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Mapping QTLs for seed dormancy and the Vp1 homologue on chromosome 3A in wheat

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Abstract A major component of the observed genetic variation for pre-harvest sprouting in wheat (*Triticum aestivum* L.) appears to be the level of seed dormancy. Group 3 chromosomes have received attention as carrying the *R* genes for seed-coat color and the *taVp1* genes that are orthologous to the maize *Vp1* gene which encode a dormancy-related transcription factor. The objectives of the present study were to map quantitative trait loci (QTLs) for seed dormancy on chromosome 3A and to investigate an association between *taVp1* or *R-A1* and the QTLs detected. A mapping population in the form of recombinant inbred lines developed from the cross between the highly dormant Zenkoujikomugi (Zen) and Chinese Spring (CS) was utilized. Nineteen marker loci, including *taVp1*, were mapped on chromosome 3A. The *taVp1* locus was located in the middle of the long arm, about 85 cM from the centromere. The population was evaluated in duplicate by growing them under controlled environment conditions. Two QTLs for seed dormancy, designated as *QPhs.ocs-3A.1* and *QPhs.ocs-3A.2*, were identified on the short and long arms, respectively. *QPhs.ocs-1* explained 23–38% of the phenotypic variation and the Zen allele had a striking effect on maintaining dormancy. *QPhs.ocs-2*, with a minor effect, was detectable only at the dormancy-breaking stage. Although *QPhs.ocs-2* was loosely linked to *taVp1* by around 50 cM, they are clearly distinct genes. Zen and CS carry the white *R-A1a* allele, and no QTL effect was detected in the vicinity region of *R-A1*. Hence it was concluded that the high dormancy associated with chromosome 3A of Zen is ascribable to *QPhs.ocs-1* on the short arm but is not due to the direct contribution of either the *taVp1* or *R-A1* locus.

Keywords *Triticum aestivum* · Pre-harvest sprouting · Seed dormancy · QTL mapping · *Vp1*

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Introduction

Pre-harvest sprouting (PHS) is a problem in many wheat-growing areas, resulting in a downgrading of quality and severe limitations in end-use application. Tolerance to PHS is therefore highly desirable. As a major component of the observed genetic variation for PHS appears to be the level of seed dormancy, efforts to elucidate the mechanisms of seed dormancy and dormancy breakage have been ongoing for a long time. Dormancy is generally expressed as a quantitative trait governed by many genes, and in a few cases these genes have been mapped to specific chromosome regions. As a result of the progress with molecular approaches, DNA markers have made it possible to identify individual genetic factors controlling such complex traits as seed dormancy, and several genes or chromosomal regions affecting dormancy have been identified in wheat genomes by means of molecular markers (Anderson et al. 1993; Flintham et al. 1999; Roy et al. 1999; Zanetti et al. 2000; Kato et al. 2001; Mares and Mrva 2001; Groos et al. 2002).

On the other hand, relatively little is known about the molecular events underpinning the expression of dormancy. The red flavonoid pigment in the testa layer of the maternal seed coat has long been recognized as a genetic marker for seed dormancy and tolerance to PHS (Nilsson-Ehle 1914). It is known that the seed-color *R* genes are located as homoeologous loci on the long arms of group 3 chromosomes (Sears 1954; Nelson et al. 1995; Flintham and Gale 1996). Using near-isogenic lines for the *R* genes, Flintham et al. (1999) and Watanabe and Ikebata (2000)

found that these genes have direct effects on dormancy. Several quantitative trait locus (QTL) analyses also demonstrated QTLs for PHS that are co-localized with the *R* genes (Nelson et al. 1995; Groos et al. 2002). Moreover, the presence of other dormancy genes on group 3 chromosomes independent of *R* genes was suggested by cytogenetic analyses (Mares 1996; Miura et al. 1996; Flintham et al. 1999).

McCarty et al. (1991) showed that the maize transcription factor VIVIPAROUS-1 encoded by the *Vp1* gene plays a critical role in the induction and maintenance of dormancy. Jones et al. (1997) isolated the *afVp1* clone from wild oat (*Avena fatua*) – *afVp1* being the orthologous gene to the maize *Vp1* – and indicated a significant role of *afVp1* in the control of dormancy. In wheat, the *Vp1* homologues, designated *taVp1*, have been mapped on the long arms of group 3 chromosomes (Bailey et al. 1999), and researchers have suggested that they have possible effects on seed dormancy (Nakamura and Toyama 2001; Groos et al. 2002). On the other hand, McKibbin et al. (1999) reported that the abundance of *taVp1* transcripts was similar in developing embryos of dormant and non-dormant genotypes and was not related to the germination potential. So there is no conclusive evidence for the contribution of *taVp1* to seed dormancy in wheat.

