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Chromosomal location of a race-specific resistance gene to *Mycosphaerella graminicola* in the spring wheat ST6

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Abstract Septoria tritici blotch, caused by *Mycosphaerella graminicola*, is a serious foliar disease of wheat worldwide. Qualitative, race-specific resistance sources have been identified and utilized for resistant cultivar development. However, septoria tritici blotch resistant varieties have succumbed to changes in virulence of *M. graminicola* on at least three continents. The use of resistance gene pyramids may slow or prevent the breakdown of resistance. A clear understanding of the genetics of resistance and the identification of linked PCR-based markers will facilitate the recovery of wheat lines carrying multiple septoria tritici blotch resistance genes. The resistance gene in ST6 to isolate MG2 of *M. graminicola* was mapped with microsatellite markers in two populations, ST6/Erik and ST6/Katepwa. Bulk segregant analysis identified a marker on chromosome 4AL putatively linked to the resistance gene. A large linkage group was identified in each population using additional microsatellite markers mapping to chromosome 4AL. The resistance gene in ST6 mapped to the distal end of chromosome 4AL in each mapping population and was designated *Stb7*. Three of the microsatellite loci, *Xwmc313*, *Xwmc219* and *Xgwm160*, mapped within 3.5 cM of *Stb7*; however, none flanked *Stb7*. *Xwmc313* was the closest and mapped 0.3 and 0.5 cM from *Stb7* in the crosses ST6/Katepwa and ST6/Erik, respectively. WMC313 will be very useful for marker-assisted selection of *Stb7* in Canadian breeding programs because the ST6 allele of *Xwmc313* was not identified in any of the Canadian common wheat cultivars tested.

Keywords Disease resistance gene · Linkage analysis · Microsatellite marker · *Triticum aestivum* · *Mycosphaerella graminicola*

Introduction

Septoria tritici blotch, caused by the ascomycete fungus *Mycosphaerella graminicola* (Fuckel) J. Schröt. in Cohn (anamorph: *Septoria tritici* Roberge in Desmaz.), is a major foliar disease of wheat (*Triticum aestivum*) in Canada and other wheat-producing areas of the world (Eyal et al. 1987; Gilbert et al. 1998). The temperature range and the leaf wetness period are important environmental factors affecting the development of septoria tritici blotch (Magboul et al. 1992). Severe epidemics of septoria tritici blotch result in considerable yield losses and shriveled seed unfit for milling (Eyal 1981). The development of resistant cultivars will provide an economically and environmentally sound method of control of this disease.

Septoria tritici blotch resistance has been reported as either a qualitative (Wilson 1979; Somasco et al. 1996; McCartney et al. 2002) or quantitative trait (Jlibene et al. 1994; Simon and Cordo 1998). A gene-for-gene interaction was recently reported for the septoria tritici blotch resistance gene *Stb6* (Brading et al. 2002). It is unknown whether other *Stb* genes (*Stb1*, *Stb2*, *Stb3*, *Stb4* and *Stb5*) are involved in gene-for-gene interactions. The breakdown of septoria tritici blotch resistance in Australia, Israel and the USA (Eyal et al. 1973; Ballantyne and Thomson 1995; Cowger et al. 2000) is also consistent with a gene-for-gene interaction and underlines the need for an improved understanding of this pathosystem.

Pyramiding resistance genes involved in gene-for-gene interactions may provide durable septoria tritici blotch resistance. Resistance gene pyramids can be developed by screening breeding populations for resistance to isolates with specific virulence patterns and/or with DNA-based markers linked to resistance genes. Markers are particularly useful for gene pyramiding when pathogen races

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carrying single avirulence genes are not available. In the absence of such races, the number of resistance genes in a plant may not be phenotypically distinguishable. Markers are also useful to maintain genetic advance in breeding populations when disease epidemics cannot be established. Currently, only two septoria tritici blotch resistance genes have been mapped to chromosomal locations in wheat. *Stb5*, derived from *Triticum tauschii*, maps near the centromere on chromosome 7DS (Arraiano et al. 2001; Simon et al. 2001). *Xgwm44* and the anthocyanin pigment gene *Rc3* flank *Stb5* and mapped 7.2 and 6.6 cM from *Stb5*, respectively. *Stb6* mapped to the distal portion of chromosome 3AL with *Xgwm369* mapping 2 cM from *Stb6* (Brading et al. 2002). The markers identified in these studies will be useful for marker-assisted selection (MAS). The resistance genes, *Stb1*, *Stb2*, *Stb3* and *Stb4*, have not been assigned to chromosomes.

