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QTL analysis of tolerance to a German strain of BYDV-PAV in barley (*Hordeum vulgare* L.)

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Abstract One hundred and forty six barley doubled-haploid lines (DH lines) were tested for variation in grain yield, yield components, plant height, and heading date after artificial infection with a German isolate of barley yellow dwarf virus (BYDV-PAV-Braunschweig). Of these 146 lines 76 were derived from the cross of the barley yellow dwarf virus (BYDV) tolerant cultivar 'Post' to cv 'Vixen' (*Ryd2*) and 70 from the cross of Post to cv 'Nixe'. Phenotypic measurements were gathered on both non-infected plants and plants artificially inoculated with BYDV-PAV by viruliferous aphids in pot and field experiments for three years at two locations. For all traits a continuous variation was observed suggesting a quantitative mode of inheritance for tolerance against BYDV-PAV. Using skeleton maps constructed using SSRs, AFLPs and RAPDs, two QTLs for relative grain yield per plant after BYDV infection, explaining about 47% of the phenotypic variance, were identified in Post × Vixen at the telomeric region of chromosome 2HL and at a region containing the *Ryd2* gene on chromosome 3HL. In Post × Nixe, a QTL was found in exactly the same chromosome 2HL marker interval. In this cross, additional QTL were mapped on chromosomes 7H and 4H and together these explained about 40% of the phenotypic variance. QTL for effects of BYDV infection on yield components, plant height, and heading date generally mapped to the same marker intervals, or in the vicinity of the QTL for relative grain yield, on chromosomes 2HL and 3HL, suggesting that these regions are of special importance for tolerance to the Braunschweig isolate of BYDV-PAV. Possible applications of marker-assisted selection for BYDV tolerance based on these results are discussed.

Keywords Barley (*Hordeum vulgare* L.) · Barley yellow dwarf virus (BYDV-PAV) · Tolerance · QTL analysis

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Introduction

Barley yellow dwarf virus (BYDV) was first described in California (Oswald and Houston 1951) and is today one of the most economically important diseases of cereals world-wide causing yield losses up to 40% in barley (Lister and Ranieri, 1995). The isometric virus particles with a size of 25–30 nm (Hewings 1995) are persistently transmitted by aphids. Due to serological and molecular differences, several serotypes of BYDV have been distinguished (Rochow 1970). Serotypes RMV and RPV are classified as cereal yellow dwarf polerovirus while serotypes PAV and MAV belong to barley yellow dwarf luteovirus (Pringle 1998; Mayo 1999). In Northern Europe *Rhopalosiphum padi* and *Macrosiphum* (*Sitobion*) *avenae* are the most-prevalent vectors making BYDV-PAV and BYDV-MAV transmitted by these aphids especially important in this region (Plumb and Johnstone 1995). Major symptoms caused by BYDV, which is phloem-limited and causes vascular degradation of the phloem sieve tube, are dwarfing of shoots and roots and leaf yellowing. Furthermore, the number of ears per plant and kernel weight are reduced, heading-date is delayed, and the plants are more susceptible to abiotic stress and fungal diseases compared to healthy plants (D'Arcy 1995; Huth 1995). BYDV infections can be reduced by sowing cereals when aphid abundance is low and by application of insecticides. Since monitoring of aphid populations in the field is very laborious, most sprayings are prophylactic. For economical and ecological reasons, cultivation of highly BYDV-

tolerant cultivars with satisfactory grain yield would be advantageous.

In contrast to the soil-borne yellow mosaic inducing viruses (BaMMV/BaYMV), no complete resistance (immunity) to BYDV encoded by single genes (cf. Bauer et al. 1997; Graner et al. 1999; Ordon et al. 1999) is known in barley (Huth 1992; Habekuss 1994). Genes conferring tolerance, i.e. *ryd1* derived from the cultivar 'Rojo' (Suneson 1955) and *Ryd2* identified in Ethiopian landraces (Rasmusson and Schaller 1959), were found soon after the discovery of the disease. However, due to its low efficiency, *ryd1* has been only rarely used in barley breeding. In contrast, *Ryd2* has been incorporated into several barley cultivars such as 'Atlas 68' (Schaller and Chim 1969) and Vixen (Parry and Habgood 1986) as well as into more-recent breeding lines (Burnett et al. 1995; Delogu et al. 1995). *Ryd2*, which confers tolerance to BYDV (Skaria et al. 1985), has been mapped to the centromeric region of the long arm of chromosome 3H (Collins et al. 1996), and PCR-based markers have been developed (Ford et al. 1998; Paltridge et al. 1998). The level of tolerance depends to some extent on the genetic background, the virus isolate and the environmental conditions (Catherall and Hayes 1966; Qualset 1975; Schaller 1984). Besides this, several authors suggest the presence of multiple alleles at the *Ryd2* locus (Catherall et al. 1970; Chalhoub et al. 1995).

Reliable selection for BYDV tolerance requires artificial inoculation using viruliferous aphids (Qualset 1984; Comeau 1992) facilitating transmission of a well-defined isolate to plants at the same developmental stage (Baltenberger et al. 1987). Tolerance cannot be determined based on the virus content of infected plants only, as ELISA values do not always correlate with symptom expression (Henry and Vivar 1998) and yield losses (Huth 1995; Scheurer et al. 2000). Rather, an assessment of tolerance should incorporate additional parameters such as plant height and especially grain yield after BYDV infection (Qualset 1992), of which the latter, from the breeder's and farmer's points of view, is the most important character.

Besides *Ryd2*, different sources of tolerance such as that found in the cultivar Post (Burnett and Mezzalama 1990; Burnett et al. 1995; Huth 1995) have been identified. For effective breeding of BYDV-tolerant cultivars knowledge of the genetics of tolerance is a prerequisite. QTL analyses, followed by the establishment of marker-based selection procedures, are of special importance in breeding for tolerance to barley yellow dwarf virus as a reliable bioassay is both time consuming and laborious. QTL analysis has been widely applied in barley for different pathosystems (e.g. Hayes et al. 1996; Pecchioni et al. 1996; Qi et al. 1998; De la Pena et al. 1999; for a review see Ordon et al. 1998) and tolerance to BYDV-MAV and BYDV-PAV has recently been studied, via QTL analysis, by Toojinda et al. (2000). BYDV tolerance QTL have also been mapped in oat (*Avena sativa*, Jin et al. 1998). In barley, the use of homozygous doubled-haploid lines (DHs, Foroughi-Wehr and Wenzel 1990; Devaux et al. 1996), which allow reliable and replicated tests for tolerance and facilitate the efficient use of dominant marker systems (Powell et al. 1996), provide a powerful approach for QTL mapping studies. The aims of this study were (1) to analyse the inheritance of tolerance against a German strain of BYDV-PAV, and (2) to identify the chromosomal location of 'genes' or QTL contributing to this trait.

Materials and methods

Genetic analyses were carried out on 76 F_1-anther culture-derived doubled-haploid lines (DHs) of a cross between Post and Vixen and 70 DH lines of a cross between Post and Nixe, which mainly served for verification purposes of QTL which derived their positive alleles from Post. Post is a six-rowed cultivar which was derived from CIMMYT and has proven to be highly tolerant against German strains of BYDV-PAV (Huth 1995). The two-rowed cultivar Vixen, which carries the *Ryd2* gene, derived from the cross ('Coracle'× 'Igri') × Igri was released in the UK in 1986 (Parry and Habgood 1986). The six-rowed cultivar Nixe (Pedigree: 'Hauter'×'Dura'× Breeding line×'Barbo'×'Banteng', Baumer and Göppel 1994) was registered in Germany in 1990 and is resistant to barley mild mosaic virus (BaMMV) and barley yellow mosaic virus (BaYMV) due to the presence of *rym4* (Ordon et al. 1995). In previous experiments, Post displayed a high level of tolerance against a German isolate of BYDV-PAV (Braunschweig isolate) while Vixen and especially Nixe showed lower levels of tolerance (Huth 1995).

