Theor Appl Genet (2000) 100:1217–1224

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Location and mapping of the powdery mildew resistance gene *MlRE* and detection of a resistance QTL by bulked segregant analysis (BSA) with microsatellites in wheat

Received: 30 August 1999 / Accepted: 11 November 1999

Abstract Powdery mildew (*Blumeria graminis* f. sp. *tritici*) is one of the most damaging diseases of wheat (*Triticum aestivum*). The objective of this study was to locate and map a recently identified powdery mildew resistance gene, *MlRE*, carried by the resistant line RE714 using microsatellites uniformly distributed among the whole genome together with a bulked segregant analysis (BSA). The bulks consisted of individuals with an extreme phenotype taken from a population of 140 F_3 families issued from the cross between RE714 (resistant) and Hardi (susceptible). The population had been tested with three powdery mildew isolates at the seedling stage. Qualitative interpretation of the resistance tests located the *MlRE* gene on the distal part of the long arm of chromosome 6A. A subsequent quantitative interpretation of the resistance permitted us to detect another resistance factor on a linkage group assigned to chromosome 5D, which was constructed with microsatellites for which a polymorphism of intensity between bulks was observed. This quantitative trait locus (QTL) explained 16.8–25.34% of the total variation. An interaction between both the resistant factor (*MlRE* and the QTL) was found for only one of the isolates tested. This study shows the advantage of making a quantitative interpretation of resistant tests and that the use of microsatellites combined with BSA is a powerful strategy to locate resistance genes in wheat.

Communicated by F. Salamini

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Key words *Triticum aestivum* · *Blumeria graminis* f. sp. *tritici* · QTL mapping · Molecular markers · Disease resistance

Introduction

Powdery mildew, caused by the pathogen *Blumeria graminis* ex E.O. Speer f.sp. *tritici*, is one of the major diseases of wheat (*Triticum aestivum*) and causes important yield loss in wheat growing areas with a temperate climate (Bennet 1984). Fungicide treatments exist but often result in the apparition of resistant races of powdery mildew (Jorgensen 1988). One of the most environmentally safe ways to control powdery mildew is to cultivate resistant varieties. Since 1960 (Powers and Sando), investigations on the genetic determinism of powdery mildew resistance in wheat have identified 24 genes (McIntosh et al. 1998; Shi et al. 1998), sometimes with different allelic forms, that provide race-specific resistances generally from the seedling stage.

Identifying molecular markers linked to resistance genes is a key factor in using the latter efficiently for further investigations. First, markers permit assisted selection, which is an attractive approach when pathological tests are costly, time-consuming and dependent on environmental conditions (Michelmore 1995). Second, fine mapping may be one of the first steps to be conducted for cloning and for a better understanding of the interactions between host and pathogen. Among the numerous plant-pathogen interactions described, more than 10 resistance genes have been cloned and, based on the properties of their products, several classes of genes have been identified (Bent 1996). Finally, knowing the precise location of genes on chromosomes enables relations with other disease resistance genes and homoeoallelism relationships among genomes to be studied, as has been done for wheat, which belongs to the tribe of *Triticeae* (Spielmeyer et al. 1998).

Genetic markers are a powerful tool since they allow disease resistances to be broken up into their components

by quantitative analysis and quantitative trait locus (QTL) mapping (Young 1996). Disease resistance systems have generally been considered to be monogenic or oligogenic resistances, with race-specific genes, or to be polygenic, with several minor factors of equivalent weight. However, several studies have shown that resistance may be the combination of one (or two) major factor(s) and few minor ones (Dirlewanger et al. 1994; Pecchioni et al. 1996). In those cases, distributions of resistance levels in segregating populations are neither bimodal nor normal, but intermediate cases are represented. Depending on the objective, two interpretations are possible. When having genetic markers for the major gene is a priority, qualitative interpretation is possible after a careful removal of individuals with intermediate reactions (Concibido et al. 1996; Kreike et al. 1994; Dirlewanger et al. 1994). Or, when a complete analysis is wanted, quantitative methods, notably QTL mapping, are effective in detecting both major and minor factors (Dirlewanger et al. 1994; Pecchioni et al. 1996).

