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Dissection of genetic components of preharvest sprouting resistance in white wheat

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Abstract Preharvest sprouting (PHS) in rain-affected wheat (*Triticum aestivum*) is a major constraint to the production of high-quality wheat, especially in regions where white grain wheat cultivars are preferred. To characterize quantitative trait loci (QTLs) for PHS resistance and seed dormancy (SD), we evaluated 162 recombinant inbred lines developed from the cross between PHS-resistant white wheat landrace Tutoumai A and PHS-susceptible white wheat cultivar 'Siyang 936' for PHS resistance and SD in field and greenhouse experiments. Composite interval mapping (CIM) identified four QTLs for PHS resistance and long SD that explained up to 45 and 40.8% of the phenotypic variation in five PHS and four SD experiments, respectively. *Qphs.pseru-4A.1* was detected in three of the five PHS experiments, and *Qphs.pseru-5B.1*, *Qphs.pseru-5B.2*, and *Qphs.pseru-4B.1* were detected in two of the five PHS experiments, respectively. All four QTLs for PHS resistance also affected SD. *Qphs.pseru-4A.1* was significant in all four SD experiments; the other three QTLs were detected only in one experiment. Additive and epistatic effects were observed for PHS resistance and SD. Besides three additive QTLs for PHS resistance and two for long SD, an additional 11 and 10 QTLs were detected with epistatic effects on PHS resistance and SD, respectively. The major genetic component of PHS resistance was SD, and other genetic factors may also contribute to PHS resistance in this population.

Keywords Genetic components · Preharvest sprouting · Quantitative trait loci · Seed dormancy wheat

Introduction

Preharvest sprouting (PHS) of wheat (*Triticum aestivum* L. em Thell.) is the germination of grains in a physiologically matured wheat spike prior to harvest. It results from a combination of specific and appropriate moisture and humidity conditions and can be particularly severe if there is a long period of rainfall or a long period of high humidity after rainfall after the wheat matures. White wheat is more susceptible than red wheat (Wu and Carver 1999; Amano and Torada 2002; Chen et al. 2008). The exposure of grain to high moisture at ripening

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triggers a series of physiological processes, including the release of hydrolytic enzymes such as α-amylase. Increased amylase activity facilitates the hydrolysis of grain carbohydrate reserves, resulting in poor flour quality. Flour from sprouted wheat has decreased thickening power, and bread baked from sprouted wheat may result in collapsed loaves (Mansour 1993; Kottearachchi et al. 2006). Therefore, PHS reduces not only the grain yield but also the nutritional and processing quality, rendering grain unusable for many commercial products.

Resistance to PHS in plants is a complex trait, and its expression is significantly affected by environment. Seed dormancy (SD) has long been regarded as the major factor that delays or prevents PHS (Mares and Mrva 2001) and has been the focus of research on PHS resistance (Amano and Torada 2002; Kottearachchi et al. 2006; Tan et al. 2006). Other factors have also been proposed as potential contributors to overall PHS resistance, including germination inhibitory substances residing in the chaff tissue (Derera and Bhatt 1980; Gatford et al. 2002), physical barriers to water penetration in a spike (Gale 1989), and spike morphologies, such as structure of the wheat spike and awns, erectness of the spike, openness of the florets, and tenacity of the glumes (King and Richards 1984). However, how many of these other factors contribute to overall PHS is still unknown. Both SD and PHS resistance are controlled by several quantitative trait loci (QTLs) (Paterson and Sorrells 1990; Mares 1996; Flintham et al. 2002). Consequently, simultaneously mapping QTLs for both PHS resistance and SD should reveal the genetic relationship between the two traits.

Red grain wheat usually shows a higher level of PHS resistance than white grain wheat (Seshu and Sorrells 1986; Debeaujon et al. 2000). Three genes for red grain (*Red*) have been associated with long SD in wheat and mapped as homoeologous loci to the long arms of chromosome group 3, and flanking markers for some *Red* genes have also been reported (Nelson et al. 1995; Flintham et al. 1999; Groos et al. 2002; Kulwal et al. 2004, 2005; Kuraparthy et al. 2008). White wheat production is increasing in many countries because of end users' preference and other economic benefits, such as a higher flour extraction rate (McCaig and Depauw 1992) and fewer visible bran specks, which improve the appearance and acceptability of steam bread and noodle products in the Asian market (Amano and Torada 2002; Tan et al. 2006; Imtiaz et al. 2008; Ogbonnaya et al. 2008). Resistance to PHS is imperative for the successful production and marketing of white wheat in PHS-favorable environments, such as Australia and the USA (Morris and Paulsen 1989; Wu and Carver 1999; Imtiaz et al. 2008). The identification of QTLs for PHS resistance in white wheat will facilitate the genetic improvement of PHS resistance in white wheat cultivars.

Various mapping populations have been used to map QTLs for PHS resistance on different chromosomes of white wheat (Anderson et al. 1993; Kato et al. 2001; Mares and Mrva 2001; Osa et al. 2003; Mori et al. 2005; Mares et al. 2005; Torada et al. 2005; Tan et al. 2006; Chen et al. 2008; Imtiaz et al. 2008; Liu et al. 2008). One QTL on the short arm of 3A showed a major effect on PHS resistance in both red and white wheats (Osa et al. 2003; Mori et al. 2005; Liu et al. 2008). Another major QTL was identified on 4AL of Chinese landraces and Australian and African germplasm lines (Kato et al. 2001; Mares and Mrva 2001; Mares et al. 2005; Torada et al. 2005; Chen et al. 2008; Ogbonnaya et al. 2008). In addition to these two major QTLs, additional QTLs with minor effects have also been reported on 3D (Imtiaz et al. 2008), 4B and 4D (Kato et al. 2001), 5B (Tan et al. 2006), 6B and 7D (Roy et al. 1999), and other chromosomes (Anderson et al. 1993). Tutoumai A is a Chinese landrace with a high level of PHS resistance, and in greenhouse experiments, Chen et al. (2008) demonstrated that it has the major QTL on 4A for both PHS resistance and long SD. However, this QTL was found to explain only part of the genetic variation for PHS resistance; thus, other QTLs may also contribute to the overall PHS resistance in Tutoumai A. The objectives of this study were to: (1) identify additional QTLs for PHS resistance and SD in Tutoumai A through genome-wide marker screening, (2) characterize interactions between QTLs for SD and PHS and between QTL and environments by phenotyping a recombination inbred line (RIL) population in multiple environments, and (3) elucidate the genetic relationship between SD and PHS.

Materials and methods

Plant materials

A population of 162 F_6 RILs was derived from a cross between the white PHS-resistant Chinese landrace Tutoumai A and white PHS-susceptible Chinese cultivar 'Siyang 936' by single-seed descent. Both parents and their RILs were evaluated for PHS resistance in two field experiments (2005 and 2006) at Jiangsu Academy of Agricultural Sciences (JAAS), Nanjing, China, and in three greenhouse experiments (from 2005 to 2007) at Kansas State University (KSU), Manhattan, KS, USA. Seed dormancy was evaluated in four experiments from 2004 to 2006 in the same two locations.

