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ORIGINAL PAPER

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Identification of quantitative trait loci for race-nonspecific resistance to tan spot in wheat

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Abstract Tan spot, caused by *Pyrenophora tritici-repentis* (Ptr), is an economically important foliar disease in the major wheat growing areas throughout the world. Multiple races of the pathogen have been characterized based on their ability to cause necrosis and/or chlorosis on differential wheat lines. In this research, we evaluated a population of recombinant inbred lines derived from a cross between the common wheat varieties Grandin and BR34 for reaction to tan spot caused by Ptr races 1–3 and 5. Composite interval mapping revealed QTLs on the short arm of chromosome 1B and the long arm of chromosome 3B that were significantly associated with resistance to all four races. The effects of the two QTLs varied for the different races. The 1B QTL explained from 13% to 29% of the phenotypic variation, whereas the 3B QTL explained from 13% to 41% of the variation. Additional minor QTLs were detected but not associated with resistance to all races. The host-selective toxin Ptr ToxA, which is produced by races 1 and 2, was not a significant factor in the development of disease in this population. The race-nonspecific resistance derived from BR34 may take precedence over the gene-for-gene interaction known to be associated with the wheat–Ptr system.

Introduction

Tan spot of wheat, caused by *Pyrenophora tritici-repentis* (Died.) Drechs. (Ptr) [anamorph *Drechslera tritici-repentis* (Died.) Shoem.] is a major foliar disease of bread wheat (*Triticum aestivum* L.) and durum wheat (*T. turgidum* L. var. *durum*) in the United States and other major wheat growing areas throughout the world. Typical symptoms include a tan-colored, diamond-shaped necrotic lesion with a small, dark-brown infection site. Lesions are often surrounded by chlorotic halos (Weise 1987).

Lamari et al. (1995) proposed a race classification system based on necrotic and/or chlorotic symptoms produced on a set of wheat differential genotypes. Race 1 isolates produce both chlorosis and necrosis, race 2 produces necrosis only, races 3 and 5 produce chlorosis only (but on different host lines), and race 4 is avirulent. Additional races with various virulence combinations found in races 1–5 have since been described (Lamari et al. 2003). Races 1 and 2 produce the well-characterized host-selective toxin Ptr ToxA (Tomás and Bockus 1987; Lamari and Bernier 1989b; Tuori et al. 1995), race 3 produces Ptr ToxC (Effertz et al. 2002), and race 5 produces Ptr ToxB (Orolaza et al. 1995).

Reports regarding the inheritance of resistance to tan spot have ranged from qualitative (Lee and Gough 1984; Lamari and Bernier 1989b, 1991; Sykes and Bernier 1991; Gamba and Lamari 1998; Gamba et al. 1998) to quantitative (Nagle et al. 1982; Elias et al. 1989; Faris et al. 1997, 1999; Friesen et al. 2003; Cheong et al. 2004). Sensitivity to Ptr ToxA produced by race 2 (nec+ chl−) isolates is conditioned by a single dominant gene (Lamari and Bernier 1989b) designated *Tsn1* on the long arm of chromosome 5B (Faris et al. 1996; Anderson et al. 1999). It was suggested that sensitivity to Ptr ToxA and susceptibility to tan necrosis caused by the fungus were controlled by the same gene (Lamari and Bernier 1989b). However, other experiments have indicated that importance of Ptr ToxA in causing disease is dependant on host genetic background (Friesen et al. 2003).

Resistance to chlorosis induced by races 1 and 3 was found to be controlled by a major QTL designated *QTsc.ndsu-1A* on the short arm of chromosome 1A in the International Triticeae Mapping Initiative (ITMI)

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population (Faris et al. 1997, 1999). Effertz et al. (2001) confirmed the association of *QTsc.ndsu-1A* with resistance to race 3 in a different population. In addition, the gene (*Tsc1*) conditioning sensitivity to Ptr ToxC was mapped to the *QTsc.ndsu-1A* locus in the ITMI population (Effertz et al. 2002). Friesen and Faris (2004) investigated resistance to race 5 in the ITMI mapping population and mapped the Ptr ToxB-sensitivity gene, designated *Tsc2*, to the short arm of chromosome 2B. The *tsc2* allele was responsible for the effects of a major QTL associated with resistance to tan spot caused by race 5. Therefore, all major tan spot resistance genes and QTLs reported until now have been race-specific and implicated in decreased toxin sensitivity.

