New seed dormancy loci detected from weedy rice-derived advanced populations with major QTL alleles removed from the background

Heng Ye a, Michael E. Foley b, Xing-You Gu a,∗

a Plant Science Department, South Dakota State University, Brookings, SD 57007, United States
b USDA-Agricultural Research Service, Biosciences Research Laboratory, Fargo, ND 58105-5674, United States

Abstract

Domestication of cereal crops from wild relatives tended to eliminate seed dormancy. Our objective was to identify all the detectable seed dormancy loci that differentiated between a weedy and a cultivated line of rice to understand the impact of domestication on distribution of dormancy genes. Three additional dormancy quantitative trait loci (QTLs) were identified from two BC1F1 plant-derived F2 populations, in which the five previously known QTL-containing segments from the weedy rice donor were substituted by the genome of the recurrent parent. The three new QTLs that accounted for 8–11% phenotypic variance in the F2s were confirmed with three BC1F2 plant-derived F3 populations. Counting the seven previously identified QTLs, the parental lines differentiate at a total of ten dormancy loci that vary in dominance from dominant, partially dominant, co-dominant, to recessive types. The weedy and cultivated parents contribute dormancy alleles to eight and two of the ten loci, respectively. The two loci were newly detected. We concluded that a large proportion (8/10) of seed dormancy genes have been eliminated during domestication. The minor proportion (2/10) of dormancy genes retained in cultivars could be masked by those with a relatively large effect in a primary segregating population.

1. Introduction

Dormancy is a key adaptive trait for wild species as it promotes the survival of seed-bearing plants in diverse environments by distributing germination over time. Domestication of cereal crops from wild relatives tended to reduce seed dormancy in cultivars to synchronize germination and seedling establishment [1,2]. Some cultivars retain a certain degree of seed dormancy, but many are nondormant. Lack of seed dormancy may lead to pre-harvest sprouting (PHS) when crop harvest is delayed by humid conditions. Genes responsible for seed dormancy and PHS have been identified as quantitative trait loci (QTLs) in barley (Hordeum spp.), rice (Oryza spp.), wheat (Triticum spp.), and some other crops. Early research identified dormancy QTLs differentiated between cultivars [3–5]. Currently, cultivars were often crossed with wild relatives to identify dormancy genes that might have been eliminated during domestication and breeding [6–12]. Validation of the reported dormancy QTLs and identification of additional ones will provide more candidate genes to improve cereal crops for resistance to PHS and also provide a deep insight into genetic and evolutionary mechanisms underlying the domestication-related trait.

Rice (O. sativa L.) is rich in genetic variation for seed dormancy, has a small genome size, and shares genome synteny with the other cereals in the Poaceae (grass) family [13]. The genetic and genomic characters make rice an ideal model system for research on seed dormancy in grasses [14,15]. Seed dormancy QTLs were reported for cultivated [5,16–20], wild [6–8,21], and weedy [22,23] rice on 11 of the 12 chromosomes (excluding chromosome 10). Attempts have been made to find orthologous relationships between some rice and barley or wheat dormancy QTLs [5,8,24]. However, detection of orthologous genes for seed dormancy in cereal crops using comparative genetic/genomic approaches will depend on the quality of QTL mapping information, particularly in the rice model system.

Weedy rice is a noxious weed accompanying cultivated rice worldwide [2]. Its seeds may be dormant in the soil seed bank for seasons to years. We have developed weedy rice (O. sativa spontanea) as a model system to investigate genetic and evolutionary mechanisms for seed dormancy in grasses. Initially, we identified six seed dormancy QTLs from a backcross BC1F1 population developed from the cross between a cultivated and a weedy rice line [22]. The current research was conducted to identify additional seed dormancy loci from the BC1F1 plant-derived advanced generations, in which several previously detected QTL alleles from the weedy rice were removed by marker-assisted selection. By combining mapping information from our previous [22] and current research, a
more complete representation of the genetic complexity of seed dormancy and the impact of domestication on the distribution of dormancy genes is obtained for this model system.