As the success of testing for BYDV tolerance entirely depends on the infection rate, DH lines were artificially infected by viruliferous aphids (*Rhopalosiphum padi*) in the greenhouse at the single-leaf stage using the Braunschweig strain of BYDV-PAV. A minimum of five aphids fed on each plant resulting in a 100% infection rate. Aphids were killed after a four-day inoculation period by an insecticide (Pirimor TM). Due to the fact that the virus content estimated by DAS-ELISA is only weakly correlated with yield losses caused by the virus isolate used (Huth 1995; Scheurer et al. 2000), the level of tolerance of parents and DH lines was assessed by measuring kernel yield per plant, thousand-kernel weight, ears per plant, kernels per ear, and plant height on infected and non-infected controls of the same DH lines in field (Braunschweig, Lower Saxony, Germany) and pot experiments (Rauischholzhausen, Hesse, Germany) in the growing seasons 1996/97, 1997/98 and 1998/99, followed by calculating the relative performance of each line for the respective trait, [i.e. (infected variant/healthy control)×100]. Additionally, the heading date was recorded in pot experiments. For better comparison of the results between lines, data were calculated on the single-plant level.

For field experiments at Braunschweig, 15 plants of each DH line were artificially infected in September and 12 of these plants, as well as non-infected control plants of the same DH line, were taken to the field at the end of October. For pot experiments plants were artificially infected in the same way, but infected plants as well as their healthy controls were vernalised for 6 weeks in a growth chamber (4°C) and transferred to Mitscherlich pots (6 l, soil:sand mixture 2:1, 0.4 g P, 1.6 g K, 0.2 g Mg, 1.5 g $CaCo_3$) in March in two replications of five plants per pot and variant (infected/control) and DH line. During the growing season plants were sprayed with insecticides and fungicides regularly in order to protect controls from BYDV infection and all plants from fungal diseases. In addition, trials were protected against bird damage by nets.

All data were analysed by ANOVA and the segregation of each trait was tested for a fit to a Gaussian distribution by Kolmogorov-Smirnoff statistics (α=5%) using the software package SPSS 8.0 (SPSS Iinc., 1998).

Genomic DNA of barley leaves was extracted according to Doyle and Doyle (1990). In order to identify markers, Post, Vixen and Nixe were screened for polymorphisms by 25 *Eco*RI+

3/*Mse*I+3 AFLP primer combinations, 280 RAPD primers, and 59 SSRs. Molecular analyses using these techniques were performed as described by Ordon et al. (1995) for RAPDs, Vos et al. (1995) and Schiemann et al. (1999) for AFLPs, and Liu et al. (1996) and Russell et al. (1997) for SSRs. AFLP and SSR detection was carried out on a DNA sequencer (LiCor L-4200S-2, MWG Biotech, Ebersberg). RAPDs and AFLPs were named according to the respective primer or primer combination and fragment size, or numbered from the smallest to the biggest fragment (AFLPs). To align maps, markers which showed segregating polymorphic RAPD or AFLP fragments of the same size in both populations were preferred for mapping. Additionally, the STS cMWG680 (Graner and Tekauz 1996), the *V*-locus (number of rows) and *Ryd2* using the CAPS marker YlpPCRM (Ford et al. 1998) were scored in Post × Vixen. Post × Nixe was screened for resistance to BaMMV (*rym4)* using mechanical inoculation in the greenhouse followed by DAS-ELISA (Ordon and Friedt 1993).

Construction of genetic linkage maps was performed according to Schäfer-Pregl et al. (1999) with the software package MAPMAKER (Lander et al. 1987) using Haldane's (1919) mapping function. SSRs and morphological markers with known chromosomal locations were used as anchor markers to assign linkage groups to chromosomes. Two- and three-point analyses were conducted at LOD 3.0 and markers were assigned to linkage groups defined by anchor markers using the "assign" command starting at a LOD 5.0 followed by LOD 4.0 and 3.0. The "order" command (LOD 3.0) was used to order markers within linkage groups and those markers without unique placement were integrated by the "build" command starting at LOD 5.0 and ending at LOD 1.75. For the construction of the map all markers which were uniquely placed by this procedure were chosen. All mapped markers were tested for the expected 1:1 segregation ratio using a χ^2 goodness of fit test.

QTL analysis of barley yellow dwarf virus tolerance was performed with the software package PLABQTL 1.0 (Utz and Melchinger 1996) employing the method of composite interval mapping (CIM). For detection of putative QTL a LOD threshold of 3.0 was chosen for declaring significance. QTL analyses were carried out for each environment (data not shown) and across all environments with cofactors obtained by the PLABQTL procedure *cov SELECT*. QTL positions were determined at the maximum of the LOD plot curve. The explained phenotypic variance of each QTL and of all detected QTL was calculated in a simultaneous fit.

Results

Construction of genetic maps

Out of 87 polymorphic RAPD fragments and 181 polymorphic AFLP loci, a skeleton map of Post × Vixen was constructed by eliminating co-segregating markers and those linked closer than 1.5 cM. The resulting map had an average marker spacing of 12.1 cM covering 1,328 cM and comprised 56 AFLP markers, 33 RAPD markers, 25 SSRs, and one morphological (*V*-locus), CAPS and STS marker (Fig. 1). YlpPCRM (Ford et al. 1998), which is very closely linked to the *Ryd2* locus, was mapped on the long arm of chromosome 3H as reported by Collins et al. (1996) and the *V*-locus was integrated on chromosome 2H. However, few locus-specific polymorphic

