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Genetic Mapping of Wheat Curl Mite Resistance Genes *Cmc3* and *Cmc4* in Common Wheat

R. Malik, G. L. Brown-Guedira,* C. M. Smith, T. L. Harvey, and B. S. Gill

ABSTRACT

To diversify the genetic base of resistance in wheat (*Triticum aestivum* L.) to the wheat curl mite (WCM), *Aceria tosichella* Keifer, resistance to this pest was transferred from the dipolid goatgrass *Aegilops taushcii* (Coss.). Schmal. to the hard red winter wheat germplasm KS96-WGRC40 by backcrossing to the cultivar TAM 107. KS96WGRC40 has WCM resistance derived from both *Ae. tauschii* and rye (*Secale cereale* L.). The objectives of this study were to determine if a unique WCM resistance gene was transferred from *Ae. tauschii* to KS96-WGRC40 and to determine the chromosome and linkage map locations of the WCM resistance genes in the germplasm. The rye-derived WCM resistance gene in TAM 107 and KS96WGRC40, designated *Cmc3*, is present on wheat–rye translocation T1AL•1RS. Marker analysis of a segregating F_2 population revealed that the rye-specific microsatellite marker SCM09 can be used to select wheat lines carrying the 1RS segment and *Cmc3*. Allelism tests indicated that the *Ae. tauschii*-derived WCM resistance gene in KS96WGRC40, designated *Cmc4*, segregated independently of the *Cmc1* gene previously transferred from this species. Molecular and cytogenetic analyses located *Cmc4* distally on chromosome 6DS flanked by markers *Xgdm141* (4.1 centimorgans, cM) and *XksuG8* (6.4 cM). The linked markers may be used in wheat breeding programs for the selection of lines resistant to WCM and for gene pyramiding.

THE WHEAT CURL MITE is an arthropod pest of wheat which vectors the *Wheat streak mosaic virus* (WSMV) (Slykhuis, 1955; Nault and Styer, 1970). The WSMV causes significant yield losses in wheat growing areas of the U.S. and the Canadian Great Plains (Sim and Willis, 1988; Bockus et al., 2001). In addition, nonviruliferous WCM infestations may reduce wheat grain yields by as much as 17% (Harvey et al., 2000). Mite resistant cultivars show lower rates of WSMV infection than mite susceptible cultivars (Conner et al., 1991; Harvey et al., 1994), demonstrating the importance of host resistance to an arthropod vector in controlling a plant virus.

Two genes conferring resistance to WCM have been named, *Cmc1* transferred to wheat chromosome 6D from *Aegilops tauschii* (Coss.). Schmal. (syn. *Ae. squarrosa* L.; *Triticum tauschii*) (Thomas and Conner, 1986; Whelan and Thomas, 1989) and the *Cmc2* gene transferred to 6D from *Agropyron elongatum* (Host). Beauv. (Martin et al., 1976; Whelan and Hart, 1988). An unnamed gene was transferred to chromosome 6A of wheat from *Haynaldia villosa* (L.) Schur as a T6AL•6VS translocation (Chen et al., 1996). In addition, WCM resistance was transferred to the hard red winter wheat cultivar TAM 107 from 'Amigo' wheat (Cox 1991), which contains the wheat–rye translocated chromosome T1AL•1RS (Lapitan et al., 1986; Schlegel and Kynast, 1987). The rye segment in the translocation chromosome was derived from 'Insave F. A.' rye through the triticale 'Gaucho' (CI15323) (Sebesta et al., 1994a,b). Although Wood et al. (1995) demonstrated that Insave F. A. rye and Gaucho triticale both are resistant to WCM, cosegregation of the T1AL•1RS chromosome with WCM resistance has not been demonstrated and the WCM resistance gene(s) has not been named. The cultivar TAM 107 has been widely grown in the western part of the southern Great Plains and WCM collections from western Kansas have been identified that are able to colonize TAM 107 (Harvey et al., 1999).

