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ORIGINAL PAPER

Identification and molecular mapping of two QTLs with major effects for resistance to Fusarium head blight in wheat

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Abstract Fusarium head blight (FHB) is a devastating disease of wheat worldwide. Novel sources of resistance are critical for improving FHB resistance levels in wheat. From a large-scale evaluation of germplasm for reactions to FHB, we identified one wheat accession (PI 277012) that consistently showed a high level of resistance in both greenhouse and field experiments. To characterize the FHB resistance in this accession, we developed a doubled haploid (DH) mapping population consisting of 130 lines from the cross between PI 277012 and the hard red spring wheat cultivar 'Grandin'. The DH population was then evaluated for reactions to FHB in three greenhouse seasons and five field environments. Based on a linkage map that consisted of 340 SSR markers spanning 2,703 cM of genetic distance, two major quantitative trait loci (QTLs) for FHB resistance were identified on chromosome arms 5AS and 5AL, with each explaining up to 20 and 32% of the variation in FHB severity, respectively. The two QTLs also showed major effects on reducing the percentage of Fusarium damaged kernels (FDK) and deoxynivalenol (DON) accumulation in seeds. FHB resistance has not previously been reported to be associated with this particular genomic region of chromosome arm 5AL, thus indicating the novelty of FHB resistance in PI 277012. Plant maturity was not associated with FHB resistance and the effects of plant height on FHB resistance were minor. Therefore, these results suggest that PI 277012 is an excellent source for improving FHB resistance in wheat. The markers identified in this research are being used for marker-assisted introgression of the QTLs into adapted durum and hard red spring wheat cultivars.

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Introduction

Fusarium head blight (FHB), caused by *Fusarium graminearum* Schwabe [teleomorph *Gibberella zeae* (Schw.) Petch], is a devastating disease of wheat (*Triticum aestivum* L.) in most wheat-growing regions worldwide. The disease can cause serious losses in grain yield and have devastating effects on seed and bread-making quality. The toxic fungal secondary metabolite deoxynivalenol (DON) is produced by the pathogen and is harmful to consumers' health in

addition to its detrimental effects on end-use quality. Development and utilization of FHB-resistant wheat cultivars are generally recognized as the most effective and economical strategy to control the disease. Therefore, the identification of sources with high levels of FHB resistance is a critical step toward the development of resistant cultivars. Particularly, the utilization of a wide range of resistance sources in wheat breeding may be necessary for developing wheat cultivars with sustainable and durable resistance to FHB.

FHB resistance in wheat is a very complex trait, and resistance components include the previously classified Type I (resistance to initial infection) and Type II (resistance to fungal spread from the infected floret along the rachis) (Schroeder and Christensen 1963) categories, as well as the recently added category for resistance to DON contamination and accumulation (Mesterhazy et al. 1999). In most of the current wheat breeding programs, the most important and widely used FHB resistance source is the Chinese cultivar 'Sumai 3' and other lines derived from Sumai 3, such as Ning 7840 and Ning 8331 (reviewed by Buerstmayr et al. 2009). The high level of FHB resistance in Sumai 3 may be largely due to the fact that it harbors both Type I (Buerstmayr et al. 2003) and Type II (Anderson et al. 2001; Waldron et al. 1999; Zhou et al. 2002) resistance as well as resistance to DON accumulation (Lemmens et al. 2005). The Brazilian cultivar 'Frontana' has been suggested as a FHB resistance source as well (Schroeder and Christensen 1963), but recent reports indicate that it has only moderate Type I resistance that might be related to its morphologic traits such as hard glumes and narrow flower opening (Buerstmayr et al. 2009). Other proposed resistance sources such as the Swiss cultivar 'Arina' (Ruckenbauer et al. 2001; Paillard et al. 2004) and the Chinese landrace 'Wangshuibai' (Jia et al. 2005; Lin et al. 2006) either showed only moderate resistance or had undesired agronomic traits and thus have not been widely used. Therefore, Sumai 3 remains the most widely accepted resistance source in most breeding programs. It is essential to identify additional acceptable sources of resistance in order to obtain durable resistance by diversifying and stacking resistance genes.

Strong genotype-by-environment ($G \times E$) interactions associated with FHB resistance (Campbell and Lipps 1998; Fuentes et al. 2005) also complicate phenotypic evaluation and genetic analysis of FHB resistance. Quantitative trait loci (QTLs) analysis can allow the identification of genomic regions associated with resistance, and the markers linked to those regions can be used in marker-assisted selection schemes without the added concern of environmental effects. To date, numerous FHB resistance QTLs have been identified on all of the wheat chromosomes except chromosome 7D (reviewed by Buerstmayr et al. 2009). However, only three QTLs that are located on chromosome arms 3BS (*Fhb1*) (Anderson et al. 2001; Bai et al. 1999; Liu et al. 2006; Waldron et al. 1999), 5AS (*Qfhs.ifa-5A*) (Buerstmayr et al. 2002; Somers et al. 2003) and 6BS (*Fhb2*) (Anderson et al. 2001; Cuthbert et al. 2007) are the most validated major QTLs. Buerstmayr et al. (2009) summarized over 100 QTLs from 52 reports and found that only 22 QTLs were detected in more than one mapping population. One reason for a significant portion of the reported QTLs not being repeatable may be due to the fact that their effects were too small or they were falsely identified due to phenotypic bias caused by environmental effects. Previous experiments indicate that QTLs with larger effects are the most reliable, and diagnostic markers linked to major QTLs, if developed, are useful for assisting in the selection of the underlying resistance factors (Liu et al. 2008).

In an effort to identify new sources of robust FHB resistance, we have evaluated a large number of tetraploid (*T. turgidum* L.) accessions since 2005 (Xu et al. 2007; Oliver et al. 2008, Chu et al. 2010). During the evaluation, we found that a hexaploid spring wheat line PI 277012, which was mistakenly classified as emmer wheat [*T. turgidum* subsp. *dicoccum* (Schrank ex Schübler) Thell (http://www.ars-grin.gov/cgi-bin/npgs/acc/display.pl?1206987)], consistently exhibited a high level of FHB resistance in both greenhouse and field environments (Xu et al. 2010; Chu et al. 2010). It showed a similar level of type II FHB resistance as Sumai 3, but marker haplotype analysis indicated that the line does not carry *Fhb1* from Sumai 3 (Xu et al. 2010). The objective of this study was to identify and map QTLs associated with resistance to FHB in PI 277012.

