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ORIGINAL PAPER

Genetics and molecular mapping of genes for race-specific all-stage resistance and non-race-specific high-temperature adult-plant resistance to stripe rust in spring wheat cultivar Alpowa

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Abstract Stripe rust, caused by *Puccinia striiformis* f. sp. *tritici*, is one of the most widespread and destructive wheat diseases worldwide. Growing resistant cultivars is the preferred control of the disease. The spring wheat cultivar 'Alpowa' has both race-specific, all-stage resistance and non-race-specific, high-temperature adult-plant (HTAP) resistances to stripe rust. To identify genes for the stripe rust resistances, Alpowa was crossed with 'Avocet Susceptible' (AVS). Seedlings of the parents, and F_1, F_2 and F_3 progeny were tested with races PST-1 and PST-21 of *P. striiformis* f. sp. *tritici* under controlled greenhouse conditions. Alpowa has a single partially dominant gene, designated as *YrAlp*, conferring all-stage resistance. Resistance gene analog polymorphism (RGAP) and simple sequence repeat (SSR) techniques were used to identify molecular markers linked to *YrAlp*. A linkage group of five RGAP markers and two SSR markers was constructed for *YrAlp* using 136 F_3 lines. Amplification of a set of nulli-tetrasomic Chinese Spring lines with RGAP markers *Xwgp47* and *Xwgp48* and the two SSR markers indicated that *YrAlp* is located on the short arm of chromosome 1B. To map quantitative trait loci (QTLs) for the non-race-specific HTAP resistance, the parents and 136 F_3 lines were tested at two sites near Pullman and one site near Mount Vernon, Washington, under naturally infected conditions. A major HTAP QTL was consistently detected across environments and was located on chromosome 7BL. Because of its chromosomal location and the non-race-specific nature of the HTAP resistance, this gene is different from previously described genes for adult-plant resistance, and is therefore designated *Yr39*. The gene contributed to 64.2% of the total variation of relative area under disease progress curve (AUDPC) data and 59.1% of the total variation of infection type data recorded at the heading-flowering stages. Two RGAP markers, *Xwgp36* and *Xwgp45* with the highest R^2 values were closely linked to *Yr39*, should be useful for incorporation of the non-race-specific resistance gene into new cultivars and for combining *Yr39* with other genes for durable and high-level resistance.

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Introduction

Stripe rust (yellow rust), caused by *Puccinia striiformis* Westend. f. sp. *tritici* Eriks., is a major disease that causes substantial losses to wheat production worldwide (Stubbs 1985; Chen et al. 2002; Wan et al. 2004; Chen 2005). The use of resistant cultivars is the most effective, economical, and environmentally friendly means to control stripe rust. Race-specific and non-race-specific resistance are two major types of resistance to wheat stripe rust. All-stage resistance (called

seedling resistance) is generally race-specific and qualitatively inherited, whereas high-temperature, adult-plant (HTAP) resistance is non-race-specific, durable, and is often quantitatively inherited (Qayoum and Line 1985; Chen and Line 1995a, 1995b; Line and Chen 1995; Line 2002; Chen 2005).

Numerous mainly race-specific resistance genes, designated *Yr*, conferring resistance to stripe rust have been identified (McIntosh et al. 1998, 1999, 2001; Chen 2005). A few genes conferring non-race-specific HTAP resistance have been identified (Chen and Line 1995a, 1995b; Chen 2005; Uauy et al. 2005; Chicaiza et al. 2006). Development of molecular markers has revolutionized genetic analysis of quantitative disease resistance. Quantitative trait loci (QTLs) analysis in wheat has been partially limited because of the large genome size and numerous repeated sequences. Bulk segregant analysis (BSA) (Michelmore et al. 1991) is a quick method to identify molecular markers closely linked to genes of interest. Degenerate primers derived from conserved motifs of cloned resistance genes such as leucine-rich repeats (LRR), nucleotide-binding sites (NBS) and serine/threonine kinase domains have been used to develop molecular markers known as resistance gene analogs (RGAs) (Leister et al. 1996; Kanazin et al. 1996; Yu et al. 1996; Feuillet et al. 1997). Chen et al. (1998) improved the RGA approach by using high-resolution polyacrylamide gel electrophoresis and sensitive silver staining referred to as resistance gene analog polymorphism (RGAP). This technique was used for mapping genes for resistance to various diseases including stripe rust (Chen et al. 1999; Toojinda et al. 2000; Shi et al. 2001; Yan et al. 2003; Pahalawatta and Chen 2005a, 2005b).

Many wheat cultivars in the Pacific Northwest (PNW) of the United States have combinations of HTAP and all-stage resistances to stripe rust (Allan et al. 1966; Qayoum and Line 1985; Line and Chen 1995; Line 2002; Chen 2005). 'Alpowa' (PI 566596), a soft white spring wheat cultivar developed by Dr. C. Konzak from the cross Fielder/Potam 70/2/Walladay/3/Walladay/Potam 70 and released in 1994 (http://www.laughlintrading.com/extended%20variety%20descriptions/Alpowa.htm), was reported to carry HTAP resistance to stripe rust (Chen et al. 2003). Partly due to its durable HTAP resistance to stripe rust, Alpowa is the most widely grown spring wheat cultivar in the western United States and the leading spring wheat cultivar in Washington in 2003, 2004 and 2005, where it occupied 39.9, 39.6 and 42.7% of the area of spring wheat, respectively (http://www.nass.usda.gov/wa/whtvar05.pdf). Despite the popularity of Alpowa, genes conferring stripe rust resistance in Alpowa were not determined. The objective of this study was to identify the genes conferring different types of stripe rust resistance in Alpowa through genetic analysis and molecular mapping.