The objective of this study was to determine the chromosomal location of the septoria tritici blotch resistance gene in ST6 and to identify microsatellite markers useful for marker-assisted selection of septoria tritici blotch resistance.

Materials and methods

Plant material

A race-specific resistant *T. aestivum* line ST6 (a selection of cv Estanzuela Federal) was crossed to two susceptible *T. aestivum* lines, Katepwa and Erik. One hundred F_{2:3} families of ST6/Katepwa, 100 F_{2:3} families of Katepwa/ST6, 55 F_{2:3} families of ST6/Erik and 55 F_{2:3} families of Erik/ST6 were generated previously (McCartney et al. 2002). A set of 36 Canadian wheat cultivars representative of different wheat classes was used to evaluate the utility of microsatellite markers for wheat breeding in Canada.

Disease evaluation

Wheat seedlings of the F_{2:3} families were evaluated for disease reaction previously (McCartney et al. 2002). Briefly, 20 F₃ families of 20 individuals, ten individuals of each control wheat line (Salamouni, ST6, Katepwa, Erik, Coulter and 4B1149), and four to ten F₁ individuals of the cross being evaluated, and its reciprocal, were included in each inoculation experiment. All seeds were germinated in the dark prior to planting (2 days at 4 °C, 2 days at room temperature) to ensure uniform emergence. Seedlings were grown in the greenhouse prior to inoculation, where temperature ranged from 18 °C at night to 25 °C during the day and supplemental lighting provided a 16-h photoperiod (260 μE m⁻² s⁻¹). Seedlings were evaluated for disease reaction with a single-spore culture of *M. graminicola* isolate MG2, which was isolated from a Manitoba wheat field (Grieger 2001). The MG2 culture was grown on yeast malt agar (Eyal et al. 1987) under fluorescent lights for 7 days. A 10⁷ conidia per ml suspension was sprayed on seedlings at the three-leaf stage. Plants were incubated in a humidity chamber that maintained continuous leaf wetness for 67 to 72 h. Wheat seedlings were moved to a growth cabinet set at 21/19 °C (day/night) with a 16-h photoperiod (390 μE m⁻² s⁻¹) and relative humidity between 70 and 80%. Disease ratings were made 17 days after inoculation. A 0 to 5 scale was used to evaluate seedling reactions (McCartney et al. 2002). This rating scale was modified slightly from the scale developed by Rosielle (1972). Reaction types 0 to 3 were resistant and 4 and 5 were susceptible. F₃ families were classified as

homozygous resistant, segregating, or homozygous susceptible. Data was tested for goodness of fit to specific genetic ratios using chi-square analysis (Strickberger 1985). Data from reciprocal crosses were tested for homogeneity prior to pooling data (Strickberger 1985).

DNA extraction

Leaf tissue was collected and lyophilized from the youngest (non-inoculated) leaf tissue of F₃ and parental plants 22 days after inoculation. F₂ individuals were reconstituted by pooling equal amounts of leaf tissue from 20 individuals of a particular F₃ family for DNA extraction. Leaf tissue was placed in a 15-ml falcon tube (Fisher Scientific, Pittsburgh, Pa.) with six 3-mm tungsten carbide beads (Qiagen, Mississauga, Ont.) and ground into a powder by shaking in a paint shaker for 15 min. DNA was extracted with a DNeasy 96 Plant Kit (Qiagen, Mississauga, Ont.). DNA was quantified by fluorimetry using Hoechst 33258 stain.

Bulked segregant analysis

A total of 279 microsatellite-marker primer pairs were screened using the bulked segregant analysis (BSA) strategy on the cross ST6/Erik (Michelmore et al. 1991). The resistant and susceptible bulks were created by combining equal amounts of DNA from ten homozygous resistant F₃ families and ten homozygous susceptible F₃ families, respectively. Each of the microsatellite markers was screened on the parental lines and the two bulks to identify putatively linked markers.