Materials and methods

Plant materials

The wheat accession PI 277012 used in this study was originally obtained from the USDA-ARS National Small Grain Collection in Aberdeen, Idaho. It has consistently showed a high level of resistance to FHB in both greenhouse and field evaluations since 2007 (Xu et al. 2010). PI 277012 was originally developed in Estacion Experimental de Aula Dei, Zaragoza, Spain, and has a pedigree of 'Extremo Sur'/'Argelino'//*T. timopheevii*. It is currently classified as *T. dicoccum* (Schrank ex Schübler) Thell (http://www.ars-grin.gov/cgi-bin/npgs/acc/display.pl?1206987). However, we verified that PI 277012 is actually a hexaploid ($2n = 6x = 42$, AABBDD genomes) on the basis of plant morphology (Fig. 1), seed storage protein analysis, and cytogenetic analysis (Xu et al. 2010; http://www.uky.edu/Ag/Wheat/wheat_breeding/New%20Folder/Steve%20Xu.pdf). To identify and map QTLs for resistance to FHB in PI 277012, we

Fig. 1 Spike morphology of wheat accession PI 277012 at about 3 weeks post point-inoculation for Fusarium head blight in the greenhouse

developed a doubled haploid (DH) population consisting of 130 lines from a cross between the hard red spring wheat cultivar 'Grandin' (PI 531005), which is susceptible to FHB, and PI 277012 using the methods described in Chu et al. (2008).

Evaluation for reaction to FHB

The 130 DH lines derived from the cross Grandin × PI 277012 and their parents were evaluated for resistance to FHB in both greenhouse and field trials. A randomized complete block design was used in all experiments. Greenhouse assessments were conducted in three growing seasons including the fall of 2009 (09GH) and the early spring (10GH1) and summer (10GH2) of 2010. In each growing season, the DH lines and their parents were grown in deep tree pots with two plants per pot. The greenhouse was supplemented with artificial light for a 16-h photoperiod with the temperature maintained between 22 and 25°C. Two replicates were used in each greenhouse experiment and each replicate consisted of two pots (four plants each). Therefore, a total of eight plants per genotype were evaluated in each greenhouse experiment. The inoculum was prepared at a concentration of 50,000 spores mL^{-1} from three strains of pathogenic *F. graminearum*. FHB inoculations were performed using the single-spikelet inoculation method described by Stack et al. (2002), by injecting 10 μL of the suspension into one floret of a single central spikelet per spike at anthesis. Approximately, 25–30 spikes per line in each replicate were inoculated. The inoculated spike was then covered by a 5 in. plastic bag with light misting for 72 h to keep the humidity high. Disease severity was scored as percentage of diseased spikelets per spike through visually counting the number of diseased and total spikelets per spike at 21 days post inoculation.

Field evaluations were performed in 2 years with two locations in 2009 including Fargo (09Far) and Prosper (09Pro), North Dakota. The second year (2010) included the locations of Fargo (10Far), Prosper (10Pro), and Langdon (10Ldn), North Dakota. Two replicates were used in each field location in 2009 due to a limited number of seeds, but three replicates were used for all field locations in 2010. In all field experiments, the DH lines and their parents were grown in hill plots (12–15 seeds per hill) with four hills in a 1.2 m row and spaced 0.30 m apart. The grain inoculum method of inoculation described by Stewart (2003) was used. At about 3 weeks before the earliest lines started to flower, autoclaved corn (*Zea Mays* L.) seeds infected with three strains of pathogenic *F. graminearum* were evenly distributed among field plots at a rate of about 36 g m^{-2}. Plots were then misted for 5 min in 15-min intervals for 12 h daily (4:00 p.m. to 4:00 a.m.), until 14 days after anthesis of the latest maturing lines. Because Langdon had much lower air temperatures than Fargo and Prosper, the mist cycles in Langdon were set to 5 min per 60 min and discontinue completely when the maximum daily air temperatures were less than the 30-year normal and precipitation events occurred (Oliver et al. 2008). Disease severity for each line was scored at 21 days post anthesis in four environments (09Far, 10Far, 10Pro, and 10Ldn). Due to the low temperature and dry weather in Prosper during early and mid July of 2009, the FHB disease was not well developed until the fourth week post anthesis. Thus, disease severity for each line was scored at 28 days post anthesis in the Prosper nursery in 2009. The disease severity was recorded as the percentage of infected spikelets on each head estimated based on the scale of ten categories of infection (0, 7, 14, 21, 33, 50, 66, 75, 90, and 100%) described by Stack and McMullen (1998). Ten spikes were scored in each plot, and the disease severity was calculated by averaging the severities of all heads.

In addition, the percentage of Fusarium damaged kernel (FDK) and the content of deoxynivalenol (DON) produced by *F. graminearum* were determined for all field experiments. To prepare the seed samples used for scoring FDK and DON content, all the spikes in each hill in all field experiments were hand harvested at maturity. The harvested spikes from the Fargo and Prosper nurseries were threshed using a Wintersteiger LD 180 Laboratory Thresher (Wintersteiger, Salt Lake City, UT, USA). The air current for separating the kernels from chaff and other residues in the thresher was adjusted to the lowest setting to retain all the light scabby kernels. The threshed kernels

were then carefully cleaned by hand. The spikes harvested in Langdon were initially threshed using a Hege small plot combine (Wintersteiger Hege, Ankeny, IA, USA) with blower fan speed low. The kernels were then carefully separated from the chaff by hand. An approximately equal amount of grain from each line of each replicate within each environment was combined, and a random sample of 200 seeds from this mixture was used to estimate FDK and reported as a percentage of diseased seeds. Seed sample sizes of approximately 20 g of each line taken randomly from the mixed grain samples were used for DON analyses. DON content was determined as the amount of DON (mg/kg) in the wheat kernels using gas chromatography–mass spectrometry (GC–MS) according to Mirocha et al. (1998) and Fuentes et al. (2005). For testing distribution of disease severity, FDK and DON content in the DH population, the Shapiro–Wilk normality test was performed using PROC UNIVARIATE (SAS Institute 2004).

To determine if FHB resistance was related to plant height and maturity, days to heading and plant height data were collected for all field experiments. Days to heading were recorded as the number of days from planting to the emergence of spikes in about 50% of the plants. Plant height was recorded by measuring the plants from the base to the tallest spike at maturity, excluding the awns. Correlations between FHB resistance and plant height or maturity were calculated using Statistical Analysis System version 9.1 (SAS Institute 2004).

