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ORIGINAL PAPER

Quantitative trait loci for non-race-specific, high-temperature adult-plant resistance to stripe rust in wheat cultivar Express

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Abstract Wheat cultivar Express has durable, high-temperature adult-plant (HTAP) resistance to stripe rust (*Puccinia striiformis* f. sp. *tritici*). To elucidate the genetic basis of the resistance, Express was crossed with 'Avocet Susceptible' (AVS). A mapping population of 146 F_5 recombinant inbred lines (RILs) was developed using single-seed descent. The RILs were evaluated at two sites near Pullman in eastern Washington and one site near Mount Vernon in western Washington in 2005, and were evaluated near Pullman in 2006 under natural stripe rust infection of predominant races virulent on seedlings of Express. Infection type (IT) and disease severity (DS) were recorded three times for each line during each growing season. The DS data were used to calculate relative area under the disease progress curve (rAUDPC) values. Both IT and rAUDPC data showed continuous distributions, indicating that the Express HTAP resistance was controlled by quantitative trait loci (QTL). Resistance gene analog polymorphism (RGAP) and simple sequence repeat (SSR) techniques were used to map the HTAP resistance QTL. Three QTL were detected with significant additive effects, explaining 49.5–69.6% of the phenotypic variation for rAUDPC. Two of the QTL explained 30.8–42.7% of the phenotypic variation for IT. The three QTL were mapped to wheat chromosomes 6AS, 3BL and 1BL, and were designated as *QYrex.wgp-6AS*, *QYrex.wgp-3BL* and *QYrex.wgp-1BL*, respectively. *QYrex.wgp-6AS* and *QYrex.wgp-3BL*, which had higher effects than *QYrex.wgp-1BL*, were different from previously reported QTL/genes for adult-plant resistance. Markers *Xgwm334–Xwgp56* and *Xgwm299–Xwgp66* flanking the two major QTL were highly polymorphic in various wheat genotypes, suggesting that these markers are useful in marker-assisted selection.

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Introduction

Stripe rust (yellow rust), caused by *Puccinia striiformis* Westend. f. sp. *tritici* Eriks., is one of the most destructive diseases of wheat worldwide (Stubbs 1985; Wellings and McIntosh 1990; Chen 2005). Growing resistant cultivars is the most effective method to control the disease, but new races that develop rapidly in the fungal pathogen population can render race-specific resistances ineffective. More than 70 officially and provisionally designed genes for stripe rust resistance have been described (McIntosh et al. 1999, 2001; Chen 2005). However, the majority of these genes confer race-specific resistance and most of them are no longer individually effective. Because race-specific resistance genes usually do not provide long-term protection of crops from the disease, scientists have been seeking and using non-race-specific resistance since the late 1950s in the Pacific Northwest of the United States (Qayoum and Line 1985; Line 2002; Chen 2005). High-temperature,

adult-plant (HTAP) resistance to stripe rust, which is non-race-specific, has been widely used to develop wheat cultivars with durable resistance in the US Pacific Northwest and other regions. Although HTAP or adult-plant resistance has been reported in numerous wheat cultivars, only a few genes or quantitative trait loci (QTL) for durable resistance have been mapped (Boukhatem et al. 2002; Bossolini et al. 2006; Lagudah et al. 2006; Rosewarne et al. 2006; Lin and Chen 2007).

Quantitative traits are assumed to be controlled by multiple genes, but the genes segregate according to Mendel's laws (Tanksley 1993). The effect of each locus in quantitative traits can be estimated through QTL mapping. Quantitative resistance to stripe rust has been characterized by QTL analysis in wheat (Bariana et al. 2001; Boukhatem et al. 2002; Suenaga et al. 2003; Bossolini et al. 2006; Lagudah et al. 2006; Rosewarne et al. 2006; Lin and Chen 2007) and in barley (Chen et al. 1994; Toojinda et al. 2000; Castro et al. 2003; Yan and Chen 2008). Researchers reported that QTL each with small effects can contribute collectively to high levels of adult-plant resistance to stripe rust.

Many wheat cultivars grown in the US Pacific Northwest have HTAP resistance (Line 2002; Chen 2005). The hard red spring wheat cultivar Express (PI 573003), released by the Western Plant Breeders Inc. in 1991 (http://www.ars-grin.gov/cgi-bin/npgs/acc/search.pl?accid=PI+573003), was added to the set of wheat genotypes to differentiate races of *P. striiformis* f. sp. *tritici* in 1998 as new races appeared to overcome the all-stage (also called seedling or overall) resistance in Express (Line 2002; Chen 2005). Even though express-virulent races have become predominant throughout the US since 2000, the cultivar has remained moderately to highly resistant to stripe rust in various regions. Chen et al. (2003) reported that Express has non-race specific HTAP resistance.

Previously, we identified two genes, *YrExp1* and *YrExp2*, conferring race-specific, all-stage resistance in Express and mapped them on chromosomes 1BL and 5BL, respectively (Lin and Chen 2008). The objectives of the present study were to determine the mode of inheritance and number of genes for HTAP resistance in Express, identify QTL contributing to the reduction of disease severity and infection type, map the HTAP resistance QTL on chromosomes, and determine polymorphisms of molecular markers flanking the QTL in other wheat genotypes for marker-assisted selection.

Materials and methods

Plant materials

'Express', a hard red spring wheat cultivar developed by Western Plant Breeders Inc. in the United States, has both race-specific all-stage and non-race-specific HTAP resistance to stripe rust (Chen et al. 2003; Lin and Chen 2008). 'Avocet Susceptible' (AVS), an Australian spring wheat selection, is highly susceptible to predominant races of *P. striiformis* f. sp. *tritici*. A cross was made between AVS and Express using Express as the male parent (Lin and Chen 2008). A total of 146 F_5 recombinant inbred lines (RILs) developed from AVS/Express were used in field experiments in 2005. The same RILs, which were harvested through bulking the seed from one row of each F_5 line in one of the 2005 fields, were evaluated in the field experiment in 2006 and used for DNA extraction. To determine the mode of inheritance of HTAP resistance in Express, F_1 and F_2 plants of AVS/Express were also tested in the field in 2006. 'Chinese Spring' and its 21 nullitetrasomic lines were used in polymerase chain reaction (PCR) studies to locate molecular markers to chromosomes. To determine the usefulness of the markers flanking resistance QTL in marker-assisted selection, 42 wheat genotypes, in addition to AVS and Express, were used to test the polymorphisms at the marker loci.