In most species, bulked segregant analysis (BSA, proposed by Michelmore et al. 1991) is the classical way to find genetic markers of disease resistance genes. In wheat, there are only a few examples of disease resistance genes that have been tagged using BSA, mainly because of the large size of the wheat genome, low polymorphism levels and existence of numerous repeated sequences. By means of BSA, however, random amplified polymorphic DNA (RAPD) markers for *Pm18* (Hartl et al. 1995) and *Pm25* (Shi et al. 1998) and amplified fragment length polymorphism (AFLP) markers for a *Septoria tritici* blotch resistance gene (Goodwin et al. 1998) were obtained.

In wheat, the recent development of microsatellite markers is motivated by their codominance, high level of polymorphism, chromosome specificity and ease of manipulation (Röder et al., 1995, 1998). During the last 10 years, RFLP markers have been extensively used for mapping in wheat and while these are well-adapted for studying relations between genomes of *Triticeae*, their polymorphism is very limited (Chao et al. 1989; Cadalen et al. 1997). RAPD markers have the same drawbacks coupled with reproducibility problems (Devos and Gale 1992). Consequently, development of new markers like AFLP and microsatellites which show a much higher level of polymorphism is a necessity, especially when working in an intraspecific context. Moreover, microsatellites are, in most cases, chromosome-specific, which is a considerable advantage in allohexaploid wheat.

Winter wheat line RE714 possesses two race-specific resistance genes, *Pm4b* and *MlRE* (Robe and Doussinault 1996). *MlRE* is particularly interesting since it has a residual effect on adult plant resistance (Chantret et al. 1999). The aims of the study reported here were to locate and map the powdery mildew resistance gene *MlRE* using bulked segregant analysis performed with a set of microsatellites uniformly distributed among the whole genome of wheat. Subsequent to this, we will determine if a quantitative interpretation of resistance allowed the detection of other minor factors.

Materials and methods

Plant material

A population of 140 F_3 families derived from the cross between RE714 and Hardi (a susceptible cultivar which does not possess any race-specific gene) was used. Renan, which possesses the race-specific resistance gene *Pm4b*, and *Triticum dicoccum* 119, the donor of *MlRE* (Robe and Doussinault 1996), were used as the control lines for the corresponding genes in seedling tests.

Fungal material

Three *Blumeria graminis* f. sp. *tritici* isolates were used for seedling tests: 96–18, 95–9 and 93–27. They were collected in France from 1993 to 1996 and purified from single-pustule progenies. These three isolates were chosen because they have a virulence response to resistance gene *Pm4b* and an avirulence response to resistance gene *MlRE*. They possess different virulence spectra with respect to other powdery mildew resistance genes (observed with several differential lines; data not shown) and also slight aggressiveness differences.

Disease assessments

Disease assessments were made at the seedling stage. Each F_3 family was evaluated (without replicates) by testing 13 F_3 plant populations with isolates 93–27, 95–9 and 96–18. 93–27 was the isolate with which the *MlRE* gene was originally identified by Robe and Doussinault (1996). Five tests were done to evaluate 13×140 plants. For each test, the stability of the isolates was verified with differential lines (data not shown). Plants were inoculated and scored on a 0–9 scale (0: no visible symptoms; 9: heavy sporulation) based on infection type, number of colonies and intensity of sporulation, as previously described (Robe and Doussinault 1996).

Microsatellites and RFLP assay

F_3 plants were grown under glasshouse conditions. About 5 weeks after sowing, an equal quantity of leaf tissue was taken from 13 F_3 plants of each family. DNA extraction, as well as the digestions, Southern blotting and hybridization were carried out using the method described by Lu et al. (1994). Polymerase chain reaction (PCR) amplifications of microsatellites were performed as described in Röder et al. (1995) migration and visualization was as described in Tixier et al. (1997).

The probe used, ksuD27, is a *T. tauschii* genomic clone obtained from B.S. Gill, Manhattan, Kansas. Microsatellite primer pairs used were those recently isolated by Röder et al. (1998).

BSA

DNA bulks of 9 resistant and 9 susceptible families were made. For each family, plant material was bulked before DNA extraction.

