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Genome-wide identification of QTL conferring high-temperature adult-plant (HTAP) resistance to stripe rust (*Puccinia striiformis* f. sp. *tritici*) in wheat

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Abstract High-temperature adult-plant (HTAP) resistance to stripe rust (caused by *Puccinia striiformis* f. sp. *tritici*) is a durable type of resistance in wheat (*Triticum aestivum* L.). This study identified quantitative trait loci (QTL) conferring HTAP resistance to stripe rust in a population consisting of 169 $F_{8:10}$ recombinant inbred lines (RILs) derived from a cross between a susceptible cultivar Rio Blanco and a resistant germplasm IDO444. HTAP resistance was evaluated for both disease severity and infection type under natural infection over two years at two locations. The genetic linkage maps had an average density of 6.7 cM per marker across the genome and were constructed using 484 markers including 96 wheat microsatellite (SSR), 632 Diversity Arrays Technology (DArT) polymorphisms, two sequence-tagged-site (STS) from semi-dwarf genes *Rht*1 and *Rht*2, and two markers for low molecular-weight glutenin gene subunits. QTL analysis detected a total of eight QTL significantly associated with HTAP resistance to stripe rust with two on chromosome 2B,

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two on 3B and one on each of 1A, 4A, 4B and 5B. QTL on chromosomes 2B and 4A were the major loci derived from IDO444 and explained up to 47 and 42% of the phenotypic variation for disease severity and infection type, respectively. The remaining five QTL accounted for 7–10% of the trait variation. Of these minor QTL, the resistant alleles at the two QTL *QYrrb.ui-3B.1* and *QYrrb.ui-4B* derived from Rio Blanco and reduced infection type only, while the resistant alleles at the other three QTL, *QYrid.ui-1A*, *QYrid.ui-3B.2* and *QYrid.ui-5B*, all derived from IDO444 and reduced either infection type or disease severity. Markers linked to 2B and 4A QTL should be useful for selection of HTAP resistance to stripe rust.

Keywords HTAP resistance · Yellow rust · QTL mapping · *Triticum aestivum*

Introduction

Stripe rust (yellow rust), caused by *Puccinia striiformis* Westend. f. sp. *tritici* Eriks., is one of the most destructive diseases of wheat (*Triticum aestivum* L.) in the world (Stubbs 1985; Chen et al. 2002; Chen 2005; Milus et al. 2009). The use of host resistance is the most effective, economical and environmentally safe means of control of stripe rust. Two major types of host resistance to stripe rust have been defined: all-stage resistance (AS resistance, also known as overall or seedling resistance) and high-temperature adult-plant (HTAP) resistance (Chen 2005). In contrast to AS resistance, HTAP resistance tends to be race non-specific and durable, and thus has been widely used to develop stripe-rust-resistant cultivars in the Pacific Northwest and other wheat-growing regions in the USA (Johnson 1992; Line 2002; Lin and Chen 2009). However, evaluation of HTAP resistance can be expensive and results are often affected by plant growth stage, temperature and disease pressure (Chen 2005). Some of the difficulties occur because HTAP resistance is expressed at later plant growth stages and becomes effective when daytime temperatures are between 25 and 30°C (Qayoum and Line 1985; Line and Chen 1995). HTAP resistance is generally incomplete and multi-genic (Milus and Line 1986a, b). Molecular markers tightly linked to HTAP resistance offer an alternative to phenotypic selection and are useful for pyramiding both AS and HTAP resistance.

Quantitative trait loci (QTL) controlling HTAP resistance have been identified in different resistant sources. Chromosome arm 2BS is associated with HTAP resistance detected in diverse wheat backgrounds, including Kariega hard red spring wheat (Ramburan et al. 2004), Louise soft white spring wheat (Carter et al. 2009) and Luke soft white winter wheat (Guo et al. 2008). This suggests that resistance alleles carried on 2BS are widely utilized in cultivar development. Additional QTL in other chromosomal regions include 2BL in Luke (Guo et al. 2008), 4A and 7D in Kariega (Ramburan et al. 2004) and 6BL in a soft white winter cultivar Stephens (Santra et al. 2008). Lin and Chen (2009) mapped three QTL in the hard red spring wheat cultivar Express located on chromosome arms 1BL, 3BL and 6AS. In addition, Lu et al. (2009) detected five QTL from four chromosomal regions 2DS, 4BL, 5BL and 7D in the cross of the Italian cultivars Libellula and Strampelli.