Materials and methods

Plant material and cross

To study genetics of, and to develop molecular markers for, stripe rust resistance, 'Alpowa' was crossed with an Australian spring wheat 'Avocet Susceptible' (AVS) selection. F_1 and F_2 plants were grown in a greenhouse for seed production. F_1, F_2 and F_3 generations and parents were used in seedling tests in the greenhouse for identifying the gene(s) for all-stage resistance. The parents and the 136 F_3 lines that were used in the greenhouse seedling tests were used in field tests for determining their phenotypes of the HTAP response for molecular mapping genes of the all-stage and HTAP resistances.

Pathogen isolates and disease assessments

Greenhouse evaluation of seedlings for stripe rust resistance

Seedling tests were conducted under controlled greenhouse conditions. Fifteen *P. striiformis* f. sp. *tritici* races (PST-1, PST-3, PST-17, PST-18, PST-21, PST-23, PST-37, PST-43, PST-45, PST-59, PST-61, PST-78, PST-81, PST-98 and PST-100), which represent various spectra of virulences (Chen 2005), were chosen to test seedlings of Alpowa and AVS. Races that were avirulent on seedlings of Alpowa but virulent on AVS were selected to evaluate seedlings of the F_1, F_2 and F_3 populations.

Seed of the parents and F_1, F_2 and F_3 progenies were planted in plastic pots (5 × 5 × 5 cm) filled with a potting mixture of 6 peat moss:2 perlite:3 sand:3 potting soil mix:4 vermiculite with lime, Osmocote 14-14-14, and ammonium nitrate added at 1.7, 3.3, and 2.2 g/L, respectively. About 15 seeds for each parent, 3 seeds for F_1, 300 seeds for F_2, and 15 seeds for each of the 136 F_3 lines were planted with about 15 seeds in each pot except for the F_1.

Seedlings at the two-leaf stage (about 10 days after planting) grown in a rust-free greenhouse (diurnal temperature cycle gradually change from 10°C at 2:00 am to 25°C at 2:00 pm with the 16 h light/8 h dark cycle) were uniformly dusted with a mixture of urediniospores of the selected *P. striiformis* f. sp. *tritici* race and

talc (Sigma, St Louis, MO, USA) at a ratio of approximately 1:20. After inoculation, plants were placed in a dew chamber at 10°C for 24 h and then transferred to a growth chamber operating at 16 h light and 8 h dark with diurnal temperatures gradually changing from 4°C at 2:00 am to 20°C at 2:00 pm (Chen and Line 1992a, 1992b). A set of wheat genotypes used to differentiate races of *P. striiformis* f. sp. *tritici* was included in the tests to confirm the race identity. Infection type (IT) data were recorded 18–21 days after inoculation based on a 0–9 scale, in which 0 = no visible symptom, 1 = necrotic and/or chlorotic flecks without sporulation, 2 = necrotic and/or chlorotic blotches or stripes without sporulation, 3 = necrotic and/or chlorotic blotches or stripes with trace sporulation, 7 = necrotic and/or chlorotic blotches or stripes with abundant sporulation, 8 = chlorosis behind the abundantly sporulating area, and 9 = abundant sporulation with no chlorosis or necrosis (Line and Qayoum 1992).

Field evaluation of adult-plants for stripe rust resistance

During the 2005 crop season, the parents and 136 F_3 lines were grown at two field sites near Pullman in eastern Washington and one site near Mt. Vernon in western Washington to evaluate stripe rust severity and infection type under naturally infected conditions. The two sites near Pullman were about 4 km away from each other with similar weather conditions. The Mt. Vernon site was different in environment and race composition. The nurseries were planted on 7 April and 10 April, 2005 at sites 1 and 2 of Pullman and 22 April at Mt. Vernon. At each site, about 30 seeds for each of 136 F_3 lines and the parents were planted in 1-m rows planted 20 cm apart. Each plot was surrounded by susceptible spring wheat 'Lemhi' as a spreader. Weeds were controlled by application of herbicides in the early growth stages and by mechanical weeding in the late stages. Because of limited seed quantities, replications were not possible at each site, and three sites were preferred in order to assess the mapping population under different environmental conditions and different pathogen race compositions, as well as to reduce the risk of failure of a natural epidemic at any single site.

Infection type and rust severity were recorded at boot, heading-flowering, and dough growth stages, when the severity on 'AVS' had reached approximately 30, 60 and 95%, respectively. A 0–9 scale for infection type similar to that described for the seedling tests was used and disease severity of each line was recorded as an average of all plants within each row. Data on rust severity were recorded in percentages 0, 2, 5, 10, 15, 20, 30, 40, 50, 60, 70, 80, 90 and 100.

PCR amplification, electrophoresis, silver staining and photography

Genomic DNA was isolated from more than 20 plants of two-leaf seedlings for each of the parents and F_3 lines using the cetyltrimethyl ammonium bromide (CTAB) method (Saghai-Maroof et al. 1984). The RGAP method described previously (Chen et al. 1998; Shi et al. 2001), and the SSR technique described by Roder et al. (1998) were followed. The RGA and SSR primers used in this study are listed in Table 1. PCRs were performed in a GeneAmp® PCR System 9700 Thermo-cycler. The 15 µl reaction mixtures consisted of 30 ng of template DNA, 1.5 µl Mg-free 10× PCR Buffer (Promega, Madison, WI), 0.6 unit of Taq DNA polymerase (Promega, Madison, WI), 5 mM of $MgCl_2$, 0.2 mM each of dCTP, dGTP, dTTP and dATP (Sigma Chemical Co., St. Louis, MO), and 30 ng of a single primer synthesized by Operon Biotechnologies, Inc. After 5 min of denaturation at 94°C, amplifications were programmed for 40 consecutive cycles each consisting of 1 min at 94°C, 1 min at either 45, 50, 55 or 60°C (45°C for RGA primers, 50, 55 or 60°C for SSR primers depending on the individual primers), 2 min at 72°C and followed by a 7 min extension step at 72°C. After amplification, 6 µl of formamide loading buffer [98% formamide, 10 mM EDTA (pH 8.0), 0.5% (W/V) xylene cyanol, and 0.5% (W/V) bromophenol blue] was added to the PCR products followed by another 4 min denaturation at 94°C for electrophoresis in 5% polyacrylamide gels. After electrophoresis, the gel was silver-stained according to the recommendation of the Promega manufacturer and produced using a silver-sequence automatic processor-compatible film (Promega, Madison, WI).

