Theor Appl Genet (1998) 97: 1225–1234

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Mapping of quantitative trait loci affecting *Drechslera teres* resistance in barley with molecular markers

Received: 26 May 1998 / Accepted: 9 June 1998

Abstract Resistance loci for seedling-stage resistance to net blotch disease (*Drechslera teres*) in barley were mapped with molecular markers in an F_2 population derived from a cross between the susceptible barley cultivar 'Arena' and the resistant Ethiopian landrace 'Hor 9088'. Disease reactions were scored with first and second leaves of 2-week-old plants 7 and 9 days after inoculation with a single spore-derived isolate. For linkage analysis, 22 RFLP markers and 284 AFLP markers were used. The seven linkage groups covered 1153.3 cM with an average marker interval of 3.76 cM. The resistance was determined to be inherited in a quantitative manner. Altogether, 12 QTLs were mapped with positions depending on the leaf used for testing and the time period after infection. Heritability in the broad sense ranged between 0.21 and 0.37.

Key words *Hordeum vulgare* · *Drechslera teres* · Resistance · AFLP · Quantitative trait loci

Introduction

Net blotch is one of the most important foliar diseases of barley (*Hordeum vulgare* L.) worldwide. It is caused by *Drechslera teres* [Sacc.] Shoem. f.sp. *teres* Smedeg. (Teleomorph: *Pyrenophora teres* Drechs. f.sp. *teres* Smedeg.). Infection is favored in times of higher moisture levels and cooler temperatures (Tekauz 1986). It causes a net-type lesion which is characterized by dark brown blotches with a net-like pattern. These lesions are accompanied by chlorosis. The disease can cause yield losses of 20–30% in susceptible cultivars (Tekauz 1990). The fungus overwinters on the seed or on crop residues on the field. Spores are produced on infected plants and are spread by wind or rain. These spores are mainly responsible for epidemical spread of the disease (Seidel 1992).

Breeding for disease resistance is an aim in controlling this disease. The reaction of barley cultivars to *D. teres* is usually tested at the seedling stage in bioassays when plants are inoculated with the pathogen. A resistant seedling reaction is considered desirable because of generally good agreement with adult response in the field (Tekauz 1986). In previous studies, several barley genotypes and crosses were evaluated for resistance to *D. teres*.

The inheritance of disease resistance was first studied by Heschele (1928). He suggested a simple Mendelian mode. Schaller (1955) proposed a major gene (*Pt1*) conferring seedling resistance, which was located in barley cultivar 'Tifang' ('C.I. 4407-1'). The incompletely dominant character of this gene is shown in crosses with the varieties 'Atlas 46' or 'Atlas' ('C.I. 4118'). Mode and Schaller (1958) found two additional resistance genes, called *Pt2* and *Pt3*. The dominant gene *Pt2* was linked to *Pt1* at 2.57 recombination units and was located in the cultivars 'Ming' ('C.I. 4797'), 'Harbin' ('C.I. 4929') and 'Manchurian' ('C.I. 739') whereas *Pt3* seemed to be unlinked to the other genes. The gene *Pt3* was found in the varieties 'C.I. 4922' and 'C.I. 2750'. Khan and Boyd (1969) identified a single resistance gene (*Pta*) originating from three Manchurian barley cultivars ['Manchuria' ('C.I. 2335'), 'Ming' and 'Tifang']. In two Ethiopian varieties ('C.I. 5797' and 'C.I. 9819') the authors found two resistance genes, one of them was allelic to *Pta*.

Bockelman et al. (1977) examined trisomic plants and showed that in barley cultivar 'Tifang' resistance is mediated by one gene, called *Rpt1a*, which is located on chromosome 3H, while the cultivar 'C.I. 7584'

Communicated by G. Wenzel

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contained a single resistance gene on chromosome 2H, named *Rpt3d*. Two additional resistance genes (*Rpt1b* and *Rpt2c*) were located on chromosomes 1H and 3H in the variety 'C.I. 9819'. Wilcoxson et al. (1992) reported on oligogenic resistance originating from the cultivar 'C.I. 11531', which is used in the Minnesota Breeding Program.

In recent years, molecular markers have been employed for the mapping of net blotch resistance genes. A single dominant gene from the cultivar 'Igri' was localized on chromosome 3H (Graner et al. 1996). In contrast, earlier reports described the quantitative inheritance of this trait (e.g. Arabi et al. 1990; Steffenson and Webster 1992). Recently, Steffenson et al. (1996) reported three quantitative trait loci (QTLs) for seedling resistance. One was located on chromosome 4H and the others on chromosome 6H. For the resistance of adult plants, the authors found seven QTLs, with one QTL on each of the following chromosomes 2H, 4H, 5H, 6H and 7H and two QTLs on chromosome 3H. Both, seedling and adult plant resistance descended from the variety 'Steptoe'.