Field evaluation for HTAP resistance

Field experiments were conducted during the crop seasons of 2005 and 2006 under natural disease infection. In 2005, the parents and 146 F_5 RILs of AVS/Express were sown on 7th April at Tukey Farm, 10th April at the Observatary (OB) Hill site near Pullman in eastern Washington, and 22nd April at Mount Vernon in western Washington. The distances are about four kilometers between the Tukey and OB Hill sites and more than 500 km between the Mount Vernon and Pullman sites, which have different races of *P. striiformis* f. sp. *tritici* and different weather patterns. The predominant races in these locations have been those virulent on seedlings of Express since 2002 (Chen 2005, 2007), and therefore resistance detected on Express and the F_5 lines was primarily HTAP resistance. The experimental plots were completely randomized with three replications at each site. In 2006, the F_1, F_2 and F_5 progenies and parents were planted at the Whitlow Farm site near Pullman on 25th April. To reduce the time and cost of field testing and hasten the molecular marker identification, the 2006 field experiment was only at one site. Twenty F_1 and 200 F_2 seeds were space-planted about eight cm apart for facilitating note-taking of individual plants. For the 146 F_5 RILs tested in 2005 and 2006, about 30 seeds from each line were planted in a 1 m row with 20 cm apart between rows. Susceptible spring wheat cultivar 'Lemhi' was planted as a spreader around each plot. Standard practices for fertilization and weed control common to the region were used for field management. Infection type (IT) and disease severity (DS) were recorded at boot, heading-

flowering, and soft dough stages, when rust severities on AVS reached approximately 30, 60 and 95%, respectively. Infection types were based on the 0–9 scale described by Line and Qayoum (1992). Disease severity was assessed visually using the percentage of infected leaf area. Both IT and DS of F_1 and F_2 progenies of individual plants were recorded. For the parents and F_5 RILs, IT was recorded as a single value for homozygous lines and as two or more values for segregating lines, but later analyzed as average scores. Average DS was recorded for each line.

Statistical analyses

Chi-squared tests were used to determine the goodness of fit of observed numbers of plants/lines in each categorized group to expected segregation ratios for the phenotypic IT and DS data. Severity data were used to calculate area under the disease progress curve (AUDPC) for each F_5 RIL and the parents according to the formula:

$$\text{AUDPC} = \sum_i \left[(\chi_i + \chi_{i+1})/2\right] t_i$$

where χ_i is the DS value on date i, t_i is the time in days between dates i and $i + 1$ (Chen and Line 1995a, b). Relative AUDPC (rAUDPC) values were calculated for each line and parent as a percentage of the mean AUDPC value of the susceptible parent, AVS (Lin and Chen 2007).

Using the SAS statistical package (SAS Institute, Cary, NC, USA), an analysis of variance (ANOVA) was performed to estimate genetic and environmental effects with the 146 F_5 RILs, sites, line × site and years. The ANOVA results were used to estimate the heritability (h^2) of DS and IT. Heritability was computed as $\sigma_g^2/(\sigma_g^2 + \sigma_{ge}^2/E + \sigma_e^2/Er)$ (Yang et al. 2005), with σ_g^2, the line variance; σ_{ge}^2, the line × site interaction variance; σ_e^2, the error variance; E, the number of sites; and r, the number of replications per line.

Phenotypic correlation coefficients between DS and IT at each site, between sites and between generations were calculated on a mean basis using the Microsoft Excel analytical tool (Microsoft, Redmond, WA, USA).

RGAP and SSR marker analyses

The same DNA of the parents and the F_5 RILs used in the study of all-stage resistance (Lin and Chen 2008) were used in this study. The RGAP and SSR procedures were as described in Lin and Chen (2007, 2008). The RGA and SSR primers used in the current study were previously presented in Lin and Chen (2008). RGAP markers were designated using the *Xwgp* series (Shi et al. 2001). Chi-squared tests were used to analyze segregations of molecular markers for goodness of fit to expected ratios. A 9:7 ratio for presence and absence of the band was used for dominant markers, and a 7:2:7 ratio for presence of the Express band, both bands and the AVS band for co-dominant markers, as expected for a F_5 generation.

Bulk segregant analysis and linkage map construction

Based on IT and DS data of various sites in both 2005 and 2006, equal amounts of DNA from the 15 most susceptible lines were mixed to form the susceptible bulk (SB) and equal amounts of DNA from the nine most resistant lines were mixed to form the resistant bulk (RB). A total of 978 resistance gene analog (RGA) primer combinations were screened on the parents, RB and SB. Primer pairs showing polymorphism potentially associated with disease reaction were used to genotype the F_5 RILs.

All polymorphic markers were first tested by one-way ANOVA for their effects on HTAP resistance. The markers with significant effects ($P < 0.05$) on either rAUDPC or IT within/across three sites in 2005 and 2006 were used to construct linkage groups by Mapmaker ver. 2.0 (Lander et al. 1987). Linkage groups were assigned to wheat chromosomes by PCR analysis of 21 Chinese Spring nullitetrasomic lines as described in previous publications (Lin and Chen 2007, 2008). To confirm chromosomes identified in the nulli-tetrasomic analysis and to determine chromosomal regions of markers and linkage groups, SSR markers (Röder et al. 1998; Somers et al. 2004) specific to the identified chromosomes were chosen to screen for polymorphic markers in the bulk segregant analysis and selected markers were tested with the F_5 RILs.

QTL analysis

Quantitative trait locus mapping was conducted based on rAUDPC and IT data for each site and also with all sites combined. Variance analysis (VA), interval mapping (IM), composite interval mapping (CIM) and multiple interval mapping (MIM) were carried out using the Windows version of QTL Cartographer V2.5 (Wang et al. 2006). The detection of QTL was performed using IM and CIM analysis. After performing a 1,000-permutation test, a logarithm of odd (LOD) threshold of 2.5 was used for IM and CIM analysis to detect QTL. The walk speed 2.0 cM was chosen for all QTL detections. For each detected QTL, the Zmapqtl procedure was used to estimate the LOD peak, additive effects and percentage of the phenotypic variance explained. The MIM analysis was used to determine digenic QTL × QTL interactions and total phenotypic variance (total R^2) explained by all significant QTL. Due to the low level of heterozygosity of the F_5 population, only additive effects and digenic QTL × QTL interactions were estimated. The QTL were designated following the rules of

wheat QTL nomenclature (http://wheat.pw.usda.gov/ggpages/wgc/98/Intro.htm).

