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Mixed-model QTL mapping for kernel hardness and dough strength in bread wheat

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Abstract Plant breeding data comprise unbalanced phenotypic data for inbreds with complex pedigrees. As traditional methods to map quantitative trait loci (QTL) cannot exploit plant breeding data, an alternative approach is QTL mapping via a mixed-model procedure. Our objective was to validate mixed-model QTL mapping for self-pollinated crops by detecting QTL for kernel hardness and dough strength from data in a bread wheat (*Triticum aestivum* L.) breeding program. We studied 80 parental and 373 experimental inbreds genotyped for 65 simple sequence repeat (SSR) markers and three candidate loci. The methodology involved three steps: variance component estimation, single-marker analyses, and a final multiple-marker analysis with marker effects treated as fixed effects. Two QTLs for kernel hardness were detected on chromosomes 1A (close to candidate locus *GluA3*) and 5D (close to candidate locus *Ha*). Four QTLs were detected for dough strength on chromosomes 1A, 1B, 1D, and 5B. Candidate gene *GluA1*, which was associated with dough strength, was the only candidate locus found significant. Results were consistent with previously reported markers and QTLs associated with kernel hardness and dough strength. Unlike previous studies that have assumed QTL effects as random, the assumption of fixed marker effects identified the favorable marker alleles to select for. We conclude that the detection of previously mapped QTL validates the usefulness of mixed-model QTL mapping in the context of a plant-breeding program.

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Introduction

Improved bread quality is an important goal in wheat (*Triticum aestivum* L.) breeding. Kernel hardness and dough strength are two important traits affecting bread quality. A better understanding of the genetic control of these and other bread quality traits would enhance the development of superior bread wheat cultivars.

At least 13 quantitative trait loci (QTL) and nine candidate genes, mostly on chromosomes 1 and 5, have been reported for kernel hardness and dough strength in wheat (Singh and Shepherd 1988a, b; Branlard et al. 2001; Campbell et al. 1999, 2001; Perretant et al. 2000). Studies to map these QTL have used designed mapping populations, such as F_2- or backcross-derived inbred progenies or double haploids. Data routinely generated in wheat breeding programs, however, have been underutilized in gene mapping for two reasons. First, wheat inbreds in a breeding program have complex pedigrees, that is, inbreds are developed from different crosses and have different levels of relatedness. Second, plant breeding data are highly unbalanced, as inbreds are evaluated in different sets of environments.

Mixed models can account for relationships among inbreds and for unbalanced data, and can incorporate marker data (Parisseaux and Bernardo 2004). A mixed-model procedure represents an in-silico approach for gene mapping because it exploits phenotypic and genomic databases that are already available (Grupe et al. 2001). To avoid confusion with other in-silico mapping procedures (e.g., for ESTs), we refer to this approach as mixed-model QTL mapping (Arbelbide et al. 2006). Mixed-model QTL mapping has been recently studied in hybrid crops (Parisseaux and Bernardo 2004; Yu et al. 2005) and in self-pollinated crops (Arbelbide et al. 2006). The power of mixed-model QTL mapping in hybrid and self-pollinated crops was found comparable to that of other QTL mapping methods. For self-pollinated crops, power to detect QTL ranged from 0.4 to 47% depending on population size, number of QTL, heritability of the

trait, and significance thresholds used (Arbelbide et al. 2006).

Different mixed models have been proposed to map QTLs in complex pedigrees. The random model approach requires a measure of whether QTL alleles in two different inbreds are copies of the same ancestral QTL allele, that is, identical by descent (Xu and Atchley 1995). Because QTL alleles are not observable, the probability that they are identical by descent needs to be estimated from information on linked markers and from pedigree records. Crepieux et al. (2004) proposed an identity by descent QTL mapping method using plant breeding data for self-pollinated crops. Crepieux et al. (2005) used this method to identify one QTL for kernel hardness and two QTLs for dough strength from data available in a wheat breeding program. The random model approach estimates a variance component associated with the QTL and identifies the marker interval that most likely contains the QTL. This approach allows a better evaluation of the overall breeding value of an inbred and the identification of genomic regions associated with the trait. However, it does not lead to estimates of the mean effect associated with a specific marker allele linked to a QTL. The random model approach therefore does not allow the identification of the favorable QTL alleles for selection.

