Molecular Mapping of *YrSP* and Its Relationship with Other Genes for Stripe Rust Resistance in Wheat Chromosome 2BL

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ABSTRACT

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Stripe rust, caused by *Puccinia striiformis* f. sp. *tritici*, is an important disease of wheat worldwide. Resistance is the best way to control the disease. *YrSP*, a gene originally from ‘Spaldings Prolific’ wheat and providing resistance to a broad spectrum of races, is used for differentiating *P. striiformis* f. sp. *tritici* races but its chromosomal location is not clear. To map *YrSP*, a near-isogenic line (AvSYrSPNIL) was backcrossed to the recurrent parent, Avocet S. Genetic analysis of the BC₇F₁, BC₈, BC₇F₂, and BC₇F₃ progenies confirmed a single dominant gene for resistance. In total, 182 BC₇F₂ plants and their derived BC₇F₃ lines were phenotyped with an avirulent *P. striiformis* f. sp. *tritici* race and genotyped with simple-sequence repeat (SSR), single-nucleotide polymorphism (SNP), and sequence-tagged

site (STS) markers. A linkage map was constructed with 3 SSR, 17 SNP, and 3 STS markers covering 23.3 centimorgans (cM). Markers *IWA638* and *dp269* were 0.6 cM proximal and 1.5 cM distal, respectively, to *YrSP*. The gene was mapped in chromosome bin 2BL-C-0.5, physically within the proximal 50% of the chromosome 2BL arm. Allelism tests based on F₂ phenotypes indicated that *YrSP* is closely linked to but not allelic with genes *Yr5*, *Yr7*, *Yr43*, *Yr44*, and *Yr53*. Infection type data from tests with 10 historical and currently predominant *P. striiformis* f. sp. *tritici* races in the United States also demonstrated differences in specificity between *YrSP* and the other genes. The specificity of *YrSP* is useful in differentiating *P. striiformis* f. sp. *tritici* races and studying the plant–pathogen interactions, and the information of chromosomal location of the gene and its tightly linked markers should be useful in developing resistant cultivars when combined with other genes for resistance to stripe rust.

Wheat stripe rust, caused by *Puccinia striiformis* Westend. f. sp. *tritici* Erikss., is one of the most destructive fungal diseases of wheat worldwide (Stubbs 1985; Wellings 2011). The disease usually occurs in cool and temperate regions and significantly affects yield and grain quality when wheat varieties are severely infected (Chen 2005, 2014). Many measures have been used to control the disease, including cultural practices and chemical application. Compared with these methods, resistant cultivars are considered the most effective and economic means to control the disease (Chen 2005, 2013, 2014; Line 2002).

Sets of differential lines with different resistance genes are used for identifying *P. striiformis* f. sp. *tritici* races. In order to improve differentiation and maintain relevance over time, genotypes in a differential set may be changed or supplemented when new races cannot be differentiated using preexisting sets. Gassner and Straib (1932) proposed the first differential set, which included 15 varieties of common wheat, *Triticum dicoccum*, barley, and rye for differentiating races of *P. striiformis*. Although this set of differentials of multiple crops was used up to the 1980s, it did not adequately differentiate races of either *P. striiformis* f. sp. *tritici* or the barley

stripe rust pathogen (*P. striiformis* f. sp. *hordei*) (Chen et al. 1995; Stubbs 1985). Johnson et al. (1972) introduced a dual set of only wheat genotypes described as “world” and “European” differentials, the latter including ‘Spaldings Prolific’ (*YrSP*). This set of differentials was adopted in many countries in Europe and other continents (de Vallavieille-Pope and Line 1990; Wellings and McIntosh 1990). In China, India, and the United States, different sets of wheat genotypes were selected for differentiating *P. striiformis* f. sp. *tritici* races. The use of different differential sets in different countries has both advantages and disadvantages. Whereas a common set enables closer comparisons globally, more local sets tend to maintain historical continuity within regions and can be managed with greater relevance to the host cultivar genetic variation within those regions. An overriding problem with both approaches is the incomplete knowledge of the genetic basis of resistance both within and between regions. In order to achieve a greater genetic relevance, Wan and Chen (2014) recently established a new set of wheat differentials based on the Avocet S (AvS) near-isogenic lines developed in Australia, as well as single gene lines developed in the United States.

Spaldings Prolific, with resistance gene *YrSP*, has been used to differentiate races in Europe and elsewhere (Johnson et al. 1972; Stubbs 1988; Wellings and McIntosh 1990). Stripe rust resistance in the wheat genotype has been studied for more than 60 years. Manners (1950) and Stubbs (1967) reported that light and temperature affected the stripe rust reaction of Spaldings Prolific. de Vallavieille-Pope and Line (1990) observed that Spaldings Prolific had low infection types (IT) when scored earlier and high IT when scored later in monocyclic inoculation tests with some U.S. races. Straib (1934) and Allan and

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Purdy (1970) reported that resistance in Spaldings Prolific to European race 4 (40E8 or 104E9) and North American race 61-1 was conferred by a single dominant gene in each instance. In contrast, Calon nec et al. (1996) reported three genes for resistance in Spaldings Prolific, all three conferring resistance to race 2E16 and only two of them effective against race 43E138. A gene for resistance to stripe rust in Spaldings Prolific, designated as *YrSP*, was tentatively located on chromosome 2BS (Gosal 2000; McDonald et al. 2004), and Guan et al. (2005) mapped a gene (*YrSpP*) in the same variety on chromosome 2BL. We presume that *YrSP* and *YrSpP* are the same gene even if the chromosomal location is unclear (McIntosh et al. 2013).