To diversify resistance to the WCM in hard winter wheat germplasm, resistance was transferred from accession TA 2397 of the diploid relative *Ae. tauschii* to the wheat germplasm KS96WGRC40 (Cox et al., 1999) by backcrossing into a TAM 107 background. The germplasm line has WCM resistance derived from both TAM 107 and *Ae. tauschii* accession TA 2397. To characterize and name the gene(s) transferred to KS96WGRC40 from TA 2397, it is necessary to also characterize the rye-derived resistance in the recurrent wheat parent. The objectives of this study were to determine if a unique WCM resistance gene was transferred from *Ae. tauschii* to KS96WGRC40 and to determine the chromosome and linkage map locations of the WCM resistance genes in KS96WGRC40.

MATERIALS AND METHODS

Plant Material

KS96WGRC40 is a hard red winter wheat germplasm with the pedigree KS93U69*3/TA 2397 (Cox et al., 1999). KS93U69 is a leaf rust-resistant germplasm with the pedigree TAM 107*3/TA 2460. TA 2460 is a leaf rust-resistant accession of *Ae. tauschii*. The WCM resistance of KS96WGRC40 is derived from TAM 107 and from *Ae. tauschii* accession TA 2397 originally collected from Afghanistan. Other lines included in the

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Abbreviations: cM, centimorgans; WCM, Wheat Curl Mite; WSMV, *Wheat streak mosaic virus*.

study were TAM 107, 'Tomahawk', 'Wichita', TA 2397, 'Norstar', and W304, a near-isogenic line of Norstar (Norstar*4/AC PGR-16635) containing the *Cmc1* gene which was also derived from *Ae. tauschii* (Thomas and Conner, 1986). The lines W304 and Norstar were provided by J. Thomas, Agriculture Canada Research Station, Winnipeg, Canada.

To map the rye-derived WCM resistance gene in TAM 107, a population of 63 F_2 plants from a cross between TAM 107 and the susceptible cultivar 'Tomahawk' was phenotyped with WCMs collected from Montana (MT) (Harvey et al., 1999). The chi-square test (χ^2) was used to test the goodness-of-fit of the data to the expected phenotypic segregation ratio of 3 resistant plants: 1 susceptible plant.

Crosses were made between KS96WGRC40 and Wichita (WCM susceptible) lines monosomic for the wheat D-genome chromosomes. The monosomics were used as the female parent in all crosses. Monosomic F_1 plants ($2n = 41$) were identified cytologically by counting chromosomes from the root tips of seedlings as described by Endo and Gill (1984) and F_2 progeny were obtained. Only D-genome monosomics were used because the new resistance gene in KS96WGRC40 was derived from *Ae. tauschii,* the D-genome progenitor of common wheat. The F_1 plants and progeny were phenotypically screened with WCMs from the Kansas (KS) colony, which are virulent to the rye source of WCM resistance in TAM 107 and KS96WGRC40 (Harvey et al., 1999). At the two-leaf stage, 70 to 100 F_2 plants from each monosomic family were tested in two different experiments. Plants of 109 F_3 families from individual F_2 plants in non-critical crosses were also evaluated for reaction to WCMs from KS and comprised the mapping population. TAM 107, Wichita, and KS96WGRC40 were planted as controls in each flat. The chi-square test was used to test the goodness-of-fit of the data to the expected phenotypic segregation ratios.

KS96WGRC40 was crossed to the line W304, having the *Cmc1* gene for WCM resistance. Three-hundred seventy-five F_2 plants were tested with the KS strain of WCM, which is avirulent to *Cmc1* and to the *Ae. tauschii*-derived gene in KS96WGRC40 but is virulent to the rye source of resistance in KS96WGRC40 (Harvey et al., 1999). Chi-square analysis was used to test the goodness-of-fit of the data to predicted Mendelian ratios.

