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Isolate-specific QTLs of resistance to leaf stripe (*Pyrenophora graminea*) in the 'Steptoe' × 'Morex' spring barley cross

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Abstract Leaf stripe caused by the fungus *Pyrenophora graminea* represents a serious threat to grain yield in organically grown barley and in conventional Nordic and Mediterranean districts, for which resistant cultivars are necessary. A medium-density, molecular marker map derived from a 'Steptoe' (partially resistant) × 'Morex' (susceptible) spring barley cross and its derived doubled-haploid mapping population inoculated with the fungus made it possible to identify QTLs of resistance to leaf stripe. In order to investigate isolate-specificity of partial resistance, the 'Steptoe' × 'Morex' segregating population was inoculated with two highly virulent *P. graminea* isolates, Dg2 and Dg5. The present study demonstrates that partial resistance to leaf stripe of cv 'Steptoe' is governed in part by shared loci and in part by isolate-specific ones. One QTL is common to the resistance for the two isolates, on the long arm of chromosome 2 (2H), two QTLs are linked on chromosome 3 (3H), and the remaining two are isolate-specific, respectively for isolate Dg2 on chromosome 2 (2H) and for isolate Dg5 on chromosome 7 (5H). The QTL in common is that with the major effect on the resistance for each isolate, explaining 18.3% and 30.9% R^2 respectively for Dg2 and Dg5. The isolate-specific QTLs mapped in the 'Steptoe' × 'Morex' barley reference map support the assumption of Parlevliet and Zadoks (1977) that partial resistance may be due to minor gene-for-minor-gene interactions. Map comparisons of the QTLs with the known qualitative resistance genes to leaf stripe, *Rdg1* (2H) and *Rdg2* (7H), as well as with other QTLs of partial resistance in barley, show that the QTL for resistance to both isolates mapped on the long arm of chromosome 2 (2H) does not coincide with the qualitative *Rdg1* gene but is linked to it at about 30 cM. One isolate-specific QTL of resistance to *P. graminea*, mapped on the short arm of chromosome 2 (2H), is coincident with a QTL for resistance to *Pyrenophora teres* previously mapped in the 'Steptoe' × 'Morex' cross.

Keywords Barley · *Pyrenophora graminea* · Partial resistance · QTL mapping · Isolate specificity

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Introduction

Leaf stripe is a widespread seed-borne barley disease caused by the fungal pathogen *Pyrenophora graminea* (Ito and Kuribayashi) [anamorph *Drechslera graminea* (Rabenh. ex. Schlech.) Shoemaker]. The fungus survives as a mycelium in the pericarp, the hull and the seed coat, but not in the embryo. During seed germination, its growing mycelium penetrates the coleorhiza and it colonizes the plant systemically starting from the root tip (Haegi et al. 1998). The disease is particularly acute in Nordic countries (spring sowing) and in the Mediterranean's winter barley districts, where soil temperatures below 12 °C during seed germination promote the infection of the rootlet. The typical symptoms, spike sterility and chlorotic stripes on leaves, which gradually extend to the full length of the leaf and finally become necrotic, lead to severe yield reductions when seed infection is high, especially in organic farming systems (Delogu et al. 1995). A variation in pathogenicity among different fungus isolates on the same genetic material has been reported, and the selective pressure of the pathogen strains may explain the existence of different resistance genes or models (Boulif and Wilcoxson 1988; Gatti et al. 1992). Analyses of the relationships between cultivars and isolates indicate that, at least in some cases, resistance to *P. graminea* belongs to a race-specific type regulated by 'major genes' (Tacconi et al. 2001).

Some useful resistance loci to this disease have been identified only in the last few years. The first extensive