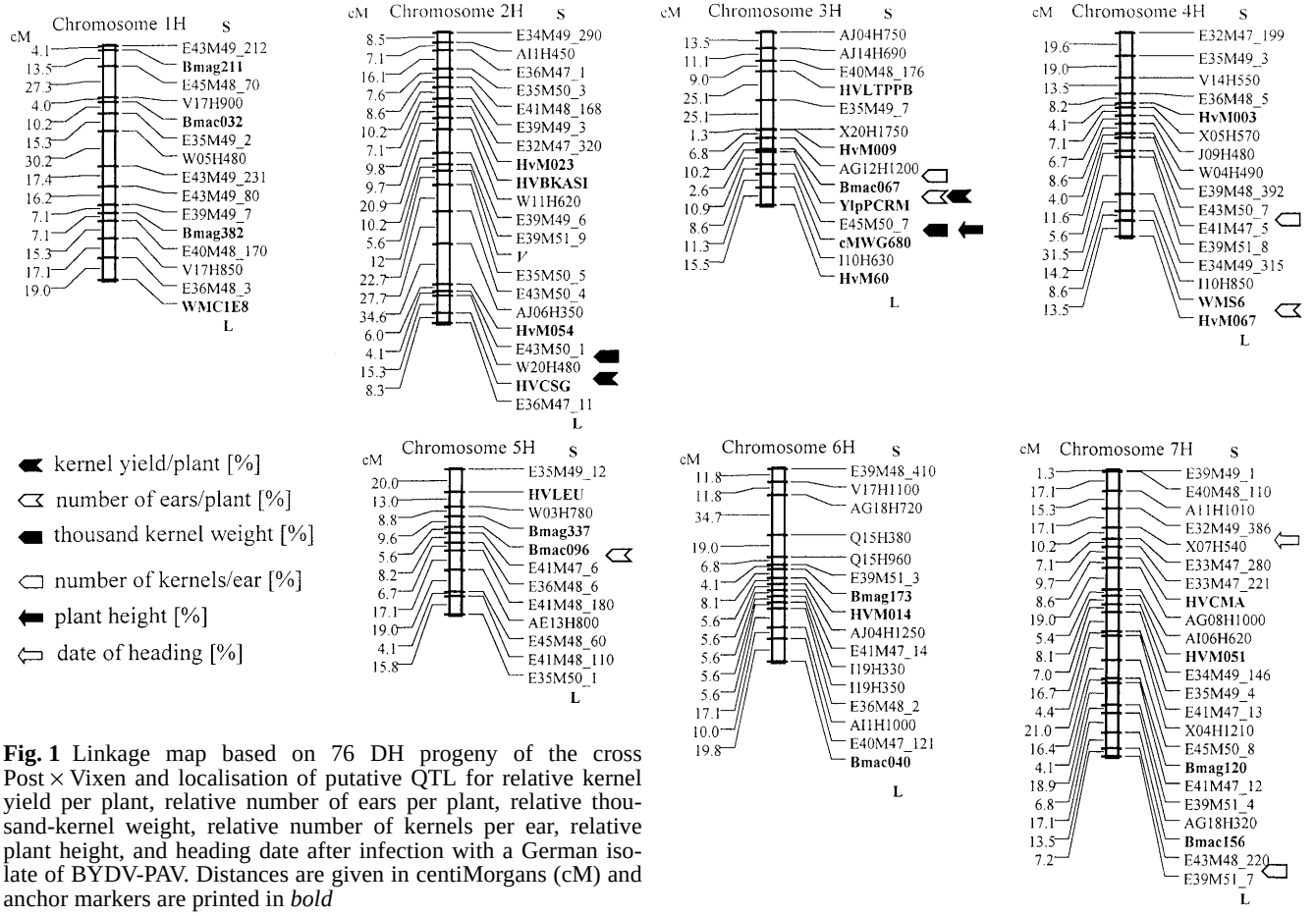

Fig. 1 Linkage map based on 76 DH progeny of the cross Post × Vixen and localisation of putative QTL for relative kernel yield per plant, relative number of ears per plant, relative thousand-kernel weight, relative number of kernels per ear, relative plant height, and heading date after infection with a German isolate of BYDV-PAV. Distances are given in centiMorgans (cM) and anchor markers are printed in *bold*

Table 1 Means, standard deviation, minima–maxima (Min-Max), and Kolmogorov-Smirnoff-statistics of parental lines and respective DH populations assessed in three years (1997–1999) at two locations (Braunschweig, Rauischholzhausen) for the traits kernel yield/plant, number of ears/plant, thousand-kernel weight, number of kernels/ear, plant height, and heading after artificial BYDV-PAV infection relative to non-infected controls of the same genotype and correlation coefficient of these traits to the relative kernel yield/plant

Trait	Post	Vixen	Nixe	Post × Vixen	Min-Max	P[a]	r[b]	Post × Nixe	Min-Max	P[a]	r[b]
Kernel yield	77.5±17.0	84.5±20.3	58.1±10.8	75.5±11.9	52.2–102.6	0.32	–	65.2±11.6	41.7–89.1	0.78	–
Ears/plant	95.0±32.6	99.1±26.3	81.7±23.8	88.5±10.4	67.6–117.2	0.91	0.61**	81.9±12.4	52.4–110.4	0.85	0.85**
TKW	96.8±9.4	89.5±4.1	90.5±5.7	93.5±3.9	84.5–103.0	0.65	0.52**	89.8±4.8	78.1–103.7	0.82	0.57**
Kernels/ear	97.4±22.4	96.6±3.6	71.5±0.9	91.7±8.9	69.4–119.2	0.51	0.41**	79.3±8.6	62.7–102.5	0.82	0.67**
Plant height	98.1±3.3	96.5±4.2	86.8±11.3	94.2±4.2	84.1–102.7	0.93	0.50**	91.8±4.8	80.3–100.0	0.25	0.55**
Heading date	101.9±6.7	100.1±4.5	110.8±5.3	106.3±4.6	97.8–119.4	0.86	–0.26*	104.3±5.4	92.9–118.8	0.91	–0.31*

[a] =P<0.05 indicates a significant deviation from a Gaussian distribution
[b] r=correlation to the relative kernel yield; *, ** correlation significant at the 0.05 and 0.01 levels, respectively

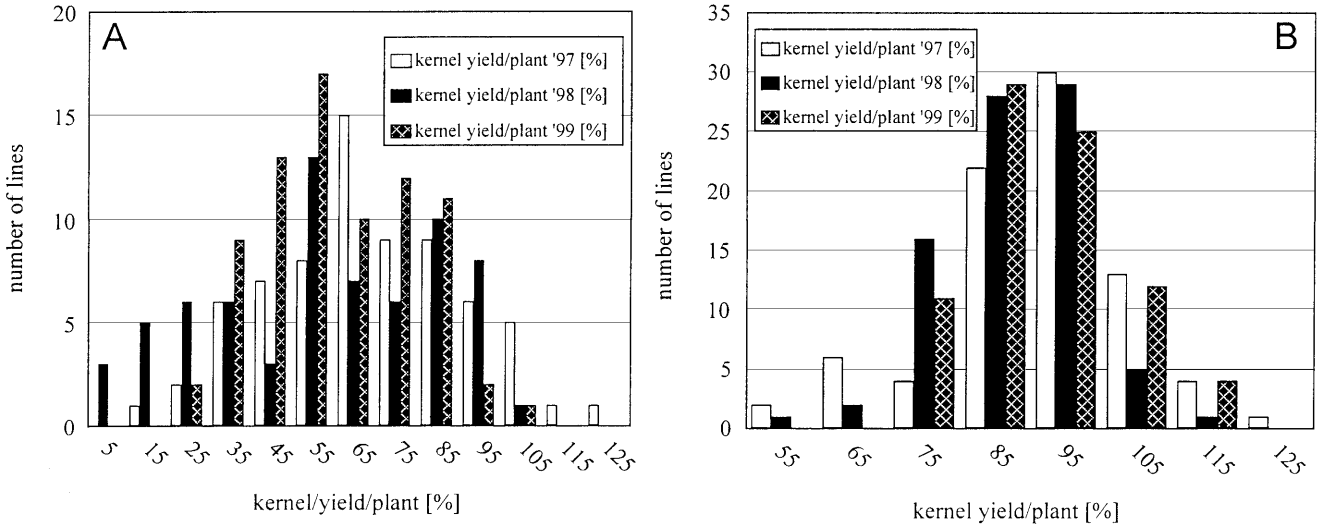

Fig. 2 Distribution of the relative kernel yield after BYDV-PAV infection of 76 DH lines of the cross Post × Vixen in field trials at Braunschweig (**A**) and pot trials at Rauischholzhausen (**B**) during seasons 1996/1997 (‘97), 1997/1998 (‘98), and 1998/1999 (‘99)

markers were identified for the long arm of chromosome 5H, so this chromosome is mainly represented by its short arm in this map. The order of the SSRs corresponds to published data of Liu et al. (1996) and the Scottish Crop Research Institute (http://www.scri.sari.ac.uk/SSR/) except for SSRs Bmag211 and Bmac032 on chromosome 1H.

