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X. Qi · G. Jiang · W. Chen · R.E. Niks · P. Stam
P. Lindhout

Isolate-specific QTLs for partial resistance to *Puccinia hordei* in barley

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Abstract By using a high-density AFLP marker linkage map, six QTLs for partial resistance to barley leaf rust (*Puccinia hordei*) isolate 1.2.1. have been identified in the RIL offspring of a cross between the partially resistant cultivar 'Vada' and the susceptible line L94. Three QTLs were effective at the seedling stage, and five QTLs were effective at the adult plant stage. To study possible isolate specificity of the resistance, seedlings and adult plants of the 103 RILs from the cross L94×'Vada' were also inoculated with another leaf rust isolate, isolate 24. In addition to the two QTLs that were effective against isolate 1.2.1. at the seedling stage, an additional QTL for seedling resistance to isolate 24 was identified on the long arm of chromosome 7. Of the eight detected QTLs effective at the adult plant stage, three were effective in both isolates and five were effective in only one of the two isolates. Only one QTL had a substantial effect at both the seedling and the adult plant stages. The expression of the other QTLs was developmental-stage specific. The isolate specificity of the QTLs supports the hypothesis of Parlevliet and Zadoks (1977) that partial resistance may be based on a minor-gene-for-minor-gene interaction.

Key words Barley · *Puccinia hordei* · Partial resistance · QTL mapping · Isolate specificity

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X. Qi (✉) · G. Jiang · W. Chen · R.E. Niks · P. Stam
Graduate School of Experimental Plant Sciences,
Department of Plant Breeding,
Wageningen Agricultural University, PO Box 386,
NL-6700 AJ Wageningen, The Netherlands

Present addresses:
X. Qi, John Innes Centre, Colney Lane, Norwich NR4 7UH, UK
e-mail: xiaoquan.qi@bbsrc.ac.uk
Fax: +44-1603456844
G. Jiang, Nanjing Agriculture University, Nanjing, China
W. Chen, Institute of Plant Protection, CAAS,
Beijing 100095, China

Introduction

In many plant-pathogen systems two types of resistance occur side by side. One is based on a hypersensitive reaction and is clearly race-specific. This race specificity has been explained by assuming a gene-for-gene interaction (Flor 1956, 1971). Van der Plank (1963, 1968) called this type of resistance a 'vertical' resistance. Molecular analysis of the cloned vertical resistance genes and the corresponding avirulence genes from several plant-pathogen systems have revealed that this model most likely holds true at the molecular level (Van den Ackerveken et al. 1992; Joosten et al. 1994, 1997; Ellis et al. 1997; see review of Hammond-Kosack and Jones 1997), although the resistance gene product itself is probably not the receptor for the corresponding avirulence gene product (Kooman-Gersman et al. 1998). Generally, this vertical resistance is associated with the hypersensitive response and is not durable [e.g. *Rph* genes in barley (Jin et al. 1995)].

The second type of resistance is quantitative, and in many cases not based on hypersensitivity. Quantitative resistance that is not based on hypersensitivity was termed 'partial' resistance by Parlevliet (1975). Partial resistance was initially considered race-non-specific and more durable, and therefore fitted Van der Plank's concept of 'horizontal' resistance. Van der Plank (1963, 1968) presumed that the quantitative resistance genes for horizontal resistance were equally effective for all pathogen isolates. However, more detailed observations showed that small but significant cultivar×isolate interactions may occur (Caten 1974; Clifford and Clothier 1974; Parlevliet 1976a, 1977). According to Parlevliet and Zadoks (1977), these interactions can only be explained by assuming a minor-gene-for-minor-gene interaction, similar to the system known in vertical resistance.

Nowadays, many quantitative traits, including quantitative resistance, have been resolved into discrete genetic loci (QTLs, quantitative trait loci). These QTLs were mapped on plant genomes by using molecular-marker

