RESEARCH

Identification of Chromosome Locations of Genes Affecting Preharvest Sprouting and Seed Dormancy Using Chromosome Substitution Lines in Tetraploid Wheat (*Triticum turgidum* L.)

Shiaoman Chao,* Steven S. Xu, Elias M. Elias, Justin D. Faris, and Mark E. Sorrells

ABSTRACT

Seed dormancy, the main factor contributing to preharvest sprouting (PHS) resistance, is a complex trait and is strongly influenced by environmental growth conditions. In this study, three sets of single chromosome substitution lines, including 36 genotypes, in a durum wheat (*Triticum turgidum* L. ssp. *durum* (Desf.) Husn.) background with donor chromosomes originating from three wild emmer (*T. turgidum* L. ssp. *dicoccoides* (Körn. ex Asch. and Graebn.) Thell.) accessions were grown in nine field environments and evaluated for seed dormancy and PHS resistance. The substitution lines involving chromosome 3A were among the most dormant genotypes. Germination tests indicated that five chromosomes contained genes influencing seed dormancy at a level comparable to 3A. Results from PHS tests showed that PHS was affected by at least eight *T. dicoccoides* chromosomes including 3A. The chromosomes harboring genes for seed dormancy did not fully correspond with those for PHS resistance. The weak correlations between PHS and dormancy observed in this study indicate that different genes are affecting these traits and they may be differentially influenced by the environment. Nonetheless, our results revealed that genes present on five chromosomes, 2A, 2B, 3A, 4A, and 7B, were found to affect both PHS resistance and seed dormancy. These genotypes thus provide useful resources for further studies on genetic interactions that contribute to the overall phenotypic variation and on genetic dissection of quantitative trait loci underlying PHS resistance.

The impact of preharvest sprouting (PHS) on reducing end-use grain quality, which leads to further economic losses, has been widely recognized by wheat (*Triticum* species) producers in the United States and throughout the world. Seed dormancy, a main factor contributing to PHS tolerance, is an adaptive trait largely affected by environmental conditions, such as temperature and moisture, during seed development and after ripening (Hagemann and Ciha, 1987; Mares, 1993; Biddulph et al., 2005). Genetic resources with seed dormancy and tolerance to PHS have been characterized and identified among both cultivated germplasm and wild relatives of *Triticum* species (Mares, 1987; Clarke et al., 1994; Hucl et al., 1996). In common wheat (*T. aestivum* L.), it has been well documented that the red kernel color has a complete association with seed dormancy (Gfeller and Svejda, 1960; Flintham, 2000; Warner et al., 2000). The red color (R) genes located on the long arms of homoeologous group 3 chromosomes in common wheat (Flintham and Gale, 1996) were found closely linked to seed dormancy in tetraploid emmer wheat (*T. turgidum* L.).
L. ssp. dicoccoides (Körn. ex Asch. and Graebn.) Thell.) as well (Watanabe and Ikebata, 2000). Several studies have indicated a considerable variation in the levels of PHS resistance present among the red wheat cultivars, and the number of R genes appears to be independent of the extent of seed dormancy (Mares and Ellison, 1990; McCaig and DePauw, 1992; Wu and Carver, 1999). Preharvest sprouting resistance was also identified in white common wheat and amber-seeded durum wheat [T. turgidum L. ssp. durum (Desf.) Husn.] cultivars that expressed comparable levels of resistance as found in the red wheat checks (Mares and Ellison, 1990; McCaig and DePauw, 1992; Clarke et al., 1994). These earlier reports showed the large genetic variation underlying PHS resistance and the strong influence from environmental factors.

