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ORIGINAL PAPER

QTL analysis of resistance to Fusarium head blight in the novel wheat germplasm CJ 9306. II. Resistance to deoxynivalenol accumulation and grain yield loss

Guo-Liang Jiang · Yanhong Dong · JianRong Shi · Richard W. Ward

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Abstract Fusarium head blight (FHB or scab) caused by *Fusarium* species is a destructive disease in wheat, not only causing dramatic decrease of grain yield and quality, but also leading to serious mycotoxin contamination in the infected grains. This study was conducted to identify and quantify quantitative trait loci (QTLs) contributing to resistance to deoxynivalenol (DON) accumulation as well as to grain yield loss in a population of 152 F_7 recombinant inbred lines (RILs) derived from the cross Veery/CJ 9306. DON content in scabby grains and relative decreases of yield components were analyzed. Two new QTLs (*QFhs.nau-2DL* and *QFhs.nau-1AS*) for resistance to DON accumulation caused by FHB in wheat were detected, and QTLs *QFhs.ndsu-3BS* and *QFhs.nau-5AS* were also validated in CJ 9306, based on a constructed genetic linkage map. On the average of three experiments, major QTLs *QFhs.ndsu-3BS* and *QFhs.nau-2DL* explained up to 23 and 20% of phenotypic variation, respectively. *QFhs.nau-1AS* and *QFhs.nau-5AS* separately explained 4–6% of phenotypic variation. The differences among years/experiments were significant for all the four QTLs. However, the QTL × environment interaction was significant only for *QFhs.nau-2DL*, but not for the others. The results suggest that simple sequence repeat (SSR) markers *Xgwm533b* associated with *QFhs.ndsu-3BS*, and *Xgwm539* associated with *QFhs.nau-2DL* could be used in marker-assisted selection to enhance resistance to DON accumulation. *QFhs.ndsu-3BS* + *QFhs.nau-2DL* and *QFhs.nau-2DL* + *QFhs.nau-5AS* would be the optimum choices for two-locus combinations. *QFhs.ndsu-3BS* was also validated in CJ 9306 for resistance to grain yield loss, explaining 8–15% of phenotypic variation. No QTLs for resistance to DON accumulation or grain yield loss independent of Type II resistance were found. By comparison, however, either of *QFhs.nau-2DL* or *QFhs.nau-5AS* alone and their combination were more contributive to resistance to DON accumulation than to Type II resistance.

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G.-L. Jiang (✉) · R. W. Ward (✉)
Department of Crop and Soil Sciences,
Michigan State University, East Lansing, MI 48824, USA
e-mail: gljiang@msu.edu; gljiang99@yahoo.com

G.-L. Jiang
State Key Laboratory of Crop Genetics and Germplasm Enhancement, Nanjing Agricultural University,
1 Weigang, Nanjing 210095, China

Y. Dong
Department of Plant Pathology, University of Minnesota,
1991 Upper Buford Circle, St Paul, MN 55108, USA

J. Shi
Jiangsu Academy of Agricultural Sciences,
Nanjing 210014, China

R. W. Ward
CIMMYT Wheat Program, Apdo. Postal 6-641,
Mexico, D.F., 06600, Mexico
e-mail: r.w.ward@cgiar.org

Introduction

Fusarium head blight (FHB or scab) caused by *Fusarium* species is a destructive disease in wheat (*Triticum aestivum* L.) and barley (*Hordeum vulgare* L.). During the past decades, tremendous economic loss resulted from frequent epidemics of this disease raised worldwide awareness of its importance (Bai and Shaner 2004; Windels 2000). In addition to a great decrease of grain yield and quality, another problem caused by FHB is mycotoxin contamination in the

infected grains, which is detrimental to the health of human beings and livestock. Developing and growing resistant cultivars is the most economic, effective and environment-friendly approach to control this disease.

The expression of resistance to FHB in wheat and barley is complex. Schroeder and Christensen (1963) first defined two types of resistance in wheat: resistance to initial infection (Type I resistance) and resistance to the spread of the pathogen within the tissue or spike (Type II resistance). Subsequently, additional types of resistance were proposed, such as resistance to mycotoxin (Type III resistance) (Miller et al. 1985), resistance to kernel infection, and tolerance (Mesterhazy et al. 1999). In breeding and research, Type II resistance is predominantly focused on due to simple, accurate and easily operated inoculation and assessment techniques. So far, a number of quantitative trait loci (QTLs) for Type II resistance have been identified in different resistant varieties, such as Sumai 3 and its derivative (Waldron et al. 1999; Anderson et al. 2001; Buerstmayr et al. 2002; Zhou et al. 2002b; Yang et al. 2005b), Wangshuibai (Lin et al. 2004; Zhang et al. 2004; Zhou et al. 2004; Mardi et al. 2005), Frontana (Han et al. 2005), Wuhan-1 (Somers et al. 2003), Chokwang (Yang et al. 2005a), Ning 894037 (Shen et al. 2003), etc. However, knowledge of other types of resistance is very limited. Therefore, characterization of the genetic basis of the different types of resistance, especially the identification and quantification of the associated QTLs and QTL combinations, is of great significance and interest for the development of resistant cultivars.

Up to now, only a few investigations have been involved in QTL analysis of resistance to mycotoxin and yield loss (Pumphrey et al. 2007; Semagn et al. 2007). Somers et al. (2003) and Yang et al. (2005b) suggested the possibility of independent QTLs or genes for resistance to DON accumulation and kernel infection from Type II resistance. More recently, Semagn et al. (2007) identified a major QTL on 2AS for DON content, but not for FHB severity in the same experiment. It is expected that QTL analysis of these types of resistance will necessarily draw more and more interest, along with increasing awareness of mycotoxin contamination and the damage of grain yield and quality.

