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ORIGINAL PAPER

The complex quantitative barley–*Rhynchosporium secalis* interaction: newly identified QTL may represent already known resistance genes

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Abstract Two barley populations, i.e. 135 doubled haploid (DH) lines of the cross 'Igri' (*rrs*1) × 'Triton' (*Rrs*1) (I × T) and 76 DH lines of the cross 'Post' × 'Vixen' (both *rrs*1) (P × V), were analysed to identify QTL for *Rhynchosporium secalis* resistance independent of the *Rrs*1 locus by using the single spore *R. secalis* isolate 271 (*Rrs*1-virulent). A major QTL with its positive allele derived from cv. 'Triton' was detected in the I × T population on chromosome 2HS explaining almost 80% of the phenotypic variance. Thus, it can be considered as an R-gene corresponding to the already described $Rrs15_{CI8288}$ on chromosome 2HS. In addition, two minor QTL were identified, one in the centromeric region of 6H in a highly polymorphic region with already several mapped R-genes and a second one at the end of the short arm of chromosome 7H which may be an allele of *Rrs*2 because of its chromosomal position. Regarding the DH population P × V different minor QTL were identified on chromosomes 6H and 7H. The first one is corresponding to the genomic region of the *Rrs*13 gene whereas the QTL on chromosome 7H maps in a genomic region where several R-genes against different pathogens have been localized. A comparison of both QTL analyses reveals no *R. secalis* isolate 271-specific resistance locus but leads to the hypothesis that two of the identified QTL may be alleles of the R-genes $Rrs15_{CI8288}$ and *Rrs*2.

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Introduction

Scald, caused by the fungus *Rhynchosporium secalis* (Oudem.) J.J. Davis, is a serious foliar disease of barley worldwide, especially in cool and temperate climates (Beer 1991). Its agronomic importance varies greatly due to variable field conditions, but yield losses of up to 40% were reported in barley (Xi et al. 2000). Furthermore, *R. secalis* infection can affect grain quality, thus discounting quality uses, e.g. for malting (Khan and Crosbie 1988). *Rhynchosporium secalis* is known to be a highly variable pathogen (Shipton et al. 1974; Ali et al. 1976; Burdon et al. 1994; Salamatini and Tronsmo 1997) with diverse virulences in local populations causing various host reactions, such as the expression of major-gene-mediated resistance, partial or quantitative resistance, and disease escape (Zhan et al. 2008). *Rhynchosporium secalis* populations can change quickly so that new plant resistances as well as fungicides, particularly triazoles (Jefferies et al. 2000), become ineffective

after several seasons of widespread use (Shipton et al. 1974; Cromey and Mulholland 1987; Taggart et al. 1999; Jefferies et al. 2000; Newton et al. 2001; Oxley et al. 2002). Therefore, and due to the fact that only moderate *R. secalis* resistance is present in released barley cultivars, there is a high demand to introduce and to combine new *R. secalis* resistances to breed barley cultivars with a more stable and durable *R. secalis* resistance.

In summary, 17 resistance gene loci have been described (Bjørnstad et al. 2002; Schweizer et al. 1995, 2004; Pickering et al. 2006), where of nine are mapped and designated by an *Rrs* prefix. Four of these R-genes derive from *H. vulgare* ssp. *spontaneum* (Abbott et al. 1995; Garvin et al. 1997; Genger et al. 2003; Yun et al. 2005) and *H. bulbosum* (Pickering et al. 2006), respectively; for an overview cf. Friedt and Ordon (2007). Besides this, numerous QTL analyses were carried out. Zhan et al. (2008) present an overview of their expected map positions by grouping them into BINs of each barley chromosome (http://barleygenomics.wsu.edu/), showing that all barley chromosomes except 5H are involved in resistance against *R. secalis*. Unfortunately, due to different mapping populations used and applied molecular markers a comparison of mapped *R. secalis*-resistances (QTL and R-genes) still remains difficult. Therefore, using defined isolates in future studies is desirable and highly recommended since it would allow unbiased comparisons between different results on *R. secalis*-resistance (Cheong et al. 2006). So, some QTL independently identified by different authors may be identical or represent different alleles of the same resistance locus or loci (Dingerdissen et al. 1996, Bjørnstad et al. 2004). The variable pathogenicity of *R. secalis* and the isolate-specific resistance reaction of barley cultivars hamper breeding progress for *Rhynchosporium* resistance. Therefore, it is of agronomic and economic interest to develop new scald resistant varieties to increase yield stability and quality of barley crop. A prerequisite hereof will be to reliably detect genomic regions responsible for pronounced *R. secalis* resistance and to develop markers facilitating efficient marker based selection.