Mapping

The linkage groups were established with MAPMAKER software (Lander et al. 1987) with a minimal LOD score of 3 and a maximal genetic distance of 30 cM. Genetic distances between markers were estimated using the mapping function of Kosambi (1944). Recombination rate standard deviation between *MlRE* loci was calculated as described by De Vienne (1998) for codominant markers in a F_2 population.

QTL mapping

For each isolate and each F_3 family, the mean resistance scores of F_3 plants were used for QTL mapping. The association of each marker genotype with the resistance values was assessed by comparing phenotypic means of the three- or two-marker classes (for codominant or dominant marker, respectively) using a single-factor analysis of variance (GLM procedure, SAS Institute Inc 1989). The probability threshold used to declare a significant association between a marker and the resistance was α=0.001. QTL effects were estimated as the percentage of phenotypic variation explained by the QTL (R^2, ratio of marker to the total sum of squares). From models with significant markers, predicted values were plotted against the corresponding residuals to verify their independence.

Results

Powdery mildew resistance tests

In spite of the fact that the three isolates had exactly the same spectra for the genes *MlRE* and *Pm4b*, some F_3 families showed different responses depending on isolate used. This observation was not caused by the slight aggressiveness differences between isolates since higher (or lower) disease scores were not always observed with the same isolate (data not shown). Consequently, each isolate was interpreted separately. Because of some experimental problems, one test (of 34 families) with 95–9 could not be interpreted.

For the majority of F_3 families (about 80%), distributions of F_3 plants represented one of the three segregation possibilities for a recessive major gene and allowed us to determine the F_2 parental plant genotype. For each isolate, when F_3 plants were all resistant (class 0 to class 3) or susceptible (class 7 to class 9), the F_2 parental plant was interpreted as being homozygous for the resistant or susceptible allele at the *MlRE* locus. When segregation was observed among plants of a F_3 family, the F_2 parental plant was interpreted as being heterozygous. For some families (about 30), however, distributions were ambiguous because of some F_3 plants displayed intermediate responses (class 4 to class 6) without a clear segregation, and this prevented us from distinguishing between the three qualitative possibilities for the F_2 parental plant. To limit error risks, we removed these families initially from qualitative analysis (Table 1). The segregation ratios obtained are presented in Table 1. Observed segregations were compared to a theoretical ratio with a chi-square test and fitted the expected 1:2:1 ratio except for isolate 96–18 for which a deficit of resistant families was observed (Table1).

Table 1 Number of F_2 parental plants obtained from the cross between RE714 and Hardi for each possible combination of alleles at the *MlRE* resistance loci with the three isolates. The theoretical numbers of individuals are given in brackets. The fit for observed distributions to theoretical segregation ratios is given as the probability of the chi-square value [P (χ^2)]

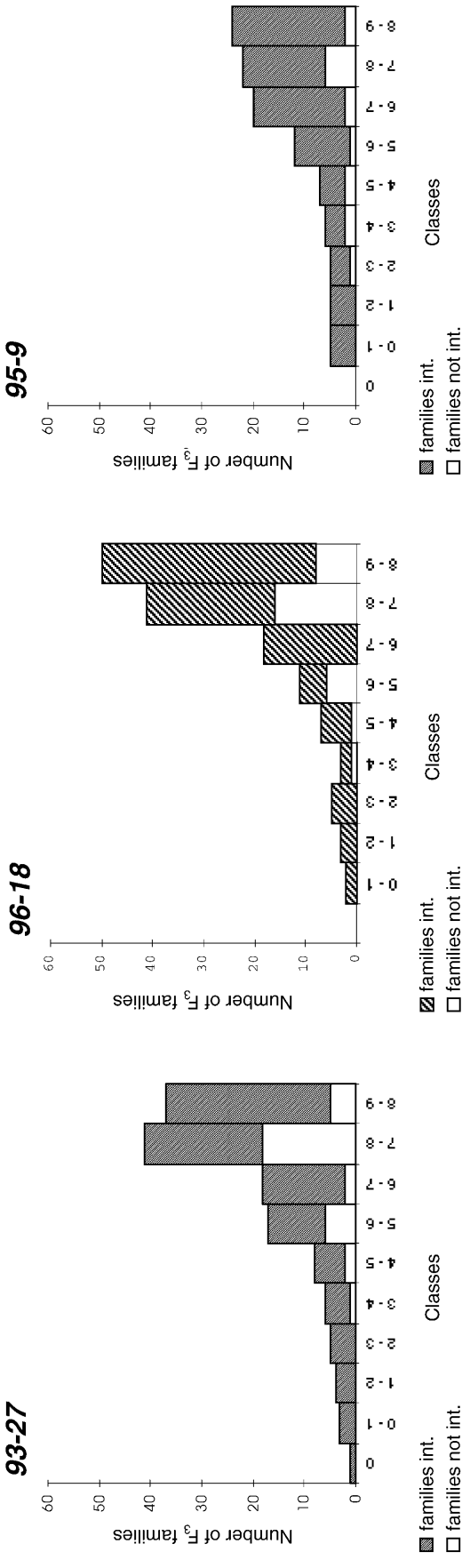
Isolate	Genotypes				Missing values	Total	Hypothesis tested[c]	P (χ^2)
	MlRE/*MlRE*	*MlRE*/o[a]	o/o	?[b]				
93–27	19 (26.5)	53 (53)	34 (26.5)	34	–	140	1:2:1	0.1197
96–18	16 (27)	49 (54)	43 (27)	32	–	140	1:2:1	0.0007
95–9	20 (22.5)	45 (45)	25 (22.5)	16	34	140	1:2:1	0.7575

