Characterization of Novel Gene *Yr79* and Four Additional Quantitative Trait Loci for All-Stage and High-Temperature Adult-Plant Resistance to Stripe Rust in Spring Wheat PI 182103

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ABSTRACT

Stripe rust, caused by *Puccinia striiformis* f. sp. *tritici*, is an important disease of wheat worldwide. Exploring new resistance genes is essential for breeding resistant wheat cultivars. PI 182103, a spring wheat landrace originally from Pakistan, has shown a high level of resistance to stripe rust in fields for many years, but genes for resistance to stripe rust in the variety have not been studied. To map the resistance gene(s) in PI 182103, 185 recombinant inbred lines (RILs) were developed from a cross with Avocet Susceptible (AvS). The RIL population was genotyped with simple sequence repeat (SSR) and single nucleotide polymorphism markers and tested with races PST-100 and PST-114 at the seedling stage under controlled greenhouse conditions and at the adult-plant stage in fields at Pullman and Mt. Vernon, Washington under natural infection by the stripe rust pathogen in 2011, 2012,

and 2013. A total of five quantitative trait loci (QTL) were detected. *QyrPI182103.wgp-2AS* and *QyrPI182103.wgp-3AL* were detected at the seedling stage, *QyrPI182103.wgp-4DL* was detected only in Mt. Vernon field tests, and *QyrPI182103.wgp-5BS* was detected in both seedling and field tests. *QyrPI182103.wgp-7BL* was identified as a high-temperature adult-plant resistance gene and detected in all field tests. Interactions among the QTL were mostly additive, but some negative interactions were detected. The 7BL QTL was mapped in chromosomal bin 7BL 0.40 to 0.45 and identified as a new gene, permanently designated as *Yr79*. SSR markers *Xbarc72* and *Xwmc335* flanking the *Yr79* locus were highly polymorphic in various wheat genotypes, indicating that the molecular markers are useful for incorporating the new gene for potentially durable stripe rust resistance into new wheat cultivars.

Wheat (*Triticum aestivum* L.) is one of the world's major food crops and its production is directly related to global food security. Stripe rust, caused by *Puccinia striiformis* Westend. f. sp. *tritici* Erikss., is one of the major factors threatening wheat production worldwide (Wellings 2011). Compared with other approaches, growing resistant cultivars is the most effective, economic, easy to use, and environmentally safe strategy to control the disease (Chen 2013). Resistance to stripe rust can be divided into different types based on various criteria, of which all-stage resistance and high-temperature adult-plant (HTAP) resistance are separated by the expression of resistance in different plant growth stages and the

influence of temperature (Chen 2013). All-stage resistance is detected at the seedling stage and is expressed throughout all growth stages. It is usually race specific, qualitatively inherited, and can be overcome by new races. In contrast, HTAP resistance becomes increasingly effective as plants become older and the weather becomes warmer. It is usually nonrace specific, quantitatively inherited, and more likely durable (Chen 2013).

To date, 78 *Yr* genes for stripe rust resistance have been permanently named and dozens of temporarily named *Yr* genes or quantitative trait loci (QTL) have been reported (Wang and Chen 2017). Because of high levels of resistance and generally simple inheritance, genes for all-stage resistance have been widely used in breeding programs throughout the world, including, for example, *Yr1*, *Yr6*, *Yr7*, *Yr9*, *Yr10*, *Yr17*, *Yr24/Yr26*, and *Yr27* (Chen 2013; McIntosh et al. 1995). Most all-stage resistance genes have been overcome by virulent races (Wang and Chen 2017). Most wheat cultivars that have exhibited durable resistance to stripe rust possess adult-plant or HTAP resistance controlled by variable numbers of QTL (Carter et al. 2009; Hou et al. 2015; Lin and Chen 2007, 2008; Paillard et al. 2012; Santra et al. 2008). Although HTAP resistance is generally durable, many HTAP resistant wheat cultivars can suffer considerable yield loss in severe epidemics as this type of resistance is incomplete, affected by plant growth stage, temperature, and inoculum load (Chen 2013, 2014). In order to improve stripe rust resistance in commercial cultivars, more genes and useful markers are needed for increasing the level and durability of resistance by combining HTAP resistance with all-stage resistance.

PI 182103, a spring wheat landrace originally from Sind, Pakistan, has exhibited a high level of resistance to stripe rust in field tests in Washington State since 2004. It has both all-stage resistance to some

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P. striiformis f. sp. *tritici* races and a high level of HTAP resistance when tested with different races under controlled conditions (Wang et al. 2012) (X. M. Chen, *unpublished data*). The objectives of the present study were to further characterize the resistance in PI 182103, map and identify genes for all-stage resistance and HTAP resistance, and determine the effects of races and environments on the resistance genes. The characterization of stripe rust resistance and discovery of tightly linked molecular markers for effective resistance genes should be useful for developing new wheat cultivars for sustainable control of stripe rust.

MATERIALS AND METHODS

Wheat lines and crosses. PI 182103 was crossed as male parent with Avocet Susceptible (AvS), an Australian spring wheat selection highly susceptible to most U.S. *P. striiformis* f. sp. *tritici* races at both the seedling and adult stages. One hundred and eighty-five recombinant inbred lines (RILs) were developed by single-seed descent. The parents and F₁ and F₆ progenies were used in seedling tests with selected races and parents and F₁, F₅, F₆, and F₇ progenies were used to phenotype stripe rust responses in field experiments. A complete set of 21 Chinese Spring (CS) nulli-tetrasomic lines, two 7B ditelosomic lines and eight 7BL deletion lines (Del7BL-1, Del7BL-3, Del7BL-5, Del7BL-6, Del7BL-7, Del7BL-9, Del7BL-10, and Del7BL-14) were used to determine the chromosomal position of a major QTL for HTAP resistance in PI 182103. A panel of 72 wheat cultivars and breeding lines was used to evaluate the polymorphisms of the markers flanked the major HTAP resistance QTL in PI 182103.

To determine the relationship of a major HTAP resistance QTL in PI 182103 mapped to chromosome 7BL with *Yr* genes previously reported on chromosome 7BL (*Yr39*, *Yr52*, *YrZH84*, *Yr59*, and *Yr67*), crosses were made between PI 182103 and wheat genotypes Alpowa (*Yr39*) (Lin and Chen 2007), PI 183527 (*Yr52*) (Ren et al. 2012a), PI 178759 (*Yr59*) (Zhou et al. 2014), PI 189747 (C591) (*Yr67 = YrC591*) (Li et al. 2009) (R. McIntosh, *personal communication*), and Zhou 8425B (*YrZH84*) (Li et al. 2006). For allelism tests, F₂ populations and selected F₃ plants in each cross were planted in a field near Pullman in 2013 and 2014.