In a previous study, Krämer (2003) observed a quantitative distribution for scald resistance in the barley cross 'Igri' × 'Triton' (60 DH lines) after artificial inoculation with a mixture of 28 pathotypes containing isolates overcoming all major resistance genes known at that time, including *Rrs*1 of 'Triton'. Thus, the aim of the current study was (1) to investigate the genetics of the quantitative *R. secalis* resistance in an extended 'Igri' (*rrs*1) × 'Triton' (*Rrs*1) population (135 DH lines) by using the virulent single spore *R. secalis* isolate 271; (2) to get information whether additional isolate 271-specific resistance QTL are present by including the moderately resistant DH population 'Post' × 'Vixen' (both without known *Rrs*-gene), and (3) to compare the results with already mapped resistance genes and QTL via the barley BIN map (http://barleygenomics.wsu.edu/) and the barley consensus map of Varshney et al. (2007).

Materials and methods

Plant material

Two barley DH populations were used in the present study: One consists of 135 anther-derived DH lines from a cross (F_1) between the susceptible two-rowed winter barley cv. 'Igri' (*rrs*1) and the resistant six-rowed winter barley cv. 'Triton' (*Rrs*1) (I × T). Resistant cv. 'Triton' is known to carry the *Rrs*1 resistance gene on chromosome 3H (Graner and Tekauz 1996). But, after infestation with virulent *R. secalis*-isolates cv. 'Triton' remained resistant indicating the presence of (an) additional resistance source(s) different from *Rrs*1 (Krämer 2003, M. Schönfeld, personal communication). The second progeny comprises 76 F_1 anther-derived DH lines of the cross 'Post' × 'Vixen' (P × V), originally developed to study BYDV-tolerance (Scheurer et al. 2001). This population was used in this study to assess isolate-specific resistance reaction because cv. 'Post' turned out to be moderately resistant against *R. secalis* isolate 271.

Rhynchosporium secalis resistance assay

Resistance assessment of each DH line, four replicates in one pot, was carried out at the seedling stage in greenhouse chambers according to Schweizer et al. (2004) with the single-spore isolate 271 of *R. secalis* in a suspension adjusted to 2–300,000 spores ml^{-1}. Each plant was assessed visually three times 14–21 days after inoculation (dai) for scald symptoms on the lamina of the second leaf according to the scale described by Jackson and Webster (1976). The final score of scald severity for each DH line was calculated by averaging the scores of four individual plants in each experiment. A total of four independent greenhouse experiments with DH population I × T and two additional idependent experiments with P × V were performed in the years 2004–2006. For inoculations, the *R. secalis* single-spore isolate 271 (from E. Sachs, collected in Strassmoos, LfL Bavaria, Germany) was selected due to its known virulence against *Rrs*1 of cv. 'Triton' (M. Schönfeld, personal communication).

To obtain clear evidence on the performance of the resistance tests Spearman's rank correlation coefficients between all experiments within each DH population were computed. Further on, Kolmogorov–Smirnov Test (P_{K-S}) was carried out to test the frequency distribution of *Rhynchosporium* resistance scores for conformity with a Gaussian distribution of each trial by using SPSS 14.0.

Assessment of *Rhynchosporium secalis* resistance of I × T in the field

With the I × T DH population an additional resistance test was carried out in a field trial with an natural unspecified mixture of pathotypes to confirm major resistance QTL detected in green house tests at the Bavarian State Research Centre for Agriculture, Freising in 2006 in blocks of six double rows without replications but with the susceptible cv. 'Nelly' as check two times in each block (double-rows 2 and 5). Visual assessment of scald symptoms was conducted per row according to the EPPO guidelines ranking from one (resistant) to nine (susceptible).

DNA marker analysis

Leaf samples of approx. 100 mg were taken from the parents and each single DH line of both populations. Genomic DNA of barley leaves was extracted according to Doyle and Doyle (1990). In order to identify molecular markers, 'Igri' and 'Triton' were screened for polymorphisms by *Eco*RI + 3/*Mse*I + 3 AFLP primer combinations, RAPD primers, and SSRs. Molecular analyses using these techniques were performed according to Ordon et al. (1995) for RAPDs, Vos et al. (1995) and Schiemann et al. (1999) for AFLPs, and for SSRs according to Ramsay et al. (2000), Li et al. (2003); cf. http://wheat.pw.usda.gov/cgi-bin/graingenes/browse.cgi. AFLP and SSR detection was carried out on a LiCor 4200S DNA sequencer (MWG Biotech, Ebersberg, Germany). RAPDs and AFLPs were named according to the respective primer or primer combination followed by the fragment size, or numbered from the smallest to the largest fragment (AFLPs). Additionally, the STS marker cMWG680 (Graner and Tekauz 1996), the *Vrs1*-locus (row-type gene of barley spike) as well as the SNP marker P1D23R (M. Röder and A. Hanemann, unpublished, on request), the STS marker GemS13 (G. Schweizer, in preparation, on request), and the SSR markers GBM1121 and GBM1281 (Stein et al. 2007) were applied on the I × T population. These additional markers were used because of their chromosomal position or since they have been reported by the cited authors to be closely related to *R. secalis* resistance. The STS marker cMWG680 maps in the centromeric region of chromosome 3H and is a selection marker for *Rrs*1 (Graner and Tekauz 1996). The markers GemS13 and P1D23R are closely linked to the resistance genes $Rrs15_{CI8288}$ and *Rrs*2 on the short arm of chromosomes 2H and 7H, respectively (Schweizer et al. in preparation, Röder et al. in preparation) whereas GBM1121 and GBM1281 were additionally used to increase the marker density on chromosome 2H. The morphological marker (*Vrs1*-locus) is known to be located on chromosome 2H too, and has been used in this study as an anchor marker.