Here, we report the identification of *P. tritici-repentis* race-nonspecific resistance QTLs in a population of recombinant inbred (RI) lines derived from the hard red spring wheat varieties Grandin and BR34.

Materials and methods

Plant materials

A segregating population of RI lines derived from a cross between the Brazilian hard red spring wheat (HRSW) BR34 (Bered et al. 2002) and the North Dakota HRSW Grandin (PI 531005) consisting of 118 $F_{7:9}$ lines was developed and provided by Dr. James Anderson, University of Minnesota, St. Paul, Minn., USA. This population, hereafter referred to as the BG population, is the subject of an extensive mapping effort (Z.H. Lui, J.A. Anderson, J.G. Hu, T.L. Friesen, J. Rasmussen, and J.D. Faris, submitted). BR34 is insensitive to Ptr ToxA and highly resistant to all races tested, whereas Grandin is sensitive to Ptr ToxA, and at least moderately susceptible to all races tested.

Fungal isolates, inoculation, and rating

Ptr races 1 (Pti2) (Friesen et al. 2002), 2 (86-124) (Friesen et al. 2003), 3 (OH99), and 5 (DW5) (Friesen and Faris 2004) were used in this study. OH99 was a recently identified race 3 isolate collected from Ohio by Pat Lipps and Jessica Engle, The Ohio State University, Columbus, Ohio, USA. For disease analysis, RI lines were inoculated with conidia of each isolate separately. Inoculations were done at the two-to-three-leaf stage. Individual lines of the BG population were planted along with parents using three 4 cm × 21-cm containers (Stuewe and Sons, Corvallis, Ore., USA) per line and three plants per container. Plants were placed in racks of 98, consisting of 20 lines, and a border of wheat plants was used to eliminate any edge effect. Cultures were grown and conidia were harvested as described by Lamari and Bernier (1989a). Spore inoculum was diluted to 3,000 spores/ml, and two drops of Tween-20 were added per 100 ml of inoculum. Plants were inoculated until runoff. Following inoculation plants were placed in 100% relative humidity in the dark at 21°C for 24 h, and then placed in a controlled chamber under a 12-h photoperiod at 21°C. Disease readings were taken at 7 days post-inoculation using the 1–5 lesion type scale developed by Lamari and Bernier (1989a) in which 1 = small dark-brown to black spots without any surrounding chlorosis or tan necrosis (resistant); 2 = small dark-brown to black spots with very little chlorosis or tan necrosis (moderately resistant); 3 = small dark-brown to black spots completely surrounded by a distinct chlorotic or tan necrotic ring, lesions generally not coalescing (moderately resistant to moderately susceptible); 4 = small dark-brown or black spots completely surrounded with chlorotic or tan necrotic zones, some of the lesions coalescing (moderately susceptible); and 5 = dark-brown or black centers that may or may not be distinguishable, most lesions consist of coalescing chlorotic or tan necrotic zones (susceptible). For each isolate, the entire mapping population and parents were inoculated in three experiments with three replications each. Therefore, a total of 27 plants per RI line were tested with each isolate.

Ptr ToxA was purified from isolate 86-124 by Dr. Steven Meinhardt, North Dakota State University, Fargo, N.D., USA, as described in Zhang et al. (1997). The parents and entire RI population were infiltrated with the toxin and scored for reaction as described in Haen et al. (2004). Purified Ptr ToxB and Ptr ToxC were not available for this study.

Molecular mapping

Over 700 molecular markers spanning the entire genome have been mapped in the BG population, and the whole genome analysis will be published elsewhere (Z.H. Lui, J.A. Anderson, J.G. Hu, T.L. Friesen, J. Rasmussen, and J.D. Faris, submitted). Briefly, DNA was isolated from parents and each RI line as described in Faris et al. (2000). Target region amplified polymorphisms [(TRAPs) *Xfcp* markers] were generated as described in Hu and Vick (2003). Briefly, TRAPs were multiplexed by using a fixed primer in combination with two random primers 5′-end labeled with different dyes (IR dye 700 or IR dye 800). Fixed primers were designed from randomly selected mapped wheat EST sequences (http://www.ncbi.nlm.nih.gov). TRAPs generate a profile similar to AFLPs, but are more user-friendly because they do not require extensive pre-PCR processing of samples. TRAPs are not locus-specific and tend to reveal multiple polymorphic fragments that represent loci scattered throughout the genome. In the BG population, an average of 24 TRAP markers per reaction was obtained.