The *YrSP* near-isogenic line AvSYrSPNIL was developed by backcrossing Spaldings Prolific and AvS in Australia (Wellings et al. 2004). AvSYrSPNIL was resistant (IT 0 to 2, severity 0 to 5%) in our germplasm nurseries and similar in response to Spaldings Prolific in various locations in the United States from 2003 to 2006, except for a reaction of IT 5 and 10% severity on Spaldings Prolific at Pendleton, OR in 2006. Susceptible reactions on both host lines appeared first in California in 2007 and, since then, a range of reactions in different locations was observed, with 100% severity at some locations near Pullman, WA in 2011 and 2012 (X. M. Chen, unpublished data). Since AvSYrSPNIL was included in the new set of *Yr* single-gene differentials in 2010, the frequencies of virulence to *YrSP* have ranged from 3.4% in 2010 to 9.8% in 2013 (Wan and Chen 2014) (A. M. Wan and X. M. Chen, unpublished data). Among the 84 races identified in the United States with the new set of 18 *Yr* single-gene differentials, 9 (10.7%) were virulent to *YrSP*; and, of 55 races identified in other countries, 8 (14.5%) were virulent (Sharma-Poudyal et al. 2013; Wan and Chen 2014) (A. M. Wan and X. M. Chen, unpublished data). The gene is still useful in developing stripe-rust-resistant cultivars in combination with other resistance genes. The objectives of this study were to map *YrSP* in AvSYrSPNIL and to determine its relationship to other *Yr* genes mapped to chromosome 2BL. The tightly linked markers for *YrSP* should be useful in marker-assisted selection (MAS) for pyramiding the gene with other effective resistance genes to develop wheat cultivars with durable resistance to stripe rust.

MATERIALS AND METHODS

Plant materials and development of genetic populations.

Seed of AvSYrSPNIL, a BC₆ line developed by backcrossing Spaldings Prolific (the resistant donor parent) to AvS in the Plant Breeding Institute, University of Sydney, were kindly provided by Dr. Colin Wellings. AvS, an Australian spring wheat genotype, is susceptible to almost all *P. striiformis* f. sp. *tritici* races identified in the United States and many other countries (Sharma-Poudyal et al. 2013; Wan and Chen 2014).

In order to study the genetic basis of resistance in AvSYrSPNIL, a cross was made between AvS and AvSYrSPNIL. BC₇F₂ seed harvested from a single BC₇F₁ plant were planted in the greenhouse, and 182 BC₇F_{2:3} lines were produced. A further backcross was made to AvS and BC₈F₂ lines were produced in the greenhouse. Two parents, BC₇F₁, BC₇F₂, BC₇F₃, and BC₈F₂ seedling lines were tested in the greenhouse to determine the inheritance of resistance to stripe rust in AvSYrSPNIL.

Because *Yr5*, *Yr7*, *Yr43*, *Yr44*, *Yr53*, and *YrSP* were previously reported on chromosome 2B (McIntosh et al. 1995, 2013), lines AvSYr5NIL (*Yr5*), AvSYr7NIL (*Yr7*), IDO377s (*Yr43*), Zak (*Yr44*), and AvS/PI 480148F₇-128 (*Yr53*) were crossed with AvSYrSPNIL and F₂ populations of 481 to 544 plants from each of these crosses were used to test for linkage with *YrSP*. 'Chinese Spring' (CS) and its nullitetrasonic or monotetrasonic lines, ditelosomic 2BL line, and 2BL deletion lines (Del2BL-4, Del2BL-5, Del2BL-6, and Del2BL-10) (Endo and Gill 1996; Sears 1966; Sears and Sears 1978) were used in marker tests to localize the specific position of *YrSP*. In total, 77 wheat cultivars and breeding lines, which do not have *YrSP* based on their pedigrees and tests with

P. striiformis f. sp. *tritici* races virulent or avirulent to the gene, were tested with the markers flanking *YrSP* to determine their usefulness in MAS for incorporating the gene into various wheat backgrounds.

Stripe rust tests. Ten *P. striiformis* f. sp. *tritici* races, representing predominant U.S. races since the 1960s (Line and Qayoum 1992; Wan and Chen 2012), and race PST-151, which has the widest virulence range, were used to test AvS and AvSYrSPNIL for reaction to stripe rust (Table 1). Race PST-100 (avirulent on AvSYrSPNIL) was used to test the various segregating materials. Urediniospores of each isolate were increased on AvS, and its virulence and avirulence pattern was confirmed by testing on the 20 U.S. differential lines following procedures as previously described (Wan and Chen 2012). Inoculations were made with fresh urediniospores mixed with talc at a 1:20 ratio when plants were at the two-leaf stage. The inoculated plants were kept in a dew chamber for 24 h at 10°C and then moved to a growth chamber, with the low temperature profile set with a diurnal temperature cycle gradually changing between 4°C at 2:00 A.M. and 20°C at 2:00 P.M. (Chen and Line 1995). IT were recorded using the 0-to-9 scale described by Line and Qayoum (1992) 20 days after inoculation.