In the framework of improving PHS levels, therefore, group 3 chromosomes have received attention as carrying the *R* genes and *taVp1* genes. In our previous study using a backcross reciprocal monosomic population it was revealed that chromosome 3A had the most striking effect on variation in seed dormancy, followed by group 4 chromosomes (Miura et al. 2002). However, the identities of the genes encoding seed dormancy remain to be discovered, and more direct information about the role of *taVp1* is needed. The objectives of the present study were to identify QTLs for seed dormancy on chromosome 3A and to investigate an association between *taVp1* and the QTLs detected.

Materials and methods

Plant material

A mapping population in the form of recombinant inbred lines (RILs) was obtained by single-seed descent (F_7 generation) from the cross between Zenkoujikomugi (Zen) and Chinese Spring (CS). Zen is a Japanese red-spring wheat which shows an extremely high level of seed dormancy and tolerance to PHS (Osanai and Amano 1993; Miura et al. 1997). CS is a red wheat and has some dormancy (Flintham et al. 1999; Warner et al. 2000), but its level is much less than that of Zen (Miura et al. 2002).

For the construction of the genetic linkage map and QTL analysis, we used 125 RILs. To determine chromosome or chromosome arm location of the molecular markers in the map construction, we used CS and its aneuploid stocks, including the CS nullisomic 3A tetrasomic 3B line and the CS ditelosomics 3AL and 3AS lines (Sears 1954).

DNA isolation and hybridization

Genomic DNA was extracted from fresh leaves of 2-week-old F_6 plants by a modified CTAB method (Murray and Thompson 1980). DNA digestion, electrophoresis and Southern blotting were performed according to the methods described by Kato et al. (1998). DNA hybridization was carried out using the non-radioisotope Gene Images labeling and detection system (Amersham Pharmacia Biotech, UK). A total of 107 restriction fragment length polymorphism (RFLP) clones were used as probes. They were chosen as already known to hybridize with DNA fragments located in group 3 chromosomes of wheat.

Following a screening of the parents for marker locus polymorphisms, chromosome assignment of the screened RFLP markers was accomplished using the aneuploid lines. For the markers that proved to be polymorphic between CS and Zen, and to establish their location on chromosome 3A, the RIL population was genotyped.

The *taVp1*-specific probe was obtained from Dr. N. Kawakami, who constructed a cDNA library from an immature embryo of a PHS-tolerant Kitakei 1354 and screened it using part of a *afVp1* homologue as a probe (N. Kawakami, unpublished data). Southern hybridization using the *taVp1* clone was carried out after the clone was labeled with α-[^{32}P]-d CTP by the random hexamer primer method (BcaBEST, Takara). The details are described in Ahmed et al. (2000).

Analysis of SSR markers

A total of 34 simple sequence repeat (SSR) primer sets, specific for wheat chromosomes 3A, 3B and 3D (Roder et al. 1998), were used for this analysis. A polymerase chain reaction (PCR) was performed in a 24-μl volume containing approximately 1 μg of genomic DNA, 10× PCR buffer [15 m*M* Mg^{2+}], 25 m*M* each dNTP, 10 μM of each primer, and 1.0 U of *Taq* polymerase (Nippon Gene). The thermocycling program consisted of an initial denaturation at 94 °C for 5 min, followed by 30 cycles of 1 min at 94 °C, 1 min at 50–60 °C, 2 min at 72 °C and a final step of 5 min at 72 °C. PCR products were separated on a 3.0% agarose gel and visualized by staining with ethidium bromide.

Evaluation of seed dormancy

The levels of seed dormancy in the RIL population (F_7 plants) were evaluated in duplicate by growing the plants under controlled environment conditions to minimize environmental variation. The trial was conducted using two plants per genotype. The RILs and parents were raised from flowering to harvesting in growth chambers maintained at 20 °C during the day and 16 °C at night and a daylength of 16 h. The experiment was replicated in August to December, 2000 (hereafter GC'00) and January to April, 2001 (GC'01).

