RESEARCH PAPER

A centromeric region on chromosome 6(6H) affects dormancy in an induced mutant in barley

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Abstract

Genetic control of seed dormancy in barley (Hordeum vulgare L.) has mostly been described in terms of quantitative variation. Although some molecular markers for dormancy QTL have been identified, the corresponding genes involved in the regulation of the process have not been cloned. Induced barley mutants may constitute useful material to study the physiology and genetics of seed dormancy. The objective of this study was to identify the genetic control of this trait in a mutant (TL43) produced in the barley cv. Triumph. This mutant was selected for reduced dormancy and reduced sensitivity to abscisic acid (ABA). Two sets of F6 barley lines were selected for high and low levels of dormancy from a cross between the original dormant parent and the sodium azide-induced non-dormant TL43 mutant. Unexpectedly, given the near-isogenic nature of these two genotypes, polymorphism was detected for an SSR located in the centromeric region of chromosome 6(6H) out of a total of 92 molecular markers evenly distributed along the genome. Fortunately, upon three cycles of intensive divergent selection, every dormant and non-dormant F5 line consistently showed the genotype for this region identical to Triumph and TL43, respectively. Based on the mutagenic effect presumably attributed to sodium azide, mostly single point mutations, it cannot be clearly established if such extensive genomic variation on chromosome 6(6H) is due to the mutagenic treatment or may be an introgression from an unknown source. The means that could originate such heterogeneity are discussed; however, regardless of its origin, this genomic region shows a strong association with the expression of seed dormancy and provides an additional genetic locus for further studies of the mechanistic basis of this complex trait. In addition, since TL43 shows reduced sensitivity to ABA, the response to this hormone was determined on the F6 seed from the two sets of selected F5 lines. The results confirmed that the initial level of dormancy in the seed lot is the most important factor in determining ABA sensitivity.

Key words: Abscisic acid sensitivity, barley mutant, chromosome 6H BIN 6, dormancy.

Introduction

Seed dormancy is defined as the absence or delay of germination in mature intact seed under favourable environmental conditions. The degree of seed dormancy within a population can be variable and may be determined by interactions among plant genotypes, maternal environments, harvest and post-harvest conditions, and germination environment (Hareland and Madson, 1989). Moderate seed dormancy is desirable in barley (Hordeum vulgare L.) to prevent premature seed germination while still on the spike of the mother plant (preharvest sprouting). However, the persistence of dormancy in the barley seed may create problems in stand establishment and in the malting process when seeds are used within a short period of time after harvest. Moreover, dormant seeds in the soil bank can become weeds in subsequent crops (Ullrich et al., 1993). Inception of dormancy is established during seed development, and its release is due to exposure for some period of time to a set of afterripening environmental conditions (Simpson, 1990).

Genetic variation for dormancy in several species has been described mostly in terms of quantitative inheritance. The availability of DNA-based molecular markers has made
the dissection of polygenic traits into their genetic components possible by genetically mapping quantitative trait loci (QTLs). In barley, the most extensive research on the genetic and environmental basis of seed dormancy has been performed in a Steptoe/Morex population by the North American Barley Genome Project. Two major dormancy QTL regions, designed as SD1 and SD2, have been identified, respectively, near the centromere and long arm telomere on chromosome 7(5H) (Ullrich et al., 1993; Han et al., 1996).

Mutants could constitute relevant material to associate physiological processes and major effects over quantitative traits, for example, seed dormancy. Genetic variation for seed dormancy has been studied in the past years in the model plant Arabidopsis thaliana because a number of mutants have been found to have a seed dormancy phenotype (Koornneef and Karssen, 1994; reviewed in Bentsink and Koornneef, 2002). Among these are the mutants in which the biosynthesis of the growth-inhibiting hormone abscisic acid (ABA) is impaired, or the sensitivity to this phytohormone is strongly reduced; the genetic, physiological, and molecular characterizations of these mutants have shown the important role of ABA in seed dormancy (reviewed in Karssen, 1995). ABA-deficient mutants such as aba1, aba2, and aba3 germinate precociously due to defects in ABA biosynthesis (Koornneef et al., 1983), while ABA-response mutants such as abi1, abi2, and abi3 show reduced seed dormancy and can germinate in the presence of ABA (Koornneef et al., 1984). Several other mutations in genes such as FUSCA3 (FUS3, Bäumlein et al., 1994), and LEAFY COTYLEDONS (LEC1 and LEC2; Meinke et al., 1994) and in genes with unknown functions, such as those disrupted reduced dormancy 1–4 mutants (rdo; León-Kloosterziel et al., 1996), have also been implicated in establishing seed dormancy.