Marker generation, linkage map construction and QTL analysis

In the survey of using 1,500 SSR (simple sequence repeat) primer pairs to detect polymorphism between PI 277012 and Grandin, 589 pairs (39.3%) amplified polymorphic bands, indicating about 40% of polymorphism between the two parents. According to the marker position in Somers et al. (2004) and Sourdille et al. (2003), a total of 280 primer sets including 50 BARC (Song et al. 2005), 85 GWM (Röder et al. 1998a, b), eight GDM (Pestsova et al. 2000), 13 CFA (Sourdille et al. 2003), 27 CFD (Guyomarc'h et al. 2002), 95 WMC (Somers et al. 2004) and two DuPw (Eujayl et al. 2002) were used to genotype the DH population for developing linkage maps. SSR fragments were amplified by PCR following the conditions described by Somers et al. (2004). SSRs were separated by capillary electrophoresis using an ABI 3130xl Genetic Analyzer (Applied Biosystems, Foster City, CA, USA) following the procedures of Chao et al. (2007).

In addition to SSR markers indicated above, one additional primer pair BF (5′-GGTAGGGAGGCGAGAGGCG AG-3′) and WR1 (5′-CATCCCCATGGCCATCTCGAG CTG-3′) described by Ellis et al. (2002) was used for detecting *Rht-B1* locus in each DH line for analyzing the genetic control of plant height in the DH population. The primer pair Qsnp2A.F (GCACTAGCTAATTCAGTGGT-TAGATTTGCTCA) and Qsnp.R (ATTCAGTGGTAG-CAACAGTTTCAGTAAGCTGG) (Simons et al. 2006) was used for determining alleles at the *Q* locus in each DH line, because PI 277012 is non-free threshing and likely harbors the *q* allele.

The computer program MAPMAKER (V2.0) for Macintosh (Lander et al. 1987) was used to perform linkage analysis with a minimum logarithm of the odds (LOD) threshold of 3.0 and the Kosambi mapping function (Kosambi 1944). Linkage groups were first identified using the ''two-point/group'' command with a minimum LOD = 3.0 and a maximum $\theta = 0.40$, which yielded several very large linkage groups. Each large group was then regrouped using a minimum of LOD = 10.0. The FIRST ORDER and RIPPLE (LOD > 3.0) commands were used to determine the most plausible order of markers within linkage groups. The TRY command was used to add markers that did not RIPPLE at an LOD > 3.0 to the established framework maps. Positions of centromeres on the maps were estimated according to the published physical maps (Sourdille et al. 2004).

For QTL analysis, composite interval-regression mapping (CIM) was performed using the computer program Map Manager QTX (Manly et al. 2001) to evaluate marker intervals associated with FHB resistance and the other traits such as plant height and days to heading. A permutation test with 1,000 permutations indicated that a LOD threshold of 3.03 in this population yielded an experiment-wise significance level of 0.05. Markers with significant ($P < 0.001$) main effects were tested against all other markers (Manly et al. 2001) in the dataset to identify significant ($P < 10^{-6}$) interactions among QTLs. To find the significance of the identified resistance QTLs under each environment, data collected from each experiment were separately used for QTL analysis, and only the QTLs that were significant in most of the environments were reported. To detect the possible G × E interactions between the resistance QTLs and environment, genotype data of peak markers for each QTL along with disease severity data were used for variance analysis through the GLM procedure in Statistical Analysis System (version 9.1) (SAS Institute 2004) with the environment set as the random effect.

Results

Linkage maps construction in the Grandin × PI 277012 DH population

A total of 340 markers generated by 280 SSR primer pairs, along with two markers specific for genes *Rht-B1* and

Q were used to assemble the 21 linkage groups (Fig. 2, Online Resource Table S1). All linkage groups were anchored to specific chromosomes according to the genetic and physical maps reported by Somers et al. (2004) and Sourdille et al. (2003), respectively. The total length of the linkage maps spanned 2,702.9 cM with an average marker density of 7.9 cM per marker (Online Resource Table S1, Fig. 2). Overall, the orders and distances of most of the SSR markers on our maps are consistent with those in genetic maps developed by Somers et al. (2004). In addition, the two markers specific for *Rht-B1* and *Q* were mapped to chromosome arms 4BS and 5AL, respectively, in positions very similar to those of previous reports (Ellis et al. 2002; Simons et al. 2006).

The total map lengths were 980.4, 870.9 and 851.6 cM for the A, B and D genomes, respectively (Online Resource Table S1). The B genome linkage maps had an average density of 7.0 cM per marker and were more dense than those of the A genome (8.0 cM/marker) and the D genome (9.1 cM/marker). Map lengths for individual chromosomes ranged from 34.6 cM (chromosome 4B) to 178.7 cM (chromosome 7B) (Online Resource Table S1, Fig. 2). Many chromosomes (1A, 2B, 2D, 3A, 4A, 4D, 5B, 6A, 6B, 7A and 7B) contained gaps greater than 30 cM (Fig. 2, Online Resource Table S1). Clustering of markers near centromeric or non-centromeric regions was observed (Fig. 2).

FHB reaction, plant height and days to heading in the DH population

Distribution of FHB disease severity varied among different experiments and transgressive segregation was observed for most experiments (Table 1). Overall, the distribution of disease severity indicated that FHB resistance in the population was quantitatively controlled because continuous variation was observed among DH lines for each experiment. Among all experiments, disease severity in Grandin ranged from 30 to 95% with most above 50%, whereas that of PI 277012 was never greater than 14% (Table 1).

FDK and DON content of DH lines showed distribution patterns similar to that of disease severity, and PI 277012 had a lower percentage of FDK and lower DON content than Grandin in almost all experiments (Tables 1, 2). Highly significant correlations among disease severity, FDK and DON content were observed. The correlation coefficient for disease severity and FDK ranged from 0.32 to 0.57 ($P < 0.01$) (Table 3) across the five field experiments, and those for disease severity and DON content, and FDK and DON content ranged from 0.20 to 0.53 ($P < 0.01$) and 0.27 to 0.64 ($P < 0.01$) (Table 3), respectively.

