Identification and Molecular Mapping of a Gene Conferring Resistance to *Pyrenophora tritici-repentis* Race 3 in Tetraploid Wheat

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ABSTRACT

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Race 3 of the fungus *Pyrenophora tritici-repentis*, causal agent of tan spot, induces differential symptoms in tetraploid and hexaploid wheat, causing necrosis and chlorosis, respectively. This study was conducted to examine the genetic control of resistance to necrosis induced by *P. tritici-repentis* race 3 and to map resistance genes identified in tetraploid wheat (*Triticum turgidum*). A mapping population of recombinant inbred lines (RILs) was developed from a cross between the resistant genotype *T. turgidum* no. 283 (PI 352519) and the susceptible durum cv. Coulter. Based

on the reactions of the Langdon-*T. dicoccoides* (LDN[DIC]) disomic substitution lines, chromosomal location of the resistance genes was determined and further molecular mapping of the resistance genes for race 3 was conducted in 80 RILs of the cross *T. turgidum* no. 283/Coulter. Plants were inoculated at the two-leaf stage and disease reaction was assessed 8 days after inoculation based on lesion type. Disease reaction of the LDN(DIC) lines and molecular mapping on the *T. turgidum* no. 283/Coulter population indicated that the gene, designated *tsn2*, conditioning resistance to race 3 is located on the long arm of chromosome 3B. Genetic analysis of the F₂ generation and of the F_{4:5} and F_{6:7} families indicated that a single recessive gene controlled resistance to necrosis induced by race 3 in the cross studied.

Tan spot of wheat, caused by the fungus *Pyrenophora tritici-repentis* (Died.) Drechs., is a destructive disease found in wheat-growing regions worldwide. The disease can cause serious yield losses by reducing the photosynthetic area of leaves, resulting in reduced grain filling and lower number of grains per head (6,38). Tan spot also causes kernel discoloration, mainly red smudge, dark smudge, and black point, thereby downgrading wheat quality (18). Changes in cultural practices, including shifts from conventional tillage and stubble burning to conservation tillage systems, shorter crop rotations, and continuous wheat cultivation, have led to an increase in the severity and incidence of tan spot. Although a number of management practices and fungicides are effective in controlling tan spot, resistant cultivars are the most effective and economical means of controlling this disease (9).

Following infection, virulent races of *P. tritici-repentis* elicit two distinct symptoms, tan necrosis and chlorosis, on susceptible wheat cultivars, both of which are under independent genetic control in the host (22,30). Based on the ability to induce necrosis or chlorosis on a set of wheat differential cultivars, isolates of the *P. tritici-repentis* presently are classified into eight races (31), although preliminary reports indicate the occurrence of three putative new races (1,2,34).

The tan spot fungus produces different host-specific toxins which have been associated with the necrosis and chlorosis symptoms induced on susceptible wheat cultivars. The well-characterized host-selective proteinaceous toxin, Ptr ToxA (7,29), produced by races 1, 2, 7, and 8 (31), is the main factor causing

necrotic symptoms in susceptible wheat cultivars. Two chlorosis-inducing host-specific toxins, designated Ptr ToxB (36), isolated from race 5, and Ptr ToxC (12), isolated from race 1, have been identified and well characterized. Recently, in preliminary reports, two different toxins simultaneously were named Ptr ToxD, one inducing necrosis (34) and the other causing chlorosis (2).

Genetic studies of tan spot of wheat have reported resistance to be either quantitatively (13,16,35) or qualitatively (10,11,22,29,30) inherited. The wheat-*P. tritici-repentis* pathosystem follows the toxin model, wherein compatible interaction between host plant and pathogen leads to susceptibility, the result of the pathogen-produced toxin and its toxin-receptor in the host (31,40). Genetic studies indicate that a single recessive gene, *tsn1*, controls resistance to necrosis caused by races 1 and 2 in both durum and common wheat and is located on the long arm of chromosome 5B (3,15). Effertz et al. (12) mapped the gene, *tsc1*, controlling insensitivity to toxin Ptr ToxC (race 3) in hexaploid wheat to the short arm chromosome 1A. Faris et al. (16), studying hexaploid wheat, observed that resistance to extensive chlorosis by race 3 was quantitative, with a major portion of the variation controlled by a quantitative trait locus or loci (QTL) on 1AS. In hexaploid wheat, Friesen and Faris (20) identified the Ptr ToxB insensitivity gene, *tsc2*, at the distal tip of the short arm of chromosome 2B, and this gene accounted for 69% of the phenotypic variation caused by race 5. In a recent report, Faris and Friesen (17) for the first time identified race nonspecific QTL on chromosomes 1B and 3B showing significant resistance against races 1, 2, 3, and 5 in a hexaploid wheat population.