Different combinations of HTAP alleles from novel loci may provide a diversity of resistance responses and a more stable pool of resistant cultivars when deployed. The hard red winter wheat germplasm, IDO444 (PI 578278), developed by the University of Idaho, exhibited a high level of stable resistance to stripe rust during several years of observation and was used in the construction of mapping populations. In this study, we used a population of recombinant inbred lines (RILs) derived from Rio Blanco × IDO444 to identify QTL conferring HTAP resistance.

Materials and methods

Plant materials

The mapping population used in this study consisted of 169 $F_{8:10}$ RILs derived from Rio Blanco × IDO444. Rio Blanco (PI 531244) is an early maturity, semi-dwarf hard white winter wheat cultivar developed by AgriPro Biosciences Inc., Kansas, USA (Wu and Carver 1999). It has been widely used as a parent in hard white winter wheat breeding programs (Carver et al. 2003; Haley et al. 2003; Martin et al.

2001). IDO444 has complex resistance to snow mold (*Typhula* spp.) and dwarf bunt caused by *Tilletia controversa* J. G. Kühn, but is generally poor for bread-baking quality (Windes et al. 1995). From tests over eight years in Pullman, Washington State, USA, Rio Blanco was susceptible to stripe rust and IDO444 expressed consistent HTAP resistance (Chen 2009, personal communication). The $F_{8:10}$ RILs were developed through single-plant-descent from the original cross between Rio Blanco and IDO444.

Evaluation of HTAP resistance to stripe rust in field trials

Stripe rust was evaluated in 2003 and 2004 at Mt. Vernon (MtV) and Pullman (PLM) in Washington State. Both locations experienced regular natural epidemics of stripe rust, yet frequently have different races (Lin and Chen 2007; Chen et al. 2010). For disease evaluation, RILs were planted in rows 1 m long and 0.2 m apart, replicated twice in randomized complete block designs, with both parental lines replicated 20 times within each block. The nursery was surrounded by spreader rows of PS 279, a stripe-rust-susceptible line. Applications of fertilizer and weed control followed standard cultivation practices. For disease evaluation, infection types (IT) were evaluated on a 0–9 scale (Line and Qayoum 1992) and disease severities (DS, percentage of infected leaf area) were visually recorded. Disease scoring was conducted at three growth stages: heading to anthesis (MtV), milk (MtV and PLM) and soft dough (PLM) stages.

Marker data generation and linkage map construction

The marker data collected included 96 wheat microsatellite (SSR), 632 Diversity Arrays Technology (DArT) polymorphisms, two sequence-tagged-site (STS) markers from the semi-dwarf genes *Rht1* and *Rht2*, and two markers for low-molecular-weight glutenin gene subunits. The SSRs were produced using Gatersleben Wheat Microsatellites (GWM; Röder et al. 1995, 1998; Ganal and Röder 2007), Wheat Microsatellite Consortium (WMC; Gupta et al. 2002), Beltsville Agriculture Research Center (BARC; Song et al. 2005) and INRA Clermont-Ferrand (CFA and CFD; Sourdille et al. 2004; Guyomarc'h et al. 2002). Total genomic DNA was extracted from seeds ground in a 2000-115 Geno Grinder (SPEX SamplePrep) using the method described by Pallotta et al. (2003) except that the SDS buffer was replaced by CTAB. DNA concentration was measured by a Nanodrop ND-1000 Spectrophotometer and adjusted to 40 ng/µl for marker generation. PCR for SSRs was performed as described by Röder et al. (1998) with minor modifications. Electrophoresis was carried out on 6% polyacrylamide gels (0.4-mm thick) in 0.5 × TBE (90 mM Tris-borate, 2 mM EDTA) at 300 volts for 3 h using an ABS gel electrophoresis system.

DArT marker assays were performed by Triticarte Pty. Ltd (Canberra, Australia; http://www.triticarte.com.au) as previously described (Wenzl et al. 2004; Akbari et al. 2006; Semagn et al. 2006). The polymorphisms of DArT markers were first evaluated on the parents Rio Blanco and IDO444 and then used for genotyping individual RILs. A total of 632 loci were scored as present or absent. Names for loci previously mapped by Triticarte Pty. Ltd include the prefix "wPt" followed by numbers corresponding to a particular clone; loci that were mapped for the first time on the current maps are presented by clone ID number.

