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Identification of QTLs for powdery mildew and scald resistance in barley

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Abstract A population of 103 recombinant inbred lines (RILs, F_9-derived lines) developed from the two-row spring barley cross L94×'Vada' was evaluated under field conditions for resistance against powdery mildew (*Blumeria graminis* f.sp. *hordei*) and scald (*Rhynchosporium secalis*). Apart from the major resistance gene *mlo* on chromosome 4 (4H), three QTLs (*Rbgq1, Rbgq2* and *Rbgq3*) for resistance against powdery mildew were detected on chromosomes 2 (2H), 3 (3H), and 7 (5H), respectively. *Rbgq1* and *Rbgq2* have not been reported before, and did not map to a chromosome region where a major gene for powdery mildew had been reported. Four QTLs (*Rrsq1, Rrsq2, Rrsq3* and *Rrsq4*) for resistance against scald were detected on chromosomes 3 (3H), 4 (4H) and 6 (6H). All four mapped to places where QTLs for scald resistance had been reported before in different populations.

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Introduction

Powdery mildew (*Blumeria graminis* f.sp. *hordei*) and scald (*Rhynchosporium secalis*) are two of the most economically important foliar diseases of barley (*Hordeum vulgare*) (Shipton et al., 1974; Balkema-Boomstra & Mastebroek, 1995). Powdery mildew and scald can be controlled by the use of fungicides, but the use of genetic resistance is the most effective, economic and environmentally sound way to control these diseases. Twenty three different major genes for resistance against powdery mildew and fifteen major genes for resistance against scald have been reported so far (Chelkowski et al., 2003; Genger et al., 2005). Almost all these R-genes give a hypersensitive type of resistance. However, nearly all the R-genes that are being deployed in commercially grown cultivars had a short effective life with the exception of *mlo* resistance gene (Hovmøller et al., 2000; Xi et al., 2002). This ephemeral effectiveness of resistance has caused breeders to look for other types of resistance such as partial resistance (PR) which appears to be more durable and race-non-specific (Niks et al., 2000). PR has been defined as a resistance causing a reduced rate of epidemic development despite a high, susceptible, infection type (Parlevliet, 1975). In the barley – *Puccinia* *hordei* pathosystem, this PR inherits polygenically (Qi et al., 1998b). Polygenically inherited PR has also been identified against powdery mildew and scald (Asher & Thomas, 1987; Kari & Griffith, 1997). The relation between QTLs for PR and major genes for hypersensitivity is still a matter of debate. QTLs for PR may be allelic variants of R-genes or may govern a separate system of defence on its own. Where PR is based on a different mechanism than post-haustorial hypersensitivity, and the genes do not map to loci known to carry also major genes for hypersensitive resistance, it has been suggested that PR has a different evolutionary origin than major-genic hypersensitivity resistance (Qi et al., 1998b). Such an assumption, of course, can never be sure, since *H. vulgare* and more in particular its wild ancestor *H. spontaneum*, probably contain more loci for R-genes than have been described. Where minor genes for PR co-localise to loci for major-genic resistance (e.g. in Backes et al., 2003), the minor genes for PR may be allelic versions of major genes for hypersensitivity, or be defeated alleles of such major genes. Evidence for the latter has been found in rice resistance to rice bacterial blight (*Xanthomonas oryzae* pv. *oryzae*) (Li et al., 2001). Conclusions on a possible common or different evolutionary origin await cloning and sequence analysis of the first gene for partial resistance.

The use of molecular markers has facilitated the construction of extensive linkage maps in barley (Graner et al., 1991; Qi et al., 1998a). By the availability of such dense linkage maps, individual gene loci (Quantitative trait loci, QTLs) for disease resistance have been identified and mapped in different genetic backgrounds (Jahoor et al., 2004; Hayes et al., 2005). QTL mapping is a highly effective means for studying genetically complex characters such as quantitatively inherited resistance. We can not only determine and compare the loci involved in a resistance, but also the relative size of their effect on the epidemic, the parent contributing the allele for resistance and the effectiveness of each locus in various plant development stages or against various pathogen isolates (Qi et al., 1998b, 1999).

