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RFLP mapping of resistance to chlorosis induction by *Pyrenophora tritici-repentis* in wheat

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Abstract Tan spot, caused by *Pyrenophora tritici-repentis*, is an economically important disease in major wheat production areas. The fungus can produce two genetically distinct symptoms on leaves of susceptible wheat genotypes: tan necrosis (nec) and extensive chlorosis (chl). Our objectives were to determine the number of genes conditioning resistance to tan spot in a population of wheat recombinant inbred lines, and map the chromosomal location of the resistance genes using RFLPs. Conidia produced by the *P. tritici-repentis* isolate Pti2 (nec + chl +) were used to inoculate seedlings of 135 recombinant inbred lines derived from the cross of the synthetic hexaploid wheat W-7984 with Opata 85. A subset of the population was inoculated with conidia produced by the isolates D308 (nec − chl +) and 86-124 (nec + chl −). Inoculated seedlings were rated on a scale of 1 to 5 based on lesion type. Necrosis-inducing culture filtrate produced by the isolate 86-124 was also used to screen the entire population. A map consisting of 532 markers was employed to identify significant associations between marker loci and tan spot resistance. The entire population was insensitive to culture filtrate produced by the isolate 86-124, and the entire subset was resistant to conidial inoculation of the same isolate. The population segregated for reaction to isolates D308 and Pti2, indicating that this population segregates for resistance to extensive chlorosis only, and not to tan necrosis. RFLP analysis indicated the presence of a gene with a major effect in 1AS, a gene with a minor effect in 4AL, and an interaction between the 1AS gene and a gene in 2DL. Together, these loci explained 49.0% of the variation in this population for resistance to tan spot produced by the isolate Pti2. Two regions one in 1BL and one in 3BL, were significantly associated with resistance to extensive chlorosis, but were not significant in the multiple regression model. It should be feasible to introgress these resistance loci into adapted genetic backgrounds by using a marker-assisted selection scheme.

Key words *Triticum aestivum* · *Pyrenophora tritici-repentis* · Quantitative trait loci · Molecular mapping · Disease resistance

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Introduction

Tan spot, caused by *Pyrenophora tritici-repentis* (Died). Drechs., occurs worldwide and is an economically important disease in major wheat (*Triticum aestivum* L. emend. Thell.) production areas including Australia and the Great Plains region of the United States and Canada (Luz and Hosford 1980; Wiese 1987). In the past decade, there has been an increase in the inoculum level of the pathogen due to the trend toward conservation tillage, which has allowed the fungus to overwinter in stubble residue (Rees 1982). The disease has caused yield losses of 3–50% (Hosford 1982; Rees and Platz 1983; Mehta and Gaudencio 1991).

Isolates of *P. tritici-repentis* differ in virulence (Luz and Hosford 1980). Susceptibility of wheat to the fungus is manifested by the development of tan necrosis and/or extensive chlorosis. Isolates of the fungus are grouped into five races based on their virulence pattern and their ability to induce tan necrosis and extensive chlorosis (nec + chl +, race 1) tan necrosis only (nec + chl −, race 2), extensive chlorosis only (nec − chl +, races 3 and 5), or avirulence (nec − chl −, race 4) (Lamari and Bernier 1989 a; Lamari et al. 1992; Lamari et al. 1996).

Necrosis-inducing isolates of *P. tritici-repentis* release a host-selective toxin in culture (Tomás and Bockus 1987; Lamari and Bernier 1989 a; Tuori et al. 1995). Sensitivity to this toxin was found to be highly associated with the induction of tan necrosis in the host, and insensitivity to the toxin and resistance to tan necrosis caused by the fungus are controlled by a common gene, or two or more closely linked genes (Tomás and Bockus 1987; Lamari and Bernier 1989 a).