In Post × Nixe, 83 RAPD and 196 AFLP fragments were polymorphic and a skeleton map with an average spacing of 8.4 cM spanning 958.5 cM was constructed consisting of 70 AFLPs, 28 RAPDs, 23 SSRs, and the resistance gene *rym4* located on chromosome 3HL (Graner and Bauer 1993). In this map, chromosomes 2H and 3H are subdivided into two linkage groups located on the long and short arms of the respective chromosomes. Furthermore, due to a lack of polymorphic markers, the map represents mainly the short arms of chromosomes 5H and 1H (data not shown, Scheurer 2001). The order of the SSRs is again in accordance with published data.

Phenotypic analysis of BYDV tolerance

A high degree of variation regarding the reaction to BYDV-PAV infection ranging form ‘highly tolerant’ to ‘non-tolerant’ was observed in both DH populations in all environments (Table 1). However, yield losses due to virus infection were more pronounced in field than in pot experiments (cf. Scheurer et al. 1998). As can be seen in Fig. 2 for the relative kernel yield in field and pot experiments of Post × Vixen, the distributions observed in all years and locations fit to a Gaussian distribution, but the average relative kernel yield in field trials was 68.5%, 57.3% and 60.5% in 1997, 1998 and 1999 in comparison to 90.9%, 87.2% and 91.0%, respectively, in the pot experiments. This may be due to the optimal water supply allowing tillers, which normally would die in the field, to develop ears and kernels in pots. However, in all experiments, a significant genotypic effect and influence of virus infection on relative grain yield and the additional parameters measured was found (data not shown, cf. Scheurer et al. 2000; Scheurer 2001). The mean parental value for kernel yield/plant was 77.5% for Post, 84.5% for Vixen and 58.1% for Nixe. In contrast to earlier results (Huth

Table 2 Putative QTL for relative kernel yield/plant, relative number of ears/plant, relative thousand-kernel weight, relative number of kernels/ear, relative plant height averaged over six environments, and relative heading date (three environments) after BYDV-PAV infection estimated on 76 DH lines of the cross Post × Vixen

Chromosome	Position [cM]	Marker interval	LOD	Simultaneous fit			Additive effects [%]	Positive allele
				σ^2_p [a]	Combined σ^2_p	σ^2_g [b]		
Kernel yield (%)								
2H	234	W20H480–HVCSG	3.3	19.6			4.0	Post
3H	104	Bmac067–YlpPCRM	7.7	31.8			7.1	Vixen
					46.8	73.7		
Ears/plant (%)								
3H	104	Bmac067–YlpPCRM	3.6	22.1			3.7	Vixen
4H	174	WMS6–HvM067	3.0	8.6			3.2	Vixen
5H	52	Bmac096–E41M47_6	4.7	16.9			3.9	Vixen
					34.0	97.7		
Thousand-kernel weight (%)								
2H	228	E43M50_1–W20H480	4.2	14.5			1.5	Post
3H	124	E45M50_7–cMWG680	5.4	27.8			2.0	Vixen
					32.4	60.2		
Kernel/ear (%)								
7H	246	E43M48220–E39M51_7	4.9	12.9			3.6	Vixen
3H	100	AG12H1200–Bmac067	6.1	17.7			4.7	Vixen
4H	100	E43M50_7–E41M47_5	4.6	16.5			3.7	Post
					34.1	81.5		
Plant height (%)								
3H	122	E45M50_7–cMWG680	3.6	19.1	19.1	46.2	2.2	Vixen
Heading date (%)								
7H	50	E32M49386–X07H540	3.4	18.5	18.5	31.0	–1.9	Post

[a] σ^2_p=phenotypic variance
[b] σ^2_g=genotypic variance

1995), Vixen turned out to be highly tolerant against the Braunschweig strain of BYDV-PAV in the three-year trials. Consequently, the average relative yield after BYDV infection was about 10% higher in Post × Vixen than in Post × Nixe.

For all investigated traits, Kolmogorov-Smirnoff statistics indicate a good fit to a Gaussian distribution (Table 1). Besides yield and yield components, plant height was also reduced by BYDV infection. However, although severe stunting to relative 45% of healthy controls was observed, e.g. in the field trials of 1997/1998 (data not shown), height was reduced on average by about 10%. In addition to these traits, relative heading date was measured in the pot experiments (Table 1). As a sign of virus infection, heading date was on average delayed in the infected variants of both populations to nearly the same extent. While Post and Vixen needed about the same time until heading in infected as in uninfected controls, the development of ears was delayed in Nixe. In general, in Post × Vixen the number of positive phenotypic transgressive segregants for all measures of tolerance was higher than in Post × Nixe, indicating that in the former cross both parents contribute positive tolerance alleles. In both populations all traits were significantly positively correlated to the relative grain yield/plant after BYDV infection, except the relative date of heading which, as expected, showed a negative correlation (Table 1).

QTL analyses

Based on estimations across six environments, the QTL listed in Tables 2 and 3 were detected for BYDV tolerance with a LOD>3.0 by composite interval mapping (CIM). Of these traits, the relative kernel yield after BYDV infection is the most important criterion from the breeder's and farmer's point of view. Across all environments two QTL for the relative kernel yield/plant were detected in Post × Vixen. The positive allele of the QTL on chromosome 2H, which explains 19.6% of the phenotypic variance, is derived from Post. This QTL is flanked by the RAPD marker W20H480 and the SSR HVCSG. The second QTL was detected on chromosome 3H in an interval defined by the SSR Bmac067 and the CAPS marker for *Ryd2*, YlpPCRM. At this QTL, the allele for a higher tolerance level is derived from Vixen. This QTL explains 31.8% of the phenotypic variance in the simultaneous fit. Its additive effect is higher (7.1%) than the QTL on chromosome 2H (4.0%). Together these two QTL explain 46.8% of the phenotypic and 73.7% of the genotypic variance of the relative grain yield after BYDV infection. In Post × Nixe, which mainly served for verification of QTL which derived positive alleles from Post, three QTL were detected for the relative grain yield per plant on chromosomes 7H, 2H and 4H. The positive additive effects of these QTL were contributed by Post. The QTL on chromosome 2H which has the

Table 3 Putative QTL for relative kernel yield/plant, relative number of ears/plant, relative thousand-kernel weight, relative number of kernels/ear, relative plant height averaged over six environments, and relative heading date (three environments) after BYDV-PAV infection estimated on 70 DH lines of the cross Post × Nixe

Chromosome	Position [cM]	Marker interval	LOD	Simultaneous fit			Additive effects [%]	Positive allele
				σ^2_p [a]	Combined σ^2_2	σ^2_g [b]		
Kernel yield (%)								
7H	12	E39M4880–E38M4860	5.9	13.5			8.4	Post
2H	28	W20H480–HVCSG	9.4	27.6			7.4	Post
4H	144	E39M487–E33M48275	3.7	9.6			4.1	Post
					39.7	75.5		
Ears/plant (%)								
7H	22	Bmag359–AE19H380	3.6	10.4			4.3	Post
2H	32	W20H480–HVCSG	6.1	28.5			5.7	Post
5H	82	Bmac096–E33M48278	3.4	16.5			4.3	Post
					40.8	59.4		
Thousand-kernel weight (%)								
2H	34	HVCSG–E36M4709	8.9	38.7	38.7	61.2	3.3	Post
Plant height (%)								
2H	28	W20H480–HVCSG	7.3	37.6	37.6	70.8	2.8	Post
Heading date (%)								
2H	28	W20H480–HVCSG	4.3	18.3	18.3	65.3	–2.7	Post

[a] σ^2_p=phenotypic variance
[b] σ^2_g=genotypic variance

largest effect (σ^2_p=27.6%) maps to exactly to the same interval (W20H480-HVCSG) as the QTL for this trait found in Post × Vixen. Together these QTL account for 39.7% of the phenotypic variance in Post × Nixe.

