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Study of the relationship between pre-harvest sprouting and grain color by quantitative trait loci analysis in a white×red grain bread-wheat cross

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Abstract In many wheat (*Triticum aestivum* L.) growing areas, pre-harvest sprouting (PHS) may cause important damage, and in particular, it has deleterious effects on bread-making quality. The relationship between PHS and grain color is well known and could be due either to the pleiotropic effect of genes controlling red-testa pigmentation (*R*) or to linkage between these genes and other genes affecting PHS. In the present work, we have studied a population of 194 recombinant inbred lines from the cross between two cultivars, 'Renan' and 'Récital', in order to detect QTLs for both PHS resistance and grain color. The variety 'Renan' has red kernels and is resistant to PHS, while 'Récital' has white grain and is highly susceptible to PHS. A molecular-marker linkage map of this cross was constructed using SSRs, RFLPs and AFLPs. The population was evaluated over 2 years at Clermont-Ferrand (France). PHS was evaluated on mature spikes under controlled conditions and red-grain color was measured using a chromameter. Over the 2 years, we detected four QTLs for PHS, all of them being co-localized with QTLs for grain color. Three of them were located on the long arm of chromosomes 3 A, 3B and 3D, close to the loci where the genes *R* and *taVp1* were previously mapped. For these three QTLs, the resistance to PHS is due to the allele of the variety 'Renan'. Another co-located QTL for PHS and grain color was detected on the short arm of chromosome 5 A. The resistance for PHS for this QTL is due to the allele of 'Récital'.

Keywords *Triticum aestivum* · Dormancy · Marker regression · Genetic mapping

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Introduction

Pre-harvest sprouting (PHS) is defined as the germination of grains in the ear before harvest and is related to an early raising of dormancy. In many wheat-growing areas, in some years it causes significant damage. PHS causes yield losses due to a diminution of grain weight, but the main problem is a decrease in bread-making quality. Sprouted wheat flour looses its thickening power and some products can not be obtained from flours milled from sprouted wheat. Breads baked from sprouted wheat will have a smaller volume and a compact interior (Mansour 1993). This decrease in quality is mainly due to early alpha-amylase activity, which can be characterized by a low Hagberg falling number.

Variation caused by environmental factors affects the study of PHS and makes the selection of resistant varieties difficult. It has been known from some time that PHS is associated with grain color (Nilsson-Ehle 1914): red-kernel wheats appear to be more resistant to PHS than white-kernel wheats. Thus, red-grain color has been used in wheat breeding programs as a marker for resistance to PHS. This association between PHS and grain color may be due either to a pleiotropic effect of the genes controlling grain color or to genetic linkage between these genes and genes affecting PHS. The red-testa pigmentation of wheat grain is controlled by three genes, named *R-A1*, *R-B1* and *R-D1*, which have been located on homoeologous group 3 by cytogenetic analyses (Sears 1944; Allan and Vogel 1965; Metzger and Silbaugh 1970). Flanking markers for the homoeologous loci have been identified (Nelson et al. 1995; Flintham and Gale 1996). Using near-isogenic lines for the *R* genes, Flintham et al. (1999) have shown that these genes have direct effects on dormancy. Recently, Bailey et al. (1999) have mapped the *taVp1* loci on chromosome arm 3L of wheat at 30 cM from the *R* loci. The *taVp1* gene is orthologous to the *VP1* gene which encodes a dormancy related transcription factor in maize (McCarty et al. 1991). Moreover, McKibbin et al. (1999) have shown that a loss of embryo dormancy was associated with a decrease in

taVp1 gene expression. Thus, it could be postulated that the relation between PHS and grain color is due to both a direct influence of the *R* genes on 'coat-imposed' mechanisms of dormancy and to the linkage of the *R* genes with the gene *taVp1*, which is related to the embryo mechanisms of dormancy.

Several QTL analyses have been conducted on PHS or dormancy in barley (Feng et al. 1999), sorghum (Lijavetzky et al. 2000), rice (Lin et al. 1998) and wheat. Using RFLP markers on wheat, Anderson et al. (1993) detected eight regions of the genome significantly associated with resistance to PHS. Roy et al. (1999) identified a microsatellite on chromosome 6B and a STS marker on 7D linked to PHS. This latter result was confirmed by Flintham et al. (1999) who found a new strong factor for resistance to PHS linked to a microsatellite of chromosome 7D and named it *Phs*. In these three studies, no influence of the chromosomes of homoeologous group 3 on PHS has been detected, even in crosses between red- and white-kernel wheats.