Linkage maps were constructed using the computer program MAPMAKER (v2.0) for Macintosh (Lander et al. 1987) with a minimum logarithm of odds (LOD) threshold of 3.0 and the Kosambi mapping function (Kosambi 1944). Linkage groups were first identified using the "two-point/group" command with a minimum LOD = 3.0 and a maximum recombination percentage $\theta = 0.40$, which yielded several very large linkage groups. Each large group was then regrouped using a minimum LOD = 10.0. The FIRST ORDER and RIPPLE (LOD > 3.0) commands were used to determine the most plausible order of markers within linkage groups. The TRY command was used to add markers that did not RIPPLE at LOD > 3.0 to the established framework maps. For QTL analysis, a subset of 317 markers was used that were spaced greater than 2 cM apart to give the most complete genome coverage.

Statistical and QTL analyses

Bartlett's χ^2-test was performed for DS and IT data collected at different growth stages in different environments (PROC GLM, SAS Institute Inc., v 9.1) to test homogeneity of error variances among the data sets. Data sets with homogeneous error variances were combined into groups for subsequent analysis. Pearson correlation coefficients (SPSS 17.3) were used to validate whether the data point distributions in the remaining heterogenous data sets were similar to those of homogeneous ones. Data sets that were both heterogeneous and uncorrelated with others were discarded. The further data analysis was performed independently for each homogeneous group.

Analysis of variances (ANOVA) for DS and IT were performed (PROC GLM, SAS Institute Inc., v 9.1). The heritability (h^2) of resistance was then estimated based on the ANOVA using the formula $h^2 = \sigma_g^2/\sigma_p^2$, where σ_g^2 and σ_p^2 represent genetic and phenotypic variances, respectively. σ_g^2 was calculated from $(\sigma_L^2 - \sigma_E^2)/r$, where σ_L^2 is mean variance of the RILs, σ_E^2 is the error variance and r equals the number of replications. σ_p^2 was obtained through summation of σ_g^2 and σ_E^2 (Lillemo et al. 2008). For QTL analysis, composite interval mapping (CIM) was performed using the computer program Map Manager QTX (Manly et al. 2001) to evaluate marker intervals putatively associated with trait phenotypes. The significance threshold was determined by a permutation test with 1,000 permutated samples (Doerge and Churchill 1996). A genome-wide significance level $P < 0.001$ for type I error with a LOD threshold of 2.84 was used as a criterion to indicate putative QTL positions in the population. Genetic loci with the most significant effect for each QTL were assembled into a multiple regression model to determine the total amount of phenotypic variation explained by all QTL (Nelson 1997).

Results

Linkage map construction

The total genetic distance of the Rio Blanco × IDO444 linkage map was 3,266 cM, with an average density of one marker per 6.7 cM (Electronic Supplementary Materials Table 1 S1 and Fig. 1 S1). The map lengths were 1,151, 1,268 and 847 cM for the A, B and D genomes, respectively (Table 1 S1). Marker distribution among the genomes was not uniform, with chromosomes 1 and 5 of the D genome having only two and three markers, respectively. The D genome coverage averaged 13.9 cM per marker, which is much less than those of the A and B genomes with densities of 6.6 and 5.1 cM per marker, respectively.

HTAP resistance evaluations

Stripe rust infection fully developed in both years at both locations. Based on Bartlett's χ^2 test, disease severity data collected at the two growth stages at MtV in both 2003 and 2004 were homogeneous ($P = 0.10$) and thus were combined. Data collected at the two stages at PLM in 2004 were also combined since their error variances were homogeneous ($P = 0.24$). Data from the two stages at PLM in 2003 were significantly different and thus each was analyzed separately. For infection type, error variances were homogeneous for data from the two stages at MtV in 2003 ($P = 0.57$), from the two stages at PLM in 2003 ($P = 0.32$) and from the two stages at MtV in 2004 ($P = 0.75$), but data from the two stages at PLM in 2004 were heterogeneous relative to all others. All DS and IT data sets were correlated ($r = 0.5$–0.9, $P < 0.0001$) except for IT data from the milk stage at PLM in 2004. The correlated data were therefore combined according to the homogeneity test for error variance and used for further analysis.

