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A major QTL conferring crown rot resistance in barley and its association with plant height

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Abstract Crown rot (CR) is one of the most destructive diseases of barley and wheat. *Fusarium* species causing CR survive in crop residue and a growing acceptance of stubble retention practices has exacerbated disease severity and yield loss. Growing resistant cultivars has long been recognised as the most effective way to reduce CR damage but these are not available in barley. In a routine screening of germplasm, a barley landrace from China gave the best CR resistance among the genotypes tested. Using a doubled haploid population derived from this landrace crossed to Franklin, we demonstrate that the CR resistance of TX9425 was conditioned by a major QTL. The QTL, designated as *Qcrs.cpi-3H*, was mapped near the centromere on the long arm of chromosome 3H. Its effect is highly significant, accounting for up to 63.3% of the phenotypic variation with a LOD value of 14.8. The location of *Qcrs.cpi-3H* was coincident with a major QTL conferring plant height (PH) and the effect of PH on CR reaction was also highly significant. When the effect of PH was accounted for by covariance analysis, the *Qcrs.cpi-3H* QTL remained highly significant, accounting for over 40% of the phenotypic variation. The existence of such a major QTL implies that breeding barley cultivars with enhanced CR resistance should be feasible.

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Introduction

Crown rot (CR), caused by various *Fusarium* species, is a severe and chronic disease of cereals found in many parts of the world (reviewed by Chakraborty et al. 2006). It has been known for a long time that both barley and wheat can be seriously affected by this disease (Wildermuth and Purss 1971). Fusarium head blight (FHB) is the other Fusarium disease affecting cereals and its economic importance stems from yield losses and the production of mycotoxins in grain. Mycotoxin contamination of grain restricts its use and threatens market access due to harmful effects on human and animal health. Recent studies in wheat have demonstrated that CR infected plants can accumulate mycotoxin in grain as well as other tissues (Mudge et al. 2006).

Crown rot has recently become more prevalent in Australia, most likely from the growing trend towards conservation farming practices involving stubble retention, as CR pathogens are carried over in residues (Wildermuth et al. 1997; Wallwork et al. 2004). Similarly, CR problems in barley and wheat have worsened in the Pacific Northwest of USA (Smiley et al. 2005). Various production practices are used in managing CR. These include crop rotation and stubble burning to reduce inoculum load (Burgess et al. 1996; Kirkegaard et al. 2004) and the use of partially resistant cultivars (Cook 1980). To breed CR resistant cultivars, significant efforts have been placed on

identifying and studying sources of resistance (Wildermuth and Purss 1971; Wallwork et al. 2004). Genetics of CR resistance from three partially resistant genotypes have been reported in wheat recently (Wallwork et al. 2004; Collard et al. 2005; Bovill et al. 2006). However, genetic study on CR resistance in barley has lagged behind and no resistant sources have been reported.

Crown rot and FHB share some key features: both can be caused by the same *Fusarium* pathogens (Akinsanmi et al. 2004), and resistant sources seem to be equally effective against different *Fusarium* pathogens (Mesterházy 1995; Liu et al. 2004). However, resistance to CR and FHB seems to be controlled by different genes. FHB resistant materials are not a good source for CR resistance (Liu et al. 2004) and the most effective allele controlling FHB resistance, *Fhb-1*, on the short arm of chromosome 3B of the spring wheat genotype Sumai 3, showed no effect on CR infection (Xie et al. 2006).

Studies of CR in barley have mainly considered disease incidence and severity with few reports considering yield losses. Smiley et al. (2005) noted an average of 13% yield reduction in eight winter malting barley cultivars, this compared with up to 61% loss in winter wheat cultivars in the same study. Recently, Daniel and Simpfendorfer (2008) found an average 20% yield loss in barley in a CR inoculated trial (~360 kg/ha) over different soil types, compared with 25% loss in bread wheat and 58% loss in durum wheat under the same conditions. These studies support the notion that barley cultivars have more severe CR symptoms than bread wheat cultivars (Wildermuth and Purss 1971; Klein et al. 1989) but they are more tolerant to CR infection and suffer less yield losses.

