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ORIGINAL ARTICLE

Identification of a major dominant gene for race-nonspecific tan spot resistance in wild emmer wheat

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Abstract

Key message **A single dominant gene found in tetraploid and hexaploid wheat controls broad-spectrum race-non-specific resistance to the foliar disease tan spot caused by *Pyrenophora tritici-repentis*.**

Abstract Tan spot is an important foliar disease of durum and common wheat caused by the necrotrophic fungal pathogen *Pyrenophora tritici-repentis*. Genetic studies in common wheat have shown that pathogen-produced necrotrophic effectors interact with host genes in an inverse gene-for-gene manner to cause disease, but quantitative trait loci (QTLs) with broad race-nonspecific resistance also exist. Less work has been done to understand the genetics of tan spot interactions in durum wheat. Here, we evaluated a set of Langdon durum—wild emmer (*Triticum turgidum* ssp. *dicoccoides*) disomic chromosome substitution lines for reaction to four *P. tritici-repentis* isolates representing races 1, 2, 3, and 5 to identify wild emmer chromosomes potentially containing tan spot resistance genes. Chromosome 3B from the wild emmer accession IsraelA rendered the tan spot-susceptible durum cultivar Langdon resistant to all four fungal isolates. Genetic analysis indicated that a single dominant gene, designated *Tsr7*, governed resistance. Detailed mapping experiments showed that the *Tsr7* locus is likely the same as the race-nonspecific QTL previously identified in the hexaploid wheat cultivars BR34 and Penawawa. Four user-friendly SNP-based semi-thermal asymmetric reverse PCR (STARP) markers cosegregated with *Tsr7* and should be useful for marker-assisted selection of resistance. In addition to 3B, other wild emmer chromosomes contributed moderate levels of tan spot resistance, and, as has been shown previously for tetraploid wheat, the *Tsn1*-Ptr ToxA interaction was not associated with susceptibility. This is the first report of a major dominant gene governing resistance to tan spot in tetraploid wheat.

Introduction

Tan spot, also known as yellow leaf spot, is a foliar disease of common wheat (*Triticum aestivum* L., $2n=6x=42$, AABBDD genomes), durum wheat (*T. turgidum* ssp. *durum* (Desf.) Husnot., $2n=4x=28$, AABB genomes) and their relatives. Tan spot is caused by the necrotrophic pathogen *Pyrenophora tritici-repentis* (Died.) Drechs. and occurs in most wheat-growing regions of the world. The practice of no-till farming adopted in the 1970s to reduce soil erosion led to an increased incidence of tan spot because *P. tritici-repentis* overwinters in stubble residue. The primary symptoms of tan spot include large necrotic lesions surrounded by chlorotic borders and sometimes extensive chlorosis extending between necrotic lesions. Large areas of necrosis and/or chlorosis coalesce in highly susceptible genotypes leaving large areas of dead leaf tissue that result in a reduced capacity to perform photosynthetic functions, which ultimately leads to reductions in yield.

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P. tritici-repentis is a member of the Pleosporales order of fungi and is known to produce necrotrophic effectors (NEs) (formerly referred to as host-selective toxins). At least three NEs are produced by *P. tritici-repentis*, including Ptr ToxA, Ptr ToxB, and Ptr ToxC (see Singh et al. 2010; Faris et al. 2013 for reviews). *P. tritici-repentis* race 1 produces Ptr ToxA and Ptr ToxC, race 2 produces Ptr ToxA, race 3 produces Ptr ToxC, race 4 is considered avirulent on wheat, and race 5 produces Ptr ToxB. Race 6 produces Ptr ToxB and Ptr ToxC, race 7 produces Ptr ToxA and Ptr ToxB, and race 8 produces all three NEs. Recognition of each of these NEs by single corresponding sensitivity genes in the host, namely *Tsn1*, *Tsc2*, and *Tsc1*, respectively, leads to a compatible interaction defined by the development of tan spot. Elimination or disruption of the dominant host allele negates recognition and leads to an incompatible (resistance) interaction. This system is referred to as inverse gene-for-gene where the pathogen essentially hijacks the plant's own defense mechanism to cause disease (Faris et al. 2010).

In addition to the inverse gene-for-gene interactions, numerous tan spot resistance QTLs have been identified (see Faris et al. 2013 for review). Most notably, a resistance QTL on the long arm of chromosome 3B was identified in the Brazilian spring wheat cultivar BR34 (Faris and Friesen 2005), and more recently in the US spring wheat cultivar Penawawa (Kariyawasam et al. 2016). In both cases, the 3B QTL conferred good levels of resistance to all known races of *P. tritici-repentis*.

Compared to common wheat, the genetics of tan spot resistance in durum wheat has been less studied. The *Tsn1*-Ptr ToxA, *Tsc2*-Ptr ToxB, and *Tsc1*-Ptr ToxC interactions have all been shown to play important roles in the development of tan spot in common wheat (Faris et al. 2013; Kariyawasam et al. 2016; Liu et al. 2017). Virdi et al. (2016) recently showed that the *Tsc2*-Ptr ToxB interaction can also play a significant role in tan spot development in durum wheat. On the contrary, there is accumulating evidence indicating that a compatible *Tsn1*-Ptr ToxA interaction plays little to no role in tan spot development in durum wheat (Chu et al. 2010; Virdi et al. 2016). The effects of a compatible *Tsc1*-Ptr ToxC interaction in durum wheat have not been investigated.

A few studies on the genetics of tan spot resistance in durum wheat have revealed relevant loci other than the three NE sensitivity genes. For example, Singh et al. (2008a, b) used a tetraploid biparental population to show that two linked recessive genes on chromosome arm 3BL conferred resistance to different isolates of the fungus. Also, Chu et al. (2010) conducted a QTL study and reported regions associated with tan spot resistance on chromosomes 3A, 3B, 5A, and 7B. In addition to the *Tsc2* locus, Virdi et al. (2016) reported resistance QTLs on chromosomes 4B and 6B using a biparental durum population. However, beyond these studies, little work has been done to dissect the genetics of tan spot resistance in durum wheat.

In polyploid wheat, disomic chromosome substitution lines consisting of a pair of chromosomes from a particular 'donor' accession substituted for a pair of homologous chromosomes in the background of a 'recipient' accession, are powerful tools for the genetic dissection of complex traits and identification of chromosomes harboring useful and/or novel genes. Dr. Leonard Joppa (USDA-ARS, Fargo, ND, retired) developed a set of disomic chromosome substitution lines in durum wheat using the wild emmer wheat [*T. turgidum* ssp. *dicoccoides* (Körn. Ex Asch. & Graebner) Aarons ($2n=4x=28$, AABB genomes)] accession IsraelA (syn. FA-15-3) as donor and North Dakota durum wheat variety Langdon (LDN) as the recipient (Cantrell and Joppa 1991). These substitution lines have been widely distributed and used for the investigation of various agronomic traits (Joppa and Cantrell 1990; Cantrell and Joppa 1991; Joppa et al. 1991; Joppa 1993; Stack et al. 2002; Singh et al. 2007).