Previous investigations suggested that CJ 9306 not only had a very high level of Type II resistance as well as good comprehensive field resistance, but also possessed high resistance to DON accumulation caused by FHB and tolerance to grain yield loss (Jiang et al. 2006a, b). Because of its excellent resistance, unique history of breeding, and complex parentage, characterization of its FHB resistance by DNA markers is very useful for understanding of the underlying genetic basis and effective utilization of this novel elite germplasm. As stated previously, therefore, the objectives of our study were: (1) to identify and localize QTLs for different types of resistance to FHB in CJ 9306 and (2) to quantify the magnitude of their effects as both individual genes alone and gene combinations for multiple loci, with the ultimate goal to develop tools and approaches (MAS procedures) utilizable in practical breeding programs (Jiang et al. 2007). In this paper, we would present the results on resistance to DON accumulation, and resistance to grain yield loss.

Materials and methods

Plant materials and experimental design

As described previously, a mapping population with 152 F_7 recombinant inbred lines (RILs) was developed from the wheat cross between CJ 9306 (highly FHB-resistant, and developed at Nanjing Agricultural University, China through multiple-parent crossing and recurrent selection with modified pedigree methods with the aid of a dominant male-sterile gene *Ta1*) and Veery (susceptible to FHB, and developed from Kavkaz/Buho//KAL/BB (CM33027) at CIMMYT, Mexico) (Jiang et al. 2007). For detailed information about the breeding history and pedigree of CJ 9306, see Jiang et al. (2006a). All the 152 RILs were grown in the greenhouse at Michigan State University in a completely randomized design with two replications, each having six plants planted in two square pots (11 × 11 cm). The two parents were planted as the controls many times at an interval of 1 week. The experiment was repeated three times, sown in December 2001, January 2002 and November 2003, and designated as Experiment 02, 02a and 04, respectively.

Disease inoculation and resistance evaluation

Single-floret inoculation was conducted immediately before or after initial anthesis (around Zadoks growth stage 60) (Jiang et al. 2006b). The inoculum was *F. graminearum* isolate PH-1 for Experiment 02 and 02a, and a mixture of two isolates PH-1 and WF-1 for Experiment 04. Six to eight spikes of each RIL were inoculated per replication. For each single batch of inoculation, the checks were included. The inoculated plants/pots were mist-irrigated in a misting chamber at 22–26°C for 3 days. Then the pots were transferred to another greenhouse compartment, and disease severity was scored once every 4 days beginning 5 days and ending 25 days after inoculation.

Deoxynivalenol (DON) test

Resistance to mycotoxin can be either the sensitivity/response of plants/tissues to fungal toxin infection (Miller

et al. 1985) or resistance to toxin accumulation. The former was defined as the ability to survive or grow under the infection of mycotoxin, which was used in bioassay or toxin screening experiments (Wang and Miller 1987). The latter was defined as the ability to reduce or limit DON accumulation within the infected/scabby grains after successful infection has been established (Jiang et al. 2006b). In this study, only resistance to DON accumulation was investigated.

After all the plants had matured, inoculated spikes and non-inoculated spikes for each replication were harvested separately and threshed carefully with a head thresher to retain the diseased kernels. Twelve to 15 scabby grains were randomly taken from the inoculated spikes to serve as a sample for DON test. DON extraction and analysis were based on a modified method of Mirocha et al. (1998). Briefly, seeds were weighed and placed into a 1-dram glass vial capped with a screw cap and extracted by soaking and shaking with 2 ml of acetonitrile/water (84/16 v/v) for 24 h. The extract was passed through a minicolumn packed with C_{18} and aluminum oxide. One and a half milliliters of the filtrate were placed into a ½-dram glass vial and evaporated to dryness under nitrogen. Twenty-five microliters of TMS reagent (TMSI/TMCS 100:1) was added, and the vial was rotated so that the reagent contacted with all residue in the vial. The vial was placed on a shaker for 10 min, and then 200 µl of isooctane were added followed by 200 µl of HPLC water to quench the reaction. After vortex, the upper layer was transferred to a GC vial. Selected ion monitoring (SIM) was used for GC/MS analysis (Shimadzu GCMS-QP2010, Shimadzu Corporation, Kyoto, Japan), with fragment ion (m/z value) of 235.10 as target ion and 259.10 and 422.10 as reference ions.

Estimation of grain yield loss

Grain yield loss caused by FHB can be defined as the amount of grain yield reduction in the field under disease conditions when compared with yield under no disease conditions. This parameter depends on many factors such as disease incidence, disease severity, kernel infection, etc. Clearly, it is difficult to directly determine and employ this measure in genetics and breeding research. Instead, the yield components would be the alternatives. In this study, the relative decreases of grain number per spike, 1,000-grain weight, and grain weight per spike were used as the parameters to evaluate the grain yield loss caused by FHB.

For Experiment 04, the total grains from inoculated spikes and non-inoculated spikes were counted separately for each pot. Grain weight per spike and 1,000-grain weight were determined as well. Then, to estimate the relative loss of grain yield caused by FHB, the relative decreases of grain number per spike, grain weight per spike (g) and 1,000-grain weight (g) were calculated using the following formula: $RD = (M_{CK} - M_{IS})/M_{CK} \times 100$, where RD = relative decrease, M_{CK} = the mean of the check (i.e., non-inoculated spikes), and M_{IS} = the mean of inoculated spikes.