Characterization of resistance in PI 182103 in the greenhouse. To assess the breadth of all-stage resistance in PI 182103, seedlings were inoculated separately with 10 *P. striiformis* f. sp. *tritici* races (PST-17, PST-21, PST-43, PST-45, PST-78, PST-100, PST-114, PST-116, PST-127, and PST-139) under a low-temperature profile, with diurnal temperatures gradually changing from 4°C at 2:00 a.m. to 20°C at 2:00 p.m. (Chen and Line 1992). These races were selected to cover all virulence factors identified thus far and to represent predominant races and race groups reported in the United States since the 1970s (Wan et al. 2017). To determine if PI 182103 has HTAP resistance, four-way tests were conducted in the greenhouse with races virulent on seedlings of PI 182103: seedling tests at low temperatures (LT, 4 to 20°C), seedling tests at high temperatures (HT, 10 to 30°C), adult-plant tests at LT, and adult-plant tests at HT as previously described (Chen 2013; Chen and Line 1995).

Plants were inoculated with fresh urediniospores mixed with talcum powder at a 1:20 ratio at the two-leaf stage for seedling plant tests or at the booting stage for adult-plant tests (Chen and Line 1995). For seedling tests, about 15 plants were used for each race test without replication. For adult-plant tests, three plants were used in each replication for each race test with three replications in a randomized block experimental design. The inoculated plants were placed in a dew chamber at 10°C for 24 h for all four-way tests, and then moved to growth chambers set for the respective temperature cycles. Light conditions were the same for all four-way tests with 8 h darkness and 16 h light cycles corresponding to the low and high temperature periods, respectively. Infection type (IT) data

were recorded 18 to 22 days postinoculation using a 0 to 9 scale as described by Line and Qayoum (1992).

Seedling phenotyping of the RIL population. To examine the inheritance of all-stage resistance in the AvS/PI 182103 population, 15 seedlings of each F₆ RIL and parents were tested with *P. striiformis* f. sp. *tritici* races PST-100 and PST-114 separately under the LT profile. The two races were chosen because they were avirulent on seedlings of PI 182103, and the former is the predominant race throughout the US while the latter is a major race in the Pacific Northwest region (Chen et al. 2010; Wan and Chen 2012). Inoculation and growth conditions, as well as IT scoring were as described above. Four F₁ plants were included in the tests to determine if the inheritance is dominant or recessive.

Field tests. Field experiments were conducted near Pullman in eastern Washington and near Mt. Vernon in western Washington in 2011 and 2012 and at Pullman in 2013 under natural infections of *P. striiformis* f. sp. *tritici*. The Pullman site has an inland climate, whereas Mt. Vernon has an oceanic climate. The two locations are about 500 km apart, and their weather conditions and *P. striiformis* f. sp. *tritici* races are different every year (Chen et al. 2010; Wan and Chen 2012).

About 30 seeds of each line and each parent were sown in a 0.5 m row with a 0.2 m space between rows. The experimental design was a randomized complete block with three replications at each location. AvS, as a susceptible check, was also planted every 20 rows to increase disease development and uniformity. The fields were managed following local cropping practices. Four F₁ seeds were planted in each location to determine the phenotype of heterozygous plants and the inheritance of the stripe rust resistance in PI 182103. Infection type and disease severity (DS) data were collected three times during each season starting when the susceptible AvS rows had uniform severity.

Phenotypic data analyses. The DS data were used to calculate the area under the disease progress curve (AUDPC), following the formula: $AUDPC = \sum_i [(\chi_i + \chi_{i+1})/2]t_i$, where χ_i is the DS value on date i , t_i is the days between dates i and $(i + 1)$ (Chen and Line 1995). Relative AUDPC (rAUDPC) was calculated for each line as a percentage of the mean AUDPC value of the susceptible parent (AvS). χ^2 tests were used to analyze the goodness of fit between observed and expected segregation ratios using the IT data. Histograms were generated for each experiment to show the distribution of IT or DS data using the Microsoft Office Excel software (Microsoft, Redmond, WA).

Statistical analysis of rAUDPC and IT data were performed for each environment using SASv.9.1 (SAS Institute, Raleigh, NC). Broad-sense heritabilities (H^2) were calculated as $H^2 = \sigma_g^2/\sigma_p^2$, where σ_g^2 is the genetic variance and σ_p^2 is the phenotypic variance; σ_g^2 was calculated from $(\sigma_L^2 - \sigma_E^2)/r$, where σ_L^2 is the mean variance of the RILs, σ_E^2 is the error variance, and r is the number of replications (Hou et al. 2015). Correlation coefficients were calculated for pairwise comparisons of IT and rAUDPC in each environment.

To determine genetic distances between two genes using an F₂ population for the allelism tests, the frequency (f) of susceptible plants in a repulsion phase cross was used to estimate the recombination value (p) using the formula $f = p^2/4$. Recombination values were converted to map distances in centimorgans (cM) for the formula $d = 1/4 \times \ln[(1 + 2p)/(1 - 2p)] \times 100$, and standard deviation (SD) was calculated using

$$SD = \sqrt{d \times (1 - d)/n}$$

where n is the number of F₂ plants (Kosambi 1944).

DNA extraction, PCR amplification and genotyping of the mapping population. Genomic DNA of the RILs and parents extracted using the CTAB (cetyltrimethylammonium bromide) method were dissolved in 1× TE buffer (10 mM Tris-HCl and 1 mM EDTA, pH 8.0) as stock solutions. DNA concentrations

were determined using both 1.0% agarose gel electrophoresis and NanoDrop ND-1000 (Thermo Scientific, Wilmington, DE). DNA stocks were diluted to 25 ng/μl with double distilled (dd)H₂O for use as PCR templates. Simple sequence repeat (SSR) polymorphisms were used to genotype the mapping population. PCR mixtures comprised of 4 μl (100 ng) of template DNA, 1.2 μl of 10× buffer (with 1.5 mM MgCl₂), 0.48 μl of 2.5 mM MgCl₂, 0.96 μl of 2.5 mM dNTP (dATP, dCTP, dGTP, and dTTP), 0.24 μl of 10 mM fluorescent-labeled M13 primer (CACGACATTGTAAACGAC), 0.12 μl of Taq DNA polymerase (5 unit/μl), 0.06 μl of forward primer (10 μM) tailed with M13, 0.30 μl of reverse primers (10 μM), and 4.64 μl of sterilized ddH₂O. These PCR reagents were purchased from Promega Inc. (Madison, WI) and primers were synthesized by Sigma-Aldrich Inc. (St. Louis, MO). PCR was run on an ABI 9700 programmed for 5 min of denaturation at 94°C, followed by 42 cycles of 1 min at 94°C, 1 min at 50 to 65°C depending on primers, and 1 min at 72°C followed by an extra extension at 72°C for 10 min. PCR products were resolved using an ABI capillary DNA sequencer 3730× (Applied Biosystems, Foster City, CA), and called using GeneMapper version 2.22 (SoftGenetics, LLC, State College, PA).