study of leaf stripe resistance was conducted by Skou and Haahr (1987). They found that the most widespread source of resistance in European spring barleys was the 'Vada resistance', a single major gene with semi-dominant behaviour that is still effective in Denmark (Skou et al. 1994), and was introgressed into cultivated barleys from *Hordeum laevigatum*, together with *MlLa* ('*Laevigatum*') mildew resistance, mainly via cv 'Vada'. Further studies conducted by Giese et al. (1993) localized the 'Vada resistance' gene on barley chromosome 2 (2H). Thomsen et al. (1997) mapped this qualitative resistance gene in cv 'Alf' on the long arm of chromosome 2 (2H) and proposed *Rdg1* as its designation. However, *Rdg1* is not the only resistance gene present in barley germplasm, and several new sources of complete resistance have been found in European and non-European germplasm (Skou et al. 1994; Pecchioni et al. 1999). Recently, a new qualitative resistance gene to *P. graminea*, named *Rdg2* and carried by a highly resistant six-rowed winter barley variety, has been mapped on the short arm of chromosome 1 (7H) (Tacconi et al. 2001). Cultivars quantitatively resistant to leaf stripe have been commonly found in spring barleys (Skou et al. 1994). A major QTL effect accounting for more than half the variation in the trait and controlling the partial resistance of the two-rowed spring barley 'Proctor' to Dg2, a highly virulent *P. graminea* isolate, was mapped to the centromere of chromosome 1 (7H) and proposed as 'Proctor resistance', by analogy to 'Vada resistance' (Pecchioni et al. 1996). Also mapped in the same, or tightly linked, position in the barley genome (Qi et al. 1996) were the *RsmMx* qualitative resistance gene for BSMV (Barley Stripe Mosaic Virus), another seed-borne disease of barley (Edwards and Steffenson 1996), and the *Rpt4* gene for resistance to the spot form of *Pyrenophora teres* (Williams et al. 1999). The former was mapped in the spring barley cross 'Steptoe' × 'Morex'. The 'Steptoe' × 'Morex' barley map is the reference map of the NABGMP (the North American Barley Genome Mapping Project) showing many QTLs for disease resistance and agronomic traits in barley (http://www.css.orst.edu/barley/nabgmp/qtlsum.htm).

The first of the two primary objectives of the present study was finding quantitative trait loci (QTLs) for partial resistance to leaf stripe (*P.graminea*) by testing 150 DH (doubled-haploid) lines derived from the 'Steptoe' × 'Morex' cross with a highly virulent *P. graminea* isolate (Dg2), and comparing the resulting data against the previously found genes and QTLs of resistance to leaf stripe. The second involved resolving the question of race-specificity of QTLs in the barley/*P. graminea* interaction. However, while a number of studies have mapped several QTLs responsible for quantitative resistance to pathogens in barley (Pecchioni et al. 1996; Steffenson et al. 1996; Toojinda et al. 1998; de la Pena et al. 1999; Arru et al. 2002), they did not address the race-specificity question, and only recently have eight QTLs for resistance to two different isolates of barley leaf rust (*Puccinia hordei*) been found, three of them being effective in both isolates and five in only one (Qi et al. 1999). This finding supports the suggestion of Parlevliet and Zadoks (1977) that partial resistance may be due to minor gene-for-minor-gene interactions but is at odds with Van der Plank (1968), i.e. that quantitative resistance is horizontal in that such genes act against all pathogen isolates. This issue was thus pursued by inoculating the DH lines derived from the cross 'Steptoe' × 'Morex' with a second highly virulent leaf-stripe isolate (Dg5).

Materials and methods

Plant and fungal sources

The *P. graminea* isolates Dg2 and Dg5 are the most virulent in a collection of 12 Italian monoconidial isolates tested on European barley varieties (Gatti et al. 1992). An artificial inoculation test in a greenhouse with monospore isolate Dg2 was performed in 1999 on an F_1-derived population of 143 out of 150 doubled-haploid (DH) lines. The DHs were developed by the Oregon State University Barley Breeding Program for the NABGMP and kindly provided by P.M. Hayes (Department of Crop and Soil Science, Oregon State University, Corvallis, Ore. 97331-4501, USA) and by B.J. Steffenson (Department of Plant Pathology, University of Minnesota, St. Paul, Minn. 55108, USA). The DHs derived from the cross between the resistant parent 'Steptoe' (hulled, six-rowed, spring barley), a high yielding, broadly adapted Coast-type feed barley, and the susceptible parent 'Morex' (hulled, six-rowed, spring barley), a midwestern Manchurian-type with a North American six-rowed malting quality standard. The highly resistant cultivar 'Onice' (hulled, six-rowed, winter barley), the partially resistant 'Rondo' (naked, six-rowed, spring barley) and 'Proctor' (hulled, two-rowed, spring barley), and the highly susceptible 'Mirco' (hulled, six-rowed, winter barley) were used as reference lines in the experiment.

A second greenhouse inoculation test was carried out in the winter 2001/02 employing 138 of the 150 DH lines and the monoconidial isolate Dg5, including the reference cultivars (except cv 'Onice'); cv 'Thibaut', which is highly resistant to isolate Dg2 but highly susceptible to isolate Dg5 (Gatti et al. 1992), was added as a control.