QTL mapping and statistical analysis

ANOVA on the basis of replication means (Sokal and Rohlf 1981) was performed for single experiment and over all combination of three experiments based on a homogeneity test, respectively. Then broad-sense heritability on a line mean basis was estimated (Fehr 1987), and the exact confidence intervals for heritability were calculated (Knapp et al. 1985).

The genetic linkage map was constructed using simple sequence repeat (SSR) markers (Roder et al. 1998; Song et al. 2002, 2005; http://www.wheat.pw.usda.gov) and JoinMap version 3.0 (van Oijen and Voorrips 2001) (Jiang et al. 2007). QTL analysis was performed in Windows QTL Cartographer version 2.0 (Wang et al. 2001–2004). Single marker analysis (SMA), interval mapping (IM), and composite interval mapping (CIM) were performed, respectively. Multiple interval mapping (MIM) was performed to detect the QTL × QTL interaction (epistasis). The QTL × environment (E) interaction was detected using the JZmapqtl program. A LOD value of 2.0 was set as the threshold value, but all the QTLs with a significant LOD value for CIM under a 1,000-permutation test were presented and claimed. As described previously, a comparison between two groups of RILs carrying marker alleles from Veery and CJ 9306 was conducted based on the results of ANOVA for group comparison of QTL/marker alleles (Jiang et al. 2007), to verify the validation of QTLs/makers and to provide information for marker-assisted selection. Likewise, a comparison of different QTL/marker combinations for multiple loci was also computed. *Q*-test (Tukey–Kramer method) was used to examine the significance of differences (Sokal and Rohlf 1981).

Results

Phenotypic analysis of resistance to DON accumulation

The differences in DON concentration among RILs were highly significant for both single experiment data and combined analysis over all three experiments ($F = 3.1$-13.1, $P < 0.01$). The environmental differences and RIL × environment interactions were also significant ($F = 35.5$ and 3.6, respectively, $P < 0.01$). The average DON content of Veery was consistently significantly higher than that of CJ 9306 (Table 1). On three experiments, the average of the differences between Veery and CJ 9306 was 107 µg g^{-1}.

Table 1 Average, range and coefficient of variation, and broad-sense heritability of resistance to mycotoxin accumulation caused by Fusarium head blight (FHB) for an F_7 recombinant inbred line (RIL) population derived from the wheat cross Veery/CJ 9306 based on deoxynivalenol (DON) concentration (μg g^{-1}) over three greenhouse experiments

Population	2002	2002a	2004	Combined
Veery	134.2 ± 15.7	83.1 ± 2.1	134.1 ± 20.6	117.1 ± 11.2
CJ 9306	21.7 ± 3.1	0.3 ± 0.1	7.7 ± 4.1	9.9 ± 3.4
RILs				
Mean ± SE	65.7 ± 4.7	43.5 ± 3.9	58.9 ± 4.4	56.4 ± 3.5
Range	0–365.3	0–424.9	0.2–274.8	0.1–277.8
CV %	85.6	111.8	92.0	75.9
$LSD_{5\%}$	88.8	52.3	41.4	33.3
h_B^2	0.674	0.849	0.924	0.705
$h_B^2$90% confidence interval	0.571–0.752	0.802–0.885	0.900–0.942	0.626–0.765

The average of DON concentration for the RIL population was 56 μg g^{-1} with a large range of variation. Frequency distribution was continuous and obviously skewed toward the resistant parent (Fig. 1). The estimates of heritability in broad sense were moderate to high, depending on environments, and the estimate on the basis of 3-year combined analysis was 0.71.

QTL mapping and analysis of resistance to DON accumulation

Four QTLs contributing to resistance to DON accumulation caused by FHB were detected in CJ 9306 by interval mapping (IM) and composite interval mapping (CIM) analyses. They were located on 3BS, 2DL, 1AS and 5AS, respectively (Table 2). The two major QTLs (*QFhs.ndsu-3BS* and *QFhs.nau-2DL*) were consistently detected by three individual experiments, and they separately explained up to 23 and 20% of phenotypic variation for the overall average of three experiments. The other two QTLs (*QFhs.nau-1AS* and *QFhs.nau-5AS*) could separately explain 4–6% of phenotypic variation. The mappings for these QTLs based on CIM analysis of DON concentration were shown in Fig. 2.

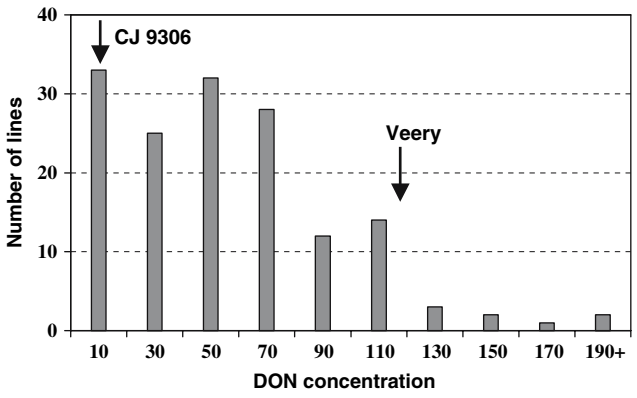

Fig. 1 Frequency distribution of 152 F_7 RILs derived from a wheat cross Veery/CJ 9306 for deoxynivalenol (DON) concentration (μg g^{-1}) in the scabby grains caused by Fusarium head blight (FHB) after single-floret inoculation over three greenhouse experiments

Multiple interval mapping (MIM) analysis did not detect significant epistatic interactions between the QTLs detected by CIM. However, by ZJmapqtl program and ANOVA of marker alleles, a significant QTL × E interaction was detected for *QFhs.nau-2DL* ($P < 0.01$). The differences among years/experiments were also significant for all the four QTLs ($P < 0.001$).

