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ORIGINAL PAPER

Molecular mapping of *Yr53*, a new gene for stripe rust resistance in durum wheat accession PI 480148 and its transfer to common wheat

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Abstract Stripe rust, caused by *Puccinia striiformis* f. sp. *tritici* (*Pst*), is one of the most damaging diseases of wheat worldwide. It is essential to identify new genes for effective resistance against the disease. Durum wheat PI 480148, originally from Ethiopia, was resistant in all seedling tests with several predominant *Pst* races in the US under controlled greenhouse conditions and at multiple locations subject to natural infection for several years. To map the resistance gene(s) and to transfer it to common wheat, a cross was made between PI 480148 and susceptible common wheat genotype Avocet S (AvS). Resistant F_3 plants with 42 chromosomes were selected cytologically and by testing with *Pst* race PST-100. A total of 157 F_4 plants from a single F_3 plant with $2n = 42$ tested with PST-100 segregated in a 3 resistant: 1 susceptible ratio, indicating that a single dominant gene from PI 480148 conferred resistance. Using the $F_{3:4}$ population and the resistance gene-analog polymorphism (RGAP) and simple sequence repeat (SSR) markers, the gene was mapped to the long arm of chromosome 2B. SSR marker *Xwmc441* and RGAP marker *XLRRrev/NLRRrev*$_{350}$ flanked the resistance gene by 5.6 and 2.7 cM, respectively. The effective resistance of the gene to an Australian *Pst* isolate virulent to *Yr5*, which is also located on 2BL and confers resistance to all US *Pst* races, together with an allelism test of the two genes, indicated that the gene from PI 480148 is different from *Yr5* and should be a new and useful gene for resistance to stripe rust. Resistant common wheat lines with plant types similar to AvS were selected for use in breeding programs.

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Introduction

Stripe rust, caused by *Puccinia striiformis* f. sp. *tritici* (*Pst*), is one of the most damaging diseases of common wheat (*Triticum aestivum* L., $2n = 6x = 42$, AABBDD genomes) and durum wheat (*Triticum turgidum* L. ssp. *durum*, $2n = 4x = 28$, AABB genomes) worldwide (Chen 2005). Stripe rust affects wheat crops in the early growth stages leading to yield losses of more than 90 % on susceptible cultivars when weather conditions are extremely favorable to the disease (Chen 2005; Sharma-Poudyal and Chen 2011). Growing resistant cultivars is the most effective, economic and environmentally friendly way to control stripe rust. Based on specificity, stripe rust resistance can be classified as race specific or race non-specific; and based on plant growth stage, it can be classified as seedling (also known as all-stage) resistance or adult plant resistance (Chen 2005, 2012). Seedling resistance is usually race specific, but often provides complete control when it confers resistance to the prevalent races. Race-specific resistance is often conferred by a single gene that follows the gene-for-gene interaction model (Flor 1971). Adult plant

resistance is usually non-race specific and often requires high temperatures to function. High-temperature adult plant (HTAP) resistance, which is expressed when plants grow old and weather becomes warm, can be controlled by a single or multiple genes. HTAP resistance is usually durable, but may not be adequate if the level of resistance is low and the weather is not favorable for expression. Because of the advantages and disadvantages of seedling resistance and HTAP resistance, it is desirable to combine both types of resistance for sustainable control of stripe rust (Chen 2005, 2012). With this in mind, more resistance genes of both types should be identified and mapped.

About 52 permanently named and more than 40 temporarily designated genes or quantitative trait loci (QTL) for stripe rust resistance have been reported (Chen 2005, 2008; McIntosh et al. 2009, 2010, 2011; Ren et al. 2012). Among the permanently named resistance genes, *Yr11*, *Yr12*, *Yr13*, *Yr14*, *Yr16*, *Yr18*, *Yr29*, *Yr30*, *Yr34*, *Yr36*, *Yr39*, *Yr46*, *Yr48* and *Yr52* confer adult plant or HTAP resistance genes, whereas the others confer all-stage resistance. Of the permanently named *Yr* genes, 14 were transferred from common wheat relatives, such as *T. aestivum* subsp. *spelta* var. album, *T. dicoccoides*, *T. tauschii*, *T. turgidum*, *T. turgidum* var. *durum*, *T. ventricosum*, *Aegilops (Ae.) comosa*, *Ae. geniculata*, *Ae. kotschyi*, *Ae. neglecta*, *Ae. sharonensis*, *Haynaldia villosa* or from more distant species, like *Secale cereale* (McIntosh et al. 1998, 2009, 2010; Chen 2005). Tetraploid wheat with the A and B genomes is a primary gene pool for desirable traits in common wheat including disease resistance (Feldman and Millet 1993; Huang et al. 2003; Blanco et al. 2001, 2008). Resistance genes *Yr7*, *Yr15*, *Yr24*/*Yr26* and *Yr36* originated from tetraploid wheat accessions (Macer 1966; McIntosh and Lagudah 2000; Ma et al. 2001; Uauy et al. 2005). Durum wheat ($2n = 4\chi = 28$, AABB genomes) is grown on approximately 17 million hectares worldwide (Abdalla et al. 1992). Mamluk (1992) reported that approximately 23 % of 216 durum wheat cultivars in West Asia and North Africa were resistant to stripe rust. Ma et al. (1995, 1997a, b) observed a wide range of seedling and adult plant resistance to stripe rust in durum wheat cultivars from various countries. Their reports indicated that there may be a number of undocumented stripe rust resistance genes in durum wheat lines, but there have been no further studies to identify the genes. Durum wheat has been frequently used as a bridging species to transfer new resistance genes from diploid wheat into common wheat because it is relatively easy to make crosses between durum wheat and common wheat (Chhuneja et al. 2008). Durum wheat–common wheat crosses should therefore be a convenient way to explore new genes for stripe rust resistance from durum wheat and an effective way to transfer them to common wheat.