PI 277012 is around 100 cm tall, which is about 20 cm taller than Grandin (about 80 cm tall). Plant height of the DH lines ranged from 50 to 120 cm (Online Resource Table S2). For days to heading, Grandin headed 46–58.5 days after planting, which is about 3–9 days earlier than PI 277012 (46–64.5 days). Within the DH population, days to heading ranged from 45 to 70 days (Online Resource Table S3). Correlation between disease severity and plant height tended to be more consistent across experiments and the correlation coefficient ranged from −0.22 in 09Far to −0.56 in 10Ldn (Table 3), whereas the correlation between disease severity and days to heading varied greatly among experiments. The correlation was significantly negative in 10Far but positive in other experiments (Table 3). Correlations among FDK and DON content with either plant height or days to heading also varied among experiments. For example, FDK was significantly (but negatively) correlated with plant height across all experiments, but only significantly correlated with days to heading in 10Far and 10Ldn. DON content was significantly correlated with plant height only in 09Far, 10Far and 10Ldn, and with days to heading only in 09Pro, 10Far, 10Pro and 10Ldn (Table 3). These inconsistent correlations between FHB reactions and morphological traits suggest that the effects of plant height and heading time on FHB are rather minor in this population. We observed that the early genotypes tended to have lighter diseases than late genotypes due to low temperature and dry weather conditions in 2009 from late June to mid July in Prosper and Fargo. The FHB infection and disease development in the DH population were probably more related to the environmental conditions than the maturity and plant height.

QTL analysis of FHB resistance and other traits

QTL analysis led to the identification of two PI 277012-derived QTLs, designated *Qfhb.rwg-5A.1* and *Qfhb.rwg-5A.2*, on chromosome arms 5AS and 5AL, respectively, which predominantly governed FHB resistance in the DH population (Fig. 3; Table 4). *Qfhb.rwg-5A.1* peaked at marker *Xbarc40* on 5AS between markers *Xcfa2104* and *Xgwm617*, and explained up to 20% of the phenotypic variation. The 5AL QTL, *Qfhb.rwg-5A.2*, peaked at marker *Xcfd39* between *Xwmc470* and *Xbarc48*, which is the interval that includes the *Q* gene (Figs. 2, 3). *Qfhb.rwg-5A.2* was significant in all experiments and explained up to 32% of the phenotypic variation (Fig. 3; Table 4). Variance analysis performed using marker genotype data of *Xbarc40* (peak marker of *Qfhb.rwg-5A.1*) and *Xcfd39* (peak marker of *Qfhb.rwg-5A.2*) along with disease severity data collected from each environment revealed that disease severity variance due to marker genotypes were highly significant (Table 5) and thus confirmed the results from QTL analysis. Disease severity variance due to environment was also highly significant, and this indicates the significant role that the environment played on FHB development. However,

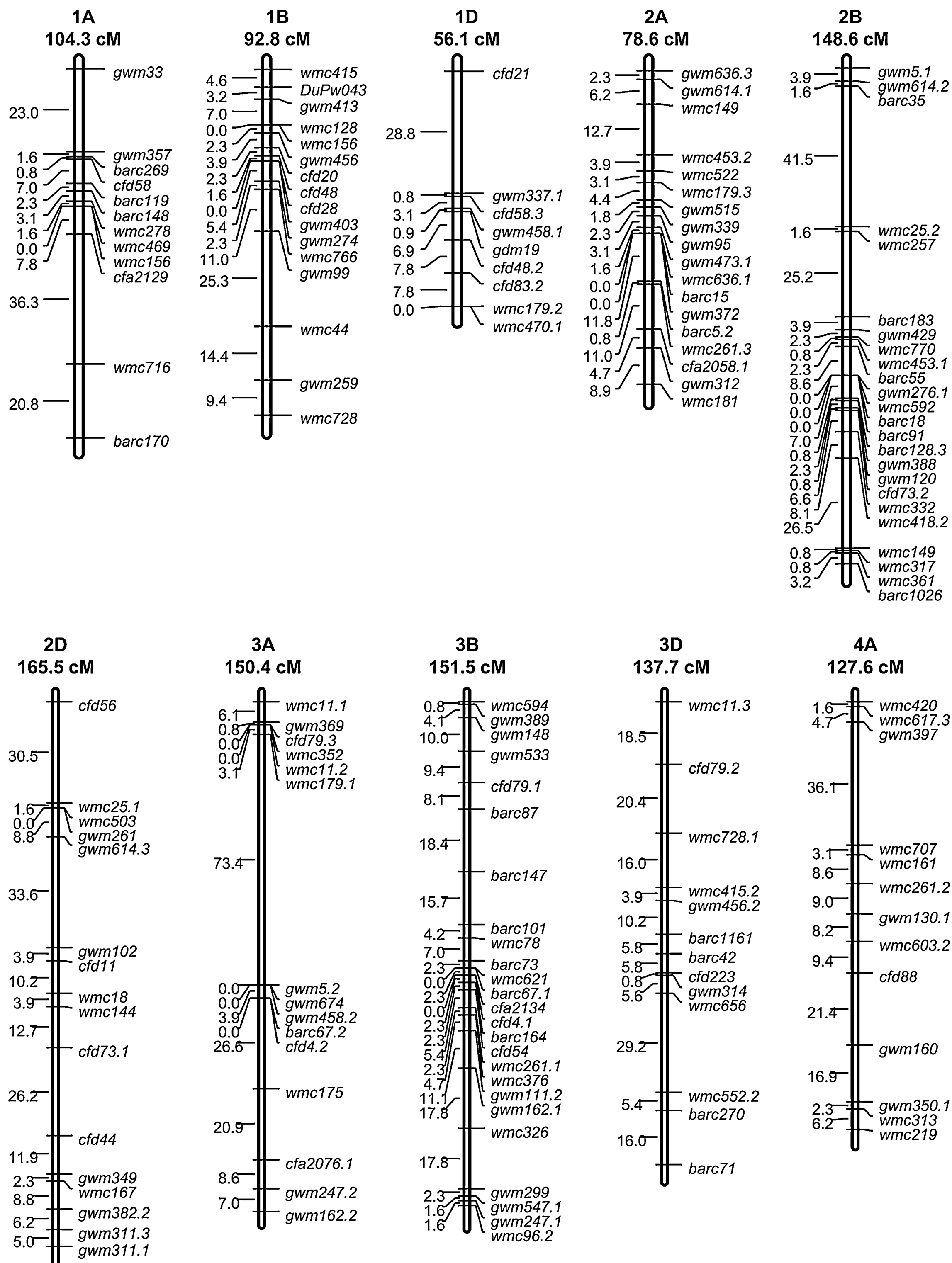

Fig. 2 Genetic linkage maps constructed in the Grandin × PI 277012 derived DH population. The positions of marker loci are shown to the right of the linkage groups and centiMorgan (cM) distances between loci are shown along the left. Linkage maps were developed using the computer program MAPMAKER (V2.0) for Macintosh (Lander et al. 1987) with a minimum LOD threshold of 3.0 and the Kosambi mapping function (Kosambi 1944)