A set of Langdon durum-*T. dicoccoides* (LDN[DIC]) disomic substitution lines was developed using the *T. dicoccoides* accession Israel-A as the donor parent (25,26). Each LDN(DIC) substitution line carries 13 pairs of chromosome from the recurrent parent (Langdon) and 1 chromosome pair from the donor parent

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(Israel-A). Agronomic, quality traits, and disease reactions of the substitution lines can be compared with the recurrent parent, Langdon, and the differences can be attributed to genes or lack of genes on the chromosome derived from the donor parent, *T. dicoccoides* accession Israel-A (4,5,23,43,44). The LDN(DIC) substitution lines can be efficient genetic stocks for determining the chromosomal location of resistance genes in the tan spot-wheat pathosystem because it follows the toxin model of the gene-for-gene hypothesis (31). Langdon is susceptible to race 3, whereas *T. dicoccoides* accession Israel-A is resistant, making it possible to identify the location of major resistance genes using the LDN(DIC) substitution lines.

Isolates of *P. tritici-repentis* races 3 and 5 and their combinations behave differentially on hexaploid and tetraploid wheat (21, 28,31). They cause chlorosis in hexaploid wheat but necrosis in tetraploid wheat. Gamba and Lamari (21), in the only published study, analyzed the genetics of resistance to necrosis induced by race 3 in tetraploid wheat and found resistance to be controlled by a single recessive gene. However, genetic location of this resistance gene is unknown. The objectives of this study were to determine the genetics of resistance to necrosis induced by *P. tritici-repentis* race 3 in a tetraploid wheat genotype different than those used by Gamba and Lamari (21), map its chromosomal location, and identify microsatellite markers closely linked to the gene or genes for resistance.

MATERIALS AND METHODS

Plant material. In the greenhouse tests, tetraploid genotypes *T. turgidum* no. 283 (PI 352519) (TT283) and durum cv. Coulter (DT-188/Dt-224/DT-182) were resistant and susceptible, respectively, to *P. tritici-repentis* race 3. To study the inheritance of resistance to the necrosis component of tan spot in tetraploid wheat, the F₂ generation and F_{4,5} families of the cross TT283/Coulter were developed and screened with *P. tritici-repentis* race 3. The F_{6,7} families also were tested with race 3 to provide additional data and provide phenotypic data for the QTL analysis. All F_{4,5} and F_{6,7} families were produced by single-seed descent. Eighty families in the F_{4,5} and F_{6,7} generations were tested for the genetic studies. The LDN(DIC) substitution lines and their parents, along with checks Erik, Glenlea, 6B-365, and 6B-662, were screened to determine the chromosomal location of the gene or genes controlling resistance to necrosis induced by *P. tritici-repentis* race 3 in tetraploid wheat.

Disease screening. Single-spore culture of isolate Ptr 331-9 (race 3) was provided by Dr. L. Lamari, University of Manitoba, Winnipeg, Canada and was used to produce spore inoculum. Prior to screening, the *P. tritici-repentis* isolate was characterized for its race designation following the procedures reported by Lamari and Bernier (28). Spore inoculum was produced by the method of Lamari and Bernier (28) and the concentration of the inoculum was 3,000 conidia/ml.

Disease testing of each population was conducted using a completely randomized design. Seed of the segregating population of the cross TT283/Coulter, substitution lines, and appropriate checks and parents were pregerminated and seed at a similar stage of germination were planted in plastic cones (3.8 cm in diameter and 20 cm in length) filled with Fison sunshine blend no. 1 (Fison Horticulture, Vancouver, BC, Canada) and grown in the greenhouse at an average temperature of 21°C with a 16-h photoperiod. Four seeds were planted in each cone. The seedlings were watered and fertilized as needed. The seedling leaves were kept dry while plants were watered to decrease the chance of saprophytic growth of microorganisms, which could interfere with the results. Using a hand-sprayer, plants at the two-leaf stage were sprayed until runoff with the conidial suspension. Following inoculation, the seedlings were moved to a mist chamber at 21°C with a 16-h photoperiod for 24 h. The chamber was misted for

16 s at 2-min intervals. Thereafter, the plants were moved to a growth chamber at 22 and 17°C (day and night) with a 16-h photoperiod.