In our previous screening of wheat germplasm for resistance to stripe rust, we identified a number of durum wheat genotypes with effective resistance to stripe rust (Chen, unpublished data). The objectives of this study were to characterize stripe rust resistance in durum wheat PI 480148 originating from Ethiopia, identify and develop molecular markers for the resistance gene(s) and to transfer the gene(s) into a common wheat background.

Materials and methods

Developing a mapping population and advanced lines

A cross was made between durum wheat PI 480148 and common spring wheat Avocet S (AvS) in early 2006 in a greenhouse using AvS as the female parent. Because of partial cross incompatibility, six heads of different AvS plants were pollinated and only 20 F_1 seeds were obtained. Six F_1 plants were grown in the greenhouse to produce F_2 seeds. In spring 2007, more than 100 F_2 seeds were space-planted in a field near Pullman, WA, and only 54 F_2 plants survived. Natural infection of stripe rust occurred in the nursery and individual plants were scored for stripe rust infection type (IT) using a 0–9 scale (Line and Qayoum 1992) and disease severity (DS) as percentage of leaf areas infected. F_3 seeds from these F_2 plants were harvested.

To identify hexaploid F_3 plants from heterozygous resistant F_2 plants, F_3 seeds were germinated on moist filter paper in petri dishes. When roots grew about 1–2 cm long, chromosome numbers of individual plants were checked using the Feulgen staining method (Schulte and Wittekind 1989; Chen et al. 1995). The chromosome numbers of 57 F_3 seedlings from 29 F_2 individuals were counted and these were inoculated with race PST-100 at the two-leaf stage. Resistant F_3 plants with 42 chromosomes were transplanted into large pots (12 × 12 × 12 cm) and grown to maturity. About 20 F_4 seeds from each selected F_3 plant were planted and the seedlings were tested for stripe rust response in the greenhouse. Based on the results, a segregating population of 157 F_4 plants derived from a single F_3 plant with 42 chromosomes was selected for genetic analysis and mapping of the resistance gene(s). F_5 and F_6 lines from the F_4 population were produced through single seed descent and evaluated for rust response and agronomic traits in fields near Pullman in 2010 and 2011.

Evaluation of parents and progeny of AvS × PI 480148 for reaction to stripe rust

Nine *Pst* races (PST-17, PST-37, PST-43, PST-45, PST-70, PST-78, PST-100, PST-127 and PST-130) were used to evaluate the stripe rust responses of PI 480148 together with

Table 1 Infection types on seedlings of Avocet S (AvS), PI 480148 and a selected F_5 line (F_5-128) from the AvS × PI 480148 cross produced by races of *Puccinia striiformis* f. sp. *tritici* under controlled greenhouse conditions

Race	Virulence formula[a]	Infection type		
		AvS	PI 480148	F_5-128
PST-17	1, 2, 3, 9, 11	9	2	2
PST-37	1, 3, 6, 8, 9, 11, 12	9	2	2
PST-43	1, 3, 4, 5, 12, 14	9	2	2
PST-45	1, 3, 12, 13, 15	9	2	2
PST-70	1, 3, 11, 12, 16, 18	9	2	2
PST-78	1, 3, 11, 12, 16, 17, 18, 19, 20	9	2	2
PST-100	1, 3, 8, 9, 10, 11, 12, 16, 17, 18, 19, 20	9	2	2
PST-127	1, 3, 5, 6, 8, 9,10, 11, 12, 13, 15, 16, 17, 18, 19, 20	9	2	2
PST-130	1, 3, 4, 8, 10, 11, 12, 16, 17, 18, 19, 20	9	2	2

[a] The virulence formulae are based on reactions on the following 20 wheat genotypes used to differentiate races of *P. striiformis* f. sp. *tritici* in the US: 1 = Lemhi (*Yr21*), 2 = Chinese 166 (*Yr1*), 3 = Heines VII (*Yr2*, *YrHVII*), 4 = Moro (*Yr10*, *YrMor*), 5 = Paha (*YrPa1*, *YrPa2*, *YrPa3*), 6 = Druchamp (*Yr3a*, *YrD*, *YrDru*), 7 = AvSYr5NIL (*Yr5*), 8 = Produra (*YrPr1*, *YrPr2*), 9 = Yamhill (*Yr2*, *Yr4a*, *YrYam*), 10 = Stephens (*Yr3a*, *YrS*, *YrSte*), 11 = Lee (*Yr7*, *Yr22*, *Yr23*), 12 = Fielder (*Yr6*, *Yr20*), 13 = Tyee (*YrTye*), 14 = Tres (*YrTr1*, *YrTr2*), 15 = Hyak (*Yr17*, *YrTye*), 16 = Express (*YrExp1*, *YrExp2*), 17 = AvSYr8NIL (*Yr8*), 17 = AvSYr9NIL (*Yr9*), 19 = Clement (*Yr9*, *YrCle*), and 20 = Compair (*Yr8*, *Yr19*) (Chen et al. 2002, 2010; Chen 2005)