Three QTL for the relative number of ears/plant were detected in the population Post × Vixen. Individual QTL explain 8.6% to 22.1% of the observed phenotypic variance. At all loci the allele for the higher number of ears was derived from Vixen. The QTL with the largest effect (σ^2_p=22.1%) coincided with the QTL for relative grain yield on chromosome 3H. In the simultaneous fit, all three QTL explained 34.0% of the phenotypic and 97.7% of the genotypic variance. In Post × Nixe three QTL were also found for this trait. The QTL on chromosome 7H maps 10 cM proximal to the QTL for relative grain yield (Table 3). The QTL on chromosome 2H has the largest effect (σ^2_p=28.5%) and maps to the same interval as the QTL for the relative grain yield. Together these QTL account for 40.8% of the phenotypic variance. Two QTL were detected for relative thousand-kernel weight in Post × Vixen. As with the relative kernel yield/plant, these QTLs map to the long arms of chromosomes 2H and 3H, respectively. While the peak of the LOD score on chromosome 2H nearly coincides with the locus of a LOD peak for relative kernel yield, the QTL for thousand-kernel weight on chromosome 3H maps 20 cM distal to the QTL for relative kernel yield. Nevertheless, for both, the favourable alleles are inherited from the same parent as for relative kernel yield/plant. The QTL on chromosome 3H has a major effect (σ^2_p=27.8%), while that from Post explains only about half this amount (σ^2_p=14.5%) of the phenotypic variance. The QTL with the positive allele inherited from Vixen also has the larger additive effect (2.0%). Together these QTL explain 32.4% of the phenotypic variance observed and 60.2% of the genotypic variance. Only one QTL, with the positive allele derived from Post, explaining 38.7% of the phenotypic variance, was detected for thousand-kernel weight in Post × Nixe. It maps to an interval adjacent to the QTL for relative grain yield and the relative number of ears per plant on the long arm of chromosome 2H. For the relative number of kernels/ear, three QTL were detected in Post × Vixen but none were found in Post × Nixe. Vixen alleles have positive effects at the QTL on chromosomes 7H and 3H, while at the QTL on chromosome 4H the positive allele derives from Post. The partial phenotypic variance explained varied from 12.9% to 17.7%. A model fitting all QTL for the relative number of kernels/ear explains 34.1% of the phenotypic variance. The genotypic variance explained was estimated at 81.5%. The position of the QTL on chromosome 3H is in an interval adjacent to the *Ryd2* region and the peak of the LOD score is only about 4 cM proximal from the position of the QTL for relative grain yield on this chromosome. Neither of the QTL on chromosomes 7H and 4H coincide with any other QTL in these regions.

Only one QTL was detected for relative plant height in both populations. In Post × Vixen, the favourable allele for plant height after BYDV-PAV infection was derived from Vixen. The QTL maps to the same region on the long arm of chromosome 3H as the QTL for the relative kernel yield/plant, the relative number of ears/plant, the relative thousand-kernel weight, and the relative number of kernels/ear. It explains 19.1% of the phenotypic and 46.2% of the genotypic variance observed. In Post × Nixe

Table 4 Additive effects of QTL for the relative kernel yield/plant on chromosome 2H and 3H estimated on 76 DH lines of the population Post × Vixen at single locations and years [Braunschweig (BS), Rauischholzhausen (RH), 1997–1999]

Map-position	Positive allele	BS 1997	RH 1997	BS 1998	RH 1998	BS 1999	RH 1999
2HL, 234 cM	Post	11.75	0.83	7.64	0.18	7.38	2.32
3HL, 104 cM	Vixen	12.35	1.11	14.72	1.71	9.51	3.89

the positive allele for plant height was contributed by Post on chromosome 2H in the interval W20H480-HVCSG, which also harbours QTL for relative grain yield and the relative number of ears per plant. There is also a QTL for relative thousand-kernel weight in an adjacent interval. The relative heading date was only measured at one location in three years. Averaged over three years, one QTL was detected in each population. In Post × Vixen the allele for a reduced delay in heading, an advantage concerning BYDV tolerance, was derived from Post. In Post × Nixe, a Post allele in the interval W20H480-HVCSG on chromosome 2H was also advantageous. These QTL explain 18.5% and 18.3% of the phenotypic variance in Post × Vixen and Post × Nixe, respectively.

In summary, QTL related to all traits which were used as measures for BYDV tolerance were detected. However, for nearly all of these traits, a significant QTL × environment interaction was observed, which may largely be explained by the experimental design, i.e. field trials and pot experi-ments. For example, concerning relative grain yield in Post × Vixen, the Post allele at the QTL on 2H always had a positive effect, as did the allele from Vixen at the QTL on chromosome 3H. However, large differences were observed concerning the additive effects in Rauischholzhausen (pot experiments) and Braunschweig (field trials) in all years (Table 4), explaining significant QTL × environment interactions. Nevertheless, the results presented here indicate that in the crosses analysed, the distal part of the long arm of chromosome 2HL as well as the *Ryd2* gene in the centromeric region of chromosome 3HL are of special importance for tolerance to infection with the Braunschweig isolate of BYDV-PAV.

Discussion

In this study, two major QTL for tolerance to BYDV, infection, i.e. relative kernel yield after BYDV-PAV infection, were identified in Post × Vixen (Table 2) and three QTL in Post × Nixe (Table 3). The QTL on the long arm of chromosome 2H with the positive allele derived from Post maps to exactly the same marker interval in both populations, i.e. W20H480 – HVCSG (cf. Tables 2, 3), suggesting that this chromosomal region is of special importance for the BYDV tolerance derived from Post. This is emphasised by the fact that in this region QTL for the relative number of ears per plant, relative thousand-kernel weight, plant height and heading date were also detected (cf. Tables 2, 3). The same holds true for the major QTL on chromosome 3HL detected in the marker interval harbouring the *Ryd2* gene. The penetrance of this locus depends to some extent on the genetic background, the environmental conditions, and the virus isolate, as shown in earlier investigations (Skaria et al. 1985; Ranieri et al. 1993).

In contrast to the results of Hayes et al. (1996) who detected distinct classes concerning *Ryd2*, in our studies about 30% of the phenotypic variance of the relative grain yield after infection by a Braunschweig isolate of BYDV-PAV is explained by this locus, which has already proven its value in released cultivars like Atlas 68 (Schaller and Chim 1968), Vixen (Parry and Habgood 1986), and cv 'Naturelle' recently released in France (Le Gouis, personal communication). Although the QTL analyses reported here were carried out on relatively small populations due to limitations in our capacity to artificially infect with viruliferous aphids, which is essential for reliable testing (cf. Qualset 1984; Comeau 1992), a major QTL on chromosome 2H has been verified and the importance of the *Ryd2* gene has been shown. It is likely that, due to the experimental design and the LOD threshold chosen, only few QTL with major effects were detected in our experiments. As a result, the genetic and phenotypic variances explained by these QTL (Tables 2, 3) are likely to be over-estimated (Melchinger et al. 1998; Utz et al. 1998).