Bulk segregant analysis

Based on phenotypic data, two sets of bulks (one for all-stage resistance and one for HTAP resistance) were constructed to detect putative resistance-related markers. For each component assessed, five most resistant and five most susceptible F_3 lines were chosen to construct the bulks by mixing equal amounts of DNA from each selected line. A total of 528 RGA primer pairs were first screened on the parents and two sets of bulks. Primer pairs showing specific bands to both Alpowa and the resistant bulks, or AVS and the susceptible bulks, were used to genotype the F_3 population. Polymorphic markers tested with all 136 F_3 lines were scored and used to construct linkage maps.

Table 1 Resistance gene analog (RGA) and SSR primers used to identify markers for stripe rust resistance in Alpowa

Primer[a]	Sequence (5'–3')	Gene	Domain	Reference
AS1-INV	CCTAACGGTGATCGCAAC	*N, Rps2*	P-loop	This study
AS3-INV	CCIGAIGGIGAICGIG	*N, Rps2*	LRR	Yan et al. (2003)
CLRR For	TTTTCGTGTTCAACGACG	*Cf9*	LRR	Chen et al. (1998)[a]
LM637	ARIGCTARIGGIARICC	*L6, N, Rps2*	P-loop	Kanazin et al. (1996)
LM638	GGIGGIGTIGGIAAIACIAC	*L6, N, Rps2*	P-loop	Kanazin et al. (1996)
NLRR For	CGCAACCACTAGAGTAAC	*N*	LRR	This study
NLRR Rev	TATAAAAAGTGCCGGACT	*N*	LRR	Chen et al. (1998)[a]
NLRR-INV1	TGCTACGTTCTCCGGG	*N*	LRR	Yan et al. (2003)
NLRR-INV2	TCAGGCCGTGAAAAATAT	*N*	LRR	This study
Pto kin1	GCATTGGAACAAGGTGAA	*Pto*	Kinase	Chen et al. (1998)[a]
Pto kin2	AGGGGGACCACCACGTAG	*Pto*	Kinase	Chen et al. (1998)[a]
Pto kin3	TAGTTCGGACGTTTACAT	*Pto*	Kinase	Pahalawatta and Chen (2005a)
Pto kin4	AGTGTCTTGTAGGGTATC	*Pto*	Kinase	Shi et al. (2001)
Pto kin1IN	AAGTGGAACAAGGTTACG	*Pto*	Kinase	Shi et al. (2001)
Pto kin2IN	GATGCACCACCAGGGGG	*Pto*	Kinase	Shi et al. (2001)
PtoFen-S	ATGGGAAGCAAGTATTCAAGGC	*Pto*	Fen	Pahalawatta and Chen (2005a)
PtoFen-AS	TTGGCACAAAATTCTCATCAAGC	*Pto*	Fen	Pahalawatta and Chen (2005b)
S2	GGIGGIGTIGGIAAIACIAC	*N, Rps2*	P-loop	Leister et al. (1996)
S2-INV	CAICAIAAIGGITGIGGIGG	*N, Rps2*	P-loop	Pahalawatta and Chen (2005a)
RLRR For	CGCAACCACTAGAGTAAC	*Rps2*	LRR	Chen et al. (1998)[a]
RLRR Rev	ACACTGGTCCATGAGGTT	*Rps2*	LRR	Chen et al. (1998)[a]
RLK-For	GAYGTNAARCCIGARAA	*LrK10*	Kinase	Feuillet et al. (1997)
Xa1NBS-R	CTCTGTATACGAGTTGTC	*Xal*	NBS	Shi et al. (2001)
XLRR For	CCGTTGGACAGGAAGGAG	*Xa21*	LRR	Chen et al. (1998)[a]
XLRR Rev	CCCATAGACCGGACTGTT	*Xa21*	LRR	Chen et al. (1998)[a]
Xa1LR-F	CTCACTCTCCTGAGAAAATTAC	*Xal*	LRR	This study
WMS11	L: GGATAGTCAGACAATTCTTGTG R:GTGAATTGTGTCTTGTATGCTTCC	–	–	Roder et al. (1998)
WMS18	L: TGGCGCCATGATTGCATTATCTTC R: GGTTGCTGAAGAACCTTATTTAGG	–	–	Roder et al. (1998)
WMS46	L: GCACGTGAATGGATTGGAC R: TGACCCAATAGTGGTGGTCA	–	–	Roder et al. (1998)
WMS131	L: AATCCCCACCGATTCTTCTC R: AGTTCGTGGGTCTCTGATGG	–	–	Roder et al. (1998)

[a] WMS11, WMS18, WMS46 and WMS131 are SSR primers and the remaining ones are RGA primers

Genetic analyses

For segregation analysis, χ^2 tests were used to analyze the inheritance of stripe rust resistance, and the polymorphic RGAP and SSR markers. The area under disease progress curve (AUDPC) was calculated for each F_3 line and the parents using all three sets of field data according to the formula: $\text{AUDPC} = \sum_i[(x_i + x_{i+1})/2]t_i$, where x_i is the severity value on date i, t_i the time in days between dates i and $i + 1$. A relative AUDPC value for each line was calculated as a percentage of AVS.

Broad-sense heritability (h^2) across the three locations was calculated using the formula $[V_{F3} - V_{p1} + V_{p2})/2]/V_{F3}$, with V_{F3}, for the F_3 population variance and V_{p1} and V_{p2} for the parental variances (Chen and Line 1995b). Phenotypic correlation coefficients of both relative AUDPC and infection type data of F_3 lines in three pair-wise combinations involving the three locations were calculated using MS Excel. The correlations between relative AUDPC and infection type data across the three locations were also calculated. Because the infection type data recorded at the heading-flowering stage best represented the HTAP resistance in the fields, this data set was used for most of the infection type analyses.