AvS and derivatives (Table 1). The nine races were selected to cover all individual virulences identified thus far and to represent predominant races in the US *Pst* population since the 1970s (Line and Qayoum 1992; Chen et al. 2002, 2010; Chen 2005; Wan and Chen 2012). PST-100, which was the most distributed race in the US in recent years (Chen et al. 2010; Wan and Chen 2012), was used to test F_3 and F_4 plants. A *Yr5*-virulent isolate (841541:430; race 360E137A-) of *Pst* from Australia (Wellings and McIntosh 1990) was used in a seedling test to determine the relationship of the resistance gene in PI 480148 and *Yr5* as both genes were mapped to chromosome 2BL and were effective against all tested US *Pst* races (Yan et al. 2003; Chen et al. 2010; Wan and Chen 2012).

Seedling tests were conducted under controlled greenhouse conditions as described by Chen and Line (1992a, b). Seeds were planted in pots (6 × 6 × 6 cm) filled with soil mixture with about seven plants per pot. Seedlings at the two-leaf stage were inoculated with selected *Pst* races and grown in growth chambers using a low temperature (4–20 °C) profile. A set of wheat genotypes used to differentiate *Pst* races in the USA was included in each test to confirm the identities of selected races. Infection type data were collected 18–21 days after inoculation based on the 0–9 scale (Line and Qayoum 1992).

RGAP and SSR analyses

Genomic DNA was extracted using the CTAB protocol (Riede and Anderson 1996). Resistant and susceptible bulks comprising equal amounts of DNA from 10 resistant and 10 susceptible F_4 plants, respectively, were used for bulked segregant analysis (Michelmore et al. 1991). The resistance gene-analog polymorphism (RGAP) method (Chen et al. 1998; Shi et al. 2001) and the SSR procedure (Röder et al. 1998) were used to identify molecular markers linked to the resistance locus.

Polymerase chain reactions were performed in a GeneAmp® PCR System 9700 thermo-cycler. A 15-μL reaction mixture contained 30 ng of template DNA, 1.5 μL Mg-free 10× PCR buffer (Promega, Madison, WI, USA), 0.6 U of *Taq* DNA polymerase (Promega, Madison, WI, USA), 5 mM of $MgCl_2$, 0.2 mM each of dCTP, dGTP, dTTP, and dATP (Sigma Chemical Co., St. Louis, MO, USA) and 30 ng of each primer synthesized by Operon Biotechnologies, Inc. (Huntsville, AL, USA). 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 primer pair), and 2 min at 72 °C followed by a 7 min extension 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 product. Following 4 min of denaturation at 94 °C, 7 μl of the PCR product and loading buffer mixture for each sample was loaded for electrophoresis in a 5 % polyacrylamide gel, and after electrophoresis, the gel was silver stained as previously described (Chen et al. 1998).

PCR fragment cloning and sequencing

PCR fragments of interest were excised from a dried polyacrylamide gel after wetting with a drop of sterile

water. The excised band was soaked in 20 μL of H_2O for at least 1 h, and the solution was used as template DNA for re-amplification with the original RGAP primers. The re-amplification product that showed a single band with the same size as the original band on a 6 % denaturing polyacrylamide gel was selected for cloning. Four microliters of re-amplified PCR products were cloned into pGEM-T Easy Vector System I (Promega) following procedures recommended by the manufacturer and transformed into bacterial cells. Three clones for each fragment were sequenced to obtain the accurate sequence.

Development of STS markers

Primers for sequence tagged site (STS) markers were designed based on the sequences of RGAP markers that were closely linked to the resistance gene using the software Primer Premier 5 (Premier Biosoft, Palo Alto, CA, USA). Potential STS markers were subjected to two testing procedures. Firstly, the STS markers were tested for polymorphism between the resistant and susceptible bulks as well as the parents. Secondly, the polymorphic bands amplified from STS markers were cloned and sequenced, and aligned with the corresponding sequences amplified from the RGAP markers using DNAMAN software (Lynnon Corporation, Pointe-Claire, Quebec, Canada). PCR amplification conditions for detecting STS markers were similar to the RGAP markers except for a varied number of cycles and annealing temperatures depending upon primers.

Statistical analysis and genetic mapping

Chi-squared tests were applied to evaluate the goodness of fit of observed segregations to specific inheritance ratios. A linkage map was constructed with the MAPMAKER program (Lander et al. 1987). Map distances in centiMorgans (cM) were calculated from recombination frequencies using the Kosambi mapping function (Kosambi 1944). For determining genetic distances between two genes using a F_2 population in an allelism analysis, the frequency (f) of susceptible plants in a repulsion phase cross was used to estimate the recombination value (p) using the formula $f = p^2/4$. For conversion to map distances, the formula $d = -1/2\ \mathrm{Ln}(1 - 2p)$ was used as previously described (Cheng and Chen 2010).

Results

Stripe rust resistance in PI 480148

PI 480148 was highly resistant (IT 2) whereas the susceptible parent, AvS, was highly susceptible (IT 9) under controlled greenhouse conditions (Table 1). PI 480148 was also highly resistant with IT 1 or 2 and DS 1–10 % in field nurseries under natural infection of *Pst* from 2005 to 2011 at the Pullman (eastern Washington) and Mount Vernon (western Washington) locations. In contrast, AvS was always highly susceptible (IT 8–9; DS 40–100 %). The results showed that PI 480148 has a high level of seedling resistance.