Inoculation test

The DHs, the two parents and the control cultivars were artificially inoculated in 1999 (Dg2) and 2001 (Dg5) at the germination stage, after Pecchioni et al. (1996). One-hundred and twenty seeds of each line were sterilized in 70% Ethanol for 30 s and 5% NaOCl for 5 min, rinsed thoroughly in deionized water and then incubated in three Petri dishes (33 seeds each) between two PDA (Potato Dextrose Agar; Liofilchem, Teramo, Italy) layers colonized by an actively growing mycelium, and in one dish (21 seeds) with PDA but with no mycelium as a control. After 20 days of incubation in the dark at 6 °C, the emerged seedlings were transplanted to pots 12-cm in diameter and grown in the greenhouse until heading at 12 °C night (10 h dark) and 20 °C day (14 h light at a quantum flux density of 28 $\mu E\ m^{-2}\ s^{-1}$). A randomized, complete-block design with three replications of 30 plants (six pots of five plants/pot) and an uninfected control per line was used. Each block was placed on a separate greenhouse bench; a fourth bench acted as the uninoculated block, where the germinated control plants (18–20 per line) were sown in one or two pots and grown in the same conditions. Plants were treated with the foliar fungicide Bayleton Combi WP (triadimefon 2%, sulphur 50%) to control powdery mildew.

At heading, infected (showing leaf stripes) and healthy plants were counted. Resistance was assessed as the incidence of infec-

tion, i.e. the percentage of infected plants. No plants showing leaf stripe symptoms were found among the uninoculated controls.

Statistical analysis

ANOVAs of the resistance data were performed using MSTAT-C software (Freed et al. 1988, DOS version 2.10). The functions 'FREQ' and 'STAT' of MSTAT-C were used to analyze the frequency distribution of infected plant numbers for each isolate and its normality; and the function 'PLOT' for correlation analysis. The resistance data, calculated as the percentage of infected plants, were transformed by an arcsine function (arcsine $\sqrt{\%}$) for ANOVA and QTL mapping. Broad-sense heritabilities ($h^2 = \sigma^2g/\sigma^2p$) were calculated for both experiments (Dg2 and Dg5) on ANOVA results.

A 223-marker base map developed by D. Mather using NAB-GMP marker data provided by Andris Kleinhofs (Washington State University) and Andrzej Kilian (Washington State University), was used (Mather 1995). Markers cover the entire length of the map with an approximate average spacing of 2–5 cM. The SMBASEv2.MAP and SMBASEv2.MRK files were downloaded by an anonymous ftp from gnome.agrenv.mcgill.ca in the directory /pub/genetics/data/basemaps.

Interval mapping of QTLs via multiple regression (Haley and Knott 1992) was then performed using the upgraded version of the PLABQTL software (Utz and Melchinger 1996). After a preliminary run performing simple interval mapping (SIM) of putative QTLs, the markers with the highest LOD value were taken as co-factors for multiple QTL mapping by means of multiple regression (composite interval mapping, CIM). This procedure was repeated until a 'stable' picture of the LOD profile was achieved; a LOD (Log-Likelihood) threshold of 2.5 was considered as evidence for the existence of a QTL.

Results and discussion

Partial resistance to two leaf stripe isolates

The ANOVA on the resistance values of parents and DHs for the two isolates showed highly significant effects of the genotypes ($P < 0.001$) for the incidence of barley leaf stripe (both expressed as a percentage and as arcsine transformed data), while no significant differences were observed for the replications. The data showed that cv 'Steptoe' was partially resistant to both isolates (Fig. 1a: respectively 14.7% and 24.8% infected plants); cv 'Morex' was susceptible (Fig. 1b: 50.0% and 88.9%). The control genotypes behaved as expected; in particular cv 'Thibaut' is susceptible only to isolate Dg5 (75.6% infected plants). Figure 1 shows the distribution of the leaf stripe resistance to each isolate calculated as percentage of the infected plants. A lower population mean (40.6%) was detected for resistance to Dg2 in the DH population (Fig. 1a), with respect to that observed for partial resistance to isolate Dg5 (population mean = 60.0%; Fig. 1b). This, together with the higher infection values for the parentals, indicates that Dg5 is more aggressive than Dg2 on these genotypes. Dg2 results, on average, more virulent than Dg5 when tested on a large collection of barley cultivars; the two isolates are the most virulent out of 12 tested (Gatti et al. 1992).