The aim of this study was to determine the genetic basis of the relationship between PHS and grain color by QTL analyses in a cross segregating for the two traits, in order to analyze the respective role of the genes *R* and *taVp1* on the resistance to pre-harvest sprouting in wheat and to identify other loci controlling PHS, unlinked to this *R-taVp1* system.

Material and methods

Plant material

A mapping population of 194 RILs (F_7 lines) was obtained by single-seed descent (SSD) from the cross between 'Renan' and 'Récital'. The variety 'Renan' has red kernels and is resistant to PHS, while 'Récital' is a white-grain wheat highly susceptible to PHS.

The 194 RILs and the parent genotypes were evaluated in a randomized trial with two replications in 1999 and 2000 at the INRA station of Clermont-Ferrand (France). Each plot measured 7.5 m^2 and plants were grown under normal field conditions.

Some RILs appeared to be heterogeneous or mixed in the field and these were discarded from further analysis, decreasing the number of studied RILs for QTLs analyses from 175 to 185 depending on the trait×year combination.

Map construction

A genetic linkage map was developed using RFLP, microsatellite and AFLP markers. This map also included some storage-protein loci.

DNA of the RILs and parents was extracted from fresh leaves of F_7 plants in 1998 according to the method described by Lu et al. (1994). For RFLP analysis, the non-radioactive probe protocol detailed in Lu et al. (1994) was used. PCR reactions for microsatellite sequences and for AFLP detection were performed using the methods described by Tixier et al. (1998) and by Bert et al. (1999), respectively.

Linkage analyses were performed using Mapmaker/exp 3.06 (Lander et al. 1987). The Kosambi mapping function (Kosambi 1944) was applied to transform recombination frequencies into additive distances in centiMorgans. Linkage groups were assigned to chromosomes via comparisons to reference maps using microsatellite loci (Cadalen et al. 1997; Röder et al. 1998).

Evaluation of PHS

The test used in this study has been developed in France by the 'Groupement d'Etude des Variétés Et Semences' (GEVES) and is used in official trials for variety registration (Renard et Lerebour in Gate 1995).

At maturity, five random spikes (cut approximately 5 cm from the base of the spike) from each plot were harvested. Harvested spikes were immersed for 4 h in test tubes in a 6% copper oxyquinoleate solution. Most of the solution was then removed, keeping just 1 cm in the bottom of the tubes, which were covered with a plastic film to ensure a moist atmosphere. The tubes were kept at 20°C for 7 days. After this time, the number of germinated and non-germinated grains were recorded for each spike.

Estimation of grain color

For each genotype, the grain color was evaluated using a chromameter (Minolta 310). This equipment decomposes color in the L*a*b color space (CIE 1931). In this color space, 'L' measures black (0) to white (100), 'a' measures green when negative and red when positive, and 'b' measures blue when negative and yellow when positive. Wang et al. (1999) have shown the efficiency of this method in determining the number of dominant alleles at the *R* loci of a variety. The measure was done on a sample of about 20 g of grains in a 55 mm Petri plate. The chromameter averages three measures by sample, and for each genotype we used four grain samples.

Statistical analyses

Analyses of variance for year, replication and genotype effects, and the calculation of correlations, were carried out using SAS 6.1 software (SAS Institute 1991). The method for detection of QTLs was carried out using Splus (1993) 'home-made' programs. First, one-way analyses of variance (ANOVA) were used to detect significant differences between genotypic classes for each marker. These significant markers were used as candidates in a multiple regression model, in order to select a subset of non-redundant markers for further use as co-variates. Then, on every linkage group, on which at least one marker was found significant, the marker regression method (Kearsey and Hyne 1994) was carried out to locate the QTLs more precisely and estimate their additive effects. Whenever the residuals of the one-QTL model remained significant, a two-QTL model was fitted using a two-dimensional scanning of the chromosome (Hyne and Kearsey 1995). When performing the marker regression on a given linkage group, the markers from the selected subset, which were located on other chromosomes, were used as co-variates in the model to reduce the residuals, and thus improve QTL detection power and accuracy, which should be very similar to that of Composite Interval Mapping (Jansen and Stam 1994). The 95% confidence intervals of the QTL locations and effects were established by bootstrapping (Visscher et al. 1996) using 200 replicates. In any case, only those replicates for which the appropriate model was detected were used for constructing the confidence interval (Lebreton and Visscher 1998).