In all data sets that were either homogeneous or heterogeneous but correlated, the two parental lines, Rio Blanco and IDO444, had consistent different responses to stripe rust, with smaller IT and DS values for IDO444 than for Rio Blanco across all locations/years (Figs. 1 and 2). The same observations were made in continuous evaluations of the two parents for HTAP resistance during 2005–2009 (data not shown). RILs had varied stripe rust reactions for DS and IT with different distribution patterns among RILs in different data sets (Figs. 1 and 2). Transgressive phenotypes for resistance and susceptibility occurred in each environment at each evaluation stage. Broad-sense heritability values based on all data sets for both DS and IT were large (0.69–0.95, Table 1), except in the soft dough stage from PLM in 2004 ($h^2 = 0.53$ and 0.45 for DS and IT, respectively).

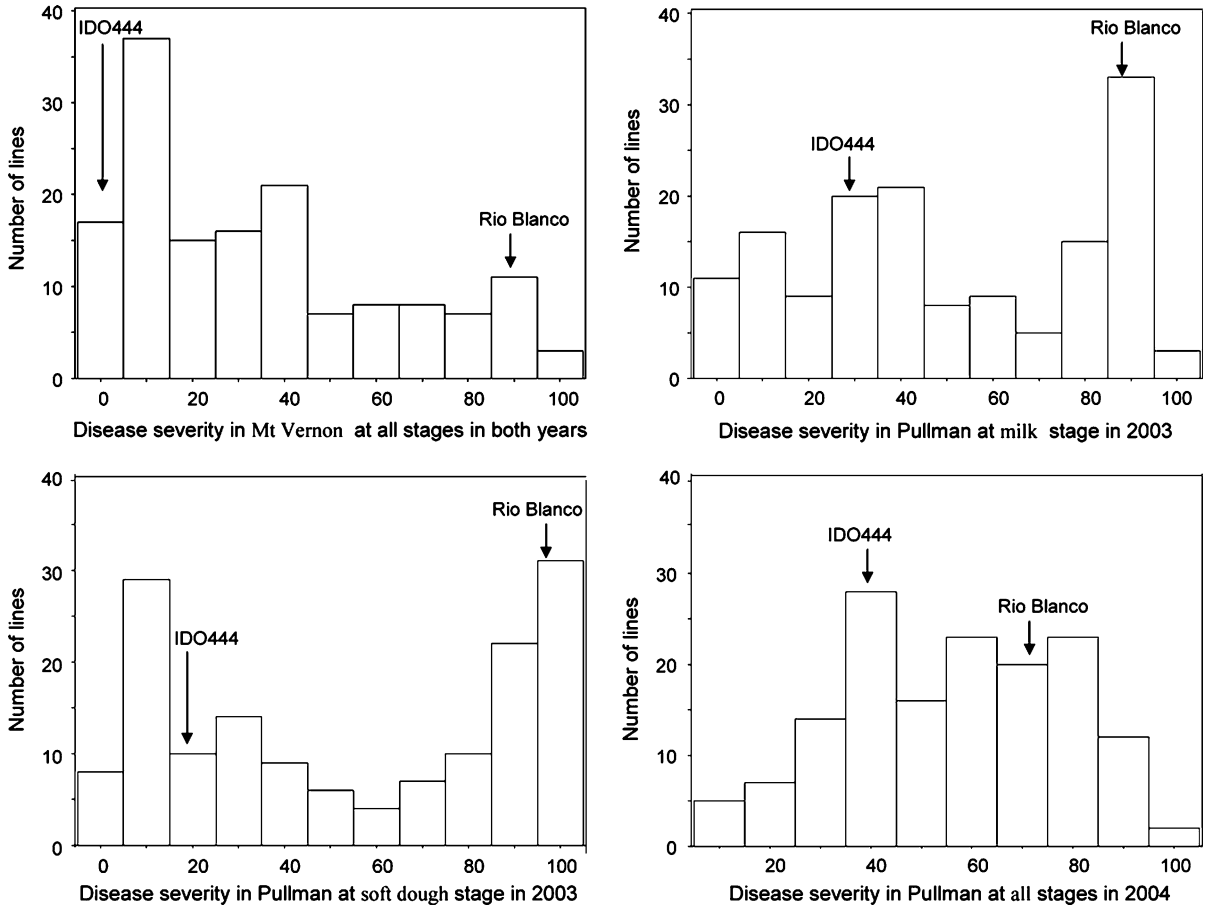

Fig. 1 Frequency distributions of disease severity in RIL population derived from RioBlanco × IDO444 in 2003 and 2004 at Mt Vernon and Pullman