In the 143 DHs the distribution of resistance to Dg2 was significantly not normal, although was normalized by the arcsine function (arcsine $\sqrt{\%}$) for the QTL analysis with PLABQTL software (Utz and Melchinger 1996). The distribution of resistance to Dg5 was not normal and significantly skewed even after algebraic transformation. Nevertheless, a previous comparison among results obtained with the parametric PLABQTL and a non-parametric QTL mapping software, released for non-normal traits (NEWQTL) on resistance data to *P. graminea* in other mapping populations, yielded substantially identical QTLs (Arru et al. 2002). This enabled us to use the PLABQTL software even for QTL mapping of the not-normally distributed resistance to isolate Dg5, after arcsine transformation. A significant transgressive segregation towards susceptibility was detected for resistance to Dg2 (Fig. 1a; $LSD_{(0.05)} = 15.19$), but not observed in the case of partial resistance to isolate Dg5. In this latter case, some lines resulted transgressively more resistant than the parent 'Steptoe' [Fig. 1b; $LSD_{(0.05)} = 13.24$].

Broad-sense heritabilities (σ^2_g/σ^2_p) for resistance to the two isolates Dg2 and Dg5, calculated on the percentage of infection, were 0.72 and 0.85 respectively. While these values may be overestimated because they come from a single year's experiment, very similar values of heritability were calculated for resistance to the same

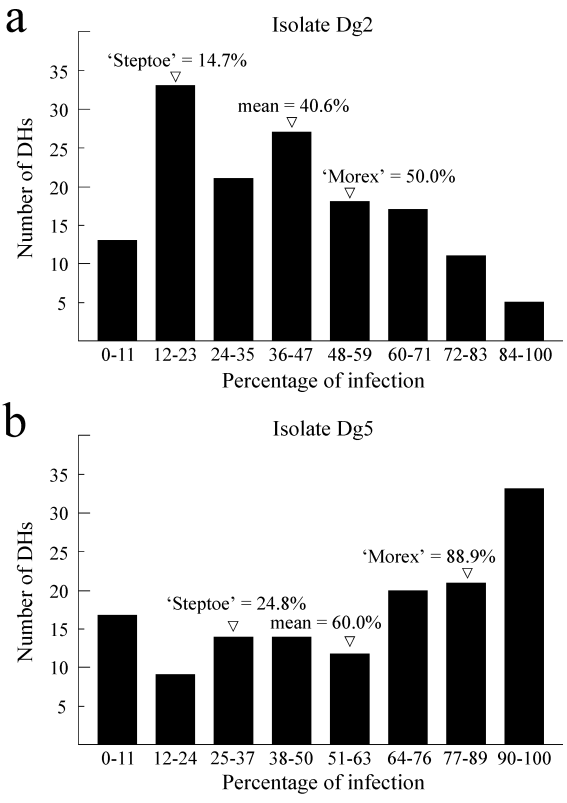

Fig. 1a, b Frequency distribution of phenotypes for resistance to leaf stripe expressed as a percentage of infection in doubled-haploid progeny derived from the cross 'Steptoe' × 'Morex'. Resistance values of the parents, and means of the population are shown and their position indicated by *arrows*. **a** Distribution of resistance to *P. graminea* isolate Dg2; **b** distribution of resistance to *P. graminea* isolate Dg5

disease for barley in the field (Delogu et al. 1989) and in the greenhouse experiments (Pecchioni et al. 1996; Arru et al. 2002), indicating a low influence of the environment on inoculation tests for this disease.

A significant correlation was found between the disease incidence of isolate Dg2 and that of isolate Dg5 ($r = 0.54$; $P < 0.001$), calculated on the percentage of infection of 135 DHs in common among the two experiments.

QTLs for partial resistance

Simple (SIM) and composite (CIM) interval mapping with the multiple regression approach were performed using the upgraded version of PLABQTL (Utz and Melchinger 1996). The CIM results, obtained using the other identified QTLs as cofactors, are presented in Table 1 and Fig. 2. The power and the precision of QTL detection by CIM analysis are improved and the bias in the estimated QTL position and effect is reduced (Jansen and Stam 1994).

Three QTLs for resistance to isolate Dg2 and three QTLs for resistance to Dg5 were detected on chromosomes 2 (2H), 3 (3H) and 7 (5H) of the barley genome. The parent 'Steptoe' contributed the resistance alleles for all the QTLs identified. The absence of resistance alleles originating from the susceptible parent is not in accordance with the transgressive segregations observed, a fact that may have been due to resistance QTLs of the susceptible parent. Nevertheless, this had already been observed for leaf stripe resistance in other DH mapping populations and did not lead to finding QTLs from the susceptible parent (Thomsen et al. 1997; Arru et al. 2002).

