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Molecular mapping of genomic regions underlying barley yellow dwarf tolerance in cultivated oat (Avena sativa L.)

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Abstract Barley yellow dwarf (BYD) is one of the most important viral diseases in small grains, including oat (*Avena sativa* L.). Breeding for BYD tolerance is an effective and efficient means to control the disease. Characterization of major sources of tolerance, and identification of marker and the trait associations, will directly benefit breeding for BYD tolerance. Genomic regions underlying BYD tolerance were mapped and characterized in an oat population consisting of 152 recombinant inbred lines from the cross of 'Ogle' (tolerant)/MAM17-5 (sensitive). Tolerance was evaluated in replicated field trials across 2 years under artificial inoculation with viruliferous aphids harboring BYD virus isolate PAV-IL. Composite interval mapping was used for quantitative trait loci (QTLs) analysis with a framework map consisting of 272 molecular markers. Four QTLs, *BYDq1, BYDq2, BYDq3* and *BYDq4*, for BYD tolerance were identified on linkage groups OM1, 5, 7 and 24, respectively. All but *BYDq2* were consistently detected across both years. Significant epistasis was found between some QTLs. The final model including the epistatic effect explained 50.3 to 58.2% of the total phenotypic variation for BYD tolerance. Some QTLs for BYD tolerance were closely linked to QTLs for plant height and days to heading. Potential problems with QTL mapping for BYD tolerance have been discussed. The identified association of markers and tolerance should be useful to pyramid favorable alleles for BYD tolerance into individual oat lines.

Keywords Quantitative trait loci · Molecular markers · Barley yellow dwarf · Oat

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Introduction

Barley yellow dwarf (BYD) was first coined by Oswald and Houston (1953) when they discovered a virus disease in barley. The disease is caused by a group of related luteoviruses collectively referred to as the barley yellow dwarf viruses. These viruses are obligately transmitted by aphids and infect all of the major cereal crops including oat (*Avena sativa* L.), as well as wild grasses (D'Arcy 1995). Losses in grain yield from BYD are related to inhibition of root formation, stunted growth, leaf discoloration and blasting abortion of florets (Comeau 1987; Kolb et al. 1991b). An effective and efficient strategy for controlling BYD is the development of resistant or tolerant cultivars (Burnett 1995).

No oat cultivar immune to BYD viruses has been found; however, a number of cultivars and germplasm lines with various levels of tolerance have been reported (Brown and Jedlinski 1978; Kolb et al. 1991a). No single major resistance or tolerance gene has been identified in classical inheritance studies (Burnett 1995). Tolerance to BYD has been shown to be conditioned by two to four, mostly additive, genes and inherited quantitatively (Brown and Poehlman 1962; Landry et al. 1984; Mckenzie et al. 1985). Improvement of quantitative tolerance to BYD should focus on accumulation of favorable alleles for tolerance into oat lines through recurrent selection (Baltenberger et al. 1988).

Classical studies of quantitative resistance, in general, could estimate the number of relevant genetic loci, the type of gene actions and heritability (Geiger and Heun 1989); however, the location of the genetic loci in the genome and the effect of individual loci are difficult to discern in these studies. With quantitative trait locus

(QTL) mapping, which is based on molecular marker technology, those issues could be addressed. QTL mapping has been used to study a variety of traits in a number of crops (Marcon et al. 1999; Pernet et al. 1999; Scheurer et al. 2001; Zhu and Kaeppler 2003b) including BYD tolerance in oat (Jin et al. 1998; Barbosa-Neto et al. 2000). Ogle, a cultivar with good tolerance to BYD (Brown and Jedlinski 1983), was widely used as both a breeding parent and a check for BYD tolerance (Landry et al. 1984; Baltenberger et al. 1988). Twenty one genomic regions were identified to be associated with BYD tolerance in a population of 84 recombinant inbred lines (RILs) from the cross of Kanota/Ogle (Barbosa-Neto et al. 2000). Ogle was significantly more tolerant to BYD virus isolate PAV-IL than Kanota; however, no PAV-IL tolerance QTL from Ogle was identified. Isolate PAV-129 was known to overcome the tolerance of Ogle; however, four QTLs for tolerance to PAV-129 were detected from Ogle in the study. Therefore, it is necessary to further investigate the QTL for BYD tolerance in Ogle by screening other populations of a larger size and with Ogle as one of the parents.