Phenotypic Screening

Seeds of each line were germinated in flats and plants at the two-leaf stage were infested with 10 aviruliferous mites per plant according to Harvey et al. (1998). The mites were counted using a dissecting microscope with 7× magnification and a fiber optic illumination system. After infestation, the flats were placed in the greenhouse and grown under an 8-h dark/16-h light period and 25 ± 5°C. Since WCM completes a generation in 10 d, seedlings were scored after 14 d as resistant (normal leaves) or susceptible (curled or trapped leaves). Plants of resistant lines and susceptible checks were tested using WCM colonies collected from Montana, Kansas and Nebraska. The WCM colonies obtained from Montana (MT) and Nebraska (NE) were originally provided by Drs. Sue Blodgett and Talat Mahamood, respectively, and the WCM colony from KS was collected in 1996 from Ellis county. All the WCM colonies were reared on the susceptible wheat cultivar Tomahawk at Kansas State University, Agricultural Research Center, Hays, KS.

Marker Analysis

Marker analysis of the rye-derived WCM resistance gene in TAM 107 was performed on the genomic DNA extracted from a population of F_2 plants from the cross between TAM 107 and Tomahawk using the microsatellite primer pair SCM09 (Saal and Wricke, 1999) specific for chromosome 1RS. For mapping the *Ae. tauschii*-derived gene in KS96WGRC40, a population of 109 F3 lines was derived from non-critical monosomic crosses segregating for resistance to WCM. Total genomic DNA was extracted from individual F_2 plants for microsatellite analyses and from 20 bulked progeny of F_2 plants for RFLP analyses according to the procedure below.

Approximately 7 g of leaf tissue was collected from 4-wk-old plants, frozen in liquid nitrogen, ground with a mortar and pestle, and transferred to 50-mL polypropylene tubes. Equal volumes of DNA extraction buffer [100 m*M* glycine, 50 m*M* NaCl, 10 m*M* EDTA, 2% SDS (w/v), and 30 m*M* sodium lauryl sarcosine, pH adjusted to 9.0 with NaOH] and phenol:chloroform:isoamylalcohol (50: 49: 1) were added to the ground leaf tissue. The resulting mixture was vigorously shaken for 20 min. at room temperature, followed by centrifugation at 5810 *g* for 15 min. The supernatant was collected in a fresh tube and the DNA was precipitated according to Sambrook and Russell (2001).

RFLP analysis was performed using *Bam*HI, *Eco*RI, *Eco*RV, *Xba*I, and *Hin*dIII restriction endonucleases according to Faris et al., 2000. The PCR was performed in a 25-μL final reaction in an MJ-100 thermocycler (MJ Research, Waltham, MA) as described by Röder et al. (1998). The amplification products were electrophoresed at 57 V for 4 h on a 2.3% (w/v) SFR (AMRESCO Inc., Solon, OH) agarose gel or on nondenaturing polyacrylamide gels ranging from 6 to 15% (w/v) at 100V for 10 to 12 h.

A total of 47 microsatellite and RFLP markers specific to chromosome 6D (Table 1) were screened for polymorphism

Table 1. Markers on wheat chromosome 6D screened for linkage to wheat curl mite resistance gene *Cmc4*.

Marker	Source
GWM55†, GWM325, GWM469.	Röder et al., 1998
GDM14‡, GDM37, GDM95, GDM125, GDM132, GDM141.	Pestova et al., 2000
WMS904§, WMS1009.	Röder, unpublished
WMC250¶, WMC256.	Varshney et al., 2000
BARC5#, BARC17, BARC21, BARC23, BARC25, BARC30, ARC123, BARC146, BARC173, BARC175, BARC183, BARC196, BARC202, BARC204.	Cregan and Song, unpublished
ABG380, ABG378, ABG484, ABG458, ABG466, ABG476.	S.A. Kleinhofs, WSU, USA
BCD9, CDO388, GLK558, GLK724.	M.E. Sorrells, Cornell University
CXP1	D. Baulcombe
KSUF36, KSUF43, KSUG8, KSUG48, KSUH4, KSUH11, KSUI28.	B.S. Gill, KSU, USA
PSR106, PSR167.	M. Gale, JI Centre, UK

† GWM = Gatersleben wheat microsatellite.
‡ GDM = Gatersleben D-genome microsatellite.
§ WMS = Wheat microsatellites.
¶ WMC = Wheat microsatellite consortium.
# BARC = Beltsville Agriculture Research Centre

between KS96WGRC40 and Wichita. Markers showing polymorphisms were then applied to the F_3 population segregating for WCM resistance and these data were used to determine linkage between the markers and WCM resistance. A genetic linkage map was constructed by converting recombination frequencies to map distance (cM) using MAPMAKER version 2.0 at LOD > 3.0 (Lander et al., 1987) and the Kosambi mapping function (Kosambi, 1944). Markers not meeting that threshold were placed in the most likely interval by means of the MAPMAKER "try" command at LOD < 3.0.