AFLPs allow rapid construction of a high-density genetic map thus facilitating the mapping of QTLs. Here, we report on the mapping of QTLs for net blotch resistance from a barley landrace. The QTL positions will be discussed in relation to previous mapping results.

Materials and methods

Plant material

Mapping was done in an F_2 population of 866 individuals derived from a cross between the susceptible variety 'Arena' and the resistant Ethiopian landrace 'Hor 9088' which was kindly provided by Professor G. Fischbeck, Freising/Weihenstephan. All plants were kept in a controlled environment at 20°C and a 16-h photoperiod in a greenhouse. After 2 weeks, the first two leaves were harvested for resistance tests. For the development of F_3 plants, F_2 individuals were self-pollinated.

Pathogen material and resistance tests

Drechslera teres f.sp. *teres*-isolate 04/6T, kindly provided by Dr. S. Hartmann, was used in this study. 'Arena' and 'Hor 9088' exhibited differential reactions to this isolate in a previous study (Hartmann 1993). The isolate was originally derived from a single conidium. Inoculum for inoculation was produced by cultivating the isolate on Sach's agar (Brandl and Hoffmann 1991) or on V-8 juice agar (Steffenson and Webster 1992) and subcultured by transferring single spores. The cultures were incubated at 20°C with a 12-h photoperiod. The optimal conidial concentration for resistance tests was determined as 10 000 conidia/ml.

A modified detached leaf test was used according to Hartleb and Krämer (1988). In each test, leaf segments were fixed on adhesive tape. The ends of the leaves were placed in filter paper moistened with benzimidazol solution (80 ppm) and then laid on plastic dishes. In each plastic dish, eight F_2 plants and leaves of the susceptible and the resistant parents were tested. Then, a conidia suspension was sprayed onto the leaves. After inoculation, plastic dishes were closed and the inoculated leaves were kept in the dark for 24 h at 100% relative humidity. They were cultivated in a growth chamber at 20°C with a 12-h photoperiod. Disease reactions were based on the amount of leaf chlorosis and necrosis in per cent and were scored 7 and 9 days after inoculation (dpi). The following traits were determined: "disease rating of the first leaf 7 days after infection" (trait 1/7), "disease rating of the first leaf 9 days after infection" (trait 2/7), "disease rating of the second leaf 7 days after infection" (trait 1/9) and "disease rating of the second leaf 9 days after infection" (trait 2/9).

Estimating heritability in the broad sense

Heritability in the broad sense was estimated between the F_2 and F_3 populations using the coefficient of regression. For this purpose, the mean of resistance-tested F_3 individuals and each parental F_2 individual was employed. Squared values of the coefficient of regression gave the heritability in the broad sense (Bos and Caligari 1995) which was determined for each trait.

Marker analysis

DNA was isolated from 134 randomly chosen F_2 plants of which 40 plants were used for RFLP analysis. All 134 probes were examined for AFLPs. The plasmids for RFLP analysis were kindly supplied by Dr. A. Graner (Gatersleben). Frozen leaves (5 g) were used for each extraction according to a modified CTAB method (Saghai-Maroof et al. 1984). For digestion, 7.5 µg of DNA was taken. The blotting was done after a modified protocol of Pillen et al. (1992). Filters were hybridized overnight at 65°C by random priming according to Feinberg and Vogelstein (1983). Seventy-five RFLP markers which had been previously mapped to the barley chromosomes were employed in the analysis (Graner et al. 1991).

For each AFLP reaction, 100 ng of DNA was double digested with *Eco*RI and *Mse*I. The pre-amplification was done with *Eco*RI- and *Mse*I-primers + 1 (*Eco*RI-A, *Mse*I-A and *Mse*I-C), each carrying one selective nucleotide. The following PCR was carried out in a Perkin Elmer 9600 thermocycler with radioactive (^{33}P)- or fluorescence-labelled *Eco*RI-primer + 3 (Schondelmaier et al. 1996; Reamon-Büttner et al. 1998).

Linkage and QTL analysis

Linkage analysis was done with the MAPMAKER/EXP. Vers. 3.0 (Lander et al. 1987) program package. The recombination units were transformed into centimorgan (cM) according to Haldane (1919). QTL analysis was utilized with PLABQTL (Utz and Melchinger 1996) which embodies that executes the idea of composite interval mapping (CIM; Jansen and Stam 1994; Zeng 1994) following the regression approach proposed by Haley and Knott (1992) with selected markers as co-factors. The degree of dominance was determined according to Stuber et al. (1987).