Linkage analysis

Genetic mapping for the DH population P × V was carried out according to the procedure described by Schäfer-Pregl et al. (1999) with the software package MAPMAKER (Lander et al. 1987) using Haldane's (1919) mapping function (Scheurer et al. 2001). In contrast, a genetic linkage map of the DH population I × T was established with the software JoinMap 3.0 (Van Ooijen and Voorrips 2001) using Kosambi's (1944) mapping function. All markers which were uniquely placed by this procedure were chosen for map construction. SSRs and morphological markers with known chromosomal locations were used as anchor markers to assign linkage groups to chromosomes. All mapped markers were tested for the expected 1:1 segregation ratio using a χ^2 goodness-of-fit test.

QTL analysis

QTL analysis of *Rhynchosporium* resistance was performed with the software package PLABQTL 1.2bic (Utz and Melchinger 1996, 2006, https://www.uni-hohenheim.de/plantbreeding/software/) employing the composite interval mapping (CIM) procedure. The LOD threshold value used to determine the genome-wide significance ($P < 0.05$) of a putative QTL was obtained empirically by permutation tests (1,000 replications) using PlabQTL 1.2bic. QTL analyses were carried out across all environments with cofactors obtained by the PLABQTL procedure. QTL positions were determined at the maximum of the respective LOD plot curve. The explained phenotypic variance of each individual QTL and the sum of all detected QTL was calculated in a final simultaneous fit (FSF). Furthermore, Bayesian information criterion (BIC), a more restrictive selection strategy compared to the F-to-enter and F-to-delete procedure of the FSF strategy, was used to choose the marker cofactors. Visualisation of QTL was carried out by using MapChart 2.2 (Voorrips 2002).

Results

Phenotypic variation of resistance against *Rynchosporium secalis* isolate 271

After resistance scoring of both DH populations a high degree of variation was observed ranging from pronounced resistance to complete susceptibility revealing transgressive lines. Focussing on the I × T population a two-peak frequency distribution was obtained for the data of the field trial ($P_{K–S} = 0.002$) and four greenhouse trials ($P_{K–S\_trial1} = 0.016$, $P_{K–S\_trial2} = 0.227$, $P_{K–S\_trial3} = 0.092$, $P_{K–S\_trial4} = 0.009$, $P_{K–S\_trials\ mean} = 0.093$) assuming that $P > 0.1$ is

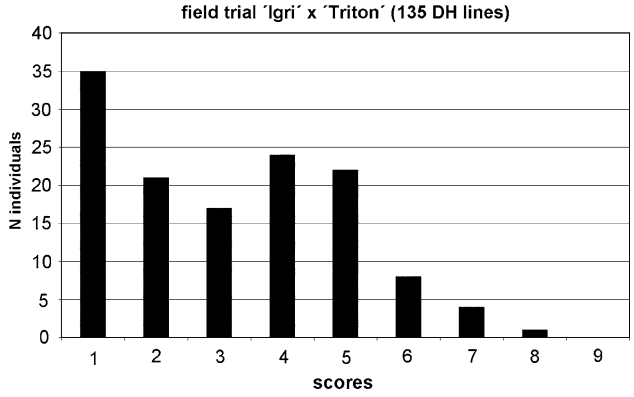

Fig. 1 *R. secalis* reaction of the 135 DH lines of the I × T population recorded in a field test at Freising in 2006

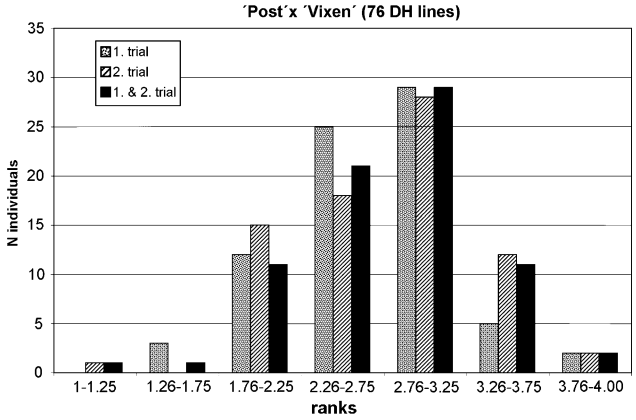

Fig. 3 *R. secalis* resistance of 76 DH lines of the cross P × V recorded in two greenhouse tests in Freising in 2005 and 2006