To evaluate the usefulness of the *Xgdm141* marker linked to the *Cmc4* gene for marker-assisted selection, hard winter wheat cultivars adapted to the central plains (including '2137', '2163', '2174', '2180', 'Alliance', 'Arlin', 'Carson', 'Cimarron', 'Custer', 'Heyne', 'Halt', 'Ike', 'Jagger', 'Kalvesta', 'Karl92', 'Lakin', 'Millenium', 'Nekota', 'Prairie Red', 'Prowers', 'Stanton', 'TAM 202', 'TAM 300', 'TAM 301', 'TAM 302', 'Tonkawa', 'Trego', 'Venango', 'Wesley', 'Wichita', 'Windstar', 'Yuma', and 'Yumar') were analyzed for polymorphism with KS96WGRC40.

RESULTS

Only KS96WGRC40 and TA 2397 were consistently resistant to the WCM collections from KS, MT, and NE (Table 2). A greater number of WCMs was observed on the line W304, which has the *Cmc1* gene, when infested with WCMs collected in NE than was observed on KS96WGRC40 and *Ae. tauschii* accession TA 2397. Harvey et al. (1999) also observed that W304 was not resistant to colonization by WCMs collected in NE. Low numbers of WCMs were observed on TAM 107 10 d after infestation with the NE and MT WCM colonies, whereas mites of the KS colony were able to reproduce on TAM 107.

Resistance to the MT strain of WCM in TAM 107 was conditioned by a single dominant gene designated *Cmc3*. The observed segregation of 44 resistant and 19 susceptible plants in an F_2 population derived from the cross between TAM 107 and Tomahawk fit a 3: 1 ratio ($P > 0.60$). The microsatellite marker *Xscm09*, located on rye chromosome arm 1RS (Korzun et al., 2001), cosegregated with WCM resistance in the population. The SCM09 primer pair amplified a 220-bp fragment in TAM 107 and all WCM resistant F_2 plants. No amplification product was detected in Tomahawk and all WCM susceptible F_2 plants (Fig. 1). Since chromosome arms 1RS and 1BS do not recombine, cosegregation of resistance and the rye-specific *Xscm09* marker in the population indicates that the WCM resistance gene is present on the T1AL•1RS translocated chromosome.

Table 2. Average number of wheat curl mites per wheat plant after 10 d of infestation with wheat curl mites from Nebraska (NE), Kansas (KS), and Montana (MT).

		WCM strains		
Wheat line	**Gene**	**NE**	**KS**	**MT**
W304	***Cmc1***	**26 ± 4†**	**11 ± 2**	**9 ± 1**
KS96WGRC40	***Cmc3* and *Cmc4***	**6 ± 2**	**5 ± 1**	**6 ± 2**
TAM 107	***Cmc3***	**13 ± 4**	**70 ± 12**	**15 ± 6**
TA2397	***Cmc4***	**4 ± 2**	**6 ± 3**	**7 ± 1**
Tomahawk	**None**	**49 ± 5**	**70 ± 12**	**84 ± 4**

† Standard error of the mean.