In a germplasm screening for CR resistance we have identified a barley landrace from China providing the best CR resistance among 54 cultivars and landraces tested. In this paper we report on experiments carried out to determine the genetic basis of resistance in this genotype using a DH population derived from a cross between this landrace and an elite Australian cultivar.

Materials and methods

Plant materials

A doubled haploid (DH) population of 92 lines was used to identify QTL conferring CR resistance and plant height (PH). The DH population was derived from TX9425 (a Chinese landrace resistant to CR) and Franklin (an elite Australian cultivar highly susceptible to CR). A linkage map for this population constructed using 412 DArT, 80 AFLP and 28 microsatellite markers is available (Wenzl et al. 2006; Li et al. 2008a).

Evaluation of crown rot resistance

A highly aggressive isolate of *Fusarium pseudograminearum* (Fp3096) collected in Northern New South Wales, Australia and maintained in the CSIRO collection (Akinsanmi et al. 2004) was used in this study. The procedures used for inoculum preparation were based on that described by Mitter et al. (2006). Specifically, plates of 1/4 strength PDA (potato dextrose agar) inoculated with Fp3096 were incubated for a week at room temperature. The mycelium was scraped and plates were incubated for a further week under a combination of cool white and black (UVA) fluorescent light with a 12 h photoperiod. Macroconidia were harvested, suspended in sterile distilled water after straining through layers of cheese cloth and spore concentration was adjusted to meet experimental requirements. Tween 20 was added (0.1% v/v) to the spore suspension prior to use.

CR reactions of the DH lines were assessed in three separate trials conducted between October 2007 and June 2008. The CR assay was based on procedures described by Li et al. (2008b). Seeds were germinated in a Petri-dish on three layers of filter paper saturated with water. Newly germinated seedlings were immersed in the conidial suspension (1×10^6 ml^{-1}) for 1 min. Three treated seedlings were sown in a 5 cm × 5 cm square punnet (Rite grow kwik pots, http://www.Gardencityplastics.com, Australia) containing autoclaved potting mix. Two replicates, each containing 15 seedlings in 5 punnets arranged in a randomized block design, were used in each of the three trials. Thirty punnets were placed in each plastic seedling tray for easy handling. After planting, the seedling trays were incubated for 24 h in a humid chamber and then transferred to a glasshouse with 25/15 (±5) °C day/night temperature and 60/80 (±10) % day/night relative humidity (RH). Seedlings were watered only when wilt symptoms appeared. CR severity was assessed at 35 days after inoculation with a 0–5 scale according to Li et al. (2008b).

Plant height

PH was measured from a 2005/2006 field trial conducted in Hobart, Tasmania (Latitude: 42°54′, South; Longitude: 147°18′, East, designated as Hobart trial) and a glasshouse trial conducted in 2007/2008 in Brisbane (Latitude: 27°30′, South; Longitude: 153°00′, East, designated as Brisbane trial), Queensland in Australia. The Hobart trial consisted of three replicates. In each of the replicates, the two parents and the DH lines were hand sown with a single row of 1.5 m long with 50 seeds. The rows were spaced 20 cm apart. Randomized block design was used in the trial. Ten plants in the middle of each row were measured for PH. The average value from the ten plants was used to represent

the height of a genotype in each of the replicates. For the glasshouse trial in Brisbane, young seedlings were vernalized for 6 weeks at 4°C and then transplanted in a glasshouse. The trial consisted of two replicates, each with four plants. Main tillers from the four plants from each replicate were measured at maturity and the average value from the four plants was used to represent the height of a genotype in each of the replicates.