In barley, only two mutant lines have apparently been reported that germinate more rapidly than the wild type: a mutant with gigantum appearance, but with no information about its dormancy properties (Visser et al., 1996); and the mutant line TL43 derived from the wild type cv. Triumph, and whose dormancy profile, endogenous ABA content during seed development, and sensitivity to ABA in mature grain throughout the afterripening period have been investigated by Romagosa et al. (2001). TL43 was selected for reduced dormancy, but has a similar amount of endogenous ABA and shows reduced sensitivity to the application of exogenous ABA compared with Triumph. Based on the extensive and detailed characterization of the mutant TL43 with respect to its germination behaviour governed by ABA, the objective of the present study was to identify the mutant genomic region induced in the Triumph seed and, consequently, to determine the impact of this region on the expression of seed dormancy and sensitivity to ABA.

### Materials and methods

#### Plant material and germination tests

The mutant line TL43 was induced in the barley cv. Triumph with sodium azide (NaN3) and was selected as described by Molina-Cano et al. (1999). Triumph and its M8-derived mutant, TL43, were crossed in the spring of 1998, with the subsequent production of 104 F2 plants in the greenhouse. Based on their germination percentage, progeny with high or low levels of seed dormancy were selected from the F2 to F4 generations in the greenhouse (20 ± 3 °C) according to a pedigree breeding scheme. Table 1 shows the selection criteria used in each cycle of selection in order finally to select five dormant and five non-dormant F2 lines, depending on the environmental conditions prevailing every generation. Spikes of the parental lines and of each F2, F3, and F4 genotype selected for high or low dormancy were harvested at physiological maturity (the time when all green colour is lost from the spike) and hand-threshed prior to being placed in storage at –20 °C to preserve dormancy. After removal from the freezer, seed was permitted to afterripen at ambient temperature (20 ± 5 °C) for 25 d prior to conducting germination analysis. Germination tests were carried out in 90 mm Petri dishes on Whatman no. 1 filter papers saturated with water. Two replications of 50 seeds each were germinated at 20 °C under standard test procedures (EBC, 1987). After 3 d, germinated seeds (coleoptile emerged through the hull) were counted and expressed as a percentage of the total (GP).

To avoid selection against dormant types, two procedures were conducted to allow advancement from the F2 to F4 generation. All seeds from lines selected for high dormancy that did not germinate at 20 °C in the germination tests were surface-sterilized in 10% sodium hypochlorite solution for 1 min and rinsed for 2 min in sterile water. Subsequently, sterilized seeds were placed in Petri dishes with Whatman no. 1 filter papers saturated with water and induced to germinate at 4 °C. After 7 d, the occasional seed that failed to germinate at 4 °C was considered non-viable. Six randomly germinated seeds of each selected line for low dormancy at 20 °C and six randomly germinated seeds of each selected line for high dormancy at 4 °C were transferred from the Petri dish in which the germination assay took place, into a tray in a cool room (4 °C) to obtain the seedlings that were, subsequently, planted in two pots with three seeds each in the greenhouse.

The F4 seed of the five most non-dormant and five most dormant genotypes and both parents were sown in the field in Lleida, Spain, in 2001. Seed of these same lines were also grown in the greenhouse during spring 2001 at Lleida. The F5 seed obtained was used to assess the release of dormancy during afterripening. Spikes of each selected line were harvested at physiological maturity and hand-threshed. Germination tests of afterripened seed, stored at room temperature

#### Table 1. Criteria for selection and genotypes selected in the F2, F3, and F4 generations based on the germination percentage of 104 F2 plants derived from the TL43/Triumph cross under greenhouse growing conditions

<table>
<thead>
<tr>
<th>Generation</th>
<th>Direction of selection</th>
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<tr>
<td></td>
<td>Low dormancy</td>
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<td>Selection</td>
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<tr>
<td>criterion</td>
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<tr>
<td>F2 seed</td>
<td>&gt;90</td>
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<td>F3 seed</td>
<td>&gt;80</td>
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<td>F4 seed</td>
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Molecular marker analyses

Genomic DNA was extracted from the leaves of greenhouse-grown seedlings of F5 lines and parents following a CTAB extraction method (Saghai-Marooti et al., 1984). Eighty SSR, seven RFLP, and seven STS markers approximately evenly distributed on the seven barley chromosomes were used to detect polymorphism between Triumph and TL43. SSR analysis was performed according to the protocols and primer sequences obtained from Ramsay et al. (2000). RFLP analyses were conducted as described in Molina-Cano et al. (1999). The STS markers were analysed according to Sayed-Tabatabaei et al. (1999), who published the sequences.