Evaluation of PHS and seed dormancy

In the greenhouse experiments, six plants per RIL were transplanted into a 13 × 13-cm Tora pot (Hummert Int., St. Louis, MO) filled with Sun Glo Metro Mix 360 soil mix (Sun Gro Horticulture, Bellevue, WA) after vernalization at 4°C in a growth chamber for 8 weeks. Plants were grown on a greenhouse bench at 22°C day/15°C night under long-day conditions (16 h) with supplemented daylight. Each experiment was arranged in a randomized complete-block design with two replicates (pots). Seven to ten spikes were harvested from each pot at physiological maturity, which is characterized by the loss of green color on the spike (Kulwal et al. 2005). Harvested spikes were air-dried at 25°C for 5 days in the greenhouse and then stored in a freezer at −20°C to maintain dormancy. After all RILs had been harvested, all spikes were dried on a greenhouse bench for two more days. Five spikes per RIL were evaluated for PHS in a moist chamber, as described by Liu et al. (2008). Sprouted and non-sprouted kernels in each spike were counted, and the percentage of visible sprouted kernels (PVSK) in a spike was calculated by dividing the number of sprouted kernels with the total number of kernels in the spike to measure PHS resistance. Fifty hand-threshed kernels from the remaining spikes were evaluated for SD in each RIL as described by Chen et al. (2008), and a weighted germination index (GI) was used to measure SD.

In the field experiments, each RIL and their parents were sowed in 4-m-long rows in a two-row plot with an inter-row spacing of .25 m. The experiments were arranged in a randomized complete-block design with two replications at JAAS, Nanjing, China. At physiological maturity, when the spike and peduncle turned yellow, 20 heads per plot (10 spikes per row) were harvested, dried for 7 days, and then stored in a freezer at −20°C until PHS evaluation. Both PHS and SD were assessed as previously described for the greenhouse experiment, with the exception that ten heads per RIL instead of five were used for PHS evaluation.

Simple sequence repeat analysis

DNA was isolated by using the CTAB method. A total of 1,543 simple sequence repeat (SSR) primers, including BARC (Song et al. 2005), GWM (Röder et al. 1998), WMC (Somers et al. 2004), GDM (Pestsova et al. 2000), CFA, CFD (Guyomarc'h et al. 2002; Sourdille et al. 2003), DUP (Eujayl et al. 2002), KSM primers developed at KSU, Manhattan, KS, and the expressed sequence tag (EST) derived PCR marker ZXQ118 developed and mapped to the 4A QTL region by (Zhang et al. 2008), were used to screen for polymorphism between the two parents. A total of 315 polymorphic markers were analyzed as described by Liu et al. (2008).

Genetic linkage map construction and QTL analysis

The initial genetic linkage map was constructed with marker data from a subset of 96 RILs that were randomly selected from 162 RILs by using JoinMap software version 3.0 (Van Ooijen and Voorrips 2001). The threshold value of logarithm of odds (LOD) score was set at 3.0 to claim linkage between markers with a maximum recombination fraction at 0.4. Recombination fractions were converted to centiMorgans (cM) using the Kosambi function (Kosambi 1944).

The initial QTL analysis was conducted with the map constructed from 96 RILs, and the markers linked to potential QTLs from the initial analysis were further screened in an additional 66 RILs. Data from 162 RILs were analyzed together to construct a linkage map for further QTL analysis. Single locus analysis was performed using the composite interval mapping (CIM) function of WinQTLCart v2.5 (Wang

et al. 2005). A permutation test of 1000 runs was conducted to identify a threshold of logarithm of odds ($s = 2.4$) for declaring significant QTLs (Doerge and Churchill 1996). Two-locus analysis with QTLMapper 2.0 (Wang et al. 1999) (http://ibi.zju.edu.cn/software) was used to determine the additive effects (a) of QTLs, epistatic interactions between QTLs (Q × Q) and interaction between QTLs and environment (Q × E). Additive and epistatic effects of QTLs were analyzed with a mixed linear model (MLM) at 1-cM walking speed, and significant effects of additive and epistatic QTLs and Q × E interactions were identified at $p \leq 0.005$ (Yang et al. 2007; Imtiaz et al. 2008). QTLs for PHS resistance (*Qphs.pseru-chromosome*) were designated according to standard nomenclature (http://wheat.pw.usda.gov/ggpages/wgc/98/Intro.htm), where 'pseru' indicates that a QTL was named by the USDA–ARS–Plant Science and Entomology Research Unit, Manhattan, KS.

Statistical analysis

Data for PHS and SD were analyzed using two-way GLM in SAS for Windows v9 (SAS Institute, Cary, NC) to determine the effects of genotypes (G), environments (E), and G × E interactions on both traits. Least significant difference (LSD) was calculated for PHS and SD (Steel and Torrie 1980).

Heritability (h^2) was estimated with the formula (Toojinda et al. 1998)

$$H^2 = \sigma_g^2 / \left(\sigma_g^2 + \sigma_{ge}^2 / n + \sigma_e^2 / nr \right)$$

where σ_g^2 is the variance among RILs, σ_{ge}^2 is the variance for G × E, σ_e^2 is the variance of environments, n is the number of environments, and r is the number of replicates. Total R^2 for a trait was calculated by multiple linear regressions with the SAS REG procedure.

Results

Phenotypic variation of PHS and SD

The PVSK of Tutoumai A and 'Siyang 936' ranged from 6.8 to 48.4% and from 43.9 to 90.8%, respectively, in the five experiments at both locations. The GI for Tutoumai A and 'Siyang 936' ranged from 18.2 to 62.3% and from 61.2 to 92.7%, respectively, in the four experiments [Electronic Supplementary Material (ESM) Table S1]. Although large variations for both PVSK and GI ratings were observed within each parent among experiments, ratings were about 35 and 40% lower for Tutoumai A than for 'Siyang 936' in each individual experiment, indicating consistent differences in PHS resistance and SD between the parents under different environments. Mean PVSK and GI values of RILs were intermediate between the two parents, and frequency distributions of both traits were continuous but the peak positions varied among experiments (Fig. 1), indicating that both traits were quantitative and their expression was significantly affected by environment. Transgressive segregation was observed in all experiments (ESM

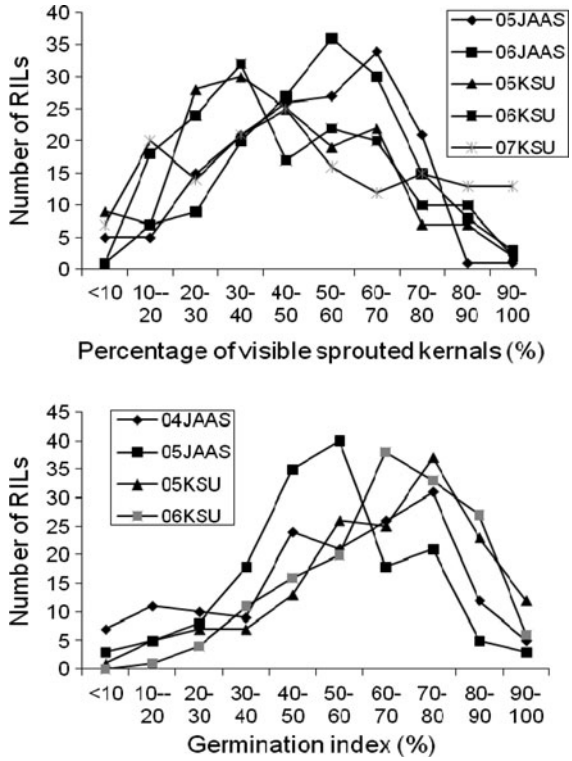


Fig. 1 Frequency distribution for percentage of visible sprouted kernels (PVSK) based on the mean value of 2005JAAS, 2006JAAS, 2005KSU, 2006KSU, and 2007KSU experiments and the mean value of the germination index in the 2004JAAS, 2005JAAS, 2005KSU and 2006KSU experiments from the RIL6 population of Tutoumai A/'Siyang 936'. *JAAS* Jiangsu Academy of Agricultural Sciences, *KSU* Kansas State University, *RIL* recombinant inbred line