The objective of this study was the identification of genes for resistance to powdery mildew (*Blumeria graminis* f.sp. *hordei*) and scald (*Rhynchosporium secalis*) in a population of 103 recombinant inbred lines (RILs) derived from a cross between L94 × ‘Vada’ and comparing the map positions of QTLs for resistance against powdery mildew and scald with those of major resistance genes and QTLs reported in literature.

Materials and methods

Plant material

The resistance tests were performed on a population of 103 recombinant inbred lines (RILs, F_9-derived lines) developed from the two-row spring barley cross L94 × ‘Vada’. L94 is an Ethiopian landrace with black and naked seeds, is very susceptible to leaf rust (Qi et al., 1998b) and resistant to powdery mildew (Jørgensen, 1992) and resistant to scald (D. Rubiales, unpublished data). The L94 line is one of the Ethiopian lines known to carry the *mlo* gene for resistance to powdery mildew (Jørgensen, 1992). ‘Vada’ is a West-European cultivar with white and covered seeds that carries a high level of PR to leaf rust (Parlevliet, 1975). It is known to possess the *MlLa* major gene for powdery mildew resistance (Giese et al., 1993), and it appears to have a low level of PR to scald (Rowling & Jones, 1976). Since virulence to *MlLa* occurs commonly in Europe (Hovmøller et al., 2000), ‘Vada’ is readily infected in the field. The 103 RILs and their two parents were used in all tests. The same mapping population had been used to map QTLs for PR to barley leaf rust (*Puccinia hordei*) (Qi et al., 1998b; 1999) and leaf stripe (*Pyrenophora graminea*) (Arru et al., 2002).

Disease assessments

Field testing was performed at CIFA experimental farm at Córdoba, Spain, over the course of three years (2002, 2003, 2004). RILs were sown in a randomised complete block design in three replicates. Each RIL was represented by 25–30 seeds in a single row of one meter long per replicate. No artificial inoculation was performed since mildew and scald incidence is usually high at this location. RILs alternated with single rows of ‘Vada’ as reference and as spreader for the two pathogens. One row of the parent L94 was sown per 20 RILs. Disease severity was estimated (one, two or three times per season, depending on pathogen and year with two weeks interval) during the heading stage as the percentage of leaf area covered by powdery mildew colonies or scald lesions.

To verify the *mlo*-carrying RILs, as determined by Qi et al. (1998a), a detached leaf test was performed. About 15 seeds per RIL were sown in 7 × 7 × 11 cm pots. A central leaf segment of about 50 mm per seedling was excised 11 days after sowing (first leaf fully expanded), and placed, adaxial surface up, in a square petri dish (12 × 12 cm) filled with 0.6% agar and 125 ppm Benzimidazole. During both growing seasons, a sample of powdery mildew was collected from the field of Córdoba and was maintained on a set of seedlings of various cultivars of which 'Vada' appeared to be the most susceptible. Two days before the inoculation of the RILs, 'Vada' stock plants were shaken to discard old spores. Fresh spores were blown from the infected 'Vada' stock over the leaf segments, in a settling tower. Every inoculation comprised 10 petri dishes together in the tower. In each petri dish, 4 segments per RIL (24 segments in total) were laid out. A glass slide was placed among the petri dishes to monitor the inoculum density, which was adjusted to give approximately 20 conidia mm^{-2}. After inoculation, petri dishes were transferred to a growth chamber at 18–20 °C and incubated in darkness for 12 h. Then they were transferred to a growth chamber with fluorescent light (12 h light/12 h dark) and 18–20 °C (Edwards, 1993). Infection type (IT) was recorded five days after inoculation, following the 0–4 scale of Mains and Dietz (1930). This scale was extended by the score 0(4) for the presence of few colonies, which is characteristic for resistance due to *mlo* (Czembor, 2001). To separate the RILs carrying *mlo* from the RILs with high level of PR, infection frequency was recorded by counting the number of powdery mildew colonies in one square cm.