Comparison of QTL/marker alleles for resistance to DON accumulation

As shown in Table 2, all the four QTLs showed positive additive effects on the resistance to DON accumulation. In comparison, the additive effects of the major QTL *QFhs.ndsu-3BS* or *QFhs.nau-2DL* were approximately twice the effects of *QFhs.nau-1AS* or *QFhs.nau-5AS*. The averages of RILs carrying alternative QTL alleles (SSR markers) associated with the resistance and the differences between them were presented in Table 3. For all the four QTLs, favorable alleles from CJ 9306 significantly enhanced the resistance in all cases without exception, although the year/environment effects were significant. On average of three experiments, the CJ 9306 alleles for either marker of *Xgwm533b* linked to *QFhs.ndsu-3BS* or *Xgwm539* linked to *QFhs.nau-2DL* could decrease DON contents in scabby grains by 44–45% (or 32 μg g^{-1}). The favorable alleles from CJ 9306 for the QTLs *QFhs.nau-1AS* and *QFhs.nau-5AS* lead to a decrease of 36 and 26% (or 24 and 18 μg g^{-1}) in DON concentration, respectively.

Effects of two- and three-locus QTL/marker combinations on DON accumulation

For two-locus combinations, the averages of RILs bearing favorable alleles at two loci for DON concentration in scabby grains were highly significantly ($P < 0.01$) smaller than those of the reciprocal genotypes (i.e., the alleles at both loci were derived from Veery) in all cases (Table 3).

Table 2 QTLs for resistance to deoxynivalenol (DON) accumulation caused by Fusarium head blight (FHB) in the novel wheat germplasm CJ 9306 detected by composite interval mapping (CIM) with an F_7 RIL population derived from the cross Veery/CJ 9306

Experiment	QTL	Interval	Chromosome	Region length (cM)	LOD	Additive effects	R^2 (%)
2002	*QFhs.ndsu-3BS*	*Xgwm533b–Xgwm493*	3BS	15.5	5.0**	22.8	13.0
	QFhs.nau-2DL	*Xgwm157–Xwmc243a*	2DL	14.9	4.4**	20.7	13.5
	QFhs.nau-1AS	*Xbarc148– Xwmc024*	1AS	15.3	1.9*	14.3	4.9
2002a	*QFhs.ndsu-3BS*	Xgwm533b–Xgwm493	3BS	15.5	2.2**	6.0	12.3
	QFhs.nau-2DL	*Xgwm157–Xwmc243a*	2DL	14.9	6.9**	20.5	17.3
2004	*QFhs.ndsu-3BS*	Xgwm533b–Xgwm493	3BS	15.5	8.9**	27.7	22.6
	QFhs.nau-2DL	*Xgwm157–Xwmc243a*	2DL	14.9	1.5*	11.7	4.6
	QFhs.nau-1AS	*Xbarc148– Xwmc024*	1AS	15.3	1.8*	13.5	5.1
	QFhs.nau-5AS	*Xgwm425–Xbarc186*	5AS	7.0	1.5*	10.8	4.2
Average (3 years)	*QFhs.ndsu-3BS*	Xgwm533b–Xgwm493	3BS	15.5	9.4**	21.6	22.8
	QFhs.nau-2DL	*Xgwm157–Xwmc243a*	2DL	14.9	6.9**	19.3	19.9
	QFhs.nau-1AS	*Xbarc148– Xwmc024*	1AS	15.3	2.7**	10.7	5.9
	QFhs.nau-5AS	*Xgwm425–Xbarc186*	5AS	7.0	2.1**	10.0	5.2

*, **Significant at $P < 0.05$ and 0.01, respectively, under a permutation test for 1,000 times

