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ORIGINAL PAPER

Genetic relationships between resistances to Fusarium head blight and crown rot in bread wheat (*Triticum aestivum* L.)

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Abstract Fusarium head blight (FHB) and crown rot (CR) are two wheat diseases caused by the same *Fusarium* pathogens. Progress towards CR resistance could benefit from FHB-resistant germplasm if the same genes are involved in resistance to these two different diseases. Two independent studies were conducted to investigate the relationship between host resistances to these two diseases. In the first study 32 genotypes were assessed and no significant correlation between their reactions to FHB and CR was detected. The second study was based on a QTL analysis of a doubled haploid population derived from a variety with resistance to both diseases. Results from this study showed that loci conferring resistance to FHB and CR are located on different chromosomes. Together, these results suggest that, despite a common aetiology, different host genes are involved in the resistance against FHB and CR in wheat. Thus, although it is possible that genes affecting both diseases may exist in other germplasm or under different conditions, separate screening seems to be needed in identifying sources of CR resistance.

Introduction

Fusarium head blight (FHB) is a serious wheat disease in many cereal growing regions. In addition to causing significant yield loss, the disease also produces mycotoxins which are harmful to humans and animals (Buerstmayr et al. 2009). Crop management and chemical control are only partially effective and growing resistant varieties is generally accepted as the most cost effective way to minimise the harm caused by FHB (Parry et al. 1995). Significant efforts have been made to identify resistant sources from large-scale germplasm screening. In China alone, at least 34,571 genotypes have been tested for FHB (Lu et al. 2001), and the total number of genotypes tested in the United States under the Wheat and Barley Scab Initiative could be even larger (R. Dill-Macky, personal

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communication). Large-scale FHB screenings have also been carried out by CIMMYT and other organisations (Gilchrist et al. 1997). Numerous studies have shown that the inheritance of FHB resistance in wheat is of a quantitative nature (Bai et al. 1999; Semagn et al. 2007). More than 50 peer-reviewed studies have been published reporting QTL for FHB resistance (Buerstmayr et al. 2009). The most extensively studied gene for FHB resistance is *Fhb1* on chromosome arm 3BS, which was found in numerous independent mapping studies based on the Chinese FHB resistant source 'Sumai 3'. This locus has been introduced into many breeding populations worldwide, including the USA (Del Blanco et al. 2003, Zhou et al. 2003; Pumphrey et al. 2007), Canada (Yang et al. 2003; McCartney et al. 2007), Australia (Xie et al. 2007) and Germany (Miedaner et al. 2006).

Crown rot (CR) is another serious cereal disease caused by *Fusarium* pathogens (Akinsanmi et al. 2004). In Australia, CR has been a chronic disease for many decades inflicting yield loss of up to 89% (Klein et al. 1991). In the Pacific North-West of the USA CR can reduce wheat yield by up to 35% (Smiley et al. 2005). Both CR and FHB have become more prevalent in recent years due to conservation farming practices involving stubble retention, as the pathogens are carried over in crop residues (Burgess 2005). CR has become increasingly important in South Africa, Italy, Egypt, Turkey, Syria, Morocco, Argentina and China (Smiley et al. 2005; Chakraborty et al. 2006). Plants infected with CR can also accumulate mycotoxins in grains and other tissues (Mudge et al. 2006), raising potential human and animal health concerns. Despite a long history of research effort (Purss 1966), breeding for crown rot resistance has been difficult due to a lack of germplasm with a high level of resistance. Until recently (Mitter et al. 2006; Li et al. 2008), methods for CR screening have been technically demanding (Wildermuth et al. 2001), where CR expression has been strongly influenced by environmental factors (Beddis and Burgess 1992). Compared to FHB only a limited number of genotypes have been screened for CR. The genetics of CR resistance have been reported in only three partially resistant genotypes in wheat (Wallwork et al. 2004; Collard et al. 2005; Bovill et al. 2006) and available results suggest that CR resistance is also quantitative in nature.

The common aetiology between FHB and CR raises the possibility that resistance to these two diseases may involve the same genes; however, there is little published information to support or refute this suggestion. If a close relationship exists, efforts to identify sources of CR resistance could be streamlined by trawling through the vast amount of data from FHB germplasm screening. We have attempted to clarify the genetic relationship between FHB and CR resistance using two independent studies: (a) by screening a panel of wheat genotypes for FHB and CR resistance; (b) by identifying and mapping QTL conferring FHB and CR resistance in a doubled haploid (DH) population derived from a genotype showing resistance to both diseases.