The seedlings were rated for their disease reactions 8 days after inoculation. The 1-to-5 lesion-type rating scale developed by Lamari and Bernier (27) was used to rate the reaction of the seedlings to tan spot. For QTL analysis, mean of the recombinant inbred lines (RILs) developed after screening at least 20 plants per line on a lesion-type scale was used. For analysis as a qualitative trait, plants with ratings of 1 to 2 and 3 to 5 were considered as resistant and susceptible, respectively.

Molecular marker analysis. DNA extraction was done on bulk of at least 12 plants per line according to the methods described by Del Blanco et al. (8). Microsatellite markers *gwm* (37), *barc*, (available online from the U.S. Wheat and Barley Scab Initiative website), and *wmc* (41), previously mapped to chromosome 3B, were used to screen the parental lines for polymorphisms. Those markers found to be polymorphic were run on the F_{6,7} RIL population. Amplification conditions for each marker were determined following the protocols described previously by Röder et al. (37) and Somers et al. (41). Polymerase chain reaction products were separated on a 6% nondenaturing polyacrylamide gel and stained with ethidium bromide as described by Shi et al. (39).

Statistical analysis. Linkage analysis and QTL mapping was performed with MapManager QTX (33). In linkage analysis, a *P* value of 10⁻⁵ was used as the cut-off value to determine linkage between loci. The QTL analysis was done using phenotypic means of the RILs. An interval mapping approach was used to generate a likelihood ratio statistic (LRS) as the measure of the significance of a possible QTL. This LRS differs from a conventional base-10 log of the likelihood ratio score by a factor of 4.6. Threshold values to determine significance, with a *P* value of ≤0.001, were calculated using a permutation test with 1,000 random permutations.

Segregation data observed in the F₂ generation and F_{4,5} and F_{6,7} families were analyzed using the χ^2 test for goodness-of-fit to various genetic models to determine the genetic control of resistance (43). A family size of a minimum of 20 plants was used to have a greater than 99% probability of correctly distinguishing between a segregating and nonsegregating family in a single gene segregation analysis (24).

RESULTS

Each of the 14 LDN(DIC) substitution lines gave clear and consistent disease reactions to race 3. Cv. Langdon and the accession Israel-A (*T. dicoccoides*) were susceptible and resistant, respectively (Table 1). The checks gave reactions expected for inoculation with race 3. All the LDN(DIC) substitution lines tested gave susceptible reaction to race 3, except the line with a substitution for 3B chromosome LDN(DIC-3B) (Table 1). This indicated that the gene or genes for resistance to necrosis caused by race 3 in tetraploid wheat was likely to be located on chromosome 3B.

Given the results from the inoculation of LDN(DIC) substitution lines, a linkage map was constructed for chromosome 3B using the F_{6,7} generation of the TT283/Coulter population. This linkage map consisted of 15 SSR loci distributed into three linkage groups (Fig. 1A). The linear order of the loci corresponds to that of previously published linkage maps of chromosome 3B (37,41).

QTL analysis for resistance to race 3 resulted in a single QTL peak in the interval defined by *Xgwm285* and *Xwmc366.2*. The LRS value at this peak was 199.1 (Fig. 1B). This QTL explained 91% of the phenotypic variation for resistance to race 3. This suggests the presence of a major single gene underlined by the QTL. The lesion type means of the 80 F_{6,7} RILs of the cross

TT283/Coulter resembled a binomial distribution (Fig. 2). Most of the RILs exhibited either resistant or susceptible lesion types, similar to parents TT283 and Coulter, indicative of qualitative inheritance. Based on these results, we included the resistance as a single locus in the linkage map. For this purpose, plants with disease ratings of 1 and 2 were considered resistant, like the TT283 parental lines, whereas plants with ratings of 3 to 5 were considered susceptible, like the Coulter parent. The resistance locus mapped on the long arm of chromosome 3B, 2.1 centi-

TABLE 1. Reaction to *Pyrenophora tritici-repentis* race 3 of Langdon-*Triticum dicoccoides* LDN(DIC) disomic substitution lines, their parents, and other checks used to determine the chromosomal location of resistance genes

