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A. J. Castro · F. Capettini · A. E. Corey · T. Filichkina · P. M. Hayes · A. Kleinhofs · D. Kudrna · K. Richardson · S. Sandoval-Islas · C. Rossi · H. Vivar

Mapping and pyramiding of qualitative and quantitative resistance to stripe rust in barley

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Abstract The identification and location of sources of genetic resistance to plant diseases are important contributions to the development of resistant varieties. The combination of different sources and types of resistance in the same genotype should assist in the development of durably resistant varieties. Using a doubled haploid (DH), mapping population of barley, we mapped a qualitative resistance gene (*Rpsx*) to barley stripe rust in the accession CI10587 (PI 243183) to the long arm of chromosome 1(7H). We combined the *Rpsx* gene, through a series of crosses, with three mapped and validated barley stripe rust resistance QTL alleles located on chromosomes 4(4H) (QTL4), 5(1H) (QTL5), and 7(5H) (QTL7). Three different barley DH populations were developed from these crosses, two combining *Rpsx* with QTL4 and QTL7, and the third combining *Rpsx* with QTL5. Disease severity testing in four environments and QTL mapping analyses confirmed the effects and locations of *Rpsx*, QTL4, and QTL5, thereby validating the original estimates of QTL location and effect. QTL alleles on chromosomes 4(4H) and 5(1H) were effective in decreasing disease severity in the absence of the resistance allele at *Rpsx*. Quantitative resistance effects were mainly additive, although magnitude interactions were detected. Our results indicate that combining qualitative and quantitative resistance in the same genotype is feasible. However, the durability of such resistance pyramids will require challenge from virulent isolates, which currently are not reported in North America.

Keywords Barley · Stripe rust · Quantitative resistance · QTL

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A. E. Corey · T. Filichkina · P. M. Hayes (✉) · K. Richardson · C. Rossi
Department of Crop and Soil Science, Oregon State University, Corvallis, OR 97331-3002, USA
e-mail: patrick.m.hayes@orst.edu

A. J. Castro
Departamento de Producción Vegetal,
Est. Exp. "Dr. Mario A. Cassinoni", Facultad de Agronomía, Universidad de la República, Ruta 3 Km.373, Paysandú 60000, Uruguay

F. Capettini · H. Vivar
ICARDA/CIMMYT, Apdo. 6-641, 06600 Mexico, DF, Mexico

A. Kleinhofs
Department of Crop and Soil Sciences,
Washington State University, 259 Johnson Hall,
P.O. Box 646420, Pullman, WA 99164-6420, USA

S. Sandoval-Islas
Instituto de Fitosanidad, Colegio de Postgraduados, Montecillo, México, 56230

D. Kudrna
Arizona Genomics Institute, University of Arizona, Tucson, AZ 85721-0036 USA

Introduction

Genetic resistance to plant diseases is a major objective of most plant breeding programs. Determining the location of the gene, or genes, controlling resistance can assist in the rapid and efficient development of new resistant varieties. The durability and stability of plant disease resistance is also a fundamental issue, as it is an important asset in new cultivars. Considering that durable resistance is a reasonable and laudable goal, the specific strategies used to achieve this goal may be improved by thorough characterization and analysis of the inheritance of resistance.

The term "qualitative resistance" designates Mendelian genes of large effect that interact on a gene-for-gene basis with the pathogen. The term "quantitative resistance", in this context, refers to resistance that shows continuous variation and is usually incomplete in expression. Qualitative resistance usually shows race-specificity, while the race specificity of quantitative resistance is still an unresolved question. The risks involved in using gene-for-gene resistance are well-known (Johnson 1981; Parlevliet 1983; Vanderplank 1963, 1978), and there is

evidence that pathogen virulence can evolve more quickly than plant breeders can deploy single resistance genes in new varieties (Parlevliet 1977). As quantitative resistance has a higher probability of durability (Parlevliet 1989), it has been proposed as an alternative to qualitative resistance. Its main limitation is that it requires extensive and accurate field-testing, which makes it more difficult to select for in plant breeding programs.

Pyramiding qualitative resistance genes with different race specificities has also been proposed as a way to increase the likelihood of resistance durability (Schaffer and Roelfs 1985). Putting multiple race-specific genes in a single genotype minimizes the probability that a single mutation in the pathogen can overcome all the resistance genes (Huang et al. 1997; McIntosh and Brown 1997; Mundt 1991). For example, Singh et al. (2001) pyramided three bacterial blight resistance genes in rice and demonstrated that this provided a wider spectrum of resistance to the pathogen population than single genes. Pyramiding quantitative resistance may also increase the probability that a variety will show durable resistance. Castro et al. (2000) have combined three resistance alleles at stripe rust resistance quantitative trait loci (QTL) in barley in the same genetic background and validated their effects in reducing disease severity.