equivalent to a Gaussian distribution (Figs. 1, 2), whereas the P × V lines fit a Gaussian distribution ($P_{K–S\_trial1} = 0.294$, $P_{K–S\_trial2} = 0.115$, $P_{K–S\_trials\ mean} = 0.667$) (Fig. 3). The estimation of Spearman's rank correlation coefficient reveals moderate to high correlations between the scores of all four greenhouse tests of the I × T population and the two trials of the P × V cross (Table 1). Therefore, all data of greenhouse trials were used for the QTL analyses.

Genetic mapping

In total 228 (I × T population) and 117 (P × V population) SSR, AFLP, RAPD, STS markers, and one morphological marker were used to generate the linkage maps. The resulting map of I × T comprises the expected seven linkage groups at an average spacing between markers of 4.72 cM. The map covers a total of 1075 cM and consists of 82 SSR,

Table 1 Spearman's rank correlation coefficients of the *Rhynchosporium secalis* resistance tests of the DH lines of the crosses 'Igri' × 'Triton' and 'Post' × 'Vixen' (2004–2006)

	Exp. 1	Exp. 2	Exp. 3	Exp. 4
'Igri' × 'Triton'				
Exp. 1	1.00	0.69**	0.80**	0.71**
Exp. 2	–	1.00	0.66**	0.65**
Exp. 3	–	–	1.00	0.63**
Exp. 4	–	–	–	1.00
'Post' × 'Vixen'				
Exp. 1	1.00	0.75**	–	–
Exp. 2		1.00	–	–

** Significant on the 0.01 level (2-tailed)

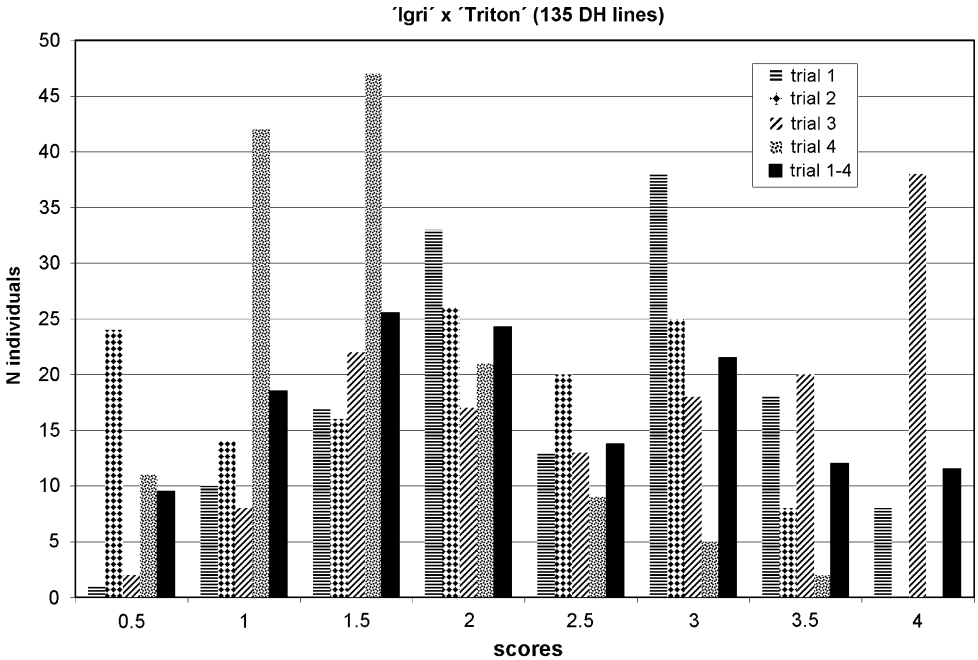

Fig. 2 *R. secalis* reaction of the 135 DH lines of the I × T population recorded in four greenhouse tests in Freising in 2004–2006

106 AFLP, 39 RAPD and STS markers as well as one morphological marker (*Vrs1*-locus). Each linkage group could be unequivocally assigned to an individual barley chromosome. Larger gaps of >25 cM were observed on the long arms of chromosomes 3H, 4H, 5H, and 7H. Deviations from the expected segregation ratio of 1:1 in favour of 'Igri' alleles were observed for markers on chromosomes 3H, 4H, and 5H. The genetic linkage map of P × V has been described already by Scheurer et al. (2001). In both maps the order of SSRs corresponds to published results of Ramsay et al. (2000), Li et al. (2003), and Varshney et al. (2007).