Few commercially grown cultivars possess resistance to tan spot (Hosford et al. 1990; Lamari et al. 1992; Mehta et al. 1992), and no complete resistance was identified among 1400 bread wheats of diverse origin (Rees and Platz 1990). A greater amount of resistance was found by Alam and Gustafson (1988) when they screened 212 accessions of eight diploid and ten polyploid species of *Aegilops*. Riede et al. (1996) screened 78 genotypes of diverse origin as seedlings and adults; 21 of these were resistant at both stages, while seven were resistant at the seedling stage but susceptible at the adult stage.

Reports on the mode of inheritance of resistance to tan spot in *T. aestivum* and *T. turgidum* L. have ranged from qualitative (1 or 2 genes) (Lee and Gough 1984; Lamari and Bernier 1991; Sykes and Bernier 1991) to quantitative (Nagle et al. 1982; Elias et al. 1989). Lamari et al. (1991) suggested that the symptoms of tan necrosis to nec+ isolates and extensive chlorosis to chl+ isolates are genetically distinct. Lamari and Bernier (1991) studied F_2 and F_3 populations and found that segregation ratios were consistent with the action of two independent genes; one, a single recessive gene controlling the development of tan necrosis, and a second, a single dominant gene controlling the development of extensive chlorosis with modifiers acting in some populations. Faris et al. (1996) determined that a single recessive nuclear gene residing in 5BL conditions insensitivity to a necrosis-inducing culture filtrate produced by the isolate 86-124 (nec+ chl−). Rees and Platz (unpublished) evaluated the 'Chinese Spring' disomic substitution series' of 'Kenya Farmer', 'Timstein', and 'Thatcher' as juvenile plants. In each series, chromosomes 1A and 5B appeared to contribute most of the resistance to conidial inoculations of *P. tritici-repentis*.

The recent development of a complete RFLP map in wheat (Van Deynze et al. 1995; Nelson et al. 1995 a, b, c; Marino et al. 1996) allows for the detection of quantitative trait loci (QTLs) in wheat. Our objectives were to determine the number of genes conditioning resistance to tan spot in a population of wheat recombinant inbred lines, and to map the chromosomal location of these resistance genes using RFLPs.

Materials and methods

Plant materials

The population was provided by M. E. Sorrells, Cornell University, Ithaca, N.Y., and consisted of 135 recombinant inbred (RI) lines derived from the cross of W-7984 [a resistant synthetic hexaploid wheat derived from the cross of 'Altar 84' durum and *T. tauschii* (Coss.) Schmal.] and 'Opata 85' a susceptible CIMMYT-bred hard red spring wheat (PI591776). W-7984 was developed by A. Mujeeb-Kazi, CIMMYT. The susceptible line ND495, provided by R. Frohberg, NDSU, Fargo, N.D., and the resistant cultivar 'Erik' (PI476849) were used as checks. Alter 84, the durum parent of W-7984, was also included in inoculation experiments.

Inoculum preparation

The *P. tritici-repentis* isolates used in this study were Pti2 (ATCC 44143, nec+ chl+), D308 (nec− chl+) and 86-124 (nec+ chl−) (Luz and Hosford 1980, Lamari and Bernier 1989 a). Isolates were stored as dried mycelial plugs at −80°C. These plugs were used to produce additional plugs that were stored at 4°C for the duration of the experiment. Inoculum was produced by placing two plugs 3 cm apart on Petri plates containing V8-potato dextrose agar (PDA) medium (150 ml V8 juice, 10 g PDA, 3 g $CaCO_3$, 10 g agar, and 850 ml distilled water). Plates were incubated for 5 days in a temperature-controlled chamber at 20°C under continuous darkness. Plates were then flooded with sterile distilled water and the hyphae flattened with a flame-sterilized glass slide. The water was then decanted and the plates incubated under fluorescent light for 24 h at 20°C followed by a 24-h dark period at 16°C. Conidia were harvested by flooding the plates with distilled water and dislodging the spores by gently scraping the colony with a flame-sterilized glass slide. Spore suspensions were blended for 3 min to separate aggregates, stirred, and the number of conidia in six 5-μL samples counted under a compound microscope to estimate spore concentration. Two drops (0.04 ml) of Tween 20 (polyoxyethylene sorbitan monolaurate) were added per 100 ml of spore suspension.