Statistical analysis

The germination profile during afterripening for the F5 seed was statistically characterized by two variables: overall level of dormancy and rate of dormancy release through afterripening. The first variable was estimated as the average GP at 15 dph (GP15). Dormancy release was determined through simple linear regression analysis with the slope of the straight-line with GP as the dependent variable and dph as the independent variable. GP15 and the slopes of straight lines of dormancy release were independently estimated for the parental lines and for each of the F5 lines comprising the two dormancy level sets from the two environments used; field and greenhouse.

Analysis of variance was carried out using standard SAS procedures (SAS Institute, 2001) to test differences among and within the two sets of lines. The genotypic mean effect (11 degrees of freedom) was partitioned into two terms to account for heterogeneity of genotypes within each dormancy type; Between-types term (with one degree of freedom) accounts for the fraction of genotypic effect due to differences between the two dormancy types; Within-types term (with 10 degrees of freedom) represents that part of the genotypic effect attributable to differences among genotypes within each dormancy type. This last term can be partitioned further into four extra terms (two for each dormancy type) in order to account for differences between the F5 lines (one degree of freedom) and the parental lines and for differences among F5 lines that belong to a particular dormancy type (4 degrees of freedom each).

The results of the response to exogenous ABA for the F5 seed were fitted to a dose–response logistic model: \( GP = a/(1 + \exp(-b \times c)) \), using Table Curve2D procedures (Jandel Scientific, 1994). The parameters \( a \) and \( b \) of the curve have a direct biological meaning: \( a \) represents the germination of the untreated seeds (GP0); and \( b \) the ABA concentration that reduces germination of the untreated control to half (LD50). Comparison of these two parameters by means of their 95% confidence intervals allows analysis of genotypic differences in the response to exogenous ABA.

Dormancy in a barley mutant

(20 ± 5 °C), were carried out at several post-harvest dates; seed grown under field conditions was germinated at 1, 7, 15, and 30 d post-harvest (dph) and seed grown in the greenhouse at 1, 15, and 30 dph. Three replicates of 50 seeds of each sampling date were germinated with the same protocol described previously. Germinated seeds counted at 3 d were scored and expressed as percentage of the total (GP).

The F5 seed from three of the dormant and three of the non-dormant selected lines, jointly with the parents, were grown in the field in Lleida in 2003. Determination of the sensitivity to exogenous ABA was performed on the F5 seed. Assays were carried out after two different post-harvest afterripening periods at room temperature (20 ± 5°C); 0 and 50 dph. Germination tests were conducted by adding ABA to the incubation medium according to the same germination protocol described previously. Two replicates of 50 seeds of size between 2.5 mm and 2.8 mm were germinated in increasing ABA concentrations in the germination medium: 0, 10⁻⁷, 10⁻⁶, 10⁻⁵, 10⁻⁴, and 10⁻³ M ([±]-cis-trans-abscisic acid, Sigma, Alcobendas, Madrid, Spain).

In order to detect the genomic modifications produced by the mutagenic treatment, the PCR products of the markers that were determined to be polymorphic between Triumph and TL43 were sequenced. The DNA fragments of interest were run in an agarose gel and subsequently isolated by using a commercial purification procedure (Amicon, Inc. Beverly, MA 01945, USA). These fragments were sequenced using an ABI373-A Stretch Sequencer (Applied Biosystems), and finally the sequences for the wild type and the mutant were compared.