Results

Genetic characterization of HTAP resistance

The susceptible parent, AVS, was susceptible (IT 9) to all *P. striiformis* f. sp. *tritici* races tested in the greenhouse under the low diurnal temperature cycle (Lin and Chen 2008) and in all field sites in 2005 and 2006. Express was susceptible (IT 9) to races PST-59, 78, 98, 100 and 111 in greenhouse seedling tests (Lin and Chen 2008), but exhibited IT 8–9 at tillering, ITs 3–5 at booting and ITs 2–3 at the flowering stage in the fields. The early development of stripe rust in the 2005 growth season allowed us to see the seedling susceptibility of Express to the natural population of *P. striiformis* f. sp. *tritici* in the field. These data agreed with the results from germplasm evaluation at more locations in 2005, 2006 and the previous several years (data not shown). In 2005 and 2006, the predominant races were PST-100, 114, 115 and 116, which were identified from infected leaf samples collected from the experimental fields (data not shown). All of these races are virulent on seedlings of Express (Chen and Penman 2006; Chen 2007). The results showed that Express has HTAP resistance. Hence, the IT data recorded during the flowering and soft-dough stages were used for the detection of HTAP resistance QTL associated with the IT data. All 20 F_1 plants had ITs 7–9 (mean IT 8) and DS of 80–100% (mean 85%), which was close to the mean and range of AVS at the Whitlow site in 2006. The 200 F_2 plants segregated in a 37R : 27S ratio based on the IT data, indicating that the HTAP resistance in AVS/Express was controlled by three partially recessive genes.

The mean DS of Express was 31% at the OB Hill Farm site, 33% at the Tukey Farm site, and 35% at the Mount Vernon site (mean 33% for all sites) in 2005 and 30% at the Whitlow site in 2006. The F_5 RILs displayed continuous variation for mean relative AUDPC and IT with skewed distribution towards susceptibility within each site in 2005 and 2006, except the Mount Vernon site. The reason that the dataset of Mount Vernon in 2005 was not continuously distributed was due to a delayed third data collection and all the susceptible lines had reached 100%. Therefore, the dataset for Mount Vernon was not included in the χ^2 test, ANOVA analysis, and QTL detection. An example of the frequency distribution of IT and relative AUDPC is shown in Fig. 1. This result demonstrated that stripe rust resistance in Express was a quantitative trait controlled by more than one gene/QTL. When lines with mean relative AUDPC less than or within two standard deviations of Express were classified as resistant, the 146 F_5 RILs segregated in a

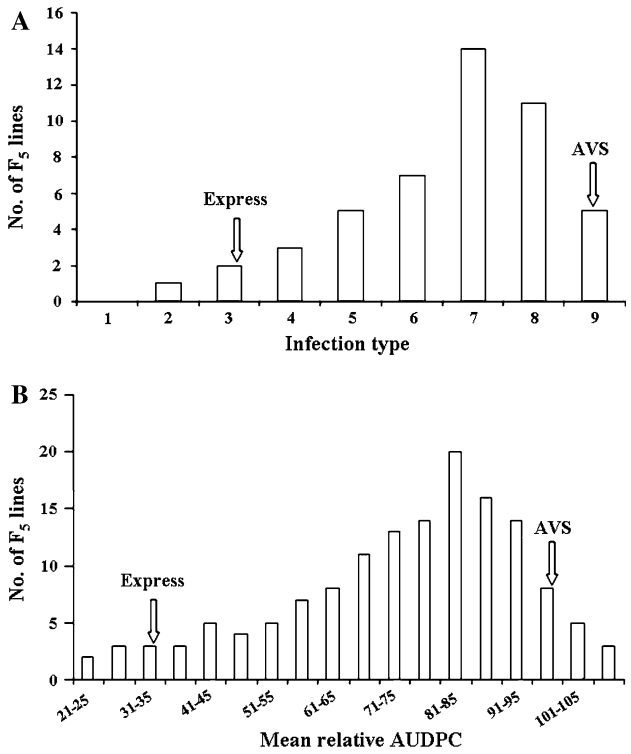

Fig. 1 Frequency distribution of stripe rust infection types (**a**) and relative AUDPC values (**b**) recorded at the soft dough stage at the Whitlow Farm site near Pullman WA in 2006, for 146 F_5 recombinant inbred lines derived from the AVS/Express cross

1R:15S ratio for four independent recessive genes ($\chi^2_{1R:15S} = 0.41–1.99$, $P = 0.15–0.52$, depending upon site and year). If the 27 of 146 F_5 RILs with a mean IT less than or within two standard deviations of Express were considered as resistant, then three recessive genes contributed to the reduction of infection type ($\chi^2_{1R:7S} = 0.46–0.64$, $P = 0.10–0.23$, depending upon site and year).

Among the different sites and between 2005 and 2006, the correlations for the mean rAUDPC ranged from 0.70 to 0.85 ($P < 0.001$) and for IT from 0.87 to 0.91($P < 0.001$), indicating that HTAP resistance in Express was highly heritable. Moderate correlations were found between mean rAUDPC and IT among different sites and between the 2 years ($r = 0.51–0.60$, $P < 0.001$), indicating some of the genes contributed to both reduced rAUDPC (DS) and IT. The correlation between rAUDPC and IT is shown in Fig. 2 using the 2006 Whitlow data.

The ANOVA results showed significant differences ($P < 0.001$) in both rAUDPC and IT among F_5 RILs, sites, line × site interactions and years, but not among replications within sites (data not shown). Therefore, mean rAUDPC and IT data of three replications were used for each site in QTL detection. Heritabilities (h^2) were 0.85 in 2005 and 0.87 in 2006 for rAUDPC, and 0.93 in 2005 and 0.95 in 2006 for IT.

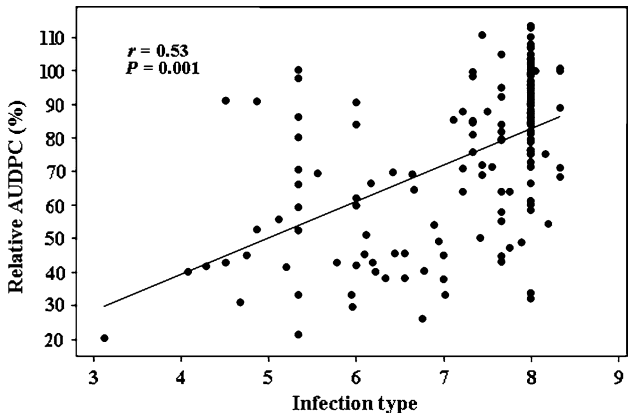

Fig. 2 Scatter plot of relative AUDPC vs. infection type at the Whitlow site in 2006 for 146 F_5 recombinant inbred lines derived from the AVS/Express cross