PCR amplification

PCR reactions were performed in 10-μl volumes and included 36 ng of template DNA, 1 U of *Taq* DNA polymerase (Gibco/BRL, Mississauga, Ont.), 1× PCR buffer (Applied Biosystems, Foster City, Calif.), 1.5 mM of MgCl₂, 200 μM of each dNTP, 20 μM of forward primer, 180 μM of 6-FAM/HEX/NED-labelled M13 primer (5'→3' CACGACGTTGTAAAACGAC; Applied Biosystems, Foster City, Calif.), and 200 μM of reverse primer. Table 1 shows the forward and reverse primer sequences and annealing temperature of the microsatellite markers that map to chromosome 4AL. GWM and WMC primer sequences were obtained from Röder et al. (1998) and Gupta et al. (2002), respectively, except for WMC283 and WMC313. Primer sequences for WMC283 and WMC313 were obtained from GrainGenes (<http://wheat.pw.usda.gov>) and Sejet Plant Breeding, respectively. All forward microsatellite primers were modified to contain a 5', 19-nucleotide M13 tail (Schuelke 2000). The reaction mixture was denatured at 94 °C for 2 min, followed by 30 cycles of 95 °C for 1 min, 51/61 °C for 50 s, 73 °C for 1 min, with a final extension step of 73 °C for 5 min. PCR amplicons were resolved in an ABI Prism 3100 Genetic Analyzer (Applied Biosystems, Foster City, Calif.) with GeneScan software and GeneScan-500 ROX as an internal size standard (Applied Biosystems, Foster City, Calif.). Genographer version 1.6.0 (<http://hordeum.oscs.montana.edu/genographer>) was used to convert chromatograms to gel images.

Linkage analysis

Microsatellite markers putatively linked to the resistance gene, based on the results of BSA, were screened on the entire mapping populations of the ST6/Erik and ST6/Katepwa crosses, and their reciprocals, to verify linkage. The chromosomal locations of these markers were previously determined in other mapping populations (Röder et al. 1998; Chalmers et al. 2001; Gupta et al. 2002; Somers, unpublished data). This information was used to select additional markers in order to identify a large linkage group.

Table 1 Primer sequences and annealing temperatures of the microsatellite markers found on chromosome 4AL

Marker	Forward primer (5'→3') ^a	Reverse primer (5'→3')	Annealing temperature (°C)
GWM160	<u>CACGACGTTG</u> TAAAACGACTTCAATTCAGTCTTGGCTTGG	CTGCAGGAAAAAAAAAGTACACCC	61
GWM397	<u>CACGACGTTG</u> TAAAACGACTGTCATGGATTATTTGGTCGG	CTGCACTCTCGGTATACCAGC	61
GWM637	<u>CACGACGTTG</u> TAAAACGACAAAGAGGTCTGCCGCTAACA	TATACGGTTTTGTGAGGGGG	51
WMC161	<u>CACGACGTTG</u> TAAAACGACACCTTCTTTGGGATGGAAGTAA	GTAAGTGAACCACTTGTAACGCA	51
WMC219	<u>CACGACGTTG</u> TAAAACGACTGCTAGTTTGTTCATCCGGGCGA	CAATCCCCTTCTACAAGTCCA	51
WMC232	<u>CACGACGTTG</u> TAAAACGACGAGATTTGTTTCATTTCATCTTCGCA	TATATTAAGGTTAGAGGTAGTCAG	61
WMC262	<u>CACGACGTTG</u> TAAAACGACGCTTTAAACAAAGATCCAAGTGGCAT	GTAACATCCAAACAAAGTCCGAACG	51
WMC283	<u>CACGACGTTG</u> TAAAACGACCGTTGGCTGGGTTATATCATCT	GACCCGCGTGTAAGTGATAGGA	51
WMC313	<u>CACGACGTTG</u> TAAAACGACGCAGTCTAATTATCTGCTGGCG	GGTCTTGTCTACTCATGTCT	51

^a The underlined portion of the forward primers is the M13 tail

Linkage analysis was conducted with Mapmaker/exp version 3.0b software (Lander et al. 1987; Lincoln et al. 1993). Map distances were converted to centimorgans using the Kosambi function (Kosambi 1944). Linkage groups and genetic-linkage maps were generated using a maximum Kosambi distance of 37.2 cM and a minimum LOD of 2.3 and 3.0 for ST6/Erik and ST6/Katepwa populations, respectively. The 'compare', 'try' and 'map' commands were used to determine the marker order and calculate the map distances.