Conidial inoculation and rating

Each RI line of the entire W-7984/Opata 85 population, parents, and checks were grown in plastic cones containing Fison sunshine blend #1 (Fison Horticulture, Vancouver, B. C.) in the greenhouse at an average temperature of 21°C with 16-h photoperiod and light intensity of 400 μmol $m^{-2}s^{-1}$ at plant height. Four seeds of each entry were planted at each of four planting dates that were staggered 3 days apart. Three weeks after the last planting date, three plants from each entry were selected based on leaf stage. This was done to minimize age effects of inoculated leaves because preliminary experiments suggested that younger leaves are more resistant than older leaves. Two experiments with three replications each were inoculated in a completely randomized design. A subset (six resistant, six moderately resistant to moderately susceptible, and six susceptible) of the population was selected based on data collected from Pti2 inoculations. This subset, parents, and checks were inoculated with isolates D308 and 86-124 in two experiments using a completely randomized design with three replications.

Selected seedlings with a fully emerged third leaf were inoculated as described in Riede et al. (1996) with a concentration of 3000 conidia per ml. After inoculation, plants were allowed to dry for 15 min, and placed in a mist chamber at 20°C with a 16-h photoperiod. The chamber was misted for 1 out of 5 min using an ultrasonic humidifier. After a 24-h wet period, plants were placed in a growth chamber at 20°C and subirrigated to avoid further leaf wetting.

The percent leaf area diseased and the lesion-type mean of the third leaf were recorded 7 days after inoculation. Lesion types were rated on a scale from 1 to 5 (1 = most resistant, 5 = most susceptible) based on the system developed by Lamari and Bernier (1989 b). Lesion type and the percent leaf area diseased were highly correlated ($r = 0.88$, $P < 0.01$), but the analysis of percent leaf area diseased had greater experimental error. Therefore, only results from the lesion-type rating system will be presented. Analysis of variance was conducted to identify significant variation among RI lines and variances were tested for homogeneity between runs of the same isolate. RI lines that showed highly resistant reactions and highly susceptible reactions within the same inoculation experiment were eliminated based on the fact that some heterogeneity may persist in the population.

Culture filtrate infiltration

The entire population was infiltrated with *P. tritici-repentis* culture filtrate produced by the isolate 86-124. This isolate was previously characterized as a producer of the necrosis toxin (nec+), and a non-inducer of extensive chlorosis (Lamari and Bernier 1989a). Plants were grown in the greenhouse as previously described. Two experiments, composed of two replications each, were evaluated in a completely randomized design. Production of culture filtrate and infiltration of plants was done as described in Faris et al. (1996). Plants were scored as sensitive or insensitive to the culture filtrate.

RFLP analysis

The number of putative QTLs associated with resistance to tan spot was estimated based on the reaction type observed on leaves inoculated with conidia produced by the isolate Pti2. Genetic maps and the DNA clones used to construct them were described in Nelson et al. (1995a, b, c), Van Deynze et al. (1995) and Marino et al. (1996). Fifty six RI lines were used to map fragments detected by clones with the prefix FBA and FBB, with the exception of FBA280 and FBA211. Fragments detected by all other clones, including FBA280 and FBA211, were mapped using 114 RI lines. The map used in this study consisted of *Glu1*, *W2'* (see Nelson et al. 1995b, and Van Deynze et al. 1995, respectively, for mapping details), and 530 RFLP markers selected to give the most complete genome coverage possible, without redundancy. A list of the markers used is available upon request. Because some of the published maps were constructed at a log likelihood ratio (LOD) of 3.0, many markers were assigned to intervals. Therefore, chromosome maps were reconstructed from raw data using MAPMAKER V2.0 (Lander et al. 1987) at a LOD of 2.0 to assign more markers to specific sites. Centimorgan distances were obtained using the Kosambi mapping function (Kosambi 1944). All markers were subjected to regression analysis using the computer program qGene V2.18 (J. C. Nelson, unpublished) to identify putative QTLs associated with tan spot resistance. Individual RFLP markers linked to putative resistance QTLs were subjected to one-way analysis of variance using the computer program DataDesk v4.1 (Data Description, Inc., Ithaca, N.Y.). Markers having significant ($P < 0.01$) main effects were tested against all other markers on the map to detect significant ($P < 0.01$) interactions. Backward elimination, forward selection, and maximum R^2 improvement stepwise regression procedures were performed on markers and interactions significant at $P < 0.01$ to identify the best multiple regression model and to eliminate markers that failed to retain significance in the model (SAS 1988). The coefficient of determination (R^2) from the multiple regression model is the proportion of the total phenotypic variance explained by the markers.