Table 1 Putative QTLs for preharvest sprouting and seed dormancy determined using composite interval mapping in recombinant inbred lines with spikes and seeds harvested from field trials in 2004, 2005, 2006 (JAAS, Nanjing, China) and 2005, 2006, 2007 (KSU, Manhattan, KS) and the mean value across the environments

QTL and its location	Marker interval	2004 JAAS		2005 JAAS		2006 JAAS		2005 KSU		2006 KSU		2007 KSU		Mean over experiments[2]	
		LOD	R^2	LOD	R^2	LOD	R^2	LOD	R^2	LOD	R^2	LOD	R^2	LOD	R^2
PHS															
Qphs.pseru-4A.1	*Xbarc170-Xgwm397*	–[a]	–	2.3	6.2	0.7	2.5	6.1*	15.6*	7.2*	17.8*	3.8*	10.7*	12.0*	25.3*
Qphs.pseru-4B.1	*Xwmc238-Xbarc20*	–	–	0.6	2.0	0.1	0.2	1.2	3.8	2.6*	5.4*	2.6*	6.1*	0.8	3.9
Qphs.pseru-5B.1	*Xwmc75-Xbarc275*	–	–	2.1	5.2	7.1*	27.0*	0.8	2.5	2.5*	5.2*	1.3	4.0	4.1*	8.4*
Qphs.pseru-5B.2	*Xbarc1176-Xwmc363*	–	–	4.6*	11.1*	2.6*	6.1*	0.2	0.3	0.5	1.0	1.0	2.7	2.4*	5.1*
Total R^2					11.2		28.3		15.6		31.5		16.6		45.0
SD															
Qphs.pseru-4A.1	*Xbarc170-Xgwm397*	2.9*	8.0*	3.1*	7.0*	–	–	6.7*	17.5*	2.5*	7.7*	–	–	10.5*	24.1*
Qphs.pseru-4B.1	*Xwmc238-Xbarc20*	0.2	0.6	0.2	1.8	–	–	4.0*	9.0*	1.0	3.5	–	–	1.1	2.0
Qphs.pseru-5B.1	*Xbarc275-Xwmc75*	0.8	1.8	5.1*	12.1*	–	–	0.6	1.0	0.5	1.3	–	–	3.9*	7.7*
Qphs.pseru-5B.2	*Xbarc1176-Xwmc363*	3.0*	7.5*	1.5	3.7	–	–	0.2	0.6	0.3	0.7	–	–	3.4*	8.1*
Total R^2			14.1		26.2				23.9		10.2				40.8

PHS preharvest sprouting, *SD* seed dormancy, *JAAS* Jiangsu academy of agricultural sciences, *KSU* Kansas State University, *LOD* logarithm of odds

[a] Trait was not evaluated in this location

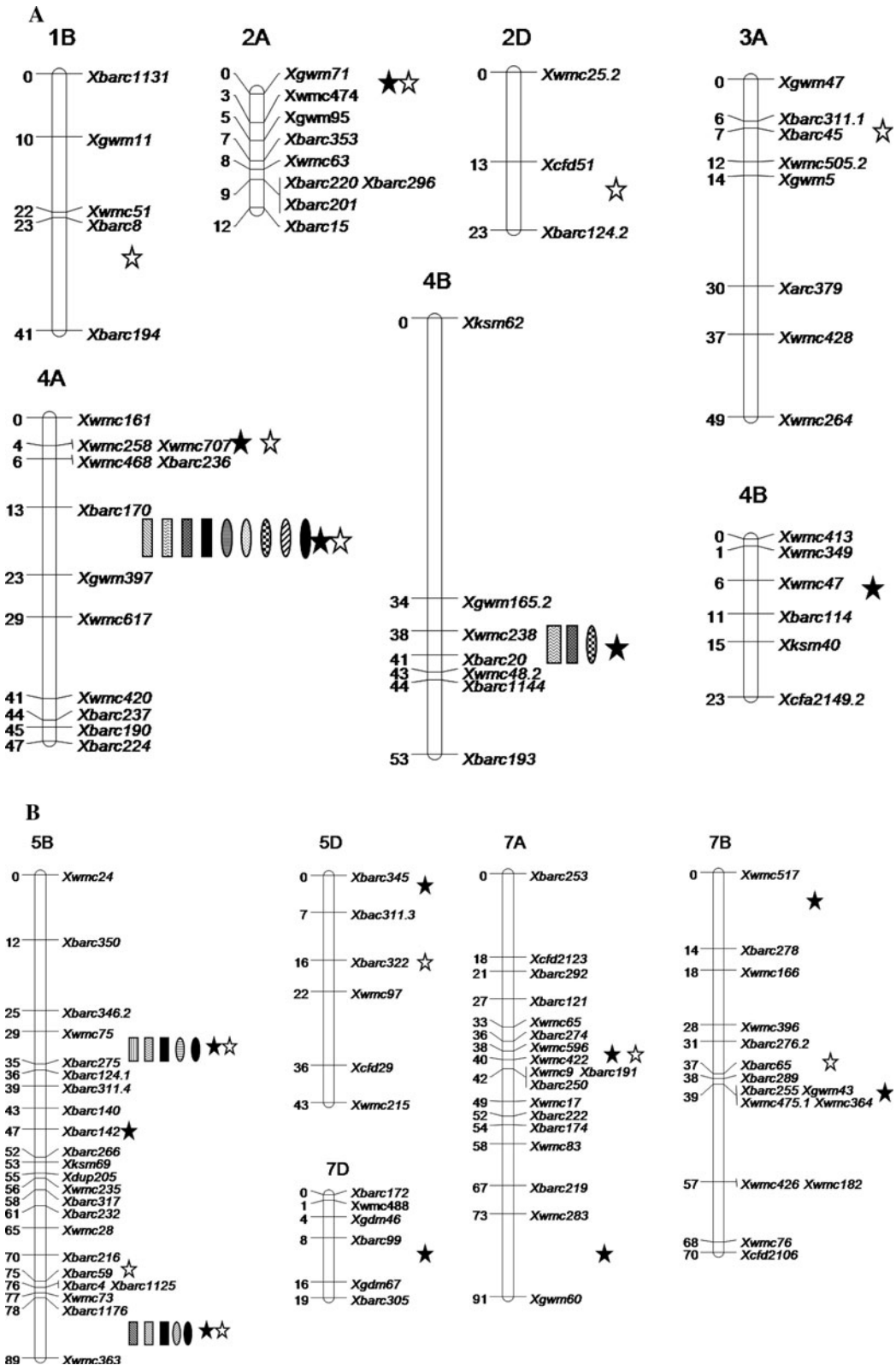
* Significant quantitative trait locus (QTL) with LOD value greater than the threshold (11.2)

Table S1), suggesting that favorable alleles governing the traits may originate from both parents.