Joppa (1993) crossed each of the chromosome substitution lines with LDN and developed segregating recombinant inbred chromosome line (RICL) populations. These populations serve as unique and useful tools because they segregate for individual recombinant chromosomes in a homozygous background allowing genetic analysis, mapping, and dissection of traits in isolation. The RICL populations have been used to evaluate traits such as grain protein content (Joppa et al. 1997), yield (Gonzalez-Hernadez et al. 2004), rachis brittleness (Nalam et al. 2006), segregation distortion (Kumar et al. 2007a, b), Fusarium head blight (FHB) resistance/susceptibility (Otto et al. 2002; Garvin et al. 2009), Septoria nodorum blotch and tan spot resistances (Faris et al. 2000; Gonzalez-Hernandez et al. 2009; Faris and Friesen 2009), and spike morphology (Faris et al. 2014b).

Here, we evaluated the LDN-IsraelA chromosome substitution lines for reaction to tan spot caused by *P. tritici-repentis* isolates representing races 1-3 and 5. Wild emmer chromosome 3B was found to harbor dominant race-nonspecific resistance and was therefore the subject of molecular and comparative mapping to determine whether the 3B resistance locus was the same as loci previously reported in hexaploid wheat. User-friendly PCR-based SNP markers developed for the locus should be useful for introgression of the gene into adapted durum backgrounds.

Materials and methods

Plant materials

The LDN-*T. turgidum* ssp. *dicoccoides* chromosome substitution lines were evaluated for reaction to tan spot using isolates Pti2 (race 1), 86-124 (race 2), 331-9 (race 3) and

DW5 (race 5). Race 4 is considered an avirulent race and was not studied. All these races have been identified in North America (Friesen et al. 2005). For brevity, the chromosome substitution lines derived from *T. turgidum* ssp. *dicoccoides* accession IsraelA will be referred to as IsA-*X*, where '*X*' refers to the *T. turgidum* ssp. *dicoccoides* chromosome being substituted for the homologous LDN chromosome. The set consists of substitution lines representing all tetraploid wheat chromosomes except for the line IsA-2B, which was not available because the IsraelA chromosome 2B poorly compensates for LDN 2B and plants are weak and mostly sterile (J.D. Faris and S.S. Xu, unpublished). In addition to LDN, IsraelA, and the substitution lines, the tan spot differential lines Salamouni, Glenlea, 6B365, and 6B662 (Lamari et al. 1995) were included in all inoculations.

The LDN × IsA-3B RICL population (hereafter referred to as the LD3B population) consisting of 91 homozygous recombinant lines was evaluated for reaction to the same isolates used to test the chromosome substitution lines. Also, a new cross between LDN and IsA-3B was made, and seven F_1 plants from this cross were evaluated for reaction to tan spot using the race 2 isolate 86-124.

A panel of 21 wheat lines was used to evaluate STARP markers developed for SNP markers associated with the QTL on 3B (see below). The panel consisted of the tetraploid and hexaploid lines listed in Table 1.

Ptr ToxA infiltrations, fungal inoculations and statistical analysis

LDN, IsraelA, and the chromosome substitution lines were infiltrated with the necrotrophic effector Ptr ToxA as described in Liu et al. (2017). Reactions were scored 5 days after infiltration as either sensitive (presence of necrosis) or insensitive (no necrosis).

Plants were grown in super-cell containers (Stuewe & Sons Inc., Corvallis, OR) consisting of racks of 98 containers as described in Liu et al. (2017). The tan spot-susceptible North Dakota hard red winter wheat cultivar 'Jerry' was planted in the outside borders of each rack to reduce any edge effect. Inoculum preparation, plant inoculations, and disease evaluations were performed as described in Liu et al. (2017). The chromosome substitution sets, and later the LD3B population were grown in completely randomized

Table 1 Tetraploid and hexaploid wheat lines used to evaluate semi-thermal asymmetric reverse PCR (STARP) markers

Wheat line	Ploidy level	Taxonomical name	Contains 3B resistance QTL	*fcp735*[a]	*fcp736*	*fcp737*	*fcp738*
Langdon	4X	*Triticum turgidum* ssp. *durum*	No	190	162	149	83
IsA-3B	4X	*Triticum turgidum* ssp. *dicoccoides*	Yes	186	166	145	87
LD3B-81	4X	*Triticum turgidum* ssp. *dicoccoides/durum* recombinant	Yes	186	166	145	87
Altar84	4X	*Triticum turgidum* ssp. *durum*	No	190	162	149	83
Lebsock	4X	*Triticum turgidum* ssp. *durum*	No	190	162	149	83
Ben	4X	*Triticum turgidum* ssp. *durum*	No	190	162	149	83
PI 94749	4X	*Triticum turgidum* ssp. *carthlicum*	No	190	162	149	83
PI 41025[b]	**4X**	***Triticum turgidum* ssp. *dicoccum***	**No**	**186**	**166**	**145**	**87**
Louise	6X	*Triticum aestivum* ssp. *aestivum*	No	190	162	149	83
Penawawa	6X	*Triticum aestivum* ssp. *aestivum*	Yes	186	166	145	87
BR34	6X	*Triticum aestivum* ssp. *aestivum*	Yes	186	166	145	87
Grandin	6X	*Triticum aestivum* ssp. *aestivum*	No	190	162	149	83
Opata85	**6X**	***Triticum aestivum* ssp. *aestivum***	**No**	**186**	**166**	**145**	**87**
Salamouni	6X	*Triticum aestivum* ssp. *aestivum*	No	190	162	149	83
Katepwa	6X	*Triticum aestivum* ssp. *aestivum*	No	190	162	149	83
Arina	6X	*Triticum aestivum* ssp. *aestivum*	No	190	162	149	83
Forno	6X	*Triticum aestivum* ssp. *aestivum*	No	190	162	149	83
Chinese Spring	**6X**	***Triticum aestivum* ssp. *aestivum***	**No**	**186**	**166**	**145**	**87**
Bobwhite	6X	*Triticum aestivum* ssp. *aestivum*	No	190	162	149	83
TA4152-60	6X	*Triticum aestivum* ssp. *aestivum* (synthetic)	No	190	162	149	83
ND495	**6X**	***Triticum aestivum* ssp. *aestivum***	**No**	**186**	**166**	**145**	**87**

[a]The numbers shown for markers *fcp735-fcp738* indicate fragment sizes in base pairs

[b]Lines in bold do not harbor resistance conferred by *Tsr7*, but they contain the resistance marker genotype for all four STARP markers indicating that they have acquired historical recombination events between *Tsr7* and the STARP markers or had mutation events that led to deactivation of *Tsr7*

designs with three seeds per container serving as an experimental unit, and two containers for each line per experiment. Each experiment was conducted at least three times; thus, the reaction of each line to each isolate consisted of 18 plants representing six replications. Disease reactions were scored seven days after inoculation using the 1–5 scale described by Lamari and Bernier (1989) where 1 is highly resistant and 5 is highly susceptible.