2. Materials and methods

2.1. Genotypes and mapping populations

Previous research detected the seed dormancy loci qSD4, 6, 7-1, 7-2, 8, and 12 from the BC1F1 (EM93-1/EM93-1/SS18-2) [22,25] and qSD1 from two BC2F2 [26] populations. The donor parent SS18-1 in this backcross is a line of weedy rice and contributes dormancy-enhancing alleles to these seven QTLs. The recurrent parent EM93-1 is a line of cultivated rice with weak seed dormancy. To identify seed dormancy loci outside the seven QTL-containing regions, the breeding scheme in Fig. 1 was used to develop single plant (i.e., BC1F1 plants #47 and #109)-derived segregation populations in the BC1F2 (i.e., F2-#47 and F2-#109) or the BC1F3 (F3-#47-101, F3-#47-133, and F3-#109-33) generations. The two BC1F1 plants (*47 and *109) were selected based on marker genotypes (Figs. 2A and 3A), in which the chromosomal regions containing qSD1, 4, 7-1, 12, and 7-2 or 8 were substituted by the genome of the recurrent parent EM93-1, but a majority of the remaining chromosomal segments are heterozygous. These heterozygous segments cover all or majority of chromosomes 2, 3, 5, 9, and 11, on which seed dormancy QTLs were reported for the other crosses [5–7,16–19]. The plants selected plants were used to confirm the QTLs detected from the F2 (Figs. 2B, C and 3B). Thus, the populations derived from these three selected plants were used to confirm the QTLs detected from the F2 populations.

2.2. Plant cultivation and seed harvest

The BC1F1 plant-derived F2 and F3 segregation populations were grown in a greenhouse to harvest seeds for dormancy assessment. To develop an unbiased segregating population, seeds from selected plants were stored at the room temperature or air dried in a greenhouse for >30 days (d) to completely break dormancy before germination. Seedlings were transplanted into 13-cm pots (with one plant per pot) that were filled with a mixture of clay soil and greenhouse medium and placed in water-tight containers (60 cm × 120 cm) to keep the soil moist. Greenhouse temperatures were set at 29/21 °C for day/night. Photoperiods were natural during the summer and supplementary light was applied in early morning and late afternoon during the winter. Plants were tagged for flowering date when the first panicle in a plant emerged from the leaf sheath. Seeds were harvested at 40 d after flowering and air dried in the greenhouse for 3 d before stored in a freezer (−20 °C) to maintain the dormancy status.

2.3. Germination

The degree of seed dormancy was measured by germination percentage with partially after-ripened seeds. The after-ripening treatment was done by leaving seed samples at the room temperature (24–25 °C) for 7 d and/or 14 d. About 50 seeds were germinated at 30 °C and 100% relative humidity in the dark for 7 d in a 9-cm Petri dish that was lined with a Whatman No. 1 filter paper and wetted with 8-ml de-ionized water. Germination was evaluated visually by protrusion of the radicle from the hull by ≥3 mm. Germination experiments were replicated 3 times. Germination percentage in a sample (y) was transformed by sin−1 y½ and mean of the three replications was used for QTL analyses.

2.4. Marker genotyping and map development

All plants from the F2 or F3 populations were genotyped with rice microsatellite (RM) markers located on heterozygous regions in the selected parental F1 or F2 plants (Figs. 2A and 3A). Genomic DNA extraction from fresh leaves, PCR amplification, and marker display were conducted using the methods described in [22]. Genotyping data were analyzed using the software MAPMAKER/EXP 3.0 [27] to develop partial linkage maps for the heterozygous regions. Parameters, including Kosambi’s mapping function [28], were same as the previously described [22]. Physical positions for makers flanking a QTL peak position were determined according to the Nipponbare genome sequence [29]. Position information was obtained by searching the Gramene database for markers or determined by Blast searching the marker primer-containing sequences against the reference genome [30].