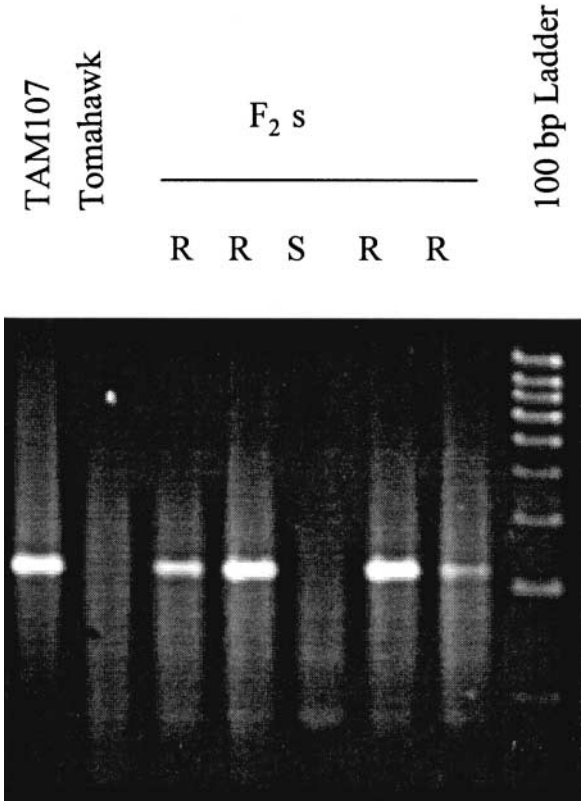

Fig. 1. Electrophoretic pattern of the dominant microsatellite marker SCM09 on WCM resistant wheat TAM 107, susceptible Tomahawk, and segregating F_2 progenies. R and S indicate resistant and susceptible phenotype.

Resistance in KS96WGRC40 to colonization by WCMs from KS, which are virulent to the *Cmc3* gene in TAM 107 (Table 2), is conditioned by a single dominant gene on chromosome 6D. All monosomic and disomic F_1 plants from the crosses between the KS96WGRC40 and the susceptible D-genome Wichita monosomic lines were resistant to WCM. The F_2 progeny from monosomic F_1 plants segregated in 3 resistant: 1 susceptible ratios, except those from the 6D and 4D monosomic crosses (Table 3). Progeny from the monosomic 6D cross significantly deviated from a 3:1 ratio ($P < 0.001$) because of an excess of resistant plants. Location of the WCM resistance gene on chromosome 6D was confirmed by subsequent molecular marker analysis. The F_2 progeny from different F_1s of the cross with monosomic 4D also significantly deviated from a 3:1 ratio ($P < 0.0002$) because of an excess of susceptible plants. Although highly deviating, chromosome 4D was counted as a noncritical cross since the critical cross in monosomic analysis has an excess of resistant plants (Nyquist, 1957). Results from both runs of the experiment were similar, eliminating the possibility of poor infestation.

The WCM resistance gene from *Ae. tauschii* in KS96WGRC40 is different from the *Cmc1* gene previously transferred from this species, although both are located on chromosome 6D. The observed segregation of 353 resistant: 22 susceptible F_2 plants from the cross KS96WGRC40 × W304 when screened with WCMs from the KS colony fit a 15 resistant: 1 susceptible ratio (χ^2 =

Table 3. Response of F_2 populations derived from monosomic F_1 plants of crosses of Wichita D-genome monosomics and KS96WGRC40 when infested with the Kansas strain of the wheat curl mite.

Chromosome	Number of Progeny		
Tested	Resistant	Susceptible	χ^2*
1D	61	29	2.5
2D	66	32	3.06
3D	67	23	0.01
4D	54	40	14.23
5D	60	21	0.04
6D	70	7	10.39
7D	61	20	0

* Value for significance at $P = 0.05$ and 1 d.f is 3.84.

0.09, $p > 0.75$). All seedlings of KS96WGRC40 and W304 were normal and seedlings of the susceptible checks, TAM 107 and Tomahawk, had tightly curled leaves. To confirm that the genes were different, the F_3 lines that segregated 3:1 with the KS WCM collection were also tested with the NE WCM collection, which is virulent to *Cmc1*. Homozygous susceptible lines were identified, indicating that they segregated only for *Cmc1* (data not shown). The *Ae. tauschii*-derived WCM resistance gene in KS96WGRC40 is designated *Cmc4*.

Out of 47 tested microsatellite and RFLP loci on chromosome 6D, 38% comprising nine microsatellites (33%) and nine RFLP probes (45%) were polymorphic between KS96WGRC40 and Wichita. Only the markers that generated clear polymorphisms were scored in the mapping population of F_3 lines. Segregation for WCM response in the population fit the expected segregation ratio of 1 homozygous resistant: 2 segregating: 1 homozygous susceptible line ($P > 0.5$).