Bartlett's Chi-squared test for homogeneity of error variances was conducted using PROC GLM in SAS program version 9.3 (SAS institute 2011) to determine whether inoculation experiments for each isolate were homogeneous among the substitution lines. Error variances were homogeneous among all experiments for each isolate (data not shown), and therefore the mean values were calculated and used for further analysis. Fisher's Least Significant Difference (LSD) test was used to determine significant differences among means of the chromosome substitution lines at the 0.05 level of probability.

Molecular mapping

We constructed a genetic linkage map of chromosome 3B in the LD3B population using simple sequence repeat (SSR) markers known to detect loci on chromosome 3B. The SSR markers were selected from the BARC (Song et al. 2005), GWM (Röder et al. 1998), WMC (Somers et al. 2004), CFA (Sourdille et al. 2003), and HBG (Torada et al. 2006) primer sets. Polymerase chain reactions (PCR) were performed as described in Röder et al. (1998), and PCR-amplified products were visualized as described in Zhang et al. (2009). Linkage analysis was conducted using the MapDisto v1.7 program (Lorieux 2012) as described in Faris et al. (2014a). The RICLs of the LD3B population segregated into discrete resistant and susceptible classes for reaction to all the isolates and were therefore classified as either resistant or susceptible. Because resistance segregated as a qualitative trait, it was mapped as a single locus relative to the molecular markers on chromosome 3B.

Race-nonspecific tan spot resistance on chromosome arm 3BL in common wheat was previously identified using biparental populations derived from BR34 × Grandin (BG population) (Faris and Friesen 2005) and from Louise × Penawawa (LouPen population) (Kariyawasam et al. 2016). Linkage maps for these populations were constructed based on SSRs and target region amplified polymorphisms (TRAPs) for the BG population and SSRs and the Illumina iSelect 9 k array for the LouPen population. Recently, the Illumina iSelect 90 k array was used to genotype both the BG and the LouPen populations. To make comparisons among the chromosome 3B linkage maps of these two populations and the LD3B population, we selected all 90 k SNP markers on chromosome 3B in common between the BG and LouPen populations, and SSR markers in both populations that were in common either with each other, or with the LD3B population, to reconstruct the 3B linkage maps in both populations using MapDisto v1.8 (Lorieux 2012). The phenotypic data, which included data from all *P. tritici-repentis* races for both populations, were re-analyzed against the new 3B linkage maps using the software program QGene v4.3 (Joehanes and Nelson 2008) to determine precisely the relative position of the 3B QTL in each population.

Development and screening of semi-thermal asymmetric reverse PCR (STARP) markers

Four SNP markers that were closely associated with the 3B QTL in the BG and LouPen populations were converted to semi-thermal asymmetric reverse PCR (STARP) markers (Long et al. 2017). The primers used for STARP marker development are presented in Table 2. PCR was carried out using a total reaction volume of 10 µl consisting of 100 ng genomic DNA, 0.9 × NH4 + buffer, 1.5 mM $MgCl_2$, 50 µM dNTPs, 0.8 M betaine, 0.04% (w/v) bovine serum albumin (BSA), 200 nM common reverse primer, 200 nM of each priming element-adjustable primer (PEA-primer 1 and PEA-primer 2), 40 nM of each asymmetrically modified allele-specific primer (AMAS forward primer 1 and AMAS forward primer 2), and 1.5 U of *Taq* DNA polymerase (without 3′ → 5′ exonuclease activity). PCR conditions were 94 °C for 5 min, followed by 6 cycles of 94 °C for 30 s and 56 °C for 2 min where the Ta/e decreased 1 °C each cycle. This touchdown cycle was followed by 40 cycles (45 cycles if using fluorescent PEA-primers) of 94 °C for 20 s, 62 °C for 30 s, 72 °C 1:30 min, followed by a final extension at 72 °C for 7 min. Amplicons were electrophoresed on 6% polyacrylamide gels, stained with GelRed™ nucleic acid stain (Biotium Inc., Fremont, CA), and visualized with a Typhoon™ FLA 9500 variable mode laser scanner (GE Healthcare Life Sciences, Marlborough, MA). For gel-free digital allele discrimination, PEA-primers 1 and 2 labeled with FAM and HEX fluorophores, respectively, were used in the STARP protocol. Fluorescence intensity of the amplified product was measured using a CFX384 Touch™ Real-Time PCR Detection System (Bio-Rad Laboratories, Inc., Hercules, CA) where STARP markers were used to genotype the three mapping populations (BG, LouPen, and LD3B) and the panel of 21 wheat lines mentioned above.

Table 2 Semi-thermal asymmetric reverse PCR (STARP) markers developed for molecular mapping on chromosome 3B

STARP marker name	Forward primer 1	Forward primer 2	Reverse primer	SNP source	Amplicon size (bp)
fcp735	**GCAACAGGAACCAGC TATGAC**-GGAAGC CCAAAATTGACCCA	GACGCAAGTGAGCAG TATGAC-GGAAGC CCAAAATTGATTCG	TGTACCTTTCCGGAA GCAATACG	IWB7782	186/190
fcp736	**GCAACAGGAACCAGC TATGAC**-TGGCTCAAC TGCTGTTAC	GACGCAAGTGAGCAG TATGAC-TGGCTCAAC TGCTGTCGT	GAGCTTCACATTGCT GTCACGT	IWB40103	162/166
fcp737	**GCAACAGGAACCAGC TATGAC**-GCATAATCG AATTAATACAAGACC	GACGCAAGTGAGCAG TATGAC-GCATAATCG AATTAATACAGAACT	GCTTCATCTCATGTG GCCTTTTTTA	IWB80906 (IWA7595)	145/149
fcp738	**GCAACAGGAACCAGC TATGAC**-GGAAAA ATATACACCAACATT CAAC	GACGCAAGTGAGCAG TATGAC-GGAAAA ATATACACCAACATT TCAT	TGGTCAATTCTTGTA CAGCTCGT	IWB80300 (IWA6794)	83/87
PEA-primers					
PEA-Primer 1	AGCTGGTT-SP9-**GCA ACAGGAACCAGC TATGAC**				
PEA-Primer 2	ACTGCTCAAGAG-SP9-GACGCAAGTGAGCAG TATGAC				

Underlined text indicates Tail 2, which provides the priming site for PEA-primer 2, containing the 4 bp insertion (red font) that allows for size discrimination on 6% polyacrylamide gels. Primers were developed using the Chinese Spring reference sequence, IWGSC RefSeq v1.0 wheat genome assembly

Bold text indicates Tail 1, which provides the priming site for priming element-adjustable (PEA)-primer 1

Results

Chromosome substitution line infiltrations with Ptr ToxA

Infiltrations with Ptr ToxA revealed that LDN was sensitive and IsraelA was insensitive as shown previously (Faris and Friesen 2009; Virdi et al. 2016). As expected, all of the substitution lines were sensitive to Ptr ToxA with the exception of IsA-5B (data not shown). In this case, the LDN 5B chromosome, which harbors *Tsn1*, was replaced by the non-*Tsn1*-containing 5B chromosomes of IsraelA, which rendered the plants insensitive to Ptr ToxA.