Materials and methods

Plant material

A set of 32 hexaploid wheat genotypes were assessed for FHB and CR reaction. Some of the 32 wheat genotypes are well-known sources of FHB or CR resistance (Table 1). A microspore-derived DH population with 153 lines were generated in this laboratory from F1 plants of 'Batavia'/'Ernie'. This DH population was used to detect QTL conferring FHB and CR reactions. 'Ernie' is a soft red winter wheat from the USA with resistances to both diseases (McKendry et al. 1995), while 'Batavia' is an Australian variety with high CR susceptibility (Brennan et al. 1994).

CR bioassay

Two isolates, one *Fusarium pseudograminearum* (CS3096) and one *F. graminearum* (CS3005), were used for assessing CR and FHB reactions. They are two of the highly aggressive *Fusarium* isolates identified from a screening of over 650 isolates collected in Queensland and New South Wales in Australia (Akinsanmi et al. 2004). The procedures described by Mitter et al. (2006) were used for inoculum preparation and for CR inoculation. Basically, seedlings were raised in punnets and they were laid horizontally on their side 10 days after emergence. A 10-μl droplet of 10^6 macroconidia/ml was placed on the stem base (0.5–1.0 cm from the soil surface) using a repeating dispensing syringe. Inoculated seedlings were incubated at near-saturated relative humidity for 48 h. Crown rot severity was assessed 35 days after inoculation. The 32 genotypes were assessed in glasshouses (settings: 25/15 (±5)°C day/night temperature and 60/80 (±10)% day/night relative humidity with natural illumination) following the methods described by Mitter et al. (2006), and the DH lines from the 'Batavia'/'Ernie' population was assessed in controlled environment facilities (CEF) (settings: 25/15°C day/night temperature, 65/95% day/night relative humidity, and a 13-h photoperiod). Three independent trials were carried out for each of the two isolates using a randomised block design. Two replicates, each with five seedlings, were used in each trial. CR severity was assessed 35 days after inoculation, using a 0 (no obvious symptom) to 5 (whole plant severely to completely necrotic) scale as described by Li et al. (2008).

Table 1 Reactions of 32 wheat genotypes to crown rot and Fusarium head blight infection

Genotype	FHB severity		CR severity	
	CS3005	CS3096	CS3005	CS3096
2-49	2.8	4.2	0.2	0.2
Abura komugi	4.1	7.9	0.8	0.7
Aso zairai (Yuubu kaf-)	1.3	6.7	0.5	0.7
Aso zairai 11	1.0	3.5	0.2	0.4
Batavia	5.1	–	4.2	4.2
Baxter	2.4	2.7	1.3	0.6
Chile	8.0	9.6	0.7	0.7
Chinese Spring	4.2	4.5	3.8	5.0
Drysdale	4.7	6.1	2.6	2.4
Ernie	2.5	2.0	0.2	0.2
Freedom	6.3	5.2	0.9	1.4
Frontana	8.1	8.0	2.5	2.1
Itou komugi	2.4	12	1.1	2.0
Janz	3.7	4.3	1.7	1.5
Kagoshima	1.2	2.5	1.2	1.2
Kennedy	4.7	5.3	1.9	2.1
Kikuchi	2.0	2.4	0.9	1.3
Lang	2.7	2.9	2.6	3.2
Ning-7840	2.3	3.6	3.1	2.5
Nobeokabouzu komugi	4.0	5.7	5.0	2.6
Nyuubai	2.2	4.7	2.9	2.9
Qiamai (Xiamai)	1.6	6.2	0.7	1.2
EGA Gregory	4.7	4.3	2.5	1.4
Shiro nankin	2.1	3.1	0.6	1.0
Shou komugi 11	1.9	3.9	1.3	1.6
Soba komugi 1B	3.3	7.5	0.7	1.1
Soba komugi 1C	6.8	12.0	0.5	0.8
Sotome	2.8	3.4	1.7	2.0
Sotome A	2.9	4.6	1.6	1.7
Sumai 3	1.6	3.1	2.1	1.4
SVP-72017	2.4	1.8	1.2	0.9
Zairai yuubou	5.6	4.3	3.8	1.5

Missing value was marked by '–'

FHB assay

FHB assessments were conducted in the CEF in two independent trials, each consisting of two replicates. Individual spikes were checked daily for flowering and FHB inoculation was carried out at anthesis. For the 32 genotypes, each replicate consisted of two plants in two separate pots. Eight to ten spikes from each of the two replicates were inoculated by inserting a 3-mm filter paper saturated with inoculum (about 10 µl suspension of 10^5 conidia/ml) into the fourth spikelet from the tip of a spike. For the DH population, each replicate consisted of five plants and point inoculation was carried out by placing a 10 µl droplet of inoculum (10^6 conidia/ml) into the spikelet in the middle of the spike. The inoculated spikes were individually covered with moistened polythene bags for 48 h and then with paper bags until disease assessment at 21 days after inoculation. The average number of infected spikelets in a replicate was used for representing FHB severity of a genotype. At maturity, data on plant height were collected by measuring the heights of the main tillers.