Effects of genotype, environment, and G × E were highly significant for both PHS and SD ($p < 0.001$, ESM Table S2). Phenotypic correlations of PHS and SD between field and greenhouse experiments varied. The correlations for both PHS and SD were high between the two field experiments ($r = 0.57$ for PHS and $r = 0.38$ for SD) and among the three greenhouse experiments (r ranged from 0.35 to 0.53), but was low between the field and greenhouse experiments, with r values ranging from 0.16 to 0.28. Heritabilities were moderately high for PHS (0.47) and SD (0.56) (ESM Table S1).

QTLs for PHS resistance and SD

The initial linkage map consisting of 315 SSRs was used for the initial QTL scan in 96 RILs. All markers in the linkage groups that associated with PHS resistance and SD were further analyzed in 66 additional RILs from the same population. The final map was constructed with 162 RILs and 118 markers from 12 linkage groups, covered all QTL regions detected in the initial map, and was used for further QTL identification. CIM detected four significant QTLs for PHS resistance in five experiments, ranging from one to three QTLs in each individual experiment, with three of these QTLs being significant for the mean values across all experiments (Table 1). *Qphs.pseru-4A.1* accounted for 10.7–17.8% of the phenotypic variation in three KSU experiments (Table 1, Fig. 2) and for 25.3% of the phenotypic variation for mean PHS over all five experiments. Another QTL, *Qphs.pseru-5B.1*, was detected in one KSU and one JAAS experiment (Table 1, Fig. 2). This QTL accounted for 8.4% of the phenotypic variation for mean PHS over five experiments,


A
1B
0 Xbarc1131
10 Xgwm11
22 Xwmc51
23 Xbarc8
41 Xbarc194
2A
0 Xgwm71
3 Xwmc474
5 Xgwm95
7 Xbarc353
8 Xwmc63
9 Xbarc220 Xbarc296
Xbarc201
12 Xbarc15
2D
0 Xwmc25.2
13 Xcfd51
23 Xbarc124.2
3A
0 Xgwm47
6 Xbarc311.1
7 Xbarc45
12 Xwmc505.2
14 Xgwm5
30 Xarc379
37 Xwmc428
49 Xwmc264
4A
0 Xwmc161
4 Xwmc258 Xwmc707
6 Xwmc468 Xbarc236
13 Xbarc170
23 Xgwm397
29 Xwmc617
41 Xwmc420
44 Xbarc237
45 Xbarc190
47 Xbarc224
4B
0 Xksm62
34 Xgwm165.2
38 Xwmc238
41 Xbarc20
43 Xwmc48.2
44 Xbarc1144
53 Xbarc193
4B
0 Xwmc413
1 Xwmc349
6 Xwmc47
11 Xbarc114
15 Xksm40
23 Xcfa2149.2
B
5B
0 Xwmc24
12 Xbarc350
25 Xbarc346.2
29 Xwmc75
35 Xbarc275
36 Xbarc124.1
39 Xbarc311.4
43 Xbarc140
47 Xbarc142
52 Xbarc266
53 Xksm69
55 Xdup205
56 Xwmc235
58 Xbarc317
61 Xbarc232
65 Xwmc28
70 Xbarc216
75 Xbarc59
76 Xbarc4 Xbarc1125
77 Xwmc73
78 Xbarc1176
89 Xwmc363
5D
0 Xbarc345
7 Xbac311.3
16 Xbarc322
22 Xwmc97
36 Xcfd29
43 Xwmc215
7D
0 Xbarc172
1 Xwmc488
4 Xgdm46
8 Xbarc99
16 Xgdm67
19 Xbarc305
7A
0 Xbarc253
18 Xcfd2123
21 Xbarc292
27 Xbarc121
33 Xwmc65
36 Xbarc274
38 Xwmc596
40 Xwmc422
42 Xwmc9 Xbarc191
Xbarc250
49 Xwmc17
52 Xbarc222
54 Xbarc174
58 Xwmc83
67 Xbarc219
73 Xwmc283
91 Xgwm60
7B
0 Xwmc517
14 Xbarc278
18 Xwmc166
28 Xwmc396
31 Xbarc276.2
37 Xbarc65
38 Xbarc289
39 Xbarc255 Xgwm43
Xwmc475.1 Xwmc364
57 Xwmc426 Xwmc182
68 Xwmc76
70 Xcfd2106

◀**Fig. 2** Linkage map and distribution of quantitative trait loci (QTLs) with main effects and epistatic effects for preharvest sprouting (PHS) resistance and seed dormancy (SD) detected by a mixed model linear approach in the RIL6 population derived from Tutoumai A/'Siyang 936'. Only the linkage maps on which QTLs have been involved are presented. *Shaded rectangles* indicate QTLs for PHS resistance detected in 2005JAAS (*large hatching*), 2006JAAS (*vertical lines*), 2005KSU (*diagonal*), 2006KSU (*wavy*), 2007KSU (*small hatching*) experiments and the mean value of the five experiments (*filled square*). *Shaded ovals* indicate QTLs for seed dormancy (GI) detected in 2004JAAS (*horizontal*) 2005JAAS (*stippled*), 2005KSU (*bold hatching*), 2006 KSU (*diagonal*) experiments and the mean value of the four experiments (*solid circle*). *Filled star* QTLs involved only in epistatic interactions for PHS resistance, *open star* QTLs involved only in epistatic interactions for seed dormancy

ranging from 5.2 to 27% in each individual experiment. *Qphs.pseru-5B.2* was detected in two JAAS experiments and explained 5.1% of the phenotypic variation for mean PHS value. *Qphs.pseru-4B.1* was significant in two KSU experiments but not significant for the mean value across the five PHS experiments. All significant QTLs jointly explained 45% of the phenotypic variation in PHS, as measured by the mean value across the five PHS experiments, with the phenotypic variation in PHS ranging from 11.2 to 31.5% in individual experiments.

All four QTLs for PHS resistance were also significant for SD in four SD experiments, ranging from one to two significant QTLs in an individual experiment (Table 1). *Qphs.pseru-4A.1* explained 7.0–17.5% of the phenotypic variation in the four experiments and 24.1% of the phenotypic variation for the mean SD across four experiments (Fig. 2). Other QTLs were detected only in a single experiment, and two of these, *Qphs.pseru-5B.1* and *Qphs.pseru-5B.2*, were also detected for the mean across all experiments (Table 1). The four QTLs together explained 40.8% of the phenotypic variation for the mean SD across all experiments, with the phenotypic variation for mean SD ranging from 10.2 to 26.2% in individual experiments.

Additive QTLs and additive Q × E interactions

Three QTLs significant for the mean PHS value detected in CIM (Table 1) also showed a significant additive effect (*a*) (Table 2). However, expression of the *a* of these QTLs on PHS resistance was significantly affected by environment (Table 2). *Qphs.pseru-4A.1* and *Qphs.pseru-5B.*2 showed a significant additive × environment interaction (*ae*) on PHS resistance in two of the five experiments, and *Qphs.pseru-5B.*1 showed a significant *ae* on PHS in one experiment. Two of the three QTLs also showed significant *a* and *ae* on SD in one of the four SD experiments (Table 2).