Inoculations of chromosome substitution lines

LDN was susceptible to tan spot caused by inoculations of all four isolates with average reaction types of 4.33, 4.38, 4.17, and 3.83 to Pti2 (race 1), 86-124 (race 2), 331-9 (race 3), and DW5 (race 5), respectively (Fig. 1, Fig. 2). In contrast, the wild emmer accession IsraelA was resistant to all four isolates with reaction types of 1.33, 1.63, 1.00, and 1.50, respectively. No chromosome substitution line was more susceptible than LDN (Fig. 2). IsraelA and the chromosome substitution line IsA-3B were significantly more resistant than LDN to all four *P. tritici-repentis* isolates

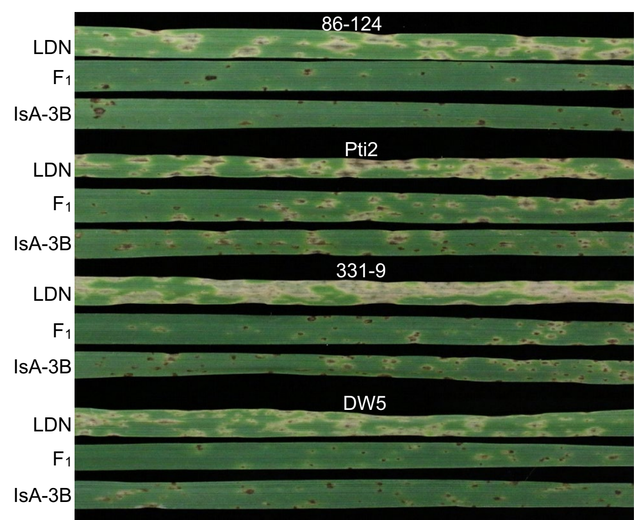

Fig. 1 Leaves of Langdon (LDN), the LDN-*Triticum turgidum* ssp. *dicoccoides* accession IsraelA chromosome 3B disomic substitution line (IsA-3B), and the F_1 derived from the cross between LDN and IsA-3B inoculated with *Pyrenophora tritici-repentis* isolates Pti2 (race 1), 86-124 (race 2), 331-9 (race 3), and DW5 (race 5)

(Fig. 2). Substitution lines IsA-3A and IsA-6A were each significantly less susceptible than LDN to three of the four isolates with IsA-3A being less susceptible to Pti2, 86-124, and DW5, and IsA-6A less susceptible to Pti-2, 86-124, and

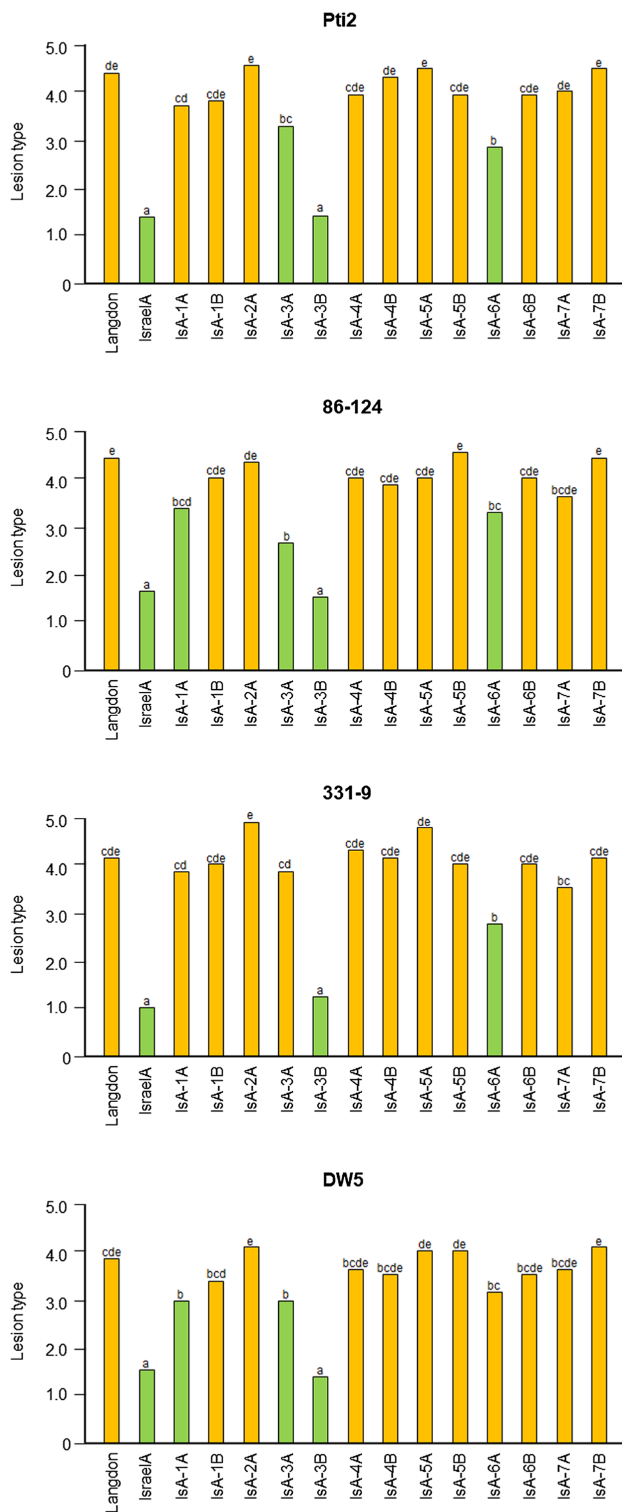

Fig. 2 Bar graphs of average lesion type reactions of Langdon, IsraelA, and the chromosome substitution lines for *Pyrenophora tritici-repentis* isolates Pti2 (race 1), 86-124 (race 2), 331-9 (race 3), and DW5 (race 5). For each isolate, bar columns labeled with different lowercase letters are significantly different at $P < 0.05$. Bars colored in green indicate substitution lines that were significantly different from Langdon

331-9. The substitution line IsA-1A was less susceptible than LDN to isolates 86-124 and DW5, but it did not differ for Pti2 and 331-9.