Taken together, the QTLs found for resistance to Dg2 accounted for a significant portion of phenotypic variance (41.4%), and those for resistance to Dg5 explained even a larger part of variation in the trait (70.5%; Table 1). Three out of six QTLs were located on chromosome 2 (2H), and the most important QTLs for resistance to both isolates coincide to a position on the long arm of that chromosome; they respectively explain 18.3% and 30.9% of the resistance (Table 1). This underlines the importance of chromosome 2 (2H) for the partial resistance to the disease, together with chromosome 1 (7H; Arru et al. 2002). In this case 'Steptoe' did not carry QTLs for resistance on chromosome 1 (7H), thus differentiating, with respect, to the model of partial resistance of the spring barley cultivar 'Proctor'.

The second QTL for significance and importance for the resistance to Dg5 (LOD 7.5 and 24.7% R^2) resides in a region of chromosome 3 (3H) at position 86 cM and is linked to the QTL for resistance to Dg2 at 76 cM. The two QTLs are mapped in the same BIN region of the BIN barley map (Kleinhofs and Graner 2000) and their support intervals overlap, thus not excluding their coincidence. These results however cannot demonstrate that the two QTLs are the same, but that there is tight linkage.

A major gene of resistance to leaf stripe, namely *Rdg1*, is mapped on the long arm of chromosome 2 (2H); in Robertson's (1985) view, the most important QTL on chromosome 2 (2H), and common to two isolates, might be a 'mild' allele of this qualitative gene. The *Rdg1* 'Vada resistance' gene was mapped tightly linked to marker aMSU21, being 0.2 ± 6.5 cM distal from it (Thomsen et al. 1997). The aMSU21 STS was mapped in the 'Proctor' × 'Nudinka' AFLP/RFLP map by Pecchioni et al. (1996) close (1.1-cM distal) to marker BCD266. The 'Proctor' × 'Nudinka' map thus can act as a 'bridge' map, since it is included as well as the 'Steptoe' × 'Morex' in the 'Barley Consensus 2' map (Qi et al. 1996; http://wheat.pw.usda.gov). In the consensus map BCD266 lies at 108.7 cM (with the *Rdg1* putative position at about 110 ± 6.5 cM distal); and the peak marker of the 'Steptoe' QTL, Pcr1, instead maps at 144.4 cM, approximating the linkage with *Rdg1* to about 27–34 cM. These results exclude the fact that the *Rdg1* gene of cultivar 'Alf' and the QTL of partial resistance of the cultivar 'Steptoe' are alleles of the same locus. Nevertheless, coincidence cannot yet be ruled out since the map position of *Rdg1* is not precise. Thomsen et al. (1997) mapped it distal with respect to the marker cMWG660 and the *MlLa* gene, while proximal to them in a previous paper (Giese et al. 1993). Moreover, Thomsen et al. (1997) mapped cMWG660 quite far from the chromosome 2 (2H) end, while the same marker in the Consensus 2 map is almost telomeric, being the last marker of the chromosome (Qi et al. 1996; http://wheat.pw.usda.gov).

The other known qualitative resistance locus to leaf stripe in barley *Rdg2* resides on a different chromosome (Tacconi et al. 2001). In Robertson's (1985) view, Pecchioni et al. (1999) indicated two barley resistance genes to two other seed-borne pathogens, *RsmMx* to BSMV (Edwards and Steffenson 1996) and *Rpt4* to *P. teres* (Williams et al. 1999) as possible candidate alleles of the 'Proctor resistance' QTL to *P. graminea*, because of their perfectly coincident map positions on chromosome 1 (7H). The *RsmMx* gene had been mapped on the 'Steptoe' × 'Morex' map. Parent testing has ruled out *RsmMx* as a candidate: while cultivar 'Steptoe' was the susceptible parent to BSMV. In any case no QTLs of resistance to leaf stripe have been mapped in the 'Steptoe' × 'Morex' cross on chromosome 1 (7H). The *Rpt4* candidate gene for resistance to a very similar fungus (*P. teres*), another species of the genus *Pyrenophora*, could not be verified as a candidate since parents of its mapping population were not different for leaf stripe resistance.

In a similar work on quantitative resistance to barley leaf rust, even Qi et al. (1998) did not find a map coincidence between leaf rust QTLs and all known mapped leaf rust R (*Rph)* genes, concluding that partial resistance to fungal pathogens is probably based on different mechanisms with respect to qualitative resistance.