The use of DNA markers has allowed the genetic basis of PHS and seed dormancy to be investigated in greater detail in wheat. Depending on the crosses used in genetic mapping studies, more than 20 quantitative trait loci (QTLs) located on 18 wheat chromosomes have been reported to be significantly associated with PHS resistance and high levels of seed dormancy and also to have strong genotype × environment interactions (Flintham et al., 2002). However, a major QTL located on the long arm of chromosome 4A has been detected in several studies using both red and white wheat crosses and crosses involving synthetic wheats (Anderson et al., 1993; Kato et al., 2001; Flintham et al., 2002; Lohwasser et al., 2005; Mares et al., 2005; Torada et al., 2005; Chen et al., 2008; Imtiaz et al., 2008), indicating that this QTL may be one of the key genes controlling PHS resistance and seed dormancy. When the simple sequence repeat marker flanking the chromosome 4AL region containing the putative PHS resistance QTL were used to survey the haplotype diversity present among a set of 28 wheat cultivars and landraces originating from different countries, three main simple sequence repeat marker haplotypes were detected with each representing a group of resistant genotypes that shared a similar resistance source (Ogbonnaya et al., 2007). In their study, the two resistant durum wheat lines were found to share the same marker haplotype as the resistant synthetic wheats at the 4AL region. Compared to common wheat, genetic mapping studies of PHS resistance in durum wheat are rather limited. A significant QTL located on chromosome 1A, identified by Knox et al. (2005), may be similar to the QTL previously reported in common wheat by Anderson et al. (1993).

Aneuploid stocks involving alien or intraspecific chromosome manipulation have been exploited extensively for the identification of genes of agronomic importance in hexaploid wheat (Law et al., 1981). Single chromosome substitution lines allow the traits affected by genes on single chromosomes to be studied in a homogeneous background. Consequently they are useful for dissecting the inheritance of quantitative traits with low heritability (Nadeau et al., 2000). Various aneuploid stocks have also been developed in tetraploid wheat by transferring chromosomes from different durum wheat varieties and wild emmer accessions into the background of the durum wheat cultivar Langdon (Joppa 1993). Wild emmer is known to be a rich source of genetic variation including novel genes for disease resistance and end-use grain quality that can be used for genetic improvement of cultivated wheat. Three sets of single chromosome substitution lines involving wild emmer and Langdon were created (Joppa and Cantrell, 1990; Xu et al., 2004) and subsequently used for evaluating and characterizing genes controlling various agronomic traits, such as grain yield (Cantrell and Joppa 1991; Gonzales-Hernandez et al., 2004), end-use quality traits (Joppa et al., 1991; Steiger et al., 1996; Klindworth et al., 2009), seed dormancy (Watanabe and Ikebata 2000), and disease resistance against Fusarium head blight (caused by Fusarium graminearum Schwabe) (Stack et al., 2002; Kumar et al., 2007) and Stagonospora nodorum blotch [caused by Stagonospora nodorum (Berk.) Castell and Germano] (Haen et al., 2004; Lu et al., 2006; Faris and Friesen, 2009). The use of these stocks for identifying and locating high grain protein content genes on the short arm of 6B chromosome (Joppa and Cantrell 1990; Joppa et al., 1997) led to the cloning of the major gene encoding grain protein content of wild emmer wheat origin (Uauy et al., 2006). In this study, these three sets of substitution lines were grown under field conditions and evaluated for PHS and seed dormancy to identify chromosome locations for genes controlling these traits. These results will facilitate future studies on genetic mapping and dissection of QTLs responsible for PHS resistance and seed dormancy in wheat.

MATERIALS AND METHODS

Plant Materials

Thirty-eight single chromosome substitution lines (Table 1) were developed that involved three wild emmer wheat, T. turgidum L. ssp. dicoccoides, accessions as the chromosome donor and the durum wheat (T. turgidum L. ssp. durum) cultivar Langdon (LDN) as the recipient. The three T. turgidum L. ssp. dicoccoides accessions used were Israel A (ISA), PI 481521 (521), and PI 478742 (742). The detailed methods for producing these lines have been described previously (Joppa and Cantrell, 1990; Xu et al., 2004). All but two lines, LDN(521-5A) and LDN(742-5A), which have winter growth habit, were included in this study (Table 1).

