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ORIGINAL PAPER

Molecular mapping of genes *Yr64* and *Yr65* for stripe rust resistance in hexaploid derivatives of durum wheat accessions PI 331260 and PI 480016

P. Cheng · L. S. Xu · M. N. Wang · D. R. See · X. M. Chen

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Abstract

Key message **This manuscript reports two new genes (*Yr64* and *Yr65*) for effective resistance to stripe rust and usefulness of their flanking SSR markers for marker-assisted selection.**

Abstract Stripe rust, caused by *Puccinia striiformis* f. sp. *tritici* (*Pst*), is one of the most important diseases of wheat worldwide and resistance is the best control strategy. Durum wheat accessions PI 331260 and PI 480016 were resistant to all tested *Pst* races. To transfer the resistance genes to common wheat and map them to wheat chromosomes, both accessions were crossed with the stripe rust-susceptible spring wheat 'Avocet S'. Resistant F_3 plants with 42 chromosomes were selected cytologically and by rust phenotype. A single dominant gene for resistance was identified in segregating F_4 lines from each cross. F_6 populations for each cross were developed from single F_5 plants and used for genetic mapping. Different genes from PI 331260 and PI 480016 were mapped to different loci in chromosome 1BS using simple sequence repeat markers. The gene from PI 331260 was flanked by *Xgwm413* and *Xgdm33* in bin 1BS9-0.84-1.06 at genetic distances of 3.5 and 2.0 cM; and the gene from PI 480016 was flanked by *Xgwm18* and *Xgwm11* in chromosome bin C-1BS10-0.50 at 1.2 and 2.1 cM, respectively. Chromosomal locations and race and allelism tests indicated that the two genes are different from previously reported stripe rust resistance genes, and therefore are named as *Yr64* from PI 331260 and *Yr65* from PI 480016. These genes and their flanking markers, and selected common wheat lines with the genes should be valuable for diversifying resistance genes used in breeding wheat cultivars with stripe rust resistance.

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P. Cheng · L. S. Xu · M. N. Wang · D. R. See · X. M. Chen (✉)
Department of Plant Pathology, Washington State University, Pullman, WA 99164-6430, USA
e-mail: xianming@wsu.edu

D. R. See · X. M. Chen
Wheat Genetics, Quality, Physiology and Disease Research Unit, Agricultural Research Service, US Department of Agriculture, Pullman, WA 99164-6430, USA

Introduction

Stripe rust, caused by *Puccinia striiformis* Westend. f. sp. *tritici* Erikss. (*Pst*), causes yield losses in wheat crops worldwide (Stubbs 1985; Chen 2005; Wellings 2011). In the USA, more than 90 % losses in yield have been observed (Sharma-Poudyal and Chen 2011; Chen unpublished data). Although use of fungicides can prevent multi-million dollar losses (Line 2002), the application of fungicides adds a significant extra cost to wheat production. Resistant cultivars are the best means of stripe rust management.

All-stage resistance and adult-plant resistance, especially high-temperature adult-plant (HTAP) resistance, are the main types of resistance used in the USA and many other countries to control stripe rust (Chen 2005, 2013). All-stage resistance, which can be detected at the seedling

stage, expresses throughout all growth stages and generally provides high levels of resistance (Chen 2005). However, all-stage resistance is often race-specific and does not last long. In contrast, HTAP resistance, which is expressed as plants get older and weather becomes warmer, is non-race specific and durable. However, HTAP resistance often provides partial resistance which may not be adequate to prevent yield losses (Line 2002; Chen 2005, 2007, 2013). Gene pyramiding and multiline cultivars have been used in the USA to provide relatively durable resistance (Chen 2007). Gene deployment requires a large number of genes conferring effective resistance. So far, more than 60 permanently designated *Yr* loci and numerous temporarily designated genes have been identified in wheat, but most of them have become ineffective or provide low levels of resistance (Chen 2013; McIntosh et al. 2013; Rosewarne et al. 2013). Molecular markers have been developed for some resistance genes and marker-assisted selection has been used to produce cultivars resistant to stripe rust (Cheng and Chen 2010; Chen 2013; Rosewarne et al. 2013). Resistance genes can be combined in wheat genotypes by rapid and targeted marker-assisted background selection (Randhawa et al. 2009). However, the number of effective resistance genes with diagnostic markers is limited. New genes and more efficient user-friendly markers are needed to diversify resistance genes used in breeding programs.

Tetraploid wheat with the A and B genomes is part of the primary gene pool of hexaploid common wheat. Resistance genes *Yr7*, *Yr15*, *Yr24*/*Yr26* and *Yr36* originated from tetraploid wheats (Macer 1966; McIntosh and Lagudah 2000; Ma et al. 2001; Uauy et al. 2005). Durum wheat ($2n = 4x = 28$, AABB genomes) is grown on approximately 17 million hectares worldwide (Abdalla et al. 1992). Mamluk (1992) reported that about 23 % of 216 durum cultivars in West Asia and North Africa were resistant to stripe rust. A wide range of seedling and adult-plant responses to stripe rust was observed in durum wheat cultivars from various countries (Ma et al. 1995, 1997b). Ma et al. (1997a) studied the inheritance of stripe rust resistance in five durum cultivars developed by CIMMYT (the International Maize and Wheat Improvement Centre) and found that field resistance in those cultivars was controlled by at least three genes with additive interactions (Ma et al. 1997a). It appears that durum may harbor many genes for stripe rust resistance, most of which remain unidentified. Crossing common wheat and durum has been a recurrent effort in wheat breeding for almost a century, and durum wheat is also used in crosses to introduce desirable traits including stripe rust resistance from diploid wheat or grass species into common wheat (Chhuneja et al. 2008). Despite some hurdles it is relatively easy to transfer genes from durum to common wheat by selection among the selfed progenies. An alternate way is to produce synthetic wheat by chromosome doubling of tetraploid wheat × *Aegilops tauschii* ($2n = 14$, DD) hybrids (Kunert et al. 2007).