Statistical analysis

All statistical analyses were performed using GenStat for Windows in the 10th edition (copy right Lawes Agricultural Trust, Rothamsted Experimental Station, UK). An analysis of variance was used to detect significant genetic effects for both CR and PH in the DH population. 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 genotype was treated as a random effect and that of replicates as fixed. Heritability was estimated from the ANOVA using the formula: $h^2 = \sigma^2_G/[\sigma^2_G + (\sigma^2_e/r)]$, with σ^2_G the genetic variance, σ^2_e the residual variance and r the number of replicates per genotype (Nyquist 1991).

QTL analysis

QTL analysis was preformed using MapQTL® 5.0 (Van Ooijen 2004). The Kruskal–Wallis test (a non-parametric equivalent of the one-way ANOVA) was used in a preliminary analysis to detect associations between markers and individual traits. This was followed by interval mapping (IM) to identify the major QTL. Automatic cofactor selection was used to fit the multiple QTL model (MQM) [backward elimination ($P > 0.02$)] and to detect significantly associated markers as cofactors. For each trait, a permutation test was performed to identify the LOD threshold corresponding to a genome-wide false discovery rate of 5% ($P < 0.05$). Based on the permutation tests (1,000 permutations), a threshold LOD value was used to declare the presence of QTL in the interval MQM analyses. QTL identified in more than one trial which mapped closely to one another on the same linkage group, and with alleles derived from the same parent, were considered to represent the same QTL. A linkage map was drawn using MAPCHART (Voorrips 2002).

Results

Crown rot reaction of the DH population

Transgressive segregations of CR severities for the DH lines were apparent for all the three trials (designated as CRS1, CRS2 and CRS3, respectively). Frequency distributions for severity in the three trials were all approximately normally distributed and slightly skewed towards greater susceptibility (Table 1). The average CR ratings for two of the three trials were similar (2.15 for CRS1 and 2.17 for CRS2), although CRS1 gave a wider variation from 0.13 to 3.80 than 0.94 to 3.50 for CRS2 (Table 1). CRS3 produced the lowest average CR rating (1.83) with a narrower distribution from 0.00 to 3.70. Heritability of CR resistance was high in all trials, with h^2 ranging from 0.74 to 0.83 (Table 1).

PH of Franklin, TX9425 and their derived DH lines

The cultivar Franklin likely carries the *denso* semi-dwarfing gene (see "Discussion"). The average height of this variety was 86.3 centimetres (cm) in the Hobart trial and 83.4 cm in the Brisbane trial. The difference in height from the two contrasting environments, one in the Hobart winter sown field trial and the other in a summer sown glasshouse trial in Brisbane, was only about 3%.

TX9425 is a landrace from China and it is not clear what gene(s) conferred PH in this genotype. The average height of this genotype was 57.60 cm from the Hobart

Table 1 Crown rot severity and plant height of the two parents Franklin and TX9425 and their derived DH population

Traits	Trials	Mean for parents		DH lines				
		TX9425	Franklin	Minimum	Maximum	Mean	SD	h^2
Crown rot Severity (0–5 scale)	CRS1	1.36	3.06	0.13	3.80	2.15	0.60	0.74
	CRS2	1.70	2.92	0.94	3.50	2.17	0.58	0.83
	CRS3	1.53	3.00	0.00	3.70	1.83	0.70	0.81
Plant height (cm)	Hobart	57.60	86.32	40.95	99.30	64.07	12.58	0.76
	Brisbane	36.64	83.40	25.25	83.75	42.95	13.72	0.89

trial and 36.63 cm from the Brisbane trial, with a 36% difference in height between the two environments. Also, compared with that of Franklin, TX9425 was 36% shorter in the Hobart trial and 47% shorter in the Brisbane trial.

The distributions of PH of the DH population in the two trials were all transgressive and skewed towards the shorter parent TX9425. The height of the DH population ranged from 40.95 to 99.3 cm with an average of 64.07 cm in the Hobart trial, and from 25.25 to 83.75 cm with an average of 42.95 cm in the Brisbane trial. In both trials, the average height of the DH population was in-between the PH of the two parents (Table 1). As expected, the values of heritability of PH detected in both of the trials were high, with h^2 of 0.76 and 0.89 respectively (Table 1).