Results

Tan spot screening

The reaction of parental and check cultivars to conidial inoculations of each isolate is presented in Table 1. The RI population segregated for reaction to Pti2 (nec+ chl+) conidial inoculations, but little necrosis was observed on infected leaves at the time of rating except for the susceptible check. Susceptible genotypes had extensive chlorosis throughout the entire leaf, and resistant genotypes mimicked W-7984 and developed small-dark flecks at infection sites without surrounding chlorosis. Variances of the two runs were homogeneous indicating that means could be pooled, and the analysis of variance indicated highly significant differences among RI lines. The coefficient of variation was 18.8%. Most genotypes showed either resistant (1 or 2) or susceptible (4 or 5) lesion types, producing a histogram resembling a bimodal distribution (Fig. 1). A portion of the population was classified as moderately susceptible (lesion types 3 and 4). Nine RI lines exhibited lesion types ranging from highly resistant to highly susceptible and were not included in the analysis. These lines may be heterogeneous at a major resistance locus.

The subset of the population screened with the isolate D308 (nec− chl+) also segregated for reaction to conidial inoculations (Table 1). Extensive chlorosis was observed on susceptible genotypes and resistant genotypes had a small tan fleck with little or no surrounding chlorosis. Chlorosis was less extensive on moderately resistant and moderately susceptible genotypes. These data correlated with the Pti2 inoculations with a value of $r = 0.81$ ($P < 0.01$) indicating the functioning of common resistance genes or closely linked genes.

The entire subpopulation, both parents, and Altar 84 were resistant to conidial inoculations of the isolate 86-124 (nec+ chl−) while the susceptible check showed

Table 1 Mean lesion type of parents and checks after inoculation of seedlings with conidia from three isolates of *P. tritici-repentis*

Genotype	Isolate[a]		
	Pti2	D308	86-124
W-7984 (Resistant parent)	1.2a[b]	1.3a	1.0a
Opata 85 (Susceptible parent)	4.2c	4.5c	1.2a
Altar 84 (Durum parent of W-7984)	2.3b	2.5b	1.0a
Erik (Resistant check)	1.5a	1.3a	1.3a
ND495 (Susceptible check)	4.8d	5.0c	4.5b

[a] Pti2 (nec+ chl+), D308 (nec− chl+), 86-124 (nec+ chl−)
[b] Lesion-type means within the same column followed by the same letter are not significantly different at $P = 0.05$. Range from 1 = most resistant to 5 = most susceptible

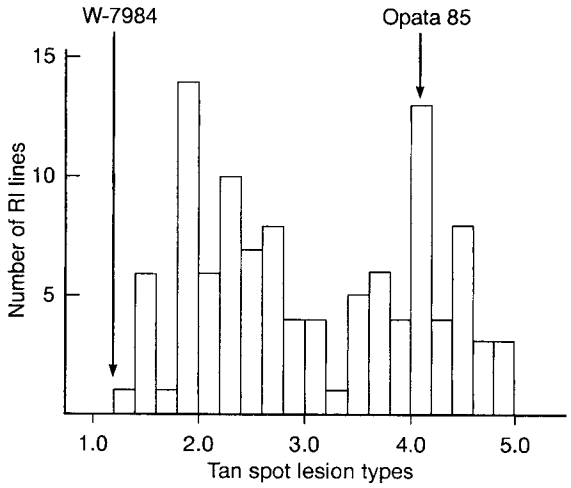

Fig. 1 Histogram of lesion-type means of the 135 recombinant inbred lines from the cross of W-7984/Opata 85 after conidial inoculations with *P. tritici-repentis* isolate Pti2. LSD (0.05) = 1.14

