color indicates additive QTL and black indicates epistatic QTL.

values for PH and TGW, and negative values for FHB and GNS suggested that alleles from YZ1 increased PH and TGW but reduced FHB resistance and GNS, or alternatively, the allele from NX188 with the effect of reducing PH and TGW but increasing FHB resistance and GNS. Three QTL (*QPH.caas-4D*, *QTGW.caas-4D* and *QFHB.caas-4D*) were mapped in the region between markers *Xpsp3007* and *DFMR2* on chromosome 4D, the position of dwarfing gene, *Rht-D1*. The allele from YZ1 for the QTL cluster reduced PH, TGW and FHB resistance or alternatively the allele from NX188 increased PH, TGW and FHB resistance. Three QTL (*QGNS.caas-5A*, *QSC.caas-5A* and *QSPI.caas-5A*) were in the region *Xgwm304–Xbarc56* on chromosome 5A. The YZ1 allele in this QTL cluster had the effect of increasing GNS and SPI and reducing SC. Five QTL (*QGNS.caas-5D*, *QPH.caas-5D*, *QSPI.caas-5D*, *QSL.caas-5D* and *QFHB.caas-5D*) were mapped between *Xgwm292* and *Xgwm269* on chromosome 5D, the location of vernalization gene *Vrn-D1*. The NX188 allele at this locus had a large effect on simultaneously increasing FHB resistance, GNS, SL, and SPN, and with low interaction with PH. Finally, four QTL (two with additive and two epistatic effects) were mapped in the *TaCK7B–Xwmc276*

Table 3 – Identified QTL with epistatic effects and QE for agronomic traits over two years.

QTL	Marker interval	Site (cM)	QTL	Marker interval	Site (cM)	AA [a]	R^2_{AA} (%) [b]	R^2_{AAE} (%) [c]
Resistance to FHB								
QFHB.caas-4B	*Xgwm0925–Xgwm0898*	84.9	*QFHB.caas-4D*	*Xpsp3007–DFMR2*	68.0	−0.2288**	0.94	0.73
Grain number per spike								
QGNS.caas-1B	*Xwmc406–cwm54*	47.6	*QGNS.caas-5B-2*	*Xgwm133–Xgwm 544*	56.3	1.5598**	6.02	0
Plant height								
QPH.caas-4D	*Xpsp3007–DFMR2*	68.0	*QPH.caas-5D*	*Xwm292–Vrn-D1*	1.1	1.0926**	0.58	0.01
QPH.caas-2A	*Xgwm425–Xgwm122*	62.9	*QPH.caas-3B*	*XwPt7015–Xgwm376*	39.6	−1.8473**	1.52	0
QPH.caas-2B	*Xgwm410–Xpsp3035*	29.4	*QPH.caas-3A*	*Xbarc218–Xwmc505*	29.4	1.6044	0.85	0
QPH.caas-4A	*Xcfd71b– Xcfd71a–2*	9.2	*QPH.caas-6A*	*XwPt7063–XwPt7486*	87.2	−2.2644	1.49	0.02
Spike length								
QSL.caas-4A.3	*Xwmc497–Xbarc70*	13.0	*QSL.caas-7B2*	*XwPt8938–TaCK7B*	95.7	0.1836**	1.68	0
QSL.caas-4A.1	*Xcfd71a.2–Xgwm397*	10.1	*QSL.caas-5D*	*Xgwm292–VrnD*	1.1	0.1316	1.33	0.16
QSL.caas-4A.1	*Xcfd71a.2–Xgwm397*	10.1	*QSL.caas-7B1*	*TaCK7B–Xwmc276*	113.0	0.1070**	0.53	0.01
Spikelet number per spike								
QSPI.caas-4B	*Xgwm6–Xwmc413*	51.4	*QSPI.caas-6B*	*Xwmc427–Xgwm518b*	39.0	−0.3434**	2.86	0.15
QSPI.caas-5B	*Xgwm408–Xwmc235*	2.6	*QSPI.caas-5D*	*Xgwm292–VrnD1*	1.1	0.1759**	0.57	2.41
Spike compactness								
QSC.caas-2B.1	*Xgwm388–Xgwm55*	72.1	*QSC.caas-2D*	*Xwmc111–Xwmc112*	74.6	−0.0584**	1.27	0
QSC.caas-2B2	*Xgwm191a–Xgwm526b*	201.6	*QSC.caas-2D*	*Xwmc111–Xwmc112*	74.6	0.1034**	0.82	0
QSC.caas-5B	*Vrn B1–Xwmc75*	0	*QSC.caas-6B*	*Xbarc79–Xbarc1008*	74.9	0.0467**	1.58	0
QSC.caas-6B	*Xbarc79–Xbarc1008*	74.9	*QSC.caas-7B*	*TaCK7B–Xwmc276*	112.0	0.0488**	2.78	0.2
Thousand-grain weight								
QTGW.caas-4B	*Xgwm0925–Xgwm0898*	84.9	*QTGW.caas-4D*	*Xpsp3007–DFMR2*	67.0	0.9569**	1.07	0.09
QTGW.caas-2A.2	*Xgwm558–Xgwm473*	78.8	*QTGW.caas-2A1*	*Xpsp3039–Pm4*	209.8	0.7373	1.88	0.23
QTGW.caas-2D	*Xcfd2– Xgwm515*	143.2	*QTGW.caas-6D*	*Xcfd42–Xgwm469*	45.7	−1.3133	3.81	0.56