Microsatellite markers *Xbarc* (Cregan and Song, http://www.scabusa.org), *Xgwm* (Röder et al. 1998), and *Xgdm* (Pestsova et al. 2000) were amplified following the procedures of Röder et al. (1998), electrophoresed through a denaturing 6% polyacrylamide gel, stained

with Sybr green II, and visualized with a Typhoon 9410 variable mode imager (Molecular Dynamics, Sunnyvale, Calif., USA). The computer program MAPMAKER, version 2.0 for Macintosh (Lander et al. 1987), was used to assemble linkage groups using a logarithm of the odds (LOD) threshold of 3.0 and the Kosambi mapping function (Kosambi 1944). Linkage groups were assigned to chromosomes by testing a number of markers on wheat aneuploid stocks. Reactions of RI lines to Ptr ToxA were assigned genotypic values and assessed using the *TRY* command to determine the most plausible position of *Tsn1* on the genetic map.

QTL analysis

The entire marker dataset, which consists of 743 markers, was tested using simple linear regression to identify markers significantly ($P < 0.001$) associated with resistance to Ptr races using the computer program Map Manager QTX (Manly et al. 2001). Simple interval mapping (SIM) (Haley and Knott 1992) and composite interval mapping (CIM) were performed using a subset of 252 markers mapping at a LOD > 3.0 and spaced approximately 10–20 cM apart to evaluate marker intervals putatively associated with resistance. The 95% confidence intervals for individual QTLs were established by bootstrapping. To determine the critical LOD threshold, we executed a permutation test with 5,000 permutations. A LOD threshold of about 3.0 in this RI population yields an experiment-wise significance level of 0.05. Markers with significant main effects were tested for significant ($P < 1.0 \times 10^{-7}$) interactions with all other markers in the dataset. Markers with significant main effects for resistance to individual races were assembled into a multiple regression model. The coefficient of determination (R^2) from the multiple regression model is the proportion of the total phenotypic variation explained by the markers.

Results

Mapping of *Tsn1*

The BG population segregated in a ratio of 59 sensitive:59 insensitive for reaction to Ptr ToxA, which fit the expected 1:1 ratio for a single gene ($\chi^2 = 0.0$, $P > 0.95$). RI lines were given genotypic values for toxin reactions, and *Tsn1* mapped to the long arm of chromosome 5B as expected (data not shown).

Reaction to conidial inoculations of Ptr races 1–3 and 5

Phenotypic scores obtained by fungal inoculations of races showed a distribution indicative of a quantitatively controlled trait (Fig. 1). Average reaction types of parents and RI lines to races 1–3 and 5 are shown in Table 1. BR34 was resistant to all four races, whereas Grandin was highly susceptible to race 1 and moderately susceptible to races 2, 3, and 5. For races 1 and 2 (Ptr ToxA-producing races), we also evaluated disease-reaction types separately for Ptr ToxA-sensitive and -insensitive lines. The mean lesion types of both sensitive and insensitive lines were moderately resistant, but toxin insensitive lines had a slightly lower average than sensitive lines (Table 1, Fig. 1).

QTL identification

Single-factor regression, SIM, and CIM analysis revealed a total of seven QTLs significantly associated with resistance to tan spot in this study, and in all cases, resistance was contributed by BR34. Genomic regions on the short arm of chromosome 1B and the long arm of chromosome 3B harbored QTLs significantly associated with resistance to all four races (Tables 2, 3; Fig. 2). The 1BS QTL, which we designated *QTs.fcu-1BS* according to McIntosh et al. (1998), was located between markers *Xgdm33* and *Xgdm125* (Table 3; Fig. 2). The effects of this QTL explained from 13% of the phenotypic variation for resistance to race 5 to 29% of the variation for resistance to race 3 (Table 3). We designated the race-nonspecific QTL on 3BL as *QTs.fcu-3BL*, which was bracketed by markers *Xbarc248.1* and *Xfcp83*. This QTL accounted for 13–41% of the phenotypic variation among the four races.