In our search for stripe rust-resistant wheat germplasm accessions, we identified a number of durum genotypes with high levels of resistance to predominant US *Pst* races under controlled greenhouse conditions and multi-year field tests under conditions of natural infection (Chen, unpublished data). In a previous study, Xu et al. (2013) identified a single dominant gene, *Yr53*, from durum wheat PI 480148, mapped the gene in the long arm of chromosome 2B and transferred it into a hexaploid wheat background. In the present study, we identified two dominant genes from durum wheat, *Yr64* from PI 331260 and *Yr65* from PI 480016, mapped both of them to the short arm of chromosome 1B and transferred them into a hexaploid wheat background. We determined that the two genes are different from each other and also different from other genes reported in 1BS. We also tested the polymorphisms of molecular markers flanking the two genes in various spring and winter wheat genotypes to determine their usefulness in marker-assisted selection.

Materials and methods

Development of mapping populations

Durum lines PI 331260 and PI 480016, used as the male parents, were crossed with the spring wheat (*T. aestivum*, $2n = 42$) line 'Avocet S' (AvS) in 2005–2006 and F_1 plants were grown in a greenhouse in 2006. F_2 plants, grown in an experimental field near Pullman, Washington, in 2007 were evaluated for stripe rust resistance, and selected plants were harvested for F_3 seeds.

Due to sterility, the numbers of seeds obtained in the F_2 and F_3 generations were too small to be used in genetic studies. In 2008, 28 F_3 seeds from AvS/PI 331260 and 20 F_3 seeds from AvS/PI 480016 were germinated on moist filter paper in Petri dishes and somatic chromosome numbers were counted in root tip cells using the standard Feulgen staining procedure (Chen et al. 1995a; Xu et al. 2013). Of the F_3 seedlings of AvS/PI 331260, 16 had $2n = 42$, 5 had $2n = 28$ and 7 had 29–41 chromosomes; and of the F_3 seedlings of AvS/PI 480016, 3 had $2n = 42$, 12 had $2n = 28$ and 5 had 29–41 chromosomes. The F_3 plants with 42 chromosomes were inoculated with urediniospores of *Pst* race PST-100 at the 3- to 5-leaf stage and grown to obtain F_4 seeds. Twenty to 40 seedlings in each F_4 line were tested with PST-100 and at least one segregating line that showed a 3 resistant:1 susceptible ratio was identified for each cross. The F_4 plants of the segregating lines were grown to obtain F_5 lines. Because sterility remained

a problem, only 27 F_5 lines of AvS/PI 331260 and 15 F_5 lines of AvS/480016 were obtained with limited seeds in each line.

F_5 lines were planted in the field in 2010; lines homozygous resistant, homozygous susceptible and segregating following natural *Pst* infection were recorded, and single-plant derived F_6 seeds were harvested. For each cross, an F_6 population derived from a single highly fertile segregating F_5 plant was selected for use as a mapping population. F_6 and parental seeds were planted in 12 × 12 × 12 cm pots, three seeds per pot, filled with soil mixture, and the plants were grown in a greenhouse. At about the 4-leaf stage, two leaves were collected from each F_6 plant or parental line for DNA extraction, and the plants were tested with race PST-100. After infection type data were recorded 20 days post-inoculation, sporulating leaf tissues were removed and the populations were grown to obtain F_7 seeds from each F_6 plant. About 15 seeds for each F_7 and parental line were planted in the field in spring 2011, and infection type and severity (percentage of leaf area infected) data were recorded at flowering. Natural infection allowed us to obtain stripe rust data without artificial inoculation. One homozygous resistant F_7 line with desirable agronomic traits such as medium height, big heads, more tillers and strong stems was selected from each cross for use as reference germplasm carrying the resistance genes in a common spring wheat background.

Pathogen isolates

Eight *P. striiformis* f. sp. *tritici* races (Table 1, Chen et al. 2010; Wan and Chen 2012) were chosen to test seedlings of PI 331260, PI 480016 and AvS based on their unique virulence patterns to determine how broad the resistance in the two resistant accessions against different races. Based on the tests, races PST-100 and PST-127 (Chen et al. 2010; Wan and Chen 2012) were selected to inoculate seedlings of the F_4, F_5 and F_6 progenies, together with the parents, in the greenhouse tests. Urediniospores of each race were increased on susceptible wheat lines and tested on the 20 wheat genotypes used to differentiate *Pst* races in the US (Chen et al. 2010; Wan and Chen 2012) or the new set of 18 *Yr* single-gene line differentials (Wan and Chen 2014) to determine purity and correctness prior to being used to test the parents and progenies of the crosses.

Evaluation of stripe rust reactions

About 5 seeds of each parent and 25–36 seeds of the F_4 or about 160 seeds of the F_6 generation for each cross were planted, 3 seeds per pot for the progenies and 5 seeds per pot for the parental lines, in the greenhouse for seedling tests and for advancing generations. Seedlings at the two-leaf stage (about 10 days after planting) grown in a rust-free greenhouse (diurnal temperature cycle gradually changing from 10 °C at 2:00 am to 25 °C at 2:00 pm with a 16 h light/8 h dark cycle) were uniformly dusted with a mixture of urediniospores of a single *Pst* race and talc 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, b). Infection type (IT) data were recorded on a 0–9 scale (Line and Qayoum 1992) 18–21 days after inoculation. Infection types 0–3, 4–6 and 7–9 were considered resistant, intermediate and susceptible, respectively.