Inheritance of stripe rust resistance derived from PI 480148

Of the 54 F_2 plants from cross AvS × PI 480148 tested with PST-100 at seedling stage under controlled greenhouse conditions, 46 were resistant and 8 were susceptible, which was not significantly different from a 3:1 ratio ($\chi^2 = 2.99$, $P = 0.08$) indicating a single dominant gene segregation. Of the 57 F_3 plants examined for chromosome number, 34 tetraploid (28 chromosomes), 11 aneuploid (29–41 chromosomes) and 12 hexaploid (42 chromosomes) plants were identified. The 12 plants with $2n = 42$ that were tested with race PST-100 in the greenhouse, were either resistant or susceptible. Of the 157 F_4 plants derived from a single F_3 plant with $2n = 42$ and IT 3 tested with PST-100 in the greenhouse, 119 were resistant (ITs 2–3) and 38 were susceptible (ITs 7–9), which well fit a 3:1 ratio ($\chi^2 = 0.05$, $P = 0.82$). These results indicated that the F_3 plant was heterozygous for a single dominant gene. A homozygous resistant F_5 line (F5-128) from a resistant F_4 plant had the same resistant reaction (IT 2) to all nine races as PI 480148 (Table 1). The combined results indicated that PI 480148 has a single dominant gene for resistance to the tested races. The gene was designated as *Yr53* for reasons presented later in this paper.

RGAP markers linked with the *Yr53* locus

Three primer pairs that generated repeatable differences in banding patterns between resistant and susceptible bulks in the bulk segregant analysis were selected to test the 157 F_4 plants. As an example, Fig. 1a, b shows the banding patterns of RGAP markers *Ptokin1/NLRR-INV1*$_{800}$ and *Ptokin2/Xa1NBS-F*$_{234}$, respectively, among the parents, bulks and some F_4 plants. All three RGAP markers were dominant, and their primers, band sizes, presence and absence in PI 480148, AvS, and Chinese Spring, and the probability (P) values for goodness of fit to single loci are shown in Table 2. The single-locus inheritance indicated that these markers are reliable for constructing a linkage group for *Yr53*.

To more easily assay these polymorphisms, we tried to convert the RGAP markers into STS markers. One STS marker, *STS2F/1R*$_{219}$ (primers: 2F-AGGGATAGGGCGTG GTT, 1R-CCACCACGTAGCAACACTTA), was successfully

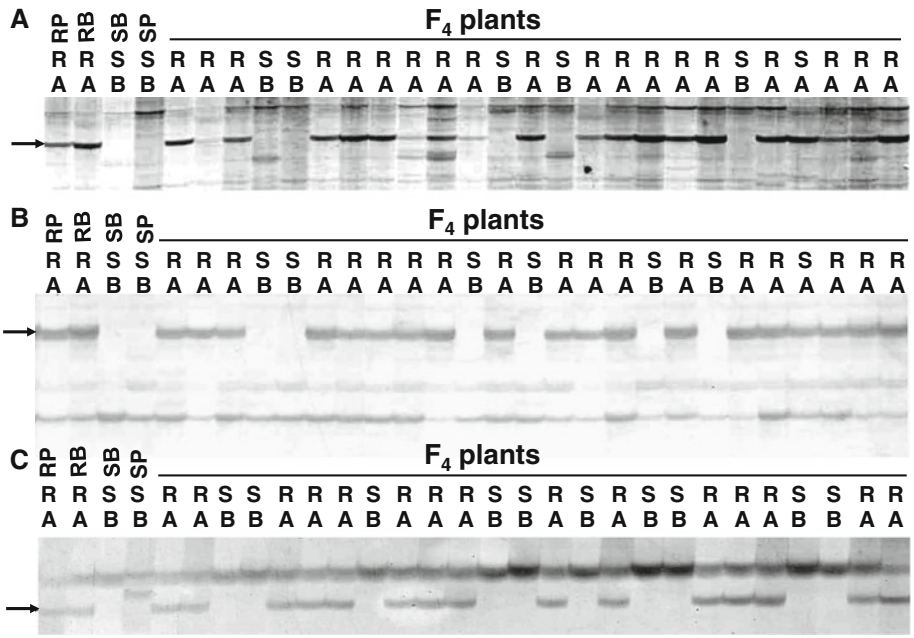

Fig. 1 Silver stained denaturing polyacrylamide gels showing segregation patterns of RGAP markers *Ptokin1/NLRR-INV1*$_{800}$ (**a**) and *Ptokin2/Xa1NBS-F*$_{234}$ (**b**) and STS marker *STS2F/1R*$_{219}$ (**c**) associated with the resistance locus detected in the F_4 mapping population from AvS/PI 480148. The STS marker was developed from RGAP marker *Ptokin2/Xa1NBS-F*$_{234}$. *RP* resistant parent (PI 480148), *RB* resistant bulk, *SB* susceptible bulk, and *SP* susceptible parent (AvS). The phenotypic reaction to stripe rust is indicated by "R" for resistant and "S" for susceptible. The genotypes are indicated by "A" for presence as in the resistant parent and "B" for absence as in the susceptible parent

developed from RGAP marker *Ptokin2/Xa1NBS-F*$_{234}$. The 219-bp STS fragment was amplified under similar conditions as the original RGAP marker except that the PCR amplification was performed for 35 cycles consisting of 94 °C for 30 s for denaturation; 55 °C for 30 s and 60 °C for 1 min for annealing; and 72 °C for 2 min for extension. Although the polymorphic band could not be detected in 5 % agarose gels, definitive bands were produced on polyacrylamide gels (Fig. 1c). DNA sequence alignments showed that the sequence of PCR products generated by the STS marker was consistent with the RGAP marker.