Interactions between QTLs and epistatic QTL × environment

A total of 14 QTLs showed nine combinations of *aa* and/or *aae* on PHS resistance (Table 3, Fig. 2). These interactions involved three QTLs that showed a significant *a* and 11 epistatic QTLs that did not show any

Table 2 Estimated additive (*a*) and additive × environmental interactions (*ae*) of QTL for PHS resistance and SD detected by using the mixed linear model and the recombinant inbred line population derived from Tutoumai A/'Siyang 936'

QTL name	Flanking interval	LOD	*a*	*ae*1	*ae* 2	*ae* 3	*ae* 4	*ae* 5	*ae* 6
PHS									
Qphs. pseru-4A.1	*Xbarc170-Xgwm397*	18.6	−6.4*****	–[a]	ns	3.9**	ns	−2.2*	ns
Qphs. pseru-5B.1	*Xwmc75-Xbarc275*	12.7	−5.0*****	–	–ns	ns	2.7*	ns	ns
Qphs. pseru-5B.2	*Xbarc1176-Xwmc363*	5.6	−2.7*****	–	−3.2*	ns	ns	−4.1**	ns
SD									
Qphs. pseru-4A.1	*Xbarc170-Xgwm397*	14.6	−5.8*****	−2.1*	–ns	–	−ns	ns	–
Qphs. pseru-5B.1	*Xwmc75-Xbarc275*	7.3	−4.0*****	ns	−3.7**	–	ns	ns	–

a, Additive effect; *ae*1, *ae*2, *ae*3, *ae*4, and *ae*5,QTL × environment (E) interaction effects for environments 1 (Nanjing, 2004), 2 (Nanjing, 2005), 3 (Nanjing, 2006), 4 (Manhattan, 2005), 5 (Manhattan, 2006), and 6 (Manhattan, 2007), respectively

[a] Trait was not evaluated in this location

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$,**** $p < 0.0001$, ***** $p < 0.00001$; *NS* not significant

Table 3 Estimated "additive × additive" epistatic (*aa*) and "additive × additive" epistasis × environment interaction (*aae*) effects of QTLs detected by two-locus analyses with QTLMapper for PHS resistance and SD in the RIL6 population derived from Tutoumai A and 'Siyang 936' (Tutoumai A/'Siyang 936')

QTL*i*[a]	Flanking interval	QTL*j*[a]	Flanking interval	LOD	$a_i a_j$ effect	*aae*1	*aae* 2	*aae* 3	*aae* 4	*aae* 5	*aae* 6
PHS											
Qphs.pseru-2A	*Xgwm71-Xwmc474*	*Qphs.pseru-7A.1*	*Xwmc422-Xwmc9*	4.1	1.6**	–[b]	ns	ns	ns	−2.7*	4.5***
Qphs.pseru-4A.1	*Xbarc170-Xgwm397*	*Qphs.pseru-4A.2*	*Xwmc161-Xwmc258*	13.8	1.5**	–	ns	ns	ns	ns	ns
Qphs.pseru-4A.1	*Xbarc170-Xgwm397*	*Qphs.pseru-5B.1*	*Xbarc275-Xwmc75*	15.0	ns	–	ns	−2.3*	ns	ns	ns
Qphs.pseru-4A.1	*Xbarc170-Xgwm397*	*Qphs.pseru-7B.1*	*Xwmc517-Xbarc278*	9.1	1.2*	–	ns	ns	ns	ns	4.7***
Qphs.pseru-4B.2	*Xwmc47-Xbarc114*	*Qphs.pseru-7A.2*	*Xwmc283-Xgwm60*	4.5	ns	–	2.7*	2.6*	ns	ns	−5.3***
Qphs.pseru-5B.3	*Xbarc142-Xbarc140*	*Qphs.pseru-7B.2*	*Xbarc255-Xgwm43*	4.2	ns	–	−4.5***	ns	ns	4.3***	ns
Qphs.pseru-5B.1	*Xbarc275-Xwmc75*	*Qphs.pseru-5D.1*	*Xbarc345-Xbarc311.3*	8.4	1.3*	–	ns	ns	2.1*	ns	ns
Qphs.pseru-5D.1	*Xbarc345-Xbarc311.3*	*Qphs.pseru-7D.3*	*Xbarc99-Xgdm67*	4.1	1.8**	–	ns	ns	ns	ns	4.0***
Qphs.pseru-4B.1	*Xwmc238-Xbarc20*	*Qphs.pseru-5B.2*	*Xbarc1176-Xwmc363*	5.6	ns	–	ns	ns	ns	ns	2.3**
SD											
Qphs-pseru-1B	*Xbarc8-Xbarc194*	*Qphs-pseru-7A.1*	*Xwmc422-Xwmc9*	5.3	−1.3*	2.7*	ns	–	ns	ns	–
Qphs-pseru-2A	*Xgwm71-Xwmc474*	*Qphs-pseru-4A.1*	*Xbar170-Xgwm397*	19.5	1.7*	ns	3.0**	–	ns	ns	–
Qphs-pseru-4A.1	*Xbarc170-Xgwm397*	*Qphs-pseru-4A.2*	*Xwmc707-Xwmc258*	17.1	1.2*	ns	ns	–	ns	ns	–
Qphs-pseru-5B.2	*Xwmc1176-Xwmc363*	*Qphs-pseru-5D.2*	*Xbarc322-Xwmc97*	10.5	2.0**	ns	2.7**	–	ns	ns	–
Qphs-pseru-5B.1	*Xbarc275-Xwmc75*	*Qphs-pseru-4A.1*	*Xbarc170-Xgwm397*	8.8	ns	4.5***	ns	–	−2.7*	ns	–
Qphs-pseru-5B.4	*Xbarc216-Xbarc59*	*Qphs-pseru-7B.3*	*Xbarc276.2-Xbarc65*	6.3	−2.8***	ns	ns	–	2.7*	−5.3***	–
Qphs-pseru-2D	*Xcfd51-Xbarc124.2*	*Qphs-pseru-3A*	*Xbarc311-Xbarc45*	6.3	−1.2*	ns	ns	–	ns	ns	–

[a] *QTLi* and *QTLj*, pair of QTLs showing interaction, $a_i a_j$ effect, the effect of additive X additive interaction across environments (e1-e6) for environments 1 (Nanjing, 2004), 2 (Nanjing, 2005), 3 (Nanjing, 2006), 4 (Manhattan 2005), 5 (Manhattan, 2006), and 6 (Manhattan, 2007); A positive value means parental type effect is greater than the recombinant effect or vice versa; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, *NS* not significant

[b] Trait was not evaluated in this location

significant *a* (Tables 2, 3). Among the nine pairs of digenic interactions for PHS resistance, epistatic QTL pairs *Qphs.pseru-2A*/*Qphs.pseru-7A.1*, *Qphs.pseru-4A.1*/*Qphs.pseru-7B.1*, *Qphs.pseru-5B.1*/*Qphs.pseru-5D.1*, and *Qphs.pseru-5D.1*/*Qphs.pseru-7D.3* showed both *aa* and *aae* in one to two KSU experiments. The QTL pair *Qphs.pseru-4A.1*/*Qphs.pseru-4A.2* showed only an *aa*. Four other QTL pairs did not show an *aa*, but had an *aae* in one to three experiments. For SD, seven pairs of digenic interactions were detected for 12 QTLs (Table 3, Fig. 2), including the two QTLs with a significant *a* and ten QTLs without a significant *a* (Tables 2, 3). Among these, six pairs showed *aa*, and five pairs also showed *aae* in at least one experiment (Table 3). One pair showed only *aae* without *aa*.