Identification of molecular markers and mapping HTAP resistance QTL

A total of 978 RGA primer pairs were screened for polymorphism among AVS, Express, RB, and SB, among which 48 primer pairs produced 71 polymorphic bands in the bulk segregant analysis. The 48 polymorphic RGA primer pairs were used to test the 146 F_5 RILs. Based on the one-way ANOVA results, only 60 markers had significant effects ($P < 0.05$) on either rAUDPC or IT within/across sites in 2005 and 2006. The 60 markers were placed on four linkage groups, which were assigned to wheat chromosomes 1B, 2D, 3B and 6A by analyzing the Chinese Spring nulli-tetrasomic lines. A total of 70 SSR primer pairs (15 on chromosome 1B, 15 on 2D, 20 on 3B and 20 on 6A) were chosen to screen for polymorphic SSR markers using bulk segregant analysis. Selected SSR markers were used to genotype the 146 F_5 RILs. SSR markers *Xgwm268* and *Xwmc631* on 1BL, *Xgwm349* and *Xgwm539* on 2DL, *Xgwm340* and *Xgwm299* on 3BL, and *Xgwm334* and *Xgwm459* on 6AS had significant effects on HTAP resistance in the one-way ANOVA tests. The chromosomal locations of these SSR markers were based on the previously published genetic linkage maps by Röder et al. (1998) and Somers et al. (2004). These results allowed us to locate the four linkage groups to wheat chromosomes 1BL, 2DL, 3BL and 6AS. All RGAP and SSR markers associated with HTAP resistance segregated in 7:2:7 ratios expected for co-dominant makers and 9:7 ratios expected for dominant markers in the F_5 RILs (Table 1). As examples, Fig. 3 shows a RGAP marker and a SSR marker in polyacrylamide gels. The established linkage groups with RGAP and SSR markers were used for QTL analysis of HTAP resistance.

For rAUDPC, four QTL associated to HTAP resistance on chromosomes 1BL, 2DL, 3BL and 6AS were detected in the 2005 and 2006 field experiments using the IM analysis and three of them on 1BL, 3BL and 6AS were confirmed by the CIM analysis. Therefore, we present and discuss the three QTL detected by both IM and CIM analyses (Table 2, Fig. 4). Based on the MIM analysis, the QTL on 6AS, designated *QYrex.wgp-6AS*, accounted for 24.5–30.9% of the phenotypic variance and was consistently detected across different sites in the two years. R^2 was as high as 33% when data were combined. The peak of this QTL was located in the marker interval *Xgwm334–Xwgp56* spanning 3.8 cM. The second QTL (*QYrex.wgp-3BL*), located on chromosome 3BL, was identified in all sites across the two years as well as in the combined data, explaining from 22.1 to 27.4% of the phenotypic variation. It was located at the interval of *Xgwm299* and *Xwgp66*. The third QTL (*QYrex.wgp-1BL*) on chromosome 1BL was detected in two field sites as well as in the combined data and explained from 6.8% (OB Hill site in 2005) to 9.4% (combined data) of the phenotypic variation. R^2 values for QTL based on the CIM analysis were lower than those based on IM (data not shown) and MIM analysis (Table 2).

QTL analysis by IM and CIM detected two QTL for IT falling in the same intervals as QTL for rAUDPC on chromosomes 6AS and 3BL. Therefore, these two QTL were interpreted to be the same as *QYrex.wgp-6AS* and *QYrex.wgp-3BL* for rAUDPC (Table 2). *QYrex.wgp-6AS* for IT (R^2 = 15.5–23.6%) showed a higher level of contribution than *QYrex.wgp-3BL* (R^2 = 8.6–18.2%). Markers significantly associated with the three QTL are listed in Table 1.

All three QTL associated with HTAP resistance showed additive effects. The negative values for additive effects of the QTL in all field sites indicated that HTAP resistance originated from the resistant parent, Express (Table 2). There was a significant digenic epistatic interaction between *QYrex.wgp-6AS* and *QYrex.wgp-3BL* for the rAUDPC data of the OB Hill, Tukey Farm and the combined data (Table 2). For IT, the digenic epistatic interaction between *QYrex.wgp-6AS* and *QYrex.wgp-3BL* could only be detected in the OB Hill and Whitlow experiments. The total phenotypic variance explained by all QTL ranged from 48.2 to 71.4% for rAUDPC and from 31.2 to 42.8% for IT. The total explained phenotypic variation increased by 1.7–3.6% (Table 2) upon including the epistatic interactions.

Phenotypic values of QTL represented by flanking markers

The phenotypic values of HTAP resistance for individual QTL and combinations represented by molecular markers

Table 1 Resistance gene analog polymorphism (RGAP) and simple sequence repeat (SSR) markers linked to quantitative trait loci and their primer pairs, size and presence (+) or absence (−) in Express, Avocet Susceptible (AVS) and Chinese Spring (CS), and probability (*P*) values for Chi-square tests of goodness of fit to 9:7 ratios for dominant markers and 7:2:7 ratios for co-dominant markers in the F_5 lines from AVS/Express