QTL analysis for the population I × T

Based on recordings of four independent greenhouse trials, three highly significant QTL for resistance to *R. secalis* isolate 271 were detected on chromosomes 2HS, 6H (centromeric region), and 7HS at a LOD score of >3.63 ($P < 0.05$, permutation test) after FSF (Fig. 4, Table 2). All three QTL derive from the resistance donor 'Triton' together explain 79.7% of the phenotypic variance. The major QTL on chromosome 2HS alone, flanked by the SSR marker GBM1281 and the STS marker GemS13, explains 77.7% (FSF) of the phenotypic variance. In addition, the two minor QTL on chromosomes 6H and 7HS each explain a partial phenotypic variance of more than 20%. Even after application of BIC all three QTL remained significant (Table 2).In order to get information whether these QTL are effective against an unspecified mixture of pathotypes an additional QTL analysis based on the data of the field trial were carried out and two QTL at a LOD threshold >6.53 corresponding to the major resistance loci of 'Triton' were detected. One on chromosome 2HS in the same marker interval as the one detected in the greenhouse trials, and the second in the centromeric region of 3H where *Rrs*1 is located. (Table 2).

QTL analysis for the population P × V

QTL analysis was run based on two greenhouse tests, i.e. each DH line was tested two times in four replicates. After simultaneous fit, two significant QTL for resistance to *Rhynchosporium* isolate 271 were detected with a LOD score >3.34 ($P < 0.05$ after permutation test) (Fig. 5, Table 2): One of them on chromosome 6H with the positive allele derived from the moderately resistant cv. 'Post', and a smaller one on 7HS with the positive allele derived from 'Vixen'. Together, both QTL explain 35.1% of the phenotypic variance. The QTL of chromosome 6H has the stronger effect explaining a partial phenotypic variance of 37.1%. Applying BIC, only the QTL on chromosome 6H was still significant explaining 30.3% of the phenotypic variance.

Discussion

Gene loci involved in Rhynchosporium resistance on chromosome 2H

The major QTL detected in the I × T population is located distally on the short arm of 2H in a genomic region where

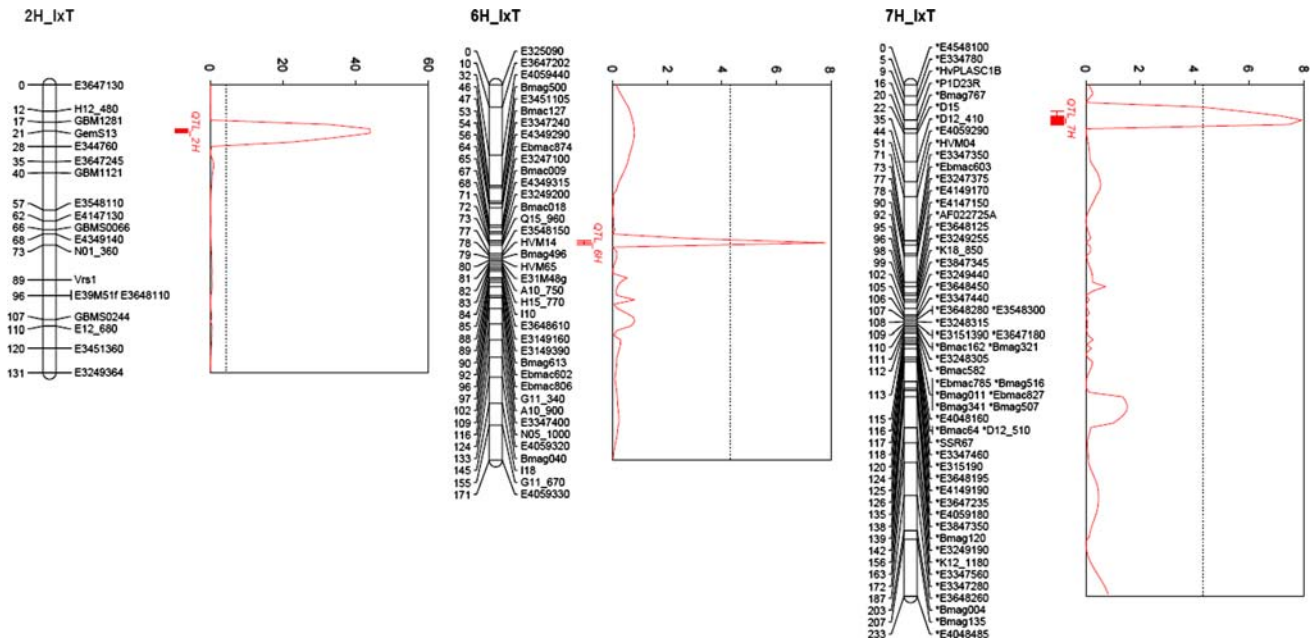

Fig. 4 Chromosomal location of QTL for *Rhynchosporium secalis* resistance in the barley DH population I × T

Table 2 LOD scores, percentages phenotypic variance explained (R^2) and additive effects of the detected QTL in the DH populations I × T and P × V