Mapping *Yr53* to chromosome 2BL

We used the three RGAP primers to amplify Chinese Spring in order to use the nulli-tetrasomic lines to determine the chromosomal location of the polymorphic fragments and thus *Yr53*. However, all of these RGAP markers were absent in Chinese Spring so a simple PCR amplification of nulli-tetrasomic lines could not be used. SSR markers were therefore screened to determine the chromosomal location and identify more markers for the gene.

A total of 216 SSR markers, selected to cover the 14 A and B chromosomes as the resistance gene is from a durum wheat, were screened among the parents and two bulks. Two markers were found to be associated to the *Yr53* locus. These were *Xwmc441* amplified with WMC441 primers (F: 5′-TCCAGTAGAGCACCTTTCATT-3′ and R: 5′-ATCACGAAGATAAACAAACGG-3′), and *Xwmc149* (F: 5′-GCTCAGATCATCCACCAACTTC-3′ and R: 5′-AGATGCTCTGGGAGAGTCCTTA-3′) (Somers et al. 2004). Both were dominant markers producing 151- and 230-bp bands, respectively, in PI 480148 and the resistant bulk, but not in AvS and the susceptible bulk. Linkage analysis using the two markers with the 157 F_4 plants indicated that they were linked to the resistance gene. The two SSR markers associated with the long arm of chromosome 2B indicated that *YrYr53* is on 2BL.

Xwmc441 produces a 159-bp band in Chinese Spring. In order to verify the identity of the PCR products amplified by the *Xwmc441* primers, the two bands from PI 480148 and Chinese Spring were cloned and sequenced. The sequences revealed that the two fragments shared a high similarity (92 %) at the nucleotide sequence, indicating that the *Xwmc441* locus has different alleles among wheat genotypes (data not shown). In addition, three more SSR markers, *Xgwm501*, *Xwgp109* and *Xbarc349* (Table 2), were identified to be loosely linked with the *Yr53* locus.

To determine the location of *Yr53* on 2BL, SSR markers *Xbarc349*, *Xwmc501*, *Xwmc441* and *Xwmc149* were used to test a set of five Chinese Spring deletion lines of 2BL (del2BL-3, del2BL-5, del2BL-6, del2BL-7 and del2BL-10) together with Chinese Spring. The target bands were present on all deletion lines, indicating that the resistance

Table 2 Molecular markers linked to *Yr53*, primer pairs, size, and presence or absence in PI 480148, AvS, and Chinese Spring (CS)

Marker[a]	Primer pair[b]	Size (bp)	Fragment			$P_{for\ 3:1}$
			PI 480148	AvS	CS	
Ptokin1/NLRR-INV1$_{800}$	Pto kin 1/NLRR-INV1	800	800	–	–	0.89
Ptokin2/Xa1NBS-F$_{234}$	Pto kin 2/Xa1NBS-F	234	234	–	–	0.61
XLRRrev/NLRRrev$_{350}$	XLRR rev/NLRR rev	350	234	–	–	0.68
Xwmc441	WMC441 F/R	151/159	151	–	159	0.54
Xwmc149	WMC149 F/R	230	230	–	–	0.33
Xgwm501	GWM501 F/R	130/140/150	130	140	150	0.25
Xwgp109	WGP109 F/R	650	650	–	–	0.12
Xbarc349	BARC349 F/R	105	105	–	105	0.10
Yr5STS7/8	Yr5STS7/8 F/R	472/478	478	472	472	0.08
STS2F/1R$_{219}$	STS 2F/1R	219	219	–	–	0.61

–, absent

[a] The first three markers are resistance gene-analog polymorphism (RGAP) markers; the next five markers are simple sequence repeat (SSR) markers; *Yr5STS7/8* is a sequence tagged site (STS) marker linked to *Yr5* (Chen et al. 2003); and STS2F/1R is a STS marker developed from *Ptokin2/Xa1NBS-F*$_{234}$ in this study

[b] Sequences of the are RGA primers are Pto kin 1: 5′-GCATTGGAACAAGGTGAA-3′, NLRR-INV1: NLRR-INV1: 5′-TTGTCAGGCCAG ATACCC-3′, Pto kin 2: 5′-AGGGGGACCACCACGTAG-3′, Xa1NBS-F: 5′-GGCAATGGAGGGATAGG-3, XLRR rev: 5′-CCCATAG ACCGGACTGTT-3′ and NLRR rev: 5′-TATAAAAAGTGCCGGACT-3′ (Chen et al. 1998; Shi et al. 2001; Yan et al. 2003). The sequence of Yr5STS7 is 5′-GTACAATTCACCTAGAGT-3′ and of Yr5STS8 5′-GCAAGTTTTCTCCCTATT-3′ (Chen et al. 2003). The sequence of primer STS 2F is 5′-AGGGATAGGGCGTGGTT-3′ and of STS 1R 5′-CCACCACGTAGCAACACTTA-3. The primer sequences of the SSR markers (Somers et al. 2004) were from the GrainGenes database (http://www.wheat.pw.usda.gov)

gene is on the chromosomal bin 0–0.35 near the centromere of 2BL.