Marker[a]	Chromosome Arm	Primer pair[b]	Size (bp)[c]	Presence (+) or absence (−) in			P^d
				Express	AVS	CS	
Xgwm334	6AS	GWM334 F/R	160/180	160	180	NT[f]	0.42
Xgwm459	6AS	GWM459 F/R	320/350	320	350	NT	0.42
Xwgp52	6AS	Pto kin1/Pto kin4	750	−	+	+	0.75
Xwgp53	6AS	Pto kin2/CLRR-INV1	940	+	−	NT	0.33
Xwgp54	6AS	XLRR Rev/Cre3LR-R	1100	+	−	+	0.13
Xwgp55	6AS	Pto kin2/Pto kin4	430	+	−	NT	0.13
Xwgp56	6AS	XLRR Rev/RLRR Rev	980	+	−	+	0.42
Xwgp57	6AS	Pto kin2/Pto kin2IN	940	+	−	NT	0.53
Xwgp58	6AS	Pto kin2/Pto kin1IN	890	+	−	+	0.88
Xwgp59	6AS	Pto kin1/Ptokin1IN	620	+	−	NT	0.18
Xwgp60	6AS	Pto kin2/PtoFen-AS	980	+	−	NT	0.13
Xwgp61	6AS	Pto kin2/PtoFen-AS	920	+	−	NT	0.10
Xgwm340	3BL	GWM340 F/R	250/260	250	260	NT	0.52
Xgwm299	3BL	GWM299 F/R	420/400	420	400	NT	0.25
Xwgp62	3BL	XLRR Rev/Pto kin4	680	+	−	NT	0.25
Xwgp63	3BL	Pto kin1/NLRR For	890	+	−	+	0.42
Xwgp64	3BL	Pto kin1IN/XLRR Rev	850	+	−	+	0.13
Xwgp65	3BL	Pto kin1IN/XLRR Rev	500	+	−	NT	0.10
Xwgp66	3BL	Pto kin2/XalNBS-F	900	+	−	NT	0.75
Xwgp67	3BL	Pto kin1/Cre3LR-F	850	+	−	NT	0.33
Xwgp68	3BL	Pto kin2/CLRR For	810	+	−	NT	0.33
Xwgp69	3BL	Pto kin2/NLRR-INV2	550	+	−	NT	0.25
Xwgp70	3BL	Pto kin2/RLRR Rev	500	−	+	+	0.75
Xwgp71	3BL	XLRR For/RLK For	560	−	+	+	0.33
Xwmc631	1BL	WMC631 F/R	220/180	220	180	NT	0.52
Xgwm268	1BL	GWM268 F/R	420/480	420	480	NT	0.53
Xwgp72	1BL	Pto kin3/S2	470	+	−	NT	0.42
Xwgp73	1BL	Pto kin3/AS1	800	+	−	NT	0.42
Xwgp74	1BL	Pto kin2/AS3-INV	800	−	+	+	0.75
Xwgp75	1Bl	Pto kin2/AS1	810	+	−	+	0.75
Xwgp76	1BL	XLRR For/S2	960	+	−	NT	0.42
Xwgp77	1BL	XLRR For/AS1	820	+	−	NT	0.63
Xwgp78	1BL	XLRR For/XLRR-INV1	470	+	−	NT	0.23
Xwgp79	1BL	Pto kin3/XalNBS For	900	+	−	NT	0.63
Xwgp80	1BL	XLRR For/PtoFen-AS	720	+	−	NT	0.88

S short arm, *L* long arm, *NT* not tested

[a] *Xgwm334*, *Xgwm459*, *Xgwm340*, *Xgwm299*, *Xwmc631* and *Xgwm268* are SSR markers and all others are RGAP markers

[b] The primer sequences were previously published in Leister et al. (1996) for AS1 and S2; Feuillet et al. (1997) for RLK For; Chen et al. (1998) for CLRR For, CLRR-INV1, Cre3LR-F, Cre3LR-R, NLRR For, Pto kin1, Pto kin2, Pto kin3, Pto kin4, Pto-kin2IN, RLRR Rev, XLRR For, and XLRR Rev; Röder et al. (1998) for GWM299, GWM340, GWM334, and GWM459; Shi et al. (2001) for Pto-kin1IN, Xa1NBS-F, and XLRR-INV1; Yan et al. (2003) for AS3-INV; Somers et al. (2004) for GWM268 and WMC631; Pahalawatta and Chen (2005) for PtoFen-AS; and Lin and Chen (2007) for NLRR-INV2

[c] Fragment size estimates were based on visual comparison with the 1 kb plus ladder DNA marker

[d] P = probability of Chi-square test for a co-dominant marker segregating 7:2:7 for the larger, both, and smaller fragments, or a dominant marker segregating 9:7 for presence and absence of the fragment in the F_5 population

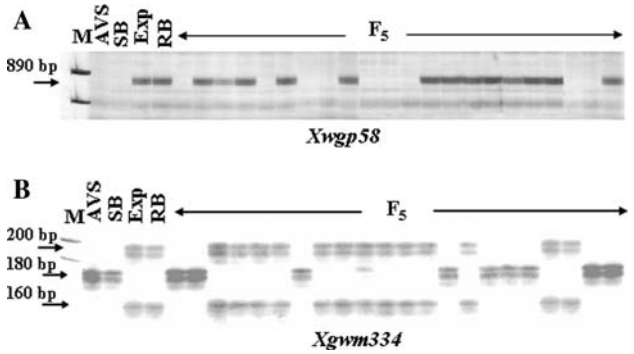

Fig. 3 Polyacrylamide gels showing resistance gene analog polymorphism (*RGAP*) marker *Xwgp58* (**a**) and simple sequence repeat (SSR) marker *Xgwm334* (**b**), which were polymorphic in the bulk segregant analysis and segregated in the F_5 mapping population of AVS/Express

are summarized in Table 3. *QYrex.wgp-6AS* flanked by markers *Xgwm334* and *Xwgp56* and *QYrex.wgp-3BL* flanked by *Xgwm299* and *Xwgp66* affected both DS and IT, whereas *QYrex.wgp-1BL* flanked by *Xwmc631* and *Xwgp78* affected only DS by 12.38–15.91%, but not IT. Therefore, only *QYrex.wgp-6AS* and *QYrex.wgp-3BL* were chosen to determine their combined effects on IT. The 146 F_5 RILs were classified into four genotypic groups. The presence of the *QYrex.wgp-6AS* and *QYrex.wgp-3BL* alleles from Express significantly reduced IT.

Based on the combinations with the presence/absence of the three QTL (*QYrex.wgp-6AS*, *QYrex.wgp-3BL* and *QYrex.wgp-1BL*) resistance alleles, the 146 RILs were classified into eight genotypic groups for examining genetic effects on DS. When the Express alleles of *QYrex.wgp-6AS* and *QYrex.wgp-3BL* were combined, the *QYrex.wgp-1BL* allele did not significantly affect DS. The DS of the RILs with one of the three resistance QTL alleles were lower than those of lines without any resistance alleles but higher than lines with any two of the three resistance alleles. This indicated that combining resistance QTL alleles improved the resistance level.

Polymorphisms of molecular markers flanking HTAP resistance QTL in wheat genotypes

To determine if the QTL-flanking markers are polymorphic across a wide range of wheat genotypes, the markers flanking the two major QTL (*QYrex.wgp-6AS* and *QYrex.wgp-3BL*) were used to test 27 spring and 15 winter wheat cultivars or genotypes (Table 4), many of which have been widely grown in the western U.S. The SSR marker *Xgwm334* and RGAP marker *Xwgp56*, flanking *QYrex.wgp-6AS*, were polymorphic in 88.1 and 40.5%, respectively, of the 42 wheat genotypes. The SSR marker *Xgwm299* and the RGAP marker *Xwgp66*, flanking

Table 2 Quantitative trait loci (QTL) detected for high-temperature, adult-plant (HTAP) resistance by composite interval mapping (CIM) and multiple interval mapping (MIM) of disease data in the AVS/Express population in 2005 and 2006 and their logarithm of odds (LOD), explained phenotypic variances (R^2%) and additive effects