QTL analysis

QTL for Crown rot reaction

MQM analyses of data from the three CR assessments identified a similar QTL conferring CR resistance at threshold LOD values of 2.7, 2.8 and 2.8, respectively. This QTL was located on the long arm of chromosome 3H flanked by markers bPb-4747 and bpb-6765 (Fig. 1). We have designated the QTL as *Qcrs.cpi-3H*, where 'crs' represents 'crown rot severity' and 'cpi', CSIRO Plant Industry following convention. The resistant allele of this locus was derived from the resistant parent TX9425. This QTL explained 34.1% (LOD 5.2), 44.5% (LOD 8.1) and 60.4% (LOD 9.8) of the phenotypic variation respectively in the three separate trials. Combining data from the three trials further improved both LOD value (14.8) and the magnitude (63.3%) of *Qcrs.cpi-3H* (Table 2). Additional loci conferring CR resistance were not detected from this DH population using data from any of the three separate trials or the three trials combined.

QTL for plant height

MQM analysis of the Hobart trial detected two QTL at a threshold LOD value of 2.8. Both QTL were located on the long arm of chromosome 3H, one (*Qph.cpi-3H.1*) flanked by markers bPb-4747 and bPb-6765 and the other (*Qph.cpi-3H.2*) flanked by markers Bmag606 and bPb-2550 (Table 2, Fig. 2). The magnitude of *Qph.cpi-3H.1* is slightly larger than that of *Qph.cpi-3H.2*. The peaks of the two PH QTL were separated by a distance of 22 cM. The proximal *Qph.cpi-3H.1* was derived from TX9425, and the distal *Qph.cpi-3H.2* was derived from Franklin.

The PH data from the glasshouse trial in Brisbane generated a single QTL at a threshold (highly significant)