Table 1 Seedling infection types of PI 331260, PI 480016 and Avocet Susceptible (AvS) to races of *Puccinia striiformis* f. sp. *tritici* tested under controlled greenhouse conditions

PST race[a]	Virulence formula	Infection type		
		AvS	PI 331260	PI 480016
PST-21	2	9	0	1
PST-43	1, 3, 4, 5, 12, 14	9	1	1
PST-45	1, 3, 12, 13, 15	9	1	1
PST-70	1, 3, 11, 12, 16, 18	9	1	1
PST-78	1, 3, 11, 12, 16, 17, 18, 19, 20	9	1	1
PST-100	1, 3, 8, 9, 10, 11, 12,16, 17, 18, 19, 20	9	1	1
PST-127	1, 2, 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	1	1

[a] The virulence formulae are based on reactions on the following 20 wheat genotypes used to differentiate races of *Puccinia 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. 2010; Wan and Chen 2012)

Because the resistance genes from PI 331260 and PI 480016 were mapped to chromosome 1B, these accessions and selected F_7 lines were tested together with wheat lines 'AvSYr10NIL' (*Yr10*), 'AvSYr15NIL' (*Yr15*) and 'AvS-Yr24NIL' (*Yr24*) carrying the genes in chromosome 1B, with seven races selected based on their virulence/avirulence reactions to the known genes.

For testing and advancing generations in the field, about 20 F_5 and 150 F_7 lines of each cross with 30–50 seeds for each line were planted in a field near Pullman, WA, USA, in 2009 and 2011 for evaluating stripe rust reactions and selecting homozygous resistant or segregating lines. Infection type and severity data were recorded at the flowering stage.

DNA extraction

Each leaf sample consisting of two fresh leaf pieces of 2 cm (about 3 mg) was pre-dried in a vacuum drier for 2–3 days before extraction of genomic DNA from each F_6 plant and parents of both crosses using a modified CTAB protocol (Riede and Anderson 1996). To each micro-centrifuge tube with a leaf sample, 100 µl 0.7 mm Zirconia beads (cat. 11079107zx, BioSpec Products, Bartlesville, OK, USA) were added. Tubes were covered with lids and placed in a Mini Beadbeater (Biospec Products, Inc.). After beating for 2 min, 560 µl pre-warmed (65 °C) 2X CTAB extraction buffer [1.4 M NaCl, 100 mM Tris-HCl pH 8.0, 2 % CTAB (hexadecyltrimethylammonium bromide), 20 mM EDTA, 0.5 % $NaHSO_3$ and 1 % 2-mercaptoethanol], was added to each tube. The tube was inverted 4–6 times. After incubation at 65 °C for 30 min, 700 µl solution of 24:1 (v/v) chloroform/isoamyl alcohol was added, vortexed thoroughly and centrifuged at 5,000 rpm for 25 min. The upper phase 600 µl solution was transferred to a new 1.5-ml microcentrifuge tube. DNA was precipitated with 500 µl of cold 70 % ethanol (−20 °C) and rinsed with 1 ml of 70 % ethanol. The air-dried DNA was dissolved in 100 µl TE (10 mM Tris–HCl and 1 mM EDTA, pH 8.0) buffer with 20 µg/ml RNase and incubated for 1–2 h in a 37 °C water bath or oven, and then stored at −20 °C. DNA was quantified through electrophoresis and with a spectrophotometer (NanoDrop ND-1000, Wilmington, DE, USA). The DNA stock solution was diluted to 30 ng/µl with sterilized ddH_2O for use as the working solution for PCR.

Bulk segregant analysis and SSR genotyping

Aliquots of DNA from 10 homozygous resistant and 10 homozygous susceptible F_6 plants were combined into resistant and susceptible bulks, respectively, for each cross. A total of 576 simple sequence repeat (SSR) markers were used for screening polymorphisms associated with the resistance locus in each cross. PCR were performed using a GeneAmp® PCR System 9700 thermo-cycler. A 12-µl reaction mixture consisted of 100 ng of template DNA, 1.2 µl Mg-free 10X PCR buffer (Promega, Madison, WI, USA), 0.6 unit of *Taq* DNA polymerase (Promega), 1.2 µl of 25 mM $MgCl_2$, 0.96 µl dNTPs (2.5 mM) (Sigma Chemical Co., St. Louis, MO, USA), 0.06 µl 10 µM forward primer with an M13 tail (5′-CACGACATTG-TAAAACGAC), 0.30 µl 10 µM reverse primer and 0.24 µl 10 µM M13 labeled primer (Applied Biosystems, Foster City, Calif., USA). M13 primers used to amplify the resistant parent, resistant bulk, susceptible bulk and susceptible parent of each cross were labeled with four fluorescent dyes: FAM (blue), VIC (green), NED (yellow) and PET (red). After 5 min of denaturation at 94 °C, amplifications were programmed for 35 cycles, each consisting of 30 s at 94 °C, 30 s at 50–61 °C (depending upon the primer pair), and 72 °C for 30 s, and 72 °C for 10 min followed by a 4 °C-holding step. The mixture of PCR products of 3.0 µl FAM, 3.0 µl VIC, 4.0 µl NED and 6.0 µl PET were added with 9 µl ddH_2O to get a 25 µl dilution. A total volume of 13 µl containing 9.93 µl formamide, 0.07 µl 445-LIZ DNA ladder (Applied Biosystems) and 3 µl diluted PCR product was denatured at 95 °C for 5 min and held at 4 °C. The sizes of the PCR products were estimated using capillary electrophoresis on an ABI3730X Genotyper (Applied Biosystems). Alleles were called using GeneMapper v3.7 software. Primer pairs showing associations with rust reaction in the bulk segregant analysis were used to genotype the F_6 population of each cross and those linked to the resistance locus were used to construct linkage maps.