Two markers closely flanking the *Cmc4* locus were identified. Microsatellite marker *Xgdm141* was 4.1 cM proximal and the RFLP marker *XksuG8* was 6.4 cM distal from *Cmc4* (Fig. 2). The microsatellite GDM141 was codominant and heterozygotes were easily differentiated from homozygotes. Primer GDM141 amplified a 135-bp fragment in KS96WGRC40 and a 120-bp fragment in Wichita (Fig. 3a). The RFLP probe KSUG8 detected a 3-kb fragment (Fig. 3b) linked to the resistance gene in KS96WGRC40 and was scored as a dominant marker. The microsatellite marker *Xwms904* was linked to *Cmc4* but because of the absence of an amplified fragment in the resistant parent (Fig. 3c), could not be placed on the genetic map with LOD > 3.0. However, all but one of the F_3 lines missing a *Xwms904* fragment were homozygous resistant to WCM. The order of all markers mapped in this study was the same as those of previously published maps (Röder et al., 1998; Boyko et al., 1999; Pestova et al., 2000; Weng et al., 2000; Boyko et al., 2002). With the exception of the *Xgdm132* microsatellite, which was not placed on the genetic map, all markers segregated in the expected 3:1 or 1:2:1 ratios.

The size of amplified products obtained with primers GWM469 and WMS904 indicated that the fragments amplified in KS96WGRC40 were derived from TA 2397 (Fig. 3c). However, microsatellite GDM141, which was proximal to *Cmc4*, amplified a 135-bp fragment in KS96-WGRC40 and TAM 107 and a 120-bp fragment in TA-

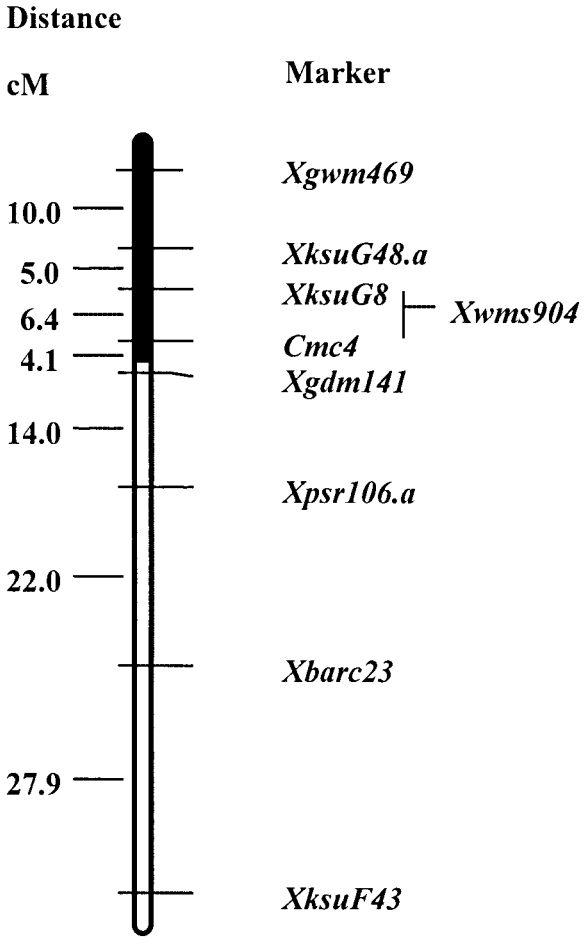

Fig. 2. Genetic map of wheat chromosome 6DS. The dark region of the chromosome represents the *Aegilops tauschii* (TA2397) segment containing *Cmc4* in common wheat germplasm KS96WGRC40. The light region represents the TAM 107 background.