2.5. QTL analysis

The Windows QTL Cartographer (v2.5) composite interval mapping (CIM) procedure [31] was used to detect QTL from the F2 and F3 populations. To run the CIM procedure, EM93-1-like homozygous, heterozygous, and SS18–2-like homozygous genotypes for a marker were coded as 1, 2, and 3, respectively, walking speed was set as 1 cM, and background markers were randomly selected at a distance of 11–13 genomic regions (Figs. 2B, C and 3B). Thus, the populations derived from these three selected plants were used to confirm the QTLs detected from the F2 populations.

Fig. 1. Breeding scheme for developing segregating populations to map and confirm new QTLs for seed dormancy.
Fig. 2. Graphic representation of genotypes. (A) BC1 F1 plant #47. The plant was selected from the BC1 F1 (EM93-1/EM93-1/SS18-2) mapping population [22] to detect seed dormancy QTLs (\(q_{SD}\)) within heterozygous chromosomal regions (hatched bars) in the F2-#47 population (Table 1). Empty bars indicate genomic regions homozygous for the chromosomal segments from the cultivated line EM93-1, which was determined by RM (rice microsatellite) genotypes at tick marked positions. (B) F2 plant #47-101. (C) F2 plant #47-133. These two plants were selected from the F2-#47 population to confirm QTLs (Table 1) in the F3-#47-101 and -133 populations. The EM93-1-derived chromosomes or chromosomal segments are not presented in the partial linkage maps. Black bars indicate genomic regions homozygous for the weedy rice line SS18-2-derived chromosomal segments. Open and black circles indicate seed dormancy loci detected in our previous [22,26] and present research, respectively.

3. Results

3.1. Seed dormancy QTLs detected from the BC1 F2 generation

The F2-*47 and F2-*109 plants varied in germination of seeds after-ripened for 7d from 0% to 98%, and the frequency distribution for each of the populations was approximately normal (Fig. 4A and B). Although several dormancy alleles were removed from the background, there was still a range of genetic variation in seed dormancy in the F2 populations as indicated by the distribution pattern. This residual genetic variation should arise from segregation at dormancy loci, including the previously detected \(q_{SD6}\) and \(q_{SD7-2}\), on the heterozygous chromosomal segments in the parents BC1 F1 plants #47 and #109 (Figs. 2A and 3A).
Five seed dormancy QTLs, including previously discovered \( qSD6 \) and \( qSD7-2 \) were identified from the two \( F_2 \) populations. These QTLs were detected by scanning the genomic region of 680 cM segregating in the \( F_2-\#47 \) (\( qSD1-2 \), 6, and 7-2 in Fig. 5A) and 1020 cM segregating in the \( F_2-\#109 \) (\( qSD1-2 \), 3, 6, and 10 in Fig. 5B) population. The QTLs \( qSD1-2 \), 3, and 10 were not detected in the primary (BC1F1) segregation population [22]. However, they accounted for 8–11% of the phenotypic variances in the BC1F2 populations (Table 1). The \( qSD1-2 \) locus, which was significant in both \( F_2-\#47 \) and \( F_2-\#109 \) populations, was located on the long arm of chromosome 1 (Figs. 2A and 3A). Of the three new QTLs, \( qSD1-2 \) and \( qSD10 \) appeared to be co-dominant because they exhibited a predominant additive effect (\( a \)) and a relatively small dominance (\( d \)) effect, and their dormancy-enhancing alleles were derived from EM93-1 (Table 1). \( qSD3 \) was a recessive locus with the SS18-2-derived allele enhancing dormancy and recessive to the EM93-1-derived allele; the degree of dominance (\( d/a \)) for the locus was less than −0.80 (Table 1).