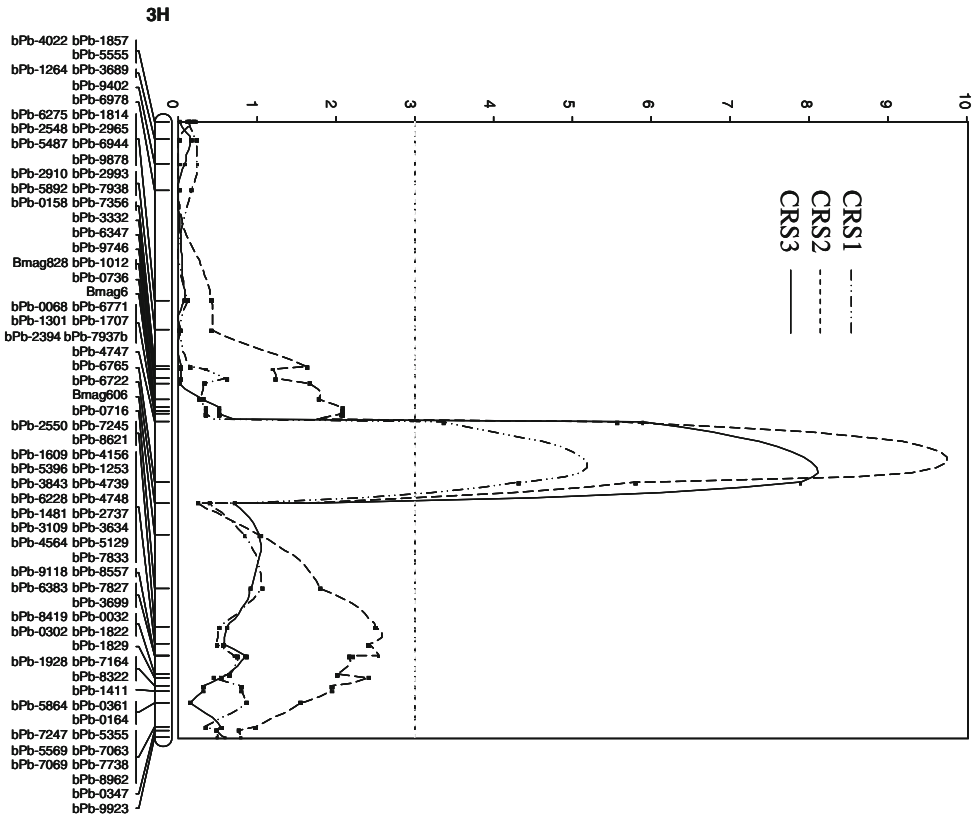

Fig. 1 QTL conferring crown rot resistance in the barley DH population of Franklin/TX9425 identified using composite interval mapping on the long arm of chromosome 3H. The LOD values from each centimorgan of the chromosome were plotted against the chromosome

Table 2 QTL for crown rot severity (CRS) and plant height (PH) identified in the DH population of Franklin/TX9425

Trials	QTL	LOD	Chr	Interval	Flanking markers	R^2 (%)	Origin
Crown rot severity							
CRS1	*Qcrs-cpi-3H*	5.2	3H	59.7–71.8	bPb-4747 and bPb-6765	34.1	TX9425
CRS2	*Qcrs-cpi-3H*	9.8	3H	59.7–71.8	bPb-4747 and bPb-6765	60.4	TX9425
CRS3	*Qcrs-cpi-3H*	8.1	3H	59.7–71.8	bPb-4747 and bPb-6765	44.5	TX9425
Combined	*Qcrs-cpi-3H*	14.8	3H	59.7–71.8	bPb-4747 and bPb-6765	63.3	TX9425
Plant height							
Hobart	*Qph-cpi-3H-1*	11.9	3H	59.7–70.9	bPb-4747 and bPb-6765	42.4	TX9425
	Qph-cpi-3H-2	8.9	3H	82.2–99.8	Bmag606 and bPb-2550	30.5	Franklin
Brisbane	*Qph-cpi-3H-1*	20.9	3H	59.7–70.9	bPb-4747 and bPb-6765	74.7	TX9425
Combined	*Qph-cpi-3H-1*	8	3H	59.7–70.9	bPb-4747 and bPb-6765	25.9	TX9425
	Qph-cpi-3H-2	14.3	3H	82.2–99.8	Bmag606 and bPb-2550	53.6	Franklin
PH adjusted CRS							
Combined	*Qcrs-cpi-3H*	8.8	3H	59.7–70.9	bPb-4747 and bPb-6765	44.8	TX9425