In contrast, considering markers as fixed effects allows the estimation of an effect for each marker allele. This inherently identifies the favorable marker alleles to select for and the inbreds that most likely have the favorable alleles at specific QTL. This approach is a first step towards gene discovery, particularly if the markers represent functionally neutral loci, such as simple sequence repeats (SSR). If the marker locus is itself a candidate gene, the analysis would provide direct information on the actual locus affecting a quantitative trait.

In this study, we analyzed the data of Crepieux et al. (2005) with a model assuming fixed marker effects rather than random QTL effects. The objective was to validate this mixed-model QTL mapping methodology for self-pollinated crops by detecting QTL for kernel hardness and dough strength from data in a wheat-breeding program.

Materials and methods

Mapping population, pedigree, and marker data sets

We studied 80 parental and 373 experimental inbreds from a Limagrain Genetics wheat breeding program (Crepieux et al. 2005). The experimental inbreds were derived from 158 F_2 populations among the 80 parents, corresponding to an average of 2.4 experimental inbreds per F_2 population. All inbreds were genotyped for 65 SSR markers corresponding to linkage groups 1A, 1B, 1D, 5A, 5B, and 5D. These linkage groups are known to contain QTLs and candidate genes for bread quality traits (Singh and Shepherd 1988a, b; Perretant et al. 2000). The inbreds were also genotyped for three biochemical markers corresponding to candidate loci for endosperm storage protein subunits *GluA1* (chromosome 1A), *GluB1* (chromosome 1B), and *GluD1* (chromosome 1D) (Fig. 1; Singh and Shepherd 1988a, b). In addition, the remaining chromosomes were genotyped for 46 SSRs that were used to estimate genetic relationships based on genome-wide marker similarity. Marker locations were obtained from previously published maps (Crepieux 2004).

Phenotypic data

The 373 experimental inbreds were evaluated for kernel hardness and dough strength in 2002 (Crepieux et al. 2005). A total of 339 inbreds were evaluated at a Limagrain Genetics breeding station near Clermont-Ferrand, France, and 62 inbreds were evaluated at Chartainvilliers (close to Paris). There were 28 inbreds in common between the two locations. Kernel hardness was evaluated on a score of 1 to 100 (1 = very soft, 100 = very hard) by near-infrared reflectance spectroscopy. Dough strength (W in J 10^{-4}) was evaluated by alveograph tests.

Mixed-model analysis

The data available for the mixed-model analysis consisted of phenotypic data on the inbreds, marker information on 114 markers for each inbred, and pedigree records describing the relationship among inbreds. The linear model was:

$$\mathbf{y} = X\boldsymbol{\beta} + \mathbf{Zu} + \mathbf{Tv} + \mathbf{Wm} + \mathbf{e}$$

where $\mathbf{y}$ = 401 × 1 vector of phenotypic observations; $\boldsymbol{\beta}$ = 2 × 1 vector of fixed effects associated with locations; $\mathbf{u}$ = 373 × 1 vector of random polygenic effects; $\mathbf{v}$ = 746 × 1 vector of random genotype by environment effects; $\mathbf{m}$ = $\mathbf{m}'$ × 1 vector of fixed effects associated with the alleles at each marker locus for a subset of m′ markers; $\mathbf{e}$ = 401 × 1 vector of residual effects associated with each observation; and $\mathbf{X}$, $\mathbf{Z}$, $\mathbf{T}$, and $\mathbf{W}$ are incidence matrices of ones and zeros relating $\mathbf{y}$ to $\boldsymbol{\beta}$, $\mathbf{u}$, $\mathbf{v}$, and $\mathbf{m}$, respectively. A phenotypic observation constituted the mean kernel hardness or dough strength of an inbred at a particular location. Polygenic effects corresponded to the sum of genotypic effects not associated with the marker or markers being considered in the model. The random vectors $\mathbf{u}$, $\mathbf{v}$, and $\mathbf{e}$ had means of zero and variances $\mathrm{Var}(\mathbf{u}) = \mathbf{G_1}V_G$, $\mathrm{Var}(\mathbf{v}) = \mathbf{G_2}V_{GE}$, and $\mathrm{Var}(\mathbf{e}) = \mathbf{R}V_R$. $\mathbf{G_1}$ is the additive relationship matrix, and V_G is the additive variance at the polygenic effects; $\mathbf{G_2}$ is the additive genotype by environment relationship matrix, and V_{GE} is the genotype by environment genetic variance at the polygenic effects; $\mathbf{R}$ is a 401 × 401 diagonal matrix with elements equal to the