The combination of both types of resistance genes (e.g., qualitative and quantitative) offers the possibility of exploiting both the complete effect of the qualitative resistance gene, or genes, with the theoretical durability of the quantitative resistance gene, or genes. In other words, one would expect that in the case of a "breakdown" of the qualitative resistance, the quantitative resistance genes present in the genotype would act as an "insurance" policy.

Stripe rust (caused by the fungal pathogen *Puccinia striiformis* f.sp. *hordei*) is a major disease of barley worldwide, and the development of resistant germplasm is a major objective of barley breeding programs where this disease is a production constraint. The lack of durability of qualitative resistance in this system has been particularly problematic (Hayes et al. 2001). In a collaborative effort to map and exploit quantitative resistance using molecular tools (reviewed by Hayes et al. 2001), we have used barley germplasm developed by ICARDA/CIMMYT with quantitative resistance (Sandoval-Islas et al. 1998). In the course of this research we have developed quantitative resistance pyramids combining three QTL alleles from two different sources (Castro et al. 2003). We were interested in combining these quantitative resistance alleles with a qualitative resistance gene. Our target as a qualitative resistance donor was CI10587 (PI 243183), an accession from the U.S. National Small Grains Collection. The location of a resistance gene in this accession has not been reported.

The objectives of the investigation reported here were to: (1) map and introgress into an adapted background, the qualitative resistance gene present in CI10587; (2) combine, in the same genetic background, the previously mentioned resistance gene with the three resistance QTL pyramided by Castro et al. (2003); (3) validate the estimates of the effects of the qualitative resistance gene and of the three QTL alleles in the new genetic background; (4) determine if there were interactions between the qualitative resistance gene and the QTL alleles.

Materials and methods

Plant material

Mapping resistance in CI10587

Ninety-four doubled-haploid (DH) lines were derived from the F_1 of the cross CI10587 × Galena, using the *Hordeum bulbosum* technique as described by Chen and Hayes (1989). CI10587 (PI 243183) is an accession from the U.S. National Small Grains Collection. It is a two-row, spring-habit barley with resistance to stripe rust. Galena is a two-row, spring-habit, proprietary malting barley belonging to the Coors Brewing Company. It is susceptible to stripe rust (Toojinda et al. 2000).

Development of resistance gene pyramids

After the phenotyping and genotyping of the CI10587 × Galena population (see below), one DH line (D3-6) was selected and crossed with var. Baronesse, a high-yielding feed barley variety well adapted to the Pacific Northwest of the USA. One hundred DH lines were derived from the F_1 of this cross, following the procedures already described. Two DH lines (D3-6/B-23 and D3-6/B-61) from this mapping population were selected, based on their stripe rust resistance and agronomic trait phenotypes, and crossed with two DH lines from the resistance QTL allele pyramid population described by Castro et al. (2003). One of these lines (BCD47) carries stripe rust resistance QTL alleles on chromosomes 4(4H) and 7(5H), while the other (BCD12) carries a stripe rust resistance QTL allele on chromosome 5(1H). Three crosses were made between the quantitative and qualitative resistance sources, and three DH populations were obtained from the F_1 of each cross following the procedures described above. Seventy DH lines were derived from the D3-6/B-23 × BCD47 cross (hereafter referred to as population BU), 77 DH lines were derived from the D3-6/B-61 × BCD47 cross (hereafter referred to as population AJ), and 85 DH lines were derived from the D3-6/B-61 × BCD12 cross (hereafter referred to as population OP). The germplasm derivation process is shown in Fig. 1.

Phenotyping

The field resistance of CI10587, Galena, and the CI10587 × Galena population to field inoculum was determined in four tests at the ICARDA/CIMMYT field station located in Toluca, Mexico. The plant material was evaluated in one-row, 3-m plots at two planting dates in 1994 and 1996. Spreader rows, planted at 5.25-m intervals and consisting of a mixture of 15 susceptible genotypes, were inoculated twice with infected plants placed in the foliage and with applications of spores suspended in oil. Infected plants and spores were collected locally. The race composition of this inoculum was not determined. Due to the fact that the stripe rust severity in these lines was either lower than 10% or higher than 80%, their reaction was rated as resistant and susceptible on a plot basis. The DH lines from the Baronesse × D3-6 cross were tested at Toluca, Mexico, following the same procedures, in 1997.

The BU, AJ, and OP populations were assessed for field resistance in four tests over 3 years at Toluca, Mexico, following the procedures described above. The DH lines and the parents were planted at one date in 1999 and 2001 and at two dates in 2000.