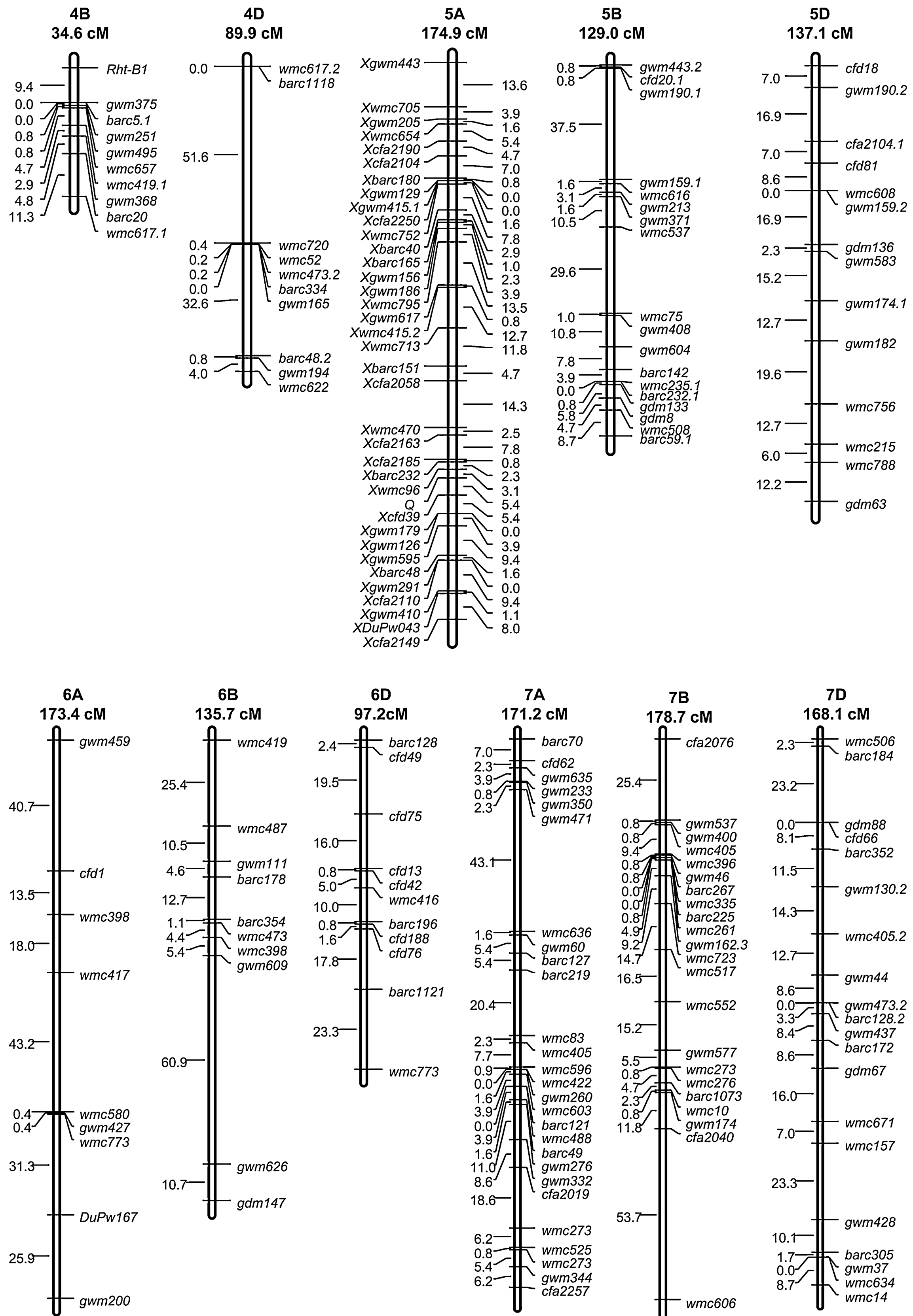

Fig. 2 continued

Table 1 Distribution of Fusarium head blight disease severity and Fusarium damaged kernel (FDK) in the doubled haploid (DH) population derived from the cross between Grandin and PI 277012

Distribution of disease severity in DH population

Range (%)	Number of DH lines[a]							
	09GH	10GH1	10GH2	09Far	09Pro	10Far	10Pro	10Ldn
0–10	1	7	17	16	3	2	1	28
11–20	35	31	36	45	31	10	5	31
21–30	31	32	22	29	15	20	18	16
31–40	20	18	15	18	13	26	32	15
41–50	16	12	12	9	16	21	28	10
51–60	12	11	11	8	14	14	15	12
61–70	5	12	7	3	15	16	13	10
71–80	7	4	5	2	9	14	13	6
81–90	2	2	4	0	10	6	4	1
91–100	1	1	1	0	4	1	1	1
Normality test (*P*)[b]	<0.01	<0.01	<0.01	<0.01	<0.01	0.13	0.07	<0.01

Disease severity (%) in parents

Parent	09GH	10GH1	10GH2	09Far	09Pro	10Far	10Pro	10Ldn
Grandin	72.0	62.2	80.0	30.0	95.0	71.0	54.0	54.0
PI 277012	12.0	11.2	7.8	7.8	12.0	14.0	13.0	7.0

Distribution of FDK in DH population

FDK (%)	Number of DH lines[a]				
	09Far	09Pro	10Far	10Pro	10Ldn
0–10	60	3	0	0	3
11–20	44	20	2	0	30
21–30	16	39	10	8	35
31–40	7	32	31	24	23
41–50	3	15	39	19	16
51–60	0	11	21	17	8
61–70	0	8	11	25	8
71–80	0	1	12	23	6
81–90	0	1	4	13	1
91–100	0	0	0	1	0
Normality test (*P*)[b]	<0.01	<0.01	0.18	0.05	<0.01

FDK (%) in parents

Parent	09Far	09Pro	10Far	10Pro	10Ldn
Grandin	20.1	72.5	59.0	76.0	55.0
PI 277012	4.6	19.5	21.0	38.0	15.0

[a] 09GH, 10GH1, and 10GH2 indicate experiments conducted in the greenhouse in the fall of 2009 and spring and summer of 2010, respectively. 09Far and 09Pro, and 10Far, 10Pro, and 10Ldn represent field experiments conducted in Fargo and Prosper in 2009 and in Fargo, Prosper and Langdon in 2010, respectively

[b] Normality test was performed under PROC UNIVARIATE and the Shapiro–Wilk test was used (SAS Institute 2004)

G × E interactions were not significant for either of the two QTLs (peak markers) (Table 5), suggesting that the effects of the two QTLs were less affected by the environment.