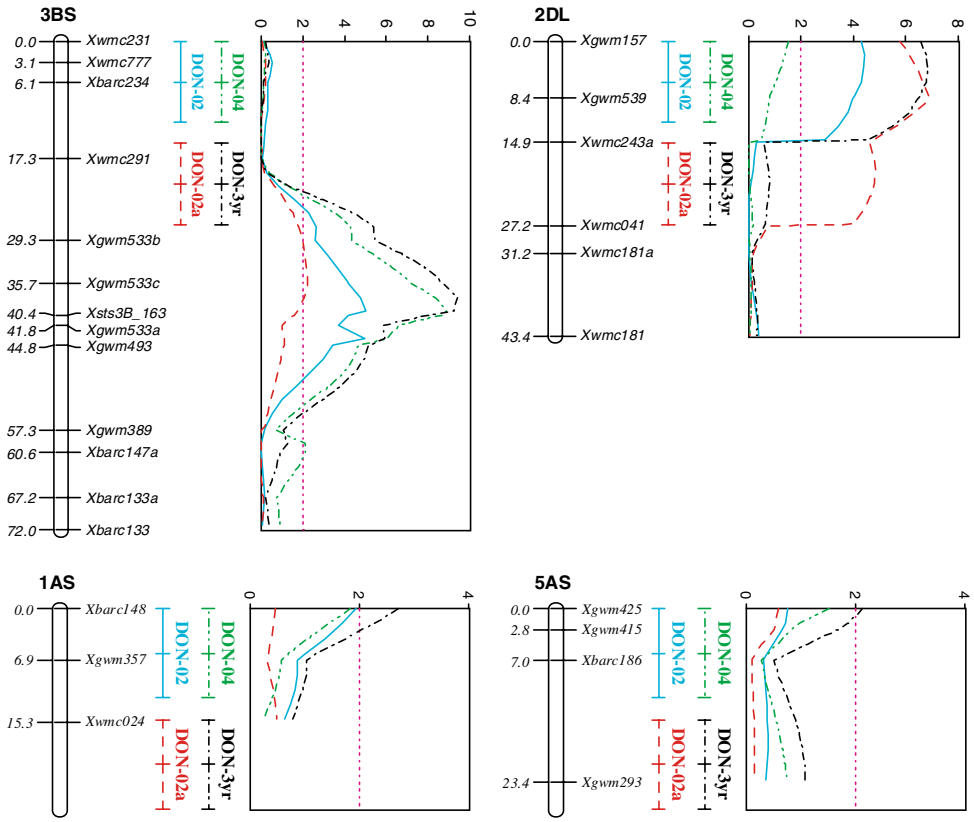

Fig. 2 QTL mapping for resistance to mycotoxin accumulation caused by Fusarium head blight in an F_7 RIL population derived from the wheat cross Veery/CJ 9306 based on CIM analysis of deoxynivalenol (DON) concentration in the scabby grains after single-floret inoculation in the greenhouse

Likewise, the genotypes/RILs bearing favorable alleles at both loci were significantly ($P < 0.05$) or highly significantly better than those genotypes/RILs with favorable alleles at either locus only, with only a few exceptions. Among all the possible two-locus combination, *QFhs.ndsu-3BS* + *QFhs.nau-2DL* was the best one, leading to a greatest decrease of DON accumulation, 59 μg g^{-1} or 66% on average of three experiments, compared with the reciprocal genotypes without favorable alleles at either locus. The combination *QFhs.nau-2DL* + *QFhs.nau-5AS* reduced DON concentration by 53 μg g^{-1} or 65%.

Comparisons of three-locus combinations indicated that, based on a favorable combination for two major QTLs, *QFhs.ndsu-3BS* + *QFhs.nau-2DL*, the addition of another QTL *QFhs.nau-1AS* or *QFhs.nau-5AS* could further decreased the DON contents to some extent, but not signifi-

Table 3 Average of RILs carrying different alleles of QTLs and QTL combinations for FHB resistance in the F_7 RIL population derived from the wheat cross Veery/CJ 9306 for deoxynivalenol (DON) concentration (μg g^{-1}) over three greenhouse experiments

QTL (marker) or QTL combination	Allele[a]	2002	2002a	2004	Average
QFhs.ndsu-3BS (Xgwm533b)	V	82.4 ± 6.7[c]	56.6 ± 7.6	78.6 ± 7.8	72.9 ± 5.8
	CJ	46.7 ± 5.5	33.2 ± 4.3	41.3 ± 5.1	40.9 ± 3.9
	Difference [b]	35.8****	23.4***	37.3****	32.0****
QFhs.nau-2DL (Xgwm539)	V	81.6 ± 6.4	62.7 ± 6.3	65.5 ± 6.7	70.5 ± 5.0
	CJ	44.9 ± 5.2	20.4 ± 2.8	51.1 ± 5.8	38.8 ± 4.0
	Difference	36.6****	42.3****	14.4*	31.7****
QFhs.nau-1AS (Xbarc148)	V	76.6 ± 6.6	52.0 ± 7.2	71.8 ± 7.4	66.8 ± 5.6
	CJ	51.0 ± 6.9	33.1 ± 5.1	42.7 ± 5.9	42.7 ± 5.1
	Difference	25.6**	18.9**	29.1***	24.1****
QFhs.nau-5AS (Xgwm425)	V	78.6 ± 7.1	51.7 ± 8.0	74.0 ± 8.3	67.9 ± 6.1
	CJ	58.0 ± 6.4	39.5 ± 4.6	49.6 ± 5.4	50.0 ± 4.5
	Difference	20.5*	12.2*	24.4***	17.9****
3BS + 2DxL	V + V	104.3 ± 8.3	78.7 ± 11.4	87.7 ± 11.2	90.0 ± 7.8
	V + CJ	48.5 ± 7.3	24.7 ± 5.0	65.5 ± 11.2	46.2 ± 7.0
	CJ + V	53.0 ± 7.2	49.8 ± 6.7	45.1 ± 8.4	50.7 ± 5.7
	CJ + CJ	41.5 ± 8.5	16.4 ± 3.4	36.2 ± 6.0	30.9 ± 5.2
	R^2 (%)	21.4**	20.2**	11.2**	25.9**
3BS + 1AS	V + V	92.6 ± 8.6	67.4 ± 12.3	95.3 ± 10.7	84.7 ± 8.2
	V + CJ	62.8 ± 12.6	35.1 ± 9.0	56.5 ± 13.2	52.4 ± 10.1
	CJ + V	54.1 ± 9.2	35.4 ± 6.6	45.0 ± 9.2	45.8 ± 6.7
	CJ + CJ	40.9 ± 8.2	32.4 ± 6.7	35.1 ± 5.5	36.1 ± 5.6
	R^2 (%)	14.6**	7.0**	16.9**	17.9**
2DL + 5AS	V + V	94.5 ± 9.8	74.6 ± 13.7	75.7 ± 13.5	81.7 ± 9.6
	V + CJ	74.7 ± 9.5	58.3 ± 6.2	58.5 ± 7.8	64.8 ± 6.3
	CJ + V	60.9 ± 8.8	28.0 ± 4.8	72.1 ± 9.4	53.1 ± 6.5
	CJ + CJ	35.0 ± 5.3	13.3 ± 2.9	36.5 ± 7.0	28.3 ± 4.3
	R^2 (%)	13.7**	16.0**	5.2*	16.3**
3BS + 1AS + 5AS	CJ + CJ + V	53.3 ± 18.7	52.6 ± 17.6	41.0 ± 8.7	49.0 ± 12.2
	CJ + CJ + CJ	33.5 ± 9.9	25.3 ± 6.4	33.8 ± 8.4	30.8 ± 6.7
	R^2 (%)	16.7**	5.7*	19.6**	18.1**
2DL + 5AS + 1AS	CJ + CJ + V	51.6 ± 9.9	23.5 ± 5.3	45.3 ± 12.0	40.1 ± 7.8
	CJ + CJ + CJ	14.3 ± 5.6	8.0 ± 5.2	25.6 ± 9.8	16.0 ± 6.3
	R^2 (%)	16.3 **	11.1**	9.5**	16.9**