After the resistance genes in both PI 331260 and PI 480016 were determined in chromosome 1BS, 20 additional SSR markers specific to 1BS (Röder et al. 1998) were tested for polymorphism using genomic DNA from the parents and F_6 lines to confirm chromosomal locations of the resistance genes and to identify more linked markers. The primer sequence information of SSR markers for 1BS tested in this study was obtained from the GrainGenes 2.0 website (http://wheat.pw.usda.gov/cgi-bin/graingenes/browse.cgi?class=marker). DNA of Chinese Spring and ditelosomic lines 1BL and 1BS and five 1BS deletion lines (Sourdille et al. 2004) were used to further determine the chromosomal bins containing the two genes.

A total of 140 spring and winter wheat cultivars or breeding lines were used in evaluating polymorphisms of molecular markers flanking the resistance genes to determine their usefulness for marker-assisted selection.

Data analyses

Chi-squared tests were conducted to determine goodness of fit of the observed numbers of plants or lines to predicted

segregation ratios of progenies to establish the number of stripe rust resistance genes, mode of inheritance and genetic relationships of genes. Marker distance in centiMorgan (cM) was calculated according to the Kosambi mapping function (Kosambi 1944) using the MAPMAKER program (Lander et al. 1987). Linkage maps were constructed using MapDraw v 2.2 for each cross (Liu and Meng 2003). Chi-squared tests were also used to determine the goodness of fit to a single-locus model for each marker in the F_6 populations. To determine genetic distances between two genes using an F_2 population in allelism test, 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 of recombination values to map distances, the Kosambi's formula $d = 1/4 \ln[(1 + 2p)/(1 - 2p)]$ was used as previously described (Kosambi 1944).

Results

Phenotypic and genetic characterization of stripe rust resistance

The seedling IT data for PI 331260, PI 480016 and AvS tested with eight *Pst* races are given in Table 1. Both durum lines were resistant to all races, whereas AvS was susceptible.

The numbers of resistant (ITs 0, 1 or 2) and susceptible (ITs 7, 8 or 9) plants in F_6 from greenhouse tests with PST-127 and the F_7 lines in naturally infected field tests are shown in Table 2. The F_6 populations of both AvS/PI 331260 and PI 480016 segregated in 3 resistant:1 susceptible ratios, and segregation of the F_7 lines fitted 1:2:1 ratios of homozygous resistant, segregating and homozygous susceptible lines. For both crosses, all F_7 lines derived from F_6 plants scored susceptible in greenhouse seedling tests were homozygous susceptible at the adult-plant stage in the field and F_7 lines derived from resistant F_6 plants were either homozygous resistant or segregating. These data showed that each of the mapping populations had a dominant gene for effective all-stage resistance to stripe rust.

SSR markers

Of the first 576 SSR markers tested in the bulk segregant analysis, *Xbarc137* and *Xgwm18* were polymorphic between the bulks and parents of AvS/PI 331260. *Xbarc137* was a dominant marker producing a 266 bp peak in AvS. *Xgwm18* was co-dominant producing a 205 bp peak in PI 331260 and 207 bp peak in AvS. *Xbarc187* and *Xgwm498* produced peaks specific to both PI 480016 and the resistant bulk or both AvS and susceptible bulk. *Xbarc187* was co-dominant producing a 199 bp peak in PI 480016 and a 203 bp peak in AvS. *Xgwm498*, also co-dominant, produced a 173 bp peak in PI 480016 and a 175 bp peak in AvS. The four markers localized both resistance genes to chromosome 1BS.

Because *Yr15* and *Yr24/Yr26* were previously mapped to chromosome 1BS (Chague et al. 1999; Ma et al. 2001; Li et al. 2006), markers linked to these genes were assayed on the F_6 lines of both crosses. Linkage analysis using the markers with the F_6 populations of both crosses indicated that both genes were linked to *Yr15* and *Yr24/Yr26*, confirming the location of both genes on chromosome 1BS. To identify more markers for linkage maps and to find closer markers, 20 additional markers including those linked to *Yr15* and *Yr24/Yr26* were screened. Overall, a total of 40 markers specific to chromosome 1B were screened; 12 were associated with the resistance gene in AvS/PI 331260 and 15 with the resistance gene in the AvS/PI 480016 using bulk segregant analysis. Ten markers were used to test the entire F_6 populations of AvS/PI 331260 and 9 markers were used to test the AvS/PI 480016 population, of which 8 markers were used in testing both populations.

Construction of linkage maps

The relative locations of *YrPI331260* and *YrPI480016* can be compared from common markers on the chromosome 1BS linkage maps shown in Fig. 1a, b. The closest markers flanking *YrPI331260*, *Xgwm413* and *Xgdm33*, were 3.5 cM distal and 2.0 cM proximal to the gene, respectively. The

Table 2 F_6 plants and F_7 lines segregation for seedling resistance to races PST-127 of *Puccinia striiformis* f. sp. *tritici* in AvS/PI 331260 and AvS/PI 480016

Crosses	Generation	Observed number of F_6 plants or F_7 lines[a]			Expected ratio	*P*
		Resistance	Segregating	Susceptible		
AvS/PI 331260	F_6	112	–	34	3:1	0.63
	F_7	35	80	31	1:2:1	0.46
AvS/PI 480016	F_6	114	–	58	3:1	0.32
	F_7	44	72	40	1:2:1	0.75

[a] For genetic analysis, F_6 was treated as F_2 and F_7 was treated as F_3. See the text for development of the segregating populations