The same two QTLs were significantly associated with FDK and DON content (Table 6). For FDK, *Qfhb.rwg-5A.1* and *Qfhb.rwg-5A.2* explained up to 14 and 12% of the variation, respectively, and for DON content, they explained up to 16 and 10% of the phenotypic variation, respectively. In addition, a QTL on chromosome 4B (*QHt.rwg-4B*) that peaked at the *Rht-B1* locus within the interval *Rht-B1*–*Xwmc657* and associated with plant height (see below) was also associated with FDK in the DH population.

Table 2 Distribution of deoxynivalenol (DON) content in the doubled haploid (DH) population derived from the cross between Grandin and PI 277012

DON content (ppm)	Number of DH lines[a]				
	09Far	09Pro	10Far	10Pro	10Ldn
0–2	88	20	0	7	13
2–4	26	44	0	41	59
4–6	14	28	0	43	37
6–8	2	10	2	26	10
8–10	0	11	7	11	7
10–12	0	8	8	1	4
12–14	0	5	5	0	0
14–16	0	2	11	1	0
16–18	0	1	10	0	0
18–20	0	1	8	0	0
20–22	0	0	19	0	0
22–24	0	0	10	0	0
24–26	0	0	10	0	0
26–28	0	0	9	0	0
28–30	0	0	8	0	0
30–32	0	0	6	0	0
32–34	0	0	5	0	0
34–36	0	0	5	0	0
36–38	0	0	2	0	0
>38	0	0	5	0	0
Normality test (*P*)[b]	<0.01	<0.01	0.09	0.03	<0.01

DON content (ppm) in parents

Parent	09Far	09Pro	10Far	10Pro	10Ldn
Grandin	4.3	2.4	23.9	4.7	4.1
PI 277012	0.2	1.5	10.7	5.0	3.1

[a] 09Far and 09Pro, and 10Far, 10Pro and 10Ldn represent field experiments conducted in Fargo and Prosper in 2009, and in Fargo, Prosper and Langdon in 2010, respectively

[b] Normality test was performed under PROC UNIVARIATE and the Shapiro–Wilk test was used (SAS Institute 2004)

Table 3 The correlation coefficient among traits of disease severity (DS), deoxynivalenol (DON) content, Fusarium damaged kernels (FDK), plant height (Hgt), and days to heading (DTH) in the doubled haploid population derived from the cross between Grandin and PI 277012

Correlation	Coefficient determinant of correlation (*r*)				
	09Far	09Pro	10Far	10Pro	10Ldn
DS-Hgt	−0.22*	−0.29**	−0.48**	−0.46**	−0.56**
DS-DTH	0.34**	0.31**	−0.35**	0.16*	0.16*
DS-FDK	0.32**	0.35**	0.52**	0.53**	0.57**
DS-DON	0.34**	0.53**	0.20*	0.35**	0.39**
FDK-Hgt	−0.63**	−0.44**	−0.62**	−0.66**	−0.56**
FDK-DTH	0.02[NS]	0.01[NS]	0.28**	-0.08[NS]	0.12*
FDK-DON	0.64**	0.44**	0.57**	0.27**	0.49**
DON-Hgt	−0.32**	−0.09[NS]	−0.15*	−0.08[NS]	−0.20*
DON-DTH	−0.03[NS]	0.31**	0.35**	0.29**	0.19*

09Far and 09Pro, and 10Far, 10Pro and 10Ldn represent field experiments conducted in Fargo and Prosper in 2009 and in Fargo, Prosper and Langdon in 2010, respectively

NS non significant

* and ** indicate significance at level of $P < 0.05$ and 0.01, respectively

This QTL was significantly associated with FDK in all field experiments and explained up to 15% of the phenotypic variation (Table 6).

QHt.rwg-4B was significant in all field experiments for plant height and explained up to 73% of the phenotypic variation, which indicated that plant height was largely governed by the *Rht-B1* locus. However, *Qfhb.rwg-5A.2* also showed minor effects on plant height in experiments 09Far, 09Pro and 10Far where it explained 5, 4, and 4% of the variation, respectively (Table 6). Days to heading in this DH population were controlled by two QTLs (*QEet.rwg-2B* and *QEet.rwg-7D*) located on chromosomes 2B and 7D, respectively. *QEet.rwg-2B* peaked at marker *Xbarc183* within the interval *Xwmc257–Xwmc770* and explained up to 15% of the trait variation. *QEet.rwg-7D* peaked at marker *Xgdm67* within the interval *Xbarc172–Xwmc671* and explained up to 10% of the variation (Table 6).

Discussion

Since the mid 1990s, bread wheat and durum wheat production in North America and many other wheat-growing regions in the world have been seriously threatened by FHB. The epidemics of this disease have caused serious economic losses in Canada and the United States over the past 16 years. Because the utilization of FHB-resistant cultivars would be an effective approach for protecting wheat against FHB, new sources of FHB resistance have been in high demand by wheat breeders. Particularly, resistance sources that carry QTLs that confer high levels of resistance are most desirable for genetically improving wheat for resistance to FHB. In this study, we found that the high level of resistance in the wheat accession PI 277012 is controlled by two major QTLs located on chromosome 5A, and were consistently detected in different environments. The two QTLs are not tightly associated with undesirable height or maturity levels. Therefore, PI 277012 is a promising FHB resistance source for current and future wheat breeding.