A third QTL was detected by single-factor regression and CIM on the short arm of chromosome 3B, but this

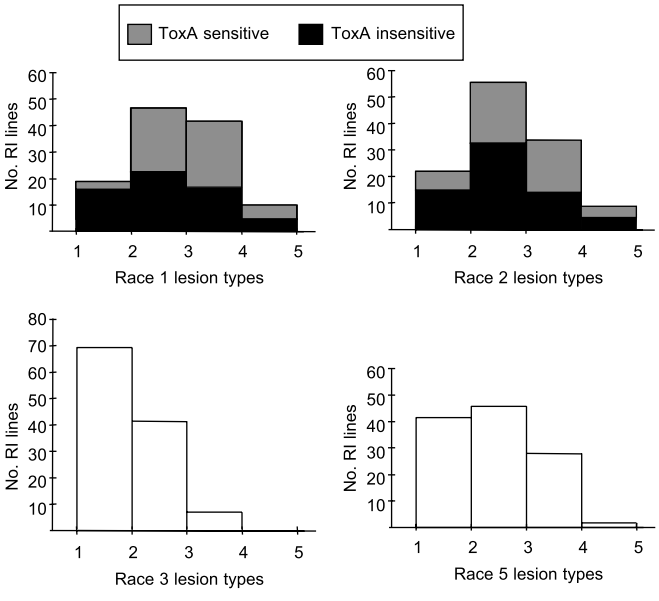

Fig. 1 Histograms of lesion type means in the BG population after inoculation with *Pyrenophora tritici-repentis* (Ptr) races 1–3 and 5. For races 1 and 2, toxin-sensitive and -insensitive lines are indicated in *gray* and *black*, respectively. *White bars* for races 3 and 5 refer to the whole population

Table 1 Lesion type means of Grandin, BR34, and the BG population to conidial inoculations of *Pyrenophora tritici-repentis* (*Ptr*) races (*R*) 1–3 and 5. *RI* Recombinant inbred line

Race	BR34	Grandin	RI pop. average	RI pop. range	Ptr ToxA-insensitive lines	Ptr ToxA-sensitive lines
1	1.33	4.33	2.75	1.33–4.83	2.55	2.95
2	1.50	3.83	2.68	1.50–4.50	2.47	2.90
3	1.17	3.50	1.81	1.00–3.33	1.70	1.93
5	1.00	3.33	2.36	1.00–4.17	2.23	2.50

Table 2 Single-factor regression analysis of individual markers associated with BR34-derived resistance to Ptr R1, R2, R3, and R5 in the BG population

Marker	Chromosome	R^2 value[a]			
		R1	R2	R3	R5
Xgdm125	1BS	0.27***	0.14***	0.28***	0.12***
Xgwm614	2DS	NS	0.13*	NS	NS
Xfcp83	3BL	0.18***	0.26***	0.13***	0.40***
Xfcp114	3BS	NS	NS	0.12***	NS
Xbarc190	4AL	NS	NS	NS	0.07**
Xfcp231	5AL	0.11*	NS	NS	0.07**
Xbarc126	7DS	NS	NS	0.09*	NS

Significance levels: ***$P < 0.00001$, ** $P < 0.0001$, * $P < 0.0005$, NS nonsignificant
[a]Multiple regression model R^2 values for R1 = 0.48, R2 = 0.45, R3 = 0.47, R5 = 0.55

Table 3 Composite interval mapping analysis of QTLs associated with resistance to tan spot caused by Ptr R1, R2, R3, and R5 in the BG population

QTL	Marker interval	Chromosome-peak position (cM)	95% CI (cM)[a]	R^2				Logarithm of the odds			
				R1	R2	R3	R5	R1	R2	R3	R5
QTs.fcu-1BS	*Xgdm33–Xgdm125*	10.0	4.0–19.0	0.27	0.14	0.29	0.13	8.05	5.47	8.61	5.94
QTs.fcu-3BL	*Xbarc248–Xfcp83*	128.0	121.0–154.0	0.17	0.24	0.13	0.41	6.75	7.18	5.03	13.51
QTs.fcu-3BS	*Xfcp311–Xfcp114*	55.0	48.0–72.0	NS	NS	0.12	NS	NS	NS	4.60	NS