Substitution line, genotype	Disease score ^a	Reaction to race 3 ^b
Glenlea	1.73	Resistant
Erik	1.56	Resistant
6B-662	1.80	Resistant
6B-365	4.50	Chlorotic
Coulter	4.43	Necrotic
<i>T. turgidum</i> no. 283	1.72	Resistant
Langdon	4.14	Necrotic
<i>T. dicoccoides</i> (Israel-A)	1.66	Resistant
LDN (DIC-1A)	4.25	Necrotic
LDN (DIC-2A)	4.43	Necrotic
LDN (DIC-3A)	4.50	Necrotic
LDN (DIC-4A)	4.25	Necrotic
LDN (DIC-5A)	4.39	Necrotic
LDN (DIC-6A)	3.10	Necrotic
LDN (DIC-7A)	4.00	Necrotic
LDN (DIC-1B)	4.00	Necrotic
LDN (DIC-2B)	3.75	Necrotic
LDN (DIC-3B)	1.80	Resistant
LDN (DIC-4B)	3.29	Necrotic
LDN (DIC-5B)	4.14	Necrotic
LDN (DIC-6B)	4.38	Necrotic
LDN (DIC-7B)	3.63	Necrotic

^a Based on mean score of at least 12 plants.
^b Plants were rated on a 1-to-5 lesion-type rating scale developed by Lamari and Bernier (26). Plants with rating of 1 to 2 were considered resistant and those with rating of 3 to 5 were considered susceptible. In susceptible plants, the type of symptom observed (i.e., necrotic or chlorotic) was noted.

morgans (cM) distal to the SSR locus *Xgwm285* (Fig. 1). The proposed designation for this locus is *tsn2*, following the guidelines for loci designation by McIntosh et al. (32).

To confirm the previous findings, a genetic segregation analysis was done using disease data from previous generations of the TT283/Coulter population. The F₂ generation segregated in a 1:3 resistant/susceptible ratio, suggesting that a single recessive gene controls resistance to necrosis induced by race 3 (Table 2). The F_{4:5} family segregation data fit a ratio of 7:2:7 homozygous resistant/segregating/homozygous susceptible, supporting the hypothesis of monogenic control of resistance (Table 2). This hypothesis was further confirmed by the F_{6:7} family segregation, which fit a 31:2:31 homozygous resistant/segregating/homozygous susceptible ratio in the cross studied (Table 2).

DISCUSSION

Tan spot is one of the most destructive leaf spot diseases of wheat in the northern Great Plains of the United States. Therefore, both private and public wheat-breeding programs have given

Fig. 2. Histogram of necrosis score means of 80 recombinant inbred lines (RILs) derived from the cross *Triticum turgidum* no. 283/Coulter after inoculation with *Pyrenophora tritici-repentis*, race 3.

Fig. 1. **A**, Linkage map of chromosome 3B for a population of 80 recombinant inbred lines derived from the cross *Triticum turgidum* no. 283/Coulter. Molecular marker loci (on the right) were distributed into three linkage groups. Genetic distances (on the left) are expressed in centimorgans. **B**, Quantitative trait loci (QTL) analysis for resistance to *Pyrenophora tritici-repentis* race 3. The graph shows the likelihood ratio statistics (LRS) of the significance of possible QTL. Dotted line represents the threshold value for a *P* value of 0.001.

TABLE 2. Disease reaction of F₂ generation and F_{4:5} and F_{6:7} families of cross *Triticum turgidum* no. 283/Coulter studied to determine the genetics of resistance to necrosis induced by *Pyrenophora tritici-repentis* isolate Ptr 331-9 (race 3)

Generation or families	Total	Resistant	Segregating	Susceptible	Ratio tested	χ^2 value	P value ^a
F ₂	230	63	...	167	1:3	0.70	0.40
F _{4:5}	80	31	9	40	7:2:7	1.27	0.53
F _{6:7}	80	29	2	49	31:2:31	5.26	0.07

^a Probability of obtaining deviations from the expected ratio by chance alone. A probability value of >0.05 indicates that segregation in the observed population does not differ significantly from the expected ratio.

major emphasis to breeding for resistance to tan spot. Genetic studies and chromosomal location of resistance genes provide vital information in the development of resistant cultivars. Various genetic studies have reported resistance to tan spot to be quantitatively (13,16,35) or qualitatively (10,11,22,29,30,40) inherited. However, comparison of these studies cannot be made due to the lack of standardization of factors such as method of inoculation, rating scales, symptoms studied, isolates used, and the environmental conditions for disease development.