The two FHB-resistance QTLs identified in PI 277012 both influence Type I and II resistance mechanisms because

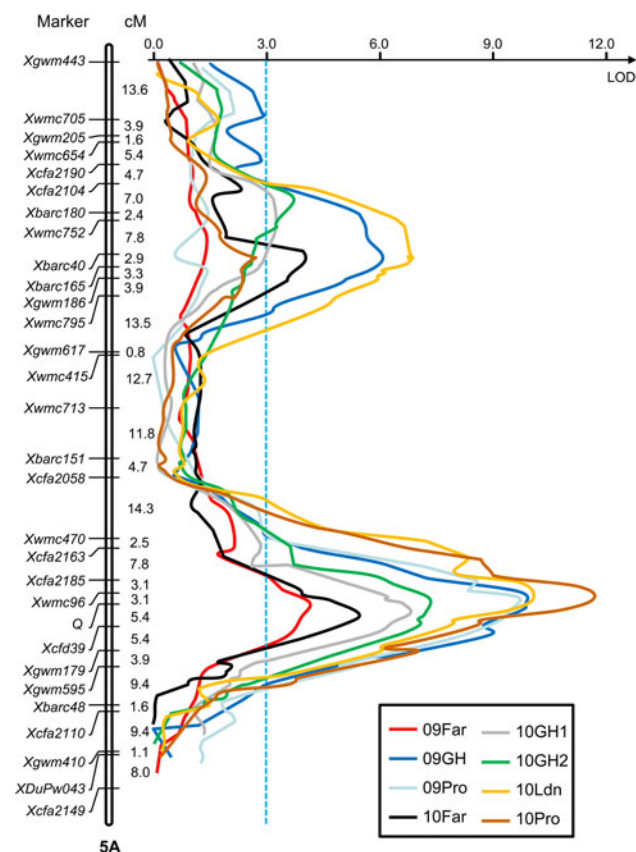

Fig. 3 Two major QTLs on chromosome 5A governed resistance to Fusarium head blight (FHB) in wheat accession PI 277012. QTL analysis was performed through composite interval mapping in the computer program Map Manager QTX (Manly et al. 2001). The positions of marker loci are shown to the left of the linkage map and centiMorgan (cM) distances between loci are shown along the right. The vertical dotted line indicates the logarithm of the odds (LOD) significance threshold of 3.03. R^2 and LOD values of the QTLs are shown in Table 4. 09GH, 10GH1 and 10GH2 indicate experiments conducted in greenhouse in fall of 2009, spring and summer of 2010, respectively. 09Far and 09Pro, and 10Far, 10Pro and 10Ldn represent field experiments conducted in Fargo and Prosper in 2009, and in Fargo, Prosper and Langdon in 2010, respectively

Table 4 Parameters of the two major QTLs controlling resistance to FHB in the doubled haploid population derived from the cross between Grandin and PI 277012 under eight environments

Experiment[a]	*Qfhb.rwg-5A.1*			*Qfhb.rwg-5A.2*		
	LOD[b]	R^2	Additive effect[c]	LOD[b]	R^2	Additive effect[c]
09GH	6.41	0.20	−8.39PI	10.76	0.32	−10.47PI
10GH1	5.07	0.14	−7.47PI	7.33	0.20	−8.86PI
10GH2	3.89	0.11	−7.18PI	7.35	0.20	−9.57PI
09Far	2.01	0.06	−3.77PI	3.91	0.12	−5.30PI
09Pro	2.33	0.08	−6.92PI	11.48	0.32	−14.19PI
10Far	3.93	0.11	−6.64PI	5.02	0.14	−7.34PI
10Pro	2.02	0.06	−4.69PI	11.89	0.31	−10.20PI
10Ldn	6.96	0.18	−9.35PI	9.72	0.24	−10.86PI

QTL analysis was performed through composite interval mapping in the computer program Map Manager QTX (Manly et al. 2001). The 5AS QTL *Qfhb.rwg-5A.1* peaked at marker *Xbarc40* within the interval between *Xcfa2104* and *Xgwm617*, and the 5AL QTL *Qfhb.rwg-5A.2* peaked at marker *Xcfd39* within the interval between *Xwmc470* and *Xbarc48* (Fig. 3)

LOD logarithm of the odds

[a] 09GH, 10GH1 and 10GH2 indicate experiments conducted in greenhouse in the fall of 2009, spring and summer of 2010, respectively. 09Far and 09Pro, and 10Far, 10Pro and 10Ldn represent field experiments conducted in Fargo and Prosper in 2009, and in Fargo, Prosper and Langdon in 2010, respectively

[b] The average critical LOD threshold of 3.03 was obtained through 1,000 permutations yielded an experiment-wise significance level of 0.05

[c] Additive effect indicates the percentage of disease severity decreased by gene or QTL. The letters PI following behind indicate that the parent PI 277012 contributed the resistance effects

they were both significantly associated with FHB resistance in the field and in the greenhouse, where the spread infected corn grains in field experiments essentially allows the evaluation of both Type I and Type II resistance and the single-spikelet inoculation method used in the greenhouse experiments allows the evaluation of Type II resistance. The two QTLs also showed significant effects on reducing DON content in the seed and thus have resistance to DON accumulation.

Based on the position of marker *Xbarc180*, *Qfhb.rwg-5A.1* appears to be located in the same region of chromosome arm 5AS as the QTL *Qfhs.ifa-5A* identified in Sumai 3 (Buerstmayr et al. 2002; Somers et al. 2003). The two QTLs may be the same or their effects may be conferred by different alleles of the same locus. However, no previous studies have reported FHB resistance genes in the region of chromosome arm 5AL that contains *Qfhb.rwg-5A.2*. Therefore, *Qfhb.rwg-5A.2* represents a novel FHB resistance QTL in wheat. PI 277012 contains *T. timopheevii* in its parentage (http://www.ars-grin.gov/cgi-bin/npgs/acc/display.pl?1206987). There is possibility that the two QTLs for FHB resistance were originally derived from *T. timopheevii*.

Correlation analysis showed that reactions to FHB in the DH population had positive but low correlation with days to heading in four experiments, and a negative correlation in one experiment (Table 3). The FDK data were significantly correlated with days to heading in only two experiments (Table 3). QTL analysis showed that days to heading in this population were governed by *QEet.rwg-2B* and *QEet.rwg-7D*, neither of which was associated with FHB resistance, which further suggests that FHB resistance in PI 277012 is not associated with days to heading. *QEet.rwg-2B* corresponds to the *Ppd-B1* because *Xwmc257*, which is within the peak interval of this QTL, is only 1.7 cM from *Xgwm257* (Somers et al. 2004), a marker closely linked to

Table 5 Variance analysis for detecting G × E interaction of the two major QTLs *Qfhb.rwg-5A.1* (peaked at marker *Xbarc40*) and *Qfhb.rwg-5A.2* (peaked at marker *Xcfd39*) derived from PI 277012

Source of variance	Degree of freedom	*P*
Qfhb.rwg-5A.1 (*Xbarc40*)	1	<0.01
Qfhb.rwg-5A.2 (*Xcfd39*)	1	<0.01
Environment	7	<0.01
Qfhb.rwg-5A.1 × environment	7	NS
Qfhb.rwg-5A.2 × environment	7	NS
Qfhb.rwg-5A.1 × *Qfhb.rwg-5A.2*	1	NS
Qfhb.rwg-5A.1 × *Qfhb.rwg-5A.2* × Environment	7	NS

NS non significant

Ppd-B1 for photoperiod insensitivity (Mohler et al. 2004). *QEet.rwg-7D* may be the same QTL identified in a population derived from Savannah × Rialto (Griffiths et al. 2009) based on the position of the common marker *Xbarc172*.