QTL analysis

Eight QTL from six genomic regions were associated with DS and/or IT for HTAP resistance in the RIL population (Table 2). Among them, *QYrid.ui-2B.1* and *QYrid.ui-2B.2* on chromosome 2B and *QYrid.ui-4A* on chromosome 4A explained up to 20–30% of the phenotypic variation for both DS and IT, with resistant alleles derived from IDO444 (Table 2 and Fig. 3). These major QTL were significant in all data sets for both IT and DS from MtV at two growth stages across both years. For data collected at PLM, *QYrid.ui-4A* was significant for both DS and IT, but the two QTL on 2B were significant only for IT and explained a smaller amount of trait variation. In the multiple regression model the three major QTL explained up to 47 and 42% of the total trait variation for DS and IT, respectively.

The remaining five QTL, *QYrid.ui-1A* located on chromosome arm 1AS, *QYrrb.ui-3B.1* on 3BS, *QYrid.ui-3B.2* on 3BL, *QYrrb.ui-4B* on 4BS and *QYrid.ui-5B* on 5BL had relatively small effects on stripe rust response, with each explaining about 7–10% of the trait variation (Table 2). Of these minor QTL, *QYrrb.ui-3B.1* and *QYrrb.ui-4B* were conditioned by Rio Blanco, whereas QTL *QYrid.ui-1A, QYrid.ui-3B.2* and *QYrid.ui-5B* were all derived from IDO444. Four minor QTL, *QYrid.ui-1A*, *QYrrb.ui-3B.1*, *QYrrb.ui-4B* and *QYrid.ui-5B*, were significant only for IT, and one minor QTL, *QYrrb.ui-3B.2*, was significant only for DS. Moreover, the minor QTL were significant only in one or some data sets. *QYrid.ui-1A* was detected only in the IT data set from PLM at the two stages in 2003, *QYrrb.ui-3B.1* was significant only in IT data set collected at the soft dough stage in PLM in 2004, *QYrid.ui-3B.2* was revealed only in DS data from the