Results

Current map

The current map for the population 'Renan'×'Récital' comprises 436 markers on 34 linkage groups of 2 or more markers. Total map length at the present time is 2,259 cM. The linkage groups are distributed throughout the wheat genome, but chromosomes 1D, 4A, 4B, 4D and 7D are still not well-covered.

Fifty three markers deviated significantly from the expected ratio for RILs (1:1) at $P<0.01$. Most of them

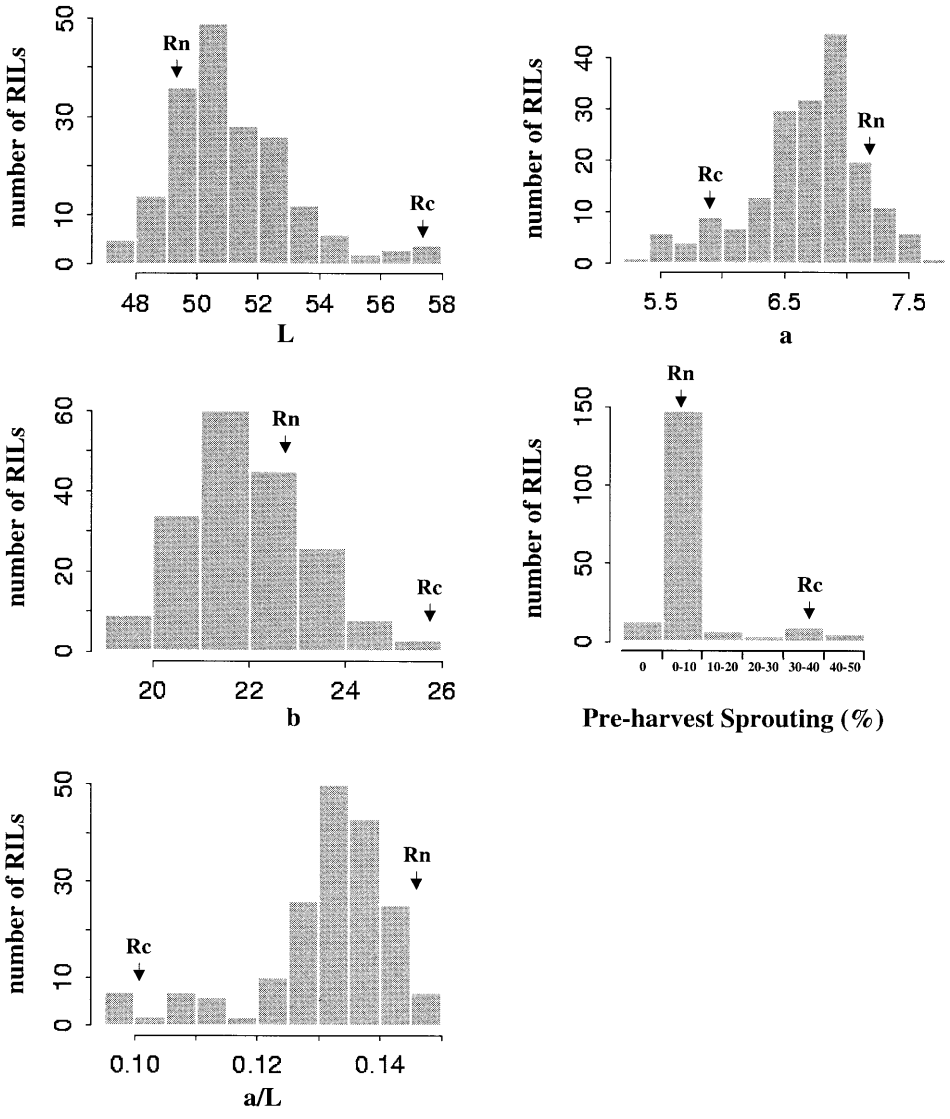

Fig. 1 Histograms of the distribution of components for grain color and PHS among the RILs derived from the cross between 'Renan' (*Rn*) and 'Récital' (*Rc*). *L*, *a* and *b* are the components of color in the L*a*b color space (CIE 1931). *L* measures black (0) to white (100); a, green (–) to red (+); and b, blue (–) to yellow (+)

Table 1 Analysis of variance for pre-harvest sprouting and grain color

	PHS			Grain color		
	SS	*df*	*F*-value	SS	*df*	*F*-value
Year	1,212.79	1	26.15***	93.25	1	4,212.25***
Block (year)	261.02	2	2.8 ns	19.05	2	430.33***
Genotype	534,760.37	188	61.33***	597.97	184	146.81***
Genotype*year	24,556.64	174	3.04***	136.33	175	35.19***
Error	159,266.34	3434		55.90	2,525	
R-square of model	0.94			0.78		