When compared to yield and other quantitatively inherited traits, only few QTL with large effects have been detected in most studies regarding quantitative resistance in barley. For example, for *Pyrenophora graminea*, two QTL explaining 58.5% and 29.3% of the phenotypic variance were detected (Pecchioni et al. 1996), and two QTL conferring resistance to *Cochliobolus sativus* in the adult stage accounted for 70.1% of the phenotypic variance (Steffenson et al. 1996). Similar outcomes have been reported for powdery mildew (Backes et al. 1996), bacterial leaf streak caused by *Xanthomonas campesrtris* pv. *hordei* (El Attari et al. 1998), and cereal aphids in barley (Moharramipour et al. 1997). For quantitatively inherited virus resistance in other cereals such as in maize, one major and three minor QTL encoding resistance to maize streak virus (MSV) were detected by Welz et al. (1998) with similar results obtained by Pernet et al. (1999a). Although the latter authors detected more QTL in additional studies, some were located in the same genetic intervals as in the previous study (Pernet et al. 1999b). For sugarcane mosaic virus (SCMV), two major and three minor QTL

Table 5 Marker genotypes of loci flanking QTL for BYDV tolerance on Post and Vixen and on German winter barley cultivars

Cultivar	QTL on chromosome 2H[a]		QTL on chromosome 3H[b]	
	HVCSG	W20H480	Bmac067	YlpPCRM
Post	A	A	A	A
Vixen	B	B	B	B
Asorbia	C	B	C	A
Arizona	B	B	B	A
Arcona	B	B	D	A
Baretta	B	B	D	A
Brunhild	C	A	C	A
Hanna	B	B	B	A
Igri	B	B	B	A
Kira	C	B	D	A
Juwel	C	B	B	A
Jolante	C	B	D	A
Magie	A	B	D	A
Nixe	B	B	C	A
Posaune	B	B	D	A
Sarah	C	B	D	A
Tokyo	B	B	D	A

[a] Positive allele derived from Post
[b] Positive allele derived from Vixen

were found in maize (Xia et al. 1999), and for resistance to wheat streak mosaic virus (WSMV) and high plains virus (HPV) three and two major QTL, respectively, were identified (Marcon et al. 1999).

In the present study two and three QTL for relative grain yield after BYDV infection have been detected, which explain about 47% and 40%, respectively, of the phenotypic variance in the populations analysed (Tables 2, 3). Similar results were obtained in oat where three loci explaining 47% of the phenotypic variance for BYDV tolerance were identified (Jin et al. 1998). Using plant height reduction and tillering scores as parameters for BYDV tolerance, Toojinda et al. (2000) detected QTL on chromosomes 7H, 4H and 1H explaining 43%, 39% and 32% of the phenotypic variance of tolerance to BYDV-MAV and 18% and 17% of the tolerance to BYDV-PAV. Therefore, phenotypic variance explained for BYDV tolerance in the present study is in the same range as in previously published data. QTL for BYDV tolerance were also mapped on chromosomes 4H and 7H in our studies (Tables 2, 3). However, the precise location of the QTL on chromosome 7H does not correspond to the map position given by Toojinda et al. (2000). In general, chromosome 7 of cereals may be of special interest for BYDV tolerance because in both *Thinopyrum intermedium* and wheat (*Triticum aestivum*) tolerance has been assigned to this chromosome (Singh 1993; Larkin et al. 1995; Hohmann et al. 1996). In the crosses analysed in the present study, QTL on chromosomes 4H and 7H are of minor importance in comparison to those on chromosomes 2H and 3H which, due to the phenotypic variance explained and the fact that QTL were estimated in independent samples (cf. Melchinger et al. 1998), may at least be suited for a marker-based pre-selection of more-tolerant genotypes, thereby reducing the number of plants to be tested by artificial inoculation followed by long-lasting field tests.

It is important to note that for the respective QTL, all flanking markers are PCR-based and, therefore, well suited to meet the high-throughput requirements of practical barley breeding [i.e. Bmac067 and YlpPCRM (Ford et al. 1998) concerning the *Ryd2* region, and HVCSG (Liu et al. 1996) and W20H480 concerning the QTL mapped on chromosome 2HL]. With the exception of W20H480, all flanking markers are also co-dominant and therefore suited for use in heterozygous segregating populations. In order to obtain information about the potential use of the markers flanking the QTL in practical barley breeding programmes, a set of recent barley cultivars – including the cultivars used here and high yielding German winter barley cultivars – have also been genotyped (Table 5). For HVCSG, polymorphism between Post and all German cultivars tested so far except cv 'Magie' has been observed. In the main, this is also the case for the RAPD-marker W20H480. Polymorphism between Vixen and all German cultivars was found for YlpPCRM, while concerning Bmac067 the same allele as in Vixen was detected in some cultivars. As a consequence, it appears that these markers, especially YLpPCRM, are well-suited to monitoring the introgression of positive alleles derived from Post and Vixen (*Ryd2*) into adapted breeding lines in order to improve BYDV tolerance, similar to the situation demonstrated already in barley [e.g. for *Puccinia striiformis* f.sp. *hordei* by Toojinda et al. (1998)]. Furthermore, since closely linked PCR-based markers for *rym4* and *rym5* are already available (cf. Graner et al. 1999; Ordon et al. 1999), by exploiting the markers described in this paper in tandem with those for *rym4*/*rym5*, it will be possible to simultaneously screen for multiple virus resistance genotypes using MAS-based tests.

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References

Backes G, Schwarz G, Wenzel G, Jahoor A (1996) Comparison between QTL analysis of powdery mildew resistance in barley based on detached primary leaves and on field data. Plant Breed 115:419–421

Baltenberger DE, Ohm HW, Foster JE (1987) Reactions of oat, barley and wheat to infection with barley yellow dwarf isolates. Crop Sci 27:195–198

Bauer E, Weyen J, Schiemann A, Graner A, Ordon F (1997) Molecular mapping of novel resistance genes against barley mild mosaic virus (BaMMV) Theor Appl Genet 95:1263–1269

Baumer M, Göppel W (1994) Gerste 1994, Sorten, Züchter, Ursprungsland, Zulassungsjahr, Abstammung. Bayerische Landesanstalt für Bodenkultur und Pflanzenbau, Freising-München

Burnett PA, Mezzalama M (1990) The barley yellow dwarf screening programm at CIMMYT. In: Burnett PA (ed) World perspectives on barley yellow dwarf. CIMMYT, Mexico D.F., Mexico, pp 434–440

Burnett PA, Comeau A, Qualset CO (1995) Host plant tolerance or resistance for control of barley yellow dwarf. In: D'Arcy CJ, Burnett PA (eds) Barley yellow dwarf. Forty years of progress. APS Press, St. Paul, Minnesota, pp 321–343

Chalhoub BA, Sarrafi A, Lapierre HD (1995) Partial resistance in the barley (*Hordeum vulgare* L.) cultivar Chikurin Ibaraki 1 to two PAV-like isolates of barley yellow-dwarf virus: allelic variability at the *Yd2* gene locus. Plant Breed 114:303–307

Catherall PL, Hayes JD (1966) Assessment of varietal reaction and breeding for barley yellow dwarf virus in barley. Euphytica 15:39–51

Catherall PL, Jones AT, Hayes JD (1970) Inheritance and effectiveness of genes in barley that condition tolerance to barley yellow dwarf virus. Ann Appl Biol 65:153–161

Collins NC, Paltridge NG, Ford CM, Symons RH (1996) The *Yd2* gene for barley yellow dwarf virus resistance maps close to the centromere on the long arm of barley chromosome 3. Theor Appl Genet 92:858–864