Results

In total, 866 F_2 individuals were tested for resistance to net blotch isolate 04/6 T. For disease scoring 7 or 9 days after inoculation of first and second leaves, a continuous distribution of their phenotypic frequencies was found (Table 1). The F_2 individuals showed varying phenotypes from resistance with no or few

Table 1 Reaction (amount of leaf chlorosis in %) of detached leaves of F_2 plants, susceptible parent 'Arena' and resistant parent 'Hor 9088' after inoculation with a single spore-derived *D. teres* f.sp. *teres* isolate 04/6T. Disease reactions were scored with first and second leaves 7 and 9 days after inoculation

Amount of leaf chlorosis in %	Number of leaves											
	Trait 1/7[a]			Trait 2/7[b]			Trait 1/9[c]			Trait 2/9[d]		
	F_2 plants	Arena	Hor 9088	F_2 plants	Arena	Hor 9088	F_2 plants	Arena	Hor 9088	F_2 plants	Arena	Hor 9088
0	341	8	766	166	0	680	568	70	858	422	0	842
10	113	16	61	88	0	73	113	74	7	142	27	7
20	72	7	8	80	0	90	63	103	0	83	41	8
30	30	16	0	43	0	6	18	68	0	36	72	8
40	41	20	0	36	0	16	20	30	0	25	51	0
50	53	78	0	65	23	0	14	212	0	32	107	0
60	39	54	0	52	46	0	13	48	0	27	38	0
70	27	54	0	38	35	0	3	56	0	14	63	0
80	54	166	0	80	70	0	3	122	0	18	154	0
90	9	117	0	48	62	0	1	21	0	8	155	0
100	86	329	0	169	629	0	49	61	0	58	157	0

[a] Disease scoring of the first leaf 7 days after inoculation
[b] Disease scoring of the first leaf 9 days after inoculation
[c] Disease scoring of the second leaf 7 days after inoculation
[d] Disease scoring of the second leaf 9 days after inoculation

Table 2 Distribution of RFLP and AFLP markers and genetic lengths of the seven barley chromosomes of the cross 'Arena' × 'Hor 9088'

	Chromosome							Total
	1H	2H	3H	4H	5H	6H	7H	
Length in cM	96.2	192.6	217.0	145.0	114.5	154.9	233.1	1153.3
Number of markers	29 (9.5%)	51 (16.6%)	73 (23.8%)	32 (10.5%)	26 (8.5%)	44 (14.4%)	51 (16.7%)	306
Loci defined by:								
RFLP markers	3	2	4	4	2	2	5	22
AFLP markers	26	49	69	28	24	42	46	284

disease symptoms up to totally attacked leaves (100% disease symptoms). The diseased leaf area ranged between 0 and 40% for the resistant parent, while the susceptible parent had an up to 100% attacked leaf area depending on the leaf tested and the time of scoring.

For RFLP analysis, 30 RFLP markers (40%) were found to be polymorphic, 44 monomorphic and one repetitive. For linkage analysis, 22 of the polymorphic RFLP markers were employed. Mapped RFLP markers served as anchors for the subsequent AFLP marker map. For AFLP analysis, 35 different *Eco*RI-*Mse*I primer combinations were analyzed. The average number of bands visible on gels was 60, with eight being polymorphic. A total of 284 AFLP markers were mapped. One per cent of the AFLP markers scored dominant with the rest co-dominant. Of the RFLP markers, 40% were found to be polymorphic. The polymorphism rate of the AFLP markers was 13.3%. For co-dominant marker analysis, 10% of the markers showed distorted segregation.

Segregation data were used for linkage analysis with the computer program MAPMAKER/EXP. (Lander et al. 1987). The resulting seven linkage groups covered 1153.3 cM. In total, 306 markers were mapped. The average marker interval was 3.76 cM. Chromosome 7H was the largest chromosome with 233.1 cM, and chromosome 1H the smallest with 96.2 cM (Table 2).

Segregation data of the molecular markers in combination with the data of the resistance test were used for QTL analysis (Table 3). The QTL positions on the chromosomes, their genetic effects, their LOD values and their explained phenotypic variances are given in Fig. 1. Different QTL positions were identified depending on the trait investigated.