Linkage map construction and QTL analysis

Linkage maps were constructed using MAPMAKER MACINTOSH ver. 2.0 (Lander et al. 1987). Two-point analysis with a logarithm of the odds (LOD) threshold of 3.0 or greater was used to determine linkage relationships among markers whereas multipoint analysis was used for determining the best locus order in the linkage groups. Map distance (in centimorgans) was calculated according to the Kosambi mapping function (Kosambi 1944).

Computer program Qgene (Nelson 1997) was performed to scan the linkage groups for the presence

of HTAP resistance QTL based on both relative AUDPC and infection type data for F_3 lines in the individual nurseries separately, and also in combination.

Determination of chromosomal locations of molecular markers and linked resistance genes

The nulli-tetrasomic lines of 'Chinese Spring' (Sears 1966) were used to locate RGAP markers related to stripe rust resistance on wheat chromosomes and then the SSR markers from those specific chromosomes were screened to position the markers and linked resistance loci to specific chromosomal locations.

Results

Stripe rust resistance

Race-specific all-stage (seedling) resistance

In the seedling tests, AVS was susceptible (IT 8–9) to all 15 tested races while Alpowa was only resistant (IT 2) to PST-1 and PST-21. When tested with PST-1, all three F_1 seedlings had IT 3. Of 300 F_2 seedlings, 60 had IT 2, 153 had IT 3, and 87 had ITs 8 and 9. The segregation fit a 3 resistant (ITs 2 and 3):1 susceptible (IT 8) ratio ($\chi^2 = 4.0$, $P = 0.05$) or a 1 (IT 2):2 (IT 3):1 (ITs 8–9) ratio ($\chi^2 = 4.98$, $P = 0.08$), suggesting that a single partially dominant gene conferred resistance detected in the seedling stage. The F_3 population had 23 resistant, 80 segregating and 33 susceptible lines, fitting a 1:2:1 ratio ($\chi^2 = 5.71$, $P = 0.06$), and further confirmed that a single gene conferred resistance. Similar results were obtained in the tests with PST-21. The F_3 seedling also had IT 3. Of 240 F_2 plants, 46 had IT 2, 134 had IT 3, and 60 had ITs 8 and 9, and the segregation fit a 3 resistant (ITs 2 and 3) :1 susceptible ratio (ITs 8 and 9) ($\chi^2 = 2.56$, $P = 0.11$) or a 1 (IT 2):2 (IT 3):1 (ITs 8–9) ratio ($\chi^2 = 4.90$, $P = 0.09$). When we first tested F_1, F_2 and F_3 generations with PST-21, the 136 F_3 lines had almost the same ITs as those inoculated with PST-1. Only two lines, which had IT 8 on all plants when inoculated with PST-1, appeared segregating (i.e. five plants with IT 3, four plants with IT 5, and two plants with IT 8) when inoculated with PST-21. All 136 F_3 lines were retested with PST-21 and the results exactly matched those with PST-1. The results obtained from tests with both PST-1 and PST-21 indicated that the same gene in Alpowa confers resistance to both PST-1 and PST-21.

Table 2 Observed number of F_3 lines in AVS/Alpowa segregating in infection types (IT) produced by natural infection of *Puccinia striiformis* f. sp. *tritici* in field plots

Location	Note	Number of F_3 lines[a]			$\chi^2_{1:2:1}$	P
		Res IT 2, 3	Seg IT 2–9	Sus IT 7–9		
Tukey Farm	1st	33	65	38	0.63	0.73
	2nd	31	64	41	1.94	0.38
	3rd	33	58	45	5.06	0.08
OB Hill Farm	1st	35	69	32	0.16	0.92
	2nd	36	63	37	0.75	0.68
	3rd	35	62	39	1.29	0.52
Mt. Vernon	1st	33	64	39	1.00	0.61
	2nd	36	60	40	2.12	0.34
	3rd	32	60	44	4.00	0.14

[a] *Res* resistant, *Seg* segregating and *Sus* susceptible

HTAP resistance evaluated in the greenhouse and fields

Alpowa was tested, together with other wheat cultivars or breeding lines, in the greenhouse in seedling stage under the low diurnal temperature cycle and adult-plant stage under a high-diurnal temperature cycle. Races PST-100, PST-101 (Chen 2005), and PST-111 (Chen unpublished data) were used in both seedling and adult-plant tests. Alpowa was susceptible (IT 8) to the three races in the seedling tests, but was resistant to moderately resistant (IT 2–5) in the adult-plant tests, depending upon individual plants. In contrast, Lemhi, used a susceptible check, had IT 8 or 9 in all tests. The results indicated that Alpowa has HTAP resistance.

The susceptible parent, AVS, had IT 8 or 9 at all stages when the data were collected at all three field locations. In contrast, Alpowa had ITs 2 and 3 at tillering and IT 2 at boot and flowering data collections. These data agree with the data from germplasm plots over several years (data not shown). The IT data consistently indicated single gene inheritance (Table 2).

Both relative AUDPC and infection type data for the adjusted means of the F_3 lines across different locations and individual locations (data not shown) had a continuous distribution (Fig. 1), suggesting quantitative inheritance. The relative AUDPC value for AVS was treated as 100% for each location whereas the values for Alpowa were 27% for Pullman site one, 23% for Pullman site two and 20% for the Mt. Vernon site with an average value of 23%. As shown in Fig. 1a, the relative AUDPC of the F_3 lines averaged across the three locations varied from 21 to 114%.

Broad-sense heritability was calculated as 87%. For the relative AUDPCs of F_3 lines among the three locations, high correlations ($P < 0.01$) were found among all three locations (data not shown).