DNA extraction and polymerase chain reaction amplification. Genomic DNA was extracted from leaf tissue of each BC₇F₂ plant or parent using the cetyltrimethylammonium bromide method (Riede and Anderson 1996). DNA was dissolved in Tris-EDTA buffer (10 mM Tris-HCl and 1 mM EDTA, pH 8.0). DNA quality was determined using 1.0% agarose gel electrophoresis, and the DNA concentration was measured using NanoDrop ND-1000 (Thermo Scientific, Wilmington, DE). The stock DNA solution was diluted to 25 ng/μl with sterile deionized distilled (dd)H₂O for use as template in polymerase chain reaction (PCR).

Simple-sequence repeat (SSR) markers were used to map *YrSP*. PCR was performed in volumes of 12 μl comprising 4 μl (100 ng) of template DNA, 1.2 μl of 10× buffer (with 1.5 mM MgCl₂), 0.48 μl of MgCl₂ (25 mM), 0.96 μl of mixed dNTP with 2.5 mM each, 0.24 μl of M13 primer (10 μM) with fluorescent dye, 0.12 μl of Taq DNA polymerase (5 U/μl), 0.06 μl of forward primer with M13 tail (10 μM), 0.3 μl of reverse primer (10 μM), and 4.64 μl of sterilized ddH₂O. Buffer, dNTP, and Taq DNA polymerase were purchased from Promega Corp. (Madison, WI). M13 fluorescent primers were

TABLE 1. Infection types of Avocet S (AvS) and AvSYrSPNIL (*YrSP*) produced by 10 races of *Puccinia striiformis* f. sp. *tritici* under controlled conditions

Race	Virulence formula ^b	Infection type ^a	
		AvS	<i>YrSP</i>
PST-3	1,3	8	1
PST-17	1,2,3,9,11	8	1
PST-43	1,3,4,5,12,14	8	1
PST-45	1,3,12,13,15	8	1
PST-78	1,3,11,12,16,17,18,19,20	8	1
PST-100	1,3,8,9,10,11,12,16,17,18,19,20	8	1
PST-114	1,3,4,8,9,10,11,12,14,16,17,18,19,20	8	1
PST-116	1,3,4,5,8,9,10,11,12,14,16,17,18,19,20	8	1
PST-127	1,2,3,5,6,8,9,10,11,12,13,15,16,17,18,19,20	8	8
PST-139	1,2,3,5,6,8,9,10,11,12,13,15,17,18,19	8	1
PST-151	1,2,3,4,5,6,8,9,10,11,12,13,14,15,16,17,18,19,20	8	8

^a Infection types were scored based on a 0-to-9 scale (Line and Qayoum 1992).

^b Virulence formulae are based on reactions on the following 20 wheat genotypes used to differentiate races of *P. striiformis* f. sp. *tritici* in the United States: 1 = Lemhi (*Yr21*), 2 = Chinese 166 (*Yr1*), 3 = Heines VII (*Yr2* and *YrHVII*), 4 = Moro (*Yr10* and *YrMor*), 5 = Paha (*YrPa1*, *YrPa2*, and *YrPa3*), 6 = Druchamp (*Yr3a*, *YrD*, and *YrDru*), 7 = AvSYr5NIL (*Yr5*), 8 = Produra (*YrPr1* and *YrPr2*), 9 = Yamhill (*Yr2*, *Yr4a*, and *YrYam*), 10 = Stephens (*Yr3a*, *YrS*, and *YrSte*), 11 = Lee (*Yr7*, *Yr22*, and *Yr23*), 12 = Fielder (*Yr6* and *Yr20*), 13 = Tyee (*YrTye*), 14 = Tres (*YrTr1* and *YrTr2*), 15 = Hyak (*Yr17* and *YrTye*), 16 = Express (*YrExp1* and *YrExp2*), 17 = AvSYr8NIL (*Yr8*), 17 = AvSYr9NIL (*Yr9*), 19 = Clement (*Yr9* and *YrCle*), and 20 = Compair (*Yr8* and *Yr19*) (Chen et al. 2010; Wan and Chen 2012).

ordered from Applied Biosystems (Grand Island, NY). SSR primers developed by Röder et al. (1998) and Somers et al. (2004) were synthesized by Sigma-Aldrich Inc. (St. Louis).

PCR was performed in an ABI-9700 thermal cycler, programmed for 5 min of initial denaturation followed by 42 cycles, each cycle consisting 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 of 10 min at 72°C. PCR products were detected on ABI capillary DNA sequencer 3730 (Applied Biosystems, Foster City, CA), and the genotypes were analyzed using GeneMarker (V2.2; SoftGenetics, LLC., State College, PA).