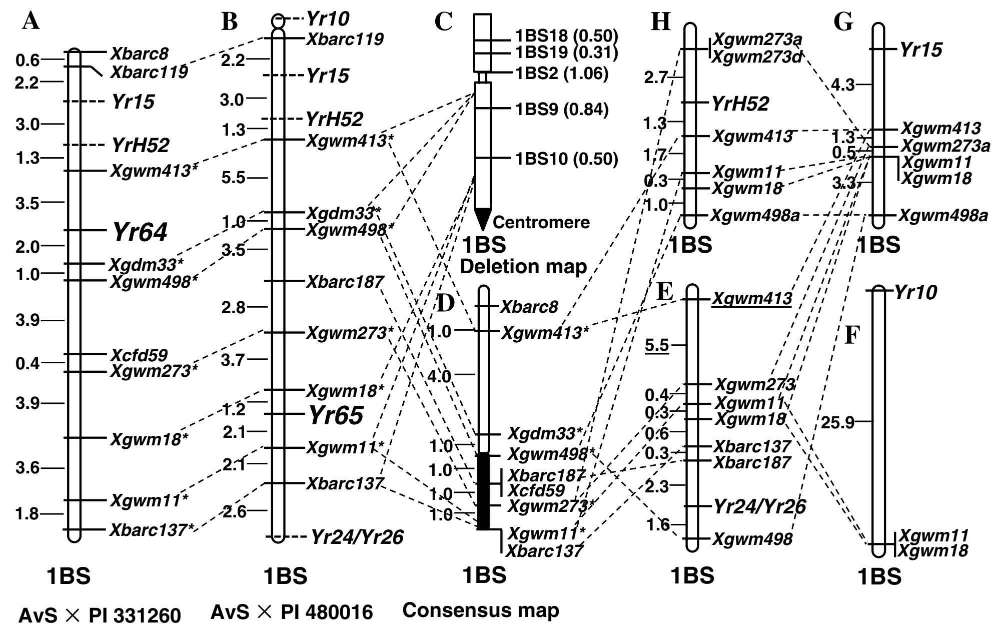

Fig. 1 Linkage maps for *Yr64* (*YrPI331260*) (**a**) and *Yr65* (*YrPI480016*) (**b**) on the short arm of chromosome 1B and comparisons with the consensus map of Somers et al. (2004) (**c**), the deletion map of Sourdille et al. (2004) (**d**), *Yr10* (Wang et al. 2002) (**e**), *Yr15* (Peng et al. 2000) (**f**), *Yr24*/*Yr26*/*YrCH42* (Li et al. 2006; Wang et al. 2008) (**g**), and *YrH52* (Peng et al. 2000) (**h**) based on common markers. The *dashed lines* connect common markers on different maps and a star *asterisk* indicates the markers that were tested on Chinese Spring and the ditelosomic and 1BS deletion lines

Table 3 Amplified SSR markers on Chinese Spring (CS), CS ditelosomic lines, CS deletion lines and their allele sizes (bp)

Chinese Spring lines	*Yr64* flanking markers	*Yr65* flanking markers	Additional markers	Chinese Spring lines	*Yr64* flanking markers	*Yr65* flanking markers
	Xgwm413	*Xgdm33*	*Xgwm18*	*Xgwm11*	*Xgwm498*	*Xgwm273*
Chinese Spring	109	148	203	212	175	182
CS Ditelo 1BL	–[a]	–	–	–	–	–
CS Ditelo 1BS	109	148	203	212	175	182
CS del 1BS-18	109	148	203	212	175	182
CS del 1BS-19	109	148	203	212	175	182
CS del 1BS-2	109	148	203	212	175	182
CS del 1BS-9	–	–	203	212	–	182
CS del 1BS-10	–	–	203	212	–	182

[a] The expected fragment was not amplified

closest markers flanking *YrPI480016* were *Xgwm18* (1.2 cM distal) and *Xgwm11* (2.1 cM proximal). Tests of the Chinese Spring 1BS deletion lines with six SSR markers, including the closest flanking markers for each of the two genes (Table 3), placed *YrPI331260* in bin 1BS9-0.84-1.06 and *YrPI480016* in bin C-1BS10-0.50 (Fig. 1c). The markers in these two linkage maps had similar orders to common markers in the consensus map (Somers et al. 2004) (Fig. 1d). For comparison, *Yr26* (*Yr24*) was reported to be either 4.1 cM (Ma et al. 2001) or 9.1 cM (Wang et al. 2008) from *Xgwm413*; either 1.9 cM (Ma et al. 2001) or 3.2 cM (Li et al. 2006; Wang et al. 2008) from *Xwgm18*/*Xwgm11*; and either closer to the centromere region or in 1BL (Zhang et al. 2013) (Fig. 1e). Based on the positions of *Xgwm11* and *Xgwm18*, *Yr24*/*Yr26*/*YrCH42* is 3.1 cM proximal to *YrPI480016*. *Yr10* was reported to be near the end of 1BS and 25.9 cM distal to *Xwgm18*/*Xwgm11* (Wang et al. 2002) (Fig. 1f). The position of *Xgwm413*, reported to be tightly linked (Murphy et al. 2009) or 4.3 cM proximal (Peng et al. 2000) to *Yr15* (Fig. 1g), indicates that *Yr15* is about 3.5 cM distal to *YrPI331260* and 6.5 cM distal to *YrPI480016*. Similarly, the position of *Xgwm413* about 1.3 cM proximal to *YrH52* (Fig. 1h) placed the *YrH52* locus 4.8 cM distal of *Yr64* and 22.6 cM distal of *Yr65*. These data indicated that *YrPI331260*, *YrPI480016*, *Yr24*/*Yr26* and *Yr15* should be at different loci and the gene order and distances should be *Yr10*–19.8 cM–*Yr15*-3.0 cM–*YrH52*–4.8 cM–*YrPI331260*–16.4 cM–*YrPI480016*–3.1 cM–*Yr24*/*Yr26*/*YrCH42* (Fig. 1). *YrPI331260* was therefore named *Yr64* and *YrPI480016* was named *Yr65*.