Comeau A (1992) The use of artificial inoculation with viruliferous aphids in barley yellow dwarf virus research. In: Comeau A, Makkouk KM (eds) Barley yellow dwarf in West Asia and North Africa, pp 177–182

D'Arcy JC (1995) Symptomatology and host range of barley yellow dwarf. In D'Arcy CJ, Burnett PA (eds) Barley yellow dwarf. Forty years of progress. APS Press, St. Paul, Minnesota, pp 107–127

De la Pena RC, Smith KP, Capettini F, Muehlbauer GJ, Gallo-Meagher M, Dill-Macky R, Somers DA, Rasmusson DC (1999) Quantitative trait loci associated with resistance to *Fusarium* head blight and kernel discoloration in barley. Theor Appl Genet 99:561–569

Delogu G, Cattivelli L, Snidaro M, Stanca AM (1995) The *Yd2* gene and enhanced resistance to barley yellow dwarf virus (BYDV) in winter barley. Plant Breed 114:417–420

Devaux P, Zivy M, Kilian A, Kleinhofs A (1996) Doubled haploids in barley. In: Scoles G, Rossnagel B (eds) Proc Vth Int Oat Conf and VIIth Int. Barley Genet Symp. Invited Papers, Saskatoon, Canada, University Extension Press, Saskatoon, Sakatchewan, pp 213–222

Doyle JF, Doyle JL (1990) Isolation of plant DNA from fresh tissue. Focus 12:13–15

El Attari H, Hayes PM, Rebai A, Barrault G, Dechamp-Guillaume G, Sarrafi A (1998) Potential of doubled-haploid lines and localization of quantitative trait loci (QTLs) for partial resistance to bacterial leaf streak (*Xanthomonas campestris* pv *hordei*) in barley. Theor Appl Genet 96:95–100

Ford CM, Paltridge NG, Rathjen JP, Moritz RL, Simpson RJ, Symons RH (1998) Rapid and informative assays for *Yd2*, the barley yellow dwarf virus resistance gene, based on the nucleotide sequence of a closely linked gene. Mol Breed 4: 23–31

Foroughi-Wehr B, Wenzel G (1990) Recurrent selection alternating with haploid steps – a rapid breeding procedure for combining agronomic traits in inbreeders. Theor Appl Genet 80:564–568

Graner A, Bauer E (1993) RFLP mapping of the *ym4* virus resistance gene in barley. Theor Appl Genet 86:689–693

Graner A, Tekauz A (1996) RFLP mapping in barley of a dominant gene conferring resistance to scald (*Rhynchosporium secalis*). Theor Appl Genet 93:421–425

Graner A, Streng S, Kellermann A, Schiemann A, Bauer E, Waugh R, Pellio B, Ordon F (1999) Molecular mapping and genetic fine-structure of the *rym5* locus encoding resistance to different strains of the barley yellow mosaic virus complex Theor Appl Genet 98:285–290

Habekuss A (1994) Evaluation of winter barley for resistance to barley yellow dwarf virus. Genet Pol 35B:199–202

Haldane JBS (1919) The combination of linkage values and the calculation of distance between the loci of linked factors. J Genet 8:299–309

Hayes PM, Prehn D, Vivar H, Blake T, Comeau A, Henry I, Johnston M, Jones B, Steffenson B, St. Pierre CA, Chen F (1996) Multiple disease resistance loci and their relationship to agronomic and quality loci in a spring barley population. J Agric Genet, http://www.ncgr.org/research/jag/papers96/paper296/indexp296.html

Henry M, Vivar H (1998) Resistance to BYDV in barley. BYDV-Newslett 1998, http://www.cimmyt.mx/Research/Wheat/BYDVNews7-2.htm#barley

Hewings AD (1995) Purification and virion characterization of barley yellow dwarf viruses. In: D'Arcy CJ, Burnett PA (eds) Barley yellow dwarf. Forty years of progress. APS Press, St. Paul, Minnesota, pp 165–179

Hohmann U, Badaeva K, Busch W, Friebe B, Gill BS (1996) Molecular cytogenetic analysis of *Agropyron* chromatin specifying resistance to barley yellow dwarf virus in wheat. Genome 39:336–347

Huth W (1992) Gelbverzwergung der Gerste: Widerstandsfähigkeit der Wintergerste gegenüber der Virose. Bericht Arbeitstagung Saatzuchtleiter, Gumpenstein 43:53–60

Huth W (1995) Möglichkeiten und Grenzen der Züchtung von Getreidesorten mit Resistenz gegenüber den Gelbverzwergungsviren – aus virologischer Sicht. Bericht Arbeitstagung Saatzuchtleiter, Gumpenstein 46:31–42

Jin H, Domier LL, Kolb FL, Brown CM (1998) Identification of quantitative loci for tolerance to barley yellow dwarf virus in oat. Phytopathology 88:410–415

Lander ES, Green P, Abrahamson J, Barlow A, Daly MJ, Lincoln SE, Newburg L (1987) MAPMAKER: an interactive computer package for constructing primary genetic linkage maps of experimental and natural populations. Genomics 1:174–181

Larkin PJ, Banks PM, Lagudah ES, Appels R, Chen X, Xin Z, Ohm HW, McIntosh RA (1995) Disomic *Thinopyrum intermedium* addition lines in wheat with barley yellow dwarf virus resistance and with rust resistances. Genome 38:385–394

Lister RM, Ranieri R (1995) Distribution and economic importance of barley yellow dwarf. In: D'Arcy CJ, Burnett PA (eds) Barley yellow dwarf. Forty years of progress. APS Press, St. Paul, Minnesota, pp 29–53

Liu ZW, Biyashev RM, Saghai Maroof MA (1996) Development of simple sequence repeat DNA markers and their integration into a barley linkage map. Theor Appl Genet 93:869–876

Marcon A, Käppler SM, Jensen SG, Senior L, Stuber C (1999) Loci controlling resistance to high plains virus and wheat streak mosaic virus in a B73 x Mo17 population of maize. Crop Sci 39:1171–1177

Mayo MA (1999) Development in plant virus taxonomy since the publication of the 6th ITCV report. Arch Virol 144:1659–1666

Melchinger AE, Utz HF, Schön CC (1998) Quantitative trait locus (QTL) mapping using different testers and independent population samples in maize reveals low power of QTL detection and large bias in estimates of QTL effects. Genetics 149: 383–403

Moharramipour S, Tsumuki H, Sato K, Yoshida H (1997) Mapping resistance to cereal aphids in barley. Theor Appl Genet 94:592–596

Ordon F, Friedt W (1993) Mode of inheritance and genetic diversity of BaMMV resistance of exotic barley germplasms carrying genes different from '*ym4*.' Theor Appl Genet 86:229–233

Ordon F, Bauer E, Friedt W, Graner A (1995) Marker-based selection for the *ym4* BaMMV-resistance gene in barley using RAPDs. Agronomie 15:481–485

Ordon F, Wenzel G, Friedt W (1998) II. Recombination molecular markers for resistance genes in major grain crops. Prog Bot 59:49–79

Ordon F, Schiemann A, Pellio B, Dauck V, Bauer E, Streng S, Friedt W, Graner A (1999) Application of molecular markers in breeding for resistance to the barley yellow mosaic complex. J Plant Dis Protection 106:256–264

Oswald JW, Houston BR (1951) A new virus disease for cereals, transmissible by aphids. Plant Dis Reporter 35:471–475

Paltridge NG, Collins NC, Bendahamane A, Symons RH (1998) Development of YLM, a codominant PCR marker closely linked to the *Yd2* gene for resistance to barley yellow dwarf disease. Theor Appl Genet 96:1170–1177