Bulk segregant analysis was used in initial SSR marker screening. Based on the IT data of BC₇F₂ individual plants and corresponding BC₇F₃ lines, 10 homozygous resistant and 10 homozygous susceptible BC₇F₂ plants were selected to make resistant and susceptible bulks, respectively. The resistant and susceptible bulks were constructed with equal amount of DNA, as described previously (Michelmore et al. 1991). The DNA samples from AvSYrSPNIL, AvS, and the two bulks were used for screening markers associated with *YrSP*.

To increase the marker intensity on wheat chromosome 2B, the wheat 9K single-nucleotide polymorphism (SNP) chip was used to screen AvSYrSPNIL and AvS, as described by Cavanagh et al. (2013). The wheat 9K SNP detection was conducted at the United States Department of Agriculture–Agricultural Research Service (USDA-ARS) Bio-Sciences Research Laboratory, Fargo, ND. In total, 40 polymorphic SNP markers were identified and used to test the 182 BC₇F₂ plants using a Sequenom MassArray system at the USDA-ARS, Wheat Genetics, Quality, Physiology, and Disease Research Unit (Pullman, WA).

Data analyses. Goodness-of-fit of observed data to theoretically expected segregation ratios was assessed using χ^2 tests. The IT data for resistant $\times$ resistant crosses were used to calculate the genetic distance between two genes according to the Kosambi formulae: $f = p^2/4$ and $d = 1/4 \ln[(1 + 2p)/(1 - 2p)]$, where f is the observed frequency of susceptible F₂ plants, p is the recombination value, and d is the map distance in morgans (Kosambi 1943). The standard error was estimated as the square root of $dx(1 - d)/n$, where n is the total number of F₂ plants in the test.

Linkage analysis was conducted based on marker genotypes and response phenotypes of BC₇F_{2,3} lines using software JoinMap 4.0 (Voorrips 2002). Markers were placed in the same linkage group, with a log of the likelihood threshold of 3.0. The Kosambi mapping

function was used to convert recombination values to map distances (Kosambi 1943). The genetic map was drawn using software Mapdraw V2.1 (Liu and Meng 2003).

RESULTS

Genetic analysis of stripe rust resistance in AvSYrSPNIL. AvSYrSPNIL was highly resistant (IT 1) to all races, except PST-127 and PST-151, to which it was susceptible (IT 8). AvS was susceptible (IT 8) to all races (Table 1).

In the test with PST-100, the BC₇F₁ plants were resistant, showing necrotic spots with trace to light sporulation (IT 3 to 4), indicating that the resistance in AvSYrSPNIL was partially dominant. The segregations of the BC₇F₂ population and its derived BC₇F₃ population showed that the resistance in AvSYrSPNIL was controlled by a single gene (Table 2).

Molecular markers and linkage map. The location of *YrSP* was previously reported as chromosome 2B but the chromosome arm is unclear. Therefore, SSR markers covering chromosome 2B and sequence-tagged site (STS) markers linked to the *Yr5* locus on the long arm of chromosome 2B (2BL) were screened on the parents and bulks to identify those associated with the *YrSP* locus. Of 62 SSR markers and 10 STS markers screened, 3 SSR (*Xwmc441*, *Xgwm388*, and *Xwmc332*) and 3 STS (*PWC17/18*, *dp269*, and *Yr5STS7/8*) markers were associated with the *YrSP* locus (Table 3). All six markers were codominant (Table 4). As an example, the fragments of *Xwmc441*, 181 bp in AvSYrSPNIL and 191 bp in AvS, are shown in Supplementary Figure S1. Because the three SSR markers were located in 2BL (Somers et al. 2004), we presumed that *YrSP* was also located in chromosome 2BL (Fig. 1A).

To determine the within-chromosomal location of *YrSP*, the six markers were screened on CS and its 21 nulli- (or mono)tetrasomic lines, ditelosomic 2BL line, and 4 2BL deletion lines (del2BL-4, del2BL-5, del2BL-6, and del2BL-10). The CS alleles of the six markers were detected in all of these lines, except N2BT2A-CS (Supplementary Table S1). The results confirmed the location on 2BL and indicated that *YrSP* was in chromosome bin 2BL-C-0.5 (Fig. 1B).

To identify more closely linked markers, AvS and AvSYrSPNIL were screened for SNP markers using the wheat 9K SNP chip. In all, 40 SNP markers (0.44%) were polymorphic and 11 (IWA series) located on 2BL were selected to test the BC₇F₂ plants. All except

TABLE 2. Reactions of parents and progenies of cross AvS $\times$ AvSYrSPNIL tested with race PST-100 in seedling stage under controlled conditions and χ^2 tests^a

Generation	Number of plants or lines			Expected ratio (Res:Sus or Res:Seg:Sus)	χ^2	<i>P</i>
	Res	Seg	Sus			
AvSYrSPNIL (BC ₆)	15	0	0	1:0	NA	NA
AvS	0	0	16	0:1	NA	NA
BC ₇ F ₁	3	0	0	1:0	NA	NA
BC ₇ F ₂	134	0	48	3:1	0.18	0.67
BC ₇ F ₃	44	90	48	1:2:1	0.20	0.91
BC ₈	14	0	16	1:1	0.13	0.72

^a Res = resistant, Seg = segregating, Sus = susceptible, and NA = not applicable.