[a]CentiMorgan position of the 95% confidence interval for the QTL

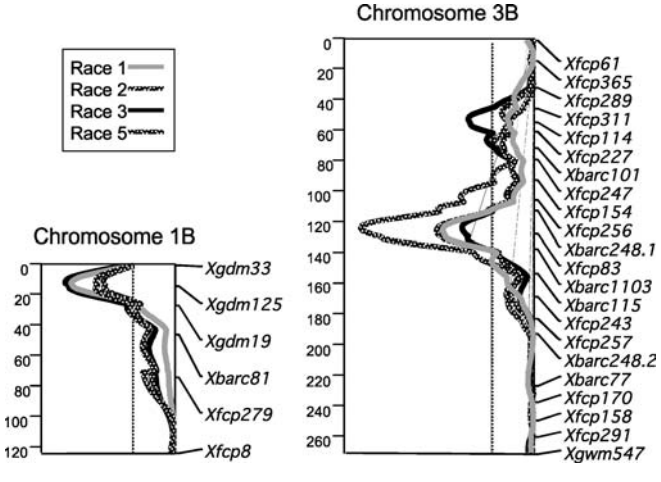

Fig. 2 Interval regression maps of chromosomes 1B and 3B indicating QTLs significantly associated with resistance to Ptr races 1–3 and 5. The *dotted line* represents the logarithm of the odds (LOD) significance threshold of 3.0. The LOD values for the QTL peaks are presented in Table 3. A centiMorgan scale is indicated to the *left* of the maps

QTL was specifically associated with resistance to race 3 (Tables 2, 3; Fig. 2). This QTL, designated *QTs.fcu-3BS*, accounted for 12% of the variation for resistance to race 3.

Four QTLs had relatively minor effects and were detected by single-factor regression but not CIM. Marker *Xfcp231* on the long arm of chromosome 5A was significantly associated with resistance to races 1 and 5 and explained 11% and 7% of the phenotypic variation, respectively (Table 2).

The remaining three putative minor QTLs were race-specific. One QTL was detected by marker *Xgwm614* on the short arm of chromosome 2D and accounted for 13% of the variation for resistance to race 2 (Table 2). Marker *Xbarc190* on the long arm of chromosome 4A explained 7% of the variation for resistance to race 5, and marker *Xbarc126* on 7DS accounted for 9% of the variation for resistance to race 3 (Table 2).

The *tsn1* gene, which confers insensitivity to Ptr ToxA produced by races 1 and 2, was not significantly associated with resistance at the significance thresholds used in this study. For race 1, *tsn1* had a *P*-value of 0.03

and an R^2 value of 0.04, and for race 2, it had a *P*-value of 0.006 and an R^2 value of 0.06.

No significant interactions involving the markers underlying QTLs, or any other markers in the dataset, were identified. With the exception of markers *Xbarc190-4AL* and *Xbarc127-7DS*, which were associated with resistance to races 5 and 3, respectively, all other markers retained significance in multiple regression models. The total amount of variation explained by models for each race ranged from 45% for race 2 to 55% for race 5 (Table 2).

Discussion

In this work, we identified two QTLs, *QTs.fcu-1BS* and *QTs.fcu-3BL*, associated with resistance to tan spot caused by Ptr races 1–3 and 5. To our knowledge, this is the first report of the identification and mapping of QTLs for race-nonspecific resistance to tan spot, and also where resistance is not clearly explained by decreased toxin sensitivity. Resistance to tan spot in wheat is generally thought of as race-specific following a gene-for-gene system involving the recognition of pathogen-produced host-selective toxins by specific genes in the host (Lamari et al. 2003). For example, the genes *tsn1*, *tsc1*, and *tsc2*, which confer insensitivity to Ptr ToxA, Ptr ToxC, and Ptr ToxB, respectively, underlie major QTLs on chromosomes 5BL, 1AS, and 2BS for resistance to races 2, 3, and 5, respectively (Effertz et al. 2002; Cheong et al. 2004; Friesen and Faris 2004). Our research suggests that BR34 harbors (a) resistance mechanism(s) that does/do not follow a gene-for-gene model, and instead recognize multiple pathogenic races of Ptr.

Tsn1 confers sensitivity to Ptr ToxA, which is produced by races 1 and 2. Ptr ToxA is considered to be a major factor in the development of disease by these races (Lamari and Bernier 1989b; Friesen et al. 2003). However, our results indicate that sensitivity to Ptr ToxA was a nonsignificant factor in disease development, which suggests that *tsn1* plays at most a very minor role in conditioning resistance in the BG population. It is possible that the race-nonspecific resistance mechanisms harbored by BR34 decrease toxin activity and render plants resistant before the manifestation of necrosis by the toxin. It is also possible that the toxin is at least partially contained, which would lead to a high level of resistance regardless of toxin sensitivity. Further investigations regarding toxin–wheat interactions at the molecular level are needed to determine the mode of action of the toxin. Also, molecular investigations of race-nonspecific and non-host specific resistance are needed to resolve the mechanisms governing these interactions.