Linkage map for *Yr53*

A linkage map consisting of three RGAP markers, five SSR markers and two STS markers was constructed for *YrPI480148* (Fig. 2) using the phenotypic and genotypic data of the 157 F_4 plants. An RGAP marker (*XLRRrev/NLRRrev*$_{350}$) and a SSR marker (*Xwmc441*) were the closest markers flanking *Yr53* with map distances of 2.7 and 5.6 cM, respectively.

Relationships of *Yr53* to other *Yr* genes on 2BL

Yr genes that have been reported on chromosome 2BL include *Yr5*, *Yr7*, *Yr43* and *Yr44* (Yan et al. 2003; Chen 2005; Sui et al. 2009; Cheng and Chen 2010). To determine the relationships of *Yr53* with these genes, PI 480148 was tested together with wheat genotypes AvSYr5NIL (*Yr5*), AvSYr7NIL (*Yr7*), IDO377s (*Yr43*) and Zak (*Yr44*) with two US races (PST-100 and PST-127) and an Australian race (360E137A-). A wheat genotype possessing *YrQz* was not available. The reactions shown in Table 3 indicate that *Yr53* is different from *Yr5*, *Yr7*, *Yr43* and *Yr44*.

In order to obtain the relative map position of *Yr53* with other *Yr* genes on chromosome 2BL, polymorphic markers linked to *Yr5* (*Yr5STS7/8*), *Yr43* (*Xgwm501*, *Xwgp109*) and *Yr44* (*Xgwm501*) (Chen et al. 2003; Sui et al. 2009; Cheng and Chen 2010) were used to analyze the F_4 population. The genetic distances from *Yr53* to *Yr5*, *Yr43* and *Yr44* were estimated as 21.2, 16.9 and 10.9 cM, respectively (Fig. 2).

Because both *Yr53* and *Yr5* were the only genes mapped to 2BL that were effective against all US races tested to date, we crossed a selected F_5 line (F5-128) from the cross AvS × PI 480148 with AvSYr5NIL (*Yr5*) for an allelism test. A population of 185 F_2 plants was tested with race PST-100 at seedling stage in the greenhouse. Three F_2 plants were susceptible (IT 8), indicating that the resistance genes were not allelic; however, the segregation ratio also differed from 15:1 ($P = 0.009$) indicating linkage. The genetic distance between the two genes was estimated as 35.6 ± 6.7 cM using the formula $d = -1/2\ \ln(1 - 2p)$, where p is the recombination frequency and estimated by the formula of observed F_2 frequency of susceptible plants $= p^2/4$. This result is not inconsistent with the relationships shown in Fig. 2. One hundred and forty-seven F_3 lines derived from the F_2 plants were tested with the Australian *Pst* isolate virulent on *Yr5* at the seedling stage under controlled greenhouse conditions and with markers *Yr5STS7/8*, *Xwmc175*, *Xwmc441* and *STS2F/1R219*. The F_3 population segregated at 47 homozygous resistant, 63 segregating, and 36 homozygous susceptible lines, which

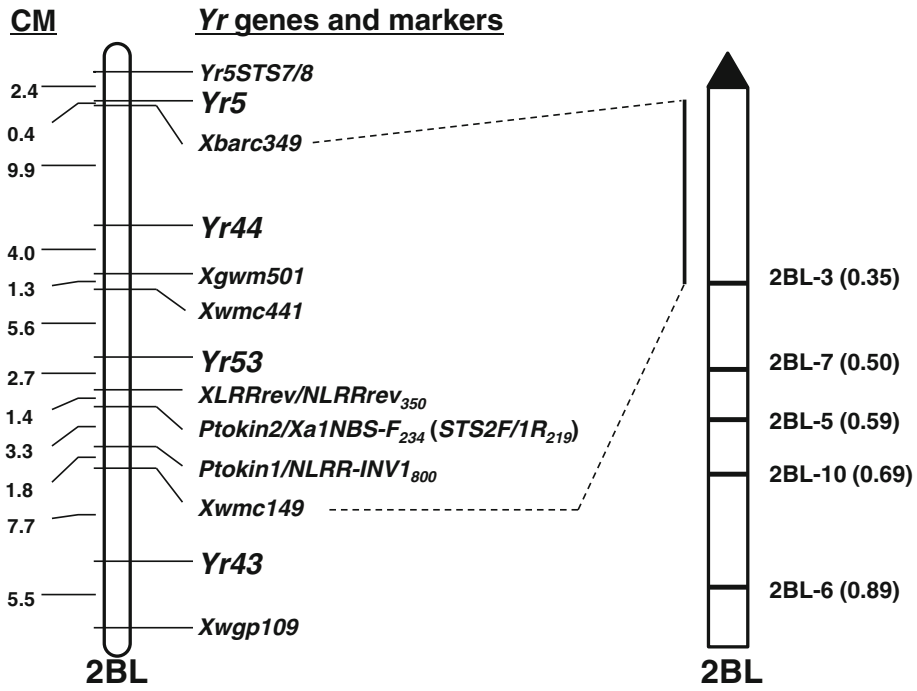

Fig. 2 Linkage map for *Yr53* on chromosome 2BL. The map distances of *Yr53* to resistance genes *Yr5*, *Yr43* and *Yr44* were based on common markers in Cheng and Chen (2010) and Sui et al. (2009)