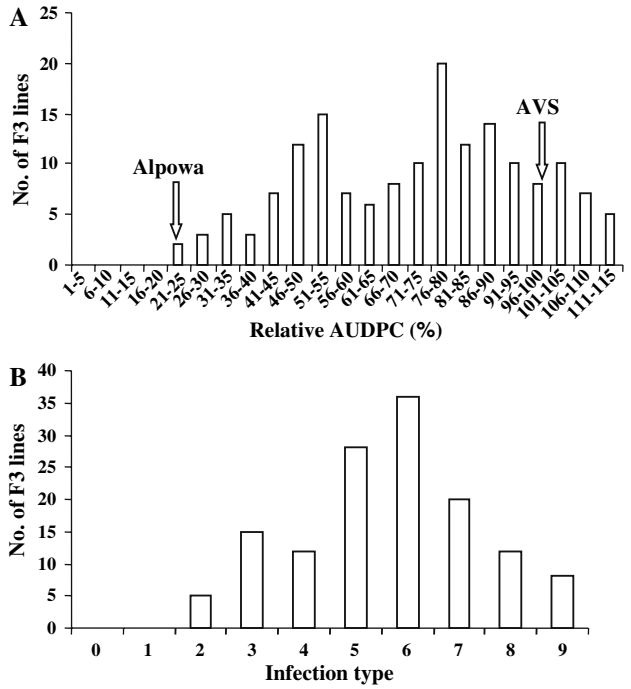

Fig. 1 Frequency distribution of (**a**) stripe rust relative AUDPCs averaged over three locations and (**b**) infection type collected at heading-flowering stage at site 1 near Pullman, WA, for 136 F_3 lines derived from a cross between 'AVS' and 'Alpowa'. Arrows indicate the values of the parental lines

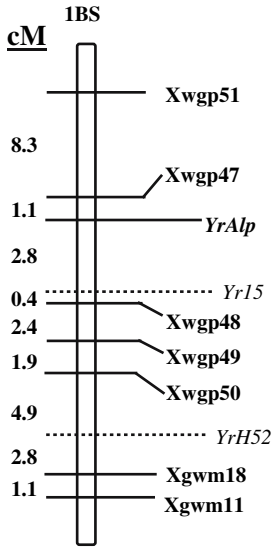

Fig. 2 A linkage map for *YrAlp* on chromosome 1BS based on the AVS/Alpowa mapping population of 136 F_3 lines. All markers were resistance gene analog polymorphism (RGAP) markers except for *Xgwm11* and *Xgwm18* that were simple sequence repeat (SSR) markers. All RGAP markers were present in Alpowa except for *Xwgp47* present in AVS

Relationship between all-stage (seedling) resistance and HTAP resistance in 'Alpowa'

The greenhouse seedling and field adult-plant IT data at the three sites fit a model of two independent genes ($df = 8$, χ^2 ranged from 9.21 to 13.4, and P value ranged from 0.1 to 0.3). These results indicated independence of the race-specific all-stage resistance detected in the seedling tests and the HTAP resistance detected in the field. The single gene for race-specific all-stage resistance was named *YrAlp* and that for HTAP resistance detected in the field, *Yr39*.

Molecular mapping of the all-stage resistance gene *YrAlp*

Of 528 RGA primer pairs screened for polymorphisms between the parental cultivars, 155 (29.8%) were polymorphic. The polymorphic RGA primer pairs were used to detect polymorphisms between resistant and susceptible bulks established for the all-stage resistance; 9 primer pairs generated polymorphisms. Segregations of the 9 RGA primer pairs were then determined for all 136 F_3 lines. Four polymorphic markers (*Xwgp48*, *49*, *50* and *51*) present in Alpowa and one marker (*Xwgp47*) in AVS were linked to the *YrAlp* locus at genetic distances ranging from 1.1 to 16.3 cM (Fig. 2).

Nulli-tetrasomic lines representing all 21 Chinese Spring chromosomes were tested with RGAP markers *Xwgp47* and *Xwgp48* to identify the critical chromosome. Marker *Xwgp47* gave about 1120-bp fragment in AVS and Chinese Spring and no fragment in Alpowa, whereas marker *Xwgp48* amplified about 450-bp fragment in Alpowa and Chinese Spring and none in AVS. All nulli-tetrasomic lines, except N1BT1A, showed the target band indicating that *YrAlp* is located on chromosome 1B. SSR primers from 1B were screened to confirm the chromosome location and region. *YrAlp* was located on 1B distally to *Xgwm18* (Fig. 2). All markers, including five RGAP markers and two SSR markers, segregated in 3:1 ratios (Table 3), indicating that these markers were reliable for constructing the linkage map. According to the high-density wheat SSR consensus wheat map (http://www.graingenes.org/cgi-bin/ace/pic/graingenes?name=Tturgidum-SSR-1B&class=Map), both *Xgwm11* (460 bp) and *Xgwm18* (400 bp) are located on 1BS, suggesting that *YrAlp* is on the short arm of 1B.

QTL mapping of *Yr39* for HTAP resistance on chromosome 7BL

A total of 25 primer pairs from the 155 polymorphic RGA primer pairs gave amplification differences between the resistant and susceptible bulks established for HTAP responses and were used to further analyze the 136 F_3 lines. Analyses of individual markers detected consistent and significant associations between 13 RGAP markers and the HTAP resistance

Table 3 RGAP and SSR markers linked to the all-stage resistance locus or the HTAP resistance QTL to stripe rust and their primer pairs, size, presence (+) and absence (−) in Alpowa, Avocet Susceptible (*AVS*) and Chinese Spring, number of F_3 lines of the AVS/Alpowa cross with or without the bands, and χ^2 and probability values for goodness of fit to a 3 presence:1 absence ratio