Genetic analysis and mapping of the tan spot resistance in IsA-3B

Evaluation of the LD3B population for reaction to tan spot caused by all four *P. tritici-repentis* isolates revealed that responses of the RICLs could be clearly classified as either a reaction type less than 2.0 or a reaction type greater than 3.0, and therefore each RICL was classified in a qualitative manner, i.e., as either resistant (R) or susceptible (S) (Supplementary Table 1). Each RICL had identical scores for all isolates, indicating the same gene was likely conditioning race-nonspecific resistance. However, the segregation of the gene did not fit the expected 1:1 ratio, and instead, the population segregated 31 resistant:60 susceptible ($\chi^2 = 9.24$, $P = 0.002$).

To determine whether the resistance conferred by IsA-3B was dominant or recessive, we evaluated seven F_1 plants derived from the LDN × IsA-3B cross along with eleven LDN plants and six IsA-3B plants with isolate 86-124. In this experiment, the F_1 plants had an average reaction type of 1.14, whereas LDN and IsA-3B had average reaction types of 3.33 and 1.16, respectively, indicating that the resistance derived from IsA-3B was dominant (Fig. 1).

A total of 44 SSR markers were mapped in the LD3B RICL population resulting in a linkage map 177.1 cM in length (Fig. 3). Twenty-eight of the markers in the proximal region of the chromosome had segregation ratios that deviated significantly ($P < 0.05$) from the expected 1:1 ratio. The race-nonspecific tan spot resistance gene, which we designated as *Tsr7*, mapped within this region of distorted markers and cosegregated with SSR markers *hbg218* and *wmc69*. Comparisons of common markers on the LD3B map and the deletion-based physical maps reported by Sourdille et al. (2004) indicated that *gwm376* was located in the most proximal deletion bin on the long arm of chromosome 3B. Because *Tsr7* mapped 1.2 cM distal to *gwm376* on the LD3B map, *Tsr7* is likely located in the proximal region of 3BL as well.

Re-mapping and analysis of tan spot resistance on chromosome 3B in the BG and LouPen populations

Genotyping of the BG and LouPen populations with the wheat Illumina 90 k iSelect array led to the identification of 934 and 340 SNP markers on chromosome 3B in each of the populations, respectively. Of these, a total of 148 SNP markers were common to both maps and were used to reconstruct the 3B linkage maps along with SSR markers

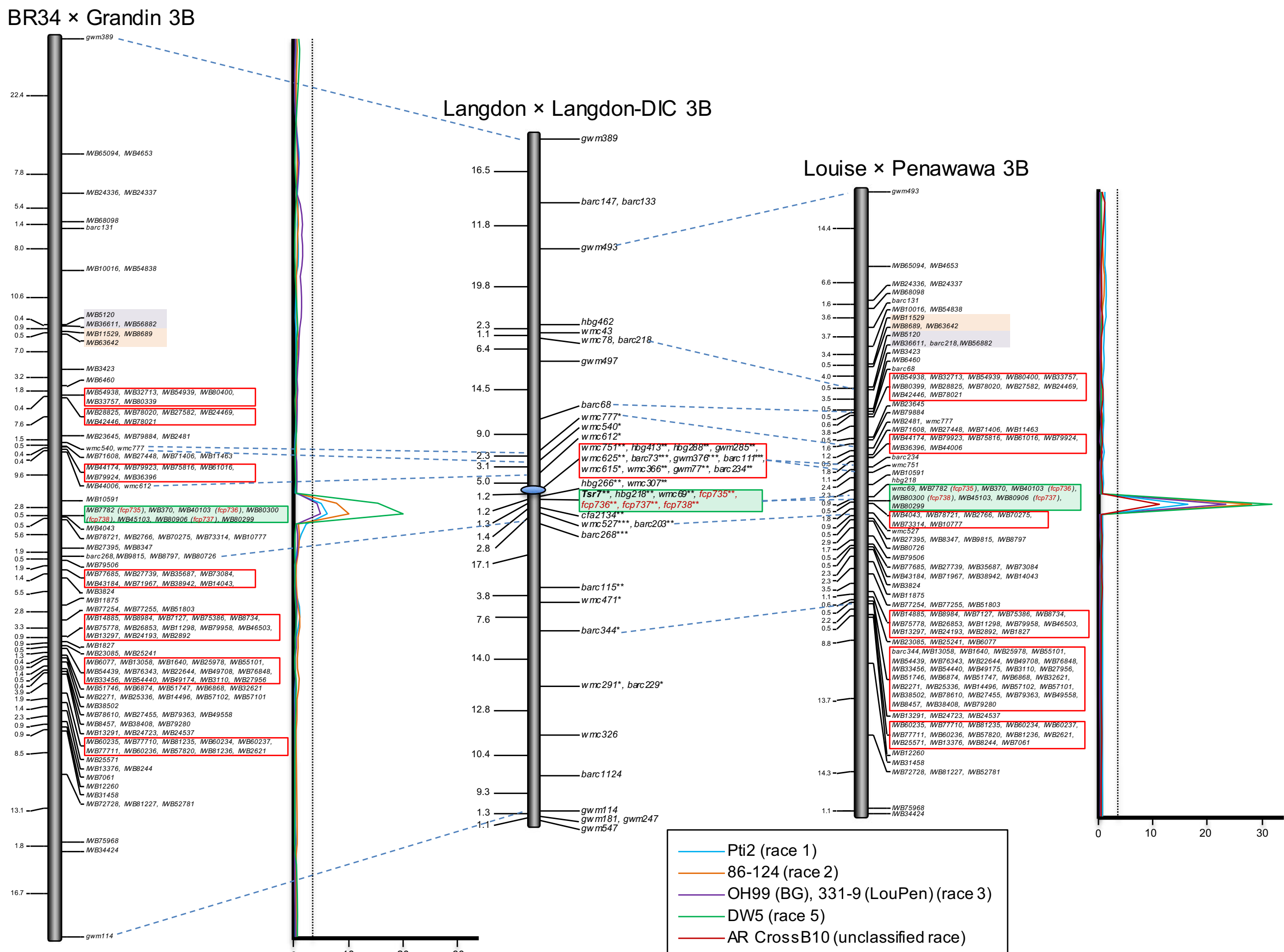

Fig. 3 Genetic linkage maps of chromosome 3B developed in the BR34 × Grandin (BG) population (left), the Langdon × Langdon—*Triticum turgidum* ssp. *dicoccoides* disomic chromosome substitution line (LD3B) population (middle), and the Louise × Penawawa (LouPen) population (right). Markers are shown on the right of the linkage maps, and genetic distances are on the left. Composite interval mapping regression curves for reaction to tan spot caused by *Pyrenophora tritici-repentis* are shown to the right of the BG and LouPen maps with the LOD scale on the *x* axis. The boxed legend describes the different *P. tritici-repentis* isolates used. The phenotypic data for tan spot in the BG and LouPen populations were published in Faris and Friesen (2005) and Kariyawasam et al. (2016), respectively. The blue dashed lines indicate common SSR markers across maps. The orange and purple shaded regions on the BG and LouPen maps represent markers involved in an inversion between the two maps. Markers outlined in a red line indicate markers occupying the same locus. The green shaded markers represent the locus that defines the peak of the tan spot race-nonspecific resistance QTL on the BG and LouPen maps, and the *Tsr7* locus on the LD3B map. The blue elliptical object indicates the centromere on the LD3B map

that were in common between the two maps and/or with the LD3B map. The resulting maps consisted of 154 and 158 markers for the BG and LouPen populations, respectively (Fig. 3). These markers accounted for 50 loci spanning 174.95 cM on BG chromosome 3B, and 44 loci spanning 120.15 cM on LouPen chromosome 3B. The markers were perfectly collinear between the BG and LouPen 3B maps with one exception; an apparent inversion involving markers *IWB5120*, *IWB36611*, *IWB56882*, *IWB11529*, *IWB8689*, *IWB63642*, and *barc218* occurred on the short arm (Fig. 3). Comparisons of all common SSR markers between the BG and LouPen chromosome 3B maps with the LD3B map indicated all were perfectly collinear.