[a] Epistatic effects. Positive value indicates parent effect is greater than the recombinant effect; a negative value indicates the opposite. [b] Percentage of variation explained by epistatic QTL. [c] Percentage of variation explained by QE interaction. ** Significance at P = 0.001.

region on chromosome 7B. *TaCK7B* is a cytokinin-oxidase/dehydrogenase gene controlling cytokinin levels in plant tissues [21].

3.3. MAS for developing elite lines with improved FHB resistance and agronomic traits

MAS was carried out to select elite lines with high FHB resistance and good agronomic traits. Among them, FHB was treated as first priority. Six elite lines were selected based on this criterion (Table 5). All had better agronomic traits (Table 6) than the others. No significant differences were detected between the observed and predicted values for all seven traits with SPI in the 2004–2005 cropping season ($P = 0.05$) as the only exception. These results indicated a high efficiency of MAS in this study (Table 5). For example, for FHB resistance, RIL-151 and RIL-164 carried all five resistance alleles, and showed the best FHB resistance. RIL-31, RIL-68, RIL-130 and RIL-169 possessed four of the five resistance alleles). For other agronomic traits, these lines carried more favorable alleles than others. The lines should be useful as parents for conventional breeding and MAS because germplasm with both good FHB resistance and other agronomic traits is rare.

4. Discussion

4.1. Analysis of QTL for FHB resistance

Numerous sources of FHB resistance that have been genetically mapped to chromosomes are from many countries in Asia, North America, South America, and Europe [9]. In this study, we identified five additive QTL associated with FHB resistance on chromosomes 2D, 4B, 4D, 5B and 5D. Among them, *QFHB.caas-4D* and *QFHB.caas-5D* showed larger effects

Table 4 – Additive QTL clusters and their effects on FHB and agronomic traits.

Chr.	Cluster interval	Trait						
		GNS	PH	SC	SPI	SL	TGW	FHB
2D	*Xwmc111–Xwmc112*		4.3070**	−0.1301**		0.4892**		−0.3679**
4B	*Xgwm0925–Xgwms0898*	−1.8482**	8.0044**				2.1747**	−0.4341**
4D	*Xpsp3007–DFMR2*		−8.4021**				−2.1228**	0.5727**
5A	*Xgwm304–Xbarc56*	1.0243**		−0.0267**	0.2200**			
5D	*Xgwm292–Vrn-D1*	−1.3031**	−1.5159**		−0.9349**	−0.3119**		0.6017**
7B	*TaCK7B–Xwmc276*			−0.0580**		0.1831**		

** Indicates significance at P = 0.001. GNS: grain number per spike; PH: plant height; SC: spikelet compactness; SPI: spikelet number per spike; SL: spike length; TGW: thousand-grain weight; FHB: Fusarium head blight.

Table 5 – Genotypes of six selected RILs.

Locus	Marker	RIL-31	RIL-68	RIL-130	RIL-151	RIL-164	RIL-169
QTGW.caas-2A	*Xpsp3039*	B	B	B	A	A	B
QGNS.caas-2B	*Xgwm429*	B	A	A	A	B	A
QTGW.caas-2B	*Xgwm191a*	B	A	A	A	A	A
QFHB.caas-2D	*Xwmc112*	A	B	A	A	A	B
QTGW.caas-3D	*C19L34.3D*	A	A	—	B	B	—
QFHB.caas-4B	*Xgwm 0898*	B	A	A	A	A	A
QGNS.caas-4B	*Xcfd39a*	B	A	A	A	B	A
QFHB.caas-4D	*DFMR2*	B	B	B	B	B	B
QGNS.caas-5A	*Xbarc56*	B	B	B	B	B	A
QPH.caas-5A	*Xwmc327*	—	A	B	A	A	A
QFHB.caas-5B	*Xwmc28*	B	B	A	B	B	B
QGNS.caas-5B	*Xgwm 408*	—	B	B	B	—	B
QFHB.caas-5D	*Xgwm 292*	B	B	B	B	B	B