Molecular marker analysis of the DH population

DArT assay was carried out as described by Jaccoud et al. (2001) and Wenzl et al. (2004) in the laboratory of Diversity Array Technology Propriety Limited (Canberra, Australia). A total of 1,074 DArT markers were scored. A quality parameter Q, which is the variance of the hybridisation intensity between allelic states as a percentage of the total variance, was calculated for each marker. Only markers with >80% Q were selected for linkage analysis.

In addition, the parents of the DH population were screened with 1,358 SSR markers. Of the 523 polymorphic markers, 104 were selected to genotype the 153 progeny with at least four well-separated SSR on each of the 21 chromosomes. SSR were amplified using [$33^{-\alpha}$P]dCTP following manufacturer's instruction (Multiplex-Ready Marker User Handbook, version 2.0). Samples were separated on 43-cm long 4% polyacrylamide gel containing 7 M urea with a 350 (TAMRA) size standard.

Statistical analysis

Statistical analyses were performed using GenStat for Windows in the 10th edition (copy right Lawes Agricultural Trust, Rothamsted Experimental Station, UK). For each trial, the following mixed-effects model was used:

$$Y_{ij} = \mu + r_i + g_j + w_{ij}$$

where Y_{ij} = observation on the jth genotype in the ith replication; μ = general mean; r_i = effect due to ith replication; g_j = effect due to the jth genotype; w_{ij} = error or genotype by replication interaction, where the effect of replicates was treated as random and that of genotypes fixed. The effects of replicate and genotype for each trait were determined using ANOVA, and broad-sense heritability from the DH population was estimated using the formula: $h^2 = \sigma_G^2/[\sigma_G^2 + (\sigma_e^{2/r})]$, where σ_G^2 is the genetic variance, σ_e^2 is the residual variance and r is the number of replicates per genotype (Nyquist 1991). Pearson correlation coefficient was estimated between traits and trials. Mean values for each DH line within each trial were used for QTL analysis. Data for each trait were combined from different trials by using a multifactor analysis of variance,

where trials were considered as a higher level of treatment compared to the models described for each trial. A single factor analysis of co-variance (ANCOVA) was performed to investigate the effect of plant height (PH) on CR and FHB reactions using combined data for each trait.

Linkage and QTL analyses

Mendelian segregation was tested by Chi-square goodness-of-fit to a 1:1 ratio at $p = 0.05$ significance level. Linkage analysis was carried out with the JoinMap4 program (Van Oiijen 2006). To distribute loci into linkage groups, LOD thresholds from 3 to 10 were tested, until a threshold with the optimum number of markers in linkage groups maintaining linkage order and distance was obtained. Threshold values of LOD 0.1 and REC (recombination frequency) 0.45 were used for marker order analysis. Framework maps were constructed by using only non-distorted markers and distorted markers were added in a second step to integrate into the map frameworks. Known chromosomal locations of some of the SSR markers and a subset of the DArT markers were used to assign linkage groups to wheat chromosomes.

The Kruskal–Wallis test was used in a preliminary analysis to detect association between markers and individual traits. Following the interval mapping analyses, permutation tests (1,000 permutations for each trial) were carried out to determine the threshold of QTL presence corresponding to a genome-wide false discovery rate of 5% ($p < 0.05$). Markers most closely linked to the putative QTL identified were selected as co-factors for further QTL detection with a multiple QTL model (MQM) using the MapQTL® program (Version 5.0, van Ooijen 2004). QTL identified in more than one trial and located close to one another on the same linkage group, with alleles derived from the same parent, were considered to represent the same QTL. QTL maps were drawn using MapChart 2.1 (Voorrips 2002).

Results

Relationship between CR and FHB reactions assessed using a panel of genotypes

Crown rot severities of the 32 genotypes caused by both the *F. graminearum* isolate CS3005 and the *F. pseudograminearum* isolate CS3096 had the same range (from 0.2 to 5.0) with similar average severities (1.7 for the former and 1.6 for the latter, Table 1). The Pearson correlation coefficient between CR severities caused by the two isolates among the 32 genotypes was highly significant at $r = 0.80$.