QTL[a]	Position (cM)	Chr.	Observatory Hill (2005)				Tukey Farm (2005)				Whitlow (2006)				Average			
			CIM			MIM	CIM			MIM	CIM			MIM	CIM			MIM
			LOD	R^2%	AE[b]	R^2%	LOD	R^2%	AE[b]	R^2%	LOD	R^2%	AE[b]	R^2%	LOD	R^2%	AE[b]	R^2%
Relative AUDPC																		
1. *QYrex.wgp-6AS*	16.5	6AS	8.6	14.0	−11.1	26.8	5.8	10.9	−8.8	24.5	9.8	15.9	−11.9	30.9	10.5	19.1	−13.3	32.6
2. *QYrex.wgp-3BL*	30.1	3BL	8.4	13.1	−10.3	26.1	5.2	9.1	−7.8	22.1	6.5	11.2	−9.1	24.8	9.9	16.4	−12.5	27.4
3. *QYrex.wgp-1BL*	24.8	1BL	3.1	4.5	−4.6	6.8	–	–	–	–	4.1	5.2	−6.1	7.8	4.4	5.9	−6.8	9.4
Digenic interaction			–	–	1 × 2	1.7[c]	–	–	1 × 2	3.6[c]	–	–	–	–	–	–	1 × 2	1.9*
Total R^2%			–	–	–	61.7	–	–	–	48.2	–	–	–	63.6	–	–	–	71.4
Infection type																		
1. *QYrex.wgp-6AS*	16.5	6AS	3.6	8.3	−0.470	15.5	5.6	13.0	−0.598	22.2	5.4	12.1	−0.523	21.8	5.7	13.4	−0.612	23.6
2. *QYrex.wgp-3BL*	30.1	3BL	3.4	7.4	−0.405	13.2	2.6	6.0	−0.318	8.6	5.0	11.0	−0.506	18.2	2.9	6.9	−0.359	10.2
Digenic interaction			–	–	1 × 2	2.1[c]	–	–	–	–	–	–	1 × 2	2.7[c]	–	–	–	–
Total R^2%			–	–	–	31.2	–	–	–	31.6	–	–	–	42.8	–	–	–	34.1

[a] Only QTL with LOD > 2.5 are shown

[b] AE = additive effect. A negative additive effect value implied that the Express allele decreased the disease score

[c] Significant at $P = 0.05$

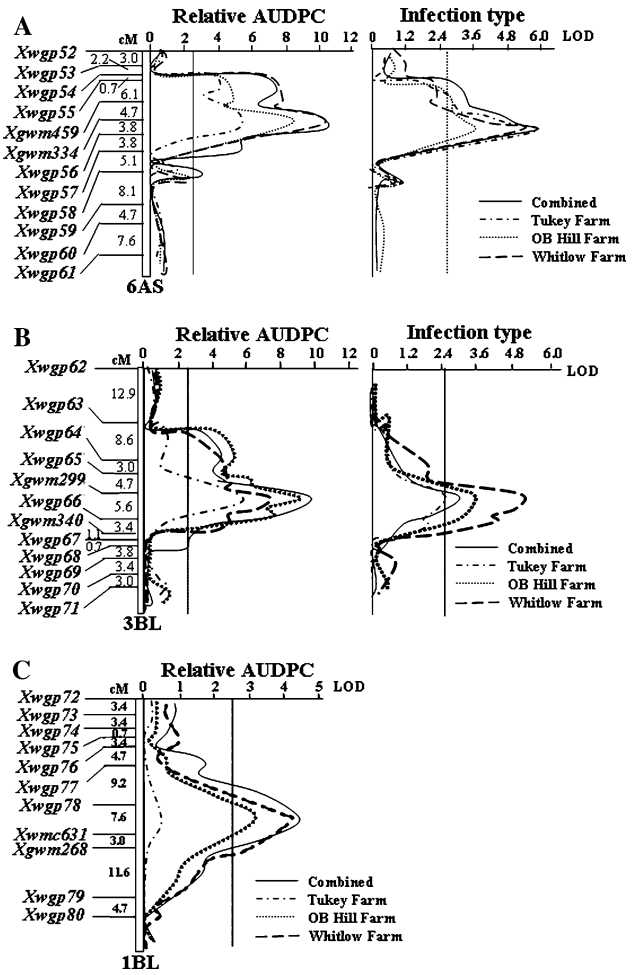

Fig. 4 High-temperature adult-plant (*HTAP*) resistance QTL on chromosomes 6AS (*QYrex.wgp-6AS*), 3BL (*QYrex.wgp-3BL*) and 1BL (*QYrex.wsu-1BL*) identified by composite interval mapping. Positions (in cM) of the molecular markers along the chromosome are shown on the vertical axes

QYrex.wgp-3BL, were polymorphic in 95.2 and 85.7%, respectively, of the 42 wheat genotypes. When the flanking markers were used in combination, they detected polymorphism in 95.2 and 97.6% of the wheat genotypes for *QYrex.wgp-6AS* and *QYrex.wgp-3BL*, respectively.

Discussion

HTAP resistance in wheat cultivars is non-race specific and durable (Qayoum and Line 1985; Chen 2005). Spring wheat cultivar Express, although seedlings susceptible to most races identified since 1998 (Chen et al. 2002; Chen 2005, 2007), is resistant in the field under natural infection by predominant races that are virulent on seedlings of Express. The early development of stripe rust in 2005 allowed us to observe susceptible reactions on Express before the tillering stage. It is evident from the race data and field observations that the recorded resistance in Express and the progeny lines was HTAP resistance.

The frequency distribution of mean rAUDPC and IT in the F_5 RILs showed a continuous variation, which confirmed that HTAP resistance is quantitatively inherited. The similar IT and DS data of F_1 plants to those of the susceptible parent, AVS, indicated that the HTAP resistance in Express were controlled by recessive genes. Similar results reported by Milus and Line (1986a, b) and Chen and Line (1995a, b) indicated that HTAP resistance was a quantitative trait and partially recessive in cultivars Gaines, Nugaines, Luke, Stephens and Druchamp. The recessiveness of HTAP resistance genes may be a general rule, with an exception of the single partially dominant genes controlling HTAP resistance in 'Alpowa' wheat (Lin and Chen 2007) and 'Bancroft' barley (Yan and Chen 2008).

Using QTL analysis, we detected three QTL with significant additive effects on HTAP resistance to stripe rust, explaining 48.2–71.4% of the phenotypic variation for rAUDPC. Two of the three QTL explained 31.2–42.8% of the phenotypic variation for IT in a simultaneous fit model. Further, the total explained phenotypic variation increased by 1.7–3.6% due to the significant digenic epistatic interactions detected. These results support the finding that F_1 plants had IT and DS, close to the mean and range of the susceptible parent, AVS, thereby clearly revealing that HTAP resistance was not merely due to additive effects. Epistatic interactions for HTAP resistance in wheat were previously reported by Chen and Line (1995a). However, as a general rule, additive effects are more important for HTAP resistance.