linkage maps (Paterson et al. 1988; Tanksley 1993; Young 1996). In barley, two QTLs for quantitative resistance to powdery mildew were identified by using the 'Proctor'/'Nudinka' RFLP map (Heun 1992) and later, by using the 'Igri'/'Danilo' map, two QTLs were detected for resistance to powdery mildew based on field data (Backers et al. 1996). One major-effect and one minor-effect QTL for resistance to barley stripe rust (*Puccinia striiformis* f.sp. *hordei*) were mapped on barley chromosomes 7L and 4L (Chen et al. 1994). Several QTLs for resistance to leaf rust, stripe rust, mildew and *Rhynchosporium* were mapped on barley chromosomes by using 59 doubled-haploid lines derived from a spring barley cross between cv 'Blenheim' and the line E224/3 (Thomas et al. 1995). One major, one moderate and two minor QTLs conferring quantitative resistance to barley leaf stripe (*Pyrenophora graminea*) were identified and mapped on barley chromosomes by Pecchioni et al. (1996). By using the high-denisty 'Steptoe'/'Morex' RLFP map, alleles of two or three unlinked loci were found to confer resistance to the net blotch pathogen (*Pyrenophora teres* f. *teres*) at the seedling stage, and seven QTLs were identified for resistance at the adult plant stage. A single gene was found to control resistance to the spot blotch (*Cochliobolus sativus*) pathogen at the seedling stage and two QTLs were detected for resistance at the adult plant stage (Steffenson et al. 1996). Recently, by using a high-density AFLP linkage map (Qi et al. 1998b), six QTLs for partial resistance to barley leaf rust (*Puccinia hordei*) isolate 1.2.1. have been identified in a recombinant inbred population from a cross between cultivar 'Vada' and the line L94 (Qi et al. 1998a). Three QTLs, *Rphq1*, *Rphq2* and *Rphq3*, were effective at the seedling stage, and five QTLs, *Rphq2*, *Rphq3*, *Rphq4*, *Rphq5* and *Rphq6*, were effective at the adult plant stage. These QTLs acted predominantly in an additive fashion; all of the resistance alleles derived from the partially resistant parent 'Vada'.

In the investigations cited above, the question of the race-specificity of partial resistance was not addressed. With the QTL approach, however, we are in a position to investigate to what extent QTLs that contribute to quantitative resistance are isolate- or rare-specific in their action. Such an approach may throw light upon the existence of the minor-gene-for-minor-gene interaction hypothesized by Parlevliet and Zadoks (1977) as a basis for quantitative resistance. The object of the present study was to resolve this question for the barley-barley leaf rust system. To this end seedlings and adult plants of the mapping population derived from the cross of L94×'Vada', used in our earlier study, were inoculated with another rust isolate, isolate 24. We reasoned that a comparison of the QTLs for resistance to the two different isolates would reveal the race-specificity of partial resistance genes if such specificity exists.

Materials and methods

Plant materials

A set of 103 F_9 recombinant inbred lines (RILs) derived from a cross of L94×'Vada' that was used to map QTLs for resistance to barley leaf rust (*P. hordei*) isolate 1.2.1. (Qi et al. 1998a) was also used in this study. L94 is extremely susceptible, and 'Vada' has a high level of partial resistance to *P. hordei* (Parlevliet 1975, 1976b).

Leaf rust

Barley leaf rust isolate 24 was collected about 5 km south-east of Aalten in Achterhoek of the Netherlands in October, 1974. Isolate 1.2.1. which was used in our previous research (Qi et al. 1998a) was a monospore culture derived from isolate 1–2 which was collected in Wageningen in September 1971 (Parlevliet 1976a). A monospore subculture of both isolates was stored in liquid nitrogen. Fresh inoculum was produced on adult plants of the susceptible line L98. The isolates were reproduced in isolated greenhouse compartments in order to maintain their purity. The two isolates were tested on the differential series proposed by Clifford (1977) to which CI 1234, *Pa9* (*Raph9*), and Triumph, *Pal2* (*Rphl2*), were added.

Map construction

Qi et al. (1998b) contructed a dense linkage map covering the barley genome (1062 cM), containing 566 AFLP markers (Qi and Lindhout 1997). A skeletal map with a uniform distribution of markers with about 5-cM intervals was extracted and used for mapping QTLs for resistance to leaf rust isolate 24.

Disease evaluations at the seedling and at the adult plant stage

Leaf rust isolate 24 was used to inoculate seedlings and adult plants of the 103 RILs and the two parents, L94 and Vada. The method of evaluation for resistance to isolate 24 was the same as for that to isolate 1.2.1. (Qi et al. 1998a). Seeds from the mapping population were sown in two rows in small trays (30×30 cm). In each tray both parents were included. About 10 days after sowing, the seedling leaves were fixed horizontally to the soil. Four to five seedlings per RIL were used for inoculations. Fresh urediospores (about 150 spores per cm^2 leaf area) were diluted ten-times with lycopodium spores and dusted over the adaxial sides of the seedling leaves in an inoculation tower. After incubation at a relative humidity of 100% overnight, the trays were moved into a greenhouse where the temperature was set at about 18°C. The latent period (LP50) of each plant was evaluated by estimating the period (in h) at which 50% of the ultimate number of postules became visible. The relative latent period in seedlings (RLP50S) was calculated relative to the LP50 of L94, where L94=100 (Parlevliet 1975). Three replications were conducted in the course of 2 years (1996 and 1997). Since ANOVA did not show significant year×line interaction, the average values of the three replications were used for the further analysis.