*** significant at the 0.001 probability level, *ns* not significant

were mapped on chromosomes 2A, 2B, 2D and 6B. On all of these chromosomes, the markers were skewed towards the 'Récital' allele. Deviations from the expected ratio have been observed on chromosome 6B in other studies: in the cross Courtot×Chinese Spring by Cadalen et al. (1997) and in the cross NY6432–18×'Clark's Cream' by Campbell et al. (1999). An hypothesis for this deviation was the existence, on this chromosome, of a 'pollen killer' gene (Ki), which would be brought by 'Récital' in our cross.

Phenotype analyses

The variance analysis for PHS and grain color showed significant genotype/year interactions for all characters (Table 1). Nevertheless, the correlations between years

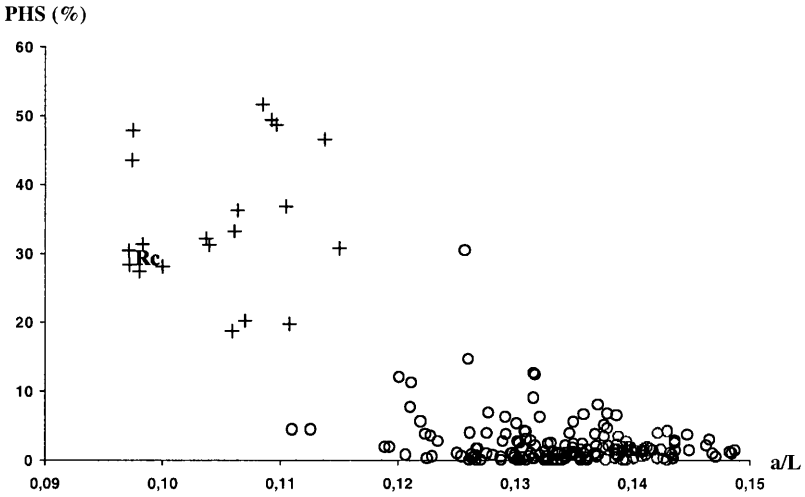

Fig. 2 Relation between PHS and grain color in the population 'Renan'×'Récital' (R^2=0.57): +, white grain lines; ○, red grain lines

Table 2 Significant markers for pre-harvest sprouting and grain color

Markers	Location	Pre-harvest sprouting						Grain color (a/L ratio)					
		1999		2000		Mean		1999		2000		Mean	
		R-square	Additive value	R-square	Additive value	R-square	Additive value	R-square	Additive value	R-square	Additive value	R-square	Additive value
Xfbb293	3A	5.5**	2.9	7.9***	3.0	7.1***	3.1	4.5**	0.003	7.1***	0.002	5.6**	0.003
Xgwm403	3B	7.4***	3.4	7.2***	3.0	7.8***	3.3						
Xbcd131	3B	6.7***	3.2	11.4***	3.7	7.6***	3.3	26.7***	0.007	19.8***	0.004	24.9***	0.006
Xgwm3	3D	10.0***	3.9	10.1***	3.6	10.8***	3.9	13.3***	0.005	7.1***	0.002	11.6***	0.004
Xbcd1871	5A	7.9***	–3.3	7.1***	–2.9	8.0***	–3.1	7.5***	–0.004	14.0***	–0.003	10.7***	–0.004
Xgwm134	5B	4.2**	2.4										
Xmta17	1B	4.4**	–2.6										
Xfbb274	7A							4.6**	–0.003	4.4**	–0.002	5.6**	–0.002

** and ***, significant, respectively, at the 0.01 and 0.001 probability levels
[a] The sign of additive value corresponds to the parent with the favorable allele :+ for 'Renan' and – for 'Récital'

Table 3 Results of bootstraps on 'marker regression' for QTLs to pre-harvest sprouting

Chromosome	1999			2000			Mean		
	P[a]	Additive value[c]	Location on chromosome (cM)	P	Additive value	Location on chromosome (cM)	P	Additive value	Location on chromosome (cM)
3A	0.52	1.0<2.5<4.8	128<134<182	0.88	0.8<2.6<4.8	125<127<145	0.85	1.2<3.2<5.4	125<130<158
3B	0.90	2.4<4.4<6.7	77<98<107	0.89	2.2<4.0<6.0	78<101<119	0.84	2.0<3.9<6.0	76<98<110
3D	0.92	1.6<4.5<7.5	128<152<177	0.96	1.5<3.7<6.2	140<156<171	0.97	1.7<4.3<6.6	146<155<166
5A	0.60	–4.5<–2.5<–0.9	17<35<58	0.48	–3.5<–1.6<0.8	19<40<64	0.68	–3.8<–1.8<0.1	24<40<67
R[b]	25.2%			30.3%			27.2%		