Parry AL, Habgood RM (1986) Field assessment of the effectiveness of a barley yellow dwarf resistance gene following its transference from spring to winter barley. Ann Appl Biol 108:395–401

Pecchioni N, Facciolo P, Toubia-Rahme H, Valè G, Terzi V (1996) Quantitative resistance to barley leaf stripe (*Pyrenophora graminea*) is dominated by one major locus. Theor Appl Genet 93:97–101

Pernet A, Hoisington D, Franco J, Isnard M, Jewell D, Jiang C, Marchand JL, Reynaud B, Glazmann JC, de Leon DG (1999a) Genetic mapping of maize streak virus resistance from the Mascarene source. I. Resistance in line D211 and stability against different virus clones. Theor Appl Genet 99:524–539

Pernet A, Hoisington D, Dintinger J, Jewell D, Jiang C, Khairallah M, Letourmy P, Marchand JL, Glazmann JC, de Leon DG (1999b) Genetic mapping of maize streak virus resistance from the Mascarene source. II. Resistance in line CIRADD390 and stability across germplasm. Theor Appl Genet 99:540–443

Plumb RT, Johnstone GR (1995) Cultural, chemical and biological methods for the control of barley yellow dwarf. In: D'Arcy CJ, Burnett PA (eds) Barley yellow dwarf. 40 years of progress. APS Press, St. Paul, Minnesota, pp 107–127

Powell W, Baird E, Booth A, Lawrence M, MacAulay M, Bonar N, Young G, Thomas WTB, McNicol JW, Waugh R (1996) Single locus and multi-locus molecular assays for barley breeding research. In: Scoles G, Rossnagel B (eds) Proc Vth Int Oat Conf and VIIth Int Barley Genet Symp. Invited Papers, Saskatoon, Canada, University Extension Press, Saskatoon, Sakatchewan, pp 174–181

Pringle CR (1998) Virus taxonomy – San Diego 1998. Arch Virol 143/7:1449–1459

Qi X, Niks RE, Stam P, Lindhout P (1998) Identification of QTLs for partial resistance to leaf rust (*Puccinia hordei*) in barley. Theor Appl Genet 96:1205–1215

Qualset CO (1975) Sampling germplasm in a centre of diversity: an example of disaese resistance in Ethopian barley. In: Frankle OH, Hawkes J (eds) Crop genetic resources of today and tomorrow. Cambridge University Press, Cambridge, UK, pp 81–96

Qualset CO (1984) Evaluation and breeding methods for barley yellow dwarf resistance. In: Burnett PA (ed) Barley yellow dwarf. Proceedings of the Workshop, Mexico D.F., Mexico, pp 93–99

Qualset CO (1992) Developing host plant resistance to barley yellow dwarf virus: an effective control strategy. In: Comeau A, Makkouk KM (edse) Barley yellow dwarf in West Asia and North Africa, pp 115–130

Ranieri R, Lister RM, Burnett PA (1993) Relationship between barley yellow dwarf virus titer and symptom expression in barley. Crop Sci 33:968–973

Rasmusson DW, Schaller CW (1959) The inheritance of resistance in barley to the yellow dwarf virus. Agron J 51:661–664

Rochow WF (1970) Barley yellow dwarf virus: phenotypic mixing and vector specificity. Science 167:875–878

Russell JR, Fuller JD, Macaulay M, Hatz BG, Jahoor A, Powell W, Waugh R (1997) Direct comparison of levels of genetic variation among barley accessions detected by RFLPs, AFLPs, SSRs and RAPDs. Theor Appl Genet 95:714–722

Schäfer-Pregl R, Borchardt DC, Barzen E, Glass C, Mechelke W, Seitzer JF, Salamini F (1999) Localization of QTLs for tolerance to *Cercospora beticola* on sugar beet linkage groups. Theor Appl Genet 99:829–836

Schaller CW (1984) The genetics of barley yellow dwarf virus in barley. In: Burnett PA (ed) Barley yellow dwarf. Proceedings of the Workshop, CIMMYT, Mexico, pp 93–99

Schaller CW, Chim CI (1968) Registration of Atlas 68 barley. Crop Sci 9:521

Scheurer KS (2001) Genetische Analyse der Vererbung von Toleranz der Gerste (*Hordeum vulgare* L.) gegenüber Barley Yellow Dwarf Virus (BYDV). PhD thesis University Giessen, Verlag Shaker, Aachen, Germany

Scheurer KS, Huth W, Habekuss A, Friedt W, Ordon F (1998) Züchtung auf Toleranz der Gerste gegen Barley Yellow Dwarf Virus. Vortr Pflanzenzüchtg 42:51–53

Scheurer KS, Huth W, Friedt W, Ordon F (2000) First results on BYDV tolerance in barley estimated in pot experiments. J Plant Dis Protection 107:427–432

Schiemann A, Dauck V, Friedt W, Streng S, Graner A, Ordon F (1999) Establishment of a fluorescence-based AFLP technique and rapid marker detection for the resistance locus *rym5*. Barley Genet Newslett 29, http://wheat.pw.usda.gov/ggpages/bgn/29/a29–01.html

Singh RP, Burnett PA, Albarran M, Rajaram S (1993) BDV1 – a gene for tolerance to barley yellow dwarf virus in bread wheats. Crop Sci 33:231–234

Skaria M, Lister RM, Foster JE, Shaner G (1985) Virus content as an index of symptomatic resistance to barley yellow dwarf virus in cereals. Phytopathology 75:212–216

Steffenson BJ, Hayes PM, Kleinhofs A (1996) Genetics of seedling and adult plant resistance to net blotch (*Pyrenophora teres* f. *teres*) and spot blotch (*Cochliobolus sativus*) in barley. Theor Appl Genet 92:552–558

Suneson CA (1955) Breeding for resistance to barley yellow dwarf virus in barley. Agron J 47:283

Toojinda T, Baird E, Booth A, Broers L, Hayes P, Powell W, Thomas W, Vivar H, Young G (1998) Introgression of quantitative trait loci (QTLs) determining stripe rust resistance in barley: an example of marker-assisted line development. Theor Appl Genet 96:123–131

Toojinda T, Broers LH, Chen XM, Hayes PM, Kleinhofs A, Korte J, Kudrna D, Leung H, Line RF, Powell W, Ramsay L, Vivar H, Waugh R (2000) Mapping quantitative and qualitative resistance genes in a doubled haploid population of barley (*Hordeum vulgare*). Theor Appl Genet 101:580–589

Utz HF, Melchinger AE (1996) PLABQTL: a program for composite interval mapping of QTLs. J Agric Genomics vol. 2, http://www.ncgr.org/ag/jag/

Utz HF, Schön CC, Melchinger AE (1998) Bewertung der Aussichten markergestützter Selektion bei quantitativen Eigenschaften. Vortr Pflanzenzücht 43:38–48

Vos P, Hogers R, Bleeker M, Reijans M, Van De Le T, Hornes M, Frijiters A, Pot J, Peleman J, Kuiper M, Zabeau M (1995) AFLP: a new technique for DNA fingerprinting. Nucleic Acids Res 23:4407–4414

Welz HG, Schechert A, Pernet A, Pixley KV, Geiger HH (1998) A gene for resistance to the maize streak virus in the African CIMMYT maize inbred line CML202. Mol Breed 4:147–154

Xia XC, Melchinger AE, Kuntze L, Lübberstedt T (1999) Quantitative trait loci mapping of resistance to sugar cane mosaic virus in maize. Phytopathology 89:660–667