[a] o, Hardi allele at the *MlRE* locus
[b] ?, Families with F_3 plants showing intermediate responses so that the genotype could not be determined
[c] Hypothesis is tested against observed values for *MlRE*/*MlRE*, *MlRE*/o and o/o genotypes

Table 2 Statistical model joining the two QTLs detected for resistance to isolates 93–27, 96–18 and 95–9 in the F_3 family population obtained from the cross between RE714 and Hardi.

Isolate	Locus			Means[b]			R^2 total (%)	Number of individuals
		P[a]	R^2	A	H	B		
93–27	*XksuD27*	0.0001	24.12	5.13[a]	6.55[b]	7.97[c]		
	Xgwm174	0.0001	25.34	4.99[a]	6.84[b]	7.81[c]	46.60	129
96–18	*XksuD27*	0.0001	31.60	5.43[a]	6.90[b]	8.53[c]		
	Xgwm174	0.0001	20.64	5.81[a]	7.03[b]	8.04[c]		
	inter[c]	0.0001	8.67	–[d]	–[d]	–[d]	60.34	129
95–9	*XksuD27*	0.0001	36.96	4.17[a]	6.13[b]	7.90[c]		
	Xgwm174	0.0001	16.82	4.68[a]	6.26[b]	7.26[c]	49.93	129

Means followed by the same symbol are not significantly different at the 0.01 probability level
[a] P is the probability associated with significance test on Fischer value
[b] A, Homozygous for RE714 allele; B, homozygous for Hardi allele; H, heterozygous
[c] inter, Interaction between loci *XksuD27* and *Xgwm174*
[d] Comparison of means in Fig. 4



Because of the presence of F_3 plants with intermediate response and to take the entire population into account, we made a quantitative interpretation of the seedling tests. For each family and for each isolate, the mean of the disease scoring for the 13 F_3 plants was calculated. Distributions obtained are presented in Fig. 1. They are not Gaussian as the resistance is controlled by, at least, one major gene (*MlRE*), but intermediate classes are represented. Means of families for which a qualitative interpretation was not carried out were between 3 and 9 (Fig. 1). Furthermore, standard deviations calculated from the resistance scores of those families were between 1 and 3, whereas the standard deviations of others were more extreme: between 0 and 4.5 (data not shown). All those elements suggest the existence of other resistant factors.

BSA

A total of 109 microsatellites were tested on RE714, Hardi and on the two bulks. Among them, 8 microsatellites could not be interpreted, 36 did not reveal polymorphism between RE714 and Hardi, 60 revealed polymorphism between the parents but not between bulks and 5 were polymorphic between the parents and between bulks. Among these 5 microsatellites, 2 (gwm174 and gwm583, on chromosome 5D) showed an intensity polymorphism between bulks, and 3 (gwm617, gwm427 and gwm494 (one locus), on chromosome 6AL) showed a clear polymorphism between bulks (Fig. 2).