A: source of QTL allele from YZ1; B: source of QTL allele from parent NX188.

than other QTL, explaining 7.01% and 12.87% of the phenotypic variation, respectively. Korean cultivar, Chokwang, was reported to carry *Qfhs.ksu-5DL.1* for type II FHB resistance [22]. A minor QTL (R^2 = 4%) on chromosome 5DL was reported in a RIL population derived from a cross between European winter wheat cultivars Renan and Recital [23]. While SSR marker *Xgwm292* was closely linked to *QFHB.caas-5D* in this study, the same QTL for type II resistance was detected in a Wangshuibai/Wheaton RIL population [24]. This indicated that *QFHB.caas-5D* conferred type II FHB resistance. In a similar region to *QFHB-caas-4D*, another QTL conferred Type I resistance using a different population [25,26]. Thus, *QFHB-caas-4D* identified in this study was probably associated with Type I resistance. In addition, *QFHB-caas-4B* was in the same region to that reported by Buerstmayr et al. [10]. It therefore should be a reliable locus for FHB resistance.

4.2. *Correlation between FHB resistance and agronomic traits*

Mechanisms of FHB resistance in wheat can be addressed from the viewpoint of morphology, physiology and biochemistry. Negative correlations between visual FHB symptoms and some agronomic traits such as plant height have been reported [2,9]. Co-localizations were also found between FHB resistance and QTL for plant height and spike architecture in barley [27]. In this study, the locations of *QPH.caas-2D*, *QPH.caas-4B* and *QPH.caas-4D* were the same as *QFHB.caas-2D*, *QFHB.caas-4B* and *QFHB.caas-4D*, respectively. *QFHB.caas-4D* was located in the interval *Xpsp3007–DFMR2*, and *QFHB.caas-2D* was located between *Xwmc11* and *Xwmc112*. Wheat dwarfing genes *Rht-B1* and *Rht8* are located on chromosomes 4D and 2D, respectively. DFMR2 was used for detecting *Rht-B1* allelic variation [28]. Compared with the high density wheat integration map [29], *Xwmc112* was very close to *Xgwm261* which is closely linked to *Rht8*. Since plant height was reduced, the probability of soil surface spore infection was increased, and the high humidity environment was conducive to FHB disease development. In the same or a similar interval between *Xgwm292* and *Vrn-D1*, there were five additive QTL conferring different traits, including *QFHB-caas-5D* (Fig. 1). These co-localizations showed that linkages may exist between genes for FHB resistance and agronomic traits that are independent of pleiotropic effects.

4.3. *Simultaneous QTL detection and MAS*

Gene/QTL detection and MAS are often carried out separately. Although hundreds of genes/QTL have been detected, progress in

Table 6 – Agronomic traits of six YZ1/NX188 RILs with superior resistance to FHB in RILs.

Trait	Year	RIL-31	RIL-68	RIL-130	RIL-151	RIL-164	RIL-169	P-value
Resistance score	2004–2005	2	2	2	2	2	2	0.0614
	2005–2006	2	2	2	2	2	2	0.3173
Grain number per spike	2003–2004	65.6	56.4	51.2	52.4	56.0	60.0	0.6188
	2004–2005	50.0	40.4	42.8	44.0	46.4	50.4	0.9919
Plant height (cm)	2003–2004	70.4	89.2	85.4	98.6	87.2	75.2	0.9133
	2004–2005	77.6	83.2	90.4	90.4	92.6	77.2	0.2802
Spike length (cm)	2003–2004	10.6	10.0	9.7	12.7	12.5	10.7	0.2886
	2004–2005	11.2	8.5	10.1	11.1	11.0	9.3	0.3689
Spikelet numberper spike	2003–2004	23.0	22.6	20.4	22.8	22.4	23.6	0.4655
	2004–2005	23.4	23.2	23.4	23.6	25.0	23.4	0.0431*
Spike compactness	2003–2004	2.17	2.26	2.10	1.80	1.79	2.21	0.0868
	2004–2005	2.09	2.73	2.32	2.13	2.27	2.52	0.6284
Thousand-grain weight (g)	2003–2004	51.29	53.33	41.62	51.84	49.30	44.80	0.0922
	2004–2005	52.17	47.14	42.01	48.32	46.58	43.05	0.9913