Field Experiments

Due to seed shattering before harvest, late maturity for ISA, and winter growth habit for 521 and 742, three wild emmer chromosome donors along with two winter lines (521-5A and 742-5A) could not be accurately evaluated in our field conditions. Altogether, 36 substitution lines and LDN were grown...
Seed Germination and PHS Test

Seed germination tests were conducted using materials collected in five environments, including 2005 Fargo, 2005 Prosper, 2006 Fargo, 2006 Prosper, and 2008 Ithaca. Germination tests were performed by placing hand-threshed and visibly healthy seed treated with thiram [bis(dimethylthio-carbamoyl) disulfide] on two sheets of Whatman no. 1 filter paper wetted with 5 mL of distilled water in 9-cm-diameter petri dishes. Two replicates of approximately 50 seeds for each genotype were incubated in the dark at 20°C with 100% relative humidity. Germinated seeds with visible shoot and root radicals were counted and removed daily for 7 d. Seeds not germinated after 7 d were transferred to 4°C for three additional days to assess seed viability. Both percent germination rate (GR) and germination index (GI) (Walker-Simmons 1987) were calculated and used to evaluate the degree of dormancy.

For the PHS test, five spikes from each line were harvested at physiological maturity, as evidenced by the loss of green color from the glumes, and dried indoors for 5 d at ambient humidity and temperature. Due to slight differences in maturity, spikes were not always harvested on a single day. When the harvest was split among different days the harvest date for the line was recorded and taken into account by standardizing the mean across harvest dates. After drying for 5 d, PHS phenotyping was conducted as described by Anderson et al. (1993).

Kernel color was determined using a NaOH treatment (Dowell 1997). Three replicates of five seeds were soaked in 5% NaOH for about 12 h and visually compared to controls at 30 min, 2 h, and 12 h.

Statistical Analysis

To determine if germination data and PHS scores collected from multiple year and location combinations (or environments) could be pooled for combined analysis of variance (ANOVA), Bartlett’s test for homogeneity of variance in different environments was performed separately for GR, GI, and PHS scores. To assess the relationships between seed dormancy and PHS, Spearman’s rank correlation coefficients were calculated by pair-wise comparisons of GR, GI, and sprouting scores over all nine environments evaluated. The ANOVA was based on the pooled GR and GI data using PROC GLM, the general linear model procedure, including genotype (G), environment (E), and G × E in the model. To identify substitution lines with higher levels of dormancy, Dunnett’s method was used to compare genotypes examined in this study against ISA-3A as the proxy, one of the most dormant genotypes in the test. For PHS scores, Student’s t test was used to estimate least significant difference (LSD) between two means of PHS scores obtained for 36 genotypes grown in each of the four environments at the 5% significance level. All the analyses were performed using SAS (v.9.1, SAS Institute, Cary, NC) and JMP (v.8) statistical software (http://www.jmp.com).

RESULTS

Evaluation of Seed Dormancy

The materials collected in five environments, 2005 Fargo, 2005 Prosper, 2006 Fargo, 2006 Prosper, and 2008 Ithaca, were evaluated for levels of seed dormancy using the germination tests as measured by both GR and GI. Some genotypes with identical GR (e.g., 75%) differed greatly in GI (e.g., 0.28 vs. 0.46). Although GR and GI are highly correlated ($R^2 = 0.90$), GI tends to better differentiate the rates of germination among genotypes.

Variances of GI across all five environments were homogeneous ($\chi^2 = 3.082$, df = 4, $P > 0.5$), while those of GR were not ($\chi^2 = 13.595$, df = 4, $P < 0.01$). However, when the error variances for GR from each of the five environments were estimated, less than a threefold difference (ranging from 179.40 to 404.91) was observed. Based on these test results, both GI and GR over all environments were subsequently combined for further analysis.

The ANOVA based on the pooled GR and GI data revealed that the differences among genotypes were strongly influenced by the environment, as shown by highly significant ($P < 0.0001$) E and G × E terms (Table 2). The evidence

Table 1. Single chromosome substitution lines developed using a durum wheat cultivar Langdon (LDN) as a recipient and three wild emmer wheat accessions (DIC) as donors, and their designated names.