The 5 microsatellites which were polymorphic between the two bulks, 2 others chosen from chromosome 6AL (gwm570 and gwm169), and the probes ksuD27, chosen because it is located on the distal part of chromosome 6AL, and gbxG103, located on chromosome 5D, were tested on the whole population. This allowed us to build two linkage groups, one corresponding to the long arm of chromosome 6A and the other to a part of chromosome 5D. The three loci for the gene *MlRE*, corresponding to the three powdery mildew isolates, were linked to the markers assigned to the distal part of the chromosome 6AL (Fig. 3). The four loci assigned to chromosome 5D remained independent of loci for the gene *MlRE* at a LOD=5.25. Markers assigned to chromosome 6A were tested on *T. dicoccum* 119 and those for which the allele of RE714 was the same as the allele of *T. dicoccum* 119 are shown enclosed in boxes in Fig. 3.

Fig. 1 Distributions of the resistance scores of the F_2 parental plants as estimated by the means of the resistance scores of F_3 plants of the same family at the seedling stage, with isolates 93–27, 96–18 and 95–9. *families int.* Families for which qualitative interpretation has been done, *families not int.* families for which qualitative interpretation has not been done

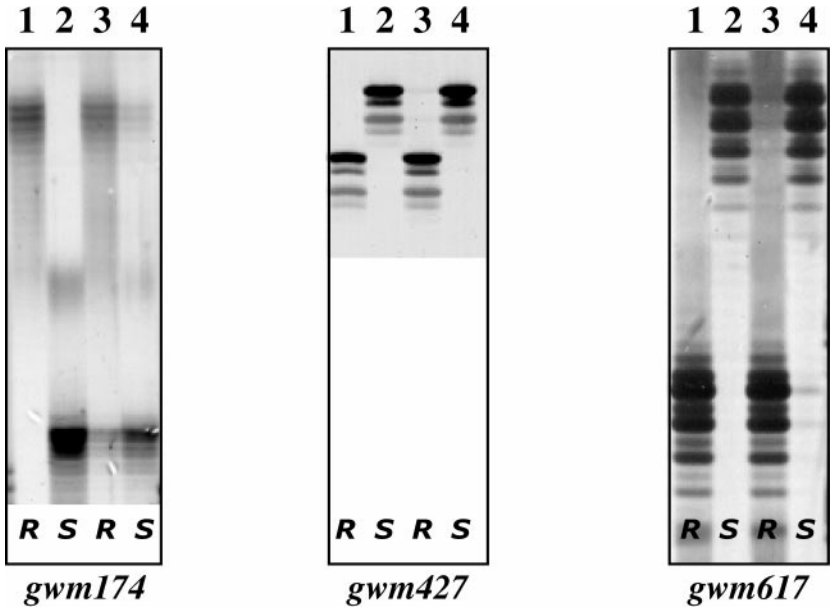


Fig. 2 PCR products of microsatellites gwm174, gwm427 and gwm617 on RE714 (*1*), Hardi (*2*), resistant bulk (*3*) and susceptible bulk (*4*). gwm174 shows intensity polymorphism between bulks; gwm427 and gwm617 show distinct polymorphism between bulks, *R* Resistant, *S* susceptible