Disease rating of the first leaf 7 dpi

For trait 1/7, QTL analysis resulted in three QTLs. They were localized on chromosomes 4HL and 6H. Based on the definition of Stuber et al. (1987), the QTL

Table 3 QTL detected for resistance to net blotch based on PLABQTL analysis, their chromosome positions, genetic effects, LOD values and explained phenotypic variances

Chromosome	Marker interval	Genetic effects			LOD[e]	R^{2f}
		a (additive)	d (dominant)	d/a		
Trait 1/7[a]						
6H	I3738_1-E4250_6	− 32.6	9.1	− 0.3	8.9	26.9
6H	E3847_9-I4140_9	− 18.6	11.9	− 0.6	3.4	12.4
4HL	I4140_6-MWG 517	27.7	10.5	0.4	3.3	12.1
Trait 2/7[b]						
6H	E4548_17-E3551_4	− 30.6	19.5	− 0.6	7.7	22.6
6H	E4048_1-E3847_9	− 23.7	14.5	− 0.6	3.0	10.3
3HL	E4547_13-E4047_4	− 22.3	− 0.5	0.02	3.0	19.2
Trait 1/9[c]						
2HS	E3748_2-E4050_7	28.9	2.7	0.1	4.9	16.6
2HL	I4133_7-E4449_D	− 9.9	− 7.6	0.8	4.4	15.1
1HL	E4547_12-MWG 943	14.6	3.2	0.2	2.9	10.6
Trait 2/9[d]						
4HS	I4232_8-I3738_5	− 12.5	− 0.6	0.1	5.3	16.8
3HS	E4048_2-E4048_10	14.1	1.9	0.1	3.2	10.8
3HS	I3738_6- I4240_8	1.5	− 12.3	− 8.4	2.8	9.7

[a] Disease scoring of the first leaf 7 days after inoculation
[b] Disease scoring of the first leaf 9 days after inoculation
[c] Disease scoring of the second leaf 7 days after inoculation
[d] Disease scoring of the second leaf 9 days after inoculation
[e] LOD score
[f] R^2: Phenotypic variation explained by QTLs in %

on chromosome 4HL showed a partial dominant allele effect from the susceptible parent. The two QTLs on chromosome 6H showed a partial recessive effect originating from the resistant parent. With the 'final simultaneous fit' procedure of the program PLABQTL, these three QTLs accounted for 24% of the total phenotypic variance with a LOD of 8.2.

Disease rating of the first leaf 9 dpi

After QTL analysis of trait 2/9, three QTLs could be mapped on chromosomes 3HL and 6H. All three QTLs originated from the resistant parent. The QTL on chromosome 3HL showed an additive allele effect and those QTLs on chromosome 6H a partial recessive inheritance. With the 'final simultaneous fit' procedure, with a LOD of 10.5, they accounted for 28% of the phenotypic variance.

Disease rating of the second leaf 7 dpi

For trait 2/7, QTL analysis resulted in three QTLs. They were localized on chromosomes 1HL and 2H. The QTLs on chromosomes 1HL and 2HS showed an additive allele effect, each descending from the susceptible parent. The QTL on chromosome 2HL was characterised by a partial dominant inheritance coming from the resistant parent. With the 'final simultaneous fit' procedure, with a LOD of 6.5, they explained 19.3% of the total phenotypic variance.

Disease rating of the second leaf 9 dpi

After QTL analysis of trait 2/9, three QTLs could be mapped on chromosomes 3HS and 4HS. The QTLs on chromosomes 3HS (from the susceptible parent) and 4HS (from the resistant parent) each showed an additive allele effect. The other QTL on chromosome 3HS expressed an over-recessive inheritance coming from the susceptible parent. With the 'final simultaneous fit' procedure, these QTLs accounted for the 30% of the phenotypic variance with a LOD of 11.4. Resulting values for heritability in the broad sense ranged from 0.21 to 0.37 depending on the trait.

Discussion

We mapped QTLs for net blotch resistance in a cross between a susceptible cultivar and a resistant landrace of barley. The landrace originated from Ethiopia, where other resistances to net blotch had been previously found (Buchannon and McDonald 1965; Khan and Boyd 1969; Khan and Portman 1979; Arabi et al. 1990). Skou and Haahr (1987) observed a moderate to high resistance in Ethiopian barley that varied greatly