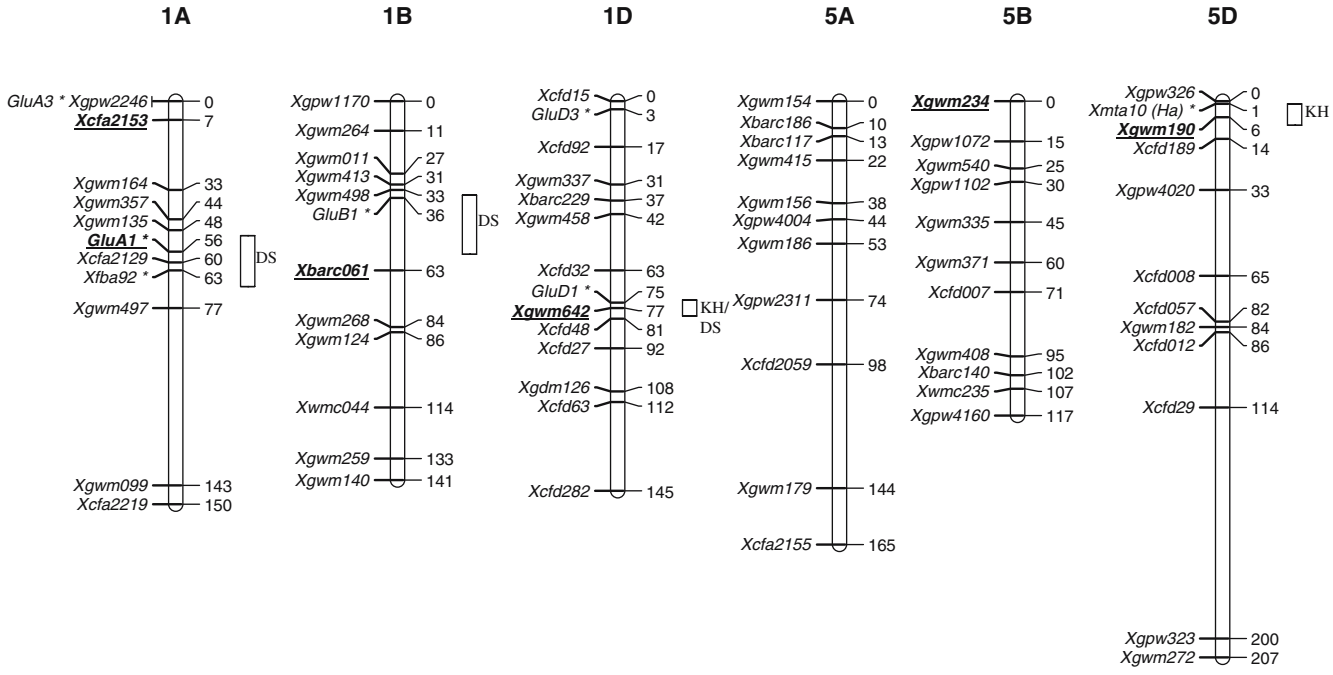

Fig. 1 Genetic map of 65 SSR loci and three candidate loci on homoeologous linkage groups 1 and 5 of wheat (adapted from Crepieux 2004). Significant markers are indicated in *bold* and *underlined*. QTL reported by Crepieux (2004) are indicated by *sidebars* (*KH* kernel hardness; *DS* dough strength). Loci previously reported to have significant effects are indicated by an *asterisk*

inverse of the number of locations of a particular inbred, and V_R is the residual variance. The relationship matrix $\mathbf{G_1}$ comprised twice the coefficient of coancestry among inbreds. The coefficients of coancestry were estimated using the tabular method (Emik and Terrill 1949). Parental contributions were estimated based on marker similarity between a parent and a derived inbred, adjusted by the proportion of the marker similarity between the parents as described by Bernardo et al. (2000). The coefficient of coancestry between inbreds ranged from 0.18 to 0.96, with a mean of 0.47. The relationship matrix $\mathbf{G_2}$ was an identity matrix, assuming that genotype by environment effects were uncorrelated among inbreds and locations. Marker effects were considered fixed as proposed by Kennedy et al. (1992). Mixed-model equations were solved as described by Arbelbide et al. (2006) to obtain BLUE values of fixed effects $\boldsymbol{\beta}$ and $\mathbf{m}$, and BLUP values of random effects $\mathbf{u}$ and $\mathbf{v}$. Confidence intervals ($P < 0.05$) were constructed for $\hat{\mathbf{m}}$, where $\mathrm{Var}(\hat{\mathbf{m}}) = C_{11}V_R$ as described by Henderson (1984). Restricted maximum likelihood estimates of V_R, V_G, and V_{GE} were obtained as described by Henderson (1984, p. 200).