Re-evaluation of the tan spot disease ratings in the BG and LouPen populations (Faris and Friesen 2005; Kariyawasam et al. 2016) revealed a locus at the seven cosegregating SNP markers (*IWB7782*, *IWB370*, *IWB40103*, *IWB80300*, *IWB45103*, *IWB80906*, and *IWB80299*) that defined the peak of the race-nonspecific tan spot resistance QTL (Fig. 3). In the BG population, the LOD values for tan spot reaction types ranged from 5.0 to 20.4 and explained from 17.5 to 55.0% of the variation in disease. LOD values ranged from 11.5 to 32.7 and the QTL explained from 24.4

to 55.1% of the variation in the LouPen population. SSR marker *wmc69*, which cosegregated with the *Tsr7* locus in the LD3B population, also cosegregated with the seven SNP markers that defined the QTL peak in the LouPen population. Therefore, the tan spot resistance locus in all three populations was determined to be at the same genetic position on chromosome arm 3BL, providing strong evidence that the same gene likely conferred the observed race-nonspecific resistance in each population.

Development of STARP markers for *Tsr7*

STARP markers were developed based on the 90 k iSelect SNP markers *IWB7782*, *IWB40103*, *IWB80300*, and *IWB80906*, and the STARP markers were designated as *fcp735*, *fcp736*, *fcp737*, and *fcp738*, respectively. The four markers were used to genotype the BG and LouPen populations, and the allelic segregation of the markers was compared to the segregation of the corresponding 90 k iSelect SNP marker. The STARP marker segregation was identical to the corresponding SNP marker in each case (Fig. 3), indicating that the STARP markers detected the targeted loci. The STARP markers were then used to genotype the LD3B population, and linkage analysis indicated that all four markers cosegregated with the *Tsr7* locus (Fig. 3).

The four STARP markers were then used to genotype a small panel of wheat lines for which the status of the 3B resistance QTL was known from past research (Faris et al. 1997, 2012; Faris and Friesen 2005; Chu et al. 2008, 2010; Tadesse et al. 2010; Kariyawasam et al. 2016; JD Faris and TL Friesen unpublished data). Of the 21 lines, IsA-3B, LD3B-81, Penawawa, and BR34 were known to carry the 3B resistance QTL, whereas the remaining 17 lines were known to lack the QTL. Genotyping of the 21 lines indicated that the 3B QTL-containing lines shared the same marker allele for all four STARP markers. Among the lines that lacked the 3B QTL, 13 carried an allele different from the one harbored by the 3B QTL-containing lines, and four had the same allele as the resistant lines (Table 1, Fig. 4).

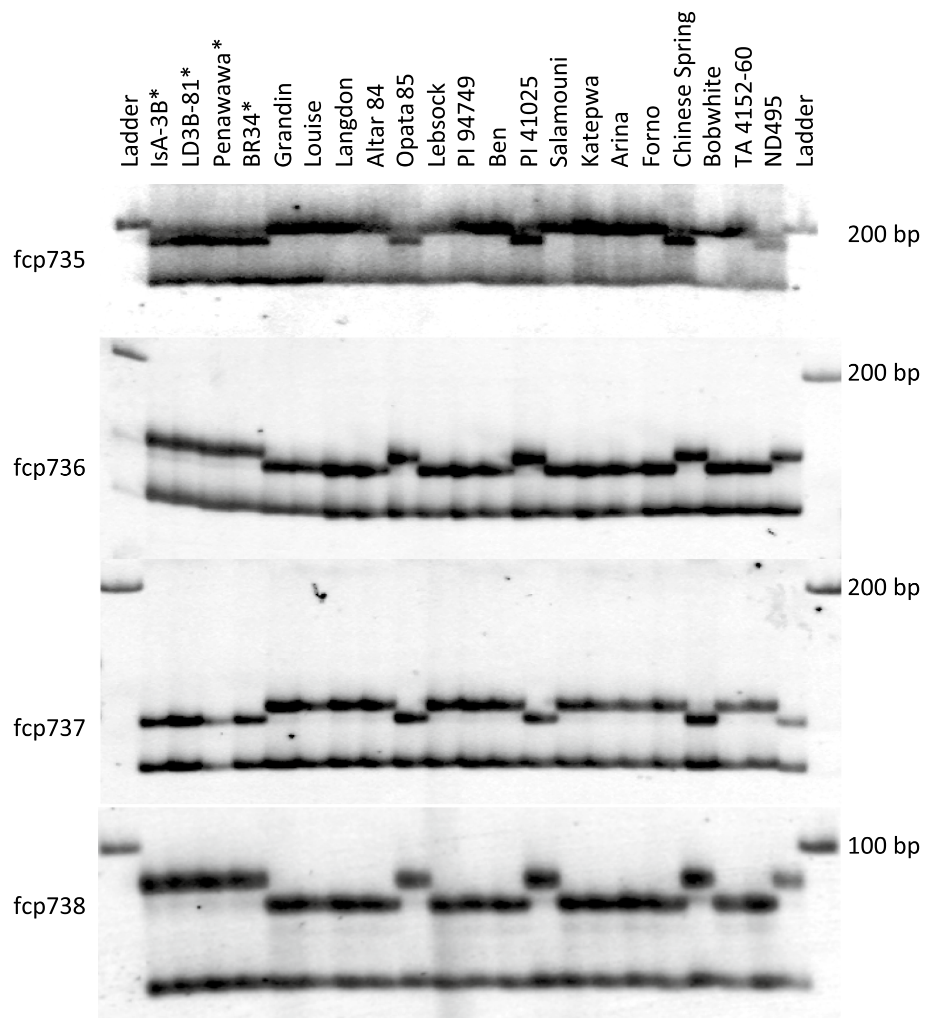

Fig. 4 Polyacrylamide gel electrophoresis assays of the four STARP markers *fcp735*, *fcp736*, *fcp737*, and *fcp738* evaluated on the 21 wheat lines described in Table 1. The asterisks denote lines that are known to carry *Tsr7*, and lines without an asterisk are known to not carry *Tsr7* based on previous genetic studies

This indicated that Opata 85, PI 41025, Chinese Spring, and ND495 have a recombination event(s) between the STARP marker loci and *Tsr7*, or the gene was deactivated by mutation without changing the marker haplotype.