Marker[a]	Primer pair	Size (bp)	Presence (+) and absence (−)			No. of F_3 lines		Test for 3:1	
			Alpowa	AVS	CS	+	−	χ^2	*P*
Xwgp33	S2-INV/XLRR Rev	872	+	−	ND[b]	100	36	0.16	0.69
Xwgp34	NLRR Rev/XalNBS Rev	960	+	−	ND	97	39	0.98	0.32
Xwgp35	LM637/XalLR For	980	+	−	ND	97	39	0.98	0.32
Xwgp36	Pto kin1/RLK For	830	+	−	+	97	39	0.98	0.32
Xwgp37	Ptokin4/XLRR-R	910	+	−	ND	92	44	3.92	0.05
Xwgp38	LM638/S2-INV	870	+	−	ND	97	39	0.98	0.32
Xwgp39	S2/Pto kin1	450	+	−	ND	95	41	1.92	0.17
Xwgp40	CLRR For/NLRR-INV1	910	+	−	ND	97	39	0.98	0.32
Xwgp41	Pto kin2/Pto kin3	440	+	−	ND	93	43	3.18	0.07
Xwgp42	Pto kin1/PtoFen-AS	840	+	−	ND	92	44	3.92	0.05
Xwgp43	Pto kin1/toFen-S	820	+	−	ND	98	38	0.63	0.43
Xwgp44	S2-INV/NLRR-INV2	875	+	−	ND	98	38	0.63	0.43
Xwgp45	Pto kin1/XLRR For	940	+	−	ND	97	39	0.98	0.32
Xwgp46	Pto kin1/AS1-INV	390	−	+	+	98	38	0.63	0.43
Xwgp47	RLK For/Pto kin2IN	1120	−	+	+	98	38	0.63	0.43
Xwgp48	Pto kin1/Pto kin3	450	+	−	+	97	39	0.98	0.32
Xwgp49	Pto kin3/XLRR Rev	980	+	−	ND	94	42	2.51	0.11
Xwgp50	RLRR Rev/XalLR For	800	+	−	ND	99	37	0.35	0.55
Xwgp51	Pto kin1IN/Pto kin2IN	1110	+	−	ND	98	38	0.63	0.43
Xgwm11	WMS11	460	+	−	ND	96	40	1.41	0.23
Xgwm18	WMS18	400	+	−	ND	99	37	0.35	0.55
Xgwm43	WMS43	950	+	−	ND	99	37	0.35	0.55
Xgwm131	WMS131	180	+	−	ND	95	41	1.92	0.17

[a] All *Xwgp* markers were RGAP markers and *Xgwm* were SSR markers

[b] *ND* no data

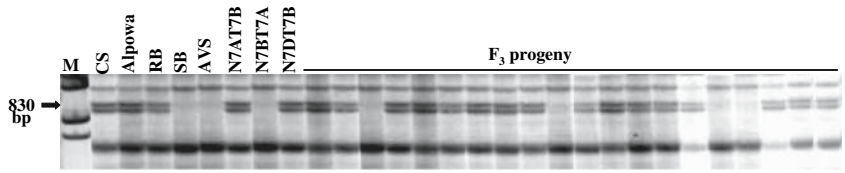

Fig. 3 A polyacrylamide gel showing a resistance gene analog polymorphism (*RGAP*) marker *Xwgp36*. *M* 100-bp DNA ladder. *CS* Chinese Spring. *AVS* 'Avocet Susceptible'. *RB* the resistant bulk and *SB* susceptible bulk of selected F_3 lines. *N7AT7B*, *N7BT7A* and *N7DT7B* are Chinese Spring nulli-tetrasomic lines. The F_3 generation was derived from the AVS/Alpowa cross

with data from each of the three locations and the combined data. As an example, Fig. 3 shows that marker *Xwgp36* was present in Alpowa and the resistant bulk, but not in AVS and the susceptible bulk, and segregated in the F_3 population. The 13 RGAP markers were mapped to one linkage group (Fig. 4). Marker *Xwgp46* (390 bp), present in AVS but not in Alpowa, also was mapped to this linkage group.

Analyses of nulli-tetrasomic Chinese Spring lines with markers *Xwgp36* and *Xwgp46* assigned the linkage group for *Yr39* to chromosome 7B (Fig. 3). Subsequently, eight SSR primers selected from 7B were used to screen the two parents and two bulks for polymorphisms. Two polymorphic SSR markers (*Xgwm43* and *Xgwm131*) were further analyzed in the F_3 population and they showed significant associations with the HTAP resistance (Fig. 4, Table 4). Because these two markers were reported on wheat chromosome 7BL (http:/www.graingenes.org/cgi-bin/ace/queryEdit/graingenes? arg1=WMS*&run=1&query=microsatellites&arg2=7B), the linkage group and therefore the *Yr39* HTAP resistance gene were located on 7BL. All of the markers showing significant associations with the HTAP resistance showed 3:1 ratios for dominant inheritance (Table 3). Together with marker *Xwgp46*, associated with susceptible allele, the genetic linkage group for the HTAP resistance gene consisted of 14 RGAP markers and 2 SSR markers and spanned 59.8 cM (Fig. 4). The primer pairs and fragment sizes of the markers in the *Yr39* linkage group, together with markers for the *YrAlp*