Results

Physiology of seed dormancy: differential selection for germination and ABA sensitivity

The level of seed dormancy of the 104 TL43/Triumph F2 genotypes is depicted in Fig. 1. The mean and standard error for GP of the population was 67 ± 2.6 % with a range of 5–100%. The GP for Triumph and TL43 were 19% and 94%, respectively. The shape of the TL43/Triumph F2 population histogram was skewed to high GP values. Although this result suggests a recessive inheritance for seed dormancy, the continuous variation observed for GP seems to indicate that environmental factors affect the expression of this trait.

In order to develop two sets of recombinant inbred lines (RILs) differing in their expression of seed dormancy, a divergent selection scheme for high or low level of dormancy was performed (Table 1). The five most dormant and the five most non-dormant genotypes throughout F2, F3, and F4 generations comprised the two dormancy types. Based on the average GP of the two sets, large differences in dormancy were detected (Fig. 2). The average GP of the five lines selected for high dormancy and the average GP of the five lines selected for low dormancy were almost equal to the GP for Triumph and TL43, respectively. Triumph and the dormant lines exhibited a GP lower than 50% at each

![Fig. 1. Frequency distribution of germination percentage of the F2 population derived from the TL43/Triumph cross under greenhouse conditions.](https://academic.oup.com/jxb/article-abstract/56/409/47/467347)
Average of the five F5 lines

ripening (afterripening, analyses of variance for the fraction of variation, for variation within the genotypic effect (80.4% and 85.8% of the variables analysed. Similarly, when comparing the F5 a particular dormancy type were not significant for either genotypic differences among the F5 lines that belong to the expression of seed dormancy during afterripening were observed within each dormancy type seems to confirm that the realized heritability for seed dormancy is high. Large selection responses found in early generations indicates that the heritability for seed dormancy is high.

On the basis of GP measured incrementally from 0–30 d afterripening, analyses of variance for GP at 15 d afterripening (GP 15) and for the slopes of the simple linear regression lines of dormancy release for Triumph, TL43, and F5 lines were calculated for both field and greenhouse data (Table 2). The genotype main effect was partitioned to determine the homogeneity in expression of dormancy of the two sets of selected lines. Highly significant differences (P ≤0.01) were observed between the dormant and non-dormant types for GP 15 and for dormancy release. Genotypic differences among the F5 lines that belong to a particular dormancy type were not significant for either of the variables analysed. Similarly, when comparing the F5 lines to the parents within each dormancy type, no significant differences were found between Triumph and the five most dormant lines, or between TL43 and the five most non-dormant lines for either GP 15 or dormancy release. Such consistent results reveal that differences between dormancy types were responsible for most of the variation within the genotypic effect (80.4% and 85.8% of the fraction of variation, for GP 15 and dormancy release, respectively). In addition, the little genotypic variation observed within each dormancy type seems to confirm that two homogeneous sets of F5 lines significantly differing in the expression of seed dormancy during afterripening were successfully developed and selected.

Response curves of the F6 seed to different concentrations of ABA in the germination medium are shown in Fig. 3. Data were fitted to a dose–response logistic model defined by the germination of the untreated seeds (GP 0) and the ABA concentration that reduces germination of the untreated control to half (LD 50). Genotypic differences in ABA sensitivity were detected by comparing the 95% confidence intervals for GP 0 and LD 50. Very good levels of fit were obtained (r 2 ranging from 0.96 to 0.99). Response to exogenous ABA was determined in two post-harvest dates: 0 and 50 dph. On the 0 dph seed, Triumph and the three dormant selected genotypes had a significantly different ABA response profile from TL43 and the three non-dormant genotypes (Fig. 3A). Although, the non-dormant lines are able to maintain germination at ABA concentrations ten-times higher than the dormant genotypes, these differences in ABA response are clearly determined by the initial level of dormancy in the untreated seed (GP 0 value). No significant differences were obtained among genotypes for LD 50. In fact, when genotypes release from dormancy on the 50 dph seed, i.e. similar GP 0 value for the dormant and non-dormant lines, ABA responses were comparable (Fig. 3B).