Conventional selection for BYD tolerance is complicated since it is necessary to inoculate plants by infesting them with aphids harboring BYD viruses, and it is difficult to accurately assess the tolerant plants. This is especially challenging for recurrent selection of BYD tolerance, in which a large amount of labor is required to inter-mate the selected plants. Genetic gain for BYD tolerance has largely depended on the effectiveness of selection. Molecular markers are insensitive to environment and can be used in plant breeding to identify desirable recombinants among progeny of a cross (Tanksley et al. 1989). Therefore, marker-assisted selection may provide an attractive approach for indirect selection of BYD tolerance to speed the accumulation of favorable alleles for tolerance into individual oat lines.

Information regarding the number, location and effect of genomic regions associated with BYD tolerance, and epistasis between these regions, would facilitate breeding for BYD tolerance. Mapping QTLs for BYD tolerance should be useful to gain a better understanding of the organization of genes for BYD tolerance in the hexaploid oat genome. This study was conducted, therefore, to identify the number, position and effect of QTL and QTL interactions that underlie BYD tolerance. Linkage relationships were also determined between QTL associated with BYD tolerance and QTL controlling plant growth reported in a previous study (Zhu and Kaeppler 2003b).

Materials and methods

Plant materials

A mapping population of 152 $F_{5:6}$ recombinant inbred lines (RIL) used in this experiment was derived from a cross between two hexaploid cultivated oat (*A. sativa* L.) genotypes, 'Ogle' and MAM17-5, with different responses to the BYD viruses (Table 1).

Table 1 Mean scores of barley yellow dwarf for two parents, Ogle and MAM17-5, and their progeny population of recombinant inbred lines (RILs) in 1999 and 2001

Year	Parents		RILs	
	Ogle	MAM17-5	Mean	Range
1999	3.6 ± 1.2	6.8 ± 0.6	5.3 ± 1.0	2.0–7.7
2001	3.9 ± 0.5	7.2 ± 0.3	5.8 ± 0.9	3.0–8.0

Ogle has good BYD tolerance and was developed in the spring oat breeding program at the University of Illinois (Brown and Jedlinski 1983). MAM17-5 is a BYD sensitive oat line and was selected in the spring oat breeding program at the University of Wisconsin-Madison (Moustafa et al. 1992). The mapping population was developed using the single-seed descent method.

Evaluations for BYD tolerance

Tolerance of RILs to BYD virus infection was evaluated at the University of Illinois Crop Sciences Research and Education Center, Urbana, Il. in 1999 and 2001. The 152 RILs and the two parents, Ogle and MAM17-5 (controls), were planted into a 3-replication hill plot trial in the field for the two growing seasons of 1999 and 2001. Replicated trials with ten plants per hill plot were planted each season in a randomized complete block design.

The hills were inoculated at Feekes growth stage 1 or 2 (Large 1954) by infesting them with viruliferous oat-bird cherry aphids (*Rhopalosiphum padi* L.) as the vector harboring an Illinois isolate of the PAV strain (PAV-IL) of BYD virus (Hewings et al. 1992). Approximately 20–30 aphids were placed onto each hill by dispensing an equal volume of aphid and corn meal mixture so that essentially all of the plants in a hill were infected. The plants at Feekes growth stage 10.6 were rated for BYD tolerance using a 0-to-9 scale. The 0-to-9 BYD ratings were assigned by visually estimating leaf discoloration, stunting and blasting of the florets in the panicles (Qualset 1984). On this scale, hills rated as 0 exhibit no leaf discoloration, no stunting and no blasting of the florets. Hills rated as 9 fail to head and are stunted so severely that the plants are almost dead.

In order to evaluate the growth of the RILs in the absence of BYD virus infection, the same population was planted at the West Madison Agricultural Experiment Station, Madison, Wis. in 1999 and 2000 in a previous study (Zhu and Kaeppler 2003b). The plants in the study were not infested with viruliferous aphids, and BYD symptoms were negligible. Days to heading and plant height were recorded in these plots.