lesions of types 4 and 5 (Table 1). The entire population, including both parents and Altar 84, was insensitive to culture filtrate produced by the isolate 86-124, and the susceptible check ND495 developed necrosis 24 h after inoculation (data not shown). These results suggest that this population is segregating for resistance to extensive chlorosis, but not for resistance to tan necrosis.

RFLP analysis

Lesion-type means were regressed on the RFLP marker data to identify loci associated with tan spot resistance. Five regions were significant at the 0.01 level of probability (Table 2). The chromosomal locations of all significant loci are shown in Fig. 2. The marker *Xbcd120* detected a fragment in the long arm of chromosome 2D and, while the main effects of this locus were non-significant, it had a significant interaction with *XGli1* (Table 3). This interaction explained 11.5% of the variation.

Three of the five significant loci were eliminated from the multiple regression model after stepwise regression.

Table 2 Coefficients of determination and *P* values of five unlinked markers significant at the $P < 0.01$ level of probability following inoculation of the W-7984/Opata 85 recombinant inbred population with conidia produced by the isolate Pti2. *Xbcd120* and the *XGli1* · *Xbcd120* interactions are included in the multiple regression model

RFLP Marker	Chrom.	Source of Res.	R^2	*P*
XGli1	1AS	W-7984	0.350	< 0.0001
Xfba211	4AL	W-7984	0.107	0.0006
Xfbb1[a]	4AS	W-7984	0.137	0.0098
Xpsr903	3BL	W-7984	0.086	0.0086
Xcdo346	1BL	Opata 85	0.076	0.0048
Xbcd120	2DL		0.000	0.9185
XGli1 · *Xbcd120*	1AS/2DL		0.115	0.0004

[a] Probed on only 56 recombinant inbred lines

The main effects of *XGli1* and *Xfba211* in addition to the interaction between *XGli1* and *Xbcd120* explained 49.0% of the variation for resistance to *P. tritici-repentis* isolate Pti2 (Table 4).