[a] P=Percentage of significant models using bootstrap re-sampling
[b] R=Part of phenotypic value explained by multiple regression with all QTLs
[c] The sign of additive value correspond to the parent with the favorable allele :+ for 'Renan' and – for 'Récital'

were very high (0.89 for PHS, 0.72 for grain color). Therefore, the detection of QTLs was done on the results of the 2 years independently, as well as on the average over the 2 years.

Figure 1 shows the distribution of the 2-years average for the different variables obtained with the chromameter and the percentage of germination. The components of color obtained with the chromameter appeared to fit a normal distribution, while the percentage of germination was skewed, most of the RILs having low values for PHS.

For the study of red pigmentation of the testa, the 'a' value of the chromameter seemed to be the most appro-

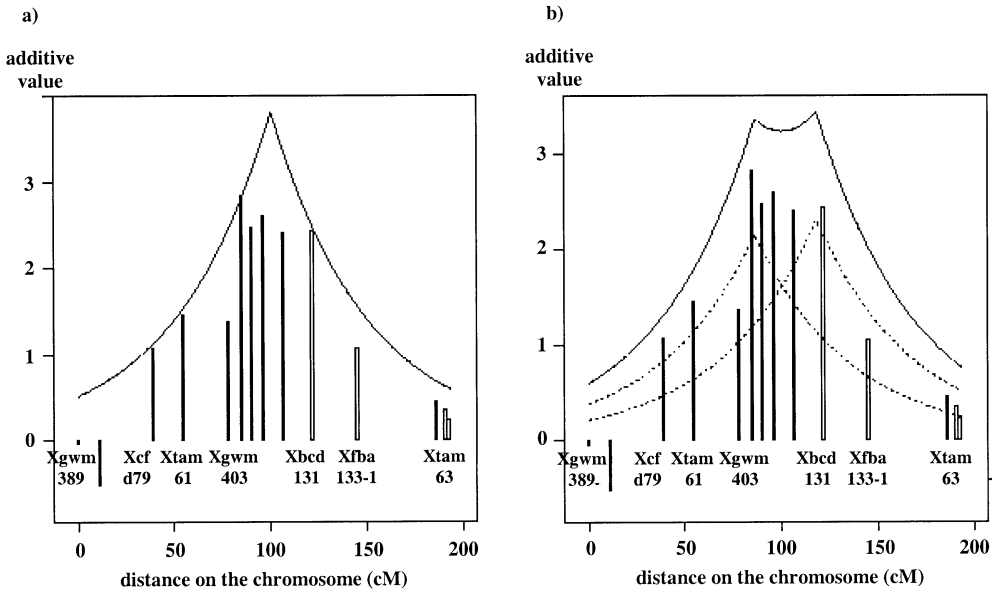

Fig. 3 Marker regression on the chromosome 3B for PHS, (**a**) with a one-QTL model, (**b**) with a two-QTL model. Model 2QTLs/1QTL: *F* value=226.5 ($P<10^{-6}$)