Table 4 Infection types on wheat genotypes with *Yr* genes on the short arm of chromosome 1B, or possibly close to the centromore on 1BL, produced by races of *Puccinia striiformis* f. sp. *tritici* (*Pst*)

Wheat line (*Yr* gene)	Infection types produced by *Pst* race[a]						
	PSTv-16	PSTv-23	PSTv-26	PSTv-27	PSTv-38	PSTv-40	PSTv-41
AvSYr10NIL (*Yr10*)	8	8	8	8	2	8	8
AvSYr15NIL (*Yr15*)	0	1	1	1	1	1	0
AvSYr24NIL (*Yr24*)	8	8	8	8	7	8	8
PI 331260 (*Yr64*)	0	0	1	1	1	1	1
PI 660064 (*Yr64*)	2	2	2	2	2	2	2
PI 480016 (*Yr65*)	1	1	0	1	1	1	1
AvS/PI 480016 F_7-12 (*Yr65*)	2	2	2	2	2	2	2
Avocet S (AvS)	8	8	8	8	8	8	8

[a] PSTv races were differentiated using a set of wheat *Yr* single-gene lines (Wan and Chen 2014)

Table 5 Numbers of tested and susceptible F_2 plants and estimated genetic distance in crosses for allelism tests of *Yr15*, *Yr24*, *Yr64* and *Yr65* mapped on chromosome 1BS or close to the centromere

Yr gene involved	Cross	No. of F_2 plants		Distance (cM)[a]
		Total	Susceptible	
Yr24/*Yr65*	AvSYr24NIL × AvS/PI 480016 F_7-12	299	1	7.1 ± 12.2
Yr65/*Yr15*	AvS/PI 480016 F_7-12 × AvSYr15NIL	300	2	14.1 ± 12.2
Yr15/*Yr64*	AvSYr15NIL × PI 660064	288	3	21.7 ± 0.9
Yr64/*Yr65*	PI 660064 × AvS/PI 480016 F_7-12	298	4	24.9 ± 6.2
Yr24/*Yr64*	AvSYr24NIL × PI 660064	280	6	33.8 ± 3.0

[a] Map distance was estimated using Kosambi's function $d = 0.25 \ln [(1 + 2p)/(1 - 2p)]$, where p is recombinant value and calculated using $f = p^2/4$ where f is the frequency of susceptible plants

Comparison of *Yr64* and *Yr65* with other *Yr* genes on 1BS using *Pst* races

To compare *Yr64* and *Yr65* with other *Yr* genes on chromosome 1BS, PI 331260, PI 660064 (a selected homozygous resistant F_7 line of AvS/PI 331260), PI 480016, AvS/PI 480016 F_7-12 (a selected homozygous resistant F_7 line of AvS/PI 480016), AvSYr10NIL (*Yr10*), AvSYr15NIL (*Yr15*) and AvSYr24NIL (*Yr24*) were tested with seven *Pst* races with virulence to *Yr24*/*Yr26* (Wan and Chen 2014). The lines with *Yr64*, *Yr65* and *Yr15* were resistant, whereas the line with *Yr24* was susceptible to all seven races and the line with *Yr10* was susceptible to all tested races except PSTv-38 (Table 4).

Because *Yr15*, *Yr24/26*, *Yr64* and *Yr65* were mapped or reported on chromosome 1BS, we made pair-wise crosses among PI 660064 (*Yr64*), AvS/PI 480016 F_7-12 (*Yr65*), AvSYr15NIL (*Yr15*) and AvSYr24NIL (*Yr24*) for allelism tests. The *Yr10* near-isogenic line was not included among the crosses because it conferred a reaction pattern distinct from those of *Yr15*, *Yr24*/*Yr26*, *Yr64* and *Yr65* to *Pst* isolates collected in North America and other countries (Sharma-Poudyal et al. 2013; Chen and associates, unpublished). In addition, *Yr10* was known to be located near the *Rg1* and *Gli-B1* loci in the end of chromosome 1BS [Metzger and Silbaugh 1970; Bariana et al. 2002; Wang et al. 2002]. A population of 280–300 F_2 plants from each cross was tested with race PST-127 at the seedling stage. Clear segregation in each cross (Table 5) indicated that the four genes were not allelic and deviations from 15:1 segregation ratios confirmed repulsion linkage in each case. The estimated genetic distances among the four genes were: *Yr24*–(7.1 ± 12.2 cM)–*Yr65*–(14.1 ± 12.2 cM)–*Yr15*–(21.7 ± 0.9 cM)–*Yr64*. The 24.9 ± 6.2 cM genetic distance between *Yr64* and *Yr65* and the 33.8 ± 3.0 cM distance between *Yr24* and *Yr64* generally supported the linkage order. Although these linkage relationships supported the gene order, the genetic distances are much higher than shown in Fig. 1.