P-value, *t*-test between observed and predicted values at $P = 0.05$. * Significant at $P = 0.05$.

utilization MAS has been slow. The main reason for this was that markers detected in one population are often not applicable to other populations. In the present study, we combined gene/QTL detection and MAS using a RIL population. The advantages of this approach are as follows: (1) all the markers detected are efficient for MAS, and do not need to be validated again; (2) Gene/QTL detection and MAS are carried out simultaneously, shortening the time of MAS; (3) the genotypes of all selected new varieties/elite lines are known, a feature that will be helpful in further genetic improvement. For example, of the five QTL for FHB resistance, there are four favorable alleles for FHB resistance in RIL-169, and only favorable allele *QFHB.caas-2D* was absent. To further improve its FHB resistance, RIL-169 and RIL-151 can be crossed in order to add *QFHB.caas-2D* in a genetic background that is largely shared with RIL-169 (Table 5). New varieties with better FHB resistance and agronomic traits than RIL-169 will be easily bred. To carry out QTL detection and MAS simultaneously, the precondition is to construct a segregating population with both target traits and a better background of traits of agronomic importance. In the present study, six elite lines were selected from a cross of well adapted varieties. In conclusion, the results from this study suggest that QTL detection and MAS can be integrated using appropriate populations. This approach will significantly accelerate MAS in the future.