Table 3 Infection types on wheat genotypes with *Yr* genes on chromosome 2BL produced by races of *Puccinia striiformis* f. sp. *tritici* (*Pst*)

Genotype	*Yr* gene	Infection type produced by *Pst* race		
		PST-100	PST-127	360E137A-[a]
AvSYr5NIL	*Yr5*	2	2	8
Lee	*Yr7*	8	8	2
IDO377s	*Yr43*	8	8	2
Zak	*Yr44*	8	8	2
PI 480148	*Yr53*	2	2	2

[a] Australian isolate (841541:430) that is virulent to *Yr5*

was not significantly different from an expected 1:2:1 ratio ($\chi^2 = 4.36$, $P = 0.11$). Based on the rust reaction and molecular marker data, three lines were identified to have both *Yr53* and *Yr5* at the homozygous state. Based on all of these results, the gene from PI 480148 was found to be different from previously named *Yr* genes on chromosome 2BL and, therefore, is permanently designated as *Yr53*.

Polymorphism of molecular markers closely linked to *Yr53* in various wheat genotypes

A total of 54 wheat genotypes representing breeding lines and current grown cultivars in the western US were tested with race PST-100 and the *Yr53*-flanking markers, *XLRRrev/NLRRrev*$_{350}$, *Xgwm441* and the STS marker (*STS2F/1R*$_{219}$) developed from RGAP marker, *Ptokin2/Xa1NBS-F*$_{234}$. When tested with PST-100, 59.3 % of the genotypes were resistant (Table 4). When tested with the markers, only seven (13.0 %) genotypes had a similar size of the *XLRRrev/NLRRrev*$_{350}$ fragment, eight (14.8 %) genotypes had the similar size of the *Xgwm44* fragment, and seven (13.0 %) had the similar size of *STS2F/1R*$_{219}$. None of the genotypes had two or three of the markers together and 32 (59.3 %) genotypes did not have any of the marker fragments. Thus, when any two of the markers are used in combination, they can detect polymorphism in 100 % of the tested genotypes.

Discussion

PI 480148 was collected from Shewa, Ethiopia in 1973 and deposited in the USDA Small Grains Collection in 1983 (http://www.ars-grin.gov/cgi-bin/npgs/acc/search.pl?accid=PI+480148). The line was first evaluated by our program in 2005 and showed high resistance in all greenhouse and field tests. In the present study, genetic analysis showed that resistance in a hexaploid derivative of PI 480148 is controlled by a dominant gene and molecular makers mapped the gene to chromosome 2BL. We showed that the gene is different from other permanently named genes on 2BL, including *Yr5*, *Yr7*, *Yr43* and *Yr44* (Yan et al. 2003; Smith et al. 2007; Sui et al. 2009; Cheng and Chen 2010). *Yr7* is ineffective against most races identified since the 1970s and *Yr43* and *Yr44* have become ineffective to most races identified since 2000 in the US (Line and Qayoum 1992; Chen 2005, Chen et al. 2010; Wan and Chen 2012),

Table 4 Infection types produced by *Puccinia striiformis* f. sp. *tritici* race PST-100 on various wheat genotypes and polymorphisms of molecular markers flanking the *Yr53* locus in these genotypes

Wheat genotype		IT	Presence (+) and absence (−) of markers		
Name	ID no.		*XLRRrev/NLRRrev*$_{350}$	*Xgwm441*	*STS2F/1R*$_{219}$
AvS	**WG00001**	**9**	**−**	**−**	**−**
PI 480148	**PI480148**	**2**	**+**	**+**	**+**
02W50076	02W50076	2	+	−	−
Blanca Grande	PI 631481	2	+	−	−
BZ9M03-1044	BZ9M03-1044	2	+	−	−
Hank	PI 613585	2	+	−	−
Lolo	PI 614840	2	+	−	−
Solano	DA900229	2	+	−	−
WA7990	WA007990	2	+	−	−
95WV10616	95WV10616	0	−	+	−
Buck Pronto	T0001052	2	−	+	−
BZ903-455WP-D	BZ903-455WP-D	2	−	+	−
BZ999-339	BZ999-339	2	−	+	−
Jefferson	PI 603040	2	−	+	−
Joaquin	YU-999-178	2	−	+	−
WA7998	PI 638537	2	−	+	−
Waikea	BZ998447	2	−	+	−
01W20172	01W20172	0	−	−	−
Alpowa	PI 566596	8	−	−	+
Alta Blanca	Alta Blanca	2	−	−	+
Alturas	PI 620631	8	−	−	+
Bersee	PI 168661	8	−	−	+
Compair	PI 325842	8	−	−	+
DA901-66	DA901-66	2	−	−	−
Eden	PI 630983	8	−	−	−
Edwall	PI 477919	8	−	−	−
Express	PI 573003	8	−	−	−
Expresso	Expresso	0	−	−	−
Fielder	CI 017268	8	−	−	−
Jeff/Pronto	Jeff/Pronto	3	−	−	−
Jerome	IDO00566	2	−	−	+
Kelse	PI 653842	2	−	−	−
Louise	PI 634865	8	−	−	−
Macon	PI 617072	8	−	−	−
Nick	BZ698031	8	−	−	−
Otis	PI 634866	8	−	−	−
Scarlet	PI 601814	8	−	−	−
Tara 2002	PI 617073	2	−	−	−
UI Cataldo	IDO00642	8	−	−	−
UI Pettit	IDO00632	8	−	−	−
UI Winchester	IDO00578	8	−	−	+
WA7964	WA007964	2	−	−	−
WA7986	WA007986	0	−	−	−
WA7987	WA007987	2	−	−	−
WA7988	WA007988	3	−	−	−
WA8007	WA008007	8	−	−	−
WA8008	PI 653841	5	−	−	−
WA8010	WA008010	3	−	−	−