Polymorphism of flanking markers for *Yr64* and *Yr65*

Five markers, *Xgwm413*, *Xgwm498* and *Xgdm33* closely linked to *Yr64* and *Xgwm11* and *Xgwm18* flanking *Yr65*, were tested with the 70 spring and 70 winter wheat cultivars and breeding lines developed in the US Pacific Northwest to determine their polymorphisms. Because two fragments were found in 61 (43.57 %) of the wheat lines when tested with *Xgdm33*, the lack of the specificity of this marker makes it unsuitable for marker-assisted selection due to the fact that its primers amplify homoeologous sequences in 1A, 1B and 1D. The specificity of *Xgwm18*

was increased when we changed the annealing temperature from 50 to 55 °C. The fragment sizes of *Xgwm413*, *Xgwm498*, *Xgwm11* and *Xgwm18* are provided in Supplement Table 1. Of the 140 wheat lines, 133 (95.00 %) had *Xgwm413* alleles and 138 (98.57 %) had *Xgwm498* alleles different from the *Yr64* donor; and 136 (97.14 %) had *Xgwm11* alleles and 134 (95.71 %) had *Xgwm18* alleles different from the *Yr65* donor. None of the wheat cultivars and breeding lines had the same-size fragments of both *Xgwm413* and *Xgwm498*, indicating that the combination of the two markers can 100 % differentiate the *Yr64* haplotype from other allelic haplotypes. Only three wheat lines had both *Xgwm11* and *Xgwm18* marker fragments at the same size as those of the *Yr65* donor, indicating that when used in combination, the two markers can distinguish the *Yr65* haplotype from other allelic haplotype at a probability of 97.86 % correctness.

Discussion

Durum lines PI 331260 and PI 480016 were collected from Shewa, Ethiopia, in 1967 and 1973, and deposited in the USDA Small Grains Collection in 1968 and 1983, respectively (http://www.ars-grin.gov/cgi-bin/npgs/acc/display.pl?1374952; http://www.ars-grin.gov/cgi-bin/npgs/acc/display.pl?1374952). These accessions were first evaluated by our program in 2005 and have shown high resistance levels in all greenhouse and field tests since then. Resistance in both lines was characterized as broad-spectrum all-stage resistance to all tested races, including those virulent to *Yr10* and *Yr24/Yr26* (Table 4). In the present study, genetic analysis showed that resistance in hexaploid populations developed from crosses of AvS with PI 331260 and PI 480016 was controlled by single dominant genes. However, it is important to note that the original durum accessions may have additional resistance genes to those reported here.

Both *Yr64* and *Yr65* were mapped on chromosome 1BS but in different bins. Associations with SSR markers and/or tests of allelism showed that the genes were different from previously named *Yr* genes on 1BS, viz. *Yr10*, *Yr15* and *Yr24/Yr26* (Chague et al. 1999; Peng et al. 2000; Ma et al. 2001; Li et al. 2006; Wang et al. 2008; Murphy et al. 2009). Genes *Yr64*, *Yr65* and *Yr15* confer resistance to all *Pst* races identified so far in the USA. However, *Yr10* and *Yr24*, which are now believed to be the same gene as *Yr26* (Li et al. 2006; McIntosh et al. 2013), are ineffective against several races (Table 4). *Yr10*, originally from Turkish hexaploid wheat accession PI 178383, has been incorporated into many wheat cultivars grown in the US Pacific Northwest (Metzger and Silbaugh 1970; Chen et al. 1995b). The only other documented source of *Yr10* is a *T. vavilovvii* accession (Bariana et al. 2002). The donor of *Yr15* is an Israeli wild emmer wheat accession (*T. dicoccoides*) (Peng et al. 2000). *Yr24* was derived from *T. turgidum* subsp. *durum* accession K733 (McIntosh and Lagudah 2000) and *Yr26* was assumed to be from durum line γ80-1, a γ-radiated mutant (Ma et al. 2001), seed of which is no longer available. PI 331260 and PI 480016 are both Ethiopian durum landraces. Based on the origins and seedling reactions to the *Pst* races tested, it can be concluded that both *Yr64* and *Yr65* are different from *Yr10*, *Yr15* and *Yr24/Yr26*.

The SSR markers common to this study and previous studies can be used to compare relative distances of *Yr64* and *Yr65* to *Yr10*, *Yr15* and *Yr24/Yr26*. *Yr64* and *Yr65* were estimated to be 14.8–15.2 cM apart based on markers shared by the two linkage maps, and were localized to different bins of chromosome 1BS (Fig. 1a–c). Wang et al. (2002) mapped *Yr10* from PI 178383 at the terminal region of chromosome 1BS, 25.9 cM from *Xgwm11* and *Xgwm18*. McIntosh et al. (1996) localized *Yr15* in chromosome 1BS about 7 cM from the centromere and 34 cM proximal to *Yr10*. Peng et al. (2000) mapped *Yr15* 4.3 cM from *Xgwm413*, and close linkages of *Yr15* to *Xgwm413* and *Xgwm273* were determined using the AvS + Yr15 near-isogenic line (Murphy et al. 2009). McIntosh and Lagudah (2000) located *Yr24* in chromosome 1B by monosomic analysis and postulated its position in 1BS by its linkage (4 cM) with *Yr15*. Zakari et al. (2003) proposed the gene order *Yr15-Yr24-Xgwm11* based on selection of *Yr15* in combination with the *Xgwm11* allele associated with *Yr24*. Li et al. (2006) reported that *Yr24* (*YrCH42*) is 1.6 cM to *Xgwm498* and 2.3 cM to *Xbarc187*, 2.6 cM to *Xbarc137*, 3.2 cM to *Xgwm18*, 3.5 cM to *Xgwm11* and 3.9 cM to *Xgwm273*. *Yr26* was first reported in chromosome 6AS by Yildirim et al. (2000) who erroneously assumed that it was associated with *Pm21*. There is now ample evidence to conclude that *Yr24* and *Yr26* are the same gene (Chen and associates, unpublished data; Li et al. 2006; Ma et al. 2001; McIntosh et al. 2013). Recent work suggests that the gene is in deletion bin C-1BL-6-0.32 or possibly within the centromere (Wang et al. 2008; Zhang et al. 2013). As shown in Fig. 1, the genetic distances of the common markers are generally similar to those in the present study except for switches in marker order. Our marker order is most consistent with the consensus map (Somers et al. 2004; Sourdille et al. 2004).