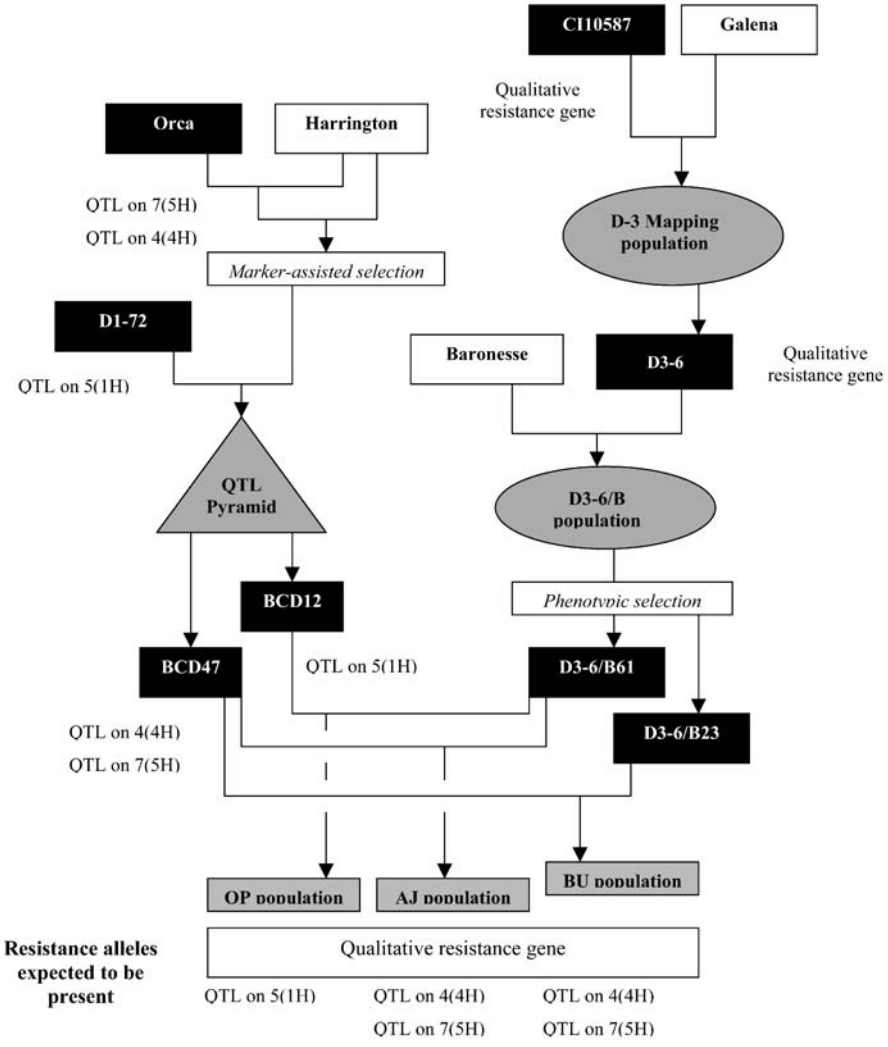

Fig. 1 Schematic showing the development of the qualitative/quantitative disease resistance populations. *Black boxes* represent resistance sources and *white boxes* represent susceptible parents

Stripe rust was rated as percentage severity on a plot basis rather than as resistant:susceptible because a range of disease severities was seen in the germplasm.

Genotyping

In the CI10587 × Galena population, restriction fragment length polymorphism (RFLP) markers were first mapped on chromosome 4(4H), 5(1H) and 7(5H) in regions where stripe rust resistance QTL had been detected in other mapping populations (Chen et al. 1994; Toojinda et al. 2000). When no associations were detected between stripe rust reaction and marker genotypes at these loci, additional RFLPs of known location throughout the genome were used for bulked segregant analysis (BSA) (Michelmore et al. 1991). When a non-random pattern of association of RFLP alleles on the long arm of chromosome 1(7H) with stripe rust reaction was observed, other RFLP loci mapping to this region in other mapping populations were assayed on the 94 DH lines. RFLP markers were assayed as described by Kleinhofs et al. (1993). The RFLP marker nomenclature follows that employed by the North American Barley Genome Mapping Project (Kleinhofs et al. 1993). Sequence-tagged site (STS) markers on chromosome 1(7H) were assayed, following the protocols of Mano et al. (1999). SSR markers were added to this map, as described below, to facilitate alignment with other mapping populations.

The resistance gene pyramid populations (AJ, BU, OP) were genotyped using SSR markers of known position on chromosomes 1(7H), 4(4H), 5(1H), and 7(5H). DNA was extracted from leaf tissue of 2- to 3-week-old plants (one plant per genotype) using the Qiagen DNAeasy 96 plant kit (Valencia, Calif.). After screening all available polymerase chain reaction (PCR)-based markers mapping to those regions, we were able to map 20 markers in the BU population, 22 in the AJ population, and 23 in the OP population. SSRs were assayed as described by Liu et al. (1996) and Russell et al. (1997). The SSR primer sets were developed and mapped by Ramsay et al. (2000), Liu et al. (1996) and Becker and Heun (1995). The reverse primers were labeled with FAM, TET, NED, or HEX fluorescent dyes. DNA amplifications were performed using either a Perkin-Elmer 9600 (Foster City, Calif.) or MJ Research PTC-100 (Waltham, Mass.) thermal cycler. PCR reactions were carried out in a 10-μl reaction mix containing 37.5 ng of template DNA, 1× PCR buffer, 0.025 U *Taq* DNA polymerase (Qiagen), 0.2 n*M* dNTPs and 0.1 pmol forward and reverse primers. Information on primer sequences and PCR amplification conditions for each set of primers are available at http://www.scri.sari.ac.uk/ssr (Ramsay et al. 2000), in Liu et al. (1996) and in Becker and Heun (1995). PCR-amplified fragments from differentially labeled SSR primers and with non-overlapping fragment sizes were simultaneously analyzed in the same gel lane and separated on an ABI Prism 377 DNA sequencer (Applied Biosystems, Foster City, Calif.) at the Oregon State University, Central Service Lab, or on an ABI Prism 3700 DNA sequencer at OMIC, Portland, Oregon. GENE SCAN and