Discussion

Relationship between PHS resistance and SD in wheat

Resistance to PHS in wheat is a complex trait controlled by several QTLs in addition to some nongenetic factors (Gatford et al. 2002; Mares and Mrva 2001). The sprouting index (SI), a visual estimation of the germination rate of intact spikes on a 1–6 or 1–10 scale, has been used as a standard method for evaluating wheat PHS resistance in many studies (Anderson et al. 1993; Kulwal et al. 2004, 2005). Chen et al. (2008) recently used the percentage of sprouted seeds in a spike to reflect PHS. Similarly, Imtiaz et al. (2008) used visibly sprouted seeds (VSS) to measure germination rate by determining the percentage of germinated seeds in more than 200 total seeds threshed from several treated spikes. After comparing SI with VSS, Imtiaz et al. (2008) concluded that VSS gave a more accurate PHS rating than SI. In this study, we used PVSK in a spike to reflect overall PHS resistance for both greenhouse- and field-grown plants. The phenotypic data should be as accurate as VSS.

However, testing intact spikes may require a specific facility that can control temperature and moisture. The results of several studies indicate that SD is the major genetic component of PHS resistance and that conditions for testing SD are more flexible. Thus, SD has been used to directly measure overall PHS resistance in many QTL mapping studies (Kato et al. 2001; Osa et al. 2003; Mares et al. 2005) and proposed as a reliable alternative in the absence of a facility for assaying intact spikes (Imtiaz et al. 2008). To date, most QTL mapping studies have used germination data from either intact spikes (PHS) or threshed seeds (SD), but not both, for PHS phenotyping. It is therefore difficult to determine the contribution of SD to overall PHS resistance in these studies. Our previous study using both GI of threshed seeds (SD) and intact spikes (PHS) from two greenhouse experiments identified a QTL on chromosome 4A that has a significant effect on both SD and PHS resistance (Chen et al. 2008). Using similar methods, Imtiaz et al. (2008) also found the 4A QTL from a red synthetic wheat line that has an effect on both long SD and PHS resistance. In this study, we evaluated the mapping population for both intact spike PHS and SD of threshed seeds harvested from both field (JAAS) and greenhouse (KSU) experiments and found that PHS resistance and long SD were mainly controlled by the same four QTLs. We therefore conclude that SD is the major contributor to PHS resistance in this population. Other factors may also contribute to PHS resistance, but their effect is small and may be more vulnerable to environmental variations.

QTLs for SD and PHS resistance in wheat

In this study, four QTLs were detected for SD (Table 1). Among these, *Qphs-pseru-4A.1* showed a significant effect on SD in both field and greenhouse experiments. This QTL has also been reported to affect SD in several sources of red and white wheat cultivars (Kato et al. 2001; Mares and Mrva 2001; Mares et al. 2005; Torada et al. 2005; Chen et al. 2008; Imtiaz et al. 2008; Ogbonnaya et al. 2008). As such, it can be considered to be a stable major QTL for long SD across different environments.

The other three QTLs for SD were likely minor QTLs, and they mainly showed a minor effect on SD in one of the four SD experiments. Two QTLs on 5B, *Qphs.pseru-5B.1* and *Qphs.pseru-5B.2*, showed a minor effect on mean SD across four experiments; thus, these QTLs may be less vulnerable to environmental variation than the other QTL. We could not determine whether the map position of the 4B QTL is the same as that reported by Kato et al. (2001)

because common markers were not identified between the two studies. However, this is the first report of this QTL from white wheat. Therefore, SD in Tutoumai A is likely to be controlled by a major QTL and several minor QTLs. The same four QTLs for long SD also showed significant effects on PHS resistance in at least two experiments (Table 1). With respect to SD, *Qphs.pseru-4A.1* showed the largest effect on overall PHS resistance, indicating that PHS resistance of *Qphs.pseru-4A.1* was mainly due to long SD. Although the QTL for PHS resistance was not significant in the two field experiments from JAAS, China, the QTL for SD from both JAAS experiments was highly significant, and the LOD value of the QTL in the 2005 JAAS PHS experiment almost reached a significant level (Table 1). The mean across all five experiments indicates the QTL from the two JAAS experiments also contributed to the overall PHS resistance QTL because the coefficient of determination for the mean of all five experiments, including the two JAAS experiments, was much higher than that from each individual experiment. The discrepancy between PHS and SD in the JAAS experiments may be because the SD and PHS evaluations were conducted in different years and environments or because environmental conditions at JAAS were better for SD evaluation than for PHS phenotyping. The results of this study are similar to those reported elsewhere, namely, the QTL on 4A from several sources has been found to have a major effect on PHS resistance (Kottearachchi et al. 2006), SD (Kato et al. 2001; Mares et al. 2005), or both (Chen et al. 2008; Imtiaz et al. 2008; Ogbonnaya et al. 2008). These results indicate that *Qphs.pseru-4A.1* is present in several different sources, including several white wheat cultivars of different origins. As such, it can be widely used to breed PHS-resistant white wheat. In this study, we were unable to detect the second QTL on 4A reported by Ogbonnaya et al. (2008) and Imtiaz et al. (2008). This discrepancy may be due to different sources of resistance being used in the two studies or to a lack of polymorphic markers in the QTL region to separate the QTL into two individual QTLs in the current study, although the newly mapped markers in the QTL region in other studies were also used in this study (Ogbonnaya et al. 2008; Zhang et al. 2008).

Another QTL, *Qphs.pseru-5B.1*, had significant effect on mean PHS and mean SD across all experiments, although it was only significant for PHS resistance in two of the five PHS experiments and for long SD in only one of four experiments. This may be because SD and PHS were evaluated in different environments or because factors other than SD may also contribute to PHS resistance (Gale 1989; Gatford et al. 2002; King and Richards 1984). Two additional SD QTLs were also significant in two PHS experiments (Table 1). Therefore, we conclude that PHS resistance of these QTLs was mainly due to extended SD. However, these QTLs appeared to be unstable and expressed only minor effects on PHS resistance in certain environments.

Two QTLs on 5B were detected for long SD and PHS resistance in this study. Groos et al. (2002) detected a QTL for PHS resistance on 5B in red wheat, but we were unable to determine whether this is the same as the one detected on 5B in this study because the markers linked with the QTL that Groos et al. (2002) detected were not mapped in our study. Tan et al. (2006) reported two QTLs on the proximal and distal region on 5BL in white wheat. For the proximal QTL (*Qphs.pseru-5B.1*), the same marker was not available in both studies [Tan et al. (2006) and ours] and, therefore, we were unable to determine whether they were the same QTL. For the QTL on the distal region (*Qphs.pseru-5B.2*), one marker *Xbarc59*, which was located on the proximal side near the QTL, was mapped in both studies; therefore, they were probably the same QTL. As these two QTLs were detected in white wheat in both studies, they were probable stable QTLs for PHS resistance and long SD although they were found to have a minor effect in the studies. The results also indicate that *Qphs.pseru-5B.1* and *Qphs.pseru-5B.2* are present in white wheat cultivars of different origins. Two QTLs for PHS resistance and SD were also mapped on 5HL in barley (Ullrich et al. 2008; Prada et al. 2004) which probably showed homologous loci for PHS resistance and SD in both wheat and barley group 5 chromosomes.

In addition to the major QTL located on 4A for both PHS resistance and long SD detected in the former study, three other minor QTLs were detected for both traits. Our identification of these minor QTLs provides breeders with more options in breeding for PHS resistance. Although these minor QTLs explained a small part of the phenotypic variation in each experiment in our study, they were able to

provide further strong justification to the unaccounted proportion of phenotypic variation in other experiments or different populations. These minor QTLs can be pyramided together to enhance the PHS resistance in breeding programs.