During the experiment period, we determined the virulence factors present in the powdery mildew population by exposing a set of 17 of the most commonly used differentials of Pallas-near-isogenic lines (Hovmøller et al., 2000) to powdery mildew spores collected in the field following the procedure described above.

Powdery mildew severity was scored twice every year. During 2002 no epidemic of powdery mildew developed. For each RIL the average severity over both evaluations was calculated and used for QTL-mapping. Scald severity was estimated once in 2002 and three times in 2004 and the average severity over the three evaluations was calculated and used for QTL-mapping. Plant height and flowering dates were scored for RILs and parental lines.

Statistical analysis and mapping

The ANOVA was calculated by using the PROC GLM in SAS programme (SAS Institute 1988). The wide-sense heritability (h^2) for the level of infection by two pathogens was estimated.

A dense marker map of L94 × Vada was constructed with JoinMap 3.0 (Marcel et al., unpublished). A skeletal map with uniformly distributed markers (approximately 5 cM per marker interval) was extracted (Fig. 1). All the markers on the skeletal map fitted in the dense map during the first or exceptionally the second round of JoinMap. This new skeletal map was used for the QTL analysis.

A computer software package, MAPQTL version 5.0 modified from version 3.0 (Van Ooijen & Maliepaard, 1996), was used for interval mapping. In the region of the putative QTLs, the markers with the highest LOD values (peak markers) were used as co-factors for running a multiple-QTL mapping programme, the MQM method (Jansen & Stam, 1994). When LOD values of some markers on other regions reached the significance level, the MQM was repeated by adding those new 'peak markers' as co-factors until a stable LOD profile was reached. The restricted MQM method (rMQM) was used to determine the values of the LOD, phenotypic variation, additive effect and the confidence interval for the detected QTLs. A LOD value of 3 was chosen as threshold value for declaring a QTL (Qi et al., 1998b). The non-parametric option of MapQTL was used to test which markers contributed significantly to the severities observed.

Results

Powdery mildew

Few changes in the virulence composition of the powdery mildew population were detected during the two years' experiments. The powdery mildew population collected in 2003 was avirulent to *Mla3, Mla6, Mla13, Mla14, MlRu3*, *Mlat* and *mlo*. The mildew population collected in 2004 was avirulent to *Mla3, Mla13, MlRu3* and *mlo*. Therefore in both years virulence to the *MlLa* resistance gene was present in the mildew population. As expected *mlo* was effective to both years' mildew populations.

In the seedling test, the 103 RILs segregated for infection type. 52 RILs showed infection type 0 or 0(4) and 51 RILs showed infection type 3 or 4. The resistant RILs (IT 0, 0(4)) showed an infection frequency of 0 to 12 colonies per cm^2 and the susceptible RILs (IT 3–4)