GENOTYPER software (PE Applied Biosystems) were used for automated data collection and to determine the allele sizes in base pairs, based on the internal standard.

Genome mapping and QTL analysis

For mapping the gene conferring resistance in CI10587, we directly mapped the trait as a marker in the CI10587 × Galena population. Linkage analyses for all populations were performed using GMENDEL 3.0 (Holloway and Knapp 1994) following procedures described by Toojinda et al. (2000). In the three pyramiding populations (AJ, BU and OP), QTL were mapped using the multi-trait option implemented in MULTIQTL version 1.5. Each dataset was analyzed with 1,000 permutations in order to establish the significance of the QTL. A bootstrap simulation (with 1,000 samples) was used for the assignment of each significant QTL to a defined marker interval.

Genome regions, revealed by the QTL scans, that determine resistance to stripe rust were used in performing a QTL analysis analogous to candidate gene analysis, as described by Castro et al. (2002a), where the genotypes of the flanking markers are used as independent variables. Recombinant genotypes were not included in the analysis. Therefore, the independent variables had two levels each, with each level corresponding to a parental genotype. The treatment design was a 2 × n factorial, where n is the number of genome regions detected. The difference between parental marker class means estimates the additive effect of the QTL flanked by the markers. Double crossovers between the QTL and marker loci downwardly bias estimates of the effects. Thus, differences between parental marker genotype means are conservative estimates of the effects of QTL residing in the n chromosomal regions. We performed a joint analysis considering the QTL effects as nested in populations. We also performed the analysis pooling together the AJ and BU populations, as the QTL source was the same (BCD-47). Because the results of this joint analysis gave the same results as the individual population analyses we present the former. Statistical analyses were performed using the SAS GLM procedure (2001).

Results

Mapping of qualitative resistance in CI10587

The reactions to stripe rust of the DH lines from the CI10587 × Galena cross were extremely consistent across environments. Based on the reaction data pooled across environments, the ratio of resistant:susceptible plants was 55:45, which fits a 1:1 ratio ($P > 0.30$ from a chi-square test). In a DH population, the expectation of segregation of alleles at a single locus is a 1:1 ratio. We systematically tested several RFLP markers of known position through the genome in order to detect associations with the trait. A non-random pattern of association of RFLP alleles on the long arm of chromosome 1(7H) with stripe rust reaction