Marker analysis and map construction

Sources of restriction fragment length polymorphism (RFLP) clones, microsatellite or simple sequence repeat (SSR) primers, and amplified fragment length polymorphism (AFLP) primers were reported elsewhere (Zhu and Kaeppler 2003a). AFLP analysis was performed according to the protocol provided by the manufacturer (Gibco-BRL Life Technology, Inc., Gaithersburg, MD.) with minor modifications (Zhu and Kaeppler 2003a). SSR analysis was performed according to Chin et al. (1996), except that the separation and detection of the amplified products were done on polyacrylamide sequencing gels. RFLP analysis followed a standard protocol (Zhu and Kaeppler 2003a). For identification of QTLs, a framework linkage map (OM map) of 34 linkage groups with 272 molecular markers was developed using the most informative markers (Zhu 2002), and the OM map was used in this study. The genetic distance in the map, however, was in Haldane's centi-Morgan units preferred by PlabQTL (Utz and Melchinger 1996).

Data analysis

Analysis of variance for trait data was performed by using the General Linear Model Procedure (Proc GLM) of SAS (SAS 1990). Broad-sense heritability was calculated as $h^2 = \sigma_g^2/(\sigma_g^2 + \sigma_{gy}^2 + \sigma_e^2)$, where $\sigma_g^2 = (MS_{genotype} - MS_{genotype\text{-}by\text{-}year})/(Reps \times Years)$, $\sigma_{gy}^2 = (MS_{genotype\text{-}by\text{-}year} - MS_{error})/Reps$ and $\sigma_e^2 = MS_{error}$. Mean values across three replications for each RIL were used to determine phenotypic correlations in the RIL population and to conduct QTL analysis. Pearson correlation coefficients were calculated by using the Correlation Procedure (Proc Corr) of SAS. The significance of genotype by year interaction was examined.

Computer program PlabQTL (Utz and Melchinger 1996) was used in QTL analysis. In order to determine the critical log likelihood of odds (LOD) threshold, permutation tests were performed with 1,000 random reshuffles of observation recommended by Churchill and Doerge (1994). All regions with LOD > 3.5, corresponding to an experiment-wise error rate of 0.05 (a comparison-wise error of approximately 0.00016) from the QTL analysis, were considered significant and included in the final model. Composite interval mapping (CIM) (Zeng 1994) with the cov SELECT option was used for QTL detection. CIM is an extension of interval mapping with some selected markers also fitted in the model as cofactors to control the genetic variation of other possibly linked or unlinked QTLs. An *F*-to-enter value of 7.0 was used for the step-wise regression to pre-select cofactors. The cov SELECT option uses all markers in the pre-selection as cofactors. The QTL position, given as centi-Morgans from the top of a linkage group, was determined when the LOD score reached its maximum. A support interval with a LOD fall-off of 1.0 was given for each QTL. QTLs with an overlapping support interval are assumed to be the same QTL for the same trait. The additive effect of a QTL was calculated by PlabQTL as (mean of the homozygous MAM17-5 class – mean of the homozygous Ogle class)/2. The additive by additive epistasis was estimated using the Model AA command. The phenotypic variance explained by the QTL model was estimated by the adjusted coefficient of determination (R^2_{adj}), which accounts for the number of predictors in the final model. The phenotypic variance explained by an individual QTL or an individual QTL by QTL interaction (predictor) was calculated as R^2_i (Exp.%) = Partial $R^2_i \times R^2_{adj}$/(sum of Partial R^2_i with i = 1 to n), with n = total number of predictors in the final model. The partial R^2_i – the partial coefficient of determination was estimated for the ith predictor.

Results

Assessment of BYD tolerance

Analysis of variance indicated highly significant differences between the two parents and among the 152 RILs for BYD tolerance. MAM17-5 consistently displayed higher scores than Ogle in both years (Table 1), corroborating that the two parents differed in genes controlling the trait. BYD scores for the 152 RILs showed an approximately normal distribution (Fig. 1), which agrees with previous reports that BYD tolerance is a quantitative trait (Brown and Poehlman 1962; Landry et al. 1984; Mckenzie et al. 1985). Transgressive segregation was observed, indicating that the two parents carried complementary alleles for BYD tolerance (Table 1 and Fig. 1).