To identify additional markers to increase mapping resolution, PI 182103 and AvS were screened with the 9K wheat single nucleotide polymorphism (SNP) chip (Cavanagh et al. 2013) at the USDA-ARS Biosciences Research Laboratory, Fargo, North Dakota. From 3,000 SNP markers polymorphic between PI 182103 and AvS, 79 SNPs on chromosomes 7B, 5B, 4D, 3A, and 2A were selected to genotype the 185 RILs. SNP genotyping of the RILs was conducted in a Sequenom MassARRAY iPLEX platform at the USDA-ARS Wheat Health, Genetics and Quality, Research Unit, Pullman, WA.

Construction of linkage maps and QTL analysis. SSR and SNP markers were used to construct linkage groups and rAUDPC and IT data were used to analyze and map QTL. Linkage groups of molecular markers were constructed using JoinMap 4.0 (Van Ooijen 2006). Chromosome identifications of linkage groups were made using a consensus map (Somers et al. 2004) (<https://wheat.pw.usda.gov/>), and chromosomal bins of SSR markers were determined based on the deletion map of Sourdille et al. (2004). QTL were detected using the interval mapping (IM) and multiple-interval mapping (MIM) function in IciMapping 3.0 (Meng et al. 2015). IM was used to select markers significantly associated with the stripe rust phenotypes for subsequent use as cofactors in MIM analysis. A walk speed of 2.0 cM was used to detect QTL. A logarithm of odds (LOD) threshold value was determined by permutation test at 1,000 iterations. A significance level of 5% was used to declare QTL in the MIM analysis. Phenotypic variance (R^2) was used as a measure of the effect of a QTL. Additive effects of QTL were calculated using interval mapping (IM) (Li et al. 2007).

RESULTS

Characterization of stripe rust resistance in PI 182103.

When tested at seedling stage under controlled greenhouse conditions, AvS was susceptible to all 10 *P. striiformis* f. sp. *tritici* races and PI 182103 was resistant to races PST-17, PST-21, PST-43, PST-45, PST-78, PST-100, PST-114, and PST-116 and moderately resistant to races PST-127 and PST-139 (Supplementary Table S1). The susceptible reactions of F₁ plants to the three races tested (PST-100, PST-114, and PST-127) indicated that the resistance was recessive. The seedling reactions of PI 182103 and AvS tested with race PST-114 are shown as examples in Supplementary Fig. S1.

To further characterize the resistance, PI 182103 and AvS were tested with race PST-127 in the four-way tests described above. PI 182103 was moderately resistant at seedling stage in the LT and HT tests, but became highly resistant at adult-plant stage in LT and HT tests (Table 1). In contrast AvS and F₁ plants were susceptible (IT 8) in all four-way tests. The results indicated that PI 182103 had a moderate level of resistance to some *P. striiformis* f. sp. *tritici* races

such as PST-127 at the seedling stage and a high level of resistance at the adult-plant stage, thus indicating the presence of HTAP resistance.

To determine the inheritance of all-stage resistance in PI 182103, seedlings from all 185 F₆ RILs from AvS × PI 182103 were tested separately with PST-100 and PST-114 in the LT profile. The RIL population had ITs ranging continuously from IT 2, similar to the resistant parent, to IT 8, similar to AvS (Supplementary Fig. S2), indicating that the all-stage resistance of PI 182103 was quantitatively inherited.

AvS was highly susceptible in all field tests, whereas PI 182103 was moderately resistant at the stem elongation stage and later became highly resistant in 2011 and 2012 at Mt. Vernon where infection always began at the seedling stage (Table 2). Similarly, at Pullman PI 182103 was moderately resistant at the seedling stage and became highly resistant in 2011 when stripe rust appeared on winter wheat crops in early March, much earlier than the normal start of stripe rust in May. However, in 2012 and 2013 as stripe rust infection started after stem elongation in May, only resistant reactions were observed on PI 182103 (Table 2). The responses of PI 182103 and AvS at Pullman in 2012 are shown in Supplementary Figure S3. The frequency distributions of IT and rAUDPC data of the 185 RILs at the five environments were continuous (Supplementary Figs. S4 and S5), indicating that the adult-plant resistance in PI 182103 was controlled by QTL. F₁ plants grown at Pullman in 2011 were susceptible, indicating that resistance was recessive.

Phenotypic variation, correlation coefficients, and heritability of resistance. The population rAUDPC means at Mt. Vernon were lower than those at Pullman (Table 2), indicating that more QTL were effective at Mt. Vernon than at Pullman. The correlation coefficients of the IT and rAUDPC data within and between different environments (location and year) were calculated.

High correlation coefficients (≥ 0.50) were detected for 29 of 45 possible trait–environment comparisons and low correlation coefficients (< 0.50) were observed for the 16 remaining pair-wise comparisons (Supplementary Table S2). However, all of the correlations were significant ($P < 0.01$). In general, the IT and rAUDPC data were highly correlated at Mt. Vernon over the 2 years and the rAUDPC data were highly correlated at Pullman over the 3 years. In contrast, the correlation coefficients were highly variable for the IT data over the 3 years at Pullman, and between the IT and rAUDPC data at Pullman and between the IT data at Pullman and rAUDPC data at Mt. Vernon. These results indicated that different QTL were involved in the IT and rAUDPC responses and they were affected differently by different *P. striiformis* f. sp. *tritici* races and/or environments in the different locations and years. Although differences were recorded, the IT and rAUDPC data were highly correlated across the 3 years at Pullman ($r^2 = 0.80$, $P < 0.001$) and between the 2 years at Mt. Vernon ($r^2 = 0.86$; $P < 0.001$) (Fig. 1).

ANOVA revealed significant differences among RILs, among or between environments (location and/or year), and interactions

TABLE 1. Infection types on PI 182103, Avocet Susceptible (AvS), and F₁ AvS × PI 182103 inoculated with race PST-127 in the four-way test under controlled temperature conditions

Wheat	Infection type			
	Seedling ^a		Adult-plant ^a	
	LT ^b	HT ^b	LT ^b	HT ^b
PI 182103	5	4	2–3	1
AvS	8	8	8	8
F ₁	8	8	8	8

^a Seedlings were inoculated at the two-leaf stage and adult plants at the boot stage. After inoculation, plants were kept in a dew chamber with 100% humidity in dark at 10°C for 24 h and then moved to growth chambers with different temperature settings.