Data analysis

Data analysis comprised three steps as described by Arbelbide et al. (2006): variance component estimation, single-marker analyses at $P < 0.05$, and multiple-marker analysis at $P < 0.001$. If adjacent markers were significant in the single-marker analyses, only the marker with the smallest P-value was selected to reduce multicollinearity in the model. Marker selection in the multiple-marker model was conducted by backwards elimination, and a stringent significance level of $P < 0.001$ was used to control the false discovery rate for QTL (Bernardo 2004). Equations were solved to obtain new estimates of V_G, V_{GE}, and V_R, and BLUE values of marker effects. Assuming only additive genetic effects, the marker effects were estimated as the maximum difference between marker allele effects (Parisseaux and Bernardo 2004). We considered this criterion meaningful to plant breeders, who are most interested in the extremes in a given population. Broad-sense heritability was estimated as $H = V_G/V_P$, where V_P is the phenotypic variance on an entry mean basis.

Results

The average number of alleles per marker was 5.6 (Table 1), with a minimum of two and a maximum of 19 alleles per marker. The number of alleles per candidate locus was three for *GluA1*, eight for *GluB1*, and three for *GluD1*. The map distance between a candidate locus and its nearest SSR marker was 3–4 cM (Fig. 1). Kernel hardness had a mean score of 69.8, and ranged from 15.0 to 119.0 with a standard deviation of 17.8. Dough strength had a mean of 215.9 J 10^{-4}, and ranged from 53.0 to 541.0 with a standard deviation of 84.6. Broad sense heritability was 0.82 and 0.62 for kernel hardness and dough strength, respectively.

Table 1 Number of SSR markers, average number of alleles per marker, and average distance among markers for wheat linkage groups 1 and 5 (adapted from Crepieux 2004)

Chromosome	Length (cM)	Number of markers	Average number of alleles per marker	Average distance between markers (cM)
1A	150	10	5.8	15.0
1B	141	12	7.1	11.8
1D	145	13	4.1	11.2
5A	165	11	6.1	15.0
5B	117	11	5.5	10.6
5D	207	11	5.1	18.8
Total	925	68	5.6	13.7

For kernel hardness, a total of five markers were found significant in the single-marker analyses at $P < 0.05$: *Xgpw2246* and *Xcfa2153* on chromosome 1A; *GluD1* on chromosome 1D; and *Xgwm190* and *Xgwm272* on chromosome 5D. Only two markers remained significant after backwards elimination in the multiple-marker analysis: *Xcfa2153* and *Xgwm190* (Fig. 1, Table 2). Both markers had similar estimated effects. Candidate locus *GluD1* was therefore significant ($P < 0.02$) only in the single-marker analyses, whereas candidate loci *GluA1* and *GluB1* were not significant in any step.

For dough strength, a total of 20 markers were found significant in the single-marker analyses: *Xgwm164*, *Xgwm357*, *GluA1*, *Xcfa2129*, and *Xcfa2219* on chromosome 1A; *Xgpw1170*, *Xgwm264*, *GluB1*, *Xbarc061*, and *Xwmc044* on chromosome 1B; *Xcfd32*, *GluD1*, *Xgwm642*, *Xcfd48*, *Xcfd27*, *Xgdm126*, and *Xcfd63* on chromosome 1D; *Xbarc117* on chromosome 5A; *Xgwm234* on chromosome 5B; and *Xgpw323* on chromosome 5D. Only four of these markers remained significant in the multiple-marker analysis: *GluA1*, *Xbarc061*, *Xgwm642*, and *Xgwm234* (Fig. 1, Table 2). The estimate of marker effects was highest for *Xbarc061*, lowest for *GluA1*, and intermediate for *Xgwm642* and *Xgwm234*. The three candidate loci therefore showed highly significant associations with dough strength in the single-marker analyses, but only *GluA1* was significant in the multiple-marker model.