TABLE 3. Simple-sequence repeat (SSR) primers and *Yr5* sequence-tagged site (STS) primers used to identify markers for the stripe rust resistance gene in the population of cross Avocet S $\times$ AvSYrSPNIL

Primer	Primer sequence (5'–3')		Tm (°C) ^a	Reference
	Forward	Reverse		
<i>Xwmc441</i>	TCCAGTAGAGCACCTTTTCATT	ATCACGAAGATAAACAAACGG	61	Somers et al. 2004
<i>Xgwm388</i>	CTACAATTCTGAAGGAGAGGGG	CACCGCGTCAACTACTTAAGC	60	Röder et al. 1998
<i>Xwmc332</i>	CATTTACAAAGCGCATGAAGCC	GAAACTTTGGGAACAAGAGCA	61	Somers et al. 2004
<i>dp269</i>	CTGCTGTACCGCTCTCC	AGTCACACGCCCTACTCTCC	55	Fang et al. 2008
<i>Yr5STS7/8</i>	GTACAATTCACCTAGAGT	GCAAGTTTCTCCCTATT	56	Chen et al. 2003
<i>PWC17/18</i>	GGAACTAAGTCTGGCAGC	CCGGTATTATCTGTATTCC	55	This study

^a Annealing temperature.

TABLE 4. Fragment sizes or polymorphic nucleotide sites of markers linked to the *YrSP* locus and χ^2 tests of markers segregating in the mapping population of AvS $\times$ AvSYrSPNIL

Marker ^b	Primer pair	Size (bp) or nucleotide ^a		χ^2 (1:2:1)	P value
		AvSYrSPNIL	AvS		
<i>Xwmc441</i>	WMC441F/R	181	191	4.17	0.12
<i>PWC17/18</i>	PWC17/18F/R	242	265	5.42	0.07
<i>dp269</i>	dp269 F/R	198	209	2.38	0.30
<i>Yr5STS7/8</i>	Yr5STS8/7 F/R	505	492	1.57	0.46
<i>Xgwm388</i>	GWM388 F/R	230	226	3.95	0.14
<i>Xwmc332</i>	WMC332 F/R	184	191	7.04	0.03
IWA638	wsnp_BQ172173B_Aa_2_2	T	C	2.48	0.29
IWA2676	wsnp_Ex_A2203_4129271	T	C	1.48	0.48
IWA2678	wsnp_Ex_A2203_4130096	T	C	1.60	0.45
IWA2872	wsnp_Ex_B24135_33382318	T	C	2.09	0.35
IWA3075	wsnp_Ex_B26818_36041748	T	C	2.09	0.35
IWA4866	wsnp_Ex_c9025_15039930	T	G	3.13	0.21
IWA4900	wsnp_Ex_c9345_15516291	G	A	1.60	0.45
IWA6121	wsnp_JD_A6010_7167084	C	T	1.60	0.45
IWA6561	wsnp_Ku_c15498_24122936	G	A	2.64	0.27
IWA4909	wsnp_Ex_B942_1806632	T	C	0.52	0.77
IWA6122	wsnp_JD_A6010_7167159	C	T	1.17	0.56

^a Sizes of SSR and STS markers include the 19-bp M13 sequence.

^b *Xwmc441*, *Xgwm388*, and *Xwmc332* are SSR markers (Röder et al. 1998; Somers et al. 2004); *PWC17/18* (M. N. Wang, unpublished), *Yr5STS7/8* (Chen et al. 2003), and *dp269* (Fang et al. 2008) are STS markers tightly linked to *Yr5*; and those with prefix IWA are single-nucleotide polymorphism (SNP) markers (Cavanagh et al. 2013).

Fig. 1. Linkage map and chromosomal location of *YrSP* and comparisons with different linkage maps of other *Yr* genes on the long arm of 2B (2BL). A, Consensus map of 2BL (Somers et al. 2004); B, deletion map of 2BL (Sourdille et al. 2004); C, Linkage map of *YrSP*; D, linkage map of *Yr7* (Yao et al. 2006); E, linkage map of *Yr5* (Murphy et al. 2009); F, linkage map of *Yr43* (Cheng and Chen 2010); G, linkage map of *Yr44* (Sui et al. 2009; Cheng and Chen 2010); and H, linkage map of *Yr53* (Xu et al. 2013). Dotted lines connected the same markers in different maps.

Xwmc332 were normally inherited and linked with the *YrSP* locus and, hence, were suitable for linkage mapping (Table 4).

All markers listed in Table 4 were used to construct a linkage map for the *YrSP* locus based on the BC₇F₂ genotypes (Fig. 1C). The nearest markers flanking *YrSP* were *IWA638* (0.6 centimorgans [cM] proximal) and *dp269* (1.5 cM distal).

Relationship of *YrSP* with other *Yr* genes on 2BL. To date, five permanently named *Yr* genes—*Yr5*, *Yr7*, *Yr43*, *Yr44*, and *Yr53*—have been mapped to chromosome 2BL. In order to determine the relationship of *YrSP* with these genes, the reference lines were compared for differential reactions to *P. striiformis* f. sp. *tritici* races, tests of allelism, and closely linked SNP markers.