^b LT = low temperature profile: diurnal cycle gradually changing from 4°C at 2:00 a.m. to 20°C at 2:00 p.m.; and HT = high-temperature profile: diurnal cycle gradually changing from 10°C at 2:00 a.m. to 30°C at 2:00 p.m.

between RILs and environments for both the IT and rAUDPC data from Pullman, Mt. Vernon, and Pullman and Mt. Vernon combined ($P < 0.0001$), except for environment (over years) at Pullman ($P = 0.22$) (Table 3).

The estimated broad-sense heritability (H^2) values were high, ranging from 0.88 using the rAUDPC data to 0.96 using the IT data at Pullman ($P < 0.001$) (Table 3). When the Pullman and Mt. Vernon data were combined, the estimated H^2 value was 0.89 for IT and 0.94 for rAUDPC.

QTL for stripe rust resistance. Of 836 SSR and 9,000 SNP markers screened on AvS and PI 182103, 473 SSR and 3,000 SNP markers were polymorphic. The polymorphic SSR markers were

screened on the susceptible and resistance bulks and parents, revealing 85 SSR markers on chromosomes 2AS, 3AL, 4DL, 5BS, and 7BL associated with stripe rust resistance. Forty-four SSR markers and 50 SNP markers were selected based on their robustness and chromosome position to genotype the 185 RILs for constructing linkage maps. These markers produced five linkage groups assigned to chromosomes 2AS, 3AL, 4DL, 5BS, and 7BL (Fig. 2). The shortest group was chromosome 4DL (23.2 cM) and the longest was 2AS (106.6 cM). The 2AS linkage map comprised 17 markers, 3AL 11 markers, 5BS 21 markers, 7BL 21 markers, and 4DL 10 markers.

In the IM analyses for mapping QTL for resistance to stripe rust using software IciMapping 3.0, a LOD threshold 2.5 was used to

TABLE 2. Infection types (IT) and relative areas under disease progress curve (rAUDPC) for parents and minima, maxima and means of IT and rAUDPC among recombinant inbred lines (RILs) from AvS × PI 182103 tested for stripe rust response under conditions of natural infection at Pullman and Mt. Vernon in 2011 to 2013

Location	Year	Data	AvS	PI 182103	RILs					
					Minimum	Maximum	Mean	SD ^a	CV ^b	P
Pullman	2011	IT	8	2	2	8	4.5	8.1	1.8	<0.001
		rAUDPC	100	12	11	95	49.1	24.1	0.5	<0.001
	2012	IT	8	2	2	8	3.9	11.2	2.9	<0.001
		rAUDPC	100	15	6	101	48.9	27.1	0.6	<0.001
	2013	IT	8	2	2	8	4.8	2.2	0.5	<0.001
		rAUDPC	100	7	6	118	42.3	46.5	1.1	<0.001
Mt. Vernon	2011	IT	8	2	2	8	3.5	8.1	2.3	<0.001
		rAUDPC	100	15	15	93	41.7	25.8	0.6	<0.001
	2012	IT	8	2	2	8	2.9	11.1	3.9	<0.001
		rAUDPC	100	18	11	100	39.3	28.9	0.7	<0.001

^a SD, standard deviation.

^b CV, covariance.

Fig. 1. Scatter plots of correlation of infection type (IT) and relative area under the disease progress curve (rAUDPC) of 185 F_7 recombinant inbred lines from cross AvS × PI 182103 at A, Pullman and B, Mount Vernon following natural infection of *Puccinia striiformis* f. sp. *tritici*.

TABLE 3. Analysis of variance and heritability (H^2) of infection type (IT) and mean relative area under the disease progress curve (rAUDPC) among recombinant inbred lines (RILs) from AvS × PI 182103 tested under natural infection of *Puccinia striiformis* f. sp. *tritici* at Pullman and Mt. Vernon in 2011 to 2013

Location	Variance or H^2	IT				rAUDPC			
		df	MS	F	P	df	MS	F	P
Pullman	RIL	184	33.8	42.6	<0.0001	184	3,580.0	7.7	<0.0001
	Year	2	69.2	87.3	<0.0001	2	504.1	1.5	0.22
	RIL × year	368	2.4	3.1	<0.0001	368	764.7	2.1	<0.0001
	Error	1,110	0.8			1,110	261.4		
	H^2		1.0		<0.0010		0.9		<0.0010
Mt. Vernon	RILs	184	18.2	28.2	<0.0001	184	2,547.1	14.1	<0.0001
	Year	1	58.1	90.1	<0.0001	1	3,241.1	17.9	<0.0001
	RIL × year	184	1.9	2.9	<0.0001	184	394.2	2.2	<0.0001
	Error	740	0.7			740	180.8		
	H^2		0.9		<0.0010		0.9		<0.0010
Pullman and Mt. Vernon	RIL	184	30.7	36.4	<0.0001	184	5,220.0	21.0	<0.0001
	Location	1	134.1	159.0	<0.0001	1	20,968.9	84.5	<0.0001
	RIL × location	184	8.7	10.3	<0.0001	184	549.8	2.2	<0.0001
	Error	2,035	0.8			2,035	248.3		
	H^2		0.9		<0.001		0.9		<0.001

declare QTL with the field data and 3.0 with the greenhouse seedling tests based on permutation tests. A total of five QTL were detected with the seedling IT data and the IT and rAUDPC field data. The positions of the QTL on chromosomes 2AS, 3AL, 4DL, 5BS, and 7BL in the linkage groups are shown in Figure 2. As an example, the map for the 7BL QTL showing the LOD scores for all the data are given in Figure 3.

In the seedling tests, the frequencies of IT data were similar in both the PST-100 and PST-114 tests, indicating that resistance to these races was controlled by the same genes. QTL were identified on chromosomes 2AS, 3AL, and 5BS (Fig. 2), and all three were detected in the tests with PST-100 and PST-114 (Table 4). The QTL *QyrPI182103.wgp-2AS* mapped between SSR marker *Xgwm312* and SNP marker *IWA5585*, and it explained 8.2% of the phenotypic variation for PST-100 and 5.0% for PST-114. The 3AL QTL, *QyrPI182103.wgp-3AL*, was located between SSR marker *Xgwm2* and SNP marker *IWA899*, and it explained 8.1% of the phenotypic variation for PST-100 and 5.4% for PST-114. The 5BS QTL, *QyrPI182103.wgp-5BS*, was located between SSR markers *Xgwm213* and *Xgwm540*, and it explained 14.2% phenotypic variation for PST-100 and 8.2% for PST-114. These three QTL showed mostly additive effects (AE), and the AE values ranged from -0.54 to -0.88 (Table 4). *QyrPI182103.wgp-2AS* and *QyrPI182103.wgp-3AL* were detected only in seedling tests, whereas *QyrPI182103.wgp-5BS* was also detected in the field tests.