Inoculation of adult plants took place when the flag leaves were just unfolded. Fresh urediospores (about 150 spores per cm^2 leaf area) of isolate 24 were diluted 10-times with lycopodium spores and dusted over the plants in the incubation room. Afterwards, a relative humidity of 100% was set and plants were kept in the incubation room overnight. The next day, the plants were placed in a greenhouse at about 15–18°C. The relative latent period in young flag leaves (RLP50A) was measured in the same way as the RLP50S. One experiment with three pots per RIL was carried out in 1997. Three to six young flag leaves per pot were used for measuring the LP50. Because of the large number of RILs and their differences in maturity, five inoculations were conducted in the course of about $1^1/_2$ months with one-week intervals. The

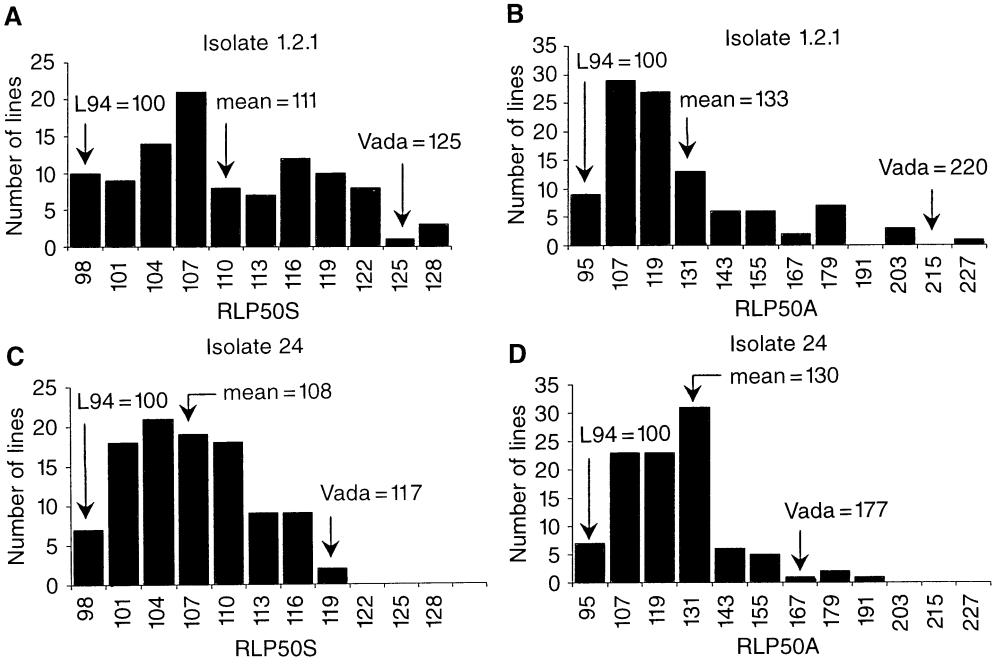


Fig. 1A–D Frequency distribution of phenotypes for the two measures of leaf rust resistance in 103 RILs derived from the cross L94×Vada. **A** RLP50S of isolate 1.2.1., **B** RLP50A of isolate 1.2.1., **C** RLP50S of isolate 24, **D** RLP50A of isolate 24. Values of L94 and Vada, and population mean values are shown by *arrows*. The values indicated on the *x*-axis are the lower limit of each category

plants were grouped and inoculated according to the stage when the flag leaves were just unfolded. In each inoculation, several L94 and 'Vada' plants were always included as controls.

Statistical analysis

ANOVAs were calculated by using the PROC GLM program (SAS Institute 1988). Wide-sense heritabilities (h^2) for RLP50S and RLP50A were estimated based on the results from ANOVAs. A computer program, MapQTL version 3.0, developed by Van Ooijen and Maliepaard (1996), was applied for interval mapping (Lander and Botstein 1989) and multiple-QTL mapping (MQM, Jansen 1993). Interval mapping was initially used to detect the region of putative QTLs. The marker with the highest LOD value was then taken as a co-factor for running a multiple-QTL mapping (MQM) program. This was repeated until a 'stable' LOD profile was reached. A LOD value of 3.0 was chosen as a significant threshold value for declaring a QTL. In this paper, results obtained with the MQM method are presented.