Genetics of seed dormancy: linkage of dormancy with molecular markers

A total of 92 markers with a well-spaced distribution on the barley genome (ranging from 10 to 15 markers in each of the seven chromosomes) were screened to identify polymorphism between Triumph and the induced mutant TL43. Using a Triumph/Morex 147-point map as a base (Prada et al., 2004), 80 SSR and seven STS markers placed in approximately 20 cM intervals in the map, jointly with five RFLP markers previously analysed (Molina-Cano et al., 1999), were used. Of all the markers initially analysed, only one SSR locus, HVM65, resulted in polymorphism between Triumph and TL43. HVM65 is located in the centromeric region of the chromosome 6(6H) BIN 6. The consensus comparative BIN map (Kleinhofs and Graner, 2001) obtained by dividing the Steptoe/Morex map into approximately 10 cM intervals allows the placement of many markers mapped on different crosses in their appropriate BINs. Six additional SSR markers in BIN 6 on chromosome 6(6H) (Ramsay et al., 2000) were analysed and two of them, EBrac639 and Bmag0344a, revealed additional polymorphism between Triumph and TL43.

To check the linkage between this region located on chromosome 6(6H) and the expression of seed dormancy, individual genotyping of the F5 lines belonging to the two dormancy types was conducted for HVM65, EBrac639, and Bmag0344a. Remarkably, all dormant and all non-dormant lines consistently showed the allele for the three SSR identical to Triumph and TL43, respectively. No recombination was found among the three loci for all the genotypes within each dormancy type. Therefore, based on these results a strong association between this region on chromosome 6(6H) and the expression of seed dormancy was established.
The putative genomic changes elicited by sodium azide in Triumph seed were determined by nucleotide sequencing of the three mutant SSRs in TL43 compared with the progenitor Triumph sequences. However, DNA sequence analysis of the three polymorphic loci revealed changes in the repeat lengths of the dinucleotide SSRs. The HVM65 primer gave PCR-amplified products of 131 and 129 bp for TL43 and Triumph, respectively, due to a 2 bp GA insertion in the mutant. The EBmac639 primer gave PCR-amplified products of 164 and 150 bp for Triumph and TL43, respectively, this time due to a 14 bp (TG)7 deletion in the mutant. Likewise, the Bmag0344a primer gave PCR-amplified products of 165 and 163 bp for Triumph and TL43, respectively due to a 2 bp CT deletion in the mutant.

**Discussion**

In a previous study, the dormancy and ABA-response properties of TL43 and Triumph were analysed under low and high dormancy-inducing conditions in Lleida (Spain) and Dundee (Scotland), respectively. TL43 had reduced dormancy and reduced sensitivity to ABA compared with Triumph (Romagosa *et al.*, 2001).

The high selection response for barley seed dormancy in this study is consistent with literature reports that the
The high heritability of seed dormancy in the TL43/Triumph-derived lines allowed for divergent selection of two sets of F2 lines greatly differing in their level of seed dormancy starting in the F2 generation (Fig. 2). Germination tests to determine the ABA response were individually performed in the selected dormant and non-dormant genotypes (Fig. 3). The initial level of dormancy played a key role in the sensitivity to ABA; when seed dormancy was completely released, no genotypic differences were detected in the ABA-response profile. Therefore, response to exogenous ABA decreased with dph. In addition, ABA sensitivity was determined on post-harvested seed of cvs Morex and Steptoe (data not shown), which could be considered, respectively, as the non-dormant and dormant standard cultivar to check dormancy properties in barley. The results of this analysis confirmed that the higher the initial level of germination, the lower the sensitivity to maintain germination at the same ABA concentration. Therefore, low dormancy and reduced sensitivity to ABA seem to be expressions of a common phenomenon rather than independent phenotypical events.

To date, Olsen et al. (1993) and Druka et al. (2003) have provided the most extensive information for barley with respect to natural and mutagen-induced sequence alterations produced by sodium azide. Those authors detected single base substitutions only, randomly scattered on the sequence that codifies for a specific polypeptide within one NaA-treated barley genotype.

In the present study, given the near-isogenic nature of Triumph and TL43, polymorphism between the two genotypes was unexpectedly displayed in an SSR (HVM65) in chromosome 6 (6H) BIN 6. Further, two SSRs (EBmac639 and Bmag0344a) which appear closely linked to HVM65 in the barley consensus map (Kleinhofs and Graner, 2001) resulted in the identification of additional polymorphism. The PCR products of the polymorphic markers were sequenced, and differences for HVM65 were attributable to a GA insertion, whereas for EBmac639 and Bmag0344a deletions of the (TG)7 and CT repeat SSR motifs, respectively, were observed.