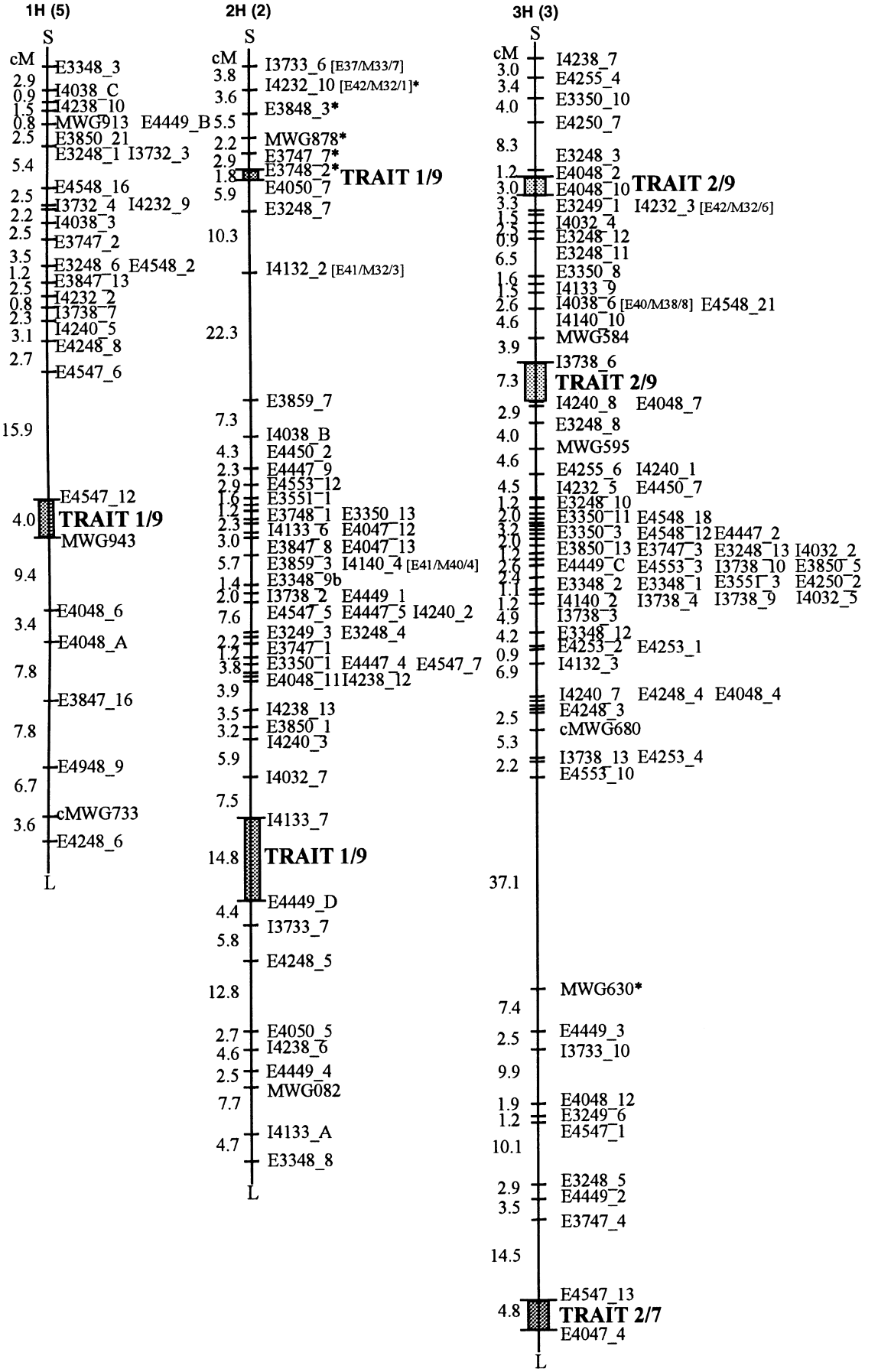

Fig. 1 See page 1231 for legend

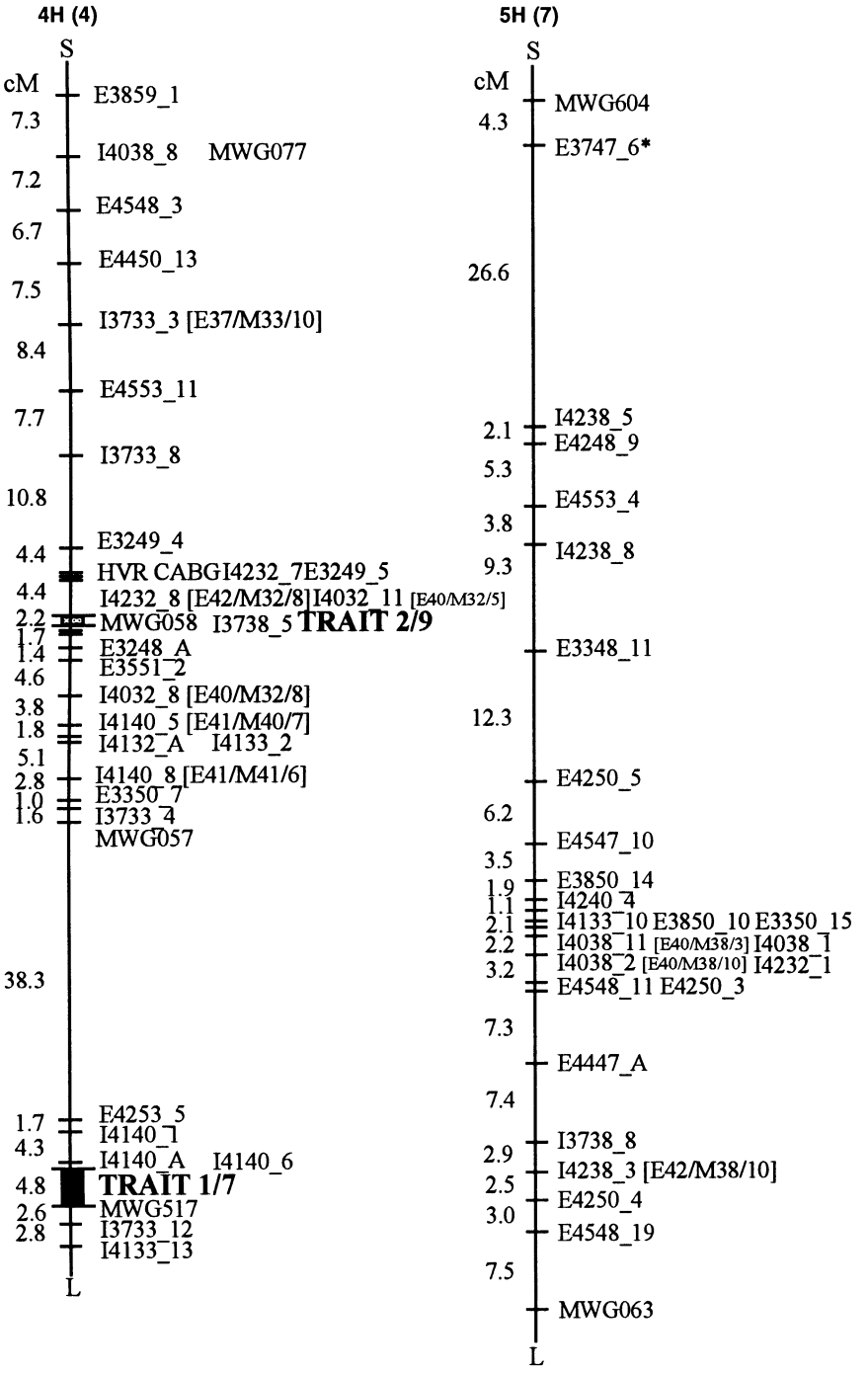

Fig. 1 See page 1231 for legend

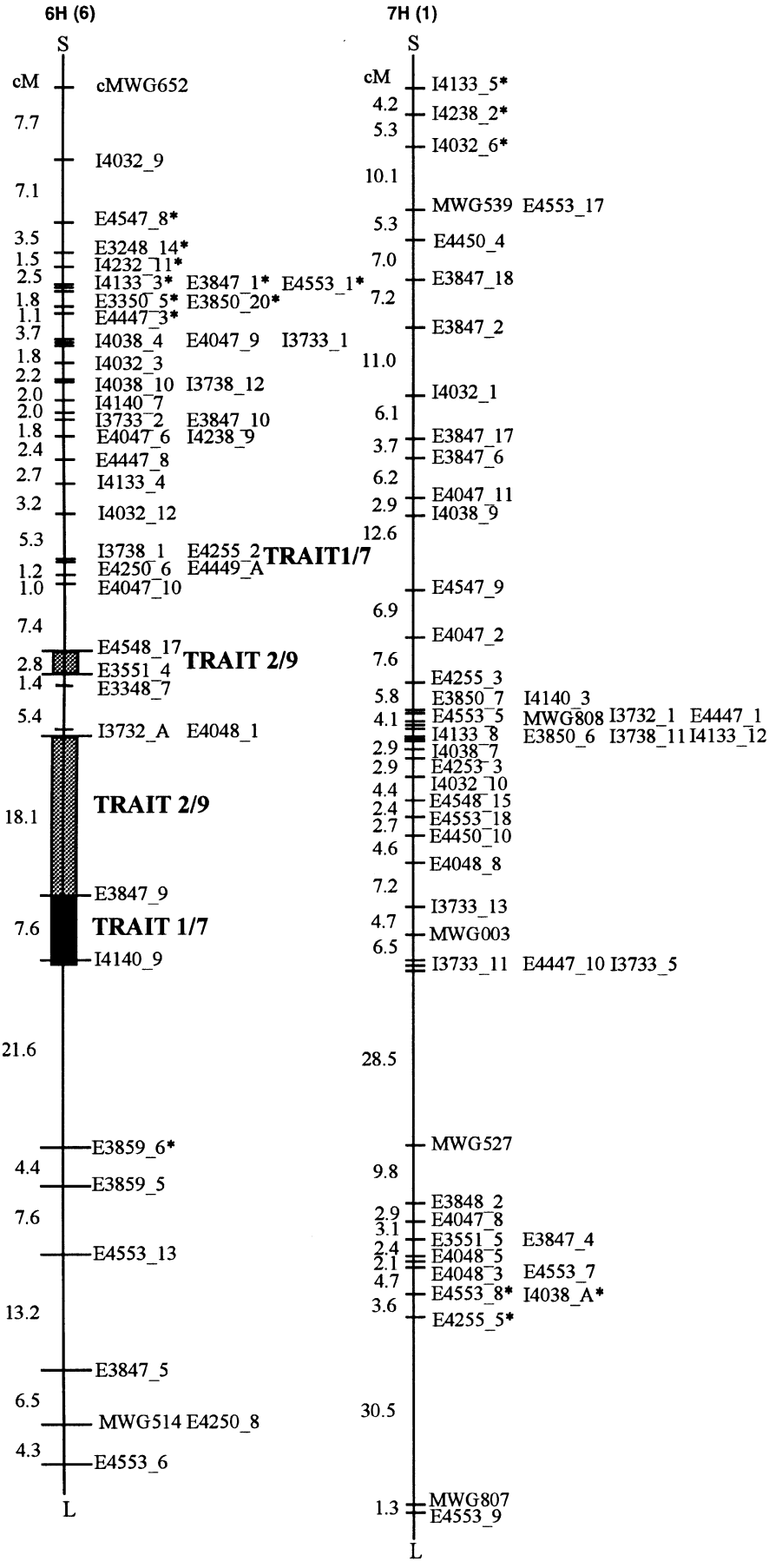

Fig. 1 Linkage map of barley based on the 'Arena' × 'Hor 9088' cross. Map distances are given in centimorgans (cM) according to Haldane (1919). Chromosomes are orientated with the short arm on the top ('*S*'). Common AFLP markers with Becker et al. (1995) are given in *square brackets* ([ ]) behind AFLP marker designations of this work. Markers showing distorted segregation are indicated by "*". QTL positions are indicated by different TRAIT *numbers and colours*, with TRAIT 1/7 being "disease rating of the first leaf 7 days after infection", TRAIT 2/7 being "disease rating