Results and discussion

Partial resistance to two leaf rust isolates

Partial resistance to a *P. hordei* isolate, isolate 24, was investiged in this study. Analysis of the RLP50S of isolate 24 obtained from three replications in the course of 2 years (data not shown) did not show significant year×line interaction. The average values of three replications were used for QTL mapping. The identification of QTLs for partial resistance to isolate 1.2.1. was described in our previous paper (Qi et al. 1998a). The results demonstrated that the expression of genes for partial resistance to barley leaf rust were insensitive to environmental conditions. A test on a differential set of barley cultivars (data not shown) indicated that the two isolates differed at least in their virulence spectrum to the hypersensitivity resistance genes *Rph5*, *Rph8*, *Rph9* and *Rph2*. The relative latent period of isolate 24 was shorter in both seedlings and adult plants of 'Vada' than that of isolate 1.2.1. The average relative latent period of isolate 24 on the 103 RILs was also lower than that of isolate 1.2.1. (Fig. 1). This indicates that isolate 24 is more aggressive than isolate 1.2.1.

In the 103 RILs, both RLP50S and RLP50A of isolate 24 at the seedling and the adult plant stages were approximately normally distributed (Fig. 1C, D). The relative latent periods of the RILs were between the values of the two parents, indicating an absence of transgression. The wide-sense heritabilities for RLP50S and RLP50A were 0.61 and 0.70 respectively.

A high correlation was found between the RLP50S of isolate 1.2.1. and that of isolate 24 (Table 1). A moderate correlation was observed between the RLP50A and RLP50S of isolate 24. The correlation between the RLP50A of the two isolates was weak but statistically significant.

QTLs for partial resistance to isolate 24

A multiple-QTL mapping method (MQM, Jansen 1993) was applied to identify QTLs for partial resistance to barley leaf rust isolate 24. A LOD value of 3.0 was set as the threshold value for declaring a QTL. Seven QTLs (Table 2 and Fig. 2) were detected by using a skeleton map extracted from a high-density AFLP map (Qi et al. 1998b). Three QTLs for RLP50S were identified, that collectively explained 45% of the phenotypic variance. Notably, when the 2-years' data sets were used separately for QTL mapping, the three QTLs were consistently identified with an identical ranking order of allelic effects (data not shown). Six QTLs were detected for

Table 1 Correlation coefficients (*r*) among two measures of partial resistance to two isolates of leaf rust in 103 RILs derived from the cross L94×Vada

Item	RLP50S-1.2.1.[a]	RLP50A-1.2.1.[a]	RLP50S-24[b]
RLP50A-1.2.1.[a]	0.42**		
RLP50S-24[b]	0.81**	0.37**	
RLP50A-24[b]	0.66**	0.40**	0.69**

** $P \leq 0.01$

[a] RLP50S-1.2.1. and RLP50A-1.2.1. are the RLP50 of isolate 1.2.1. measured on the 103 RILs in the seedling stage and in the adult plant stage respectively

[b] RLP50S-24 and RLP50A-24 are the RLP50 of isolate 24 measured on the 103 RILs in the seedling stage and in the adult plant stage respectively

Table 2 Summary of QTLs for partial resistance to leaf rust isolate 24

QTLs	RLP50S			RLP50A		
	LOD	Exp%[a]	Add[b]	LOD	Exp%	Add
Rphq7	4.7	6.3	1.4	–	–	–
Rphq2	16.3	29.2	2.9	5.3	10.9	6.3
Rphq3	6.6	9.4	1.8	6.5	13.7	7.5
Rphq4	–[d]	–	–	4.5	9.1	6.0
Rphq8	–	–	–	4.7	9.4	6.4
Rphq9	–	–	–	4.2	7.1	5.2
Rphq10	–	–	–	3.1	6.1	4.7
Total[c]		**44.9**	**6.1**		**58.6**	**36.1**

[a] The proportion of the explained phenotypic variance

[b] Effects of the alleles from 'Vada'

[c] Sum of the values of the significant QTLs (**bold** font)

[d] Only data with a LOD≥3.0 are presented

RLP50A (Table 2), together explaining 59% of the phenotypic variance. Comparison with the wide-sense heritabilities (0.61 and 0.70 for RLP50S and RLP50A, respectively) suggested that most of the genetic variance was explained by these QTLs. The resistance alleles of the seven QTLs all originated from the partially resistant parent, 'Vada'. No resistance allele was identified originating from L94. This result is in accordance with the absence of clear transgression in the RILs.