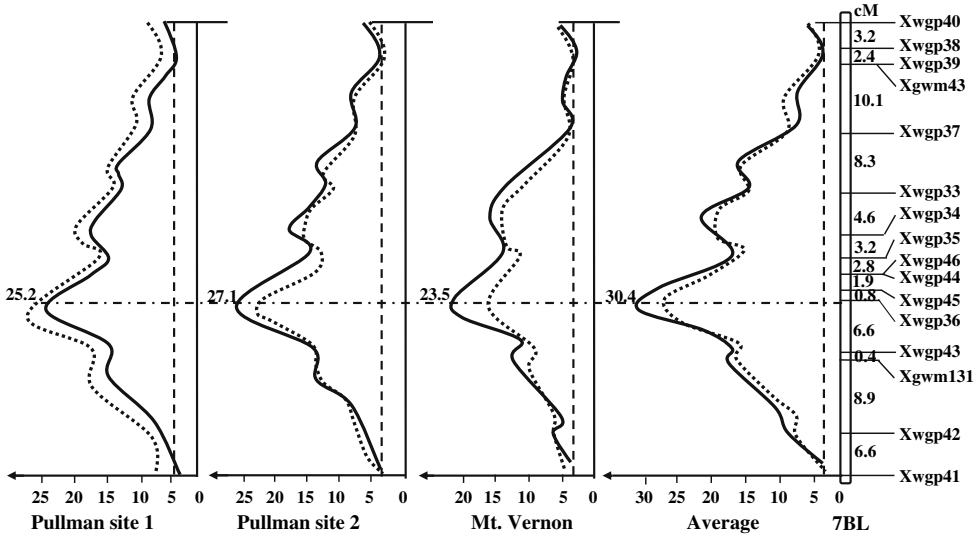

Fig. 4 Interval analysis of HTAP resistance QTL on chromosome 7BL (*Qhtap.wsu-7BL*) from the F_3 population of AVS/Alpowa, with SSR and RGAP markers assigned to chromosome 7BL. The HTAP QTL scan for each location and combined data (*Pullman sites 1 and 2*, *Mt.Vernon* and *average*) is represented separately. The positions (in cM) of the molecular markers are shown on the chromosome 7BL along the vertical axis. The dot line represents the logarithm of the odds (LOD) significance threshold of 3.0. LOD values are shown on the horizontal axis and the maximum value is shown at the peak. The solid line contours are based on relative AUDPCs and the dot line contours are based on heading-flowering stage infection type data

Table 4 Coefficients (R^2) and LOD values of RGAP and SSR markers linked to the HTAP resistance gene *Yr39* based on individual and combined relative area under disease progress curve (*AUDPC*) and infection type (*IT*) data of the AVS/Alpowa F_3 population at three locations

Marker[a]	Pullman site 1				Pullman site 2				Mt. Vernon				Average			
	Relative AUDPC		IT[b]		Relative AUDPC		IT[b]		Relative AUDPC		IT[b]		Relative AUDPC		IT[b]	
	R^2	LOD	R^2	LOD	R^2	LOD	R^2	LOD	R^2	LOD	R^2	LOD	R^2	LOD	R^2	LOD
Xwgp41	13.7	4.4	14.9	4.8	13.7	4.4	16.4	5.3	18.1	5.9	15.6	5.0	17.1	5.5	17.7	5.8
Xwgp42	25.3	8.6	27.9	9.7	26.3	9.0	25.7	8.8	23.2	7.8	20.9	6.9	28.6	9.9	26.3	9.0
Xgwm43	36.9	13.6	42.3	16.2	40	15.0	38.9	14.6	37.9	14.1	31.8	11.3	43.7	17	41.5	15.8
Xwgp43	38.6	14.4	44.3	17.3	42.3	16.2	41.1	15.7	38.1	14.2	30.3	10.7	45.4	17.9	42.3	16.3
Xwgp36	52.1	21.7	53.7	22.8	58.9	26.3	54.3	23.2	54.2	23.0	45.3	17.8	63.0	29.4	57.5	25.3
Xwgp45	47.6	19.1	50.9	21	54.9	23.5	51.5	21.4	50.4	20.7	43.3	16.8	58.3	25.8	54.0	22.9
Xwgp44	39.2	14.7	41.8	15.9	45.6	17.9	40.3	15.2	43.7	16.9	38.3	14.3	48.9	19.8	44.4	17.3
Xwgp46	39.2	14.7	41.8	15.9	45.6	17.9	40.3	15.2	43.7	16.9	38.3	14.3	48.9	19.8	44.4	17.3
Xwgp35	37.7	13.9	40.5	15.3	42.9	16.6	38.4	14.3	41.7	15.9	35.8	13.1	46.5	18.5	41.2	15.7
Xwgp34	40.9	15.6	43.8	17	45.7	18.1	42.6	16.4	41.9	16.1	40.4	15.3	49.0	19.9	46.3	18.4
Xwgp33	33.8	12.2	34.6	12.6	37.1	13.7	35.1	12.8	39.4	14.8	36.9	13.6	41.8	16.0	39.6	14.9
Xwgp37	23.8	8.0	25.3	8.6	25.1	8.5	27.6	9.5	18.8	6.1	14.8	4.7	25.9	8.9	29.5	10.3
Xgwm131	14.8	4.7	15.1	4.8	14.8	4.7	14.4	4.6	13.5	4.3	12.8	4.0	16.5	5.3	18.8	6.2
Xwgp39	14.8	4.7	15.1	4.8	14.8	4.7	14.4	4.6	13.5	4.3	12.8	4.0	16.5	5.3	18.8	6.2
Xwgp38	17.8	5.8	17.9	5.8	19.0	6.2	17.9	5.9	17.7	5.8	16.8	5.4	20.8	6.9	20.8	6.9
Xwgp40	19.1	6.2	18.5	6	19.5	6.4	22.1	7.4	23.3	7.9	20.7	6.9	23.4	7.9	22.2	7.4

[a] *Xgwm43* and *Xgwm131* were SSR markers and all others were RGAP markers

[b] Infection type (IT) data were those recorded at the heading-flowering stages

linkage group, are given in Table 3. The determination coefficients (R^2) and LOD values of RGAP and SSR markers linked to *Yr39* using data from each location, and the combined data, are presented in Table 4. The peak of the HTAP resistance was between the RGAP markers *Xwgp36* and *Xwgp45* with a LOD value of 30.4 based on the relative AUDPC means across the three sites (Fig. 4).