In both trials, flowering date was recorded on a genotype basis to examine the degree of seed dormancy at a given number of days post-anthesis (DPA). All plant materials were harvested at 45 DPA to coincide with physical maturity. Harvested spikes were allowed to air-dry for about 1 week until the moisture content of the seed was approximately 14%, and dried spikes were gently hand-threshed. To elucidate the degree of dormancy at the dormancy-breaking stage, germination tests were further carried out in GC'01 using seeds that were harvested at 45 DPA and stored 4 weeks further at room temperature (GC'01-Late).

Germination tests were performed in plastic petri dishes on paper filters wetted with distilled water at 20 °C in the dark. Duplicates of 50 seeds were tested in each trial. Germinated seeds were recorded daily and removed. Germinability, measured as cumulative percentage germination for 10 days, was used to estimate the degree of seed dormancy.

Statistical analysis

A linkage map was constructed using MAPMAKER/EXP 3.0 (Lander et al. 1987), and the recombination frequencies were converted to centiMorgans (cM) using the Kosambi mapping function (Kosambi 1944) based on the genotype data of RFLP and SSR markers.

Percentage germination was transformed to arcsine and subjected to statistical analyses. The chromosomal location of QTLs for seed dormancy was determined by the simple interval mapping method using the QGENE program (Nelson 1997). A log-likelihood (LOD) score threshold of 3.0 was used to identify regions containing putative loci associated with seed dormancy.

Results

Genetic map of chromosome 3A

The chromosome 3A map illustrated in Fig. 1 was constructed on the basis of the genotypic classifications for the RIL population. Nineteen marker loci covering approximately 250 cM were mapped. RFLP analyses of ditelosomic stocks available in CS demonstrated that the centromere was assigned within the marker interval between *Xgwm5* on the short arm and *Xpsr394* on the long arm. As a result, five loci consisting of four RFLP markers and a SSR locus were mapped on the short arm, and ten RFLP and three SSR markers were assigned together with *taVp1* on the long arm. Chi-square analysis indicated that all RFLP and SSR markers segregated in a 1:1 ratio. This suggests that the polymorphic marker loci are not linked to factors affecting genetic fitness in single seed descent.

When the current linkage map was compared with previously published maps of group 3 chromosomes, the marker order revealed was comparable with that of the maps compiled by the International Triticeae Mapping Initiative (Sorrells and Anderson 1997). In our map, however, there were two large gaps – a 50.9-cM interval between *Xbcd1380* and *Xfbb370* on the short arm and a 52.9-cM interval between *taVp1* and *Xgwm1071* on the long arm.

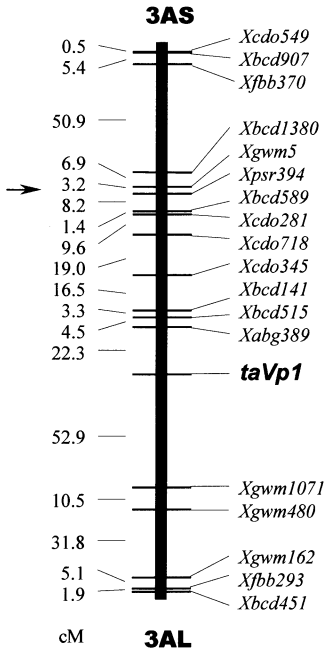

Fig. 1 The genetic linkage map of chromosome 3A with RFLP and SSR markers constructed in this study. *Arrow* Centromere

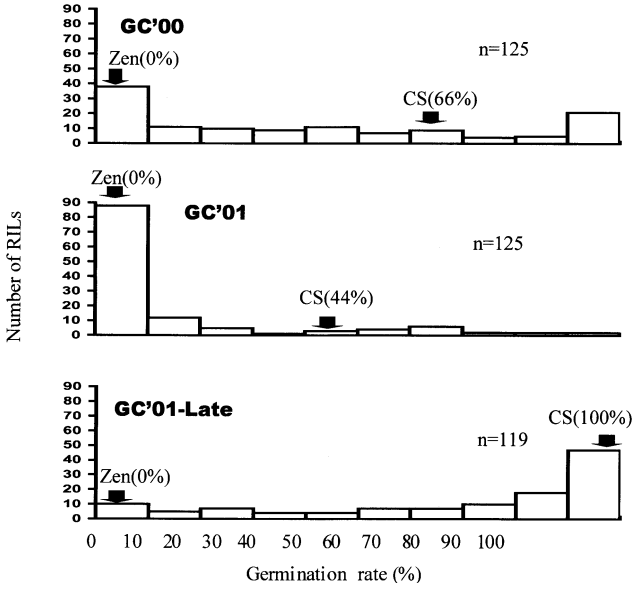

Fig. 2 Frequency distributions for cumulative percentage germination for 10 days at 20 °C in 125 or 119 RILs in GC'00, GC'01 and GC-01-Late trials