Discussion

In this study, 373 inbreds developed from 158 different crosses in a wheat-breeding program were used to validate candidate loci and identify SSR markers for kernel hardness and dough strength. The methodology involved three steps: variance component estimation; single-marker analyses at $P < 0.05$; and multiple-marker analysis at $P < 0.001$ with selected markers found significant in the single-marker analyses. As we discussed in a companion article (Arbelbide et al. 2006), single-marker analyses prior to multiple-marker analyses prevented overparameterization in the model (i.e., the number of marker effects exceeding the number of equations). The mixed-model procedure easily incorporated information from phenotypic, marker and pedigree records. Despite the relatively small population size, a total of six different markers were identified as associated with kernel hardness or dough strength. The comparisons below show that these results were consistent with those from previously reported experiments.

Two markers were found associated with kernel hardness. Of these, *Xcfa2153* showed the stronger evidence for association. This marker has not been previously associated with kernel hardness, but it has been reported to be located close to the end of the long arm of chromosome 1A (Paillard et al. 2003), where candidate locus *GluA3* has also been reported (Somers et al. 2004). We speculate that variation for kernel hardness due to *GluA3* was captured by a significant association with marker *Xcfa2153*, because *Xcfa2153* is 1–7 cM from the end of chromosome 1A (Paillard et al. 2003). Marker *gpw2246*, which was reported at the same location as *GluA3*, was found significant only in the single-marker analyses. Likewise, candidate locus *GluD1* was significant ($P < 0.05$) in the single-marker analyses but not in the multiple-marker model.

We also found marker *Xgwm190* significant for kernel hardness. This locus has been previously mapped to the end of chromosome 5D (Röder et al. 1998; Paillard et al. 2003; Somers et al. 2004). Despite not having been previously reported as associated with kernel hardness, *Xgwm190* has been mapped very close to the *mta10*

Table 2 Significant marker–trait associations from multiple-marker models for kernel hardness and dough strength in wheat

Marker	Chromosome	Number of alleles	Additive effect[a]	*P*-value
Kernel hardness				
Xcfa2153	1A	10	29.8 (23.9, 35.7)[b]	4.0E-05
Xgwm190	5D	5	35.2 (27.2, 43.2)	6.2E-04
Dough strength				
GluA1	1A	3	12.2 (3.7, 20.7)	6.5E-08
Xbarc061	1B	8	126.6 (114.3, 138.9)	5.8E-12
Xgwm642	1D	4	75.8 (66.0, 85.6)	3.1E-17
Xgwm234	5B	7	47.6 (31.7, 61.5)	2.9E-04

[a]Maximum difference between effects of marker alleles
[b]95% confidence intervals in parentheses

locus, which has been reported strongly associated with kernel hardness and linked to grain hardness locus *Ha* (Perretant et al. 2000; Fig. 1). Marker *Xgwm190* had the highest estimated effect, which was consistent with results reported for *mta10* and other loci linked to the major locus *Ha* (Sourdille et al. 1996; Perretant et al. 2000; Campbell et al. 2001; Charmet et al. 2001).

For dough strength, candidate locus *GluA1* showed strong association (Table 2, Fig. 1). This result was in agreement with those of Perretant et al. (2000) who reported association of a nearby locus, *fba92*. Campbell et al. (2001) found similar results where locus *GluA1* was associated with mixograph peak height, a dough strength parameter that describes the torque applied to the dough sample at maximum resistance. Candidate locus *GluB1* was selected in the single-marker analyses but eliminated in the multiple-marker analysis. Marker *Xbarc061* on chromosome 1B showed strong association with the trait and had the highest estimated effect. Marker *Xbarc061* is located near the proximal end of chromosome 1BL (Crepieux et al. 2005). Crepieux et al. (2005) reported a QTL for dough strength in the interval between *Xbarc061* and *GluB1*. Results showed that when *Xbarc061* was in the model, candidate locus *GluB1* was no longer significant. We speculate that *Xbarc061* accounted for variation due to the joint effects of *GluB1* and other possible QTL in the region.