Five genotypes—AvSYr5NIL (*Yr5*), AvSYr7NIL (*Yr7*), IDO377s (*Yr43*), Zak (*Yr44*), AvS/PI 484480F₇-128 (*Yr53*), and AvSYrSPNIL (*YrSP*)—were tested with 10 selected *P. striiformis* f. sp. *tritici* races. AvSYr5NIL and AvS/PI 484480F₇-128 were resistant to all races but with slightly different ITs. AvSYr7NIL was susceptible to all races, except resistant to PST-3. IDO377s was resistant to six races and Zak was resistant to three races. AvSYrSPNIL was resistant to all races, except PST-127 (Table 5). These results indicated that *YrSP* differed in specificity from the other five genes.

Susceptible F₂ plants were identified in all five crosses between AvSYrSPNIL and each of the five lines. The segregations of resistant to susceptible plants in the F₂ populations of these crosses did not fit ($P < 0.0001$) a 15:1 ratio, which is expected for two independent genes on different chromosomes, and, thus, indicated that *YrSP* was linked to all of the other five genes involved in the populations. Based on the

frequencies of the susceptible plants, the genetic distances between *YrSP* and the other genes were estimated to be 12.6 to 18.6 cM (Table 6).

To further compare *YrSP* with the other *Yr* genes on chromosome 2BL, AvSYrSPNIL (*YrSP*), *T. spelta* album (*Yr5*), AvSYr5NIL (*Yr5*), AvSYr7NIL (*Yr7*), IDO377s (*Yr43*), Zak (*Yr44*), *T. durum* PI 480148 (*Yr53*), and AvS/PI 480148 F₇-128 (*Yr53*) were tested with six SNP markers linked with *YrSP*. Markers *IWA2872*, *IWA3973*, and *IWA6561* distinguished AvSYrSPNIL from all other lines, except IDO377s and Zak (Table 7). Markers *IWA1489* and *IWA6317* shared the same allele in AvSYrSPNIL, AvSYr7NIL, and Zak but different alleles were present in the other gene lines. Marker *IWA6122* could not differentiate among AvSYrSPNIL, *T. spelta* album, and AvSYr7NIL but could differentiate AvSYrSPNIL from the remaining lines. Thus, when used in combination, these SNP markers could be used to distinguish AvSYrSPNIL from lines carrying the other *Yr* genes on 2BL. The SNP marker haplotypes, along with the response spectra and allelism tests, indicated that *YrSP* was different from other named *Yr* genes in chromosome 2BL.

Polymorphism of *YrSP* markers in different wheat backgrounds. To determine how useful markers *Xwmc332*, *dp269*, and *Xwmc441* might be in MAS to incorporate *YrSP* to wheat cultivars, 77 wheat cultivars and breeding lines were assayed with the markers. The specific band of *dp269* associated with *YrSP* was present in 28 genotypes and absent in 49 (Supplementary Table S2). For *Xwmc441*, eight genotypes had the target amplicon (89.61% polymorphic). The expected amplicon of *Xwmc332* was not present in 66 genotypes. Only two lines (OR2080156H and

TABLE 5. Infection types of wheat lines with *Yr* genes located in the long arm of chromosome 2B tested with 10 races of *Puccinia striiformis* f. sp. *tritici* in the greenhouse

Genotype	<i>Yr</i> gene	Infection types produced by <i>P. striiformis</i> f. sp. <i>tritici</i> races ^a									
		PST-3	PST-17	PST-43	PST-45	PST-78	PST-100	PST-114	PST-116	PST-127	PST-139
AvSYr5NIL	<i>Yr5</i>	1	1	1	1	1	1	1	1	1	1
AvSYr7NIL	<i>Yr7</i>	2	8	8	8	8	8	8	8	8	8
IDO377s	<i>Yr43</i>	2	2	2	8	2	2	2	8	8	8
Zak	<i>Yr44</i>	2	8	2	2	8	8	8	8	8	8
AvS/PI480148F ₇ -128	<i>Yr53</i>	2	2	2	2	2	2	2	2	2	2
AvSYrSPNIL	<i>YrSP</i>	1	1	1	1	1	1	1	1	8	1
AvS	None	8	8	8	8	8	8	8	8	8	8

^a Infection types were scored based on a 0-to-9 scale (Line and Qayoum 1992).

TABLE 6. Reactions of F₁ and F₂ progenies in allelism tests between the wheat lines with *YrSP* and five *Yr* genes located in the long arm of chromosome 2B

Cross ^b	Reaction of F ₁ plants	Number of F ₂ plants ^a			χ^2 (15:1)	<i>P</i>	Recombinant rate	Distance (cM)
		Total	Res	Sus				
<i>YrSP/Yr5</i>	R	522	520	2	30.7	3.0×10^{-8}	0.124	12.6 ± 1.5
<i>YrSP/Yr7</i>	R	506	502	4	25.7	3.9×10^{-7}	0.178	18.6 ± 1.7
<i>YrSP/Yr43</i>	R	544	541	3	30.1	4.0×10^{-8}	0.149	15.3 ± 1.5
<i>YrSP/Yr44</i>	R	535	532	3	29.6	5.4×10^{-8}	0.15	15.5 ± 1.6
<i>YrSP/Yr53</i>	R	481	479	2	27.9	1.3×10^{-7}	0.129	13.2 ± 1.5

^a Res = resistant and Sus = susceptible.