Map position of *taVp1*

A wheat cDNA clone *taVp1*, isolated by Dr. N. Kawakami (unpublished data), was used as a probe to determine the chromosomal location of the *taVp1* locus. When DNAs from CS and Zen were digested with multiple restriction enzymes, *taVp1* hybridized to three fragments in most cases, indicating that the *taVp1* gene is present as a single copy in each of the A, B and D genomes. When digested with *Hin*dIII, CS had a marker band of about 20 kbp, whereas Zen lacked it. Nullisomic and ditelosomic analyses indicated that this marker band was encoded by the gene located on 3AL. Following the genotypic classification for the RIL population, *taVp1* was mapped in the middle of the long arm of chromosome 3A (Fig. 1), 84.8 cM from the centromere.

QTLs for seed dormancy

A large difference in germination rate between parental CS and Zen was detected in GC'00 and GC'01 (Fig. 2). CS showed a 66% germination rate in GC'00 and 44% in GC'01, while complete dormancy was observed in Zen. These results confirmed a distinctive difference in the level of seed dormancy between the parents. Over two trials, the 125 RILs gave a continuous distribution of

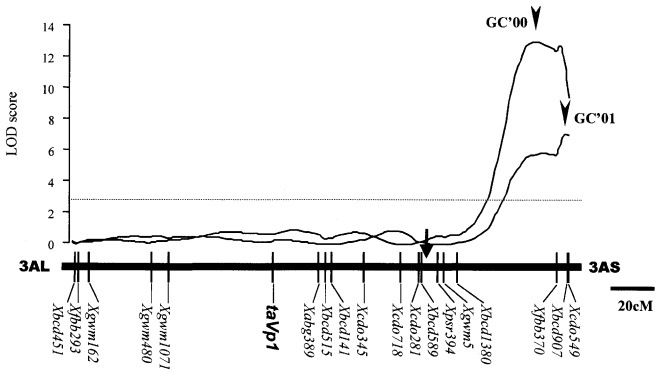

Fig. 3 QTL-likelihood curves of LOD scores showing the locations of QTL for seed dormancy in GC'00 and GC'01 trials. *Arrow* Centromere

germination rates ranging from 0% to 100%. RILs expressing high dormancy predominated, especially in GC'01.

Using QGENE analysis we deduced one putative QTL associated with seed dormancy. The QTL, designated *QPhs.ocs-3A.1*, was identified within the marker intervals between *Xbcd1380* and *Xfbb370* (GC'00), *Xfbb370* and *Xbcd907* (GC'01) in the terminal region of the short arm (Fig. 3). The LOD score of the QTL peaks was larger in GC'00 (13.05) than in GC'01 (7.15). The percentages of phenotypic variation explained by this QTL were 38.2% in GC'00 and 23.3% in GC'01 (Table 1). The Zen alleles at the *QPhs.ocs-1* contributed to keeping seed dormancy.

QTL at the dormancy-breaking stage

When germination tests were carried out in GC'01-Late using seeds of 119 RILs harvested at 45 DPA and successively stored 4 weeks at room temperature, CS and most RILs had high germination rates with dormancy breaking, while Zen and some RILs still maintained complete or strong dormancy (Fig. 2). Figure 4 shows an interval mapping in GC'01-Late. One peak was identified on the long arm. The QTL on the long arm, designated as *QPhs.ocs-3A.2*, was identified within the marker interval between *Xcdo345* and *Xbcd141*, proximal to *Xcdo345*, the LOD score was 3.60. The percentage of phenotypic variation explained by *QPhs.ocs-2* was 13.0% (Table 1). Zen alleles at both the QTLs increased dormancy. *QPhs.ocs-1* on the short arm had a small effect (LOD=2.55) at the dormancy-breaking stage.