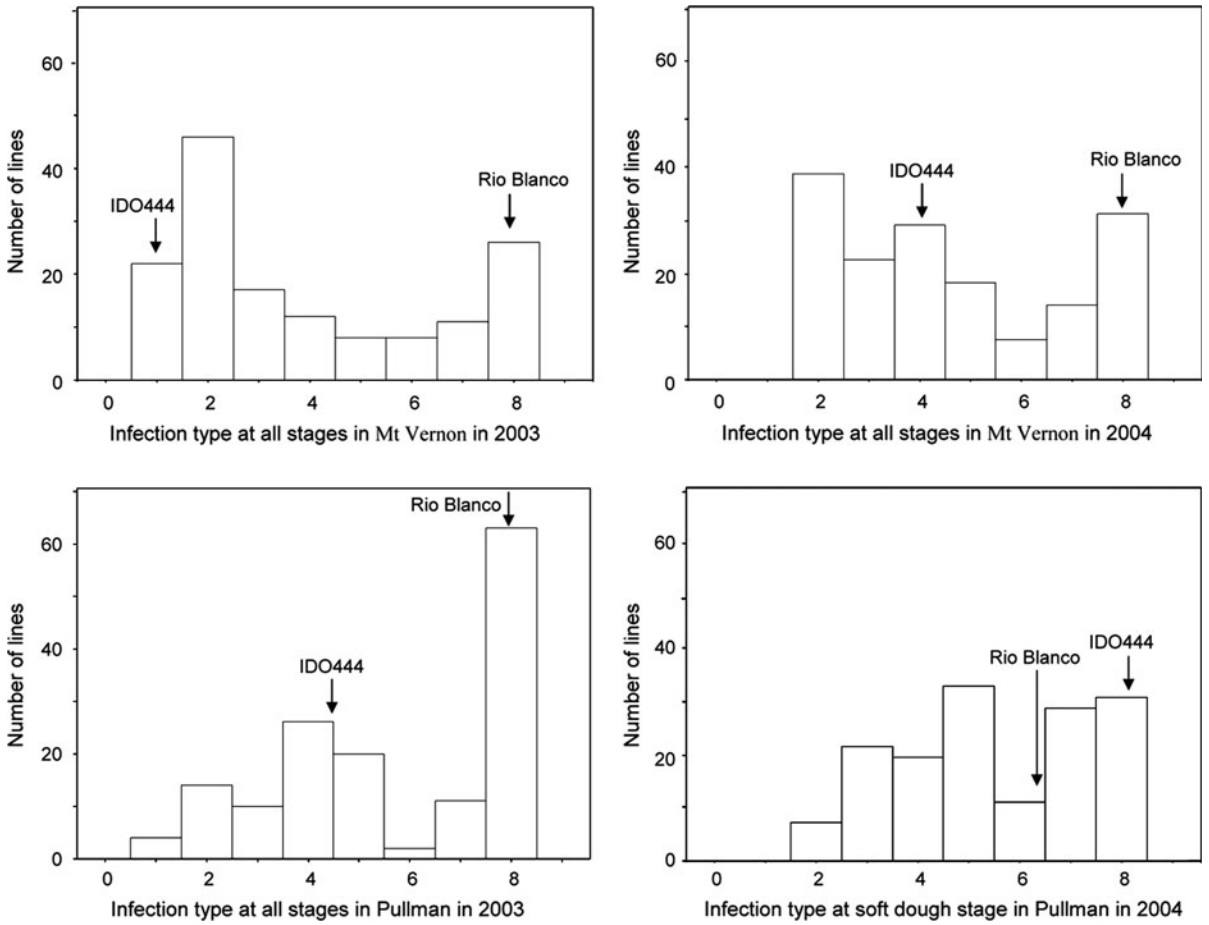

Fig. 2 Frequency distributions of infection type in RIL population derived from RioBlanco × IDO444 in 2003 and 2004 at Mt Vernon and Pullman

Table 1 Estimated heritabilities of HTAP resistance to stripe rust in a Rio Blanco × IDO444 RIL population

Trait	Heritability
Disease severity	
Mt. Vernon, two stages in both years	0.75
Pullman, milk stage in 2003	0.89
Pullman, soft dough stage in 2003	0.94
Pullman, two stages in 2004	0.53
Infection type	
Mt. Vernon, two stages in 2003	0.69
Mt. Vernon, two stages in 2004	0.88
Pullman, two stages in 2003	0.95
Pullman, soft dough stage in 2004	0.45

two stages in PLM in 2004, and both *QYrrb.ui-4B* and *QYrid.ui-5B* were significant only in the data set from MtV at the two stages in 2003.

Discussion

The Rio Blanco × IDO444 linkage maps had a total length of 3,266 cM with an average density of one marker per 6.7 cM. Based on the previously reported genetic maps (Chu et al. 2008; Quarrie et al. 2005; Somers et al. 2004; Sourdille et al. 2003), our maps had about 80% genome coverage. Our markers had similar order and distances to the linkage maps constructed using SSR and DArT markers reported by Somers et al (2004) and Triticarte Wheat Services (http://www.triticarte.com.au).

IDO444-derived major QTL for HTAP resistance (both DS and IT) were identified on chromosomes 2B and 4A. Stripe rust resistance QTL on chromosome 2B were previously reported by Carter et al. (2009) for the *QYrlo.wpg-2BS* QTL in soft white spring wheat cultivar Louise, by Ramburan et al. (2004) for

Table 2 QTL detected by composite interval mapping significantly (LOD > 2.84) associated with disease severity and infection type of stripe rust in the Rio Blanco × IDO444 RIL population evaluated at Mt.Vernon (MtV) and Pullman (PLM) in 2003 and 2004