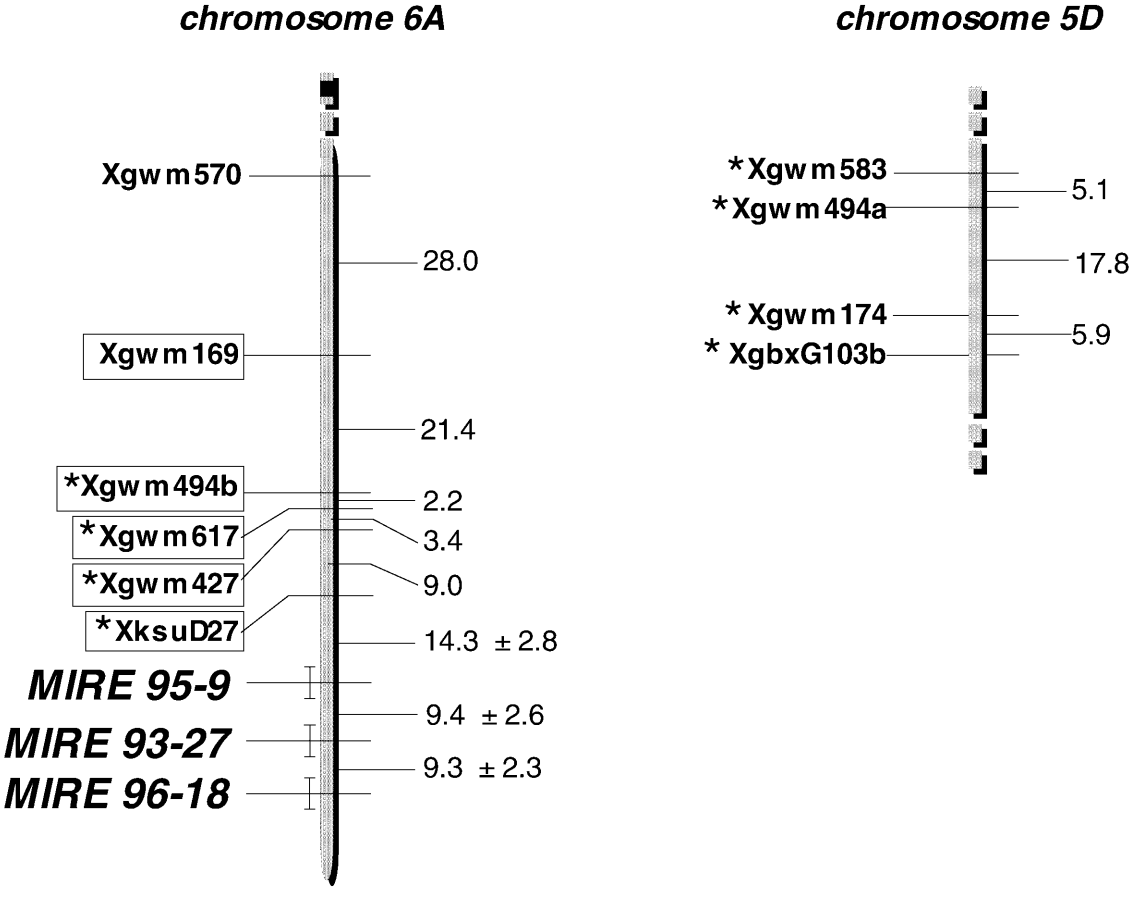


Fig. 3 Linkage groups obtained from the F_3 family population between RE714 and Hardi, with microsatellites and RFLP markers assigned to chromosomes 6AL and 5D and the three loci for *MlRE* corresponding to the three isolates used. Loci marked by a *star* are associated with resistance at a threshold of 0.0001. Loci enclosed in a *box* are those for which the allele of RE714 is the same as the allele of *T. dicoccum* 119. *Black lines* (*I*) represent the confidence intervals of the genetic distances; *black box* represents the centromere

QTL mapping

Analyses of variance were conducted on all marker loci that had been tested on the whole population (those of chromosome 6A and 5D). Two QTLs associated with resistance to the three isolates were detected at a threshold of 0.0001: one on the chromosome 6A and the other on

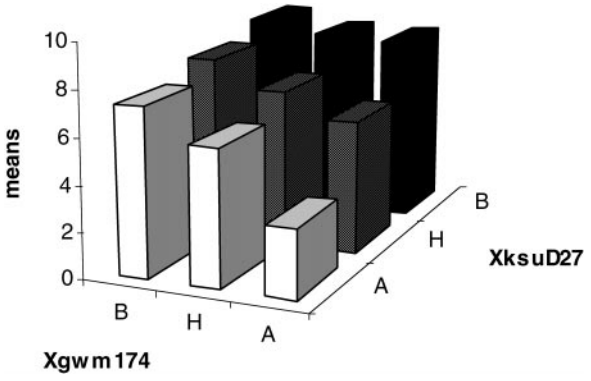


Fig. 4 Least square means of resistance to isolate 96–18 for the different genotypic classes for the interaction between loci *XksuD27* and *Xgwm174* in the F_3 family population obtained from the cross between RE714 and Hardi. *A* Homozygous for RE714 allele; *B*, homozygous for Hardi allele; *H*, heterozygous