Results

Disease reaction of F₃ families

F_{2:3} family reactions of the crosses, ST6/Katepwa, Katepwa/ST6, ST6/Erik and Erik/ST6, were reported previously (McCartney et al. 2002). Reciprocal effects were not observed allowing data from reciprocal crosses to be pooled. The observed F₃ family reactions to isolate MG2 were 42:102:56 and 28:57:25 homozygous resistant:segregating:homozygous susceptible for the ST6/Katepwa and ST6/Erik populations, respectively (McCartney et al. 2002). Both datasets fit a 1:2:1 segregation ratio, indicating that ST6 has a single incompletely dominant gene conferring resistance to isolate MG2 of *M. graminicola*. The symbol *Stb7* (Reaction to *M. graminicola* 7) is proposed to designate the resistance gene in ST6 controlling reaction to isolate MG2 of *M. graminicola*.

Marker and linkage analysis

WMC219 was the only marker, of 279 microsatellite markers, that appeared to be linked to *Stb7* in the BSA of the ST6/Erik population. The ST6 allele only amplified in the resistant bulk and the Erik allele only amplified in the susceptible bulk (Fig. 1). The PCR profiles of the ten F₃ families in the resistant bulk and the ten F₃ families in the susceptible bulk are also shown. This result demonstrated a linkage between *Xwmc219* and *Stb7*. The ST6, Erik and Katepwa alleles were 175 bp, 226 bp and 230 bp, respectively.



Fig. 1 PCR profiles produced by amplification of WMC219 in the ST6/Erik population. Numbers on the left indicated the size in bp of the amplicon. Abbreviations: *R Bulk* is the resistant bulk, *S Bulk* is the susceptible bulk, *R1–R10* are the homozygous resistant F₃ families in the resistant bulk, and *S1–S10* are the homozygous susceptible F₃ families in the susceptible bulk

WMC219 mapped to chromosome 4AL in the W7984/Opata reference mapping population (Gupta et al. 2002). The entire ST6/Erik mapping population was screened with WMC219 and other microsatellite markers that map to chromosome 4AL. Linkage analysis of these markers generated a 108.1 cM linkage group (Fig. 2A). *Xwmc313* and *Xgwm219* mapped 0.5 and 1.0 cM from *Stb7*, respectively.

The chromosomal location of *Stb7* was verified in the ST6/Katepwa mapping population (Fig. 2B). The same microsatellite markers formed a 108.3 cM linkage group in this population. *Xwmc313*, *Xwmc219* and *Xgwm160* mapped 0.3, 1.1 and 3.5 cM from *Stb7*, respectively. The linkage groups for the ST6/Erik and ST6/Katepwa populations were nearly identical (Fig. 2). No markers were identified that flanked the resistance gene. The maps of chromosome 4AL produced in this study are consistent with maps of 4AL in other mapping populations (Röder et al. 1998; Chalmers et al. 2001; Gupta et al. 2002; Somers, unpublished data). *Stb7* is located at the distal end of chromosome 4AL.

Canadian wheat cultivars representative of different wheat classes were screened with WMC313 to determine whether the marker will be useful for MAS of *Stb7* in Canadian wheat breeding programs. The ST6 allele of WMC313 was not detected in Canadian common wheat cultivars (Fig. 3). The majority of Canadian common wheat cultivars produced a 229 bp amplicon similar to Katepwa and Erik. The remaining common wheat culti-

vars had null alleles. Eight of nine Canada Western Amber Durum (CWAD) wheat cultivars had a 206 bp amplicon similar to ST6, the other CWAD cultivar Sceptre had a 227 bp amplicon.