Fusarium head blight severities of the 32 genotypes caused by CS3005 ranged from 1.0 to 8.1 with an average of 3.5. The average FHB severity caused by CS3096 was higher, ranging from 1.8 to 12.0 with an average of 5.3. The correlation coefficient between FHB severities caused by the two isolates was highly significant at $r = 0.54$. However, significant correlations between CR and FHB resistance for either of the isolates was not detected ($r = 0.20$ for CS3005 and $r = 0.00$ for CS3096). A lack of strong correlation between reactions to these two diseases was also reflected by the best known genotype for FHB resistance, 'Sumai 3', and the best known genotype for CR resistance, '2-49'. The former gave only an average CR reaction (at 1.8) among the 32 genotypes tested and the FHB reaction of the latter (at 3.5) was not among the best (Table 1).

Distribution of phenotypic data for CR, FHB and PH in the DH population from 'Batavia'/'Ernie'

Transgressive segregation for CR severity was apparent in all six trials (Table 2). The frequency distributions of CR severity in all trials were approximately normal and slightly skewed towards lower severity (not shown). The average CR severity for CS3005 was lower than that for CS3096 in all three trials, and the two trials of CR05-2 and CR05-3 generated wider ranges of disease severities than the other four trials (Table 2). Strong correlations between CR severities were detected among all, but the CR96-3 trials (Table 3).

Table 2 Crown rot and Fusarium head blight severities of the two parents 'Batavia' and 'Ernie' and a doubled haploid population derived from them

Trial	Parents		DH population				
	Batavia	Ernie	Minimum	Maximum	Mean	SD	h^2
CR05-1	3.6	1.3	0.8	3.9	2.2	0.68	0.81
CR05-2	3.9	1.0	0.0	5.0	2.0	1.22	0.86
CR05-3	5.0	2.5	0.0	5.0	1.9	1.34	0.79
CR96-1	4.2	1.8	1.0	5.0	2.7	2.15	0.78
CR96-2	3.9	1.4	1.0	5.0	2.8	0.92	0.83
CR96-3	4.5	1.2	1.0	4.8	2.6	0.81	0.76
FHB-1	3.4	1.7	1.0	12.5	4.2	2.28	0.81
FHB-2	6.8	3.6	1.0	12.5	4.1	2.51	0.74
PH-1	56.5	65.8	37.0	80.4	63.0	8.82	0.77
PH-2	58.3	59.5	33.0	77.7	61.8	7.93	0.69

The three trials conducted using the *F. graminearum* isolate CS3005 were designated as CR05-1, CR05-2 and CR05-3, respectively, and those using the *F. pseudograminearum* isolate CS3096 were designated as CR96-1, CR96-2 and CR96-3, respectively. The two trials on FHB were designated as FHB-1 and FHB-2, respectively, and the two trials on plant height were designated as PH-1 and PH-2, respectively

Table 3 Correlation coefficients among crown rot and Fusarium head blight severities and plant height obtained from the doubled haploid population of 'Batavia'/'Ernie'

Traits	CR05-1	CR05-2	CR05-3	CR96-1	CR96-2	CR96-3	FHB-1	FHB-2	PH-1	PH-2
CR05-1	1.00									
CR05-2	0.66	1.00								
CR05-3	0.68	0.98	1.00							
CR96-1	0.36	0.24	0.25	1.00						
CR96-2	0.33	0.18	0.19	0.90	1.00					
CR96-3	0.65	0.82	0.81	0.62	0.57	1.00				
FHB-1	0.26	0.20	0.23	0.00	−0.06	0.10	1.00			
FHB-2	0.27	0.24	0.27	0.02	−0.04	0.15	0.97	1.00		
PH-1	−0.10	−0.32	−0.31	−0.01	0.00	−0.25	−0.04	−0.06	1.00	
PH-2	−0.08	−0.12	−0.12	−0.11	−0.05	−0.14	0.08	0.07	0.59	1.00

The three trials conducted using the *F. graminearum* isolate CS3005 were designated as CR05-1, CR05-2 and CR05-3, respectively, and those using the *F. pseudograminearum* isolate CS3096 were designated as CR96-1, CR96-2 and CR96-3, respectively. The two trials on FHB were designated as FHB-1 and FHB-2, respectively, and those on plant height were designated as PH-1 and PH-2, respectively

Transgressive segregation for FHB severity was observed in both trials (Table 2). Frequencies of FHB severity in both trials were approximately normally distributed and slightly skewed towards lower severity. The average FHB severities of the two trials were similar and correlation between the two trials was strong ($r = 0.97$). However, the correlation between FHB and CR severities was very weak (r values ranging from -0.06 to 0.27) (Table 3).