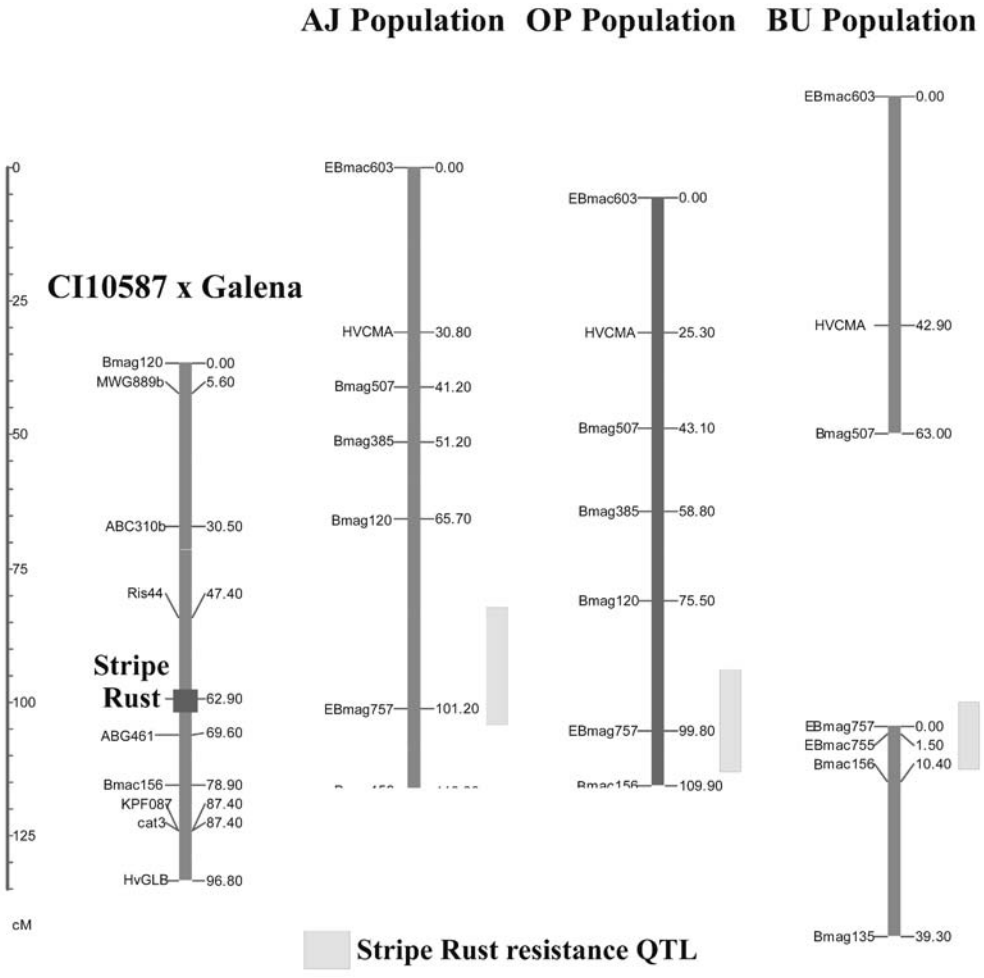

Fig. 2 Alignment of the chromosome 1(7H) maps of the CI10587 × Galena, AJ, BU and OP populations. The CI10587 × Galena map shows the linkage map location of the stripe rust resistance qualitative gene present in CI10587. The location of this gene, mapped using QTL analysis procedures, is shown in each of the derived populations. The *gaps* in the map of the BU population are due to monomorphism of the *Bmag385* and *Bmag120* markers in this population

Table 1 Summary of significance of effects of a barley stripe rust resistance gene (*Rpsx*) and QTL resistance alleles. Chromosome locations are shown in Figs. 2 and 3

Population	QTL/Gene	Resistance allele donor	LOD score	*P*-value
AJ	*Rpsx*	D3-6/B23	6.92	<0.0001
BU	*Rpsx*	D3-6/B23	11.95	<0.0001
OP	*Rpsx*	D3-6/B61	6.73	0.0009
	QTL5	BCD12	3.41	0.0136