Discussion

The present study indicates that *Stb7* is located at the distal end of chromosome 4AL and is the third *Triticum*-derived *Stb* gene to be mapped to a chromosomal location. *Stb7* confers resistance to isolate MG2 of *M. graminicola* (McCartney et al. 2002), a representative member of one of the two races identified in western Canada (Grieger 2001). The present results illustrate the utility of microsatellite markers for locating genes on chromosomes. *Stb7* may be linked to other disease/pest resistance loci, possibly in a resistance gene cluster, since a number of resistance loci have been identified on chromosome 4A. These include *H25*, *Lr28*, *Lr30*, *Pm16*, *Sr7*, *Wsm1*, *YrMin* and *YrND* (McIntosh et al. 1998).

The relationships between the described *Stb* genes have not been completely studied. *Stb1*, *Stb2* and *Stb3* are not genetically linked (Wilson 1985). *Stb4* is not linked to *Stb1*, but whether it is linked to *Stb2* or *Stb3* is unknown (Somasco et al. 1996). *Stb1*, *Stb2*, *Stb3* and *Stb4* have not been assigned to chromosomes. *Stb5* maps near the centromere of chromosome 7DS (Arraiano et al. 2001; Simon et al. 2001) and *Stb6* maps to the distal end to chromosome 3AS (Brading et al. 2002). This information and the present study reveal that *Stb5*, *Stb6* and *Stb7* are not linked to each other. However, their relationship to *Stb1*, *Stb2*, *Stb3* and *Stb4* is unknown. The *T. aestivum* line Salamouni has three additional *Stb* genes that are unlinked to *Stb7*, but their relationship to other *Stb* genes is unknown (McCartney et al. 2002). The number of allelism tests required to differentiate these genes grows rapidly as additional septoria tritici blotch resistance sources are described. Mapping *Stb* genes to chromosomal locations is most likely the quickest method of differentiating different *Stb* genes. Allelism tests can then be performed on genes that map to similar chromosomal locations.

Scoring reaction to *M. graminicola* in segregating generations is difficult and could be facilitated with MAS. Progeny tests appear to provide the most accurate phenotypic ratings (Brading et al. 2002; McCartney et al. 2002), but these are labour intensive. Individual plant

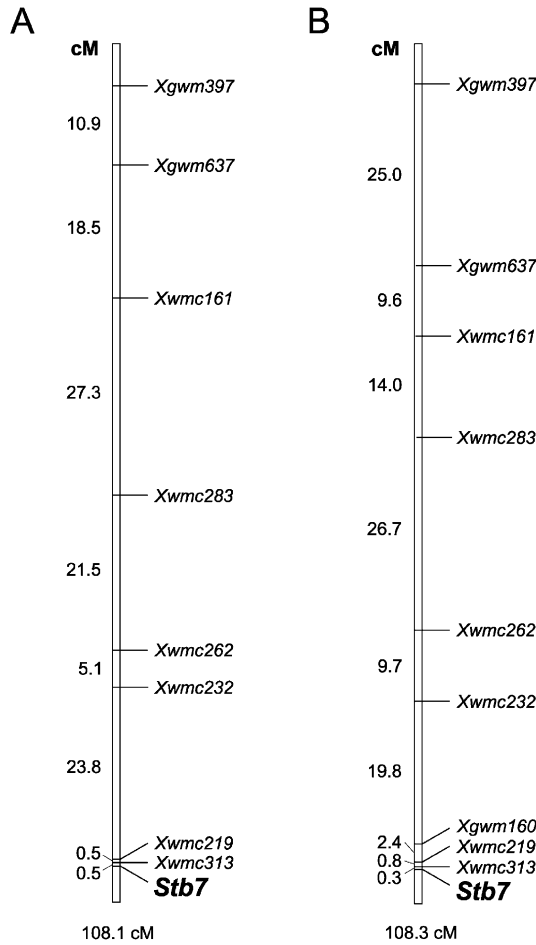


Fig. 2A, B Linkage maps displaying the microsatellite loci and *Stb7* on chromosome 4AL in the (A) ST6/Erik and (B) ST6/Katepwa crosses. *Stb7* is located at the distal end of chromosome 4AL. Distances are reported in Kosambi centimorgans and the linkage groups were established at LOD 2.3 and 3.0 for the ST6/Erik and ST6/Katepwa populations, respectively