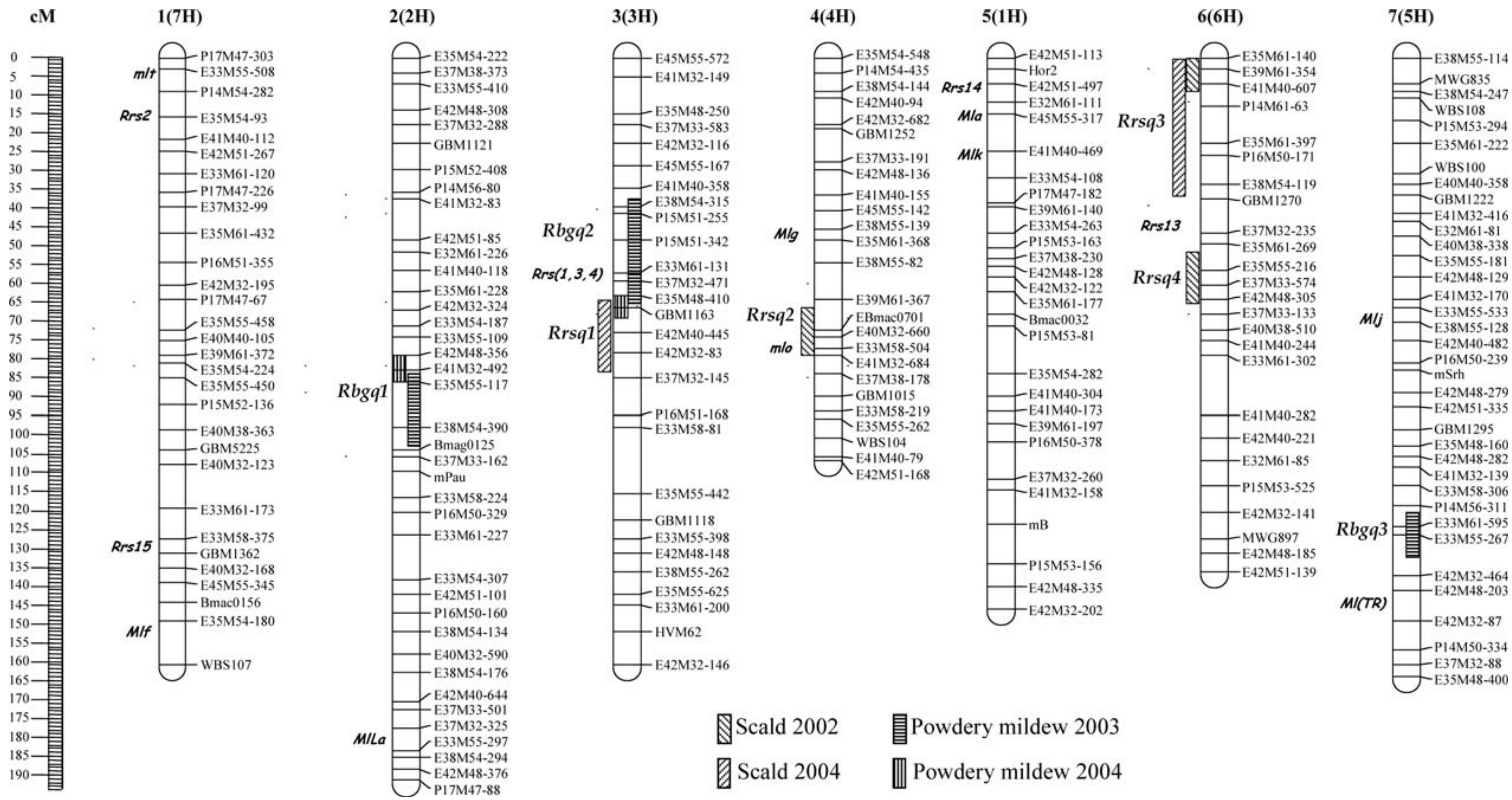

Fig. 1 Location of QTLs for resistance to powdery mildew and scald on the skeletal map of barley, based on 51 RILs for powdery mildew and 103 RILs for scald, from a cross L94 × 'Vada'. Chromosomes were oriented with the short arms to the top. Kosambi's mapping function was used. Names of QTLs are on the left of each QTL box. Boxes left to the chromosome bars are the QTLs for scald in 2002 and 2004, and boxes inside the chromosome bars are the QTLs for powdery mildew in 2003 and 2004. Length of bars corresponds to two LOD support intervals (from peak) based on the results of the rMQM method. On the left of the chromosome bars all reported loci of race specific resistance genes against powdery mildew (*Ml*) and scald (*Rrs*) are indicated