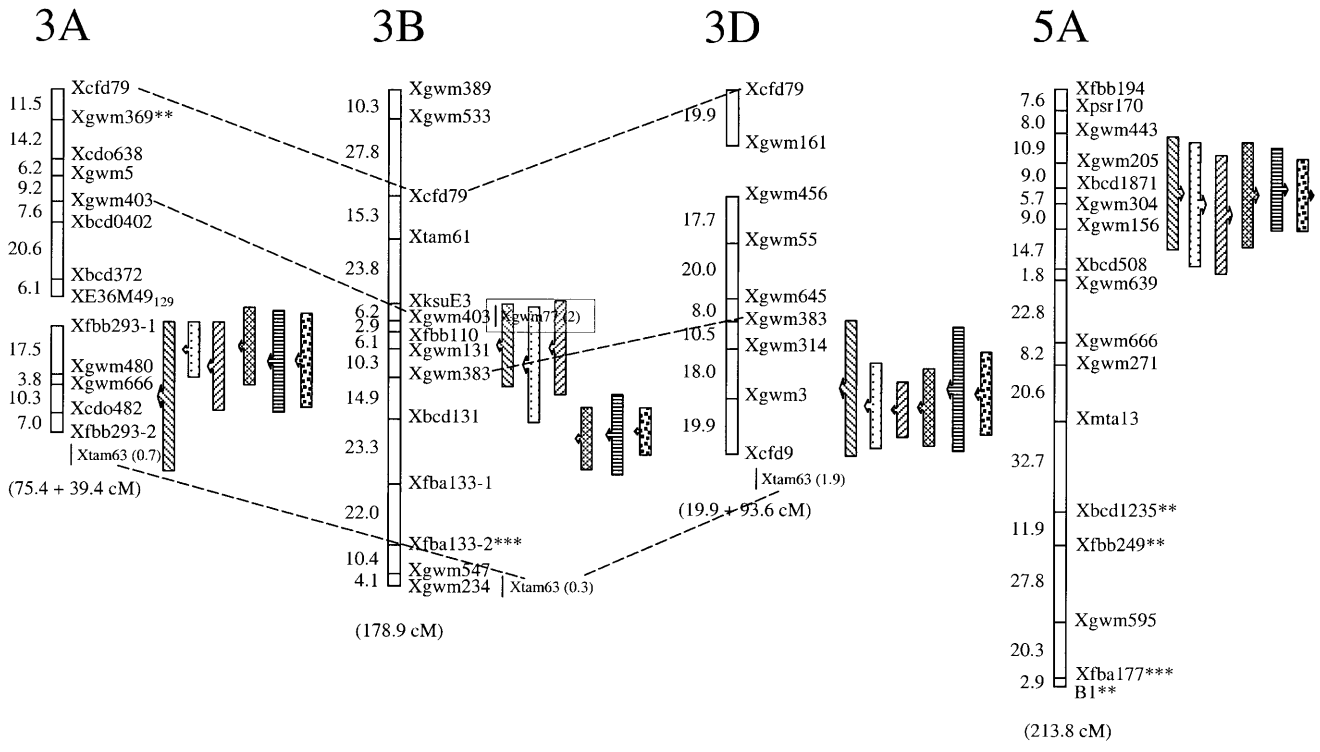

Fig. 4 Locations of QTLs for PHS and grain color in the population 'Renan'×'Récital'. PHS 1999; grain color 1999; PHS 2000; grain color 2000; PHS mean; grain color mean; – relation between homeologous loci. ** and ***: markers which deviated from the ratio 1:1 with $P<0.05$ and 0.01, respectively; the favorable allele is borne by 'Renan' when the *arrow* is on the left, by 'Récital' when it is on the right; the length of the box corresponds to the length of the confidence interval of the QTL

priate because it measures the 'red' color. However, the brightness of the grain could modify the measure of the a component. Therefore, in this study, we have analyzed the a/L ratio.

The relation between PHS and a/L was quite strong in this population as shown in Fig. 2 for the 2 years average ($R^2=0.57$). Most RILs which have a high sensibility for PHS show a value of a/L below a threshold of 0.12 and have white grains.

Table 4 Results of bootstraps on 'marker regression' for QTLs to Grain Color

Chromosome	1999			2000			Mean		
	P[a]	Additive value	Location on chromosome (cM)	P	Additive value	Location on chromosome (cM)	P	Additive value	Location on chromosome (cM)
3A	0.82	0.001<0.004<0.006	121<130<148	0.89	0<0.002<0.004	122<129<159	0.88	0.001<0.003<0.004	123<129<157
3B	1	0.005<0.008<0.01	114<120<139	1	0.002<0.004<0.006	109<124<138	1	0.004<0.006<0.008	114<121<131
3D	0.98	0.003<0.006<0.009	142<153<170	0.82	0.002<0.003<0.005	129<157<174	0.99	0.003<0.005<0.007	138<156<168
5A	0.49	–0.004<–0.002<0	19<37<57	0.96	–0.005<–0.003<–0.002	21<38<51	0.85	–0.005<–0.003<–0.001	25<36<51
R	41.1%			38.8%			43.1%		

[a] P=Percentage of significant models using bootstrap re-sampling
[b] R=Part of phenotypic value explain by multiple regression with all QTLs
[c] The sign of additive value correspond to the parent with the favorable allele: + for 'Renan' and – for 'Récital'

Detection of QTLs

For PHS

Using ANOVA, we identified seven markers significantly ($P<0.01$) linked with PHS (Table 2). Among these seven markers, five were found for the 2 years, and for the mean the others were detected only in 1999. These markers each explained between 4.2 and 11.4% of the phenotypic variance. By the 'marker-regression' method, we tested the accuracy of the putative QTLs by bootstrapping. The results are given in Table 3. The remaining QTLs were mapped on chromosomes 3A, 3B, 3D and 5A, and altogether explained about 30% of the phenotypic variance. On chromosomes 3A, 3B and 3D, the alleles for resistance to PHS come from 'Renan', whereas the positive allele on chromosome 5A was brought by the susceptible parent, 'Récital'. The QTLs on chromosomes 1B and 5B, detected only in 1999, appeared to be inconsistent through bootstraps and are not conserved in Table 3.