QTL	Chr	Data set	Marker interval[a]	LOD	AE[b]	R^2 (%)
Disease severity						
QYrid.ui-2B.1	2B	MtV, two stages in both years	*wPt-9668–Xgwm429*	12.3	−19.82ID	25.0
QYrid.ui-2B.2	2B	MtV, two stages in both years	*Xgwm429–Xbarc91*	16.5	−18.06ID	31.0
QYrid.ui-3B.2	3B	PLM, two stages in 2004	*X379646–Xgwm299*	3.1	−6.61ID	9.0
QYrid.ui-4A	4A	MtV, two stages in both years	*wPt-2983–wPt-8275*	10.8	−13.94ID	22.0
		PLM, two stages in 2004	*wPt-2983–wPt-2319*	8.1	−11.69ID	22.0
Infection type						
QYrid.ui-1A	1A	PLM, two stages in 2003	*X377889–XLMW1*	3.0	−0.72RB	9.0
QYrid.ui-2B.1	2B	MtV, two stages in 2003	*wPt-9668–Xgwm429*	4.9	−1.05ID	10.0
		MtV, two stages in 2004	*wPt-9668–wPt-8492*	5.8	−1.13ID	15.0
QYrid.ui-2B.2	2B	MtV, two stages in 2003	*Xgwm429–Xbarc91*	8.1	−1.16ID	16.0
		MtV, two stages in 2004	*X379114–Xbarc91*	3.6	−0.77ID	10.0
QYrrb.ui-3B.1	3B	PLM, soft dough stage in 2004	*X345897–wPt-3921*	3.6	−0.68RB	10.0
QYrid.ui-4A	4A	MtV, two stages in 2003	*wPt-2983–wPt-8275*	11.3	−1.35ID	26.0
		MtV, two stages in 2004	*wPt-2151–wPt-1961*	3.9	−0.63ID	8.0
		PLM, soft dough stage in 2004	*wPt-2983–wPt-6440*	5.1	−0.79ID	14.0
QYrrb.ui-4B	4B	MtV, two stages in 2003	*Xgwm165–Xgwm495*	3.0	−0.67RB	7.0
QYrid.ui-5B	5B	MtV, two stages in 2003	*X63541–X barc59*	3.3	−0.76ID	7.0

Chr chromosome

[a] Marker interval based on significance level of the marker associated with the trait

[b] *AE* additive effect; a negative value indicates the amount of disease score decreased by the gene or QTL. Letters following indicate the corresponding parent (ID = IDO444 and RB = Rio Blanco) that contributed the resistance effects

the *QYr.sgi-2B.1* QTL in wheat cultivar Kariego, and by Guo et al. (2008) for the *QYrlu.cau-2BS1* and *QYrlu.cau-2BS2* QTL in winter wheat cultivar Luke. Based on chromosomal positions, *QYrid.ui-2B.1* in this study was located in a similar region to *QYr.sgi-2B.1* in Kariego and *QYrlu.cau-2BS1* in Luke, and *QYrid.ui-2B.2* seems close to *QYrlu.cau-2BS2* in Luke and *QYrlo.wpg-2BS* in Louise. Ramburan et al. (2004) also reported a minor QTL (*QYr.sgi-4A.1*) on chromosome 4A in Kariega, and based on the position of common marker *Xgwm160*, the 4A major QTL *QYrid.ui-4A* identified in this study may be in the same region as *QYr.sgi-4A.1*.

Of the five minor QTL identified in this research, *QYrrb.ui-3B.1* was located in the same region as *QTLYrns-B1* on 3BS in an old German/UK line Lgst. 79–74 (Khlestkina et al. 2007). *QYrrb.ui-4B* may be the same locus as *QYr.caas-4BL* identified in an Italian wheat cultivar Strampelli (Lu et al. 2009) since they are located in the same region on chromosome 4B. The remaining three QTL, *QYrid.ui-1A*, *QYrid.ui-3B.2* and *QYrid.ui-5B* have not been previously reported.

The magnitude of the resistance attributed to the QTL loci varied greatly among location and year combinations. The heritability of the resistance was very large, suggesting that, although the RILs could be predictably scored for resistance, the environmental conditions such as timing of transitions to warmer spring temperatures may be affecting the induction of the resistant phenotype. Chen (2005) has indicated that the expression of HTAP resistance is often affected by plant growth stage, temperature, humidity and the inoculum load. In this research, HTAP resistance was evaluated under natural infection in field trials, which added to the variation in the trial and probably contributed to the smaller heritability values in some of the Pullman datasets. However, the environments and temperatures appear to differentially induce QTL expression as well. Since races with varied virulence and frequencies could cause disease with different levels of severity (Lin and Chen 2007), the change of the significance of the QTL could also be related to the different timing of infection and composition of *P. striiformis* f. sp.