Developmental stage-dependent expression of QTLs

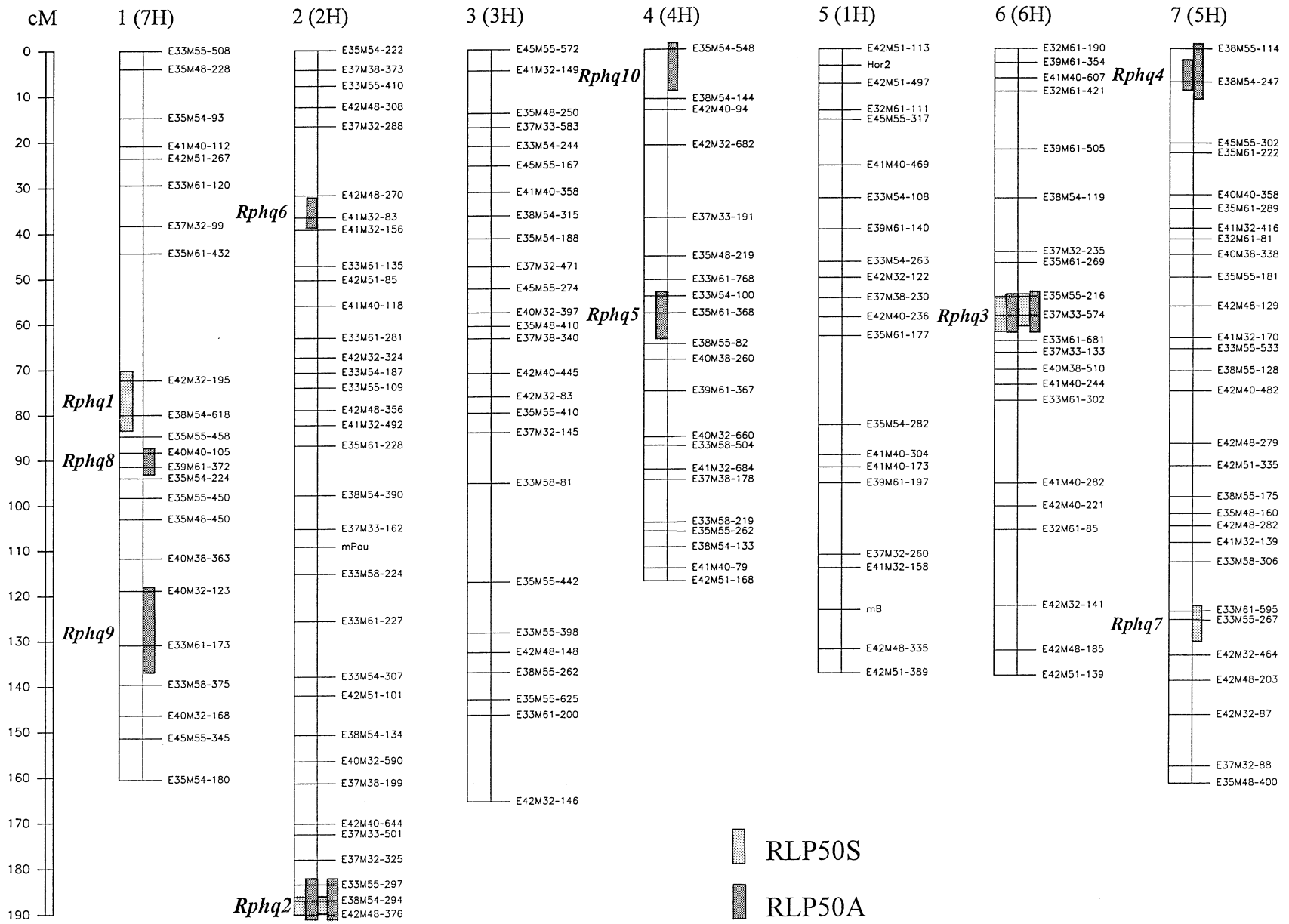
One QTL, *Rphq7*, was only effective at the seedling stage while four QTLs, *Rphq4*, *Rphq8*, *Rphq9* and *Rphq10*, were only effective at the adult plant stage (Table 2). Such a development-dependent expression of genes for partial resistance was also observed in our previous research (Qi et al. 1998a). It also agrees with the moderate correlation between RLP50S and RLP50A. As was the case with isolate 1.2.1., *Rphq2* and *Rphq3* were the only two QTLs effective at both plant development stages. However, *Rphq2* was highly effective at the seedling stage (RLP50S) but only moderately at the adult plant stage (RLP50A). *Rphq3* is the only QTL with a consistent effect at both plant stages.