Based on the field data, three QTL, *QyrPI182103.wgp-5BS*, *QyrPI182103.wgp-7BL*, and *QyrPI182103.wgp-4DL*, were identified and mapped in chromosomes 7BL, 5BS, and 4DL, respectively (Fig. 2, Supplementary Table S3). The negative AE values of the QTL in all field tests indicated that the resistant alleles were from the resistant parent, PI 182103.

QyrPI182103.wgp-5BS, close to markers *Xgwm213* and *Xgwm540*, explained phenotypic variations of 30.5% (2013) to 55.6% (2011) in IT data in all 3 years and 16.1% (2011) to 23.3% (2012) of rAUDPC variation at Pullman. This QTL was detected for IT in 2011 (explaining 26.9% of the variation) and in 2012 (10.6%) and for rAUDPC at Mt. Vernon in both 2011 (15.3%) and 2012 (14.8%). On average, the QTL explained 57.9% of the variation in IT and 20.2% in rAUDPC at Pullman, and 13.6% in IT and 15.6% in rAUDPC at Mt. Vernon. The 5BS QTL contributed more to resistance at Pullman than at Mt. Vernon and reduced IT more than rAUDPC at Pullman.

QyrPI182103.wgp-7BL, close to markers *Xwmc335* and *Xbarc72*, was significant for both IT and rAUDPC in all the field tests. This QTL explained phenotypic variation ranging from 13.9% (Mt. Vernon 2012) to 29.4% (Pullman 2011) for IT and 16.3% (Pullman 2011) to 22.0% (Pullman 2012) for rAUDPC. On average, the QTL explained 27.7% IT variation and 20.0% rAUDPC variation at Pullman, and 12.6% IT variation and 17.9% rAUDPC variation at Mt. Vernon. In general, the 7BL QTL contributed more to resistance

Fig. 2. Linkages and quantitative trait loci for resistance to stripe rust mapped to five chromosomes in cross AvS × PI 182103.

at Pullman than at Mt. Vernon, and reduced IT more than rAUDPC at Pullman but reduced rAUDPC more than IT at Mt. Vernon.

QyrPI182103.wgp-4DL, close to SSR marker *Xbarc288* and SNP marker *IWA4044*, was detected only in the tests at Mt. Vernon. It was significant for rAUDPC in 2012, explaining 11.8% of the variation, and almost significant (LOD 2.43) for rAUDPC in 2011, explaining 7.0% of the variation.

When the RILs were classified by presence of the closest markers for the three QTL, it was obvious that the effect of 7BL was greater than either 4DL or 5BS (Fig. 4). All three QTL showed mostly additive effects and RILs with more QTL had lower IT and rAUDPC. However, significant digenic epistatic interactions were observed. The combination of the 5BS and 7BL QTL resulted in high IT and rAUDPC than lines with only the 7BL QTL at Mt. Vernon, but a similar reduction in the 7BL QTL effect by the 5BS QTL was not observed at Pullman for both IT and rAUDPC. Similarly, RILs with both the 4DL and 7BL QTL had higher mean IT and rAUDPC values than RILs with only the 7BL QTL.

Physical map of *QyrPI182103.wgp-7BL*. Because *QyrPI182103.wgp-7BL* is a major QTL conferring HTAP resistance, experiments were conducted to determine its physical location on chromosome 7B. A complete set of 21 CS nulli-tetrasomic lines, two 7B ditelosomic lines, and eight 7BL deletion lines were amplified with all SSR markers linked to *QyrPI182103.wgp-7BL*. SSR markers *Xgwm333* and *Xwmc335* amplified the same bands as PI 182103 in CS. The target bands were not detected in CS N7BT7A and ditelosomic 7BS, indicating that *QyrPI182103.wgp-7BL* is located on chromosome 7BL. The targeted bands were not present in Del7BL-14 (0.14) and Del7BL-1 (0.40), but were present in the six other 7BL deletion lines, indicating that *QyrPI182103.wgp-7BL* was located in chromosome bin 7BL 0.40 to 0.45 (Fig. 5).

Relationships of *QyrPI182103.wgp-7BL* and other *Yr* genes on 7BL. Because *QyrPI182103.wgp-7BL* was located on chromosome 7BL, it was important to determine if it differed from other *Yr* genes on 7BL. Eight *Yr* genes are located on chromosome 7B, six named (*Yr2*, *Yr6*, *Yr39*, *Yr52*, *Yr59*, and *Yr67* [= *YrC59I*]) and two

Fig. 3. Quantitative trait loci mapping of *QyrPI182103.wgp-7BL* for resistance to stripe rust on chromosome 7BL based on infection type (IT) and relative area under the disease progress curve (rAUDPC) data in different environments (location/year). The numbers on the left side of the linkage map are genetic distances (cM). Markers with prefix of X are simple sequence repeat markers and those with IWA are single nucleotide polymorphism markers.

TABLE 4. Flanking markers, map positions, logarithm of odds (LOD), explained phenotypic variation (R^2), and additive effects (AE) of quantitative trait loci (QTL) for all stage resistance to stripe rust detected in recombinant inbred lines from AvS $\times$ PI182103 tested with *Puccinia striiformis* f. sp. *tritici* races of PST-100 and PST-114 under controlled conditions

QTL	Flanking markers		Position (cM)	Race	LOD	R^2 (%)	AE
	Distal	Proximal					
<i>QYrPI182103.wgp-2AS</i>	<i>IWA5585</i>	<i>Xgwm312</i>	95	PST-100	3.6	8.2	-0.7
				PST-114	2.0	5.0	-0.5
<i>QYrPI182103.wgp-3AL</i>	<i>IWA899</i>	<i>Xgwm2</i>	23	PST-100	3.4	8.1	-0.7
				PST-114	2.2	5.4	-0.6
<i>QYrPI182103.wgp-5BS</i>	<i>Xgwm540</i>	<i>Xgwm213</i>	59	PST-100	6.5	14.2	-0.9
				PST-114	3.3	8.2	-0.7

tentatively named (*YrZH84* and *yrMY37*). To determine the relationships of *QyrPI182103.wgp-7BL* with these *Yr* genes, wheat genotypes with genes on 7BL, except a line with *yrMY37* which was not available, were tested with PST-78, PST-100, PST-116, and

Fig. 4. Means of infection types and relative area under the disease progress curve (RAUDPC) values for the parents and F_7 recombinant inbred lines from cross AvS $\times$ PI 182103 with none, one, two, or three quantitative trait loci for resistance to stripe rust on chromosomes 4DL, 5BS, and/or 7BL.