<table>
<thead>
<tr>
<th>Chromosome</th>
<th>PI 481521 (521)</th>
<th>PI 478742 (742)</th>
<th>Israel A (ISA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1A</td>
<td>LDN(521-1A)</td>
<td>LDN(742-1A)</td>
<td>LDN(ISA-1A)</td>
</tr>
<tr>
<td>2A</td>
<td>LDN(521-2A)</td>
<td>n/a</td>
<td>LDN(ISA-2A)</td>
</tr>
<tr>
<td>3A</td>
<td>LDN(521-3A)</td>
<td>n/a</td>
<td>LDN(ISA-3A)</td>
</tr>
<tr>
<td>4A</td>
<td>LDN(521-4A)</td>
<td>LDN(742-4A)</td>
<td>LDN(ISA-4A)</td>
</tr>
<tr>
<td>5A</td>
<td>LDN(521-5A)</td>
<td>LDN(742-5A)</td>
<td>LDN(ISA-5A)</td>
</tr>
<tr>
<td>6A</td>
<td>LDN(521-6A)</td>
<td>LDN(742-6A)</td>
<td>LDN(ISA-6A)</td>
</tr>
<tr>
<td>7A</td>
<td>LDN(521-7A)</td>
<td>LDN(742-7A)</td>
<td>LDN(ISA-7A)</td>
</tr>
<tr>
<td>1B</td>
<td>LDN(521-1B)</td>
<td>LDN(742-1B)</td>
<td>LDN(ISA-1B)</td>
</tr>
<tr>
<td>2B</td>
<td>LDN(521-2B)</td>
<td>LDN(742-2B)</td>
<td>n/a</td>
</tr>
<tr>
<td>3B</td>
<td>LDN(521-3B)</td>
<td>n/a</td>
<td>LDN(ISA-3B)</td>
</tr>
<tr>
<td>4B</td>
<td>LDN(521-4B)</td>
<td>LDN(742-4B)</td>
<td>LDN(ISA-4B)</td>
</tr>
<tr>
<td>5B</td>
<td>LDN(521-5B)</td>
<td>LDN(742-5B)</td>
<td>LDN(ISA-5B)</td>
</tr>
<tr>
<td>6B</td>
<td>LDN(521-6B)</td>
<td>LDN(742-6B)</td>
<td>LDN(ISA-6B)</td>
</tr>
<tr>
<td>7B</td>
<td>LDN(521-7B)</td>
<td>LDN(742-7B)</td>
<td>LDN(ISA-7B)</td>
</tr>
</tbody>
</table>

†Lines with winter growth habit were not included in this study.

‡n/a, not available.
from highly significant ($P < 0.001$) Spearman’s rank correlations among five environments for GR (data not shown) and GI (Table 3) suggested that $G \times E$ interactions were likely due to changes in magnitude rather than rank.

Highly significant differences ($P < 0.0001$) in dormancy levels were observed among the 37 genotypes investigated with an overall mean of GR ranging from 44 to 97%, and that of GI from 0.21 to 0.70 (Table 4). Three genotypes, ISA-3A, ISA-3B, and 521-3A, known to carry the $R$ gene and confirmed with the NaOH test for red kernel phenotype (data not shown), were found to be among the most dormant genotypes but at different levels. Statistically both 3A genotypes had the same GR and GI values, but their dormancy levels were significantly ($P < 0.05$) higher than that found in ISA-3B. The negative result from the seed color test observed in 521-3B and added with the absence of the $R$ gene in this genotype and coincided with the high GR and GI values obtained.

Dunnett’s test was used to compare GR and GI of ISA-3A to amber-colored genotypes. For GR, only 521-2B was similar ($P > 0.05$). However, for GI, four additional genotypes had similar levels of dormancy ($P > 0.05$) including 742-1A, 521-2A, 521-4A, and ISA-7B. Taken together, the results based on germination tests indicated that *T. dicoccoides* chromosomes 1A, 2A, 2B, 3A, 4A, and 7B carry genes affecting seed dormancy.