The plant height in the DH population showed a significant correlation with FHB reaction in most field experiments (Table 3). However, QTL analysis indicated that plant height in this population is predominantly controlled by the *Rht-B1* gene on chromosome arm 4BS, which explained up to 73% of the phenotypic variation. Of the two major FHB resistance QTLs on chromosome 5A, *Qfhb.rwg-5A.1* showed no association with plant height, but *Qfhb.rwg-5A.2* showed a minor effect on plant height and explained about 5% of the variation in three experiments (09Far, 09Pro and 10Far). Therefore, these results indicate that FHB resistance effects of *Qfhb.rwg-5A.1* are not influenced by plant height, and *Qfhb.rwg-5A.2* is very weakly associated with plant height.

Even though no significant effects from the *Rht-B1* locus were found on FHB disease severity and DON accumulation in the grain, the *Rht-B1* locus played a significant role on percentage of FDK with the *QHt.rwg-4B* (QTL corresponding to *Rht-B1*) explaining up to 15% of the phenotypic variation. Based on the genotypic analysis of the *Rht-B1* locus in the DH population, average percentage of FDK in lines carrying the *Rht-B1* allele derived from Grandin is 12% more than those having the *rht-B1* allele from PI 277012. Since the short plant height can increase the possibility of pathogen infection and thus cause more kernel damage, the effect from *Rht-B1* on FDK percentage is mostly due to the plant height morphology rather than physiological resistance.

A disadvantage to using PI 277012 as a resistance source is that it carries the *q* allele, which confers non-free-threshability. Molecular mapping showed that the *Q* locus is located within the peak interval of *Qfhb.rwg-5A.2*. Thus, a relatively large breeding base population is required for breaking the linkage between *q* and the resistance gene(s) if PI 277012 is used in a breeding program. However, among the 130 DH lines evaluated in this study, one free-threshing line DH#80 carried PI 277012 alleles at both resistance QTLs (*Qfhb.rwg-5A.1* and *Qfhb.rwg-5A.2*) and the *Q* allele from Grandin made it free threshing with an average FHB disease severity of 19.7%. Therefore, DH#80 will be an

Table 6 QTLs associated with deoxynivalenol content, Fusarium damaged kernel, plant height, and days to heading in the doubled haploid population derived from the cross between Grandin and PI 277012 [a]

QTL	Chro.	Marker interval	Peak marker	LOD value[a]					R^2 value				
				09Far	09Pro	10Far	10Pro	10Ldn	09Far	09Pro	10Far	10Pro	10Ldn
Fusarium damaged kernel													
QHt.rwg-4B	4B	*Rht-B1–Xwmc657*	*Rht-B1*	4.61	3.01	5.09	5.28	4.24	0.13	0.09	0.14	0.15	0.12
Qfhb.rwg-5A.1	5A	*Xcfa2104–Xgwm617*	*Xbarc40*	3.00	NS	5.41	3.37	NS	0.08	–	0.14	0.09	–
Qfhb.rwg-5A.2	5A	*Xwmc470–Xbarc48*	*Xcfd39*	3.13	3.67	4.74	4.07	2.01	0.08	0.11	0.12	0.11	0.05
Deoxynivalenol content													
Qfhb.rwg-5A.1	5A	*Xcfa2104–Xgwm617*	*Xbarc40*	5.65	NS	4.11	2.94	2.57	0.16	–	0.14	0.07	0.06
Qfhb.rwg-5A.2	5A	*Xwmc470–Xbarc48*	*Xcfd39*	3.02	3.11	3.03	3.21	2.43	0.08	0.10	0.08	0.09	0.05
Plant height													
QHt.rwg-4B	4B	*Rht-B1–Xwmc657*	*Rht-B1*	24.12	26.89	30.54	26.89	37.13	0.53	0.52	0.59	0.52	0.73
Qfhb.rwg-5A.2	5A	*Xwmc96–Xgwm179*	*Xcfd39*	3.30	3.35	3.43	NS	NS	0.05	0.04	0.04	–	–
Days to heading													
QEet.rwg-2B	2B	*Xwmc257 –Xwmc770*	*Xbarc183*	5.61	3.12	3.64	5.47	3.11	0.15	0.10	0.11	0.15	0.09
QEet.rwg-7D	7D	*Xbarc172–Xwmc671*	*Xgdm67*	4.02	2.01	2.58	2.51	2.63	0.10	0.06	0.07	0.07	0.07

Chro. chromosome, *NS* non-significant

[a] 09Far and 09Pro, and 10Far, 10Pro and 10Ldn represent field experiments conducted in Fargo and Prosper in 2009, and in Fargo, Prosper and Langdon in 2010, respectively

extremely useful source for transferring the FHB resistance QTLs to other backgrounds for validation, introgression, and breeding purposes.

In addition to the cultivar Grandin, the accession PI 277012 was also crossed and backcrossed with hard red spring wheat varieties 'Glenn', 'Russ', and 'Reeder', and durum cultivars 'Lebsock', 'Mountrail', 'Divide', and 'Ben' (Xu et al. 2010). The BC_1F_5-derived lines which carried one or both of the two major QTLs from PI 277012 exhibited a high level of resistance to FHB, indicating that the high level of FHB resistance in PI 277012 can be steadily expressed in different genetic backgrounds (http://www.uky.edu/Ag/Wheat/wheat_breeding/New%20Folder/Steve%20Xu.pdf). Particularly noteworthy is the fact that the two QTLs from PI 277012 have been successfully transferred into durum wheat, and preliminary data indicate that PI 277012 may be an excellent resistance source for durum wheat as well.

In summary, this research resulted in the identification of two major QTLs on chromosome 5A conferring a high level of FHB resistance in wheat accession PI 277012. The two QTLs affect both Type I and II resistance, and also contribute resistance to DON accumulation. One of the QTLs mapped to a genomic region where no other FHB resistance QTLs have been previously reported. Our results also indicated that the resistance governed by the two QTLs is loosely related to plant height and days to heading, and the linkage between the locus controlling free-threshability and FHB resistance can be broken. Therefore, we proposed that PI 277012 is an excellent resistance source for improving wheat FHB resistance.

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