PST-127 in both seedling LT (4 to 20°C) and adult-plant HT (10 to 30°C) conditions, together with PI 182103, an F_8 RIL (AvS/PI182103 F8-226) and AvS (Table 5). PI 182103 was highly resistant (IT 2) to PST-78, PST-100, and PST-116 in the seedling tests, but gave an intermediate reaction (IT 5) to PST-127. PI 189747 (C591) with *Yr67* (*YrC591*) was highly resistant (IT 2) to PST-78, PST-100, and PST-127, but gave an intermediate reaction (IT 4) to PST-116. All other lines including AvS/PI182103 F8-226 (*QyrPI182103.wgp-7BL*), Kalyansona (*Yr2*), AvSYr6NIL (*Yr6*), Alpowa (*Yr39*), PI 183527 (*Yr52*), PI 178759 (with *Yr59*), Zhou 8425B (with *YrZH84*), and AvS were highly susceptible (IT 8) to all four races. In the adult-plant tests with races PST-100 and PST-127, Kalyansona (*Yr2*) and AvSYr6NIL (*Yr6*) were susceptible under both LT (4 to 20°C) and HT (10 to 30°C) conditions. These two genes for all-stage resistance were not effective against any of the tested four races (Table 5). In the LT (4 to 20°C) tests, adult plants of Alpowa (*Yr39*), PI 183527 (*Yr52*), PI 178759 (*Yr59*), and Zhou 8425B (*YrZH84*) were susceptible (IT 8), whereas PI 182103 and PI 189747 (*Yr67*) were resistant (IT 2 or 3) and AvS/PI182103 F8-226 was intermediate (IT 5). In the HT test, all of these lines were resistant (IT 2 or 3) with various levels of severity. The differential reaction patterns to different races at the seedling and adult plant stages and LT versus HT comparisons suggested that the QTL on chromosome 7BL in PI 182103 and the selected F_8 line was different in specificity from the other genes on this chromosome.

Allelism tests to verify the relationships of *QyrPI182103.wgp-7BL* and the other genes were conducted using F_2 populations from

Fig. 5. Comparisons of the linkage map of *QyrPI182103.wgp-7BL* with the consensus map (Somers et al. 2004), deletion map (Sourdille et al. 2004), and those of *Yr2* (Lin et al. 2004), *Yr6* (Li and Niu 2007), *Yr39* (Lin and Chen 2007), *yrMY37* (Ren et al. 2015), *YrC591* (Li et al. 2009), *YrZH84* (Li et al. 2006), *Yr52* (Ren et al. 2012a), *Yr59* (Zhou et al. 2014), and *Yr67* (Xu et al. 2014) (R. McIntosh, personal communication) on chromosome 7BL. Dotted lines connect the same markers in different linkage maps.

crosses of PI 182103 with Alpowa (*Yr39*), PI 183527 (*Yr52*), PI 178759 (*Yr59*), PI 189747 (*Yr67*), and Zhou 8425B (*YrZH84*) in a field at Pullman under naturally occurring disease in 2013. All crosses segregated for susceptible plants and the estimated genetic distances ranged from 5.84 cM between *QyrPI182103.wgp-7BL* and *Yr59* to 9.57 cM between *QyrPI182103.wgp-7BL* and *Yr39* (Table 6). The responses of all susceptible and selected resistant F₂ plants were confirmed by progeny testing in the field in 2014. Based on the results of these tests and locations of common markers in linkage maps for these genes (Fig. 5); the possible order of the genes is: *Yr39* – 9.57 cM – *QyrPI182103.wgp-7BL* – 5.84 cM – *Yr59* – *Yr52* – *YrZh84* – *Yr67*. The type of resistance and genetic distances from the allelism tests provide evidence that *QyrPI182103.wgp-7BL* is different from previously reported *Yr* genes on chromosome 7BL.

Polymorphisms of molecular markers flanking *QyrPI182103.wgp-7BL*. Seventy-two wheat cultivars and breeding lines from the U.S. Pacific Northwest were tested for polymorphism to determine the value of the SSR markers *Xbarc72* and *Xgwm335* flanking *QyrPI182103.wgp-7BL* for marker-assisted selection (Supplementary Table S4). The polymorphism rates were 86.1% for *Xbarc72* and 93.1% for *Xgwm335*. No line had both markers, suggesting that the markers can reliably be used in combination for marker-assisted selection.

DISCUSSION

We confirmed that wheat accession PI 182103 has both race-specific all-stage resistance and HTAP resistance. We identified three QTL, *QyrPI182103.wgp-2AS*, *QyrPI182103.wgp-3AL*, and *QyrPI182103.wgp-5BS*, in greenhouse seedling tests, but only *QyrPI182103.wgp-5BS* was also detected in field tests. Two more QTL, *QyrPI182103.wgp-4DL* and *QyrPI182103.wgp-7BL*, were

detected only at the adult-plant stage in the field tests. Thus, a total of five QTL were identified in the mapping population of AvS × PI 182103 and all were from PI 182103. *QyrPI182103.wgp-2AS*, *QyrPI182103.wgp-3AL*, and *QyrPI182103.wgp-5BS* confer race-specific all-stage resistance, whereas *QyrPI182103.wgp-7BL* clearly controlled HTAP resistance. *QyrPI182103.wgp-4DL* is a race-specific gene for either all-stage resistance or adult-plant resistance.

In both seedling tests in the greenhouse and adult-plant tests in the fields, different QTL generally exhibited additive effects. RILs with all three QTL (2AS, 3AL, and 5BS) detected in seedlings had low IT similar to PI 182103 and those with one or two QTL had higher ITs than the resistant parent. Similarly, RILs with 4DL, 5BS, and 7BL QTL had the highest level of resistance among all RILs, and gave similar reactions to PI 182103 (Fig. 4). However, RILs with 5BS and 7BL had significantly higher ITs than those with only the 7BL QTL in the Mt. Vernon experiments. Although not significant, the same trend was also observed for rAUDPC at the same location. Such a negative interaction between different QTL was not observed in the Pullman field tests, suggesting that the negative interaction between the two QTL was influenced by either different environments or *P. striiformis* f. sp. *tritici* races. As the 5BS QTL was race-specific, its effectiveness might be related to mixed pathogen populations. The 4DL QTL was detected only in the field tests at Mt. Vernon, and was not effective in tests with races PST-100 and PST-114 in the seedling greenhouse tests and in the field tests at Pullman. This gene could be race-specific and influenced by environment.