Marker *Xgwm642* on chromosome 1DL had an intermediate effect on dough strength but had the strongest association with the trait. It is located at 2.5 cM from the candidate locus *GluD1*, a grain protein storage gene (Singh and Shepherd 1988a, b; Boeuf et al. 2003; Fig. 1). *GluD1* has been previously associated with mixograph peak time, which measures time to maximum dough resistance (Campbell et al. 2001). In this study, *GluD1* was found significant in the single-marker analyses only, and was not as strongly associated with dough strength as marker *Xgwm642*. In contrast, Perretant et al. (2000) reported a QTL for dough strength associated with locus *mta10* (*GluD3*), which is located near the end of chromosome 5DS and (as previously mentioned) has been associated with kernel hardness. Perretant et al. (2000) did not find a significant effect for *GluD1* due to lack of polymorphism among the parental lines used in their experiment.

Marker *Xgwm234* on chromosome 5B was found significantly associated with dough strength. This marker has been reported at the 0 cM position (Röder et al. 1998; Paillard et al. 2003; Crepieux et al. 2005) and at the 38 cM position (Somers et al. 2004) of chromosome 5BS. These results are in agreement with Zanetti et al. (2001) who reported a QTL for dough extensibility at the end of chromosome 5BS, and a minor QTL for dough strength closer to the centromere.

Branlard et al. (2001) and Perretant et al. (2000) reported correlations between kernel hardness and dough strength. In this study the phenotypic correlation between the two traits was 0.45, but QTLs associated with both the traits were found only in the single-marker analyses (data not shown). Crepieux et al. (2005) reported a QTL for kernel hardness and dough strength in the interval flanked by markers *GluD1* and *Xgwm642* on chromosome 1D. This QTL was at the 76 cM position. In this study, candidate locus *GluD1* had only weak association with kernel hardness. Also, Crepieux et al. (2005) reported a QTL for kernel hardness at the 3 cM position (in the interval flanked by markers *gpw326* and *Xgwm190*) on chromosome 5D. Marker *Xgwm190* was identified as having a significant effect. Results agree with those of Crepieux et al. (2005) who used a random effects model for QTLs. The model considered marker effects as fixed and was able to identify the same markers as those reported as flanking markers by Crepieux et al. (2005), plus some others in regions known to be associated with either kernel hardness or dough strength.

In this study three candidate loci were tested for association with kernel hardness and dough strength. However, only one of these three candidate loci was found significant for either trait. In contrast, adjacent SSR loci were found significant. The reasons for the significance of adjacent SSR loci rather than of the candidate loci themselves are unknown. We speculate, however, that these SSR markers could have detected the effects of unknown QTL close to these known candidate genes, especially that families of similar genes are known to be found in clusters in the genome (Gill et al. 1996a, b; Dilbirligi et al. 2004). Further analysis with a larger population and with a higher density of markers in these genomic regions would be needed to confirm this speculation.

Previous experiments to detect QTL for kernel hardness and dough strength have used parental inbreds with contrasting phenotypes. Such experiments maximize variation for the trait and maximize the chance of finding significant marker–trait associations. However, given the nature of the crosses, results for discovered QTLs may not necessarily apply to elite breeding populations. Such wide crosses are unlikely in applied wheat-breeding programs, where major genes are likely to have become fixed in elite germplasm and only minor, less heritable QTL would remain segregating. In contrast, mixed-model QTL mapping utilizes elite inbreds developed in the breeding program to identify marker–trait associations. Results from this approach would therefore be directly applicable to the breeding program. Empirical studies are needed to determine the level of kernel hardness and dough strength that can be attained by pyramiding the favorable QTL alleles in a single wheat inbred. Such empirical studies would also reveal any epistasis, influence of genetic background, unfavorable linkage drag, or pleiotropy among the favorable QTL alleles.

Plant breeding data comprise massive phenotypic and genotypic information generated through heavy investments in cultivar development for many years. This implies that breeding data consist of many generations with significant accumulation of historical recombinants and a large number of progenies. The use of dense

marker maps would enable higher resolution in the QTL mapping analysis. Once QTL regions are identified, mixed-model QTL mapping can easily incorporate single-nucleotide polymorphism (SNP) data to dissect candidate genes in large populations. By using a fixed effects approach, SNP alleles or haplotypes with favorable effects can be directly identified. This comprehensive approach to QTL mapping enables the joint exploitation of plant breeding data and genomic resources, resulting in a better leverage of resources invested in cultivar development and genomics research.

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