Comparison of the locations of genes and QTLs involved in resistance to barley pathogens revealed a tendency of these genes to cluster. In barley there are both 'hetero-specific' (composed of genes acting against

Table 1 Results of the CIM analysis based on leaf stripe resistance to *P. graminea* isolates Dg2 and Dg5 for arcsine-transformed incidence data. Columns of the Table divide values related to Dg2 and Dg5. 'Position' indicates the QTL peak position in Kosambi cM on the barley chromosomes ('Chrom.'). 'Peak marker' the closest marker to the QTL peak; QTLs having the same peak marker are on the same line. 'R^2' the proportion of explained phenotypic variance; 'Add.' the estimated additive effects on the resistance (arcsine transformed values) of the alleles from 'Steptoe'. Sum 'R^2' a simple additive sum of single QTL R^2 values

Chrom.	Position		Peak Marker	LOD Dg2	LOD Dg5	R^2 (%) Dg2	R^2 (%) Dg5	Add. Dg2	Add. Dg5
	Dg2	Dg5							
2 (2H)	48 cM		ABG459	2.6		7.9		–4.1	
	168 cM	170 cM	Pcr 1	6.3	9.7	18.3	30.9	–6.4	–11.2
3 (3H)	76 cM		ABG377	5.1		15.2		–5.7	
		86 cM	ABG315		7.5		24.7		–9.4
7 (5H)		154 cM	WG908		4.2		14.8		–6.9
Sum						41.4	70.5		