The effects of *QTs.fcu-1BS* and *QTs.fcu-3BL* were significant for all races tested in this study, but their effects varied for the different races. *QTs.fcu-1BS* was the most significant QTL for races 1 and 3, explaining 27% and 29% of the variation, respectively, whereas *QTs.fcu-3BL* was the most significant QTL for resistance to races 2 and 5, explaining 24% and 41% of the variation, respectively. It is possible that some of the variation in the effects of the two QTLs for the different races is because of environmental factors or experimental error. However, we conducted replicated experiments under controlled greenhouse conditions to minimize environmental influences as much as possible. It is also possible that the resistance mechanisms underlying the QTLs vary in their capacity to recognize different races/isolates.

A third QTL (*QTs.fcu-3BS*) significantly associated with resistance to race 3 was detected on the short arm of chromosome 3B. Whereas this QTL was not significantly associated with resistance to races 1, 2, or 5 by simple linear regression or CIM, the data suggest that this genomic region harbors a minor factor for resistance to all the races (Fig. 2). We identified other putative minor QTLs on chromosomes 2DS, 4AL, 5AL, and 7DS. Of these, only the QTL on 5AL was significantly associated with resistance to more than one race (races 1 and 5). Mapping in a much larger population is needed to validate the significance of these putative QTLs.

Of the QTLs identified in this research, only two have the potential of being the same as previously identified tan spot resistance QTLs. First, Faris et al. (1997) reported a minor QTL in the proximal region of 3BL associated with resistance to race 1 in the ITMI mapping population. The position of this QTL in the ITMI population coincides with *QTs.fcu-3BL* in the BG population. In both populations, the effect of this QTL for resistance to race 1 was relatively minor, and it is possible that it is governed by a common locus. Second, the putative QTL on 4AL weakly associated with resistance to race 5 coincides with the position of a race 5 resistance QTL on 4AL in the ITMI population reported by Friesen and Faris (2004). Faris et al. (1997, 1999) also reported that this QTL was associated with resistance to race 1 in the ITMI population, but we found no indication of a 4AL QTL associated with resistance to race 1 in the BG population.

Our multiple regression models explained from 45% to 55% of the phenotypic variation for resistance to the Ptr races used in this study. It is possible that additional significant QTLs segregate in the BG population but went undetected. Although the total marker dataset consists of over 700 markers, gaps of considerable size still exist on some chromosomes, and on several chromosome arms few or no markers were available (Z.H. Liu, J.A. Anderson, J.G. Hu, T.L. Friesen, J.B. Rasmussen, and J.D. Faris, submitted). In particular, the short arm of chromosome 1A contains only two markers. Chromosome 1AS is where *Tsc1*, which confers sensitivity to Ptr ToxC (Effertz et al. 2002), and a major QTL for resistance to race 1 (*QTsc.ndsu-1AS*, Faris et al. 1997), were previously mapped in the ITMI population. Therefore, if *QTsc.ndsu-1AS* were associated with resistance in the BG population, it went undetected. The

addition of more markers to the maps, chromosome 1AS in particular, will lead to more complete genome coverage and allow all genomic regions to be surveyed for additional QTLs.

Incorporation of resistance loci underlying QTLs *QTs.fcu-1BS* and *QTs.fcu-3BL* into elite lines and cultivars would provide broad-spectrum resistance to tan spot. It may be further beneficial to pyramid these resistance QTLs together with toxin insensitivity genes *tsn1*, *tsc1*, and *tsc2* into germplasm to possibly provide a more complete level of resistance to all races/isolates in the event that the race-nonspecific QTLs identified in this research are overcome by the pathogen or are not effective against races/isolates not tested in this study. Suitable markers for *tsn1*, *tsc1*, and *tsc2* have been identified (Effertz et al. 2002; Friesen and Faris 2004; Haen et al. 2004), and the molecular markers bracketing *QTs.fcu-1BS* and *QTs.fcu-3BL* are suitable for use in marker-assisted selection schemes as well.

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