On chromosome 3B, the test of a two-QTL model was significant against a one-QTL model for the 2 years and for the mean with the full dataset. The location for these two QTLs are shown in Fig. 3 for 1999. However, because of the proximity of these two QTLs and the limited population size, the confident intervals of each of the two QTLs obtained by bootstrapping are very large, together covering most of the chromosome, and overlapped in 1999 and for the mean.

For grain color

Five markers were detected as having significant effects (with $P<0.01$) on a/L (Table 2). They were all detected for the 2 years and for the mean, and individually explained between 4.4% and 26.7% of the phenotypic variance of grain color. The results of marker-regression are shown in Table 4. Altogether, the five QTLs explained about 40% of the variation of grain color. The QTL on 7A appeared not to be consistent after bootstrapping and was not conserved in Table 4, although it was detected in the 2 years. The QTL on chromosome 5A was most influenced by year; in 1999 it was detected in only half the bootstrap samples. The QTLs on the chromosomes from group 3 were all consistent but their effects appeared unequal: the QTL on chromosome 3B appeared to have the strongest effect. 'Renan' has positive alleles for red color for the three QTLs of group 3, while the positive allele for the QTL on the chromosome 5A is brought by 'Récital'. All these QTLs for grain color co-located with QTLs for PHS. The results are summarized in Fig. 4.

Discussion

Using color components obtained by a chromameter to estimate grain color, we detected QTLs on chromosomes 3A, 3B and 3D (Fig. 4). The locations of these QTLs correspond to the locations of the wheat *R* loci. Indeed,

the *R-B1* locus has been mapped at 5 cM from the locus *Xbcd131* (Nelson et al. 1995), located in our map on chromosome 3B (Fig. 4). The most-likely location of the QTL on the chromosome 3B fits with this locus, and it can be assumed that this QTL corresponds to the *R-B1* locus. Using the probes showing homoeologous loci on the three chromosomes of group 3, such as TAM63 and GWM383, we can also postulate that the QTLs on chromosomes 3A and 3D correspond to the *R-A1* and *R-D1* loci, respectively. *R-A1* may be located in the gap on the map. Unfortunately, no polymorphic marker between the two parents has yet been found in this chromosome region. Thus, we can suggest that 'Renan' has the *R-A1b*, *R-B1b* and *R-D1b* alleles, responsible for the red grain color, whereas 'Récital' has the recessive 'white' alleles at the three loci. Moreover, the ratio of white on red grains RILs (19/166) fits the expected value for three independently segregating genes [1:7, $P(\chi_2)$=0.36]. No significant interaction between the QTLs was identified; hence the effects of the R dominant alleles seems to act essentially as additive factors.

The last QTL detected for grain color was located on chromosome 5AS and the positive allele comes from 'Récital'. The effect of this QTL was smaller than those of the *R* genes and was more influenced by year. This QTL probably has an effect on the pericarp, independently of the red pigmentation, which modifies the measure of the grain color by the chromameter.

The four QTLs detected for resistance to PHS all co-located with QTLs for grain color. Three of them were located on the long arm of chromosomes of group 3, close to the position of the *R* loci. These QTLs could be due to pleiotropic effects of the *R* genes or to the effect of the gene *taVp1*, mapped at 30 cM from the *R* loci (Bailey et al. 1999). On chromosomes 3A and 3D, the confidence intervals of the QTLs for PHS and grain color largely overlapped; thus, we cannot exclude the hypothesis that the resistance to PHS on these chromosomes was just due to a pleiotropic effect of *R-A1* and *R-D1*. Moreover the two-QTL models tested on these chromosomes were not significant. The effect of the QTL on chromosome 3A was probably underestimated due to the gap in this region of the map.