[a] *V* Homozygous alleles for Veery, and *CJ* Homozygous alleles for CJ 9306, respectively

[b] The positive value indicates that the favorable allele was from CJ 9306, and the significance of differences was on the basis of ANOVA results

[c] Mean ± standard error. The frequencies of lines with various levels of DON concentration for each genotype (or allele group) were continuously distributed, and no extremely high and low readings presented in most cases

*, **, ***, ****Significant at $P < 5,1,0.1$ and 0.01%, respectively

cant statistically. For the combinations *QFhs.nau-2DL* + *QFhs.nau-5AS* and *QFhs.ndsu-3BS* + *QFhs.nau-1AS*, however, *QFhs.nau-1AS* or *QFhs.ndsu-5AS* significantly increased the resistance (Table 3).

QTL analysis of resistance to grain yield loss

Based on the data of Experiment 04, QTL analysis was computed for the relative decreases of three yield components: grain number per spike, 1,000-grain weight, and grain weight per spike. The results indicated that the major QTL *QFhs.ndsu-3BS* was detected by IM and CIM for all the three parameters of resistance to grain yield loss in CJ 9306 (Fig. 3). The explained proportions of phenotypic variations were up to 8–15% (Table 4). The favorable alleles from CJ 9306 significantly decreased the losses of yield components caused by FHB ($P < 0.01$). In comparison, the differences between the reciprocal genotypes at this locus for the relative decrease of grain number per spike were smaller than those for 1,000-grain weight and grain weight per spike.

Discussion

QTLs for resistance to DON accumulation

Significant progress in development of QTL mapping and DNA markers enables a deep understanding of the underlying genetic basis of complex traits in plants. A joint analy-

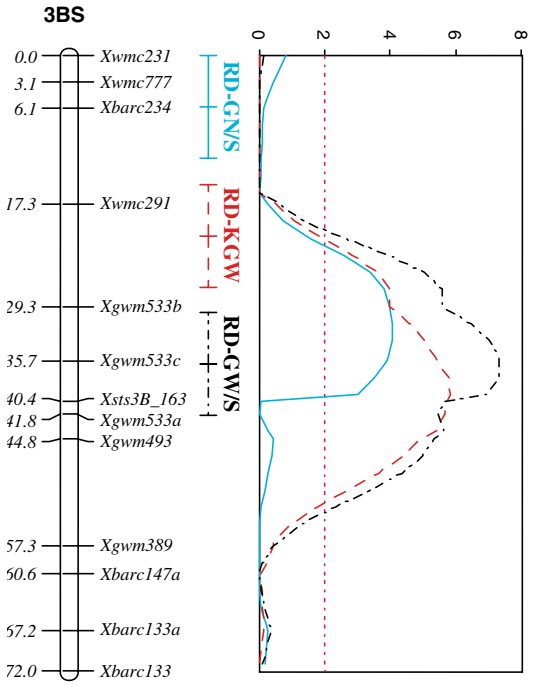

Fig. 3 QTL mapping on Chromesome 3BS for resistance to yield loss caused by Fusarium head blight in an F_7 RIL population derived from the wheat cross Veery/CJ 9306 based on CIM analysis of relative decrease in grain number per spike (RD-GN/S), 1,000-grain weight (RD-KGW), and grain weight per spike (RD-GW/S) after single-floret inoculation in the greenhouse (2004)

Table 4 LOD value, explained variation (R^2), additive effects of the major QTL on 3BS (*QFhs.ndsu-3BS*) for resistance to yield loss caused by Fusarium head blight (FHB) and the phenotypic difference between alternative alleles for the peak marker *Xgwm533b* in an F_7 RIL population derived from the wheat cross Veery/CJ 9306 (2004)

Statistics	Relative decrease (%)		
	Grain number per spike	1,000-grain weight (g)	Grain weight per spike (g)
LOD	3.9**	5.8**	7.3**
R^2 (%)	7.6	9.8	14.7
Additive effects	11.9	15.1	22.4
Veery alleles	15.6 ± 2.6	39.8 ± 2.9	47.3 ± 3.3
CJ 9306 alleles	2.8 ± 2.3	25.0 ± 2.6	25.6 ± 3.5
Difference	12.8**	14.8**	21.7**

**Significant at $P < 1\%$

sis of genotype marker segregation and phenotypic values of individuals or lines cannot only detect and localize QTLs, but may also quantify the effects of individual genes. So far, approximately 30 QTLs associated with resistance to FHB in wheat have been reported. Studies suggested that all the 21 chromosomes carried the resistance genes. However, the emphasis was placed on Type II resistance in most investigations. The technological requirements for DON testing retarded research on resistance to DON accumulation. Zhou et al. (2002a) compared the DON concentrations in two sets of Chinese Spring-Sumai 3 substitution lines. They found that chromosomes 3B and 7A from Sumai 3 reduced DON accumulation within the kernels, while 1B, 2D, and 4D from Sumai 3 increased DON concentration. Recently, Ma et al. (2006a) studied the effects of individual chromosome arms on FHB infection and DON accumulation using a set of ditelosomic lines derived from Chinese Spring. Their results suggested that chromosome arms 1DL, 2AL, 3AL, 1AL, 3BS, and 1BS might carry genes contributing to resistance to DON accumulation, whereas on 7AS, 4DS, 6AS, and 6DL there might be susceptibility factors or resistance suppressors. Somers et al. (2003) identified three QTLs on 3BS, 2DS and 5AS controlling resistance to DON accumulation in wheat cultivar Maringa. Two of them were validated in W14, except 2DS (Chen et al. 2006). More recently, Semagn et al. (2007) reported two major QTLs derived from a Norweigian line NK93604, which were located on chromosomes 1AL and 2AS.