Based on the common markers discussed above, the consensus order of these genes is: *Yr10*–(19.8 cM)–*Yr15*–(7.8 cM)–*Yr64*–(16.4 cM)–*Yr65*-(3.1 cM)-centromere/*Yr24*/*Yr26*, which is generally in agreement with the order in the genetic linkage *Yr64*–(21.7 ± 0.9 cM)–*Yr15*–(14.1 ± 12.2 cM)–*Yr65*–(7.1 ± 12.2 cM)–*Yr24*/*Yr26* based on the allelism tests. These differences in genetic distance require further investigation especially given that the region carries several important genes. For example, the

distance between *Yr15* and *Yr24* was estimated at 4 cM, but the marker bin mapping data indicates that it is physically well separated from *Yr24* and the centromere (Chague et al. 1999; McIntosh and Lagudah 2000; Li et al. 2006; Wang et al. 2008; Zhang et al. 2013). Different marker positions or orders are also found among the consensus (Somers et al. 2004) the deletion maps (Sourdille et al. 2004) and the maps for *Yr15* (Peng et al. 2000) and *Yr24*/*Yr26* (Li et al. 2006). Differences in genetic distance were also found for some of the genes and markers; for example, in the map of Li et al. (2006) *Yr24*/*Yr26* was 3.2 or 3.5 cM from *Xgwm18* or *Xgwm11*, but the distance was 9.9 cM in Wang et al. (2008). Different populations, especially tetraploid (both *T. dicoccoides* and *T. durum*) compared to hexaploid populations, may also involve different gene orders and distances due to evolutionary changes. More detailed genetic studies with these materials will be necessary. Tests of allelism clearly showed that *Yr64* and *Yr65* are different genes, and also different from other genes reported on chromosome 1BS or near the centromere on 1BL. The consensus order based on the common markers as discussed above and presented in Fig. 1 can be used as a starting point for more detailed studies and eventual cloning of the genes. For this, the recently published genomic sequences and markers for chromosome 1BS (Raats et al. 2013) will be extremely valuable.

In addition to *Yr15* and *Yr24*/*Yr26*, temporarily named genes *YrCH42* and *YrH52* were also reported on chromosome 1BS (Peng et al. 2000; Li et al. 2006). *YrCH42* in Chinese wheat cultivar Chuanmai *42*, a derivative of a synthetic wheat (*T. durum* × *Aegilops tauschii* amphilpoid) was allelic (Li et al. 2006) and considered identical to *Yr24*/*Yr26* based on specificity with the pathogen (McIntosh et al. 2013; Zhang et al. 2013). *YrH52*, derived from *T. dicoccoides* accession Hermon 52 was linked to *Xgwm413* with a map distance of 1.3 (proximal) (Peng et al. 2000). *Yr64* was estimated to be 3.5 cM proximal to *Xgwm413*, whereas *Yr65* was 21.3 cM proximal to *Xgwm413* (Fig. 1a, b). Considering the origins and distances of the molecular markers, both *Yr64* and *Yr65* appear to be different from *YrH52*.

Although both *Yr64* and *Yr65* are effective against all races identified in the USA, further work is needed to determine the effectiveness of these genes in other countries. Since these genes have been transferred to a common wheat background, PI 660064 with *Yr64* (Wang et al. 2012) and AvS/PI 480016 F_7-12 with *Yr65*, they can be more easily used in breeding programs for developing common wheat cultivars. However, it is always better to use these genes in combination with other all-stage resistance genes such as *Yr5*, *Yr15*, *Yr45* and *Yr53*, all of which are effective against all tested US *Pst* races (Yan et al. 2003; Murphy et al. 2009; Li et al. 2011; Xu et al. 2013). It is even more desirable to combine the effective all-stage resistance genes with genes for non-race-specific HTAP resistance genes such as *Yr18*, *Yr29*, *Yr36*, *Yr39*, *Yr52*, *Yr59*, *Yr62* and many other QTL as molecular markers are available for these genes or QTL (Uauy et al. 2005; Lin and Chen 2007; Ren et al. 2012; Chen 2013; Rosewarne et al. 2013; Lu et al. 2014; Zhou et al. 2014). Alternatively, the effective genes can be utilized in different wheat crops or different regions using a gene deployment strategy. Nevertheless, the genes identified in the present study should be useful in diversifying stripe rust resistance genes used in durum and common wheat breeding programs. The high polymorphism rates of the flanking markers for *Yr64* and *Yr65* in 140 spring and winter common wheat genotypes indicate that the markers can be used in marker-assisted selection when use the stripe rust resistance gene donors in breeding programs.

Author contributions PC developed the mapping population, conducted the experiments of phenotyping and genotyping the mapping population, mapping the gene to chromosomes, analyzing the data, and draft and revise the manuscript. LSX selected progeny plants with 42 chromosomes and stripe rust resistance, MNW made the initial crosses, advanced the generations, tested early generation progenies for stripe rust response, and revised the manuscript. DRS provided equipment and guidance for SSR marker analysis and revised the manuscript. XMC developed the project, selected the resistant parent, initiated the crosses, guided through the entire study, and write the manuscript.

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Conflict of interest None.

Ethical standards All experiments and data analyses were conducted in Pullman, Washington, the USA. All authors have contributed to the study and approved the version for submission. The manuscript has not been submitted to any other journal.

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