Significant genotype by environment interaction was detected from the analysis of variance, suggesting that QTL analysis for BYD tolerance should be conducted separately for each environment; however, a highly significant correlation was found between the 2 years (Table 2). Therefore, QTL analysis for BYD tolerance in this study was conducted for each year and for the average of 2 years. Both days to heading and plant height had a low, but significant, negative correlation with BYD tolerance (Table 2), suggesting that these traits may have QTLs in linkage or even in common.

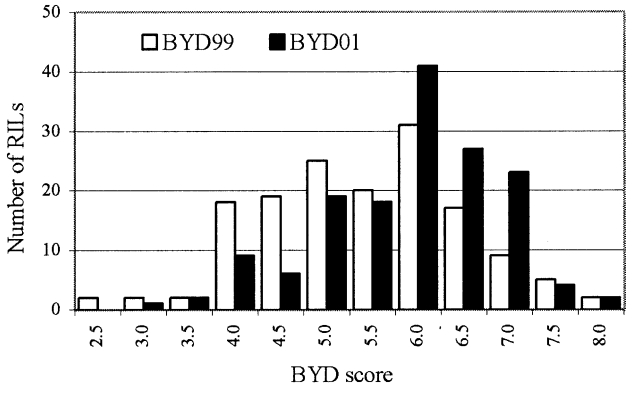

Fig. 1 Distribution of barley yellow dwarf (BYD) scores for the 152 recombinant inbred lines from the cross of 'Ogle'/MAM17-5. BYD severity was rated on replicated field plots in 1999 (BYD99) and in 2001 (BYD01) based on a 0-to-9 scale, in which 0 is highly tolerant and 9 is highly sensitive (Qualset 1984). The values next to the x-axis are the upper limit of each category

Table 2 Correlation between barley yellow dwarf (BYD) ratings in 1999 (BYD99) and 2001 (BYD01), and between BYD ratings and plant growth traits

Item	BYD01	Days to heading	Plant height (cm)
BYD99	0.75***	–0.18*	–0.29**
BYD01		–0.20*	–0.32**

*, **, and ***: significant at the 0.05, 0.01 and 0.001 levels of probability, respectively

The broad-sense heritability of BYD tolerance was estimated using data from the 2 years. The heritability for BYD tolerance was 0.58, which was in agreement with previous estimates (about 0.51) of broad-sense heritability for the *A. sativa* crosses of tolerant by susceptible (Brown and Poehlman 1962).

Genomic regions underlying BYD tolerance

Composite interval mapping was used to detect genomic regions that underlie BYD tolerance in a more precise manner. Four QTLs for BYD tolerance were identified in the mapping population (Table 3; Fig. 2). Three of the QTLs, *BYDq1, BYDq3* and *BYDq4*, located on linkage groups OM1, OM7 and OM24, respectively, were consistently detected in both years. Another QTL, *BYDq2* located on linkage group OM5, was found to be significant only in 2001. From the signs of QTL effects, three QTLs associated with BYD tolerance were contributed by Ogle, and one came from MAM17-5 (*BYDq3*),

Table 3 Summary of quantitative trait loci (QTLs) for tolerance to barley yellow dwarf in 1999 (BYD99), 2001 (BYD01) and an average of the 2 years (BYD). Data collected on 152 recombinant inbred lines derived from Ogle/MAM17-5 inoculated with the PAV-IL isolate of the BYD virus. The QTL analysis was conducted using composite interval mapping with a LOD threshold of 3.5

QTL	Linkage group	Support interval (cM)[a]	BYD99			BYD01			BYD		
			LOD	Exp. (%)[b]	Effect[c]	LOD	Exp. (%)	Effect	LOD	Exp. (%)	Effect
BYDq1 (1)	OM1	7–18	14.2	35.9	0.92	17.4	30.7	0.89	16.9	31.7	0.85
BYDq2 (2)	OM5	6–12				3.8	6.0	0.22	3.9	6.0	0.22
BYDq3 (3)	OM7	56–61	4.5	7.2	–0.27	4.3	8.7	–0.27	4.2	7.6	–0.25
BYDq4 (4)	OM24	0–5	3.7	4.0	0.23	3.9	7.7	0.24	4.0	6.6	0.24
(1) × (3)				3.2	–0.15						
(2) × (3)							5.1	0.20			
Total (%)[d]				50.3			58.2			51.9	

[a] Start and ending points of the interval from the top of the linkage group
[b] Explained phenotypic variance obtained from the simultaneous fit of all putative QTLs and significant QTL × QTL epistasis
[c] Additive effect for QTLs. Positive values indicate that Ogle provides tolerance alleles, and negative values indicate that MAM17-5 carries tolerance alleles for BYD
[d] The adjusted R^2 for the final model of simultaneous fit

which was in agreement with the finding of transgressive segregants in the RILs.