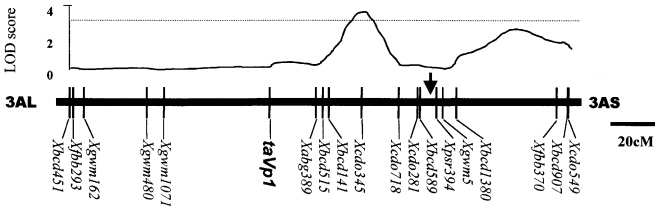

Fig. 4 QTL-likelihood curve of LOD scores showing the locations of QTL for seed dormancy in GC-'01-Late trial. *Arrow* Centromere

The proximal marker of *QPhs.ocs-2* is *Xcdo345*, which is linked to *taVp1* by 46.6 cM. Therefore, it was concluded that the QTL effect associated with the long arm is not attributable to *taVp1*. The two-way ANOVA detected no significant effect of interaction between *QPhs.ocs-1* linkage marker *Xfbb370* and *QPhs.ocs-2* linkage marker *Xcdo345* (data not shown), indicating no epistasis between *QPhs.ocs-1* and *QPhs.ocs-2*.

QTL × environment interaction

On the basis of these results, the presence of QTL × environment interaction was deduced. Using data from the three experiments, QGENE analysis was conducted and QTL × environment interaction was detected at *QPhs.ocs-1* and *QPhs.ocs-2*. However, LOD scores for QTL × environment were much smaller than the corresponding main effects (data not shown). The seed dormancy QTL in the controlled environments of the present study was somewhat affected by environment, but more importantly by the QTLs *QPhs.ocs-1* and *QPhs.ocs-2*.

Discussion

Seed dormancy in wheat is generally governed by many genes, and in a few cases these genes have been mapped to specific chromosome regions. Analysis of the Zen × CS RIL population in this study identified two QTLs, *QPhs.ocs-1* on the short arm and *QPhs.ocs-2* on the long arm of chromosome 3A, that were associated with clear differences in seed dormancy and contributed by the highly dormant parent, Zen. Expression of the *QPhs.ocs-1* effect on seed dormancy was highly repeatable under the controlled environments of the GC'00 and GC'01 trials. If it is proven that this QTL is a beneficial gene in marker-assisted selection for PHS tolerance, specific molecular markers that are easy to handle and very useful for wheat

Table 1 Location of QTLs for seed dormancy in GC'00, GC'01 and GC'01-Late trials detected by QGENE and differences in cumulative percentage germination in the Chinese Spring and Zenkoujikomugi allele classes at the proximal markers

Trial	Marker interval	LOD score	r^2	% Germination for 10 days			
				Marker	CS[a]	Zen[a]	Difference
GC'00	*Xbcd1380/Xfbb370*	13.05	0.382	FBB370	65.17	23.09	42.08
GC'01	*Xfbb370/Xbcd907*	7.10	0.233	BCD907	23.11	2.62	20.49
GC'01-Late	*Xbcd141/Xcdo345*	3.60	0.130	CDO345	71.03	51.55	19.48

[a] CS and Zen represent the means of RILs carrying Chinese Spring alleles and Zenkoujikomigi alleles, respectively

breeders should be developed. However, the deduced position of *QPhs.ocs-1* varied somewhat between trials, being between *Xbcd1380* and *Xfbb370* in GC'00, and between *Xfbb370* and *Xbcd907* in GC'01. Thus, more investigations are needed to dissect this QTL, particularly to determine its precise position via linkage to molecular markers. Moreover, the stability of the *QPhs.ocs-1* effect should be determined after the Zen × CS RIL population is evaluated with replication in several environments, because the ability to detect genetic loci accounting for phenotypic variation of seed dormancy trait is compromised by the effects of environment and genotype × environment interactions (Anderson et al. 1993). Multiple evaluations over years and locations are now in progress.