	LOD	R^2 (%)	Additive effect	FSF Part. R^2 (%)	BIC Part. R^2 (%)
'Igri' × 'Triton' (I × T): 4 greenhouse experiments					
$QTL_{Triton}Rrs2H_{271}$	49.57	81.8	−0.643	77.7	77.7
$QTL_{Triton}Rrs6H_{271}$	3.19*	10.4	−0.157	22.8	22.8
$QTL_{Triton}Rrs7H_{271}$	8.31	24.8	−0.169	20.9	20.9
Sum				79.7	79.7
'Igri' × 'Triton' (I × T): 1 field trial					
$QTL_{Triton}Rrs2H_{natural}$	13.44	38.4	−0.964	25.8	25.8
$QTL_{Triton}Rrs3H_{natural}$	6.53	20.9	−0.704	18.0	18.0
Sum				36.8	36.8
'Post' × 'Vixen' (P x V): 2 greenhouse experiments					
$QTL_{Post}Rrs6H_{271}$	3.62	19.7	0.226	37.1	32.1
$QTL_{Vixen}Rrs7H_{271}$	4.01	21.6	−0.189	9.6	ns
Sum				35.1	30.3

* LOD score $P < 0.1$

ns not significant at the 0.01 level

FSF final simultaneous fit

BIC Baysian information criterion

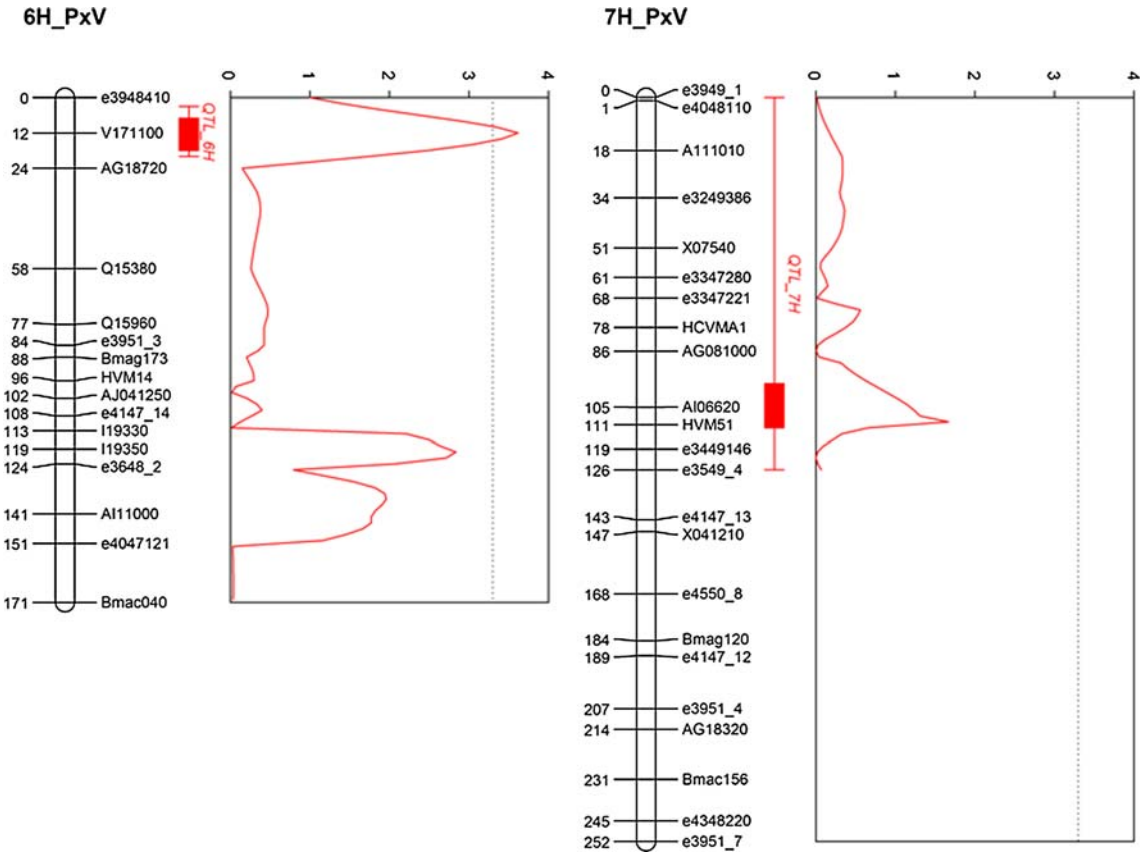

Fig. 5 Chromosomal location of QTL for *Rhynchosporium secalis* resistance in the barley DH population P × V

the scald resistance gene $Rrs15_{CI8288}$ has been detected by Schweizer et al. (2004). The closely linked STS marker GemS13, identified by Schweizer et al. (in preparation, on request), could also be mapped in the I × T population and is one of the flanking markers of the $QTL_{Triton}Rrs2H_{271}$. Therefore, it is very likely that the detected major $QTL_{Triton}Rrs2H_{271}$ is identical with or allelic to the R-gene $Rrs15_{CI8288}$. Besides this gene, a few loci for scald resistance have already been detected on the short arm of chromosome 2H (Backes et al. 1995, Grønnerød et al. 2002,