Digenic epistasis between *BYDq1* and *BYDq3* was found to be significant for BYD tolerance in 1999. A significant epistatic effect between *BYDq2* and *BYDq3* was identified in 2001. By including the epistatic effect in the model for simultaneous fit, the final model explained 50.3 to 58.2% of the total phenotypic variation for BYD tolerance (Table 3).

Relationship between BYD tolerance and plant development

Days to heading and plant height for the same population have been investigated in field plots, in which plants were not infested with viruliferous aphids and BYD symptoms were negligible, in a previous study (Zhu and Kaeppler 2003b). Three QTLs, *Hdq1, Hdq2* and *Hdq3*, associated with days to heading, and four QTLs, *Htq1, Htq2, Htq3* and *Htq4*, controlling plant height, were putatively identified in the study. QTLs, *Htq1* and *Htq3*, for plant height and *Hdq3* for days to heading were located at almost the same position as one of two QTLs, *BYDq2* and *BYDq3*, for BYD tolerance, and *Htq2* for plant height was loosely linked to *BYDq3* for BYD tolerance (Fig. 3).

On linkage group OM5, the closest marker to the peak of *BYDq2* was e8m6_16 (Fig. 2), and UMN464 was the closest marker to the peak of *Htq1* (Zhu 2002). On linkage group OM7, the closest marker to the peak of *BYDq3* was BCD1797 (Fig. 2), and BCD808b was the closest marker to the peaks of *Htq3* and *Hdq3* (Zhu 2002). Moreover, the peak of each QTL was located outside of the window flanked by the relevant closest markers (Fig. 3). The recombinants between *BYDq2* and *Htq1*, and between *BYDq3* and each of *Htq3* and *Hdq3*, were observed by examining the crossovers between the relevant closest markers. Therefore, the relationship between *BYDq2* and *Htq1*, and between *BYDq3* and each of *Htq3* and *Hdq3*, was in linkage instead of pleiotropic effects of single genes. The linkage relationship of QTL

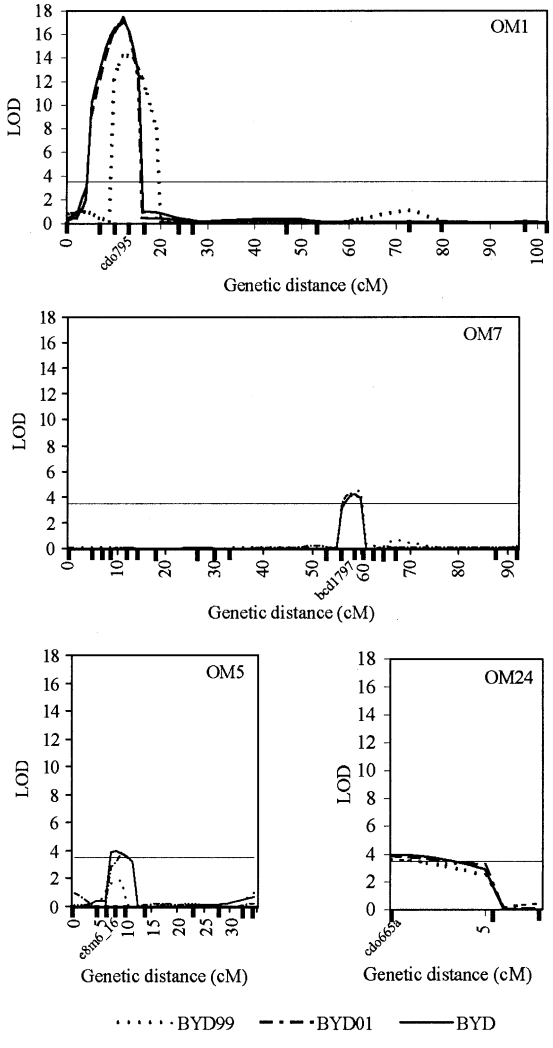

Fig. 2 LOD score profiles of QTLs for BYD tolerance in 1999 (BYD99), 2001 (BYD01) and average (BYD) on linkage groups OM1, OM5, OM7 and OM24 using the composite interval mapping method. Linkage groups correspond to the groups in Fig. 3, and are oriented with the top to the left. *Thick tick marks* on the x-axis indicate the position of molecular markers. The closest marker to the significant QTL is shown below the x-axis. A LOD threshold of 3.5 was used to declare a QTL