Transgressive segregation was also noted for PH in both trials and their frequencies were normally distributed. The average PH was similar between the two trials with a strong correlation at $r = 0.57$. Correlations between PH and CR were either non-existent or negative with r ranging from 0.00 to -0.32. Significant correlation between PH and FHB was not detected, with r ranging from -0.06 to 0.08 (Table 3). ANOVA detected highly significant effects not only between the genotypes, but also between the trials for all the four traits analysed (Supplementary Table 1). Thus, data from each of the trials were analysed separately for QTL detection (see below).

Marker segregation and linkage map of the 'Batavia'/'Ernie' DH population

A total of 956 polymorphic markers were scored in this population including 104 SSR and 852 DArT markers. About 25.9% (or 248) of these markers showed segregation distortion at $p = 0.05$. Among the distorted loci, 97 alleles (39.1%) favoured the parent 'Ernie' and the remaining 151 (60.9%) favoured the parent 'Batavia'. Of the scored markers, 57 were not included in the linkage map constructed as they could not be grouped with any marker with known chromosome location. The genetic map comprised 899 markers spanning a genetic distance of 1865.9 cM (Supplementary Fig. 1, Table 3). Of the three wheat genomes, genome D had the lowest proportion of markers covering the shortest distance. Marker densities vary between chromosomes, with chromosome 5D having the lowest density with an average of 6.7 cM between markers while 6A having the highest density with an average of 1.0 cM between markers. The overall average distance between markers across the whole map was 2.1 cM (Supplementary Table 3).

QTL for crown rot resistance

Analyses of data for both CS3005 and CS3096 identified a highly significant QTL for CR resistance on the long arm of chromosome 3B with wPt-1151 as the closest marker. The resistance allele of this locus was derived from the resistant parent 'Ernie'. This QTL explained 16.7% (LOD 3.3), 27.6% (LOD 5.9) and 28.2% (LOD 6.1) of the phenotypic variation in the three trials conducted with CS3005 (Fig. 1); and 18.7% (LOD 5.3), 24.7% (LOD 5.8) and 34.6% (LOD 7.6) in the three trials conducted with CS3096 (Fig. 2). Additional loci conferring CR resistance were detected for neither of the two different isolates.

QTL for FHB resistance

Three QTL were identified for FHB resistance from the first trial. They accounted for 15.9, 17.4 and 23.5% of the phenotypic variance at LOD values of 3.2, 3.5 and 4.4, respectively. The largest of the QTL was located on chromosome 5B with wPt-3661 as the closest marker. The

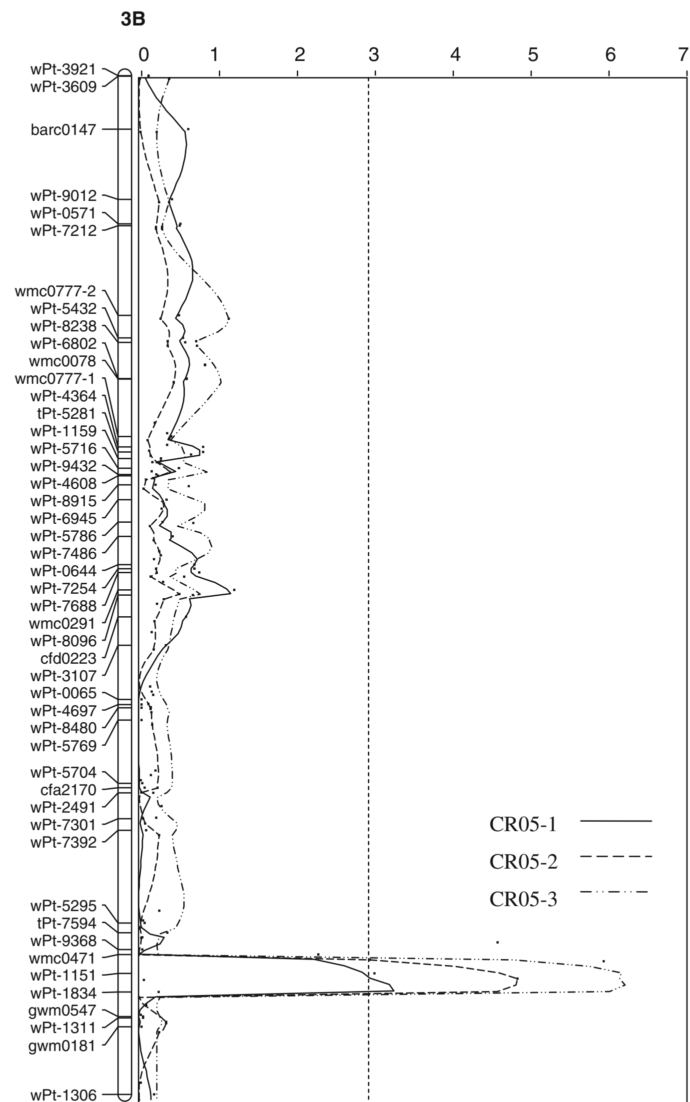

Fig. 1 QTL conferring crown rot resistance detected in three replicated trials using a *Fusarium graminearum* isolate (CS3005) in a doubled haploid population of 'Batavia'/'Ernie'. The LOD values (*x* axis) from each centimorgan were plotted against the chromosome (*y* axis), and the threshold LOD value (2.9) based on permutation test for declaring the presence of a QTL is indicated by a *dotted line*

other two were located to chromosomes 3A and 6A, respectively (Table 4). The resistant alleles of the three QTL were all derived from the resistant parent 'Ernie'.