Two sets of digenic epistasis, \( qSD6 \times qSD3 \) and \( qSD6 \times qSD7-2 \), were detected and the component epistatic effect on germination explained 4% and 12% of the phenotypic variances in the \( F_2-\#109 \) and \( F_2-\#47 \) populations, respectively (Fig. 6). The \( qSD6 \) locus was involved in both epistases, but display different interaction patterns. For example, the \( qSD3 \) genotypic difference was greater when \( qSD6 \) was homozygous for the SS18-2- than for EM93-1-derived allele (Fig. 6A), while the \( qSD7-2 \) genotypic difference was smaller when \( qSD6 \) was homozygous for the SS18-2- than for EM93-1-derived allele (Fig. 6B). The \( qSD8 \) QTL, which had significant main and/or epistatic effects on germination in the BC1F1 or BC4F2 populations [22,25], was not detectable in the \( F_2-\#109 \) population. In
addition, none of the dormancy QTLs reported on chromosomes 2, 5, 9, and 11 had a significant main or epistatic effect in the two F2 populations.

### 3.2. Seed dormancy QTLs detected from the BC1 F3 generation

As expected, the F2 plant-derived F3 populations also varied in percentage germination (Fig. 4C–E). Four of the five QTLs (excluding qSD1-2) were detected in one or two of the three F3 populations. The qSD6 and qSD7-2 alleles from SS18-2 reduced germination and accounted for a relatively larger proportion of the phenotypic variances in both F3-#47-101 and F3-#47-133 populations (14–22%) than in the F2-#47 population (8–11%) (Table 1). The recessive QTL qSD3 was significant in the F3-#47-133, but not in the F3-#47-101 population. The qSD10 locus accounted for a large proportion (42%) of the phenotypic variance (Table 1) in the F3-#109-33 population. It is noted that qSD10 is the only known dormancy QTL segregating in the population (Fig. 4E), as the other two loci (qSD1-2 and qSD3) were homozygous for SS18-2-derived alleles (Fig. 3B). The synchronized genetic background could be the major reason for the qSD10's major contribution.

The qSD6 × qSD7-2 interaction was significant in both F3-#47-101 and F3-#47-133 populations. The interaction pattern was similar to that in the F2-#47 population (Fig. 6B) and the component epistatic effect accounted for 12% and 16% of the phenotypic variances in the F3-#47-101 and F3-#47-133 populations, respectively. The qSD6 × qSD3 epistasis was not detectable in the F2-#47-101 population. There was no additional seed dormancy QTL detected in the three F3 populations.

### 3.3. QTL physical intervals

After two (BC1F1 and BC1F2) generations of marker-assisted selection, the length of individual dormancy allele-containing seg-

---

**Table 1** Summary of parameters for seed dormancy QTLs detected in populations derived from BC1 F1 plants #47 and #109.