The development of the necrosis-chlorosis model and the identification of races inducing either necrosis or chlorosis (28) have been of great value to studies of genetic control of tan spot. Differentiation between the necrotic and chlorotic components of tan spot has led to reliable and accurate assessment of resistance and susceptibility. Most genetic studies conducted since the advent of the necrosis-chlorosis model have reported inheritance of resistance to be controlled by major genes (10,11,22,29,30,40).

In recent years, resistance to tan spot also has been reported to be quantitative, but a major portion of this resistance is controlled by a single major QTL (16,20). The findings of this study, that a single gene controls resistance to necrosis induced by race 3 in tetraploid wheat, is in agreement with the findings of Gamba and Lamari (21). The QTL analysis in this study supports the previous findings that the majority of resistance is controlled by a single QTL. Resistance to tan spot of wheat is controlled by multiple major genes (21,30). Tan spot in the field appears quantitative in nature due to multigenic control of resistance, environmental factors, and the mix of races observed in field conditions; however, resistance to individual races in controlled environments generally is qualitative. The findings of this study indicate that resistance to tan spot is qualitative for tetraploid wheat, similar to the observation in hexaploid wheat (3,10,11,40). However, this does not preclude the existence of other genes with minor effects, as suggested by Faris et al. (16).

Lamari et al. (31) suggested that the wheat-*P. tritici-repentis* pathosystem conforms to the toxin model originally proposed by Ellingboe (14). In this model, a compatible interaction between host plant and pathogen leads to susceptibility; the toxin model is a mirror image of the classical gene-for-gene model (19). In the toxin model, susceptibility results when a "toxin-receptor" site is present in the host. If the presence of such a site is considered the product of a susceptibility gene, then resistance in the wheat-*P. tritici-repentis* pathosystem is due to the lack of a susceptibility gene rather than the presence of a resistance gene (3,40). Langdon is a cultivar susceptible to *P. tritici-repentis* race 3. In the LDN(DIC) substitution lines, a pair of chromosome are replaced with a chromosome derived from the donor parent *T. dicoccoides* accession Israel-A, which is resistant to race 3. Any LDN(DIC) substitution lines giving resistance is due to the absence of susceptibility genes of Langdon (i.e., indicating that the locus for resistance or susceptibility is located on that chromosome). This study, for the first time, identifies a recessive gene for resistance to necrosis induced by *P. tritici-repentis* race 3 located on chromosome 3B and further supports the prior findings that LDN(DIC) substitution lines can be utilized for identifying the chromosomal location genes controlling qualitative traits (4,5,23,25,42,44).

This gene, *tsn2*, mapping close to *Xgwm285*, may be in the vicinity of the race-nonspecific resistance locus mapped by Faris

and Friesen (17) in hexaploid wheat; however, a precise comparison is not possible due to lack of common markers between the two maps. The race-nonspecific QTL identified on chromosome 3B by Faris and Friesen (17) explained 13 to 41% of the phenotypic variation caused by spore inoculation of races 1, 2, 3, and 5 in a hexaploid wheat population, whereas *tsn2* accounts for 91% of the phenotypic variation caused by race 3 in durum wheat populations.

Durum wheat is highly susceptible to tan spot (18). Most durum breeding program, when screening for tan spot resistance, screen with race 1 isolates. However, in the case of durum wheat, resistance to necrosis induced by races 1, 3, and 5 is controlled by three independently inherited recessive genes (21). Hence, when screening with race 1, we fail to select for resistance to races 3 and 5. In order to develop resistant durum wheat, multiple screening with individual races needs to be performed. The SSR locus *Xgwm285*, located at only 2.1cM from *tsn2*, should be of great use for marker-assisted selection to introduce resistance from *T. turgidum* no. 283 into an adapted durum wheat background. Tan spot resistance is controlled by multiple major genes; hence, marker-assisted selection also can be utilized to pyramid resistance genes. It is more effective for durum wheat because two recessive resistance genes have been closely mapped. These recessive genes control resistance to necrosis caused by races 1 and 2 (*tsn1* on chromosome 5BL), and race 3 (*tsn2* mapped on chromosome 3BL in this study). Presently, efforts are going on to map additional genes controlling resistance to necrosis induced by race 5 in tetraploid wheat. This will broaden the scope of marker-assisted selection in durum wheat breeding and hasten the development of tan spot-resistant cultivars.

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