Epistatic interaction between QTLs

Major genetic components of a quantitative trait in a recombinant population include *a*, *aa*, *ae,* and *aae*. Three types of epistatic interactions were characterized in rice: interactions between two additive QTLs (Type I), between an additive QTL and a 'background' nonadditive locus (Type II), and between two epistatic loci (Type III). Type I is considered to be the major type of epistasis (Li et al. 2001; Luo et al. 2001). To date, two studies have reported on the dissection of genetic components for PHS in red wheat (Kulwal et al. 2004; Imtiaz et al. 2008) but not in white wheat. Three types of epistasis for PHS resistance were reported in one study (Kulwal et al. 2004) and two types (Type I and II) in another (Imtiaz et al. 2008). In the current study, we detected all three types of interactions for both PHS resistance and SD, which indicate that all QTLs that have a significant *a* on PHS and that SD also shows digenic interaction with some other loci with or without a significant *a* (Table 3). Additional QTLs for PHS resistance and ten QTLs for SD had significant *aa*, and they alone did not show a significant main effect. These results suggest that genetic control of SD and PHS resistance in white wheat is determined not only by additive QTL, but also by epistatic interaction among additive QTLs and epistatic QTLs.

QTL × environment interactions

In addition to the genetic effect of SD or PHS, environmental factors, such as light, temperature, and moisture during and after seed development, may also be important to PHS resistance in wheat (Alonso-Blanco et al. 2003). In our study, G × E interactions made a significant contribution to phenotypic variation in SD and PHS resistance among the RILs (ESM Table S2) and between the two parents (Tutoumai A and 'Siyang 936'), although 'Siyang 936' consistently showed higher PHS and SD ratings than Tutoumai A in all experiments. A significant G × E interaction was also reflected by the medium to low heritability of both traits (ESM Table S1) and a low correlation between Nanjing field and Manhattan greenhouse experiments. Thus, for QTL mapping, multiple tests over different years and environments may provide a more accurate estimation of QTL effects and locations, especially for QTLs with minor effects. In this study, data were collected from five PHS experiments and four SD experiments in two locations to minimize environmental effects, and four common QTLs were detected for both traits. Some QTLs were expressed only in certain environments (e.g., *Qphs.pseru-4B.1* was detected only in greenhouse experiments and *Qphs.pseru-5B.2* only in field experiments). However, most of these QTLs were repeatable QTLs for PHS resistance and SD under different environments.

To date, only two studies have reported Q × E interaction for PHS in wheat; most QTLs with a significant *a* did not have a significant *ae,* and less than half of the epistatis QTLs (*aa*) showed a significant *aae* (Kulwal et al. 2004; Imtiaz et al. 2008). In our study, all additive QTLs (*a*) for PHS resistance and SD showed *ae* interactions in one or two PHS experiments and one SD experiment (Table 2), and eight of the nine epistasis loci (*aa*) for PHS resistance and five of the seven epistasis loci for SD showed a significant *aae* (Table 3). More QTLs showed Q × E interaction in our study than has been reported in other studies (Imtiaz et al. 2008; Kulwal et al. 2004), perhaps because different populations and environments were used for QTL analysis and PHS evaluation, respectively. High Q × E interaction suggests that direct phenotyping of PHS and SD may be difficult for breeders to apply to accurately select PHS-resistant genotypes with conventional selection techniques. This makes PHS resistance a good candidate for marker-assisted selection. Epistatic QTLs may contribute significantly to overall PHS resistance of a genotype and should be given considerable attention when breeders make selections designed to improve PHS resistance in wheat.

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References

Alonso-Blanco C, Bentsink L, Hanharta CJ, Vries HB, Koornneefa M (2003) Analysis of natural allelic variation at seed dormancy loci of *Arabidopsis thaliana*. Genetics 164:711–729

Amano Y, Torada A (2002) Breeding of white-grained wheats for Japan. Euphytica 126:83–88

Anderson JA, Sorrells ME, Tanksley SD (1993) RFLP analysis of genomic regions associated with resistance to preharvest sprouting in wheat. Crop Sci 33:453–459

Chen CX, Cai SB, Bai GH (2008) A major QTL controlling seed dormancy and pre-harvest sprouting resistance on chromosome 4A in a Chinese wheat landrace. Mol Breed 21:351–358

Debeaujon IKM, Leon-Klooterziel KM, Koornneef M (2000) Influence of the testa on seed dormancy, germination, and longevity in *Arabidopsis*. Plant Physiol 122:403–413

Derera NF, Bhatt GM (1980) Germination inhibition of the bracts in relation to preharvest sprouting tolerance in wheat. Cereal Res Commun 8:199–201

Doerge RW, Churchill GA (1996) Permutation tests for multiple loci affecting a quantitative character. Genetics 142:285–294

Eujayl I, Sorrells ME, Baum M, Wolters P, Powell W (2002) Isolation of EST-derived microsatellite markers for genotyping the A and B genomes of wheat. Theor Appl Genet 104:399–407

Flintham JE, Adlam R, Gale MD (1999) Seed coat and embryo dormancy in wheat. In: Proceedings of 8th international symposium pre-harvest sprouting in cereals. Detmold, pp 67–76

Flintham J, Adlam R, Bassoi M, Holdsworth M, Gale MD (2002) Mapping genes for resistance to sprouting damage in wheat. Euphytica 126:39–45

Gale MD (1989) The genetics of preharvest sprouting in cereals, particularly in wheat. In: Derera NF (ed) Preharvest field sprouting in cereals. CRC Press, Boca Raton, pp 85–110

Gatford KT, Eastwood RF, Halloran GM (2002) Germination inhibitors in bracts surrounding the grain of *Triticum tauschii*. Funct Plant Biol 29:881–890

Groos C, Gay G, Perretant MR, Gervais L, Bernard M, Dedryver F et al (2002) Study of the relationship between pre-harvest sprouting and grain color by quantitative trait loci analysis in a whitexred grain bread-wheat cross. Theor Appl Genet 104:39–47

Guyomarc'h H, Sourdille P, Edwards KJ, Bernard M (2002) Studies of the transferability of microsatellite derived from *Triticum taushchii* to hexaploid wheat and to diploid related species using amplification, hybridization and sequence comparisons. Theor Appl Genet 105:736–744

Imtiaz M, Ogbonnaya FC, Oman J, Ginkel MV (2008) Characterization of quantitative trait loci controlling genetic variation for preharvest sprouting in synthetic backcross-derived wheat lines. Genetics 178:1725–1736

Kato K, Nakamura W, Tabiki T, Miura H, Sawada S (2001) Detection of loci controlling seed dormancy on group 4 chromosomes of wheat and comparative mapping with rice and barley genomes. Theor Appl Genet 102:980–985

King RW, Richards RA (1984) Water-uptake in relation to preharvest sprouting damage in wheat—ear characteristics. Aust J Agric Res 35:327–336

Kosambi DD (1944) The estimation of map distances from recombination values. Ann Eugen 12:172–175

Kottearachchi NS, Uchino N, Kato K, Miura H (2006) Increased grain dormancy in white-grained wheat by introgression of preharvest sprouting tolerance QTLs. Euphytica 152:421–428