<table>
<thead>
<tr>
<th>QTL</th>
<th>Chr.</th>
<th>Marker (interval)</th>
<th>LOD</th>
<th>a</th>
<th>d</th>
<th>R²</th>
<th>d/a</th>
<th>Donor</th>
<th>Population</th>
</tr>
</thead>
<tbody>
<tr>
<td>qSD1-2</td>
<td>1</td>
<td>RM3602 (40.2–42.7)</td>
<td>24.7</td>
<td>0.12</td>
<td>0.03</td>
<td>0.11</td>
<td>0.25</td>
<td>EM93-1</td>
<td>F2-#47</td>
</tr>
<tr>
<td></td>
<td></td>
<td>13.0</td>
<td></td>
<td>0.13</td>
<td>−0.03</td>
<td>0.11</td>
<td>−0.25</td>
<td>F2-#109</td>
<td></td>
</tr>
<tr>
<td>qSD3</td>
<td>3</td>
<td>RM520 (35.1–39.6)</td>
<td>15.9</td>
<td>−0.17</td>
<td>0.14</td>
<td>0.09</td>
<td>−0.82</td>
<td>SS18-2</td>
<td>F2-#109</td>
</tr>
<tr>
<td></td>
<td></td>
<td>11.7 (10.4)</td>
<td></td>
<td>−0.10 (−0.13)</td>
<td>0.14 (0.14)</td>
<td>0.11 (0.09)</td>
<td>−1.40 (−1.08)</td>
<td>F2-#47-133</td>
<td></td>
</tr>
<tr>
<td>qSD6</td>
<td>6</td>
<td>RM564B (11.8–15.3)</td>
<td>26.3</td>
<td>−0.10</td>
<td>−0.03</td>
<td>0.11</td>
<td>0.30</td>
<td>SS18-2</td>
<td>F2-#47</td>
</tr>
<tr>
<td></td>
<td></td>
<td>31.5</td>
<td></td>
<td>−0.21</td>
<td>0.06</td>
<td>0.17</td>
<td>−0.29</td>
<td>F2-#109</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>31.0 (30.0)</td>
<td></td>
<td>−0.13 (−0.45)</td>
<td>0.00 (0.07)</td>
<td>0.22 (0.39)</td>
<td>0.00 (−0.15)</td>
<td>F2-#47-101</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>18.6 (12.0)</td>
<td></td>
<td>−0.14 (−0.15)</td>
<td>0.00 (0.03)</td>
<td>0.15 (0.11)</td>
<td>0.00 (−0.20)</td>
<td>F2-#47-133</td>
<td></td>
</tr>
<tr>
<td>qSD7-2</td>
<td>7</td>
<td>RM346 (19.1–25.5)</td>
<td>15.8</td>
<td>−0.13</td>
<td>0.01</td>
<td>0.08</td>
<td>−0.08</td>
<td>SS18-2</td>
<td>F2-#47</td>
</tr>
<tr>
<td></td>
<td></td>
<td>24.4 (28.8)</td>
<td></td>
<td>−0.06 (−0.17)</td>
<td>−0.07 (−0.06)</td>
<td>0.17 (0.19)</td>
<td>1.17 (0.35)</td>
<td>F2-#109</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>14.3 (13.6)</td>
<td></td>
<td>−0.07 (−0.11)</td>
<td>−0.06 (−0.03)</td>
<td>0.14 (0.11)</td>
<td>0.86 (0.27)</td>
<td>F2-#47-133</td>
<td></td>
</tr>
<tr>
<td>qSD10</td>
<td>10</td>
<td>RM271 (12.7–18.0)</td>
<td>13.6</td>
<td>0.11</td>
<td>0.00</td>
<td>0.08</td>
<td>0.00</td>
<td>EM93-1</td>
<td>F2-#109</td>
</tr>
<tr>
<td></td>
<td></td>
<td>24.7</td>
<td></td>
<td>0.15</td>
<td>−0.06</td>
<td>0.42</td>
<td>−0.40</td>
<td>F2-#109-33</td>
<td></td>
</tr>
</tbody>
</table>

**a** Marker nearest the QTL peak position (Fig. 5). Physical intervals on the chromosome in the parentheses are estimated based on the Nipponbare genome sequence in megabase corresponding to the QTL flanking markers.

**b** Parameters derived by the WinQTLCart composite interval mapping program [31], including likelihood value (LOD), gene additive (a) and dominance (d) effects, and proportion of phenotypic variance in germination explained by the locus (R²). Germination was evaluated with the seeds after-ripened for 7 d and/or 14 d and germination data were arc-sin-transformed to estimate the parameters. Estimates based on the 14-d after-ripened seeds are listed in the parentheses. A positive/negative value indicates the SS18-2-derived allele increased/reduced the genic effect on germination. The ratio d/a estimates the degree of dominance.

**c** The parent line contributing the alleles enhancing seed dormancy.

**d** The segregation population in which the QTL was detected.
The R and F2 -# 109 (B) populations. Dormancy was evaluated by germination of 7-d after-ripened seeds. Epistatic interactions between Fig. 6. Likelihood ratio distributions for seed dormancy QTLs (qSD) in F1 -# 47 (A) and F1 -# 109 (B) populations. Dormancy was evaluated by germination of 7-d after-ripened seeds. Likelihood ratio and threshold values were derived by WinQTLCart composite interval mapping [31]. Horizontal bars at the top of each graph indicate lengths of the genomic segments on individual chromosomes (Figs. 2A and 3A) segregating in the populations. Mentions that segregated in the F3 mapping populations were reduced by >50% (Figs. 2 and 3). Based on physical positions of the flanking markers, the five dormancy QTLs could be delimited to the intervals listed in Table 1. Physical lengths of these intervals vary from 2.4 megabases (Mb) for qSD1-2 to 6.3 Mb for qSD7-2. The physical intervals for qSD1-2, 3, 7-2, and 10 are located on the long arms of chromosomes 1, 3, 7, and 10, respectively. qSD6 is located in the pericentromeric region on the short arm of chromosome 6, which was determined by Blast primer-containing sequences of the RM564 and RM541 markers against the Nipponbare genome sequence.