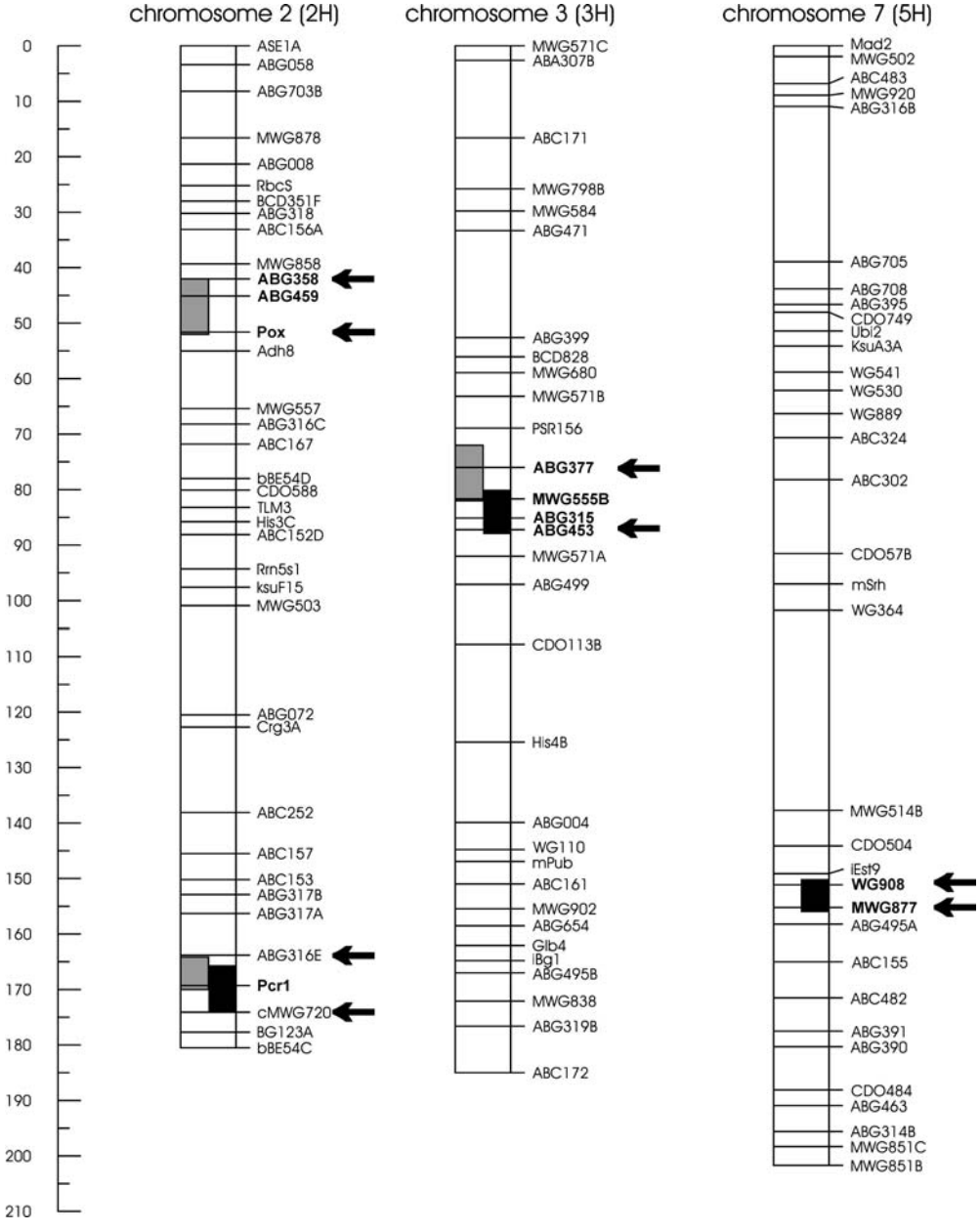

Fig. 2 Localization of QTLs for partial resistance to barley leaf stripe on the 'Steptoe' × 'Morex' base-map. Only the three chromosomes containing the detected QTLs are shown. Chromosomes are oriented with the short arm on the top. Distances (left) are given in Kosambi cM. Length of the rectangles corresponds to the two LOD support intervals identified by a LOD fall-off of 2 from the peak. *Markers in bold characters* are those located inside the two LOD support intervals; flanking markers of the QTLs useful for molecular-assisted selection are indicated by *black arrows*

Table 2 Summary of various barley pathogens with resistance QTLs coinciding with the QTLs responsible for leaf stripe resistance detected in the present study

Resistance	Chrom.	BIN[a]	Peak Marker or Interval	Authors
Leaf stripe (Dg2)	2 (2H)	4–5	ABG459	This work
Stripe rust	2 (2H)	4–5	ABG358–ABG459	Toojinda et al. (2000)
Fusarium head blight	2 (2H)	3–4	MWG858	De la Pena et al. (1999)
Fusarium head blight	2 (2H)	5	ABG459–MWG520A	De la Pena et al. (1999)
Net blotch	2 (2H)	4–5	ABG2–ABG459	Steffenson et al. (1996)
Leaf stripe (Dg2 and Dg5)	2 (2H)	14	Pcr1	This work
Leaf stripe (Dg2)	3 (3H)	8	ABG377	This work
Leaf stripe (Dg5)	3 (3H)	8	ABG315	This work
X. campestris	3 (3H)	8	ABG377–MWG555B	El Attari et al. (1998)
Leaf stripe (Dg5)	7 (5H)	12	WG908	This work
X. campestris	7 (5H)	12–13	ABC155	El Attari et al. (1998)

[a] BIN value indicates the position of the BIN marker(s) closest to the tagged QTL interval. BIN markers, following Kleinhofs and Graner (2000), are the 10-cM evenly spaced markers of 'Steptoe' × 'Morex' "Bin map" that are used as reference positions of chromosome segments

different pathogens) and 'homo-specific' (composed of genes that condition resistance against a single pathogen species) clusters of resistance genes (Graner et al. 1996; Ordon et al. 1998).

Table 2 summarizes mapping collinearities between the leaf stripe QTLs found in the present study and other QTLs of partial resistance to pathogens in barley. Data were collected from the web-based barley QTL summary (http://www.css.orst.edu/barley/nabgmp/qtlsum.htm), where the reference positions of the 'BIN map' and physical map are also given (Kleinhofs and Graner 2000; Kunzel et al. 2000). The BIN map is a 10-cM evenly spaced (Bins = 10 cM segments) reference map (Kleinhofs and Graner 2000). In the chromosome 2 (2H) region around the ABG459 marker, at least four QTLs coincide with the QTL of resistance to leaf stripe. The resistance QTL to net blotch *P. teres*; (Steffenson et al. 1996) was mapped in the same 'Steptoe' × 'Morex' population, 'Steptoe' being the resistant parent. All the other QTLs come from different mapping populations.

This region of the short arm of chromosome 2 (2H) is also an important cluster of QTLs for heading time (http://www.css.orst.edu/barley/nabgmp/qtlsum.htm), as mapped in very different barley populations. This trait to some extent might have pleiotropically influenced the resistance to such foliar diseases as stripe rust or net blotch, because they can infect and are measured on adult plants. However, given the capacity of the germinating seedling to avoid colonization by the *P. graminea* mycelium only at early growth stages (Haegi et al. 1998), it is very unlikely that resistance to leaf stripe is influenced by heading date.