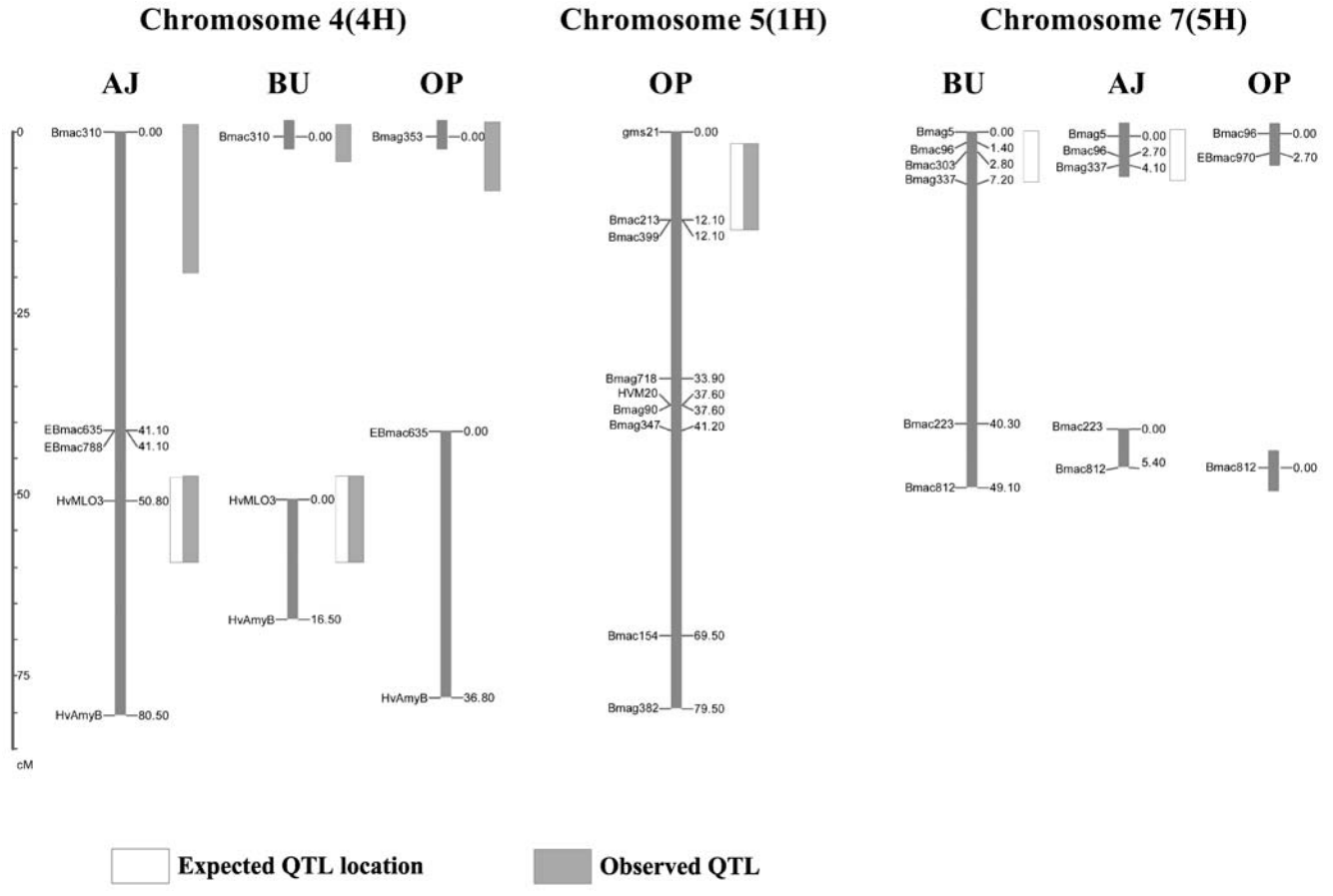

Fig. 3 Alignment of the maps of chromosomes 4(4H), 5(1H) and 7(5H) in the AJ, BU and OP populations, showing the expected and actual locations of stripe rust resistance QTL

was observed. Other RFLP loci mapping to this region in other mapping populations were then assayed on the DH lines. We mapped the stripe rust resistance gene between *Ris44* and *ABG461*, and based on known marker positions we located it on the long arm of chromosome 1(7H) (Fig. 2). In the rest of the paper we will refer to the qualitative resistance gene as *Rpsx*, pending gene assignment.

QTL analysis of qualitative and quantitative resistance pyramids

In order to determine the reaction of AJ, BU, and OP to stripe rust at the adult plant stage under field conditions, we performed QTL analyses of disease severity in these three pyramid populations. This analysis also allowed the estimation of the individual effects of the QTL alleles expected to be present in these populations and their interactions, inter se and with *Rpsx*. As described in the Materials and methods, the genotyping of these populations was restricted to markers on chromosomes 1(7H), 4(4H), 5(1H), and 7(5H).

For the three populations, a significant QTL was detected on chromosome 1(7H) (Table 1), in approximately the same region where *Rpsx* was expected to map to (Fig. 2). The resistance alleles corresponded to D3-6/B23 (in BU) and D3-6/B61 (in AJ and OP). We consider that *Rpsx* is the candidate gene for this QTL. No other QTL was detected in the AJ and BU populations in this analysis. In the OP population, an additional QTL with a smaller effect was detected on chromosome 5(1H). It was located in the expected QTL5 position (Fig. 3), since this population was expected to segregate for QTL alleles at

Table 2 ANOVA table of the candidate gene analysis for stripe rust resistance. Only QTL main effects and significant QTL × QTL interactions are presented. The r^2 for the model was 0.609

	df	M.S.	*F* value	Pr > *F*
Environment	3	11140	37.20	<0.0001
Population	1	4328	14.45	0.0002
Rpsx	1	229276	765.65	<0.0001
QTL4 (AJ/BU)	1	12728	42.50	<0.0001
QTL4B (AJ/BU)	1	1057	3.53	0.0607
QTL7 (AJ/BU)	1	141	0.47	0.4920
QTL5 (OP)	1	1508	5.03	0.0251
QTL4C (OP)	1	1820	6.08	0.0139
Rpsx × QTL4 (AJ/BU)	1	8251	27.55	<0.0001
Rpsx × QTL4B (AJ/BU)	1	1599	5.34	0.0211
QTL4 × QTL4B (AJ/BU)	1	3593	12.00	0.0006
QTL4 × QTL7 (AJ/BU)	1	2840	9.48	0.0022
Rpsx × QTL4C (OP)	1	5539	18.50	<0.0001
Rpsx × QTL4 × QTL4B (A/B)	1	3434	11.47	0.0007
Rpsx × QTL4 × QTL7 (A/B)	1	2959	9.88	0.0017
QTL4 × QTL4 × QTL7 (A/B)	1	1577	5.27	0.0220
Error	719	299		