chromosome 5D. With respect to the QTL detected on chromosome 6A, loci *Xgwm494b*, *Xgwm617*, *Xgwm427* and *XksuD27* were significant, with the peak on locus *XksuD27* (Fig. 3). For the QTL detected on the chromosome 5D, loci *XgbxG103*g, *Xgwm174*, *Xgwm494a* (resistance to 93–27) and *Xgwm583* (resistance to 93–27 and 96–18) were significant, with the peak on locus *Xgwm174* (Fig. 3). The QTL detected as being associated to markers assigned to chromosome 6A corresponded to the gene *MlRE*, already known, but analysis of variance revealed another QTL for resistance against powdery mildew at the seedling stage that was associated to markers assigned to chromosome 5D. To construct a model joining the effects of both QTLs, we chose loci *XksuD27* and *Xgwm174*: *XksuD27* was the closest locus of loci *MlRE* while *Xgwm174* was codominant with the highest part of variability explained for isolates 93–27 and 96–18 (data not shown). Results of these models are presented in Table 2. The models with both factors explained from 54.8% to 71.9% of the phenotypic variability. Statistical interaction between QTLs was tested and appeared to be significant only for resistance to isolate 96–18. Means for interaction between *XksuD27* and *Xgwm174* for resistance to isolate 96–18 is presented in Fig. 4. Only families with the allele of RE714 at both loci were clearly resistant.

Discussion

Analysis of powdery mildew resistance at the seedling stage of line RE714 was carried out by testing a population of 140 F_3 families from the cross between RE714 and Hardi, with three isolates avirulent to the gene *MlRE*. We found that the gene *MlRE*, already known (Robe and Doussinault 1996), was the major resistance factor but also that another resistant factor existed that accounted for 16–25% of the variation and was located on chromosome 5D. Qualitative interpretation of resistant tests was done first and allowed us to identify genetic markers and the chromosomal location of the gene *MlRE*. Differences among the resistance to the three isolates in pathological tests on the F_3 families allowed us to distinguish three loci for *MlRE*. These three loci turned out to be linked at 9.3 and 9.4 cM. This situation may correspond to a cluster of genes. Clusters of disease resistance genes are frequent in many species (Pryor and Ellis 1993; Michelmore 1995). Since the plant material used did not allow us to make repetitions, it would be interesting to specify distances between loci using a population composed of fixed individuals, like a single-seed descent (SSD) population. Such a population (170 SSD) is in the process of being produced.

Qualitative interpretation, (i.e. determination of the genotype at the *MlRE* locus for F_2 parental plants), was not possible for all the families since intermediate reactions (class 4 to class 6) for F_3 plants were observed in 16, 32 and 34 families, depending on the isolate. These observations led us to make a quantitative interpretation to take into account the whole population. Two QTLs were detected by means of quantitative interpretation and QTL mapping with all three isolates: one of these was on chromosome 6A, which corresponded to *MlRE*, and the other was at a new resistance locus detected on chromosome 5D. The weight of the QTL corresponding to *MlRE* was underestimated since the genetic distance between the next marker and the loci for *MlRE* was at least 14.3 cM. The second factor, on chromosome 5D, accounted for 16.82–25.34% of the variability, depending on the isolate considered and cannot be considered as 'minor'. In spite of the apparent monogenic determinism of resistance (gene *MlRE*: Robe and Doussinault 1996), another factor of resistance exists. This is supported by the presence of 20–30 F_3 families for which F_3 plants had intermediate reactions.

When a resistance is controlled by a major gene, the genotyping of all individuals of a segregating population is required to map this gene. Then, usually, the few ambiguous individuals of segregation are eliminated from the analysis or sometimes classified as resistant or susceptible. Eliminating these individuals from the analysis is probably a better choice than classifying them as resistant or susceptible since genotyping errors may lead to false estimations of genetic distances (Concibido et al. 1996). Many resistances are controlled by a major gene and one or more smaller factors: in pea, resistance to *Ascochyta pisi* race C resistance is controlled by a major QTL and a smaller one, explaining 45% and 20%, respectively, of the variability (Dirlewanger et al. 1994); in barley, resistance to leaf stripe is controlled by a major locus explaining 58.8% of the variability and by another one accounting for 29.3% of the variation (Pecchioni et al. 1996). Our study shows the advantages of making a quantitative analysis, as it enables the construction of a more realistic genetic model.