of the first leaf 9 days after infection", TRAIT 1/9 being "disease rating of the second leaf 7 days after infection" and TRAIT 2/9 being "disease rating of the second leaf 9 days after infection"

with the isolates of the fungus tested. The detached leaf resistance test employed was slightly modified from that of Hartleb and Krämer (1988). The conidial concentration was determined as 10 000 conidia/ml. This concentration was also used for resistance testing by Afanasenko et al. (1995). At 7 and 9 days after infection, disease reactions were scored on the amount of leaf chlorosis in per cent, which other authors also used (Tekauz 1986; Deadman and Cooke 1987; Cherif and Harrabi 1990; Hartleb et al. 1990; Jana and Bailey 1995; Steffenson et al. 1996).

For the mapping of resistance factors, molecular markers such as RFLPs and AFLPs were used. The polymorphism rate with AFLP markers (13.3%) was smaller than that of RFLP markers (40%). A similar observation was made by Becker et al. (1995). In their study, the polymorphism rate with genomic RFLP probes was 27.8% and with AFLP markers 11.3%. A screen for QTL positions was made with the help of the computer program PLABQTL (Utz and Melchinger 1996). The program took into account the influence of genetic effects of other genomic regions. Therefore it was possible to map linked QTLs, too. With the same data material from a corn experiment, Lübberstedt et al. (1997) detected more than twice the number of QTLs than did Schön et al. (1994) with simple interval mapping (SIM).