Two QTL were detected from the second trial, one mapped on chromosome 6A explaining 20.2% of the phenotypic variance with a LOD value of 3.8, and the other mapped to chromosome 5B explaining 22.6% of the phenotypic variance with a LOD value of 4.2. They have similar locations as two of the QTL detected from the first trial. The QTL on chromosome 3A detected in the first trial was not detected in this second trial (Table 4).

QTL for plant height

Analysis of data from the first trial detected two QTL for plant height. The major one explaining 30.1% of the phenotypic variance was located on the short arm of chromosome 2B with gwm0271 as the closest marker. The minor one explained 10.8% of the phenotypic variance and was located on chromosome 3B with wPt-1834 as the closest marker. The tall allele of the QTL on chromosome 2B originated from 'Ernie' and that of the 3B QTL was derived from 'Batavia'. The PH QTL on chromosome 3B was located to a similar region as the single QTL for CR resistance (Table 4).

Data from the second trial also revealed two QTL for plant height. One of them was located to a similar region with the 2B QTL detected in the first trial, but with higher LOD (7.8) and r^2 (33.4%) values. The second QTL, explaining 15.6% of the phenotypic variance, was located on chromosome 5B at a similar region as the 5B QTL identified for FHB resistance (Table 4).

Effect of PH on CR reaction

As the QTL conferring CR resistance on chromosome 3B was located in a similar region as one of the minor QTL

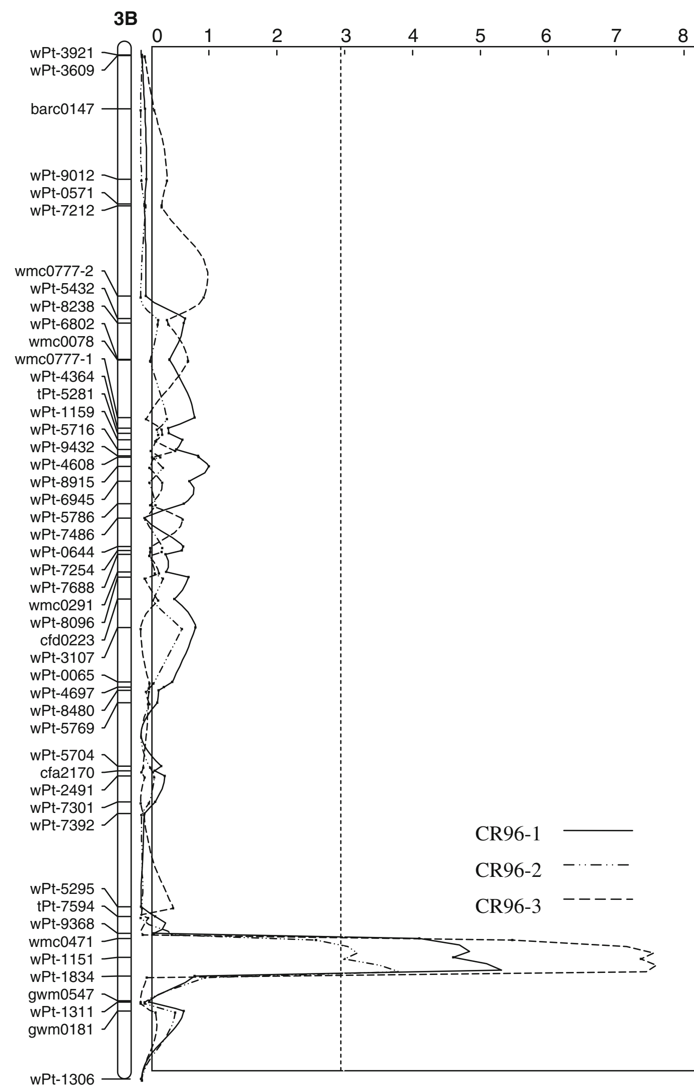

Fig. 2 QTL conferring crown rot resistance detected in three replicated trials using a *Fusarium pseudograminearum* isolate (CS3096) in a doubled haploid population of 'Batavia'/'Ernie'. The LOD values (*x* axis) from each centimorgan were plotted against the chromosome (*y* axis), and the threshold LOD value (2.9) based on permutation test for declaring the presence of a QTL is indicated by a dotted line

conferring PH, possible effects of PH on CR reaction was further analysed. This was carried out using combined data from the three CR trials with CS3005 and the three trials with CS3096 against the combined data from the two PH trials. When the effect of PH was accounted for by covariance analysis, there was very little change in the location and magnitude of the crown rot QTL on chromosome 3B (Table 4), indicating that PH had no significant effect on CR reaction.