The *Yr39* gene explained 64.2% of the phenotypic variance for the averaged relative AUDPCs and 59.1% for the averaged heading-flowering stage IT data. Because of the high-explained phenotypic variance, high-LOD scores across three locations (Fig. 4), and high R^2 values (Table 4), the *Yr39* QTL had a major effect on the HTAP response in Alpowa. Figure 4 shows an identical peak at the same chromosome region using different data sites despite the different LOD values, indicating that the *Yr39* gene contributing to the HTAP resistance is relatively insensitive to environmental conditions and different pathogen populations.

Discussion

Alpowa was released in the USA PNW in 1994 and is still the major spring wheat cultivar in that region. In several tests of wheat germplasms under controlled greenhouse conditions from 2002 to 2004, Alpowa was susceptible to races PST-29, 37, 43, 45, 78, 98, 100, 101, and PST-111) of *P. striiformis* f. sp. *tritici* tested in the seedling stage (XM Chen, unpublished data). The susceptibility of Alpowa to these races, which predominated in the area at various times from the early 1980s to 2004, suggests that Alpowa does not have effective race-specific resistance. However, Alpowa seedlings were resistant to races PST-1 and PST-21, which are the least virulent races that have been identified because PST-1 is only virulent on Lemhi (*Yr21*) and Chinese 166 (*Yr1*), and PST-21 is only virulent on Chinese 166 among the wheat genotypes used to differentiate races of *P. striiformis* f. sp. *tritici* in the USA (Line and Qayoum 1992; Chen 2005). These results show that all-stage resistance to PST-1 and PST-21 detected in seedlings is controlled by a single partially dominant gene. Although the resistance was detected in the seedling stage, it is also specifically resistant to races PST-1 and PST-21 throughout growth stages, and therefore, should be considered to be race-specific.

The all-stage resistance gene was mapped on chromosome 1BS with genetic distances of 2.8 cM from *Yr15*, and 12.4 cM from *YrH52*, based on linkage maps of Sun et al. (1997) and Peng et al. (1999). *Yr15* confers resistance to all races identified in the USA (Chen, unpublished data). Both *Yr15* and *YrH52* confer wide ranges of resistance, whereas the gene in Alpowa identified in our study confers a narrow range of resistance. Other genes reported on chromosome 1BS include *Yr9*, *Yr10* and *Yr24*/*Yr26*. In addition, *Yr29* was reported on chromosome 1BL and *Yr3a*, *Yr3c*, and *Yr21* were reported on chromosome 1B, but the arm was not determined (McIntosh et al. 1996, 1998; Chen 2005). Except for *Yr21*, all these genes have a wider virulence spectrum than the all-stage resistance gene in Alpowa. Resistance gene *Yr21* reported in Lemhi is only effective against PST-21 and ineffective against PST-1 (Chen et al. 1995; Pahalawatta and Chen 2005a). Because the all-stage resistance gene in Alpowa has a different specificity from each of the other genes, we designate this gene as *YrAlp*. Although *YrAlp* is ineffective against predominant races of the stripe rust pathogen, it should be useful in studying the plant–pathogen interactions and mechanisms of the host resistance and pathogen evolution.

Stripe rust on Alpowa has never been observed to have an IT 8 or 9 in the field at late stage of plant growth. The typical symptoms and signs caused by stripe rust infection on Alpowa at later stage of plant growth are necrotic stripes up to several centimeters long with or without sporulation along the edges. The field results obtained in this study confirmed the non-race-specific HTAP resistance in Alpowa. The HTAP resistance of Alpowa is generally adequate in the USA PNW and also should be adequate in regions with similar weather conditions and cropping systems. With early infection and favorable conditions, yield losses in our yield loss experimental plots in 2005 were 6.1% for Alpowa, which was much lower than 22.6% for the susceptible spring wheat cultivar 'Fielder' and 51.4% for the susceptible winter wheat cultivar 'Moreland' (X.M. Chen and D. Wood, unpublished data).

Using the QTL mapping approach, we identified QTL with a major effect conferring the resistance with both the relative AUDPC and IT data. In addition, genetic analysis of the IT data indicated a single gene for HTAP resistance. The major effect of the single QTL for HTAP resistance makes it relatively easy to use in breeding. The degree of HTAP resistance conferred by this QTL is similar to that of *Yr36* (Uauy et al. 2005). Alpowa and wheat lines with *Yr36* were tested in the same experiments with seedlings and adult-plants inoculated with the same races in the greenhouse (data not shown). Our results were also similar to those of Börner et al. (2000) for the *Yrns-B1* gene determining non-specific adult-plant disease resistance against stripe rust mapped on chromosome 3BS.

It is common to find one or two major QTLs in reports on disease resistance (Rector et al. 1998; Geffroy et al. 2000). When more than one QTL is involved, usually one of the QTLs has a bigger effect than the others. With such findings in mind, we took the bulk segregant analysis approach to quickly identify a major QTL. The HTAP resistance QTL explained 64.2% for

the averaged relative AUDPCs and 59.1% for the averaged heading-flowering stage IT data.

The HTAP resistance gene in Alpowa was mapped on the long arm of chromosome 7B. Resistance genes *Yr2* and *Yr6* have been reported on chromosome 7B, but these genes confer race-specific all-stage resistance (Chen 2005). Other genes conferring non-specific adult-plant and/or HTAP resistance mapped on other chromosomes include *Yr16* (2DS), *Yr18* (7DS), *Yr29* (1BL), *Yr30* (3BS), *Yr36* (6BS), *Yrns-B1* (3BS) and a QTL (6BS) in Stephens soft white winter wheat (McIntosh et al. 1998; Chen 2005; Uauy et al. 2005; Santra et al. 2006). Thus, the HTAP gene resistance in 'Alpowa' is different from previously reported genes for non-race-specific resistance and we designate it *Yr39*. The different chromosomal location of *Yr39* relative to other non-race-specific resistance genes makes it feasible to combine *Yr39* with other genes to obtain durable and high-level resistance. The molecular markers identified in this study should be useful for such combinations.

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