**Evaluation of PHS**

The same set of 36 substitution lines and LDN was evaluated for dormancy resistance to PHS in 4 yr from 2005 to 2008 in Ithaca, NY. For most of the genotypes, the dormancy levels were not consistent from year to year, indicating strong environmental influence. Bartlett’s test indicated that the variances for the PHS scores among 4 yr were heterogeneous ($\chi^2 = 30.61, df = 3, P < 0.0001$). Spearman’s rank correlation analysis further showed weak correlations among PHS scores in different environments (Table 3). As a result, the PHS data were analyzed separately.

We evaluated PHS tolerance for different genotypes based on their rank in each environment. Among the three substitution lines with red seed color, 521-3A showed the highest levels of resistance to PHS in two out of the four environments studied (Table 4). The level of PHS resistance was similar between ISA-3A and ISA-3B in all four environments, but both were lower than 521-3A. Two genotypes, ISA-4B and ISA-6A, showed consistently higher resistance in all four environments evaluated and were comparable to 521-3A. Four substitution lines exhibiting PHS resistance in three out of the four environments studied included 521-1B, 521-2A, 521-2B, and ISA-7B. Five genotypes displaying high to moderate resistance in two out of four environments were 742-1B, 742-2B, 521-4A, 521-7B, and 742-7B. These results indicate that the genes affecting PHS resistance are likely present in at least eight chromosomes in these *T. dicoccoides* accessions, including 1B, 2A, 2B, 3A, 4A, 4B, 6A, and 7B.

### Correlations between Seed Dormancy and PHS Resistance

To assess the relationship between seed dormancy and PHS resistance, 2008 Ithaca materials were subjected to both germination and sprouting tests. For these samples, a significant ($P = 0.013$) correlation was found between dormancy (GI) and PHS resistance (Table 3). In contrast, relationships between PHS resistance and seed dormancy were weak for samples grown in different geographic areas. While seed dormancy levels for samples grown in all five environments in North Dakota and New York generally agreed with each other as indicated by highly significant correlations ($P < 0.001$), PHS resistance was not consistently expressed

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**Table 2. Mean squares for seed germination tests based on germination rate (GR) and germination index (GI) data.**

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>GR</th>
<th>GI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genotype (G)</td>
<td>36</td>
<td>2507.52***</td>
<td>0.188***</td>
</tr>
<tr>
<td>Environment (E)</td>
<td>4</td>
<td>6706.43***</td>
<td>0.673***</td>
</tr>
<tr>
<td>$G \times E$</td>
<td>144</td>
<td>264.55***</td>
<td>0.017***</td>
</tr>
<tr>
<td>Error</td>
<td>370</td>
<td>98.27</td>
<td>0.008</td>
</tr>
</tbody>
</table>

***Significant at the 0.0001 probability level (F-test).

**Table 3. Spearman’s rank correlation coefficients calculated for pairwise comparisons between germination index (GI) and preharvest sprouting (PHS) scores from all nine environments investigated.**

<table>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>2005 Fargo</td>
<td>-</td>
<td>0.67***</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2005 Prosper</td>
<td>0.55***</td>
<td>0.43***</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2006 Fargo</td>
<td>0.52***</td>
<td>0.55***</td>
<td>0.42***</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2006 Prosper</td>
<td>0.46***</td>
<td>0.52***</td>
<td>0.35***</td>
<td>0.43***</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2008 Ithaca</td>
<td>-0.18</td>
<td>-0.08</td>
<td>-0.17</td>
<td>0.21*</td>
<td>-0.01</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2005 PHS</td>
<td>0.26***</td>
<td>0.33***</td>
<td>0.07</td>
<td>0.15</td>
<td>0.22*</td>
<td>0.11</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2006 PHS</td>
<td>0.19</td>
<td>0.21*</td>
<td>0.08</td>
<td>-0.03</td>
<td>0.22*</td>
<td>0.03</td>
<td>0.28*</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2007 PHS</td>
<td>0.12</td>
<td>0.16</td>
<td>-0.02</td>
<td>0.07</td>
<td>0.24*</td>
<td>0.02*</td>
<td>0.36***</td>
<td>0.19</td>
<td>-</td>
</tr>
<tr>
<td>2008 PHS</td>
<td></td>
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</table>

*Significant at the 0.05 probability level.