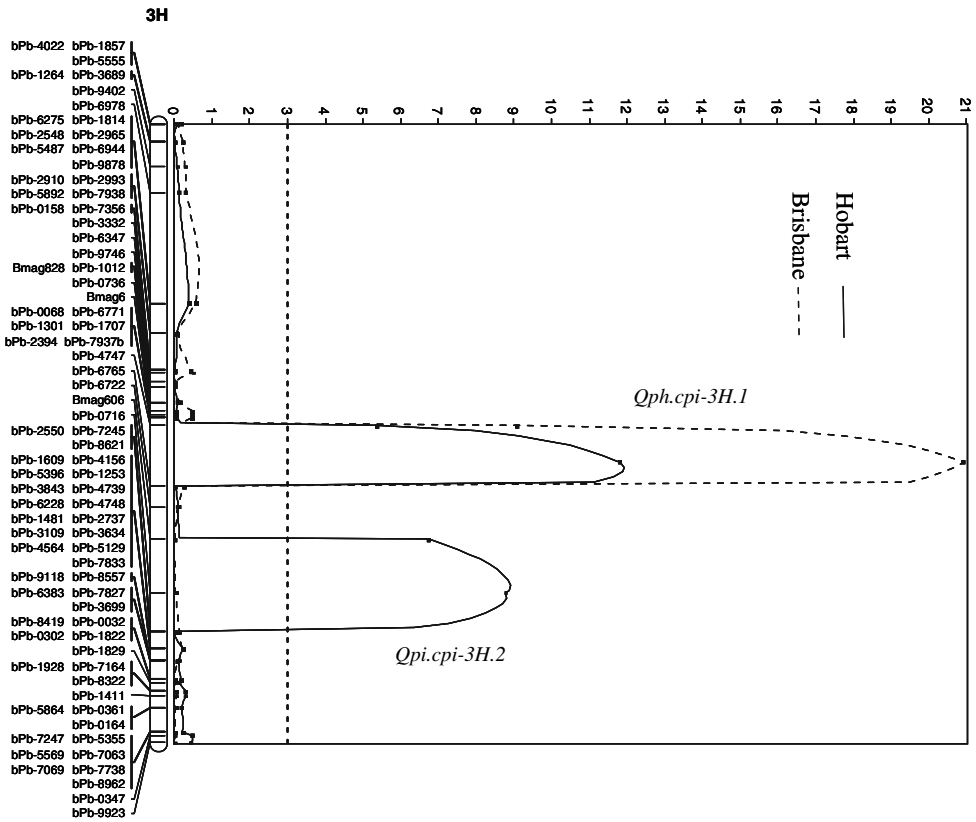

Fig. 2 QTL for plant height in the barley DH population of Franklin/TX9425 identified using composite interval mapping on the long arm of chromosome 3H. The LOD values from each centimorgan of the chromosome were plotted against the chromosome

LOD value of 2.7. This QTL had the same location as *Qph.cpi-3H.1* but was more significant with a LOD 20.9 and 74.7% R^2 (Table 2, Fig. 2). When data from the two trials were combined, the same two PH QTL (position-wise) were detected, but the effect of *Qph.cpi-3H.1* (LOD 8.0 and R^2 25.9%) was smaller than from either of the two separate trials, and the effect of *Qph.cpi-3H.2* (LOD 14.3 and R^2 53.6%) was intermediate (Table 2).

Effect of PH on CR reaction

The QTL conferring CR resistance from TX9425 (*Qcrs.cpi-3H*) was mapped to the same position as the QTL conferring PH from the same parent (*Qph.cpi-3H.1*). To test any possible effect of PH on CR reaction, combined data from the three CR trials were analysed against the combined data from the two PH trials by covariance

analysis. This analysis showed that PH had a significant effect on CR reaction. Both LOD value and the magnitude of *Qcrs.cpi-3H* were reduced (LOD from 14.8 to 8.8 and R^2 from 63.3 to 44.8) when the effect of height was accounted for (Table 2, Fig. 3). Despite this, the CR QTL remained highly significant and its position was not affected.

Discussion

Homoeologous relationship between *Qcrs.cpi-3H* and other reported loci on 3HL conferring CR and FHB resistance in barley and wheat

Comparative analysis based on co-linearity between closely related species has been routinely used to predict and locate genes/markers of interest. Apart from a few well-characterized translocations between the A and B genomes of wheat (Liu et al. 1992), gene orders between barley and wheat are highly conserved (Devos 2005). Several loci conferring CR resistance have been reported for bread wheat, and were located on chromosomes 4B (Wallwork et al. 2004), 1A and 1D (Collard et al. 2005), and 2B, 2D and 5D (Bovill et al. 2006). As chromosome 3H of barley is homoeologous with the group 3 chromosomes of wheat (Devos 2005), it seems that none of the reported wheat loci conferring CR resistance are homoeologous with *Qcrs.cpi-3H*, the first reported QTL conferring CR resistance in barley.