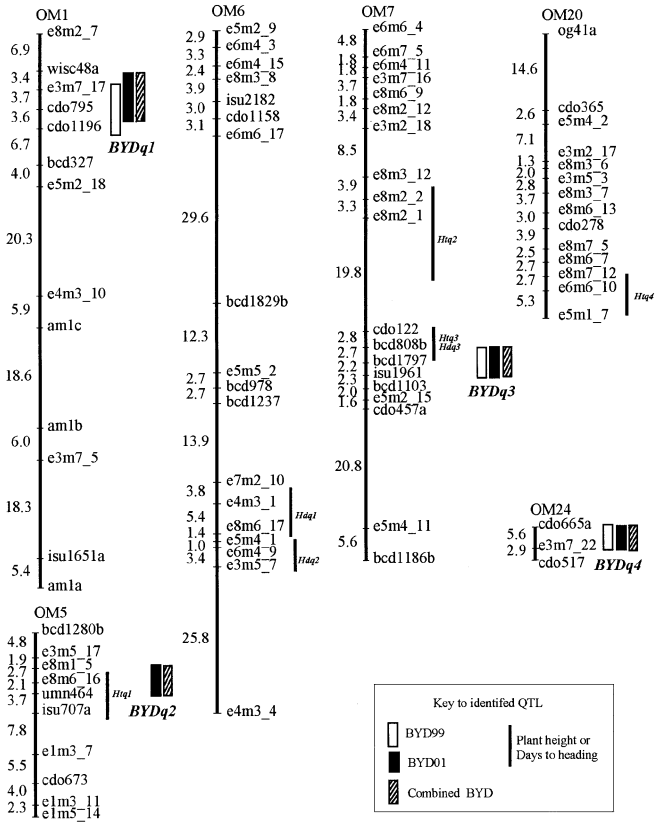

Fig. 3 Linkage groups from the framework linkage map (Zhu 2002) developed on the cross of 'Ogle'/MAM17-5 (OM), showing significant quantitative trait loci (QTL) for BYD tolerance, days to heading and plant height. Map distances are given in Haldane's centi-Morgans. QTLs for BYD tolerance are indicated to the right of linkage groups by *open boxes* (1999), *solid bars* (2001) and *crosshatch bars* (2 years combined). QTL for days to heading (*Hdq*) and plant height (*Htq*) are indicated to the right of linkage groups by *lines*. Molecular markers beginning with letter 'e' are AFLP-type, while 'am' or 'wisc' are SSR-type, and with others are RFLP-type markers

resulted in the significant negative correlation between BYD ratings and days to heading and plant height (Table 2).

Discussion

No attempt was made to isolate resistance and tolerance, so BYD tolerance was referred to in this study. In a previous QTL analysis for tolerance to BYD isolate PAV-IL, no QTL for tolerance from Ogle was identified using 84 RILs from the cross of Kanota/Ogle (Barbosa-Neto et al. 2000). In our study, three genomic regions that underlie BYD tolerance contributed by Ogle were detected through artificially inoculating with the same isolate, in addition to one conferred by the other parent MAM17-5. The identified four genomic regions explained approximately 50–58% of the total phenotypic variation for BYD tolerance. Heritability estimate (0.58) suggested that about 40% of the variation was caused by non-genetic factors. Likely QTLs with minor effects were not detected in our experiments, because of problems such as incomplete map coverage and a strict LOD threshold; however, little residual variation was left for undetected minor QTLs. In general, the number of QTLs for BYD tolerance found in this study was in agreement with the results of classical studies (Landry et al. 1984; Mckenzie et al. 1985) and another study of QTL mapping for BYD tolerance (Jin et al. 1998) in oat. The results of this study, coupled with previous reports, support the conclusion that BYD tolerance is oligogenic.