***Significant at the 0.001 probability level.
in different years at the same location (Table 3). The generally weak correlations observed between seed dormancy and PHS resistance in these materials indicated the presence of additional and complex factors influencing PHS.

**DISCUSSION**

**Chromosome Locations of QTL for Seed Dormancy and PHS Resistance**

In this study, three sets of single chromosome substitution lines involving donor chromosomes originating from three accessions of wild emmer wheat were evaluated for seed dormancy and PHS separately in multiple field environments. The use of these stocks allowed us to partition the genetic effects influencing seed dormancy and PHS resistance contributed by the donor into individual chromosomes.

Consistent with the previous findings, the three genotypes with red seed color, 521-3A, ISA-3A, and ISA-3B, showed higher levels of dormancy and PHS resistance in most of the environments studied. ISA-3A, however, germinated at a significantly slower rate than ISA-3B did, which generally agreed with the results of Watanabe and Ikebata (2000) from studying the same set of substitution lines involving wild emmer accession ISA. Despite the well-accepted notion of close association between red kernel color and seed dormancy, using a mapping population developed from a red wheat cross, Osa et al. (2003) reported no apparent linkage detected between major QTLs controlling seed dormancy

| Table 4. Combined mean values of germination rate (GR) and germination index (GI) over five environments, and mean values of preharvest sprouting (PHS) scores and their ranks in individual environment. Genotypes underlined showed large genetic effects on both seed dormancy and PHS resistance. |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Germination     | PHS score       | 2005 (rank)     | 2006 (rank)     | 2007 (rank)     | 2008 (rank)     | Overall mean    |
| LDN             | LDN(521-1A)     | LDN(521-1B)     | LDN(521-2A)     | LDN(521-2B)     | LDN(521-3A)     | LDN(521-3B)     |
| GR mean         | 84.98           | 79.70           | 69.20           | 56.94           | 43.60           | 52.10           |
| GI mean         | 0.45            | 0.44            | 0.36            | 0.32            | 0.21            | 0.44            |
| 2005 (rank)     | 3.20 (33)       | 1.17 (14)       | 0.97 (9)        | 3.33 (34)       | 0.87 (7)        | 0.75 (7)        |
| 2006 (rank)     | 1.93 (17)       | 3.93 (33)       | 2.45 (23)       | 0.67 (2)        | 1.17 (1)        | 2.50 (37)       |
| 2007 (rank)     | 2.10 (34)       | 1.20 (17)       | 0.93 (11)       | 0.50 (4)        | 0.07 (1)        | 1.60 (27)       |
| 2008 (rank)     | 1.13 (23)       | 1.47 (26)       | 0.40 (10)       | 0.33 (7)        | 1.07 (20)       | 1.60 (27)       |
| Overall mean    | 1.98            | 1.94            | 1.19            | 1.21            | 0.65            | 2.50            |

LSD (P < 0.05) 7.03 0.06 1.17 1.62 1.54 1.18
and the R gene on chromosome 3A. Further studies on this population revealed that the levels of seed dormancy appeared to be influenced by the allelic interactions between QTLs located on 3A and those detected on homoeologous group 4 chromosomes (Mori et al., 2005).