Kulwal PL, Singh R, Balyan HS, Gupta PK (2004) Genetic basis of pre-harvest sprouting tolerance using single-locus and two-locus QTL analyses in bread wheat. Funct Integr Genomics 4:94–101

Kulwal PL, Kumar N, Gaur A, Khurana P, Khurana JP, Tyagi AK et al (2005) Mapping of a major QTL for pre-harvest sprouting tolerance on chromosome 3A in bread wheat. Theor Appl Genet 111:1052–1059

Kuraparthy V, Sood S, Gill BS (2008) Targeted genomic mapping of a red seed color gene (R-A1) in wheat. Crop Sci 48:S37–S48

Li ZK, Luo LJ, Mei HW, Wang DL, Shu QY, Tabien R et al (2001) Overdominant epistatic loci are the primary genetic basis of inbreeding depression and heterosis in rice. I. Biomass and grain yield. Genetics 158:1737–1753

Liu SB, Cai SB, Graybosch R, Chen CX, Bai GH (2008) Quantitative trait loci for resistance to pre-harvest sprouting in US hard white winter wheat Rio Blanco. Theor Appl Genet 117:691–699

Luo LJ, Li ZK, Mei HW, Shu QY, Tabien R, Zhong DB et al (2001) Overdominant epistatic loci are the primary genetic basis of inbreeding depression and heterosis in rice. II. Grain yield components. Genetics 158:1755–1771

Mansour K (1993) Sprout damage in wheat and its effect on wheat flour products. In: Walker-Simmons MK, Ried JL (eds) Pre-harvest sprouting in cereals, 1992. American Association of Cereal Chemistry Press, St. Paul, MN, pp 8–9

Mares DJ (1996) Dormancy in white wheat: mechanism and location of genes. In: Noda K, Mares DJ (eds) Preharvest sprouting in cereals, 1995. Centre for Academic Societies, Osaka, pp 179–184

Mares DJ, Mrva K (2001) Mapping quantitative trait loci associated with variation in grain dormancy in Australian wheat. Aust J Agric Res 52:1257–1265

Mares DJ, Mrva K, Cheong J, Williams K, Watson B, Storlie E et al (2005) A QTL located on chromosome 4A associated with dormancy in white- and red-grained wheats of diverse origin. Theor Appl Genet 111:1357–1364

McCaig TN, Depauw RM (1992) Breeding for preharvest sprouting tolerance in white-seed-coat spring wheat. Crop Sci 32:19–23

Mori M, Uchino N, Chono M, Kato K, Miura H (2005) Mapping QTLs for grain dormancy on wheat chromosome 3A and the group 4 chromosomes, and their combined effect. Theor Appl Genet 110:1315–1323

Morris CF, Paulsen GM (1989) Registration of 5 preharvest sprouting-resistant hard white winter wheat germplasm lines. Crop Sci 29:246–247

Nelson JC, Deynze AEV, Autrique E, Sorrells ME, Yun HL, Negre S et al (1995) Molecular mapping of wheat: homoeologous group 3. Genome 38:525–533

Ogbonnaya FC, Imtiaz M, Ye G, Hearnden PR, Hernandez E, Eastwood RF et al (2008) Genetic and QTL analyses of seed dormancy and preharvest sprouting resistance in the wheat germplasm CN10955. Theor Appl Genet 116: 891–902

Osa M, Kato K, Mori M, Shindo C, Torada A, Miura H et al (2003) Mapping QTLs for seed dormancy and the Vp1 homologue on chromosome 3A in wheat. Theor Appl Genet 106:1491–1496

Paterson AH, Sorrells ME (1990) Inheritance of grain dormancy in white-kernelled wheat. Crop Sci 30:25–30

Pestsova EG, Ganal MW, Röder MS (2000) Isolation and mapping of microsatellite markers specific for the D-genome of bread wheat. Genome 43:689–697

Prada D, Ullrich SE, Molina-Cano JL, Cistu L, Clancy JA, Romagosa I (2004) Genetic control of dormancy in a Triumph/Morex cross in barley. Theor Appl Genet 109:62–70

Röder MS, Korzun V, Wendehake K, Plaschke J, Tixier MH, Leroy P et al (1998) A microsatellite map of wheat. Genetics 149:2007–2023

Roy JK, Prasad M, Varshney RK, Balyan HS, Blake TK (1999) Identification of a microsatellite on chromosomes 6B and a STS on 7D of bread wheat showing an association with preharvest sprouting tolerance. Theor Appl Genet 99: 336–340

Seshu DV, Sorrells ME (1986) Genetic studies on seed dormancy in rice. In: RI IR (ed) Rice genetics. IRRI, Manila, pp 369–382

Somers DJ, Isaac P, Edwards K (2004) A high-density wheat microsatellite consensus map for bread wheat (*Triticum aestivum* L.). Theor Appl Genet 109:1105–1114

Song QJ, Shi JR, Singh S, Fickus EW, Costa JM, Lewis J et al (2005) Development and mapping of microsatellite (SSR) markers in wheat. Theor Appl Genet 110:550–560

Sourdille P, Cadalen T, Guyomarc'h H, Snape JW, Perretant MR, Charmet G et al (2003) An update of the Courtot/Chinese Spring intervarietal molecular marker linkage map for the QTL detection of agronomic traits in wheat. Theor Appl Genet 106:530–538

Steel RGD, Torrie JH (1980) Principles and procedures of statistics. McGraw-Hill, New York, pp 172–194

Tan MK, Sharp PJ, Lu MQ, Howes N (2006) Genetics of grain dormancy in a white wheat. Aust J Agric Res 57: 1157–1165

Toojinda T, Baird E, Booth A, Broers L, Hayes P (1998) Introgression of quantitative trait loci (QTLs) determining stripe rust resistance in barley: an example of marker-assisted line development. Theor Appl Genet 96:123–131

Torada A, Ikeguchi S, Koike M (2005) Mapping and validation of PCR-based markers associated with a major QTL for seed dormancy in wheat. Euphytica 143:251–255

Ullrich SE, Clancy JA, del Blanco IA, Lee H, Jitkov VA, Han F, Kleinihiko A, Matsui K (2008) Genetic analysis of preharvest sprouting in a six-row barley cross. Mol Breed 21:249–259

Van Ooijen JW, Voorrips RE (2001) JoinMap version 3.0: Software for the calculation of genetic linkage maps. Plant Research International, Wageningen

Wang DL, Zhu J, Li ZK, Paterson A (1999) Mapping QTL with epistatic effects and QTL × environment interaction by mixed linear model approaches. Thoer Appl Genet 99:1255–1264

Wang S, Basten CJ, Zeng ZB (2005) Windows QTL Cartographer 2.5. Department of statistics, Department of Statistics, North Carolina State University, Raleigh. Available at: http://statgen.ncsu.edu/qtlcart/WQTLCart.htm

Wu JM, Carver BF (1999) Sprout damage and preharvest sprout resistance in hard white winter wheat. Crop Sci 39:441–447

Yang DL, Jing RL, Chang XP, Li W (2007) Identification of quantitative trait loci and environmental interactions for accumulation and remobilization of water-soluble carbohydrates in wheat (*Triticum aestivum* L.) stems. Genetics 176:571–584

Zhang XQ, Li CD, Tay A, Lance R, Mares D, Cheong J, Cakir M, Ma J, Appels R (2008) A new PCR-based marker on chromosome 4AL for resistance to pre-harvest sprouting in wheat (*Triticum aestivum* L.). Moleculer Breed 22: 227–236