Isolate-specific QTLs for partial resistance

The partial resistance to isolates 1.2.1. and 24 was resolved into ten QTLs. These QTLs were mapped on the barley genome (Fig. 2). Four QTLs were effective (Fig. 3A) at the seedling stage. Two of them, *Rphq2* and *Rphq3*, were consistently effective for both isolates, but had smaller effects in isolate 24 than in isolate 1.2.1. (Fig. 3A). *Rphq1* which had a weak effect in isolate 1.2.1. (found in the previous study, Qi et al. 1998a) showed no significant effect in isolate 24. In contrast, *Rphq7* on the long arm of chromosome 7, was only effective in isolate 24. Isolate-specificity of the QTLs for partial resistance was evident at the adult plant stage. Among the eight QTLs (Fig. 3B) identified for resistance to the two isolates, two QTLs, *Rphq5* and *Rphq6*, were only effective in isolate 1.2.1. and three, *Rphq8*, *Rphq9* and *Rphq10*, only in isolate 24. Three QTLs, *Rphq2*, *Rphq3* and *Rphq4* were effective for both isolates. The effects of *Rphq3* and *Rphq4* on isolate 24 were smaller than on isolate 1.2.1. while *Rphq2* had a similar effect for both isolates.

The relative latent period of isolate 1.2.1. in 'Vada' at both plant stages is much longer than that of isolate 24. Indeed, the isolate-non-specific QTLs, *Rphq2* (except for RLP50A), *Rphq3* and *Rphq4*, contributed smaller effects to isolate 24. However, more QTLs were detected for resistance to isolate 24 than to isolate 1.2.1. It seems that

Fig. 2 QTLs for partial resistance to barley leaf rust shown on a skeletal map based on 103 RILs from the cross L94×Vada. Chromosomes are oriented with the short arms at the top. Kosambi's mapping function was used. Names of QTLs are designated on the left side of each QTL. *Boxes inside* the chromosome bars are the QTLs for partial resistance to leaf rust isolate 1.2.1. *Boxes on the right side* of the chromosome bars are QTLs for partial resistance to isolate 24. *Length of bars* corresponds to two LOD support intervals (from peak) based on the results of the "MQM" method ▶


cM
1 (7H)
2 (2H)
3 (3H)
4 (4H)
5 (1H)
6 (6H)
7 (5H)
0
10
20
30
40
50
60
70
80
90
100
110
120
130
140
150
160
170
180
190
E33M55-508
E35M48-228
E35M54-93
E41M40-112
E42M51-267
E33M61-120
E37M32-99
E35M61-432
E42M32-195
E38M54-618
E35M55-458
E40M40-105
E39M61-372
E35M54-224
E35M55-450
E35M48-450
E40M38-363
E40M32-123
E33M61-173
E33M58-375
E40M32-168
E45M55-345
E35M54-180
Rphq1
Rphq8
Rphq9
E35M54-222
E37M38-373
E33M55-410
E42M48-308
E37M32-288
E42M48-270
E41M32-83
E41M32-156
E33M61-135
E42M51-85
E41M40-118
E33M61-281
E42M32-324
E33M54-187
E33M55-109
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E41M32-492
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E33M61-227
E33M54-307
E42M51-101
E38M54-134
E40M32-590
E37M38-199
E42M40-644
E37M33-501
E37M32-325
E33M55-297
E38M54-294
E42M48-376
Rphq6
Rphq2
E45M55-572
E41M32-149
E35M48-250
E37M33-583
E33M54-244
E45M55-167
E41M40-358
E38M54-315
E35M54-188
E37M32-471
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E40M32-397
E35M48-410
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Rphq10
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E42M40-94
E42M32-682
E37M33-191
E35M48-219
E33M61-768
E33M54-100
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E37M38-178
E33M58-219
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E42M51-168
Rphq5
E42M51-113
Hor2
E42M51-497
E32M61-111
E45M55-317
E41M40-469
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E39M61-140
E33M54-263
E42M32-122
E37M38-230
E42M40-236
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E41M40-304
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E39M61-197
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E41M32-158
mB
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E42M51-389
E32M61-190
E39M61-354
E41M40-607
E32M61-421
E39M61-505
E38M54-119
E37M32-235
E35M61-269
E35M55-216
E37M33-574
E33M61-681
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E33M61-595
E33M55-267
E42M32-464
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E35M48-400
Rphq4
Rphq7
RLP50S
RLP50A