Discussion

QTL conferring FHB resistance in 'Ernie'

A previous study identified that FHB resistance in 'Ernie' was controlled by four loci, located on chromosomes 2B, 3B, 4BL and 5A, respectively (Liu et al. 2007). However, none of these QTL was detected in the current study which detected three QTL, locating on chromosomes 3A, 6A and 5B, respectively. The QTL on chromosomes 6A and 5B were detected in both trials while the one on chromosome 3A reached significant levels in only one of the two trials (Table 4).

The different QTL detected from the same resistant genotype in the two different studies could be due to several possible reasons. First, QTL mapping can only detect differences between the two parents, but different susceptible parents were used in the current study and that reported by Liu et al. (2007). Second, the two studies were carried out at different environments with the use of different *F. graminearum* isolates. The possible influence of different isolates on FHB assessment is debatable. Results from some studies showed that FHB resistance is horizontal and non-species specific and non-strain specific (van Eeuwijk et al. 1995; Mesterhazy et al. 1999). These findings are enforced by performance of some well-known resistance sources, such as 'Sumai 3' which has

Table 4 QTL for crown rot and Fusarium head blight reactions and for plant height identified in the doubled haploid population of 'Batavia'/'Ernie'

Trials	Chr.	LOD	Interval (cM)	Closest markers	R^2 (%)	Origin
CR05-1	3BL	3.3	136.4–141.3	wPt-1151	16.7	Ernie
CR05-2	3BL	5.2	136.4–141.3	wPt-1151	27.6	Ernie
CR05-3	3BL	6.1	136.4–141.3	wPt-1151	28.2	Ernie
CR96-1	3BL	5.8	136.4–141.3	wPt-1151	24.7	Ernie
CR96-2	3BL	4.3	136.4–141.3	wPt-1151	18.7	Ernie
CR96-3	3BL	7.6	136.4–141.3	wPt-1151	34.6	Ernie
FHB-1	3A	3.2	4.2–5.7	wPt-1353	15.9	Ernie
	6A	3.5	0.0–1.0	wPt-6268	17.4	Ernie
	5B	4.4	66.8–74.4	wPt-3661	23.5	Ernie
FHB-2	6A	3.8	0.0–1.0	wPt-6268	20.2	Ernie
	5B	4.2	66.8–73.5	wPt-3661	22.6	Ernie
PH-1	2B	6.7	27.1–39.6	ta0249-1	30.1	Ernie
	3BL	3.1	141.8–142.3	wPt-1834	10.8	Batavia
PH-2	2B	7.8	25.8–31.1	wPt-0981	33.4	Ernie
	5B	3.1	62.4–65.3	wPt-4628	15.6	Ernie
CR05-PH	3BL	6.0	136.4–141.3	wPt-1151	27.5	Ernie
CR96-PH	3BL	8.8	139.3–145.2	wPt-1834	35.8	Ernie

The three trials conducted using the *F. graminearum* isolate CS3005 were designated as CR05-1, CR05-2 and CR05-3, respectively and the analysis of using the combined results of the three trials was designated as CR05. Similarly, the three trials conducted using the *F. pseudograminearum* isolate CS3096 were designated as CR96-1, CR96-2 and CR96-3, respectively and the analysis conducted using the combined data was designated as CR96. The two trials on FHB were designated as FHB-1 and FHB-2, respectively and the analysis of the combined data was designated as FHB; and the two trials on plant height were designated as PH-1 and PH-2, respectively and the analysis using the combined data was designated as PH. CR05-PH and CR96-PH represent QTL analyses of the combined data from the three CR trials with CS3005 and the three trials with CS3096, respectively, against the combined data from the two PH trials

consistently showed resistance to various *Fusarium* pathogens under different environments worldwide. However, a recent report by Akinsanmi et al. (2006) showed that FHB can be strain-specific and these authors reiterated the need to consider pathogen variability in the screening, selection and improvement of resistance to FHB in wheat. Third, the discrepancy could be due to the fact that accurate evaluation of FHB is notoriously difficult (Ireta and Gilchrist 1994). For example, the effect of *Fhb-1* detected in different studies varied from as low as 15.4% (Waldron et al. 1999) to as high as 57% (Buerstmayr et al. 2002). The magnitudes of the four loci conferring resistance in 'Ernie' detected by Liu et al. (2007) are all moderate, with the largest explaining only up to 19.6% of the phenotypic variance. Thus, failed detections of these QTL under different environments and with the use of different pathogen isolates could be understandable.