The three QTL detected in seedling tests together were effective against races PST-100 and PST-114. The former has been widely distributed throughout the United States since 2003 and the latter was predominant in the U.S. Pacific Northwest from 2005 to 2009 (Chen et al. 2010; Wan and Chen 2012). The QTL on 2AS and 3AL

TABLE 5. Stripe rust reactions of wheat genotypes with different *Yr* genes on chromosome 7BL to different *Puccinia striiformis* f. sp. *tritici* races under controlled conditions

Genotype	<i>Yr</i> gene	Seedling tests with different races at LT ^a				Adult-plant tests with PST-127 (PST-100) ^b			
		IT ^c				LT ^a		HT ^a	
		PST-78	PST-100	PST-116	PST-127	IT ^c	DS ^c	IT ^c	DS ^c
PI 182103	<i>QYrPI182103.wgp.7BL</i> +	2	2	2	5	2	15	2	5
AvS/PI182103F ₈ -226	<i>QYrPI182103.wgp.7BL</i>	8	8	8	8	5	70	2	25
Kalyansona	<i>Yr2</i>	8	8	8	8	8	40	8	45
AvSYr6NIL	<i>Yr6</i>	8	8	8	8	8	75	8	55
Alpowa	<i>Yr39</i>	8	8	8	8	8	45	3	35
PI 183527	<i>Yr52</i>	8	8	8	8	8	15	2	15
PI 178759	<i>Yr59</i>	8	8	8	8	8	15	2	20
PI 189747 (C591)	<i>Yr67</i> (<i>YrC591</i>)	2	2	4	2	3	25	2	15
Zhou 8425B	<i>YrZH84</i>	8	8	8	8	8	40	2	10
AvS (susceptible check)	None	8	8	8	8	8	85	8	95

^a LT, low-temperature cycle (4 to 20°C); HT, high-temperature cycle (10 to 30°C).

^b Adult-plant tests of these lines with race PST-100 at both LT and HT produced similar results to those tested with PST-127.

^c IT, infection type; and DS, disease severity (percentage of leaf areas infected).

TABLE 6. Observed numbers of stripe rust resistant and susceptible F₁ and F₂ plant crosses of PI 182103 (*QYrPI182103.wgp-7BL*) and wheat lines with *Yr* genes on chromosome 7BL tested in the field at Pullman under natural infection

Cross (P1 × P2)	Gene in P2	P1	P2	F ₁	F ₂ plants		Genetic distance (cM) ^b
					Resistant	Susceptible ^a	
PI 182103 × Alpowa	<i>Yr39</i>	2	4	2	377	14	9.6 ± 1.5
PI 182103 × PI 183527	<i>Yr52</i>	2	2	2	267	4	6.1 ± 1.5
PI 182103 × PI 178759	<i>Yr59</i>	2	2	2	292	4	5.8 ± 1.4
PI 182103 × PI 189747	<i>Yr67</i>	2	2	2	178	4	7.5 ± 2.0
PI 182103 × Zhou 8425B	<i>YrZH84</i>	2	2	2	184	3	6.4 ± 1.8

^a Susceptibility was confirmed by progeny testing.

^b Genetic distance (cM) $d = 1/4 \ln[(1 + 2p)/(1 - 2p)] \times 100$, where $p = \sqrt{(f/4)}$ and f = the frequency of susceptible plants, and standard deviation (SD) was calculated using the formula $SD = \sqrt{[d \times (1 - d)/n]}$, where n is the total number of F₂ plants (Kosambi 1944).

were not detected in any of the field experiments, indicating that they were not effective against at least some races occurring in the field. Thus, these two QTL are less useful in breeding programs.

QyrPII82103.wgp-2AS was mapped on chromosome 2AS. Bariana and McIntosh (1994) reported *Yr17* on chromosome 2AS, and Fang et al. (2011) mapped the gene to the same chromosomal arm. None of the markers in the *Yr17* and *QyrPII82103.wgp-2AS* maps were the same. Also as *Yr17* originated from *Aegilops ventricosa*, the 2AS gene in PI 182103 is not likely *Yr17*. Eriksen et al. (2004) mapped *Yr32* on chromosome 2AS with 17 markers including eight SSR markers. However, none of the SSR markers were polymorphic in the PI 182103 mapping population, indicating that *QyrPII82103.wgp-2AS* is probably different from *Yr32*. Hao et al. (2011) mapped *YrR61* on chromosome 2AS in line 26R61 with 31 markers. This QTL, defined by SSR markers *Xbarc124* and *Xgwm359*, accounted for 56% of the phenotypic variance. SSR markers *Xbarc15* and *Xgwm312* are common to both *QyrPII82103.wgp-2AS* map and the *YrR61* map. However, these markers were about 37 and 61 cM away from *YrR61*, respectively, whereas *Xgwm312* is in the middle of the 2AS QTL region of PI 182103. Thus, these two genes are likely different. Chhuneja et al. (2008) mapped *QYrta.pau-2A* on chromosome 2AS in *T. monoccoccum* accession pau14087. This QTL, accounting for 14.0% of the phenotypic variance, is defined by flanking markers *Xwmc407* and *Xwmc170*. This QTL map of Chhuneja et al. (2008) does not share any markers with the *QyrPII82103.wgp-2AS* map in the present study, indicating that the two QTL are likely different. Recently, two genes, *Yr56* (*QYr.sun-2A*) for adult-plant resistance to stripe rust in wheat line AUS 91465 and Wollaroi durum and *Yr69* in line CH7085 were mapped on chromosome 2AS (R. McIntosh, *personal communication*). *Yr56* is about 11.5 cM from SSR marker *Xbarc212* and 20.6 cM from *Xgwm512*, and *Yr69* is between SSR markers *Xwmc25* and *Xmag3807* and about 30 cM from *Yr17*. However, these markers were not polymorphic in the PI 182103 mapping population. Thus, *QyrPII82103.wgp-2AS* is likely different from *Yr56* and *Yr69*.

QyrPII82103.wgp-3AL was mapped between IWA899 and *Xgwm2* on chromosome 3AL. Chen et al. (1995b) reported a temporarily named gene, *YrTr2* on chromosome 3A in winter club wheat Tres based on monosomic analysis, but the within chromosome location was not identified. Lillemo et al. (2008) identified a QTL close to SSR markers *Xbarc310* and *Xbarc321* on chromosome 3AS in Saar. So far, no permanently named *Yr* gene has been mapped on chromosome 3A, but four QTL, *QYrst.orr-3AL_Stephens* in a U.S. winter wheat cultivar Stephens (Vazquez et al. 2012), *QYr.cau-3AL_AQ24788-83* in wheat line AQ24788-83 (Quan et al. 2013), *QYrdr.wgp-3AL_IWA6834* (Hou et al. 2015), and *QYr.cim-3A_Avocet* (Rosewarne et al. 2012) were mapped to chromosome 3AL. The relationships of these 3AL QTL need further study.