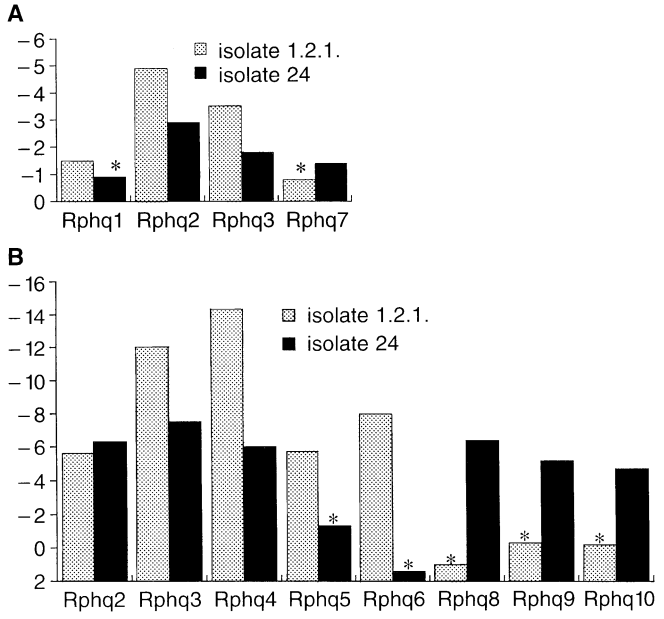

Fig. 3 Histogram of additive effects of each QTL for two leaf rust isolates in the seedling stage (**A**) and in the adult plant stage (**B**). *: indicates that the effect of the QTL is not significant

more genes (QTLs) in Vada, but each with smaller effects, were involved in resistance to isolate 24.

The map position on chromosome 1 of *Rphq8*, an isolate-specific QTL with a moderate effect on isolate 24 at the adult plant stage, coincided with a minor QTL for days to heading (*Dhl*) as shown in our previous study (Qi et al. 1998a). So far, it is still unknown whether this reflects two closely linked QTLs or a pleiotropic effect of one QTL.

The present study demonstrates that most QTLs for partial resistance are isolate-specific and show plant stage-dependent expression. Two major-effect QTLs, *Rphq2* and *Rphq3*, are expressed in both development stages and may be isolate-non-specific. Only *Rphq4* shows development stage-specific expression but is isolate-non-specific. However, the question whether these QTLs are isolate-specific as well can only be answered when a large number of isolates are tested.

Using sub-samples of a population of F_3 lines, Melchinger et al. (1998) mapped QTLs in maize using two testers showing a relatively low power of QTL detection and a large bias in estimates of QTL effects. This raises the question about the 'confidence' one can have in QTLs mapped in a single experiment. Despite the disappointing results reported by Melchinger et al. (1998), we have reasons to have confidence in our results. Firstly, theoretically, test-cross progenies have a lower power of QTL detection than RILs for QTLs with additive effects (Gallais and Rives 1993; Groh et al. 1998). Secondly, in our previous (Qi et al. 1998a) and present studies, several independent disease evaluations with replications were conducted by different persons and in different years. However, the QTLs were consistently identified with an identical rank order of the estimated effects. This indicates that these QTLs for partial resistance to barley leaf rust were relatively insensitive to environmental conditions. Because of the relative high heritabilities (h^2=0.6–0.8) of the partial resistance, any bias in mapping QTLs with the recombinant inbred population of 103 RILs used in our study could be limited (Lande and Thompson 1990).