QTLs identified in our study could be compared with previously detected QTLs using the Kanota/Ogle (KO) map (O' Donoughue et al. 1995) as a bridge. A major QTL *BYDq1*, closely linked to a RFLP marker CDO795 in this study, was located on linkage group OM1, which was homologous to linkage group 22 (KO22) of the KO map (Zhu and Kaeppler 2003a). It was interesting to find that a major QTL associated with BYD tolerance, linked to the same marker CDO795, was also mapped to KO22; however, the tolerance allele was contributed by Kanota and the isolate inoculated was PAV-NY (Barbosa-Neto et al. 2000). Another QTL, *BYDq4*, in this study was located on OM24, which was putatively homoeologous to KO11, 14 and 16. There was also a major QTL controlling tolerance to BYD isolate PAV-IL mapped to KO14; however, Kanota conferred the tolerance allele (Barbosa-Neto et al. 2000). Three major QTLs, A, C and E, conditioning tolerance to BYD isolate PAV-IL, putatively assigned onto linkage groups KO2, 36 and 8, respectively, were identified in the mapping population derived from the oat cross of Clintland 64 and IL86-5698 (Jin et al. 1998). Ogle and IL86-5698 were both developed in the spring oat breeding program in Illinois (Brown and Jedlinski 1983; Kolb et al. 1991a); however, based on comparative mapping, none of the three QTLs, A, C and E, from IL86-5698 was the same as any of the three QTLs, *BYDq1, BYDq2* and *BYDq4*, from Ogle. This result suggests that it is promising to combine different favorable alleles from Ogle and IL86-5698 into single oat lines to improve BYD tolerance.

It should not be surprising to find QTLs, *BYDq2* and *BYDq3*, displaying a linkage relationship with QTLs for plant height and days to heading. The linkage could be true because in parental development, selection most likely favored segregates in which QTLs for BYD tolerance and QTLs for tall and late plants occurred together. An alternative explanation is that stunted growth, and delay in or lack of heading in infected plants are factors considered in rating BYD symptoms. A potential problem with the association should be noted. When BYD symptoms are evaluated on a visual scale in the field, the variability in plant height and maturity among lines may interfere with the assessment of BYD symptoms. Some inherent short and early lines (leaves turning yellow earlier) could be mistakenly assessed as sensitive to BYD, while naturally tall and late lines could be mistakenly measured as BYD tolerant. Simply discarding the QTL for BYD tolerance linked to plant height or days to heading (Barbosa-Neto et al. 2000) is not an appropriate means to correct the potential problem.

Another approach might be to grow healthy control lines side by side with infected plants to aid in reducing variability in BYD ratings associated with plant height and maturity.

Inheritance analysis of virus disease resistance is complicated, since usually three kinds of organisms, host, vector and virus, and environmental factors interact with one another to result in symptom expression. Depending on viruses, vectors could be fungi (Walker et al. 1998), mites (Marcon et al. 1999), leafhoppers (Welz et al. 1998; Pernet et al. 1999) and aphids (Jin et al. 1998; Barbosa-Neto et al. 2000; Scheurer et al. 2001). No efforts were made to isolate QTLs for vector resistance and QTLs for virus resistance in this or previous QTL mapping studies. Therefore, a QTL for BYD tolerance identified in this study may, in fact, be a QTL for aphid resistance. In some studies, viruliferous vectors were removed by spraying insecticides a few days after being infested to minimize the feeding effect caused by the genetic difference in vector resistance among lines (Marcon et al. 1999; Scheurer et al. 2001); however, this action could not solve the vector inability to introduce viruses caused by vector resistance.

This study has identified four genomic regions underlying BYD tolerance in cultivated oat, especially a major QTL *BYDq1*, closely linked to RFLP marker CDO795, explaining more than 30% of the total phenotypic variation for BYD tolerance. The QTLs identified in this study were new compared with the three major QTLs, A, C and E, detected in oat (Jin et al. 1998). The results obtained in this study are of practical significance to oat breeding for disease resistance. Incorporation of marker-assisted selection into breeding programs will speed pyramiding of these favorable QTL alleles for BYD tolerance from different sources into single oat lines and enhance genetic gains.

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