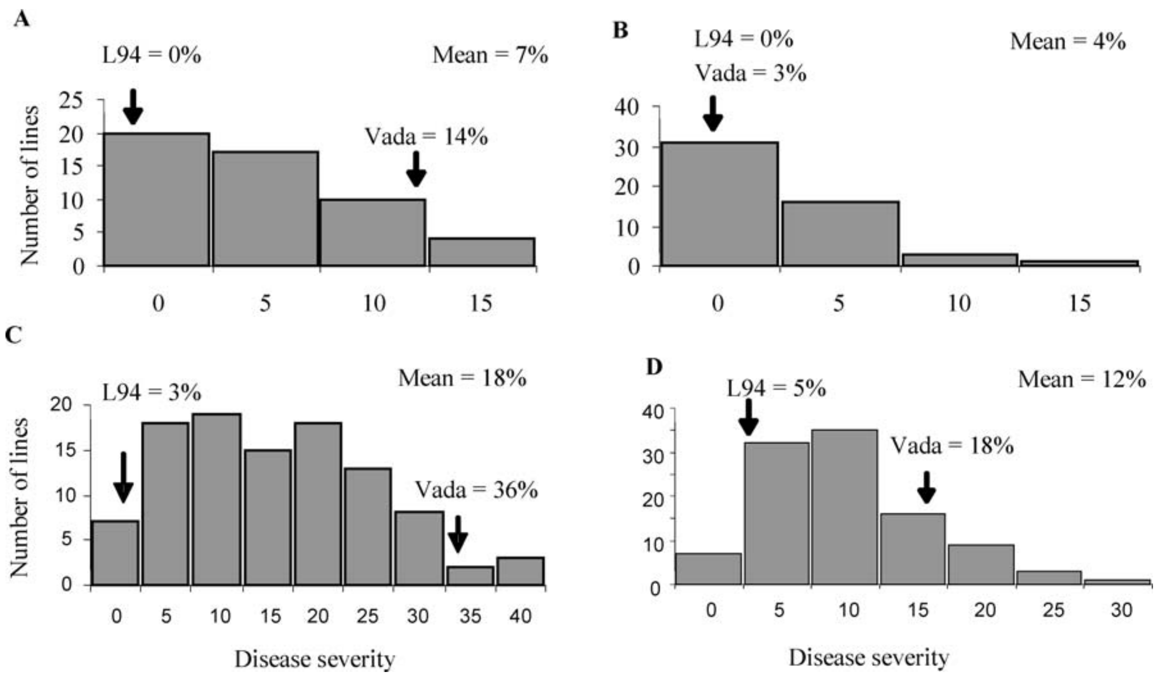

Fig. 2 (*A-D.*) Frequency distribution of phenotypes for powdery mildew (51 RILs F_9) and scald (103 RILs F_9) derived from a cross L94 × 'Vada'. (A): Powdery mildew severity in the field during 2003. (B): Powdery mildew severity in the field during 2004. (C): Scald severity in the field during 2002. (D): Scald severity in the field during 2004. The values indicated on the x-axis are the lower limit of each class

showed an infection frequency of 76 to 133 colonies per cm^2, without intermediate cases. This observed ratio of 52:51 (resistant: susceptible) does not deviate significantly from the 1:1 ratio suggesting a single gene for resistance. We mapped the resistance gene at 78.6 cM from the tip of the short arm of Chromosome 4 (4H), indicating, as expected, that this gene is the *mlo* resistance locus.

In the field the RILs showed the same observed segregation ratio. For QTL mapping only the 51 RILs with IT 3–4 (*Mlo* allele) were used. They segregated quantitatively and significantly in severity of infection ($P = 0.001$; Duncan test). Figures 2A–B shows the frequency distribution of the 51 RILs during the two years. Disease severity was higher in 2003 than in 2004 (population mean 7% and 4% respectively). L94 and 'Vada' had disease severities of 0% and 14% (2003) and of 0% and 3% (2004) respectively with RILs ranging from 0% to 16% in both years. The heritability was about 0.4 in both years. Correlation between the values for the RILs between the two years was moderate but highly significant ($r = 0.46$, $n = 51$; $P < 0.01$). The low general level of infection was unlikely to cause substantial, if any, competition for green leaf tissue with the other pathogen, scald.