4. Discussion

4.1. Number of seed dormancy QTLs

A total of ten seed dormancy loci were identified from the primary (6) and advanced (4) populations of the EM93-1/SS18-2 cross. Each of these QTLs was validated with one or more populations derived from the same cross. The three new loci explained a relatively small proportion (8–11%) of phenotypic variances in the populations, in which several known dormancy QTL alleles were removed from the genetic background. The qSD1-2 and 10 were not detectable in the BC1 F1 population grown in 2 years [22,25], suggesting that they could be masked by QTLs with a relatively large effect. Number of seed dormancy QTLs reported for the wild/weedy rice-derived segregating populations varied from three to five [7,8,21,23]. It is possible that there are undetected loci for seed dormancy in these populations due to a masking effect from the identified QTL-containing regions, as suggested above.

Considering the QTLs reported on the other chromosomes, the natural variation for seed dormancy in rice is controlled by more than ten genes. This number of genes alone explains the genetic complexity of the seed dormancy trait. We have delimited the ten QTLs to physical intervals of <7 Mbp and introduced them into the EM93-1 genetic background as nearly isogenic lines to characterize physiological and molecular mechanisms regulating primary seed dormancy and germination.

4.2. Dominance and epistasis of seed dormancy genes

Genes for seed dormancy are different in intra- and inter-locus interactions in regulating germination. The ten seed dormancy QTLs display several types of dominance (Table 2). For example, qSD3 was completely recessive (Table 1), qSD7-1 was completely dominant, qSD1-1 and 12 were co-dominant [33]; the remaining six QTLs could be partially dominant or co-dominant. The recessive nature made qSD3 undetectable in the BC1 F1 population with EM93-1 as the recurrent parent. The previously identified seven QTLs interacted with each other by two or higher orders of epistasis [22,26]. Of the three newly detected loci, only qSD3 was involved in the digenic epistasis with qSD6 (Fig. 6A), the remaining two (qSD1-2 and 10) did not interact with each other or with the other loci segregating in the populations. The latter observation will be repeated with different populations to confirm if qSD1-2 and qSD10 are independent of the other eight QTLs in regulating germination in our system.

4.3. Impact of domestication on distribution of seed dormancy genes

The parental lines SS18-2 and EM93-1 contribute dormancy-enhancing alleles to eight and two out of the ten QTLs, respectively. SS18-2 is a line of wild-like weedy rice collected from Thailand where rice cultivars co-existed with the wild rice O. rufipogon. Consequently, SS18-2 could originate from a direct adaptation of the wild rice or derive from a natural hybridization between cultivated and wild rice [2,34]. Asian cultivated rice like EM93-1 also originated from the wild progenitor [2]. The unequal allele distribution between the weedy and cultivated parental lines indicates that domestication and breeding activities retained only a small proportion (20%) of dormancy genes.
Table 2
Summary of seed dormancy QTLs differentiated between EM93-1 and SS18-2 and their allelic relation to other reported loci.