QTL conferring CR resistance identified up to date

Prior to the current study, there are only three reports on CR gene mapping and they dealt with three different genotypes with partial resistance to CR. The first was on the variety 'Kukri'. The adult plant resistance of this variety was assessed by growing plants in open-ended tubes placed in outdoor terraces. A locus on chromosome 4B near the semi-dwarfing gene *Rht1* was identified (Wallwork et al. 2004). The second was on the breeding line '2-49'. Based on a seedling assay, two QTL conferring CR resistance were detected from this genotype. One of them, located on chromosome arm 1DL, explained up to 21% of phenotypic variance. The other, located on chromosome arm 1AL, explained up to 10% of the variance (Collard et al. 2005). The third report was on the genotype 'W21MMT20', again based on a seedling assay (Bovill et al. 2006). These authors reported several putative QTL conferring CR resistances. However, none of them reached significant levels in all of the three trials conducted. The most significant QTL was located on 5D. It reached significant levels in two of the three trials and explained up to 28.0% of the phenotypic variance. The other QTL, located on 2D, reached significant level in only one of the three trials conducted and it explained 10.2% of the phenotypic variance. It is of note that none of the five loci derived from the three different resistant genotypes co-locate. They were found on five different chromosomes.

The QTL identified in this study map to yet another chromosomal region, the long arm of chromosome 3B. The

fact that the six CR QTL identified so far locate on six different chromosomes seems to suggest that the genetic mechanism for CR resistance is multigenic like that for FHB resistance. QTL conferring FHB resistance have been found on 20 out of the 21 possible chromosomes (Buerstmayr et al. 2009). However, there are more than 50 reports on FHB mapping, but only 4 on CR so far. Nevertheless, the magnitude of the CR QTL identified in this study, explaining up to 34.6% of the phenotypic variance with detection in each of the six trials with the use of two different *F. pseudograminearum* and *F. graminearum* isolates, represents the largest QTL conferring CR resistance reported in wheat to date.

Possible effects of plant height on CR reaction

Two of the three available genetic studies on CR resistance in wheat noted an effect of PH on CR reaction. Both of the studies dealt with the same semi-dwarfing gene *Rht1* and both claimed that it was the taller plants that gave better resistance (Wallwork et al. 2004; Collard et al. 2005). A recent report in barley also found significant association between PH and CR resistance. However, the barley results showed that the shorter plants gave increased resistance (Li et al. 2009). Results from the current study showed that PH is uncorrelated or negatively correlated with CR resistance, with shorter plants giving the same or better CR resistance. The different relationships between PH and CR resistance reported so far raise two further questions: whether different *Rht* genes have different effects on CR reaction, and/or whether the detected relationships are determined by the magnitude of the different *Rht* genes in different genetic backgrounds. Understanding why the different relationships exist for these two traits would facilitate the breeding of CR-resistant varieties and this forms part of our ongoing research.

Relationship between CR and FHB reaction

The common aetiology of FHB and CR prompted this study to determine a possible association between host resistances to these two different diseases. A strong association between host resistances to these two diseases could greatly enhance CR research as large volumes of FHB resistance data have been accumulated. Unfortunately, results from each of the two different aspects investigated in the studies reported here showed that such an association is unlikely: FHB and CR reactions of the panel of genotypes assessed showed no strong correlation. A similar lack of strong correlation between FHB and CR reactions was also noted among the DH lines used (Table 3); and loci conferring resistance to these two diseases do not co-locate (Table 4).

The finding that different genes could be involved in resistance to the two different diseases with the common aetiology is not totally surprising. There are at least two major differences between them. One is the time of infection: CR infects at the seedling stage while FHB infects at anthesis. It has been well established that different genes can be involved at different developmental stages of host plants in resistance to the same disease. One of the best known examples is perhaps the resistance to rust in wheat, where the difference between seedling and mature plant resistance has been well documented (Johnson and Law 1975). The other major difference between FHB and CR is the different organs they infected. Clearly, genes affecting both FHB and CR may exist in some germplasm or under certain conditions. Nevertheless, the results from this study indicate that the data accumulated from FHB research for CR research could be of limited value and separate screening seems to be essential in identifying novel sources of CR resistance.

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