Several genes/QTL in wheat have been reported for adult-plant resistance, presumably HTAP resistance, to stripe rust, including *Yr16* on 2D (Worland and Law 1986), *Yr18* on 7DS (Singh 1992; Lagudah et al. 2006), *Yr29* on 1BL (Rosewarne et al. 2006), *Yr30* on 3BS (Börner et al. 2000), *Yr34* on 5AL (Bariana et al. 2006), *Yr36* on 6BS (Uauy et al. 2005), and *Yr39* on 6BL (Lin and Chen 2007). In addition to these named genes, Boukhatem et al. (2002) identified QTL for adult-plant resistance on the centromeric region of chromosome 2B and the telomeric regions of 2AL and 7DS in wheat cultivar 'Camp Remy' and the Opata 85/synthetic hexaploid population. Börner et al. (2000) reported *Yrns-B1* in wheat line 'Lgst. 79-74' on chromosome 3BS. Santra et al. (2006) reported QTL in Stephens for HTAP resistance on 6BS. Of the three QTL for HTAP resistance detected in this study, *QYrex.wgp-1BL* was mapped to chromosome 1BL, at the same chromosomal region as *YrExp1* for race-specific all-stage resistance in Express (Lin and Chen, 2008). Because *YrExp1* is only effective against races PST-1 and 21, two old and narrowly virulent races that were not detected in

Table 3 Mean phenotypic values of stripe rust infection type (IT) and disease severity (%) for marker groups representing different QTL and their combinations for HTAP resistance in the F_5 lines of AVS/Express recorded at heading to soft dough stages at different sites in 2005 and 2006

QTL locus and markers	Marker type[a]	Mean infection type (IT) and disease severity (%) of marker group[b]							
		2005 OB Hill (F_5)		2005 Tukey (F_5)		2006 Whitlow ($F_{5:6}$)		Combined	
		IT	%	IT	%	IT	%	IT	%
QYrex.wgp-6AS									
Xgwm334	M1	5.64	48.81	5.54	49.06	5.60	47.79	5.50	47.42
	m1	7.25	66.51	7.28	66.25	7.31	66.34	7.25	66.16
	Difference	−1.61**	−17.7**	−1.74**	−17.19**	−1.71**	−18.55**	−1.75**	−18.74**
Xwgp56	M2	5.7	49.41	5.63	49.95	5.68	48.57	5.61	48.08
	m2	7.13	67.00	7.18	66.22	7.22	66.18	7.26	66.51
	Difference	−1.43**	−17.59**	−1.55**	−16.27**	−1.54**	−17.61**	−1.65**	−18.43**
QYrex.wgp-3BL									
Xgwm299	M3	5.57	48.14	5.67	49.53	5.55	48.91	5.64	47.86
	m3	7.24	65.56	7.18	66.28	7.25	65.79	7.17	66.38
	Difference	−1.67**	−17.42**	−1.51**	−16.75**	−1.7**	−16.88**	−1.53**	−18.52**
Xwgp66	M4	5.71	52.15	5.95	57.52	5.62	54.41	5.77	52.06
	m4	7.2	69.02	7.35	72.65	7.22	70.71	7.19	69.53
	Difference	−1.49**	−16.87**	−1.4**	−15.13**	−1.6**	−16.3**	−1.42**	−17.47**
QYrex.wgp-1BL									
Xwmc631	M5	7.34	58.49	7.37	66.09	7.08	58.27	7.26	57.52
	m5	7.38	71.06	7.4	70.71	7.4	72.35	7.05	73.43
	Difference	−0.04	−12.57**	−0.03	−4.62*	−0.32	−14.08**	0.21	−15.91**
Xwgp78	M6	7.26	62.96	7.26	66.96	7.37	62.88	7.15	59.63
	m6	7.41	75.34	7.48	71.26	7.38	76.40	7.15	74.30
	Difference	−0.14	−12.38**	−0.22	−4.30*	−0.01	−13.52**	0.00	−14.67**
QTL combinations for severity									
M1M2/M3M4/M5M6	Q1Q2Q3	–	39.68a	–	43.56a	–	39.61a	–	37.39a
M1M2/M3M4/m5m6	Q1Q2q3	–	46.55a	–	51.77a	–	46.32a	–	45.31a
M1M2/m3m4/M5M6	Q1q2Q3	–	58.40b	–	63.17b	–	57.72b	–	57.35b
m1m2/M3M4/M5M6	q1Q2Q3	–	59.01b	–	63.81b	–	58.94b	–	58.85b
M1M2/m3m4/m5m6	Q1q2q3	–	66.37b	–	68.56b	–	65.50b	–	63.69b
m1m2/M3M4/m5m6	q1Q2q3	–	66.55b	–	70.11b	–	68.78b	–	64.99b
m1m2/m3m4/M5M6	q1q2Q3	–	77.91c	–	86.30c	–	76.06c	–	75.98c
m1m2/m3m4/m5m6	q1q2q3	–	94.84d	–	89.79d	–	94.28d	–	91.83d
QTL combinations for infection type									
M1M2/M3M4	Q1Q2	3.81a	–	3.90a	–	3.51a	–	3.57a	–
M1M2/m3m4	Q1q2	5.71b	–	5.61b	–	5.68b	–	5.56b	–
m1m2/M3M4	q1Q2	5.83b	–	6.11b	–	5.71b	–	6.01b	–
m1m2/m3m4	q1q2	8.01c	–	8.34c	–	7.96c	–	8.10c	–

[a] M1, M2, M3, M4, M5 and M6 represent alleles in Express and m1, m2, m3, m4, m5 and m6 represent alleles in AVS corresponding to *Xgwm334*, *Xwgp56*, *Xgwm299*, *Xwgp66*, *Xwmc631* and *Xwgp78*, respectively. For convenience, Q1, Q2 and Q3 represent the resistant QTL alleles from Express and q1, q2, and q3 represent the susceptible alleles from AVS at the *QYrex.wgp-6AS*, *QYrex.wgp-3BL* and *QYrex.wgp-1BL* loci, respectively