Aneuploid analyses have detected dormancy effects and therefore implicated QTLs on the short arms of group 3 chromosomes (Mares et al. 1996; Flintham et al. 1999). However, no QTLs on the short arms of group 3 chromosomes have been described in other studies on PHS tolerance and seed dormancy of wheat using molecular markers (Anderson et al. 1993; Roy et al. 1999; Zanetti et al. 2000; Mares and Mrva 2001; Groos et al. 2002). Recently comparative mapping across wheat and barley has demonstrated the possibility of a homoeologous relationship between the seed dormancy QTL on chromosome 4AL and the barley SD4 gene (Kato et al. 2001). So far identified in barley (Oberthur et al. 1995) and rice (Lin et al. 1998; Cai and Morishima 2000), however, the homoeologous regions with wheat 3AS do not carry genes associated with seed dormancy. Thus, it is of interest to know if *QPhs.ocs-1* is a gene restricted to wheat. On the other hand, the *QPhs.ocs-2* region on the long arm of chromosome 3A is orthologous with chromosome 1 of rice (Ahn et al. 1993; Kurata et al. 1994; Gale and Devos 1998; Bailey et al. 1999). The closely linked marker of *QPhs.ocs-2* is *Xcdo345*. In rice, *Xcdo345* on the long arm of chromosome 1 is again a QTL marker associated with seed dormancy (Cai and Morishima 2000), suggesting a possible relationship between *QPhs.ocs-2* and the rice QTL linked to *Xcdo345*.

In general, red-grained wheats have a higher resistance to PHS than white-grained cultivars. Both Zen and CS are red-grained wheats. Zen has the dominant red alleles at the *R-B1* and *R-D1* loci on chromosomes 3B and 3D, respectively, and the white *R-A1a* allele on chromosome 3A (Miura et al. 2002). The red-grain color of CS is due to the dominant *R-D1b* allele (Sears 1954; McIntosh et al. 1998). Therefore, Zen and CS are polymorphic only with respect to the *R-B1* locus. Since the two parental cultivars have the recessive white-allele *R-A1a* on chromosome 3A, the genetic linkage map in this study excludes the *R-A1* locus. Groos et al. (2002) indicated that the *R-A1* gene is closely linked to *Xgwm480* in the genetic maps constructed from a white × red cross. When this SSR clone was surveyed in the present experiment, it was polymorphic between Zen and CS and mapped on the long arm with 148.2 cM apart from centromere (Fig. 1). No QTL effect was detected in the vicinity region of *R-A1, Xgwm480.*

The distance between *taVp1* and *Xgwm480* was 63 cM with the order of centromere-*taVp1-Xgwm480*, indicating that *taVp1* and *R-A1* are distinct genes. Bailey et al. (1999) indicated a loose genetic linkage between these two genes in the group 3 consensus map. Based on this evidence they proposed that genetic separation between the two loci reflect separate roles for *taVp1* and *R* in zygotic and maternal dormancy mechanisms, respectively. To date there are opposing reports with respect to the role of *taVp1* in wheat seed dormancy. McKibbin et al. (1999) reported that the abundance of *taVp1* transcripts was similar in developing embryos of dormant cv. Soleil and non-dormant cv. Boxer and was not related to the germination potential. Conversely, Nakamura and Toyama (2001) presented a different result that expression of *taVp1* differs between dormant and non-dormant genotypes. In the present study, we concluded that the high dormancy associated with chromosome 3A of Zen can be ascribed to *QPhs.ocs-1* on the short arm but is not due to the direct contribution of either *taVp1* or *R-A1* loci. However, our result does not rule out roles for the *taVp1* homoeologues in the regulation of seed dormancy expression. Holdsworth et al. (2001) showed that transcripts arising from the *taVp1* genes are not correctly spliced, suggesting that sub-optimal *Vp1* expression may not be sufficient to repress the onset of germination-related gene expression. Therefore, further analyses are needed to clarify the effects of *QPhs.ocs-1* and *QPhs.ocs-2* on *taVp1* gene expression and the biochemical functions of these QTLs.

To improve PHS tolerance, Osanai and Amano (1993) developed breeder's lines, designated as OS or OW, that were derived from the progeny of Zen by cross breeding. Those lines received attention as breeding materials to develop new cultivars that are more tolerant to PHS than Zen. Dormancy QTLs specific to the Zen genome would be attractive not only for practical breeding purposes but for analyzing genetic control mechanisms of seed dormancy and PHS tolerance. Previously we showed that the strong dormancy of Zen is ascribable to QTLs on group 4 chromosomes as well as QTLs on 3A (Miura et al. 2002). There are several reports pointing to the dormancy QTLs on group 4 chromosomes (Flintham et al. 1999; Kato et al. 2001; Mares and Mrva 2001, Noda et al. 2002), but whether those QTLs reported are identical to the Zen QTLs on group 4 are unknown. To resolve this, we are carrying out mapping studies of QTLs on group 4 chromosomes of Zen.

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