On chromosome 3B, the QTLs for PHS and grain color were not so close, although their confidence intervals slightly overlapped (Fig. 4). For PHS, a two-QTL model was found to be significant on this chromosome for the 2 years and for the mean (Fig. 3). Using this model, one QTL was located close to the locus *Xgwm403*, the other close to the locus *Xbcd131*. This latter QTL corresponds to the location of the *R-B1* loci. The first one could be due to an action of the gene *taVp1*. On a consensus map, Bailey et al. (1999) located *taVp1* (Xlars10) and Xbcd131, respectively at 30 cM and 60 cM from the centromere. By comparison with the map of Röder et al. (1998), the position of the centromer corresponds in our map to the position of Xgwm77 (Fig. 4). Hence in this area, our population shows fewer recombination events (30 cM between the centromer and Xbcd131 against 60 cM), and the gene *taVp1* could be putatively located between Xgwm131–3B and Xgwm183–3B. It is then difficult to conclude whether the second QTL on 3B is really due to *taVp1*, and the proximity of the putative position of the two genes limits the possibility of studying recombination between them in our population. On chromosome 3B, the resistance to PHS of 'Renan' is caused by simultaneous effects of the gene *R-B1* and of another gene. Further analyses are needed to check whether the second QTL is due to *taVp1* or to another gene.

The last QTL for PHS, on 5AS, has not been described in other studies on PHS (Anderson et al. 1993; Roy et al. 1999). In a cross between wheat and spelt, Zanetti et al. (2000) have found a QTL for alpha-amylase activity on chromosome 5A but located on the long arm. By hybridization, Cadle et al. (1994) have mapped an abscisic-acid responsive gene, *pMA1951*, on the short arm of group 5. Since abscisic acid is involved in embryo morphogenesis and seed dormancy (Flintham and Gale 1988) it may be suggested that the QTL found on chromosome 5A is due to an action of *pMA1951* on embryo dormancy. On the other hand, this QTL co-located with a QTL for grain color for which 'Récital' has the positive allele. We can then suppose that this resistance to PHS is due to a pigmentation not caused by the *R* genes and which has an effect on the structure of the pericarp, allowing a certain resistance to PHS for 'Récital' by a 'coat-imposed' mechanism of dormancy. The effect of this QTL could be on the vitreousness of the grain, which modifies the exterior aspect of the grain.

The selection of bread wheats resistant to PHS is one of the aims of plant breeding in wheat. Some varieties with red-grain are susceptible to PHS. In England, where only red grain varieties are used, PHS has often strong effects on wheat quality (Flintham and Gale 1988). One of the reason could be that these susceptible varieties have only one (or two) dominant(s) allele(s) at the *R* loci. Thus it is essential for plant breeders to determine the number of dominant alleles at the *R* loci in the different varieties. The use of a chromameter allows the determination of a value for grain color but it would be difficult with this measure to know exactly the number of dominant *R* alleles. Another way to achieve this could be to use locus-specific markers for the *R* loci. Adlam and Flintham (1999) have determined PCR-markers closely linked to the *R-A1*, *R-B1* and *R-D1* loci. These markers could be useful: (1) to determine the number of dominant alleles at the *R* loci in current varieties, and (2) to introduce, by marker-assisted selection, dominant alleles in varieties of agronomic and end-use quality interest, which are insufficiently resistant to PHS because of the presence of one (or two) recessive allele(s) at the *R* loci.

The cumulating dominant alleles for red color is not the only possibility to improve resistance to PHS and appears not always to be sufficient, since Flintham et al. (1999) reported a variety, with three dominant alleles for

R genes, which was susceptible to PHS. The QTLs detected in the different studies in wheat (Anderson et al. 1993; Roy et al. 1999) will be useful for the improvement of wheat for resistance to PHS. In our study, we detected a QTL on chromosome 5AS which allows a certain resistance to PHS in the susceptible parent. With this QTL, some RILs of our cross with red grain were completely resistant while 'Renan' showed an incomplete resistance to PHS. Moreover, in our population, some white-grain wheat lines are less susceptible to PHS than 'Récital' (Fig. 2). These lines could have the favorable allele at the second QTL on 3B, possibly associated to *taVp1*, and not the *R*-B1b allele. These different QTLs, not related to red-grain color, could be of particular interest for the breeding of white-grain wheat tolerant to PHS. These wheats are used for the production of noodles in Asia, where PHS can be very strong; and obtaining white wheat resistant to PHS is of first importance for plant breeders in this region.

More investigations are needed to dissect the QTLs on chromosome 3B, particularly to determine the possible role of *taVp1* as well as its precise position, and to develop specific markers for these QTLs, which could prove very useful for plant breeders.

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