In our study, four QTLs located on 3BS, 2DL, 1AS, and 5AS contributing to resistance to DON accumulation were detected in the highly FHB-resistant germplasm CJ 9306. *QFhs.ndsu-3BS* and *QFhs.nau-2DL* were consistently detected by three individual experiments and a combined analysis. Until now, no QTLs on 2DL have been claimed for resistance to DON accumulation in wheat, although a QTL for Type II resistance were reported previously (Somers et al. 2003; Mardi et al. 2005). Similarly to Type II resistance in previous report (Jiang et al. 2007), *QFhs.nau-2DL* had a significant QTL × E interaction for resistance to DON accumulation as well. For Type II resistance, the peak marker of this QTL was not consistently the same for individual experiments, and the effects were obviously smaller than those of *QFhs.ndsu-3BS* (Jiang et al. 2007). For resistance to DON accumulation, however, *QFhs.nau-2DL* exhibited a highly coincided peak marker, and was very similar to *QFhs.ndsu-3BS* in the expressed effects and explained variations. In other words, *QFhs.nau-2DL* played a greater role in resistance to DON accumulation than in resistance to fungal spread. It is clear that *QFhs.nau-2DL* in CJ 9306 had different alleles and functions/expressions from the QTL on 2DL reported by Somers et al. (2003).

In addition, no reports indicated that on 1AS there was a QTL associated with resistance to DON accumulation and other types of resistance. In this study, such a QTL (*QFhs.nau-1AS*) was detected in two of three experiments and overall combined analysis, and its phenotypic effects (differences between alternative alleles) were significant for all the cases (Table 3). Somers et al. (2003) suggested that the QTL on 5AS for DON resistance was independent of Type II resistance because that QTL was not detected for Type II resistance in the same study. However, our results indicated that *QFhs.nau-5AS* had significantly positive

effects on both of resistance to DON accumulation and Type II resistance (Jiang et al. 2007), even though it was weaker for the latter. It suggested that these two types of resistance were not completely independent of each other for this locus. It also could be supposed that the alleles of *QFhs.nau-5AS* in CJ 9306 and the QTL on 5AS in Maringa reported by Somers et al. (2003) were different, at least they differed in the functions or expressions. Compared with other major QTLs, such as *QFhs.ndsu-3BS*, this work and previous studies also suggested that this QTL was weaker for resistance to DON accumulation (Chen et al. 2006; Somers et al. 2003).

In conclusion, the QTLs *QFhs.nau-2DL* and *QFhs.nau-1AS* identified in this study were two novel QTLs/genes for resistance to mycotoxin accumulation caused by FHB in wheat, although they also significantly contributed to Type II resistance. *QFhs.ndsu-3BS* and *QFhs.nau-5AS* were not only associated with Type II resistance, but also contributed to resistance to DON accumulation. In comparison, *QFhs.ndsu-3BS* and *QFhs.nau-2DL* played a larger role as major genes. Other QTLs for Type II resistance, such as *QFhs.nau-7BS* in CJ 9306, *QFhs.nau-2BL* and *QFhs.nau-1BC* in Veery, were not detected for resistance to DON accumulation, suggesting that compared with Type II resistance, fewer genes were involved in resistance to DON accumulation in CJ 9306.

Effects of marker-assisted selection for two- and three-locus combinations

Somers et al. (2003) suggested that the genotypes fixed for two resistance QTLs had the lowest DON accumulation compared with the other three possible genotypes for the two-locus combination 3BS and 5AS. Marker-assisted selection (MAS) for favorable alleles at two loci was not significantly different from MAS for favorable alleles at either locus alone. In our study, the combination *QFhs.ndsu-3BS* + *QFhs.nau-5AS* exhibited a similar result (data not shown). For the other two-locus combinations, however, the averages of DON contents of RILs bearing favorable alleles at two loci were not only significantly smaller than those of the reciprocal genotypes (without favorable alleles at both loci) in all cases, but also significantly smaller than those of RILs with favorable alleles at only one locus in most cases (Table 3). Other previous studies also suggested that the average of the lines with favorable alleles at both loci for disease severity was significantly lower than that of the lines with favorable alleles at either locus alone (Buerstmayr et al. 2002; 2003; Steiner et al. 2004; Guo et al. 2006). Therefore, the effects of individual QTLs could be accumulated. MAS for favorable alleles at two loci would lead to better improvement than MAS for one locus only. Among all the possible two-locus combinations, *QFhs.ndsu-3BS* + *QFhs.nau-2DL* and *QFhs.nau-2DL* + *QFhs.nau-5AS* exhibited lowest DON concentration and a greatest reduce in DON accumulation as well. Thus these two combinations should be the optimum choices for MAS for resistance to DON accumulation. By comparison, the combination of *QFhs.nau-2DL* and *QFhs.nau-5AS* exhibited greater contributions to resistance to DON accumulation than to Type II resistance, as either of them did alone.