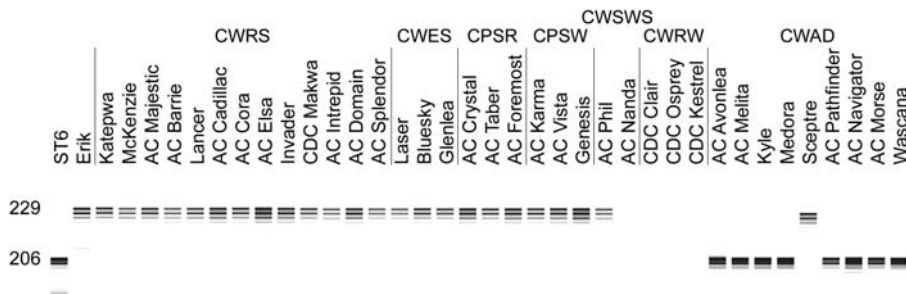


Fig. 3 PCR profiles produced by amplification of WMC313 in a set of Canadian wheat cultivars representative of different wheat classes. Numbers on the left indicated the size in bp of the amplicon. Wheat class abbreviations: CWRS is Canada Western Red Spring, CWES is Canada Western Extra Strong Red Spring,

CPSR is Canada Prairie Spring Red, CPSW is Canada Prairie Spring White, CWSWS is Canada Western Soft White Spring, CWRW is Canada Western Red Winter and CWAD is Canada Western Amber Durum

ratings can be very accurate in controlled growth rooms (McCartney et al. 2002). However, individual plant ratings in the field or greenhouse would not be as accurate due to decreased control of temperature and humidity, which would not allow for maximal expression of resistance and susceptibility. Also, foliar pathogens of wheat often occur together in the field since they require similar conditions for infection (Gilbert et al. 1998) and can be difficult to distinguish. Improper disease identification will result in inaccurate disease data and decrease the genetic gain in breeding populations. Furthermore, *M. graminicola* is known to interact with other wheat pathogens. Brokenshire (1974) reported that *Blumeria graminis* f. sp. *tritici* infection allowed *M. graminicola* to sporulate on a cultivar that is resistant to that *M. graminicola* isolate. MAS can overcome these disease-screening problems because selection is based on genotype rather than phenotype. WMC313 will be very useful for MAS of *Stb7* in Canadian wheat breeding programs given its tight linkage to *Stb7* and that the ST6 allele of *Xwmc313* is rare or absent in elite Canadian common wheat germplasm.

The production of PCR amplicons of 206 bp in length in ST6, and eight of nine CWAD wheat cultivars using WMC313 is intriguing as it suggests that *Stb7* may be commonly present in CWAD cultivars. Other data support this possibility. First, CWAD cultivars appear to have septoria tritici blotch resistance gene(s) absent in Canadian common wheat cultivars, because the prevalence of *M. graminicola* in CWAD wheat fields was significantly lower than in CWRS and CPS wheat fields in a survey of leaf-spotting pathogens in Manitoba from 1990 to 1995 (Gilbert et al. 1998). Second, the CWAD cultivar Coulter has two septoria tritici blotch resistance genes, one of which has the same race specificity as *Stb7* based on the available data (McCartney et al. 2002). If certain septoria tritici blotch resistance genes, such as *Stb7*, are common in durum cultivars and rare in common wheat cultivars or vice-versa, this would explain the host-species specialization reported previously in *M. graminicola* (Kema et al. 1996). Further research is needed to determine whether *Stb7* is present in CWAD cultivars to lend further support to this hypothesis.

Introgression of *Stb7* into Canadian common wheat germplasm has been initiated. An F₃ family was detected in the ST6/Katewpa population that is segregating for *Stb7* but is homozygous for Katewpa alleles for the majority of chromosome 4A. A recombination event has occurred between *Xwmc313* and *Xwmc219* in this line, which will limit the amount of ST6 DNA that will be introduced into Canadian breeding material.

In summary, *Stb7* was mapped to the distal end of chromosome 4AL and tagged with three tightly linked microsatellite markers, GWM160, WMC219 and WMC313. WMC313 will be useful for MAS of *Stb7* in Canadian breeding programs since it is tightly linked to *Stb7*, and the ST6 allele of *Xwmc313* is absent or rare in Canadian common wheat cultivars.

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