Two out of three QTLs found for resistance to *Xanthomonas campestris* (El Attari et al. 1998) have been mapped in the 'Steptoe' × 'Morex' spring barley cross at positions on chromosomes 3 (3H) and 7 (5H) where the remaining three QTLs for resistance to *P. graminea* have been mapped (Table 2). The two pathogens are very different in the infection pathway and 'Steptoe' is susceptible to the bacterial streak (El Attari et al. 1998).

It is of interest to speculate whether the observed clustering of QTLs implies functional significance or whether it is only a consequence of genome organization. Indeed, such a line of enquiry could also be extended to alleles contributing to partial resistances and whether they, in conferring resistance to different pathogens, encode gene products of the same or of clusters of evolutionarily related loci.

In sum, the life cycles and the mode of infection of the pathogens against which the comapping QTLs listed in Table 2 are effective are very different from leaf stripe, except that in part *P. teres* can infect the barley seedlings. It is therefore more likely that there is a cluster of partial resistance QTLs in the tagged genomic regions, rather than they represent the same loci. However, at least in the case of net blotch and leaf stripe, the introgression from cultivar 'Steptoe' on the same interval of chromosome 2 (2H) by means of molecular-assisted selection (MAS) would lead to an increase of tolerance to both pathogens.

No QTLs of resistance to other barley pathogens reside in the genomic region tagged by marker Pcr1 on chromosome 2 (2H) (Table 2). Although QTLs for resistance to leaf rust (Qi et al. 1998, 1999) and to leaf stripe (Arru et al. 2002) were mapped on the same chromosome arm, they are difficult to compare with those found in this study mapped on AFLP marker maps.

Isolate-specificicity of QTLs

Figure 2 summarizes the map locations of QTLs of resistance to isolates Dg2 and Dg5 of *P. graminea*. The present study demonstrates that partial resistance to leaf stripe is governed in part by shared loci and in part by isolate-specific ones.

One resistance QTL is common for the two isolates on the long arm of chromosome 2 (2H), two QTLs are linked on chromosome 3 (3H), and the remaining two are isolate-specific, respectively for isolate Dg2 on chromosome 2 (2H) and for isolate Dg5 on chromosome 7 (5H). The QTL in common is that with the major effect on resistance for each isolate, explaining 18.3% and 30.9% R^2 respectively for Dg2 and Dg5 (Table 1).

Qi et al. (1999) found a similar situation for partial resistance to leaf rust in barley. They found three QTLs in common for effective resistance to two isolates at the adult stage; one had a similar effect against the two in terms of R^2 while the other two were differently weighted. The remaining QTLs were isolate-specific: two were effective against the first isolate, and three were specific for the second isolate. Even in other pathogen systems very different from barley and leaf blight, like potato/*Phytophthora infestans* (Leonards-Schippers et al. 1994) and pepper/*Potyvirus* (Caranta et al. 1997), clear isolate-specificity of QTLs has been observed. The present results and these previous findings thus acquire a more general significance, supporting Parlevliet and Zadoks's (1977) suggestion of isolate-specificity of partial resistance rather than Van der Plank's (1968) horizontal resistance theory.

Yet it is still an open question and hard to prove, also because their nature of QTLs, whether these resistance genes (major or minor) all interact in a gene-for-gene manner with genes for virulence or avirulence in the pathogen populations.

It is of interest that in all the studies cited, including the present, the QTLs common to the different isolates were those with the highest effects on the resistance. In particular Caranta et al. (1997) found a single major QTL acting against the three potyvirus strains tested. This might suggest the existence in plants of separate gene classes conferring either race-specific tolerance or horizontal tolerance to different strains of pathogens.

In this connection, and for the considerations above, the development of QTL-NILs (near-isogenic lines) carrying each common and isolate-specific QTLs, and a combination of the two in a susceptible background like 'Morex', would contribute to understanding the bases of partial resistance to pathogens in plants.

Accumulation of QTLs for partial resistance in breeding programs is one of the best ways to improve crops in modern agriculture, especially in an organic one. Figure 2 indicates eight markers flanking the four genomic regions that encompass all six QTLs for resistance to the two leaf-stripe isolates. After conversion into simple PCR markers and validation of the amplified products, they can be used in a MAS scheme to introduce tolerance to both isolates into elite barley breeding lines. In fact, following only eight markers it would be possible for introgression of isolate-specific QTLs, thus conferring a wider and durable resistance to leaf stripe for barley cultivars.

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