In our material, the response of barley to pathogen attack was found to depend on leaf position (first or second leaf) and the time of disease scoring (7 or 9 days after inoculation). For all mapped positions of resistance QTLs, further loci for resistance to different pathogens were located in the same chromosomal regions according to the hypothesis of Robertson (1985). He postulated that qualitative resistance loci build the extremes of the alleles of quantitative loci. Therefore, qualitative and quantitative resistance loci should be localized in the same chromosomal region. Our results were in agreement with this hypothesis, because other resistance loci were found in the neighborhood of the mapped QTLs.

For the mapped QTL on chromosome 1H, resistances to *Erysiphe graminis* f.sp. *graminis* (*Ml-Ga* gene) and to *Cochliobolus sativus* were found in this chromosomal region (Hoissan and Sparrow 1991; Jensen et al. 1992; Steffenson et al. 1996). On chromosome 2HS, a QTL with a minor effect for resistance to *Puccinia graminae* and a QTL for resistance to net blotch of adult plants has been described (Pecchioni et al. 1996; Steffenson et al. 1996). For the characterized quantitative resistance loci on chromosome 3H, another QTL for resistance to net blotch in adult plants and distal on the long arm of this chromosome, as well as a resistance to the barley viruses BaYMV-1 and BaMMV (*ym4* gene), were found (Graner and Bauer 1993; Steffenson et al. 1996). One of the mapped QTLs for resistance to net blotch on chromosome 4H seemed to be linked to the *Ml-g* gene, that confered resistance to *E. graminis* f.sp. *hordei* (Görg et al. 1993). Another QTL for net blotch resistance both in seedlings and in adult plants was reported by Steffenson et al. (1996). The second resistance QTL of 4H was localized in a region, where the *ml-o* gene for resistance to powdery mildew and a quantitative resistance to *P. striiformis* f.sp. *hordei* were described (Hinze et al. 1991; Chen et al. 1994; Hayes et al. 1996). On chromosome 6H, the *Rph11* gene for resistance to *P. hordei*, two QTLs for resistance to net blotch in seedlings, and a resistance to *Rhynchosporium secalis* (*Rh13* gene) were found (Feuerstein et al. 1990; Abbott et al. 1995; Steffenson et al. 1996). Furthermore, genes for resistance seemed not to be equally distributed among chromosomes, but tended to build clusters. Thus, one could speculate about possible similar functions of their gene products.

For disease scoring of the first leaf 7 or 9 days after inoculation, similar QTL positions were found on chromosome 6H. One additional QTL was located for each scoring time (4HL and 3HL). No common QTL positions were found between the two leaves. For disease scoring of the second leaf, different QTL positions were mapped for disease scoring 7 or 9 days after inoculation. The three QTLs for resistance of the second leaf 7 dpi were located on chromosomes 1H and 2H. The three QTLs for resistance of the same leaf 9 dpi were mapped on chromosomes 3H and 4H. From the existence of different QTL positions, a stage-specific resistance to net blotch can be postulated. Other authors have also previously described this phenomenon. For example, Steffenson et al. (1996) commented on the localization of different QTLs for resistance to net blotch in seedlings and in adult plants. The authors found one QTL on chromosome 4H, that was in common for seedling and adult plant resistance. Tekauz (1986) reported an enhanced resistance in older plants. He suggested that younger and older leaves differ physiologically. Subsequently he described the first leaf being the most susceptible leaf (Tekauz 1990).

Similarly with Stuber et al. (1987), the detected QTLs showed additive and partial dominant genetic effects. One QTL exhibited over-recessive inheritance from the susceptible parent, which could be explained due to transgression. This means that the progeny will even be more resistant than the resistant parent. Cherif and Harrabi (1993) likewise reported transgression in barley that was infected with *D. teres* which was explained by the accumulation of resistance factors and possible additive or epistatic gene actions. The observed transgression in our study could be accounted for by additional resistance alleles from the susceptible parent 'Arena' (e.g. the QTL for trait 1/7 on chromosome 4HL). Comparable results were reported by Schön et al. (1993) in maize where two of seven QTLs for resistance to second-generation European corn borer originated from the susceptible parent.

A possible influence of the applied fungal isolate on the resistance reaction has been described (Skou and

Haahr 1987; Afanasenko et al. 1995). Since only one isolate was used in our study, we cannot clearly answer the question of whether the resistance is isolate-dependent. However, polygenic inheritance suggests that the net blotch resistance described here will be durable even in the presence of different isolates of *Drechslera teres*.

Acknowledgements We thank S. Reamon-Büttner for critical reading of the manuscript. This research was supported by the Bundesministerium für Bildung, Wissenschaft, Forschung und Technologie (BMBF), grant no. 03 10689 7.

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