^b F₁ and F₂ plants of all of the five crosses were tested with PST-3 that is avirulent to all of the *Yr* genes.

TABLE 7. Single-nucleotide polymorphism (SNP) genotypes of AvSYrSPNIL and other wheat lines carrying *Yr* genes in the long arm of chromosome 2B

Genotypes	<i>Yr</i> genes	Genotype at SNP marker loci ^a					
		<i>IWA2872</i>	<i>IWA3973</i>	<i>IWA6561</i>	<i>IWA1489</i>	<i>IWA6317</i>	<i>IWA6122</i>
AvSYrSPNIL	<i>YrSP</i>	A	A	A	A	A	A
AvS	None	B	B	B	B	B	B
<i>Triticum spelta</i> album	<i>Yr5</i>	B	B	B	B	B	A
AvSYr5NIL	<i>Yr5</i>	B	B	B	B	B	B
AvSYr7NIL	<i>Yr7</i>	B	B	B	A	A	A
IDO377s	<i>Yr43</i>	A	A	A	B	B	B
Zak	<i>Yr44</i>	A	A	A	A	A	B
PI 480148	<i>Yr53</i>	B	B	B	B	B	B
AvS/PI 480148 F ₇ -128	<i>Yr53</i>	B	B	B	B	B	B

^a A and B are used to designate the nucleotide type of AvSYrSPNIL and AvS, respectively.

OR2080228H) lacking *YrSP* had all three expected amplicons, indicating that, when the markers are used together, there would be a 97.40% likelihood of identifying *YrSP* in wheat germplasm.

The wheat cultivars or breeding lines were also tested with five *P. striiformis* f. sp. *tritici* races in the seedling stage in the greenhouse. All wheat cultivars or lines had reaction patterns different from AvSYrSP-NIL, except Glee, WB Hartline, and Malbee, which had patterns similar to AvSYrSP-NIL. Lines OR2080156H and OR2080228H, with marker alleles in common with AvSYrSP-NIL, had different reaction patterns. Thus, the combination of the marker and race test data indicates that none of the wheat cultivar or lines has *YrSP*.

DISCUSSION

Spaldings Prolific is one of the earliest wheat varieties used to differentiate *P. striiformis* f. sp. *tritici* races (Gassner and Straib 1932; Stubbs 1985). The variety was reported to have a single gene (Allan and Purdy 1970; Straib 1934) or three genes (Calonnec et al. 1996) for resistance to stripe rust. Several studies attempted to find the chromosomal location of the single gene but whether the gene is in the long arm or short arm of chromosome 2B was not clear (Gosal 2000; Guan et al. 2005; McIntosh et al. 2013). Carrying a single gene for stripe rust resistance, the near-isogenic line AvSYrSP-NIL derived from Spaldings Prolific has been selected as a differential to identify *P. striiformis* f. sp. *tritici* races (Wan and Chen 2014). The near-isogenic line is preferred over Spaldings Prolific for mapping the *YrSP* gene. In the present study, we confirmed that the line has a partially dominant gene conferring a high level of resistance against avirulent *P. striiformis* f. sp. *tritici* races. We constructed a high-resolution map with 23 markers covering 23.3 cM, with the flanking markers being 0.6 cM proximal and 1.5 cM distal to *YrSP*. Using CS deletion lines, we placed the gene in chromosome bin 2BL-C-0.5. Based on differential rust responses, allelism tests, and molecular markers, *YrSP* is different from previously reported genes in chromosome 2BL.

Yan et al. (2003) mapped *Yr5* and Yao et al. (2006) reported *Yr7* in chromosome 2BL (Fig. 1D and E). *Yr7* is linked to SSR marker *Xgwm526* at a genetic distance of 5.6 cM (Fig. 1D) (Yao et al. 2006). Because this marker is most distal in the consensus 2BL map (Somers et al. 2004) and not present in the *YrSP* map, the distance between *Yr7* and *YrSP* could not be accurately determined. Zhang et al. (2009) found no susceptible line among 143 F_3 lines of a cross between AvSYr7NIL (*Yr7*) and AvSYr5NIL (*Yr5*). They found that STS marker *Yr5STS9/10*, which is the same as *Yr5STS7/8* (Chen et al. 2003), is also in the *Yr7* line but of different alleles. Their data confirmed the location of *Yr7* in chromosome 2BL and tight linkage to the *Yr5STS7/8* or *Yr5STS9/10* marker locus. Based on these results, they concluded that *Yr7* and *Yr5* were allelic or tightly linked. Using 506 F_2 plants from a cross of AvSYrSP-NIL and AvSYr7NIL, we found four susceptible plants, suggesting linkage of 18.6 cM between *YrSP* and *Yr7*. In our map, *Yr5STS7/8* was 2.3 cM distal to *YrSP* (Fig. 1C). The *Yr5STS7/8* and *Yr5STS9/10* markers were converted from a single resistance gene analog polymorphic marker that was cosegregating with *Yr5* (Yan et al. 2003; Chen et al. 2003) but later found to be 0.3 cM from *Yr5* (Fig. 1F) (Murphy et al. 2009). *PWC1718* is an STS marker that was found to be closer to *Yr5* than *Yr5STS7/8* (M. N. Wang and X. M. Chen, unpublished data). In the present study, this marker was found 5.5 cM and the *Yr5STS7/8* marker 2.3 cM distal to the *YrSP* locus in the mapping population (Fig. 1C). The present linkage map of *YrSP* and the *Yr5* map of Murphy et al. (2009) share SSR marker *Xwmc411*, which is 3.6 cM proximal to *Yr5* and 11.4 cM proximal to *YrSP*. The linkage estimates indicate that *YrSP* and *Yr5* are different but closely linked. Among 520 F_2 plants of the AvSYrSP-NIL $\times$ AvSYr5NIL cross, we identified two susceptible plants, indicating linkage of 12.6 cM between *YrSP* and *Yr5*. Although this distance and that between *YrSP* and *Yr7* were larger than the distances based on the markers, the order of the genes is consistent. Therefore, the three resistance genes must be at different loci, with *Yr7* more distal