On the basis of a favorable combination for two QTLs, *QFhs.ndsu-3BS* + *QFhs.nau-2DL* or *QFhs.nau-2DL* + *QFhs.nau-5AS*, the third minor QTL *QFhs.nau-1AS* and/or *QFhs.nau-5AS* could further enhance the resistance to some extent, though the decreased values of DON contents were not significant in most cases. The one-major-one-minor QTL combination (*QFhs.nau-2DL* + *QFhs.nau-5AS* or *QFhs.ndsu-3BS* + *QFhs.nau-1AS*) could be significantly benefited from the addition of another minor QTL (*QFhs.nau-1AS* or *QFhs.ndsu-5AS*) (Table 3). The results on Type II resistance also suggested that additional favorable allele at the third QTL could significantly enhance the resistance in most cases (Jiang et al.2007). Therefore, it seems that the third QTL also deserved to be included in MAS, regardless of Type II resistance or resistance to DON accumulation and for both.

In this study, we used markers to define QTLs and to combine the QTLs for the purpose of simplification. However, markers per se are not the genes/QTLs although DNA markers are associated with genes/QTLs. Here we would point out that there might be some risk for doing this if the adjacent markers are over 10 cM apart. The reliability and effects of selection would be decreased as the distance between markers increases.

QTL × E and QTL × QTL interactions

The results of this study indicated that the environmental differences in DON concentration between years/experiments were highly significant for both ANOVA on an RIL basis and on individual QTLs. Genotype/RIL × year/experiment interaction was also significant (Somers et al. 2003). Even so, the correlations between years/experiments were also highly significant (the average coefficient of correlation $r = 0.46$, $P < 0.01$). Compared with Type II resistance, resistance to DON accumulation is more variable and instable. Therefore, repeated determinations of DON concentration were very important for accurate evaluation of the resistance and characterization of the associated QTLs. Among the four detected QTLs controlling resistance to DON accumulation, a QTL × E interaction was significant only for *QFhs.nau-2DL*, but not for the other three. This indicates that these QTLs are applicable to MAS in breeding programs.

Similarly to Type II resistance (Jiang et al. 2007), no significant QTL × QTL interaction (epistasis) was detected for resistance to DON accumulation in this study. It was further demonstrated that FHB resistance (Type II resistance and/or resistance to mycotoxin accumulation) in CJ 9306 was inherited predominantly in an additive-dominance model (Jiang and Ward 2006). However, some of other studies suggested significant QTL × QTL interactions (Jia et al. 2005; Yang et al. 2005b; Guo et al. 2006; Lin et al. 2006; Ma et al. 2006b). It seems that the inheritance model might differ depending on specific resources of resistance and investigated populations/crosses.

QTLs for resistance to grain yield loss

Up to now, resistance to grain yield loss caused by FHB has been involved in only a few researches (Mesterhazy 1995; Mesterhazy et al. 1999; Browne et al. 2005), especially few on QTL analysis (Pumphrey et al. 2007). For simplicity, the percentage of Fusarium-damaged kernels (FDK) was investigated (Yang et al. 2005b; Chen et al. 2006), while the decrease of 1,000-kernel weight or grain weight per spike was rarely measured (Mesterhazy et al. 1999). Based on 1 year field data in a double haploid (DH) population (DH181/AC Foremost), Yang et al. (2005b) suggested six QTLs associated with resistance to kernel infection, locating on chromosomes 1DL, 2DS, 3BS, 3BC, 4DL, and 6BS, respectively. Two QTLs on 3BS and 5AS associated with FDK were also validated in a 1 year greenhouse experiment with a DH population of W14/Pion2684 (Chen et al. 2006). More recently, Pumphrey et al. (2007) validated QTL *Fhb1* (or *QFhs.ndsu-3BS*) for scabby kernels and kernel weight of spikes harvested from the field nurseries, using near-isogenic lines developed from wheat breeding populations.

In this study, we tried to use three parameters (the relative decreases of grain number per spike, 1,000-grain weight, and grain weight per spike) to identify the QTLs for resistance to grain yield loss caused by FHB. The results indicated that the major QTL *QFhs.ndsu-3BS* was detected by all the three parameters in CJ 9306, significantly decreasing the losses of yield components caused by FHB. It further demonstrated that this QTL or gene *Fhb1* (Cuthbert et al. 2006) played an important role in resistance to kernel infection (Yang et al. 2005b; Chen et al. 2006; Pumphrey et al. 2007), in addition to Type II resistance (Anderson et al. 2001; Buerstmayr et al. 2002, 2003; Lin et al. 2004), Type I resistance (Yang et al. 2005b; Lin et al. 2006), and resistance to DON accumulation (Somers et al. 2003; Chen et al. 2006). Therefore, this QTL/gene could be widely used in MAS programs, although the effects may vary much with different backgrounds, types and parameters of resistance, inoculation techniques, and screening pressures (Pumphrey et al. 2007). No more other QTLs were detected for all the three parameters of resistance to yield loss. It suggested that it might be harder to identify the QTLs for resistance to grain yield loss, and probably there were no independent genes/QTLs from Type II resistance. More work would be helpful.

For resistance to grain yield loss, in comparison, grain weight per spike and 1,000-grain weight showed a higher heritability than grain number per spike (Jiang et al. 2006b). This study indicated that the LOD values, explained variations, and additive effects of the QTL *QFhs.ndsu-3BS* for the relative decreases of grain weight per spike and 1,000-grain weight were also greater than those for grain number per spike. Therefore, grain weight per spike and 1,000-grain weight would be more effective and valuable than grain number per spike in studies on resistance to yield loss caused by FHB.

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