Table 4 continued

Wheat genotype		IT	Presence (+) and absence (−) of markers		
Name	ID no.		*XLRRrev/NLRRrev*$_{350}$	*Xgwm441*	*STS2F/1R*$_{219}$
WA8012	WA008012	0	−	−	−
WA8015	WA008015	2	−	−	−
WA8016	WA008016	2	−	−	−
WA8017	WA008017	2	−	−	−
Wakanz	PI 506352	8	−	−	−
Wawawai	PI574538	8	−	−	−
Westbred 926	Westbred 926	8	−	−	−
Zak	PI 607839	8	−	−	−

so it is easy to distinguish the PI 480148 gene from these three genes since it is effective against all tested races. Because *Yr5* is effective against all *Pst* races identified so far in the US (Chen 2005; Chen et al. 2002, 2010; Wan and Chen 2012), we used a *Yr5*-virulent isolate from Australia (Wellings and McIntosh 1990) to demonstrate that the PI 480148 gene is effective to that isolate, indicating that they are different genes. Furthermore, we showed that *Yr5* and the gene from PI 480148 are not allelic. Tests of molecular markers closely linked to *Yr5*, *Yr43* and *Yr44* with the F_4 population of the AvS × PI 480148 cross also showed that these genes and the PI 480148 gene should be located at different loci. Based on these results, the stripe rust resistance gene in durum wheat PI 480148 and transferred to a common wheat background was named as *Yr53*.

In order to ensure that we have genetic resources to successfully counter the continuous evolution of the stripe rust pathogen, it is necessary to discover new resistance genes on a continuing basis, either in hexaploid wheat or in its relatives. Because durum wheat and common wheat have common A and B genomes, it is relatively quick and easy to introgress resistance genes into common wheat from durum wheat, compared to interspecific hybridization with most other wheat relatives. In this study, we combined chromosome counting and rust evaluation in the F_3 generation to develop a mapping population at the hexaploid level. This approach allowed us to map the gene and transfer the gene into a common wheat background simultaneously. Common wheat lines carrying *Yr53* showed the same level of resistance as the durum donor and will be easily integrated into breeding programs. Limited seed will be available from late 2012.

Although we have not found any *Pst* races virulent to *Yr53*, we consider it race-specific seedling resistance gene as it is expressed at the seedling stage under low temperatures and is also expressed at later growth stages. Further work is needed to test lines with *Yr53* in other parts of the world to determine if there are existing races with virulence to the gene. Before such information is available, the gene can be used in breeding programs, but with caution. It should be used in combination with other effective seedling resistance genes such as *Yr5*, *Yr15* and *Yr45* (Yan et al. 2003; Chen et al. 2010; Murphy et al. 2009; Li et al. 2010) and also in combination with non-race-specific HTAP resistance genes such as *Yr18*, *Yr29*, *Yr36*, *Yr39*, *Yr52* as well as others (Chen 2005, 2012, Ren et al. 2012) as molecular makers are available for these genes to assist in transferring and combining them into elite varieties.

Gene stacking has been proposed for using different genes to achieve durable resistance. Stacking genes from different sources but located on the same chromosome or chromosomal arm is even more useful because the genes will be more likely retained in coupling in a breeding program (Hu and Hulbert 1996). Both *Yr5* and *Yr53* are located on the long arm of chromosome 2B and each provides effective resistance against all *Pst* races in the US and probably other countries. Races virulent to *Yr5* are not prevalent but have been identified in India and Australia (Nagarajan et al. 1986; Wellings and McIntosh 1990). This makes *Yr53* and *Yr5* good candidates for combination. With this in mind, we made a cross between an *Yr53* line and an *Yr5* line, both in the AvS common spring wheat background. From the F_3 lines of this cross, we identified at least three lines with the combination of *Yr53* and *Yr5* at the homozygous state with molecular markers and a race test. These lines should be more useful in breeding programs to develop wheat cultivars with high-level durable resistance to stripe rust.

One of the major obstacles for marker-assisted selection is the lack of polymorphism of marker loci between the resistance donor and elite breeding lines. We tested three closely linked markers, *XLRRrev/NLRRrev*$_{350}$, *Xwmc441* and *STS2F/1R*$_{219}$, flanking the *Yr53* locus with a sample of 54 wheat cultivars and breeding lines. The markers exhibited high levels of polymorphism among the wheat genotypes. When they were used together, the combination differentiates the *Yr53* chromosomal regions in all tested wheat genotypes. Although we have not successfully

converted the RGAP marker *XLRRrev/NLRRrev*$_{350}$ into a STS marker, it is usable with polyacrylamide gel electrophoresis and could be used with other systems. We successfully converted *Ptokin2/Xa1NBS-F*$_{234}$ into a STS marker, *STS2F/1R*$_{219}$. This marker is only 1.4 cM further away from the gene than *XLRRrev/NLRRrev*$_{350}$. Compared to the original RGAP marker, the STS marker is relatively robust and easy to score. The three markers should be useful in marker-assisted selection for incorporating *Yr53* into elite wheat lines to develop wheat cultivars with effective resistance to stripe rust.

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