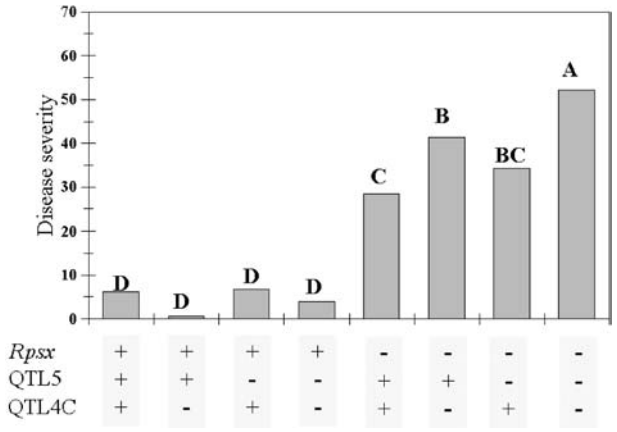

Fig. 4 Least squares means of disease severity in DH lines of the OP population classified according to the presence or absence of the resistance alleles at *Rpsx*, QTL5 and QTL4C QTL regions. *Bars* with the *same letter* are not significantly different ($P < 0.05$) based on pairwise comparisons

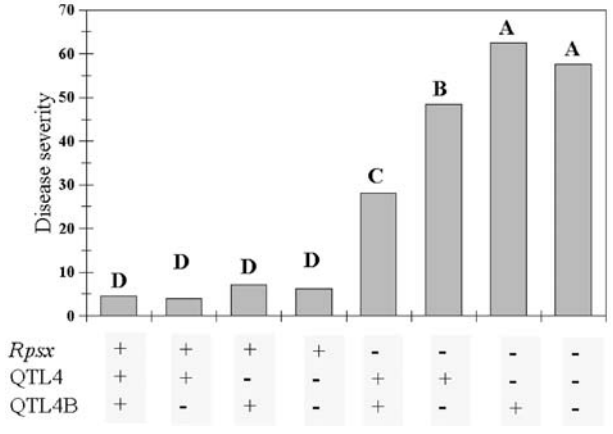

Fig. 5 Least squares means of disease severity in DH lines of the AJ and BU populations classified according to the presence or absence of the resistance alleles at *Rpsx*, QTL4 and QTL4B QTL regions. *Bars* with the *same letter* are not significantly different ($P < 0.05$) based on pairwise comparisons

this location. Through this analysis, no QTL was detected in any population at the QTL4 and QTL7 positions.

Considering that there could be a masking effect of *Rpsx* on the effect of other resistance QTL, we performed a second QTL analysis using the subset of lines with susceptible alleles at *Rpsx* in each population. The small sizes of the subpopulations (32 in AJ, 36 in BU, 37 in OP) limited the power of the QTL analysis, so we considered any QTL detected as a candidate QTL in the individual analyses. Only if the ANOVA of the pooled data confirmed the QTL did we consider it significant.

Via this analysis, we located stripe rust resistance effects in the QTL4 region in the BU and AJ populations (Fig. 3) as well as in the QTL5 region in the OP population. Another QTL candidate was mapped on chromosome 4(4H) in the AJ and BU population. This QTL (hereinafter referred to as QTL4B in order to differentiate it from the expected QR allele) was linked to *Bmac310*, more than 40 cM proximal to QTL4. A third QTL candidate was detected in the OP population linked to *Bmag353* (hereinafter referred to as QTL4C), also on chromosome 4(4H). *Bmac310* and *Bmag353* are tightly linked (Ramsay et al. 2000). The resistance alleles at these QTL candidates traced to BCD47 (in AJ and BU) and BCD12 (in OP). A complete map of BCD12 and BCD47 is not available, so it is not possible to determine if these QTL resistance alleles are identical by descent, although it is reasonable to expect so, since we have detected no resistance alleles in CI10587 or Baronesse in this region.

We used all of the QTL candidates from the original mapping populations in a candidate gene analysis, including QTL7. Although we failed to detect QTL effects in this region in any of the populations, this QTL was present in the parental line, BCD47, and was considered in the development of these populations. Considering that the same QTL were detected in AJ and BU and that the common origin of these QTL alleles is BCD47, the AJ and BU populations were pooled for a subsequent analysis. The ANOVA results (Table 2) confirmed the effects of *Rpsx*, QTL4, QTL4B, QTL4C, and QTL5, and the lack of significance of the QTL7 main effect. The qualitative nature of the *Rpsx* effect is reflected in the significant interactions with the different QTL. As seen in Figs. 4 and 5, the different QTL alleles show their effects only in the absence of the resistance allele at *Rpsx*. The disease severity of lines with the resistance allele at *Rpsx* in all the populations was significantly lower. In the absence of the *Rpsx* resistance allele, the presence of resistance alleles at QTL4 and QTL4B in AJ and BU, and at QTL5 and QTL4B in OP, significantly decreased disease severity. The significant second and third order interactions (Table 2), with the

exception of the interactions involving *Rpsx*, were magnitude interactions that do not lead to changes in estimates of individual effects.

Discussion

We have mapped a gene conferring qualitative resistance to barley stripe rust (BSR) on chromosome 1(7H) and validated its location in three mapping populations segregating for this source of resistance. Thomas et al. (1995) mapped a BSR resistance QTL in this region of the genome. The only qualitative BSR resistance gene mapped is *Yr4*, which is on the short arm of chromosome 5(1H) (von Wettstein-Knowles 1992). *Rpsx* conferred resistance to races PSH-13, PSH-14, PSH-21, and PSH-31 (R. Line, X. Chen, personal communication). However, further studies are necessary in order to establish the race specificity of *Rpsx* and its identity regarding other BSR qualitative resistance genes as these are mapped.