To determine whether the STARP markers were suitable for genotyping using a digital assay, all four were used to genotype the BG population using a real-time detection system as described in Materials and Methods. Allele discrimination for all four was successful (Fig. 5) indicating that the four STARP markers are suitable for use in either gel or gel-free genotyping assays.

Discussion

Much less work has been done to understand the genetics of tan spot resistance in durum wheat than in common wheat. The LDN-*T. turgidum* ssp. *dicoccoides* chromosome substitution lines provide valuable tools for which to dissect and study the genetics of tan spot resistance in tetraploid wheat because they allow the effects of single chromosomes to be evaluated in isolation. Four chromosomes derived from IsraelA rendered LDN more resistant than euploid LDN and therefore likely possess tan spot resistance factors. Chromosome 3B from IsraelA conferred the highest levels of resistance to all four isolates.

Detailed comparative mapping of chromosome 3B in the BG, LouPen, and LD3B populations suggested that wild emmer accession IsraelA contains the same 3B resistance QTL as the hexaploid wheat cultivars BR34 and Penawawa (Faris and Friesen 2005; Kariyawasam et al. 2016). The gene underlying the QTL, which we designate as *Tsr7*, lies in a proximal region of 3BL. *Tsr7* confers race-nonspecific resistance in a dominant fashion indicating the interaction with the pathogen is different from the canonical inverse gene-for-gene interactions involving dominant susceptibility genes with pathogen-produced NEs such as the *Tsn1*-Ptr ToxA interaction. Furthermore, *Tsr7* resistance precludes susceptibility conferred by *Tsn1* (Kariyawasam et al. 2016) making it a desirable target for cloning and deployment. The

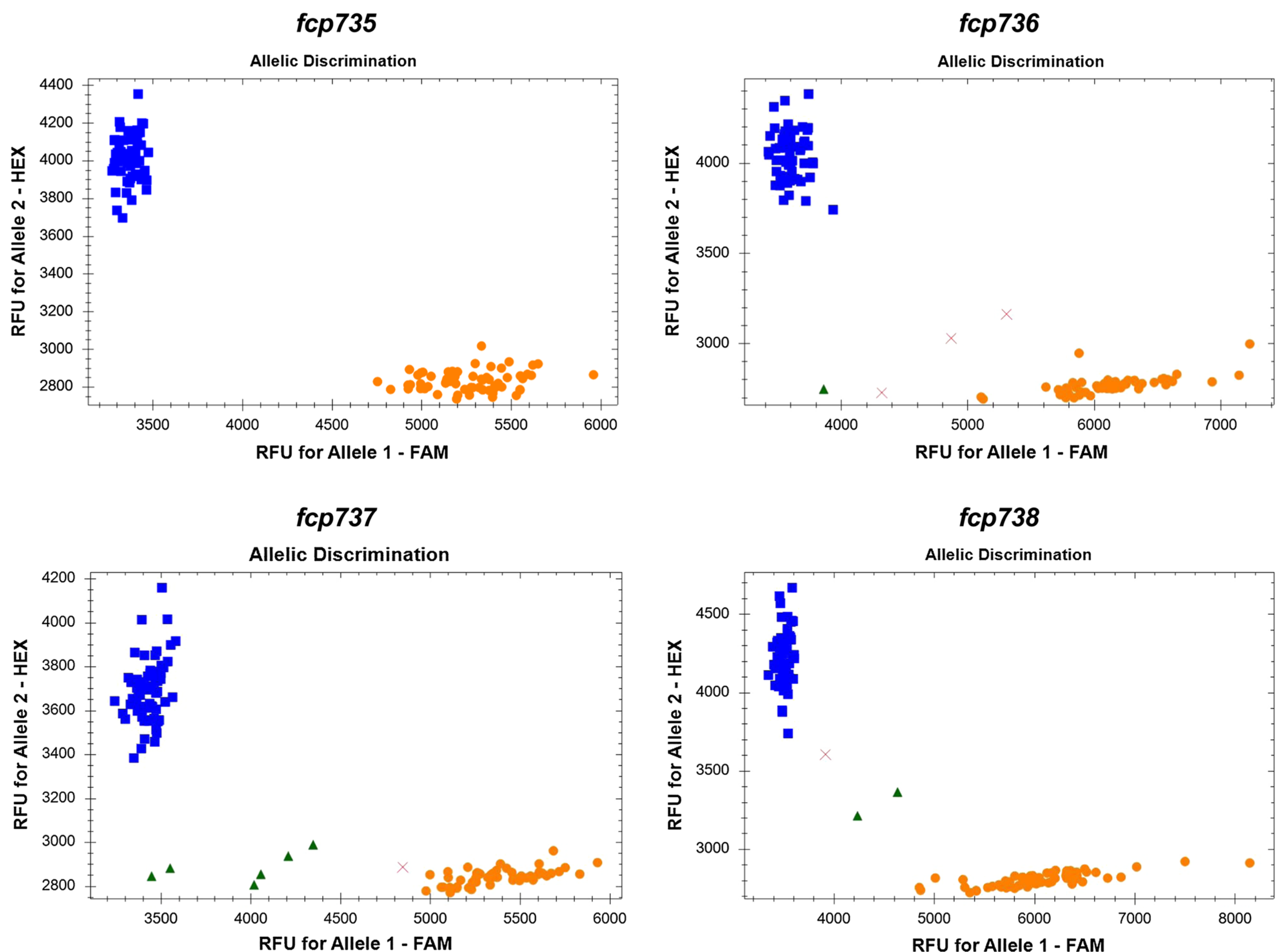

Fig. 5 Allele discrimination plots of the four STARP markers *fcp735*, *fcp736*, *fcp737*, and *fcp738* evaluated on the BG population

position of *Tsr7* in the proximal region of the chromosome would likely pose difficulties for conventional map-based cloning strategies but make it an attractable candidate for alternate strategies such as MutChromSeq (Sánchez-Martin et al. 2016).

In addition to the wheat-*P. tritici-repentis* pathosystem, other systems involve inverse gene-for-gene interactions consisting of dominant host susceptibility genes and pathogen-produced NEs. The wheat-*Parastagonospora nodorum* system involves nine host gene-NE interactions that confer susceptibility, but no dominant resistance with resemblance to *Tsr7* has been identified. However, the barley-*Pyrenophora teres* f. *teres* pathosystem consists of inverse gene-for-gene interactions involving dominant susceptibility genes and NEs as well as broad-spectrum dominant resistance genes, one of which resides on the long arm of chromosome 3H and could be homoeologous to *Tsr7* (Koladia et al. 2017). We speculate that the resistance conferred by *Tsr7* acts very early in the host–pathogen interaction and possibly prohibits penetration by the fungus or proliferation immediately after penetration, thereby precluding susceptibility conferred by the NE sensitivity genes such as *Tsn1*.