to *YrSP* than *Yr5*. The 11 SNP markers identified to be linked to *YrSP* were all mapped to chromosome 2B (Cavanagh et al. 2013). The order and distances among the markers in the present study were consistent with those reported by Cavanagh et al. (2013). The SNP markers confirmed the chromosomal location of *YrSP*.

Marker *Yr5STS7/8* is also linked to *Yr43*, *Yr44*, and *Yr53* (Fig. 1F–H). It was 33.6 cM from *Yr43* (Cheng and Chen 2010), 12.7 cM from *Yr44* (Sui et al. 2009), and 23.6 cM from *Yr53* (Xu et al. 2013). The marker was only 2.3 cM from *YrSP*. Based on this marker and other markers in the linkage map for *YrSP*, *Yr5STS7/8* was approximately 8.1 cM from *Yr43*, 6.0 cM from *Yr44*, and 18.6 cM from *Yr53*. In addition, *Xgwm388* was reported to be 22.6 cM from *Yr44* (Fig. 1G) (Sui et al. 2009) and *Xwmc411* was 5.6 cM from *Yr53* (Fig. 1H) (Xu et al. 2013). In our present map, *Xgwm388* and *Xwmc411* were 17.8 and 10.5 cM, respectively, from *YrSP*. The differences in distance could be due to different genetic backgrounds of the wheat lines. However, the gene order and relative distances were consistent. Although accurate distances among the genes and markers require further study, all these data indicate that *YrSP* is at a different locus from *Yr43*, *Yr44*, and *Yr53*.

In addition to the permanently named genes, Deng et al. (2004) located *YrQz* 5.66 cM from marker *Xgwm388* in chromosome 2B. Based on the much larger distance between *Xgwm388* and *YrSP* (17.6 cM) in the present study, *YrSP* is likely different from *YrQz*. *YrSP* has not been allocated a permanent gene designation because of conflicting data regarding its allelism, or otherwise closely linked with *Yr5* and *Yr7*. Although the present work strongly suggests that the genes are at different loci, there is already one report (Zhang et al. 2009) that the latter two genes are allelic. The present allelism tests were based on F_2 phenotypes and neither the resistant plants nor susceptible plants were progeny tested. New populations under increase will be tested as $F_{2:3}$ populations, a much more efficient process for assessing duplicate dominant genes in repulsion.

MAS has become increasingly important in breeding programs, because it permits breeders to combine genes for important traits, including multiple genes for resistance to a single disease, thereby contributing to both enhanced levels of resistance (in some situations) and resistance durability (Chen 2013; Cheng et al. 2014; Lu et al. 2014; Xu et al. 2013). However, one of the limitations of MAS for breeding is lack of closely linked diagnostic markers for targeted traits. In the present study, we identified several markers closely linked to *YrSP*. We tested *Xwmc332*, *dp269*, and *Xwmc441* in 77 wheat cultivars or lines that do not have the *YrSP* line or Spaldings Prolific in their pedigrees, and found that 97% of the cultivars or lines do not have at least one of the marker alleles identified in AvSYrSP-NIL. These markers in combination should be useful in MAS to combine *YrSP* with other effective genes. Although *YrSP* is race specific, it provides resistance to a broad range of *P. striiformis* f. sp. *tritici* races in the United States and other countries (Sharma-Poudyal et al. 2013; Wan and Chen 2014) and could be valuable when combined with other *Yr* genes but, if used alone, would be unlikely to provide durable resistance because of continuing reports of occasional virulent isolates in race surveys. Because the identified chromosome 2BL region contains several genes, we are currently attempting to combine *YrSP* with *Yr5* and *Yr53* in the same region because the latter two are effective against all *P. striiformis* f. sp. *tritici* races tested in the present and previous studies (Wan and Chen 2014; Xu et al. 2013; Yan et al. 2003). In addition to developing resistant cultivars, the results of the present study indicate that *YrSP* should be useful in differentiating *P. striiformis* f. sp. *tritici* races and studying the plant–pathogen interactions.

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