Crown rot and FHB are two different diseases that can be caused by the same pathogens (Akinsanmi et al. 2004), and research has focused on determining whether resistance genes from a host plant to these two diseases are the same (Liu et al. 2004; Xie et al. 2006). Most of the barley genotypes studied so far showed that FHB resistance is controlled by multiple genes. For example, using a population derived from the two-rowed parents, Gobernadora and CMB643, Zhu et al. (1999) found QTL for FHB resistance on six of the seven barley chromosomes. Similarly, in the Chevron/M69 population de la PeNa et al. (1999) also found QTL for FHB on 6 of the 7 barley chromosomes. Using a population derived from the cross Chevron/Stander, Ma et al. (2000) reported nine QTL for FHB resistance on five of the barley chromosomes. Although chromosome 3H was found to be involved in FHB resistance in some of the studies, the multi-loci nature of FHB resistance is in clear contrast to the single locus situation for CR resistance revealed in this first study on CR QTL in barley.

Relationship between the two PH loci detected in this study with other known PH loci on 3HL

One of the parents used in this study, Franklin, is a long-season malting cultivar derived from a cross between the

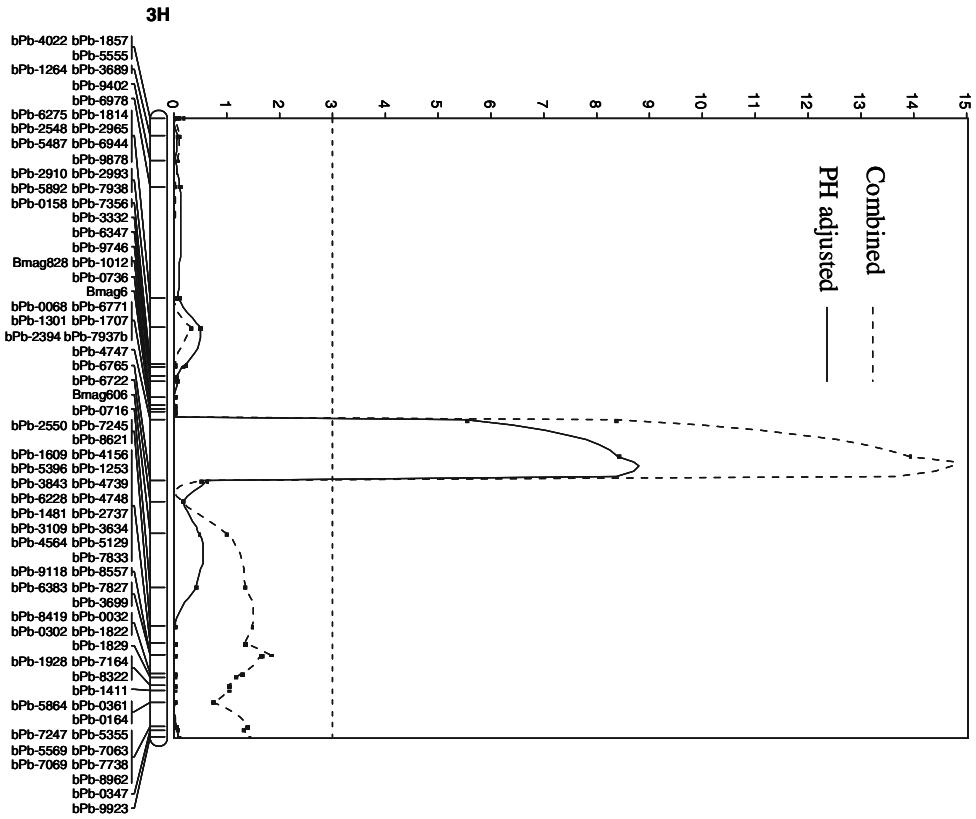

Fig. 3 QTL for crown rot severity from combined data of the three CR trials pre-(combined) and post-adjustment by plant height (PH adjusted) on the long arm of chromosome 3H. The LOD values from each centimorgan of the chromosome were plotted against the chromosome

Australian cultivar Shannon (Proctor*4/CI3208-1) and the German cultivar Triumph (Diamant/St 1402964/6) (Vertigan 1991). As it is known that Triumph carries the *denso* dwarfing gene (Laurie et al. 1995) which is widely present in malting barley cultivars (Mickelson and Rasmusson 1994), the malting cultivar Franklin likely carries this dwarfing gene. It is also known that *denso* is allelic with another dwarfing gene *sdw* which are located on the long arm of 3H (Hellewell et al. 2000). Thus, *Qph.cpi-3H.2* detected in this study very likely represents the *denso/sdw* locus.