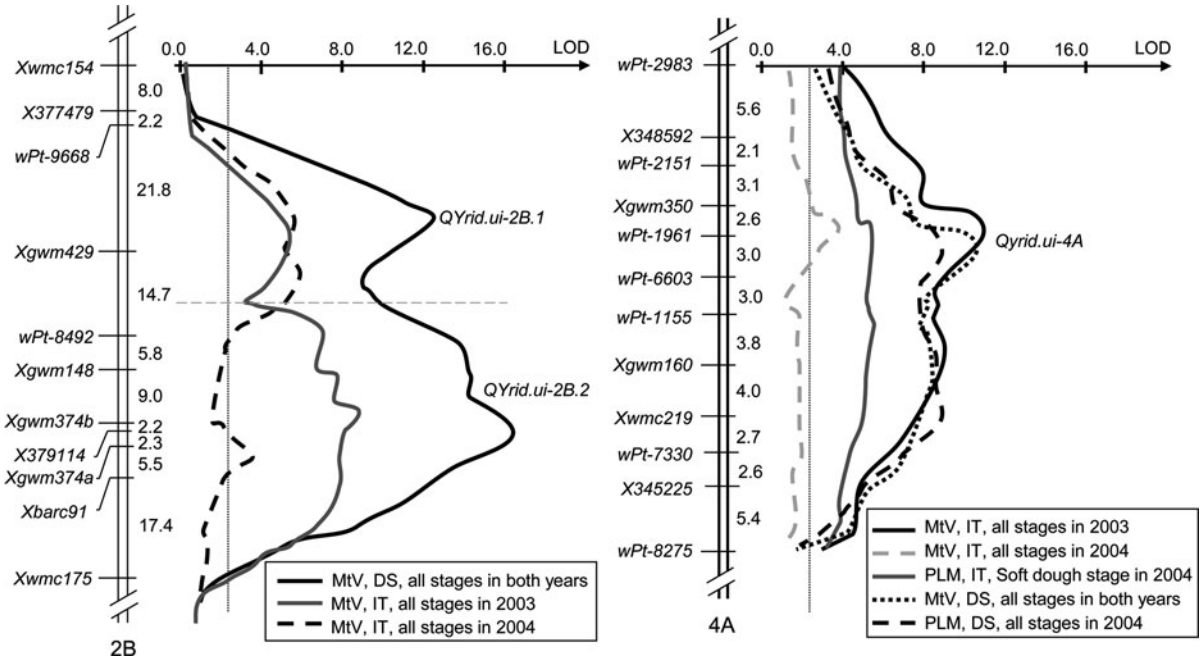

Fig. 3 Graph of chromosomal segments showing major QTL on chromosomes 2B and 4A conferring HTAP resistance to stripe rust in the RIL population from RioBlanco × IDO444 evaluated in 2003 and 2004 at Mt Vernon (*MtV*) and Pullman (*PLM*). Positions of marker loci are shown to the *left* of the linkage groups and distances (cM) between loci are shown on the *right*. The *vertical dotted line* represents the logarithm of odds (*LOD*) significance threshold of 2.84. The LOD and R^2 values for each QTL are listed in Table 2

tritici races in MtV and PLM in 2003 and 2004 reported by Chen et al. (2010). The major QTL in this study were consistent with previous reports using diverse resistance sources under different environments. Thus selection for markers linked to the resistance alleles in IDO444 should have great potential for marker-assisted selection in pyramiding different resistance genes to improve wheat adult plant resistance to stripe rust. The possibility exists of alternative alleles *QYrid.ui-2B.1*, *QYrid.ui-2B.2* and *QYrid.ui-4A* linked to the markers associated with resistance in IDO444. Co-segregation of resistance with the markers should be validated when parents other than IDO444 are used as sources of resistance. The commonality of HTAP studies identifying the regions in 2BS and 4A as sources of the resistance to stripe rust suggests that they would be valuable targets for studies of large-scale association mapping.

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