Sayed et al. 2004, von Korff et al. 2005, Zhan et al. 2008). In contrast to the QTL detected in this study, a weak QTL (LOD <3.0, not confirmed by CIM) of the Ethiopian line 'Abyssinian' (CI668) described by Grønnerød et al. (2002) is located in the centromeric region of 2H. Further on, a QTL for scald resistance on 2HS detected by von Korff et al. (2005) with the positive allele derived from the wild barley *H. vulgare* ssp. *spontaneum* ISR42-8 is not located in the genomic region of $Rrs15_{CI8288}$, too. The remaining QTL for scald resistance on chromosome 2H detected by Sayed et al. (2004) in one environment maps on the long arm with a LOD score of 2.5. By comparing the genomic region of $Rrs15_{CI8288}$ with the maps of Rostoks et al. (2005), Marcel et al. (2007), Ramsay et al. (2000), and Varshney et al. (2007), it turned out that the QTL detected by von Korff et al. (2005) must be different from those of Grønnerød et al. (2002) but still seems not to be located in the genomic region of $Rrs15_{CI8288}$.

Resistances against different other barley pathogens were mapped on chromosome 2H: Pickering et al. (1998) mapped the resistance genes *Rph.Hb* and *Mlhbl.a* in BIN1 of chromosome 2HS whereas $Rrs15_{CI8288}$ was located in BIN3 (http://barleygenomics.wsu.edu/, Williams 2003). Several *Fusarium* head blight (FHB) resistance and mycotoxin (DON) accumulation QTL were detected on 2H (Dahleen et al. 2003, Mesfin et al. 2003). All other loci are in the centromeric region of 2H, except QFhs.umn-2H.1, which is mapped on the short arm of chromosome 2H and may be located in the genomic region of $Rrs15_{CI8288}$ as shown by comparison of marker positions via common markers on the barley consensus map (Mesfin et al. 2003, Varshney et al. 2007).

Gene loci for scald resistance on chromosome 6H

Some scald resistance QTL have been described on chromosome 6HS (Zhan et al. 2008). Most of the resistance QTL (Jensen et al. 2002, Cheong et al. 2006, Shtaya et al. 2006) were located on the short arm in a genomic region where the *Rrs*13 gene was mapped, too (Abbott et al. 1992). An additional QTL (*Rrsq*4) has been described in a single experiment by Shtaya et al. (2006). Comparing these genomic regions with $QTL_{Post}Rrs6H_{271}$ detected in P × V after comparison with the barley consensus map of Varshney et al. (2007) it can be hypothesized that these QTL actually represent alleles of *Rrs*13 contributing to scald resistance with minor effects (Jensen et al. 2002, Cheong et al. 2006).

In the centromeric region of chromosome 6H, in which the $QTL_{Triton}Rrs6H_{271}$ is located, no scald resistance QTL has been described so far. Marcel et al. (2007) mapped a *Puccinia hordei* resistance QTL (*Rph*q3) of the 'L94' × 'Vada' cross in the centromeric region of 6H (BIN6), a genomic region where the *Pyrenophora teres* resistance gene Rpt_{CI9819} (Manninen 2000), the BaMMV/BaYMV resistance gene *rym*15 (Le Gouis et al. 2004), and the BYDV resistance gene *Ryd*3 (Niks et al. 2004) have been mapped. Furthermore, Marcel et al. (2007) showed co-localisation of the *Rph*q3 QTL with the defence gene homologues (DGHs) WBE103, GBS0164 (both mRNA sequences for a glycoprotein with a superoxide dismutase function), and WBE201 (similar to Xa21 homologue with a serine/threonine-protein kinase function). A comparison of the respective centromeric region of 6H with the transcript map of Stein et al. (2007) and with the map of Rostoks et al. (2005) indicates the relevance of this region for stress reaction, but not indicating potential candidate gene(s) for *R. secalis* resistance.