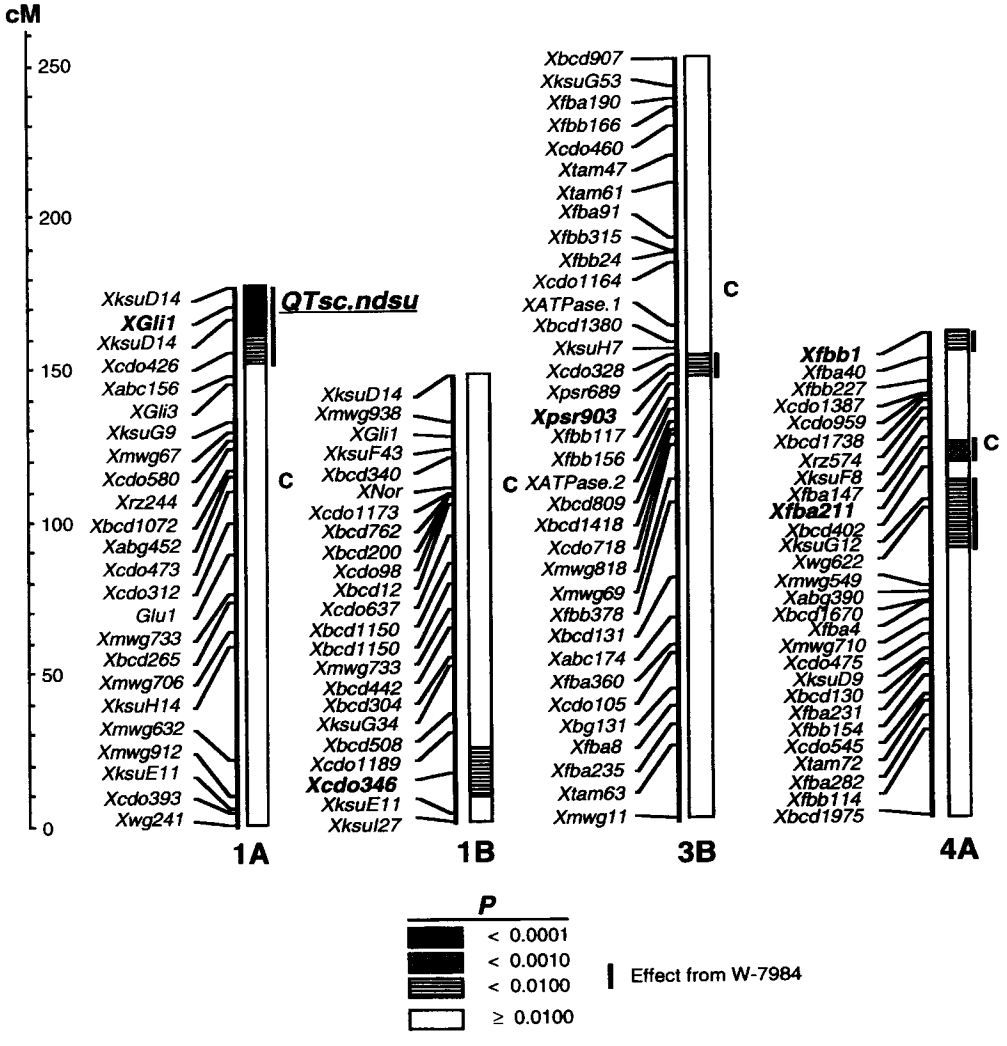

Fig. 2 Genetic linkage maps of the four chromosomes possessing putative quantitative trait loci associated with resistance to tan spot caused by the *P. tritici-repentis* isolate Pti2. Markers closely linked to putative quantitative trait loci are in *bold face*. Centromere regions are indicated with a *C*

Table 3 Number of observations and lesion-type means of the four possible classifications of recombinant inbreds for the allelic state at *XGli1* and *Xbcd120* after inoculation of the population derived from the cross W-7984/Opata 85 with conidia produced by the *P. tritici-repentis* isolate Pti2

XGli1	*Xbcd120*	No. observations	Lesion-type means[a]
Opata 85	W-7984	22	4.1d
W-7984	W-7984	24	2.4a
Opata 85	Opata 85	22	3.5c
W-7984	Opata 85	28	2.8b

[a] Numbers followed by the same letter are not significantly different at $P = 0.05$. Lower means indicate greater resistance

Table 4 Multiple regression model of quantitative trait loci controlling reaction to the *P. tritici-repentis* isolate Pti2 in seedlings of recombinant inbred lines derived from the cross W-7984/Opata 85. $R^2 = 0.49$

Source of variation	*df*	Mean square	*F* value	*P* value
XGli1	1	29.34	50.28	< 0.0001
Xbcd120	1	0.10	0.17	0.6825
Xfba211	1	4.75	8.14	0.0054
XGli1 · Xbcd120	1	6.05	10.37	0.0018
Error	91	0.58		
Total	95			

Discussion

We identified a QTL with a major effect in 1AS that is associated with resistance to tan spot caused by the *P. tritici-repentis* isolate Pti2. A QTL with a minor effect in the long arm of chromosome 4A, and an interaction between *XGli1* in 1AS and *Xbcd120* in 2DL, also contribute to the variation observed. This population segregated for resistance to chlorosis induced by *P. tritici-repentis*, but was entirely insensitive to the necrosis-inducing culture filtrate produced by the isolate 86-124. Lamari et al. (1991) also demonstrated that extensive chlorosis and tan necrosis caused by *P. tritici-repentis* are two genetically distinct symptoms. Our research suggests that resistance to the chlorosis reaction in this population is controlled quantitatively, but that a large portion of the variation is controlled by a QTL in 1AS. This also agrees with the conclusions of Lamari and Bernier (1991) that resistance to extensive chlorosis caused by *P. tritici-repentis* is controlled primarily by a single gene in the host with minor gene action occurring in some populations. Following McIntosh et al. (1994), we propose the symbol *QTsc.ndsu-1A* to designate this gene.