In 2003 no obvious transgressive segregation was detected in this population since none of the RILs showed disease severity significantly higher than 'Vada' (Duncan, $P \leq 0.05$). In 2004 a strong transgressive segregation towards susceptibility was found since many RILs showed disease severity significantly higher than in the most susceptible parent 'Vada' (Duncan, $P \leq 0.05$).

In total three QTLs for PR to powdery mildew were identified and were tentatively named as *Rbgq* (**R**esistance locus to **B***lumeria* **g***raminis* that is **q**uantitative) (Table 1 and Fig. 1). Two QTLs (*Rbgq1* and *Rbgq3*) were detected on chromosomes 2 (2H) and 7 (5H) respectively with the resistance allele from 'Vada' (Table 1). *Rbgq1* was detected for both years' data whereas *Rbgq3* was detected in the 2003 experiment only. One more QTL was detected for both years' data and was designated *Rbgq2*, located on the short arm of chromosome 3 (3H) with the resistance allele from L94. The total phenotypic variation explained by the QTLs detected in the population was 36.2% in 2003 and 35.8% in 2004.

Table 1 Summary of QTLs for disease severity of powdery mildew (*Blumeria graminis* f.sp. *hordei*) identified in the cross L94 × 'Vada'

		2003				2004			
QTL	Chromosome	cM	LOD	Exp%	Add[b]	cM	LOD	Exp%[a]	Add[b]
*Rbgq*1	2 (2H)	100.5	**3.6**	**14.1**	**1.9**	84.6	**5.1**	**15.3**	**1.5**
*Rbgq*2	3 (3H)	60.4	**3.4**	**10.2**	**−1.6**	67.3	**6.4**	**20.5**	**−1.8**
*Rbgq*3	7 (5H)	130.3	**3.9**	**11.9**	**1.6**	130.3	0.3	1.1	−0.5
Total[c]				**36.2**	**1.9**			**35.8**	**−0.3**

[a]The proportion of the explained phenotypic variation
[b]Effect of alleles from L94
[c]Sum of the values of the significant QTLs (**Bold** font)

Table 2 Summary of QTLs for disease severity of scald (*Rhynchosporium secalis*) identified in the cross L94 × 'Vada'

		2002				2004			
QTL	Chromosome	cM	LOD	Exp%[a]	Add[b]	cM	LOD	Exp%[a]	Add[b]
*Rrsq*1	3 (3H)	67.3[d]	1.9	5.0	−2.3	73.7	**4.1**	**11.1**	**−2.1**
*Rrsq*2	4 (4H)	72.7	**3.9**	**8.3**	**−3.0**	72.7[d]	2.6	7.3	−1.6
*Rrsq*3	6 (6H)	3.5	**5.3**	**13.9**	**−3.8**	3.5	**3.3**	**8.9**	**−1.8**
*Rrsq*4	6 (6H)	56.6	**4.2**	**11.2**	**3.5**	56.6	0.0	0.0	−0.1
Total[c]				**33.4**	**−5.6**			**20.0**	**−3.9**

[a]The proportion of the explained phenotypic variation
[b]Effect of alleles from L94
[c]Sum of the values of the significant QTLs (**Bold** font)
[d]Although not significant by rMQM mapping, this locus contributed significantly according to the non-parametric test

Scald

During 2002, the disease severity of the 103 RILs ranged from 0 to 43% and 1% to 32% in 2004 (Figs. 2C and D). L94 and 'Vada' had disease severities of 3% and 36% in 2002 and during 2004 it was 5% and 18% respectively. The RILs differed significantly in level of infection ($P = 0.001$; Duncan test). The heritability (h^2) was 0.58 and 0.65 for 2002 and 2004 respectively. No transgressive segregation was detected in 2002 but it was in 2004 (Figs. 2C and D). Correlation between the values for the RILs between the two years was moderate but highly significant ($r = 0.44$, $n = 103$; $P < 0.01$). The moderate level of infection was unlikely to cause substantial if any competition for green leaf tissue with the other pathogen, powdery mildew. All 103 RILs were used for QTL-mapping to scald.