The other parent, TX9425, is a landrace from China and showed marked variation in plant height when growing under different environments (MX Zhou, unpublished). This genotype was about 33% shorter than Franklin in the Hobart trial and was only about half of the height of Franklin in the Brisbane trial. Previous studies showed that the semi-dwarf gene *uz*, also located on 3HL (Tsuchiya 1986), is prevalent among Chinese barley genotypes and it segregated independently from the locus *sdw* (Zhang 1998). Thus, it was somewhat expected that two loci conferring PH were detected in this study, *Qph.cpi-3H.1* from TX9425 and *Qph.cpi.3H.2* from Franklin. As shown in Fig. 2, *Qph.cpi.3H.1* from the latter is more proximally located. However, the two loci detected in this study were not independently segregated. Rather, they are linked and the genetic distance from the peaks of the two QTL is only 22 cM. If the PH loci of *uz* and *sdw* indeed segregate independently as claimed by Zhang (1998), *Qph.cpi.3H.1* from TX9425 detected in this study may represent a new locus which is different from that of *uz*. In other words, there are at least three loci on 3HL conferring PH, including *denso/sdw, uz* and *Qph.cpi.3H.1* identified in this study. The possibility that *Qph.cpi.3H.1* from TX9425 could be new is very significant, as many of the currently widely used PH genes in barley show inferior effects on grain yield and other traits of agronomic importance (Hellewell et al. 2000). Thus, there is an urgent need to clarify if *Qph.cpi.3H.1* from TX9425 represents a new locus.

However, we could not rule out the possibility that *Qph.cpi-3H.1*is in fact the same as *uz*. It is well known that recombination rates between loci could be dramatically affected by numerous factors including the use of different populations (Liu et al. 1996). It is possible that, for some unknown reasons, the recombination rate on the long arm of chromosome 3H was somehow dramatically reduced in the population used in this study.

It is of interest to note that the two PH loci detected in this study had dramatically different responses between the two trials. As shown in Table 2 and Figure 2, the effect of *Qph.cpi-3H.2* from Franklin was detected only from the Hobart trial, while that of *Qph.cpi-3H.1* from TX9425 was detected from both trials (Table 2, Fig. 2). Similarly, compared with that of Franklin, the height of TX9425 was also changed more dramatically between the two trials. It was not unexpected that the very different environments between the field trial in Hobart and the glasshouse trial in Brisbane would significantly affect PH. However, the different responses of the two QTL between the two trials warrant further study, as it would affect its value in breeding programs.

The usability of Qcrs.cpi-3H and Qph.cpi-3H.1

There had only been one report to date on the possible association between CR infection and PH (Wallwork et al. 2004). These authors detected a CR QTL co-located with a PH locus on chromosome 4B in wheat, and found that the two traits were negatively correlated, i.e., taller plants showed to be more resistant. Surprisingly, in this first barley mapping study we also detected that QTL conferring PH (*Qph.cpi-3H.1*) and CR resistance (*Qcrs.cpi-3H*) co-located, even though this time their association is positive, i.e., shorter plants gave better CR resistance. It is not clear if the association between CR and PH in both of the reported studies is coincidental. However, one negative and one positive association between these two traits found in the two different studies suggest that PH per se may have no effect on CR resistance. The observed effect of PH on CR reaction could be due either to closely linked genes or the two traits were conditioned by the same gene(s) with pleiotropic effects. Nevertheless, it is paramount to clarity their relationship which would have significant consequences in successfully exploiting the CR locus as well as the PH QTL in breeding programs.

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