Such repeat length variation suggests that this is conventional SSR polymorphism. Therefore, it is hard to assign these genomic changes to the mutagenic effect of sodium azide (mostly point mutations). The extensive genomic alteration reported here could have been originated by other means and additional factors might need to be taken into account to explain these observations. Firstly, this heterogeneity could already be present in the original dormant treated material. In fact, it is well known that cv. Triumph, is heterogeneous for some molecular markers (G Fishbeck, personal communication), and thus TL43 may have been selected from a pre-existing variant of the original cultivar. However, such heterogeneity in chromosome 6(6H) should not be associated with the observed reduced dormancy in TL43 and the non-dormant F2 lines given the dormant behaviour exhibited by the original cv. Triumph. Secondly, this extensive genomic variation may be the result of a introgression from an unknown source. A putative source of hybridization might come from cv. Alexis because, whilst the selection of TL43 from a mutagenized population of cv. Triumph was being performed, another mutagenic experiment was developed in cv. Alexis; this derives from a Triumph cross and is phenotypically similar except for the lack of dormancy (Molina-Cano et al., 1999). In order to eliminate the possibility of accidental hybridization with Alexis, the genotypic identities of the selected alleles in chromosome 6(6H) for TL43 were checked. Results confirmed the allelic distinctiveness of TL43 in relation to Alexis, and thus discarded this cultivar as a possible source of introgression. Whether this region results from a normal out-cross from an additional genotype remains still unknown.

The three polymorphic markers included in this genomic region in chromosome 6(6H) showed a lack of recombination among them, which indicates that such a region has been maintained throughout three intensive cycles of divergent selection in the Triumph/TL43 cross. In fact, recombination within centromeric regions, where this heterogeneity is located, is highly suppressed in barley.
(Künzel et al., 2000). Given the level of resolution afforded in this study it cannot be clearly determined if either a single major locus or a gene cluster responsible for seed dormancy are present within the boundaries of these markers in chromosome 6(6H).

Regardless of its origin (either caused by mutation or present in the original seed or the result of an introgression from an unknown source), and the nature of the putative gene(s) included (either a single major locus or a gene cluster), such heterogeneity has allowed the detection of a genomic region in chromosome 6(6H) responsible for this trait. Interestingly, data presented provide additional locus/loci for further studies of the mechanistic basis for the complex regulation of seed dormancy.

Although seed dormancy in barley has been extensively studied by means of QTL mapping in segregating populations, this is the first report in which a genomic region involved in the expression of this trait has been located by using a barley mutant and a set of F₂ lines derived from the cross between the wild type and the mutant. The most widely conserved dormancy QTLs among different barley crosses has been detected near the centromere and long arm telomere on chromosome 7(5H) within the Steptoe/Morex population (Ullrich et al., 1993), the Harrington/TR306 population (Takeda, 1996), and the Triumph/Morex (T/M) population (Prada et al., 2004). Although TL43 and the T/M doubled haploid line (DHL) population were developed from the common genotype Triumph, the mechanism of genetic control for seed dormancy appears to be different. TL43 presumably has the same allele as Triumph for both major dormancy QTLs on chromosome 7(5H), while the genomic region in TL43 on chromosome 6(6H) detected in the present work has no effect in the expression of seed dormancy within the T/M DHL population.

Several QTLs that affect physiological mechanisms putatively governed by ABA in barley have been located at the same, or adjacent, position to the TL43 mutant region on this chromosome (http://www.css.orst.edu/barley/nabgrp/ QTLsum9150×.htm). Mano and Takeda (1997) identified a QTL for salt tolerance during germination in the S/M population, and a germination-rate QTL at the same location in the Harrington/TR306 cross (Takeda, 1996). Tuberosa et al. (1997) found at this site a QTL related to freezing survival and Teulat et al. (1998) detected an osmotic-adjustment QTL in an adjacent position. There is evidence that ABA has a large impact in modulating the adaptive response mechanisms of plants to abiotic stresses. Seed dormancy could be considered as an adaptive trait because it plays a major role in the ecological adaptation of wild plant species by enhancing the distribution of germination over time. It is, therefore, hypothesized that the presence of a putative gene(s) near the centromere of chromosome 6(6H) is involved in the regulation of the germination process and is strongly modified by ABA. Additional research on this chromosome 6(6H) region that affects both dormancy and ABA sensitivity may provide additional insights into the physiological pathway leading to the imposition and/or release of seed dormancy in barley.

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