The finding that a wild emmer accession and two hexaploid bread wheat varieties carry the *Tsr7* gene is intriguing, but it is difficult to discern the origin. It is possible that *Tsr7* originated in the B-genome progenitor and passed through emmer (AB tetraploid) during the evolution of hexaploid wheat. However, gene flow between hexaploids and tetraploids subsequent to the formation of hexaploid wheat has occurred frequently (He et al. 2019), and it is possible that *Tsr7* was transferred from a hexaploid to the wild emmer accession IsraelA. To support this notion, we found that IsraelA contains the domesticated form of the *Q* allele, which it acquired from domesticated tetraploid or hexaploid wheat subsequent to the domestication of wheat (Faris unpublished) indicating the possibility that IsraelA may have also acquired *Tsr7* from a domesticated wheat form. Genome-wide association studies of large, diverse collections of tetraploid and hexaploid wheat would shed more light on the frequency of *Tsr7*, and the cloning of the gene would provide more insights regarding its origin.

The STARP markers developed in this research are closely linked to *Tsr7* and provide user-friendly tools for marker-assisted selection. However, analysis of the panel of wheat lines showed that either recombination events or mutations have occurred in some lines resulting in reversal of the repulsion/coupling association of the markers and QTL. Therefore, it is important to know the marker allele/QTL status in initial material before using these markers for selection of resistance.

Chromosome 6A derived from IsraelA conferred moderate levels of resistance to Pti2, 86-124, and 331-9, and although not significant, it was slightly more resistant to DW5 than was LDN. Therefore, chromosome 6A from IsraelA appears to harbor race-nonspecific resistance gene(s) with minor effects. To our knowledge, no tan spot resistance genes have been identified on chromosome 6A in either hexaploid or tetraploid wheat. In evaluations done by Singh et al. (2006, 2007, 2008a), IsA-6A also showed less disease than other substitution lines for all the isolates they used.

Chromosome 3A from IsraelA conferred minor levels of resistance to Pti2, 86-124, and DW5. Tadesse et al. (2006) reported a single recessive gene conferring resistance to a race 1 isolate (ASC1) derived from the landrace Salamouni on chromosome 3A, and Chu et al. (2010) identified a QTL with minor effects on 3A for resistance to Pti2 and 86-124, with resistance effects derived from the durum cultivar Lebsock. In addition, Singh et al. (2008b) showed that a QTL on chromosome arm 3AS was associated with resistance to tan spot caused by a different race 1 isolate. The possibility that the chromosome 3A resistance in IsraelA might be the same as that identified in other sources will need further investigation.

The IsraelA set of chromosome substitution lines was previously screened by Singh et al. (2007) for reaction to tan spot caused by races 1, 2, 3 and 5. For that experiment, they used the same isolates for races 1-3 (Pti2, 86-124, and 331-9) as the current study and a different isolate (DW13) for race 5, and they infiltrated the entire set with Ptr ToxA. Their results showed that all the IsraelA substitution lines were sensitive to Ptr ToxA, with the exception of IsA-5B, which agreed with our findings that the recessive *tsn1* allele on chromosome 5B is present in IsraelA.

However, there were some discrepancies between the results reported by Singh et al. (2007) and our results regarding the tan spot reactions obtained from fungal spore inoculation experiments. Singh et al. (2007) reported that the IsA-5B substitution line was resistant to tan spot caused by isolates Pti2 and 86-124 (races 1 and 2) and susceptible to isolates 331-9 and DW13 (races 3 and 5), whereas IsA-3B was resistant to isolates 331-9 and DW13, but not Pti2 and 86-124. Our results differ in that we found that IsA-5B was susceptible to all four tested isolates, and IsA-3B was resistant to all four. To confirm the results of our IsA-5B inoculations, we performed additional spore inoculations on plants derived from various seed sources, including seed from Dr. L.R. Joppa's early increases of this line, and all results indicated that IsA-5B was susceptible. In further support of our findings, Virdi et al. (2016) reported that IsA-5B was susceptible to tan spot caused by 86-124 even though it was insensitive to Ptr ToxA. We do not have a plausible explanation for the discrepancy between the study by Singh et al. (2007) and the current study.

The finding that sensitivity to the NE Ptr ToxA in durum wheat is not associated with susceptibility to Ptr ToxA-producing isolates is consistent with previous research in tetraploid

wheat. As summarized in Virdi et al. (2016), studies conducted to date indicate that the *Tsn1*-Ptr ToxA interaction is not associated with the development of tan spot in tetraploid wheat. However, in hexaploid wheat, the role of the *Tsn1*-Ptr ToxA interaction can range from nonsignificant to highly significant depending on the genetic background of the host. The reasons for this are not yet known, but the phenomenon is a subject of further study in our laboratories.

Although the *Tsn1*-Ptr ToxA interaction does not appear to be associated with tan spot susceptibility in tetraploid wheat, Virdi et al. (2016) showed that the *Tsc2*-Ptr ToxB interaction is important and significantly associated with susceptibility to chlorosis produced by race 5 isolates. The *Tsc2* gene lies on the short arm of chromosome 2B (Friesen and Faris 2004; Abeysekara et al. 2010), and although the substitution line IsA-2B was not available for evaluation, effects of the *Tsc2*-Ptr ToxB interaction would not have been observed because neither LDN or IsraelA harbor *Tsc2* (Fig. 2 and Virdi et al. 2016). Similarly, the *Tsc1*-Ptr ToxC interaction was not a factor in this research because LDN does not carry *Tsc1* (Virdi et al. 2016), and IsraelA did not express chlorotic symptoms upon infection by Ptr ToxC-producing isolates (Pti-2 and 331-9) suggesting that it does not harbor *Tsc1* either. Therefore, the *Tsc1*-Ptr ToxC interaction still has not been directly evaluated in tetraploid wheat.

The results of this work indicate that chromosome 3B from wild emmer accession IsraelA harbors *Tsr7*, a gene that confers high levels of race-nonspecific resistance to tan spot of durum wheat and it is likely the same as one that is known to exist in some lines of hexaploid wheat. The markers developed in this research should prove useful for deployment of the resistance and development of durum cultivars with improved tan spot resistance.

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Author contribution statement JF and ZL initiated the study and designed the experiment. JF, SX and AC developed and/or maintained the mapping populations and genetic stocks. JF, MO, GK, and AC conducted mapping studies and/or contributed marker information and marker analysis. MO developed STARP markers. JF, MO, and GK conducted linkage analysis. ZH, MO, and GK conducted tan spot inoculations and analysis. JF and ZH wrote the manuscript and all authors contributed to the final version.

Compliance with ethical standards

Conflict of interest All authors have no conflict of interest.

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