Combining qualitative and quantitative resistance genes in the same genotype presents a challenge to the plant breeder. The qualitative resistance gene masks the effects of the quantitative resistance genes, precluding phenotypic selection for the latter. Molecular marker-assisted selection (MAS) allows for the pyramiding of qualitative and quantitative resistance genes, based on the presence of target resistance alleles as inferred from the allelic composition of linked molecular markers. However, there are no reports in the literature describing the effectiveness of pyramiding of qualitative and quantitative resistance genes. The durability of such a resistance pyramid can only be assessed in the presence of a race that is virulent to *Rpsx*. Currently, such a virulence pattern is, fortunately, not present in North America. There is evidence of a new race in the Andean region (USDA nursery results 2001) and in these tests CI10587, the donor of *Rpsx*, had a severity of 60S. One of the quantitative resistance donors also showed higher severity percentages than in our long-term tests in Mexico: the severity of Orca in the Huancayo test (donor of QTL4 and QTL7 resistance alleles) was 80S versus 30S in Mexico. Shyri, donor of QTL5, however, had severity values of 0S in Huancayo versus 10S in Mexico. All BU, AJ, and OP lines with *Rpsx* and one or more resistance QTL allele had reactions ranging from 0 to 20S. Additional tests are underway to confirm this apparent effectiveness of the qualitative and quantitative resistance pyramids. Together with our longer-term data from Mexico, these results allow us to be cautiously optimistic about the effectiveness of combining qualitative and quantitative resistance in the same genotype. It is clear from Figs. 4 and 5 that the QTL alleles on chromosomes 4(4H) and 5(1H) were indeed effective at lowering disease severity in the absence of the resistance alleles at *Rpsx*.

The results of QTL analyses of the pyramid populations (AJ, BU and OP) validate the map location of *Rpsx* and also confirm the QTL allele effects reported by Castro et al. (2002b). Of particular interest is the lack of significance of QTL7. The original mapping population estimates of QTL4 and QTL7 (Chen et al. 1994) revealed QTL7 as having a much larger effect than QTL4, but Castro et al. (2000) found a change of rank in importance of these QTL based on the estimates of QTL effects in a QTL validation population. The AJ and BU populations represent a second generation of QTL alleles from the original mapping population and confirmed that these "realized heritability" estimates of QTL effect are indeed more robust estimates of allele value than those obtained in the original mapping population. QTL effect estimates are reported to be biased, raising concerns about their use in understanding and manipulating genetic determinants of quantitative characters (Beavis 1998). We have found that QTL effect estimates based on derived populations provide better estimates of QTL effects than source mapping populations, but whether this is due to bias estimation or to genetic background is not known.

The detection of a new QTL on chromosome 4(4H) is intriguing and may help to explain some of the inconsistencies in BSR resistance QTL effects in this region of the genome. In the original QTL report (Chen et al. 1994), QTL4 had an effect on disease severity in only one environment, and the significance threshold of this QTL extended over more than 40 cM. Castro et al. (2002b) showed that there were non-coincidental seedling resistance and heading date QTL in the same region and hypothesized that the adult plant resistance QTL detected in the source mapping population was actually divisible into two QTL – one coincidental with a seedling resistance QTL, and the other coincidental with a heading date QTL.

The results of the current experiments suggest the presence of another QTL proximal to QTL4. There is evidence that resistance genes in plants occur in clusters (Ellis et al. 1998; Kanazin et al. 1996; Michelmore 1995), and QTL mapping tools are known to provide poor resolution in the case of linked QTL (Lynch and Walsh 1997). QTL analysis in a new, larger population of 422 DH lines derived from the cross of BCD47 × Baronesse is under way and should be of assistance in resolving the number of QTL on chromosome 4(4H). Preliminary results (Vales et al. 2002) reveal multiple resistance significant QTL throughout the long arm of chromosome 4.

Our results support the utility of molecular markers and QTL analysis for understanding and manipulating genes determining qualitative and quantitative resistance to barley stripe rust. We mapped one qualitative resistance gene (*Rpsx*) and validated its importance, as well as the importance of two QTL (QTL4 and QTL5) that determine quantitative resistance to barley stripe rust, in a new genetic background. We also found that, in the populations under study, the effects of QTL estimates based on derived populations were different from those in the original mapping populations. Whether this is due to bias or genetic background is not known. We found that in the case of these genes, qualitative and quantitative gene interactions and QTL × environment interaction

were not significant. Preliminary data indicate that combining qualitative and quantitative resistance genes in the same genotype can confer high levels of resistance in the presence of a race virulent on the qualitative resistance gene and causing higher severity on the qualitative resistance source.

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