<table>
<thead>
<tr>
<th>QTL</th>
<th>Allelic locus*</th>
<th>R² b</th>
<th>Dominancec</th>
<th>Epistasisd</th>
<th>Distribution*</th>
</tr>
</thead>
<tbody>
<tr>
<td>qSD1-1</td>
<td>sd1, qSD-1</td>
<td>7–22%</td>
<td>CD</td>
<td>Detected</td>
<td>Weed, wild, cultivars [8,23,26,33,35]</td>
</tr>
<tr>
<td>qSD1-2</td>
<td>qSD-1</td>
<td>4–11%</td>
<td>CD or PD</td>
<td>ND</td>
<td>Cultivars [18] (Table 1)</td>
</tr>
<tr>
<td>qSD3</td>
<td>sd3.2</td>
<td>9–11%</td>
<td>Recessive</td>
<td>Detected</td>
<td>Weed, wild [7] (Table 1)</td>
</tr>
<tr>
<td>qSD4</td>
<td>gmr4.1</td>
<td>9–11%</td>
<td>PD</td>
<td>Detected</td>
<td>Weed, wild [21,22,25,26] (Table 1)</td>
</tr>
<tr>
<td>qSD6</td>
<td>sd6</td>
<td>8–39%</td>
<td>CD or PD</td>
<td>Detected</td>
<td>Weed, wild [8,22,25] (Table 1)</td>
</tr>
<tr>
<td>qSD7-1</td>
<td>NI</td>
<td>8–16%</td>
<td>Dominant</td>
<td>Detected</td>
<td>Weed [22,25,26,33]</td>
</tr>
<tr>
<td>qSD7-2</td>
<td>SD-7, qPHS-7</td>
<td>10–22%</td>
<td>CD or PD</td>
<td>Detected</td>
<td>Weed, cultivars [16,22,25,26,35]</td>
</tr>
<tr>
<td>qSD8</td>
<td>qPSR8</td>
<td>7–43%</td>
<td>PD</td>
<td>Detected</td>
<td>Weed, cultivars [20,22,25]</td>
</tr>
<tr>
<td>qSD10</td>
<td>NI</td>
<td>8–42%</td>
<td>PD</td>
<td>ND</td>
<td>Cultivars (Table 1)</td>
</tr>
<tr>
<td>qSD12</td>
<td>NI</td>
<td>24–68%</td>
<td>CD</td>
<td>Detected</td>
<td>Weed [22,25,26,33]</td>
</tr>
</tbody>
</table>

a QTL on the same genomic region reported for the other crosses. NI, not identified yet.
b Range of the QTL contributions to the phenotypic variances in different populations.
c The dormancy-enhancing allele was co-dominant (CD), partially dominant (PD), dominant, or recessive over the dormancy-reducing allele.
d Epistatic effect was detected or not detected (ND).

Some of the eight dormancy alleles from SS18-2 are present in wild rice (e.g., qSD3 and qSD4), but were not detected in cultivated lines, whereas others (qSD1-1 and qSD7-2) were reported for both wild and cultivated rice (Table 2). This suggests that genes controlling the major domestication-related trait vary in response to human selections. qSD3 was mapped to the seed shattering locus qSH3 region, qSD4 was co-located with a cluster of QTLs for shattering (qSH4), black hull color (qBH4), and awn length (qAL4-A and qAL4-D), qSD7-1 was co-segregated with the red pericarp color QTL/gene (qPC7/Rc), and qSD12 displayed a major effect on delaying germination in a series of our research [25]. Dormancy genes at the aforementioned loci should be more responsive to selections against undesirable wild traits or for rapid germination and have a less chance to be retained in modern cultivars; these loci must hold molecular information that is important for understanding the evolution of cereal crops. In contrast, dormancy genes at the loci with a relatively small effect on germination and/or independent of the undesirable wild traits (e.g., qSD1-2 and qSD10) are more likely to distribute in cultivars, as they are less sensitive to selection. These loci are likely the major source of genes that have been used to manipulate germabliity for new cereal varieties or to enhance resistance to pre-harvest sprouting in breeding programs. Cereal crop improvements might be made by incorporating some of the wild and weedy dormancy genes, if they can be uncoupled from the undesirable traits.

Acknowledgements
We thank B. Carsrud for technical assistance. This research was supported in part by grants from National Science Foundation (0641376) and United States Department of Agriculture National Research Initiative (2008-35301-19058).

References