Another advantage of QTL detection strategy is the possibility to study genetic interactions. A significant interaction between the two QTLs was found only for isolate 96–18 (Table 2). Other authors have found differ-

ences between clones or isolates: Caranta et al. (1997) found interactions between two QTLs for only some strains of potyviruses. Comparisons of means for resistance to 96–18 indicates that only individuals with RE714 alleles at both QTLs are really resistant (Fig. 4). The interaction effect, in addition to the main effects, leads to a deficit of resistant plants, which is probably one of the explanation for the distortion observed in the qualitative interpretation of resistance to 96–18 (Table 1).

The use of microsatellites combined with BSA is probably one of the fastest and easiest strategies to locate and map resistant genes in wheat, especially in an intraspecific context or when special material (as isogenic lines) is not available. Until now there have only been a few examples of disease resistance genes tagged by BSA in wheat, unlike in a lot of other species, essentially because of a lack of polymorphism, the size of the genome and the presence of many repeated sequences. The recent development of markers likely to reveal a high level of polymorphism, such as microsatellites or AFLPs, gives the tagging of gene with BSA a better chance to succeed. In our study, 64% of the microsatellites tested were polymorphic. This polymorphism rate was equivalent to those found in others studies (Plaschke et al. 1995; Röder et al. 1995). Furthermore, microsatellites have the significant advantage of being genome-specific (Röder et al. 1995). Having one or two microsatellite markers for a gene means that it can be assigned a location immediately.

The QTL mapped on chromosome 5D was found close to microsatellite loci for which an intensity polymorphism was observed between bulks. In our study, BSA was employed to map the major gene, *MlRE*, but since we chose F_3 families with extreme disease scores to make bulks, BSA also detected a quantitative factor. BSA, initially proposed for mapping major genes (Michelmore et al. 1991), has been applied to the detection of QTLs in both a theoretical manner (Wang and Paterson 1994) and also in practice (Chagué et al. 1997; William et al. 1997). The weight of the QTL found with BSA was assessed between 11.2% and 22.8% (Chagué et al. 1997) or between 7% and 34% (William et al. 1997); the QTL we have found explains about 20% of the variability, a value which was included in these intervals. Mapping QTLs with BSA is also facilitated by the use of codominant and, especially, specific PCR markers. Specific PCR may be sensitive to the number of target sequences and so detect intensity polymorphism between bulks in spite of the existence of some heterozygous individuals in the bulks (results not shown).

The loci for *MlRE* were located on the distal part of the long arm chromosome 6A and probably on an introgressed fragment from *T. dicoccum* 119. The location of the loci for *MlRE* on a fragment of *T dicoccum* confirms the result of Robe and Doussinault (1996). With respect to the location, until now, there is no other published gene for powdery mildew resistance on chromosome 6AL, but the gene *Pm20* is on 6BL on a introgression from *Secale cereale* (Friebe et al. 1994, 1996), and it may exist an homoeoallelic relation between loci of *MlRE* and *Pm20*. This kind of hypothesis was proposed for the genes *Pm1*, *Pm9* and *Pm18* located on chromosome 7AL, for *Pm5* located on chromosome 7BL and *Mlf* located on chromosome 7HL of oat (Hartl et al. 1995). Furthermore, the leaf rust resistance gene *Lr9* is linked to ksuD27 (Autrique et al. 1995) and is probably located on the distal part of chromosome 6BL. They may derive from a common ancestor gene, as suggested by Hartl et al. (1995). It could be interesting to confirm the location of this gene by a physical means.

Acknowledgements The authors thank M.T. Pavoine and B. Charef for technical assistance. They acknowledge J.M. Jacquemin and D. Mingeot for providing ‘gbx’ probes and some genotyping data. They are also grateful to C. Boeuf and P. Barret for their advice

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