QyrPII82103.wgp-4DL was delimited by the marker interval 3.3 cM *Xbarc288-IWA4044* in chromosome 4DL. Herrera-Foessel et al. (2011) mapped adult-plant resistance gene *Yr46* on chromosome 4DL, close to markers *Xgwm165* and *Xgwm192* in wheat line RL6077. Ren et al. (2012c) mapped *QYr.caas-4DL* on chromosome 4DL. This QTL, associated with flanking markers *Xwmc331* and *Xgwm165*, accounted for 8.0% of the phenotypic variation. Mallard et al. (2005) mapped *QYr.caas-4DL* flanked by markers *Xgwm382a* accounting for 3.9-8.0% of the phenotypic variation in a Camp Remy cross. Suenaga et al. (2003) mapped *QYr.jirc-4DL* associated with marker *Xwmc399* on chromosome 4DL in Oligoculm. Because the linkage maps of *QYr.jirc-4DL* and *QyrPII82103.wgp-4DL* do not share common markers, their relationship could not be determined, but they are likely to be different. As *QyrPII82103.wgp-4DL* was detected only in the Mt. Vernon tests, it may be race-specific or influenced by specific environments.

QyrPII82103.wgp-5BS was mapped on chromosome 5BS. Although numerous stripe rust resistance genes or QTL have been reported on chromosome 5BL (Chen 2013; McIntosh et al. 2013),

few genes were reported on 5BS. Chen et al. (1995a) located *Yr19* for all-stage resistance on chromosome 5B in Compair through monosomic analysis. No molecular markers were identified for this gene, and the arm location in 5B is not clear. Bansal et al. (2011) mapped *Yr47* in an ~20 cM region between SSR markers *Xgwm234* and *Xcfd309* in chromosome 5BS in wheat line AUS28183. Because these two markers were not detected in the present study, the relationship of *QyrPII82103.wgp-5BS* and *Yr47* could not be estimated and further work is needed to determine if these two genes are different.

QyrPII82103.wgp-7BL is the most important among the five QTL identified in the present study because it was consistently detected in field tests at two locations over 3 years. Several permanently or tentatively named *Yr* genes on chromosome 7B have been reported, including *Yr2* (Lin et al. 2004), *Yr6* (Li and Niu 2007), *Yr39* (Lin and Chen 2007), *Yr52* (Ren et al. 2012a), *Yr59* (Zhou et al. 2014), *Yr67* (*YrC591*) (Li et al. 2009; Xu et al. 2014) (R. McIntosh, *personal communication*), *YrZH84* (Li et al. 2006), and *yrMY37* (Ren et al. 2015). To determine the relationships of *QyrPII82103.wgp-7BL* and other *Yr* genes on chromosome 7B, we compared the resistance spectra and types of these *Yr* genes. *QyrPII82103.wgp-7BL* is different from *Yr2* and *Yr6* by its HTAP response. *Yr2* and *Yr6* are race-specific and ineffective not only to the races used in this study, but also to most other races identified in the United States (Wang and Chen 2017). Through tests of allelism, we found that *QyrPII82103.wgp-7BL* was a locus different from *Yr39*, *Yr52*, *Yr59*, *Yr67*, and *YrZH84*, all of which except *Yr67* confer HTAP. Our results confirmed these genes on chromosome 7BL and demonstrated that they were linked in repulsion to *QyrPII82103.wgp-7BL*. Ren et al. (2015) mapped stripe rust resistance gene *yrMY37* on chromosome 7BL in Chinese cultivar Mianyang 37. Because this gene confers seedling resistance, it should be different from *QyrPII82103.wgp-7BL* that confers HTAP resistance. Based on the chromosomal locations of these genes (Fig. 5), the *Yr* genes on chromosome 7BL can be separated into two groups. The first group consisting of *Yr2*, *Yr6*, *Yr39*, *yrMY37*, and *QyrPII82103.wgp-7BL* is clustered in a more proximal chromosome region between SSR markers *Xbarc72* and *Xgwm517* (Somers et al. 2004). The second group consisting of *Yr67* (*YrC591*), *YrZH84*, *Yr52*, and *Yr59* are clustered in a more distal region between SSR markers *Xgwm577* and *Xwmc526*. As suitable markers are not available for *Yr2* and *Yr6*, and seed of Mianyang 37 was not available, further studies are needed to determine the distances between *QyrPII82103.wgp-7BL* and these genes. As *Yr2*, *Yr6*, and *yrMY37* confer all-stage resistance they should be different from the HTAP resistance gene *QyrPII82103.wgp-7BL*.

Imtiaz et al. (2004) mapped *QYr.nsw-7B* linked to SSR marker *Xgwm611* on chromosome 7BL, explaining 26% of phenotypic variation. Rosewarne et al. (2008) mapped *QYr.csiro-7BL* flanked by markers *XP32/M59* and *Xgwm344* on chromosome 7BL in common wheat Attila. Yao et al. (2009) reported *QYr.caas-7B.1* in wheat cultivar Neixiang 188 flanked by markers *wPt2994* and *wPt1856* and accounting for 6.4% of the phenotypic variation. Ren et al. (2012b) detected *QYr.caas-7BL.1* and *QYr.caas-7BL.2* in wheat line Shanghai3/Catbird. The QTL, defined by markers *XwPt-8106-Xbarc176* and *Xgwm577-XwPt-4300*, explained 3.7 to 9.1% and 14.7% of the phenotypic variation, respectively. Comparing chromosome positions and phenotypic variations, *QyrPII82103.wgp-7BL* differs from all these QTL.

As discussed above, chromosome 7BL harbors a significant number of genes or QTL conferring different types and levels of resistance to stripe rust. Without more definitive seed stocks it will be extremely difficult to determine the genetic relationships of these genes. However, we show that *QyrPII82103.wgp-7BL* is different from the present permanently named *Yr* genes. Therefore, we name *QyrPII82103.wgp-7BL* as *Yr79*. An F₁₀ line carrying only *Yr79* for HTAP resistance was deposited in the United States National Small Grains Collection and registered as PI 679609. This line gave IT 5 at

Pullman in both 2015 (20% severity) and 2016 (30% severity) and IT 4 with 40% severity at Mt. Vernon in both 2015 and 2016, in contrast to IT 8 and 95 to 100% severities for AvS (data not shown). Because HTAP resistances are usually durable (Chen 2013; Line 2002), *Yr79* will be a valuable new gene for use in breeding for durable stripe rust resistance. Polymorphic markers tightly linked to this gene should be useful for marker-assisted selection.

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