In total, four QTLs for PR were identified. They were tentatively named as *Rrsq* (**R**esistance locus to **R***hynchosporium* **s***ecalis* that is **q**uantitative) (Table 2 and Fig. 1). One QTL (*Rrsq3*) was detected both years in the same confidence interval on chromosome 6 (6H). Two QTLs (*Rrsq2* and *Rrsq4* on chromosomes 4 (4H) and 6 (6H) respectively) were detected only in the 2002 experiment. Another QTL (*Rrsq1* on chromosome 3 (3H)) was detected only in the 2004 experiment. However, the effects of *Rrsq1* in 2002 and of *Rrsq2* in 2004 were significant according to the non-parametric test. The total phenotypic variation explained by the QTLs detected in the population was 33.4% in 2002 and 20.0% in 2004. The *Rrsq2* resistance allele was located at less than 6 cM proximally to the *mlo*-recessive allele for powdery mildew resistance (Fig. 1). The resistance alleles of *Rrsq1, Rrsq2* and *Rrsq3* were from L94 while the resistance allele of *Rrsq4* was from 'Vada'.

Possible effect of crop architecture

A possible disturbing effect in measuring the resistance could be due to the crop architecture. L94 is poorly adapted to modern agronomic conditions because of its tall stature. This might cause an associated variation in microclimate between RILs. However, the correlations between severity of infection and plant height were in all cases close to zero, ruling out a substantial epidemiological effect of plant height on both pathogens.

Discussion

Many research groups have now reported QTLs for PR in barley populations (e.g. Heun, 1992; Thomas et al., 1995; Qi et al., 1998b; Arru et al., 2002). Most reported QTLs are listed in Hayes et al. (2005); Jahoor et al. (2004). The resistance of our population parents against powdery mildew was assumed to be based on the *mlo* and *MlLa* resistance genes (Giese et al., 1993; Qi et al., 1998a). The *mlo* resistance gene was mapped on the long arm of chromosome 4 (4H). The *MlLa* resistance gene has been reported to be located on the distal part of the long arm of chromosome 2 (2H) (Giese et al., 1993). The Pallas-*MlLa* near-isogenic line in our differential set indicated that in both years the powdery mildew population was virulent to *MlLa*. In agreement with this, we did not find any effect of the *MlLa* locus on the severity in the QTL-mapping, not even in 2004 when 'Vada' was almost completely resistant.

Three QTLs for resistance against powdery mildew were identified. Two of these, *Rbgq1* and *Rbgq2*, that we detected in both years on chromosomes 2 (2H) and 3 (3H), have not been reported before. The fact that they were detected in both years lends confidence to the reliability of these QTLs. The other QTL, *Rbgq3,* mapped at 130 cM from the tip of the short arm on chromosome 7 (5H) and is at a similar position as a QTL reported by Spaner et al. (1998) and by Saghai Maroof et al. (1994). It is possible that some other QTLs for powdery mildew resistance with smaller effects were not identified due to the small population size (51 RILs) and the low level of infection and, as a consequence, the modest contrast in infection severity between the RILs. In both years' data both parents contributed to the resistance, implying that in both years we should expect transgressive segregation. Such transgressive segregation occurred in 2004, but not in 2003. At present we have no explanation for this.

There is no indication that QTLs *Rbgq1* and *Rbgq2* detected on chromosomes 2 (2H) and 3 (3H) are allelic to a major gene for powdery mildew resistance since no such major gene has been mapped at these positions (Chelkowski et al., 2003; Jahoor et al., 2004). However, QTL *Rbgq3* for powdery mildew resistance on chromosome 7 (5H) co-locates with the major gene for powdery mildew resistance designated as *Ml(TR)* in the Harrington × TR306 cross (Falak et al., 1999). This gene caused an infection type 1–2 in the study of Falak et al. (1999). Since there is no *Ml*(*TR*)-Pallas near-isogenic line available, virulence against *Ml*(*TR*) has not been quantified in surveys in Europe (Hovmøller et al., 2000). Jørgensen (cited by Falak et al., 1999) reported virulence to this gene in European powdery mildew. Since *Ml*(*TR*) has been discovered recently in a Canadian breeding line, it is not likely that 'Vada' carries this resistance gene.