Other chromosomes were identified as containing genes that provided levels of dormancy and PHS resistance comparable to the levels observed in the substitution lines with red seed color, indicating that multiple genes contributed to dormancy and PHS resistance in the donor parents. Based on the germination tests, the five chromosomes other than 3A responsible for seed dormancy included 1A, 2A, 2B, 4A, and 7B, most of which have been previously reported to contain QTLs associated with seed dormancy in both durum and common wheat. For example, Knox et al. (2005) used a durum wheat cross to locate a major QTL on chromosome 1A. The identification of 2A by Mares et al. (2002) and a 2B QTL by Munkvold et al. (2009) were both based on crosses between two common white wheat cultivars. The 4A QTL has been reported in numerous studies and validated in different genetic backgrounds and is considered a critical component of seed dormancy in wheat (Mares et al., 2005). A QTL mapped on chromosome 7B has been found to be associated with late maturity α-amylase (LMA) activity occurring during later stages of seed development in cool temperatures (Mrva and Mares 2001). The low falling number (FN) is the result of higher level of LMA activity causing the degradation of starch by active α-amylase similar to the early stages of germination and has been used as an additional measurement to assess PHS resistance and seed dormancy, even though LMA is controlled by a separate genetic mechanism from PHS (Mrva and Mares 2002). Our results from PHS tests suggested that at least seven chromosomes, 1B, 2A, 2B, 4A, 4B, 6A, and 7B, in addition to 3A harbor factors associated with PHS resistance. Previous mapping studies using common wheat populations with different sources of PHS tolerance have identified QTLs on chromosomes 1B and 4B (Flintham et al., 2002), 2B (Munkvold et al., 2009), and 4A (Anderson et al., 1993; Lohwasser et al., 2005) based on PHS scores, and QTLs on 2A, 6A, and 7B using the parameters of α-amylase activity and FN to measure PHS resistance (Zanetti et al., 2000). Although most of the chromosomes we identified appeared to be the same as in previous findings, the question of whether the genes linked to seed dormancy and PHS resistance found in this set of materials are the same as those previously mapped must be resolved in future studies.

Correlations between Seed Dormancy and PHS Resistance

Seed dormancy is the main physiological mechanism contributing to PHS resistance. Because PHS was measured based on wetting the entire spike, constraints imposed by the inflorescence structures are expected to influence seed germination on spikes in addition to seed dormancy. While spike wetting tends to mimic field conditions, seed germination, on the other hand, is a direct measure to assess seed dormancy. Significant correlations between these two measures were found in previous studies using both common wheat and durum wheat genotypes (DePauw and McCaig, 1991; Wu and Carver, 1999; Mares and Mrva, 2001). The lack of correlation was also reported in a mapping study by Munkvold et al. (2009). The germination rates were shown to correlate better with FN than with the PHS scores, when these three measurements were assessed to screen wheat breeding lines with PHS tolerance (Trethowan 1995). The seasonal conditions were found to be able to induce different levels of dormancy, which greatly influenced the expression of PHS resistance, particularly for the partially dormant genotypes (Biddulph et al., 2008). Therefore, the different sources of PHS resistance in these studies as well as the complex interactions between genotypes and environments likely contribute to this inconsistency.

The sprouting test is easily performed on a large number of samples, and therefore it is often the method favored by breeders. Our results indicated that genes present on three out of eight chromosomes had effects on PHS resistance only, whereas one of the six chromosomes containing genes having large genetic effects on seed dormancy detected susceptibility to PHS. Similar findings were reported in barley (Hordeum vulgare L.), from which common QTL regions playing major roles in both traits were located on chromosomes, while different QTLs having greater genetic influences on PHS resistance than on seed dormancy were also detected (Ullrich et al., 2008). The different genetic control and environmental effects on these traits likely resulted in the weak correlations between PHS and dormancy observed in this study. Nonetheless, genes present on five chromosomes, 2A, 2B, 3A, 4A, and 7B, affected both PHS and seed dormancy. These genotypes thus provide useful resources for further studies on genetic interactions contributing to the overall phenotypic variation and on genetic dissection of QTLs underlying PHS resistance. A better understanding of genetic control of both PHS resistance and seed dormancy would help ensure grain quality through breeding improvement.

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