Altar 84 durum was moderately resistant to conidia from *P. tritici-repentis* isolates Pti2 (nec+ chl+) and D308 (nec− chl+) and was highly resistant to both conidia and culture filtrate produced by the isolate 86-124 (nec+ chl−). Four of the five resistance loci detected in this population were presumably derived from the durum parent (Altar 84) of the synthetic hexaploid. Because durum lacks the D genome, the interaction between the loci in 1AS and 2DL is not present in Alter 84. The absence of this, and possibly other unidentified factors from the D-genome chromosomes, may explain why Altar 84 exhibited only moderately resistant lesion types when inoculated with chlorosis-inducing isolates.

Other QTLs may be involved in controlling resistance to tan spot but were undetected. Host-parasite interactions in this system are highly affected by environmental conditions. We attempted to reduce these environmental effects as much as possible by conducting all studies under greenhouse conditions. Identifying more distal RFLP fragments and mapping the telomeres of chromosomes would provide a greater degree of confidence that all QTLs have been detected. Due to the lower frequency of polymorphism, the D-genome maps had an average of only 20.0 markers per chromosome compared to 29.3 and 28.0 markers for A- and B-genome chromosomes, respectively. This resulted in 11 gaps of at least 20 cM including two gaps greater than 30 cM between markers on D-genome chromosomes. Therefore, minor QTLs may have gone undetected if QTL-marker linkages were not tight enough to identify significant associations.

The gliadin locus detected by *XGli1* resides in a gene-rich region of group-1 chromosomes. Other genes mapping near *XGli1* include: *Pm3*, resistance to powdery mildew (*Blumeria graminis* f.sp. *tritici*; Ma et al. 1994) in 1AS; *Lr21*, resistance to leaf rust (*Puccinia recondita* f.sp. *tritici*), *Rg2*, red glume color (Jones et al. 1990), and *Sr33*, resistance to stem rust (*P. graminis* f.sp.*graminis*; Jones et al. 1991), all in 1DS. The *XGli1* locus may be a useful marker for the resistance genes.

Preliminary analysis of chromosome 4A indicated that one marker lies between two significant regions of the chromosome. This marker was significant at the 0.05 level of probability. Therefore, these closely linked regions may represent one QTL.

The ability to produce extensive chlorosis in susceptible wheat genotypes can be a major destructive property of many tan spot isolates (Lamari and Bernier 1989 b). However, selection for resistance to extensive chlorosis alone will not lead to cultivars possessing complete resistance to tan spot. Tan necrosis is also a significant virulence component of many tan spot isolates and should be selected for as well. The present study employed three isolates of *P. tritici-repentis* and was conducted on juvenile plants. Further work is needed to determine if the QTLs identified in this study are also involved in conditioning resistance in adult plants and to other isolates. Evaluations of other populations are required to confirm the minor QTL and the epistatic interaction reported here due to the Type-I error rate tolerated in this experiment.

Knowledge of the location of QTLs associated with tan spot resistance and the identification of markers linked to them could facilitate a marker-assisted selec-

tion scheme and aid breeders in making selections for resistant genotypes. The QTL identified in chromosome 4A resides near the centromere of the chromosome where recombination frequencies are low. This may make it difficult to obtain the desired allele in progeny without the occurrence of linkage drag. Therefore, the employment of markers in a selection scheme would be especially beneficial in a backcross breeding program to transfer the desired resistance allele to a more genetically adapted background.

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