Minor-gene-for-minor gene interaction

Isolate-specific QTLs for quantitative resistance to *Phytophthora infestans* were also identified in potato (Leonards-Schippers et al. 1994). Six of the eleven detected QTLs showed specificity to two *P. infestans* races. In mapping QTLs for resistance to bacterial wilt (*Pseudomonas solanacearum*) on the tomato genome, one of two major resistance loci was highly race-specific (Danesh and Young 1994). In addition, in the *Capsicum annuum-Potyvirus* host-pathogen system, isolate-specific effects of QTLs for resistance were clearly demonstrated (Caranta et al. 1997). In the present research, we studied the barley-barley leaf rust pathosystem and detected clear isolate-specific effects of QTLs for partial resistance. More than 20 years ago, Parlevliet (1976a) reported small but significant cultivar isolate interactions in partially resistant barley lines. This led him to propose the 'minor-gene-for-minor-gene' hypothesis to explain quantitative (horizontal) resistance (Parlevliet and Zadoks 1977). Indeed, the examples mentioned above together with the present data indicate that minor-gene-for-minor-gene interactions do occur in plant-pathogen systems.

It is still questionable whether all resistance genes (major or minor) in the host population interact in a gene-for-gene manner with genes for virulence or avirulence in the pathogen population. Our study showed that the three major-effect QTLs were effective in both rust isolates and did not show clear isolate-specific effects. Similarly, 5 of 11 QTLs in potato showed no specificity to two *P. infestans* races (Leonards-Schippers et al. 1994). In pepper, one major-effect QTL was effective in all three potyvirus isolates tested (Caranta et al. 1997). However, it is easy to hypothesize, but hard to prove, that all resistance genes are race- or isolate-specific and operate in a gene-for-gene manner. In our on-going studies of the barley-*P. hordei* system, we are developing a series of near-isogenic lines (NILs) for each of the QTLs by using marker-assisted selection. Each set of NIL-QTLs with an identical genetic background will allow numerous QTL×rust isolate combinations to be tested. In addition, these NILs will serve as starting material for the map-based cloning of QTLs for partial resistance. These on-going researches will allow us to determine whether these QTLs which are effective against both isolates are isolate-specific or isolate-non-specific.

Durability of partial resistance

The gene-for-gene theory was proposed in studies on the interaction between flax cultivars and flax rust (Flor 1956, 1971). There are numerous examples that testify to the fact that the hypersensitive resistance operating on a gene-for-gene basis is not durable [e.g. *Rph* genes in barley (Jin et al. 1995)]. This does not imply, however, that resistance based on the gene-for-gene principle can never be durable. There are at least three possible ways to explain durability in a polygenic resistance based on a minor-gene-for-minor-gene interaction.

Firstly, as Parlevliet and Zadoks (1977) argued, genes operating on a minor-gene-for-minor-gene basis would result in a higher durability of resistance than genes with additive effects that are effective to all genotypes of the pathogen. In the latter case, a mutant pathogen genotype with an increased aggressiveness would have a selection advantage on all host plants with any QTL for partial resistance and, as consequence, very soon replace the less-aggressive pathogen strain in the population. Hence, such a resistance would not be very durable. In the case where the interaction acts according to the minor-gene-for-minor-gene principle, a mutation for increased aggressiveness in the pathogen would only increase the fitness of the pathogen on those host genotypes that have the minor gene for quantitative resistance that corresponds with the mutated aggressiveness gene. In genetically diverse host populations, this would lead to a rather mild increase of the pathogen with the mutant minor gene for increased aggresiveness and, hence, the resistance would be quite durable.

Secondly, it is generally accepted that the break down of hypersensitive resistance is a consequence of the deletion (Van den Ackerveken et al. 1992) or mutation (Joosten et al. 1994, 1997) of the avirulence gene in the pathogen. This is a rather unspecific event, e.g. any mutation in the avirulence gene leading to a frame shift should result in virulence on a host genotype with the corresponding resistance gene. In partial resistance, we are concerned with a completely different plant defense system. The QTLs for partial resistance do not tend to coincide in the linkage map with the *Rph* genes for hypersensitivity (Qi et al. 1998a) and the resistance mechanism is entirely different, supporting the idea that the *Rph* genes and QTLs for partial resistance represent distinct classes of genes or gene families. The *Rph* gene resistance acts post-haustorially with hypersensitivity, whereas partial resistance is based on a pre-haustorial mechanism associated with the formation of papillae (Niks 1986). Therefore, it is very probable that the gene-for-gene specificity in partial resistance is of a different nature. Breaking down this resistance may require very specific, and therefore rare, mutations in the pathogen. This scenario would result in a higher durability of resistance.

Thirdly, a polygenic resistance *per se* has a higher probability to be durably effective. In the event that the minor genes each have a different function in defense, the pathogen can only negate this multiple barrier by step-wise genetic adaptation.

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