[b] Phenotypic data were those of the 3rd recording during the flowering to soft-dough stages. Values with "*" and "**" indicate significance at $P = 0.05$ and $P = 0.01$, respectively. Different letters indicate the values were significantly different at $P = 0.05$

fields in 2005 and 2006, and *QYrex.wgp-1BL* is expressed at the adult-plant stage, they are unlikely to be the same gene. Moreover, *YrExp1* induces a resistant reaction (IT 2) (Lin and Chen, 2008), whereas *QYrex.wgp-1BL* does not significantly affect IT, but only reduces disease severity. Neither gene is considered useful for breeding programs because *YrExp1* confers resistance to a very narrow-spectrum of races and *QYrex.wgp-1BL* contributes only a small

Table 4 Polymorphisms in selected wheat genotypes for markers flanking each of the two major quantitative trait loci (QTL) for high-temperature adult-plant (HTAP) resistance to stripe rust in Express

Genotype		Growth habit	Stripe rust[a]		Presence (+) and absence (-) of marker			
					QYrex.wgp-6AS		*QYrex.wgp-3BL*	
Name	ID no.		IT	%	*Xgwm334*[b]	*Xwgp56*	*Xgwm299*[b]	*Xwgp66*
AVS	WG00001	Spring	8–9	98.3	−	−	−	−
Express	PI573003	Spring	2–5	12.5	+	+	+	+
Expresso	Expresso	Spring	0–2	1.0	+	+	+	+
Fielder	CI017268	Spring	8–9	93.3	+	+	−	−
Nick	BZ698031	Spring	2, 5, 8	37.5	+	−	−	−
Alturas	PI620631	Spring	2, 5	5.2	+	−	−	−
Solano	DA900229	Spring	0, 2, 5, 8	4.0	+	−	−	+
Hank	PI613585	Spring	2, 5, 8	34.2	−	+	−	+
Produra	CI 017460	Spring	2, 5, 8	38.3	−	+	−	+
Tara 2002	PI617073	Spring	2, 5, 8	14.2	−	+	−	+
Jerome	IDO00566	Spring	2, 5, 8	13.5	−	+	−	−
Lolo	PI614840	Spring	2, 8	40.0	−	+	−	−
IDO377 s	IDO0377S	Spring	2, 8	28.3	−	+	−	−
Otis	PI634866	Spring	2–5	25.0	−	+	−	−
Scarlet	PI601814	Spring	5–8	51.7	−	+	−	−
Waikea	BZ998447	Spring	2, 5	9.2	−	+	−	−
Wakanz	PI506352	Spring	2	18.3	−	+	−	−
Wawawai	PI574538	Spring	2, 5, 8	38.3	−	+	−	−
UI Cataldo	IDO00642	Spring	0–2	2.5	−	+	−	−
Zak	PI607839	Spring	8	80.0	−	+	−	−
Macon	PI617072	Spring	8	66.7	−	−	−	+
Alpowa	PI566596	Spring	2–3	21.7	−	−	−	−
Edwall	PI477919	Spring	8–9	80.0	−	−	−	−
Louise	PI634865	Spring	2	13.3	−	−	−	−
Blanca Grande	PI631481	Spring	0–2	6.2	−	−	−	−
Buck Pronto	T0001052	Spring	2, 5, 8	7.5	−	−	−	−
Eden	PI630983	Spring	2, 5, 8	43.3	−	−	−	−
Jefferson	PI603040	Spring	2, 5, 8	15.8	−	−	−	−
Jeff/Pronto	JEFF/BUCK	Spring	2, 5, 8	20.0	−	−	−	−
Chinese 166	CI 011765	Winter	2, 5	9.4	−	+	−	+
Barbee	CI017417	Winter	2, 8	6.5	−	+	−	−
Cashup	PI601237	Winter	2–3	7.2	−	+	−	−
Crew	CI017951	Winter	2, 8	11.8	−	+	−	−
Jacmar	PI608016	Winter	2, 8	12.8	−	+	−	−
Omar	CI013072	Winter	8–9	51.7	−	+	−	−
Paha	CI 014485	Winter	2, 5, 8	10.7	−	+	−	−
Rely	PI542401	Winter	2, 5, 8	3.3	−	+	−	-
Rohde	PI582529	Winter	0–2	0.7	−	+	−	−
Tres	CI 017917	Winter	2, 5, 8	23.5	−	+	−	−
Druchamp	CI 013723	Winter	2	1.2	−	−	+	−
Hill 81	CI017954	Winter	0–2	1.0	−	−	−	−
Hiller	PI587026	Winter	2–5	12.8	−	−	−	−
Moro	CI 013740	Winter	2, 8	15.3	−	−	−	−
Riebesel 47–51	YR 00004	Winter	0–2	1.0	−	−	−	−

Table 4 continued

Genotype		Growth habit	Stripe rust[a]		Presence (+) and absence (-) of marker			
					QYrex.wgp-6AS		*QYrex.wgp-3BL*	
Name	ID no.		IT	%	*Xgwm334*[b]	*Xwgp56*	*Xgwm299*[b]	*Xwgp66*
Polymorphism (%)					88.1	40.5	95.2	85.7
					95.2		97.6	

[a] The stripe rust data were from the 2006 plots tested at six locations in Washington, USA. Infection types (ITs) separated by "," indicated different reactions at different locations, and connected by "−" indicated a ranges at one or more locations. The % column is the mean severity value for all six locations

[b] For SSR markers *Xgwm334* and *Xgwm299*, "+" indicates the presence of the Express marker types (i.e., alleles $Xgwm334_{160}$ and $Xgwm299_{420}$), whereas "−" indicates the absence of the Express marker alleles but the presence of either the Avocet Susceptible (AVS) marker alleles ($Xgwm334_{180}$ and $Xgwm299_{400}$) or fragments of different sizes

degree of resistance. Because *Yr29* is on chromosome 1BL (Rosewarne et al. 2006) and confers a low level of adult-plant resistance at the field locations used in the present study (X. M. Chen, unpublished data), the possibility that *QYrex.wgp-1BL* and *Yr29* are the same could not be ruled out. *QYrex.wgp-6AS* and *QYrex.wgp-3BL* are newly described genes. Individual F_5 lines were selected as representative single gene lines to study the mechanisms of HTAP resistance and as genetic stocks for breeding programs.

The usefulness of molecular markers in marker-assisted selection largely depends upon polymorphism. In this study, three of the four closest markers flanking *QYrex.wgp-6AS* and *QYrex.wgp-3BL* showed differences from Express in more than 85% of the tested wheat genotypes. The presence of the flanking alleles in 'Expresso' confirmed that it inherited at least two QTL for HTAP resistance from its Express parent. The results thus indicated that these flanking markers should be useful in introgressing and pyramiding of these resistance genes into other wheat genotypes.

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