In the present study, four QTLs for PR against *R. secalis* were detected on chromosomes 3 (3H), 4 (4H) and 6 (6H). Both the resistant and the susceptible parents (L94 and 'Vada' respectively) contributed alleles for resistance against scald.

QTL *Rrsq1* on chromosome 3 (3H) mapped at a region where a QTL has been detected in three mapping populations. It occurred in the Blenheim × E224/3 cross with the resistance allele contributed by E224/3 (Thomas et al., 1995), in the Harrington × TR306 cross with the resistance allele contributed by Harrington (Spaner et al., 1998), and in the Ingrid × Abyssinian cross with the resistance allele contributed by Abyssinian (Grønnerød et al., 2002). QTL *Rrsq1* may be the same as the QTL detected in the Ingrid × Abyssinian since both Abyssinian and L94, also known as Abyssinian 1102, are of Ethiopian origin (Jørgensen, 1992). QTL *Rrsq2* on chromosome 4 (4H) may correspond to the QTL for scald resistance detected in the Harrington × TR306 cross with the resistance allele contributed by TR306 (Spaner et al., 1998). In our population the confidence interval of *Rrsq2* contained the locus *mlo*. Further studies should be made to determine whether the recessive *mlo* locus and the QTL allele for scald resistance are genetically linked or a pleiotropic effect of *mlo*. QTL *Rrsq3* on the distal part of the short arm of chromosome 6 (6H) may correspond to a QTL at a similar mapping position in Alexis × Regatta cross (Jensen et al., 2002).

QTL *Rrsq4* detected on the proximal part of the short arm of chromosome 6 (6H) may coincide with a QTL detected in two mapping populations. It occurred in the 'Igri' × 'Danilo' cross (Backes et al., 1995), and in the Harrington × TR306 cross (Spaner et al., 1998). In conclusion, all the QTLs for PR against scald detected in the present L94 × 'Vada' cross are supported by previously mapped QTLs in various barley populations.

Major genes for resistance against scald have been mapped to barley chromosomes 1 (7H), 3 (3H), 4 (4H), 5 (1H) and 6 (6H) (Schweizer et al., 1995; Graner & Tekauz, 1996; Garvin et al., 2000; Genger et al., 2005). We found two QTLs (*Rrsq3* and *Rrsq4*) for scald resistance on chromosome 6 (6H) and it is unlikely that any one of these two QTLs co-locates with the *Rrs13* major gene for scald resistance which is reported to occur on chromosome 6 (6H) (Abbott et al., 1995). QTL *Rrsq2* detected on chromosome 4 (4H) does not coincide with any known major gene for resistance against scald. QTL *Rrsq1* detected on chromosome 3 (3H) maps at a similar position as the complex locus, *Rh*(*Rrs1*)-*Rrs3*-*Rrs4*, for scald resistance (Graner & Tekauz 1996).

Some QTLs identified in this study (*Rbgq3, Rrsq1, Rrsq2* and *Rrsq4*) were found only in one out of the two years' trials, although for *Rrsq1, Rrsq2* the non-parametric test indicated a significant contribution in both years. Effectiveness in only one trial may be due to inconsistent expression of those QTLs, to isolate specificity of those genes or to experimental error.

The present study demonstrated that this population segregated for at least three QTLs for powdery mildew resistance and four QTLs for scald resistance. At least *Rbgq1*, *Rbgq2,* for PR to powdery mildew and QTLs *Rrsq2, Rrsq3* and *Rrsq4* for PR to scald do not co-localise with known major genes for hypersensitive resistance. It would be interesting to determine their mode of action to the pathogen in order to decide whether they may constitute a separate class of genes for PR, as reported in the barley leaf rust system (Qi et al., 1998b).

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