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REFERENCES

[1] H.W. Schroeder, J.J. Christensen, Factors affecting resistance of wheat to scab caused by *Gibberella zeae* (Schw.), Phytopathology 53 (1963) 831–838.
[2] A. Mesterhazy, Types and components of resistance to Fusarium head blight of wheat, Plant Breed. 114 (1995) 377–386.
[3] G.H. Bai, G. Shaner, Management and resistance in wheat and barley to Fusarium head blight, Annu. Rev. Phytopathol. 42 (2004) 135–161.
[4] P. Chen, D. Liu, W. Sun, New countermeasures of breeding wheat for scab resistance, in: H.J. Dubin, L. Gilchrist, J. Reeves, A. McNab (Eds.), Fusarium Head Scab: Global Status and Future Prospects, CIMMYT, Mexico, 1997, pp. 59–65.
[5] J.B. Yao, W.Z. Lu, Research advances in wheat breeding for scab resistance in China, J. Agric. Sci. 16 (2000) 242–248(in Chinese with English abstract).
[6] G.H. Bai, F.L. Kolb, G.E. Shaner, L.L. Domier, Amplified fragment length polymorphism markers linked to a major quantitative trait locus controlling FHB resistance in wheat, Phytopathology 89 (1999) 343–348.
[7] G.H. Bai, G. Shaner, H. Ohm, Inheritance of resistance to *Fusarium graminearum* in wheat, Theor. Appl. Genet. 100 (2000) 1–8.
[8] H.X. Ma, G.H. Bai, X. Zhang, W.Z. Lu, Main effects, epistasis, and environmental interactions of quantitative trait loci for Fusarium head blight resistance in a recombinant inbred population, Phytopathology 96 (2006) 534–541.
[9] H. Buerstmayr, T. Ban, J.A. Anderson, QTL mapping and marker-assisted selection for Fusarium head blight resistance in wheat: a review, Plant Breed. 128 (2009) 1–26.
[10] M. Buerstmayr, K. Huber, J. Heckmann, B. Steiner, J.C. Nelson, H. Buerstmayr, Mapping of QTL for Fusarium head blight resistance and morphological and developmental traits in three backcross populations derived from *Triticum dicoccum* × *Triticum durum*, Theor. Appl. Genet. 125 (2012) 1751–1765.
[11] B.L. Waldron, B. Moreno-Sevilla, J.A. Anderson, R.W. Stack, R.C. Frohberg, RFLP mapping of QTL for Fusarium head blight resistance in wheat, Crop Sci. 39 (1999) 805–811.
[12] J.A. Anderson, R.W. Stack, S. Liu, B.L. Waldron, A.D. Fjeld, C. Coyne, B. Moreno-Sevilla, J.M. Fetch, Q.J. Song, P.B. Cregan, DNA markers for Fusarium head blight resistance QTLs in two wheat populations, Theor. Appl. Genet. 102 (2001) 1164–1168.
[13] J.A. Anderson, S.M. Chao, S.X. Liu, Molecular breeding using a major QTL for Fusarium head blight resistance in wheat, Crop Sci. 47 (2007) S112–S119.
[14] W.C. Zhou, F.L. Kolb, G. Bai, G. Shaner, L.L. Domier, Genetic analysis of scab resistance QTL in wheat with microsatellite and AFLP markers, Genome 45 (2002) 719–727.
[15] Z.Z. Liu, Z.Y. Wang, W.J. Zhao, A study on FHB resistance of wheat germplasm source, Acta Agric. Shanghai 1 (2) (1985) 75–84(in Chinese with English abstract).
[16] L. Tamburic-Ilincic, D. Falk, A. Schaafsma, Fusarium ratings in the Ontario Winter Wheat Performance Trial (OWWPT) using an index that combines Fusarium head blight symptoms and deoxynivalenol levels, Czech J. Genet. Plant Breed. 47 (2011) S115–S122.
[17] Q. Yao, Y.X. Song, R.H. Zhou, T.H. Fu, J.Z. Jia, Quantitative trait loci for adult-plant resistance against yellow rust in a wheat-derived recombinant inbred line population, Sci. Agric. Sin. 42 (2009) 4234–4241(in Chinese with English abstract).
[18] D.L. Wang, J. Zhu, Z.K. Li, A.H. Paterson, Mapping QTLs with epistatic effects and QTL × environment interactions by mixed linear model approaches, Theor. Appl. Genet. 99 (1999) 1255–1264.
[19] R.A. McIntosh, G.E. Hart, K.M. Devos, M.D. Gale, W.J. Rogers, Catalogue of gene symbols for wheat, http://grain.jouy.inra.fr/ggpages/wgc/98/Intro.htm#Intro61988.
[20] X.K. Zhang, S.J. Yang, Y. Zhou, Z.H. He, X.C. Xia, Distribution of the *Rht-B1b*, *Rht-D1b* and *Rht8* reduced height genes in autumn-sown Chinese wheats detected by molecular markers, Euphytica 152 (2006) 109–116.
[21] L. Zhang, B.S. Zhang, R.H. Zhou, L.F. Gao, G.Y. Zhao, Y.X. Song, J.Z. Jia, Cloning and genetic mapping of cytokinin oxidase/dehydrogenase gene (*TaCKX2*) in wheat, Acta Agron. Sin. 33 (2007) 1419–1425(in Chinese with English abstract).
[22] Z.P. Yang, J. Gilbert, G. Fedak, D.J. Somers, Genetic characterization of QTL associated with resistance to Fusarium head blight in a doubled-haploid spring wheat population, Genome 48 (2005) 187–196.
[23] L. Gervais, F. Dedryver, J.Y. Morlais, V. Bodusseau, S. Negre, M. Bilous, C. Groos, M. Trottet, Mapping of quantitative trait loci for field resistance to Fusarium head blight in an European winter wheat, Theor. Appl. Genet. 106 (2003) 961–970.
[24] J. Yu, G. Bai, W. Zhou, Y. Dong, F.L. Kolb, Quantitative trait loci for Fusarium head blight resistance in a recombinant inbred population of Wangshuibai/Wheaton, Phytopathology 98 (2003) 87–94.

[25] J. Yang, G. Bai, G.E. Shaner, Novel quantitative trait loci (QTL) for Fusarium head blight resistance in wheat cultivar Chokwang, Theor. Appl. Genet. 111 (2005) 1571–1579.
[26] R. Draeger, N. Gosman, A. Steed, E. Chandler, M. Thomsett, J. Schondelmaier, H. Buerstmayr, M. Lemmens, M. Schmolke, A. Mesterhazy, Identification of QTLs for resistance to Fusarium head blight, DON accumulation and associated traits in the winter wheat variety Arina, Theor. Appl. Genet. 115 (2007) 617–625.
[27] Z.X. Ma, B.J. Steffenson, L.K. Prom, N.L.V. Lapitan, Mapping of quantitative trait loci for Fusarium head blight resistance in barley, Phytopathology 90 (2000) 1079–1088.
[28] H. Ellis, W. Spielmeyer, K. Gale, G. Rebetzke, R. Richards, "Perfect" markers for the *Rht-B1b* and *Rht-D1b* dwarfing genes in wheat, Theor. Appl. Genet. 105 (2002) 1038–1042.
[29] D.J. Somers, P. Isaac, K. Edwards, A high-density microsatellite consensus map for bread wheat (*Triticum aestivum* L.), Theor. Appl. Genet. 109 (2004) 1105–1114.