Scald resistance loci on chromosome 7H

Two genes or genomic regions on chromosome 7H have been described to be associated with scald resistance [*Rrs*2 and *Rrs*12 (*Hordeum vulgare*), and *Rrs*15 (*H. spontaneum*); Zhan et al. (2008)]. The minor resistance $QTL_{Triton}Rrs7H_{271}$ is located at the end of the short arm of 7H (BIN1), while $QTL_{Vixen}Rrs7H_{271}$ is located on 7HL (BIN8-10). $QTL_{Triton}Rrs7H_{271}$ is located in the same chromosomal region as *Rrs*2 (Schweizer et al. 1995), distally on 7HS, since it is closely linked with the *Rrs*2 SNP marker P1D23R developed by Röder and Schweizer (in preparation). Cheong et al. (2006) have also reported co-localisation of the scald resistance QTL derived from Harrington with *Rrs*2. Regarding further mapped QTL in the genomic region of *Rrs*2 (Zhan et al. 2008), it can be hypothesized due to common molecular markers that there is only one—probably multiallelic—locus, *Rrs*2/*Rrs*12. Besides for scald resistance, additional R-genes are located in the same genomic region (BIN1) of 7HS, such as *Rdg*2a for barley leaf stripe (*Drechslera graminea* syn. *Pyrenophora teres* subsp. *graminea*) (Tacconi et al. 2001), *Rpg*1 (a receptor-like kinase; Brueggeman et al. 2002) for barley stem rust (*Puccinia graminis f. sp. tritici*) (Kilian et al. 1997, http://ace.untamo.net/), and *Run*1 for loose smut (*Ustilago nuda*) (http://ace.untamo.net/). These facts clearly underline the importance of the 7HS genomic region for resistance of barley against different pathogens. Madsen et al. (2003) mapped a RGA in the genomic region of chromosome 7H BIN1, but concluded that due to linked markers it is not a candidate for the *Rrs*2 locus but suggested MWG851 (Michalek et al. 1999), which belongs to a RGA class 2 (Madsen et al. 2003), or ssCH4 (Seah et al. 1998) as possible candidate genes.

In spite of the small mapping population and some gaps in the map, the minor $QTL_{Vixen}Rrs7H_{271}$ mapped in the same region as *Rph*q8 (Marcel et al. 2007) which is linked

with a DGH WBE101, a similar WIR1 protein known to be induced upon inoculation with *Blumeria graminis* f. sp. *hordei* (Jansen et al. 2005, Zierold et al. 2005) and with the non-host pathogen *P. triticina* (Neu et al. 2003). Besides this, the gene *Rpt*4 encoding resistance to *Pyrenophora teres* f. *maculata* was mapped in the respective genomic region (Williams et al. 1999, Williams 2003).

Synopsis

In summary, the newly detected QTL described in this paper turn out to be located in concordant genomic regions with resistance genes and QTL mapped before. In the I × T DH population three QTL derived from the resistant cv. 'Triton' were discovered contributing resistance against the virulent *R. secalis* isolate 271 whereof the QTL on chromosomes 2HS and 7HS seem to be alleles of already described *R. secalis* resistance loci $Rrs15_{CI8288}$ and *Rrs*2/*Rrs*12 due to their genomic map position flanked by the closely linked molecular markers GemS13 (2HS) and P1D23R (7HS). Therefore, the identified major $QTL_{Triton}Rrs2H_{271}$ in the present study seems to confirm the resistance locus described by Schweizer et al. (2004) in the accession CI8288 on chromosome 2HS. However, this has to be proven by tests for allelism. Further on, it can be hypothesized that an allele of the *Rrs2* resistance locus of chromosome 7HS has been identified with only a minor contribution to *Rhynchosporium* resistance, which has to be validated by tests for allelism. The genomic region of chromosome 6H where we have localized a minor QTL in cv. 'Triton', turned out to contain a number of QTL for resistance against different pathogens and resistance-associated transcripts.

A single field trial with a natural unspecified mixture of *R. secalis* pathotypes revealed that cv. 'Triton' is carrying two major resistance loci, the *Rrs*1 locus on chromosome 3H described by Graner and Tekauz (1996) and Krämer (2003) as well as the resistance locus on 2HS confirmed in the greenhouse against the virulent *R. secalis* isolate 271.

Regarding the small P × V DH population a major QTL for *R. secalis* resistance on chromosome 6HS derives from cv. 'Post'. By comparing the barley consensus map (Varshney et al. 2007) and the barley BIN map (http://barleygenomics.wsu.edu/) with results of QTL analysis it turned out that the significant QTL on 6HS is closely linked to the genomic region of the *Rrs*13 resistance locus and may even be allelic to *Rrs*13.

Therefore, the present study confirms the hypothesis of Bjørnstad et al. (2002) and Cheong et al. (2006) that OTL in the same genomic regions identified in different mapping populations describe alleles of the same resistance loci. Both QTL analyses show different genomic regions involved in resistance against the *R. secalis* isolate 271 pointing out that there are distinct resistance mechanisms involved in the pathogen defence to the same pathotype in both populations.

By comparing the identified relevant genomic regions with e.g. barley transcript maps of Rostoks et al. (2005), Marcel et al. (2007), and Stein et al. (2007) candidate genes migth be detected localized in respective genomic regions of the identified QTL. These sequences represent genes of interest to be mapped in the I × T and P × V populations, respectively, and to study their expression profiles. This would answer the question of putative functions of the detected genomic regions involved in scald resistance.

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