2397 (Fig. 3a), indicating that the fragment in KS96-WGRC40 linked to *Cmc4* was derived from TAM 107. During transfer of resistance from TA 2397 to wheat, recombination occurred between *Xgdm141* and *Cmc4* (Fig. 2), which may limit the usefulness of this marker to breeding programs since marker polymorphism within the D-genome of *T. aestivum* is much lower than that observed between wheat and *Ae. tauschii* (Dvórak et al., 1998). To determine if this marker would be useful to do marker-assisted selection for *Cmc4*, DNA of 34 cultivars developed by breeding programs in central plains of North America was amplified with the GDM141 primer pair. The 135-bp fragment found in KS96WGRC40 and TAM 107 was present in five of the cultivars (Fig. 4). With the exception of Pronghorn and Yumar, the presence of this fragment in the cultivars was associated with presence of TAM 107 in their pedigrees.

DISCUSSION

Our marker analysis confirms the observation of Wood et al. (1995) that resistance to WCM in TAM•107 was transferred from rye via the Amigo translocation and provides a basis to designate the gene on chromosome T1AL 1RS as *Cmc3*. Although WCMs collected from

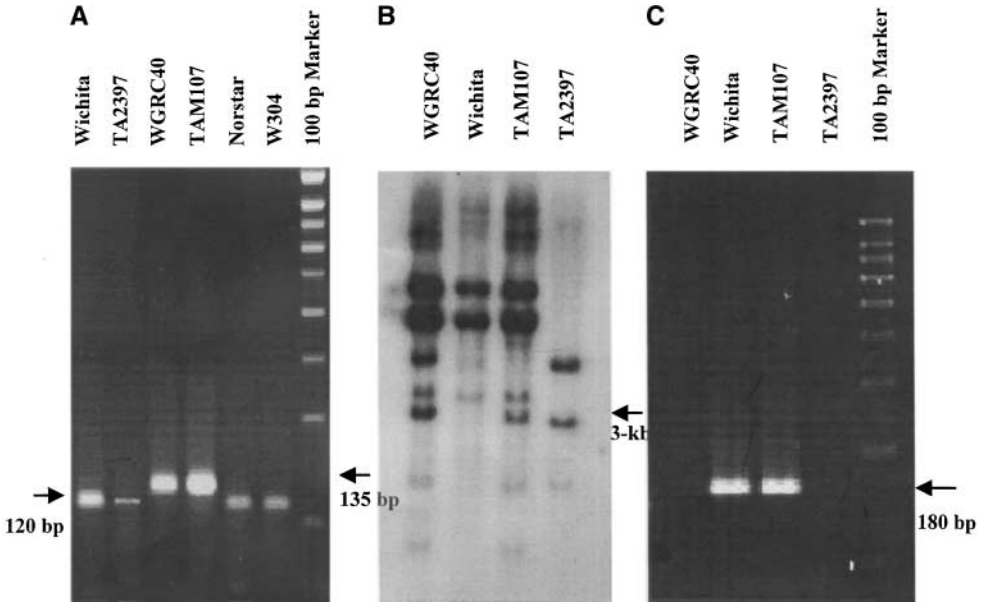

Fig. 3. Molecular markers closely linked to the *Cmc4* locus in WGRC40. DNA polymorphisms as detected in WGRC40 (*Cmc4*), Wichita, TAM 107, TA2397, W304 (*Cmc1*), and Norstar. Arrows indicate size of the polymorphic fragments. (a) A codominant microsatellite GDM141 (b) a dominant RFLP detected by restriction endonuclease (*Xba*I) digestion of the genomic DNA and hybridized to the probe KSUG8 (c) polymorphism detected by microsatellite marker WMS904.

some locations in KS have overcome *Cmc3*, this gene is effective against most WCM populations (Harvey et al., 1999) and can still be used to protect wheat in other areas.

The new WCM resistance gene transferred from *Ae. tauschii* to wheat germplasm KS96WGRC40 is designated *Cmc4*. With the exception of the *Cmc3* gene, all the reported WCM resistance genes transferred to wheat from related species (including *Cmc4*) are located on the short arm of group 6 chromosomes. This may be due to a common origin of WCM resistance genes among the Triticeae. Although both the *Ae. tauschii*-derived genes *Cmc4* and *Cmc1* are located on wheat chromosome arm 6DS (Thomas and Conner 1986), our data indicate that they are different loci.

KS96WGRC40 has *Ae tauschii*-derived genes for re-

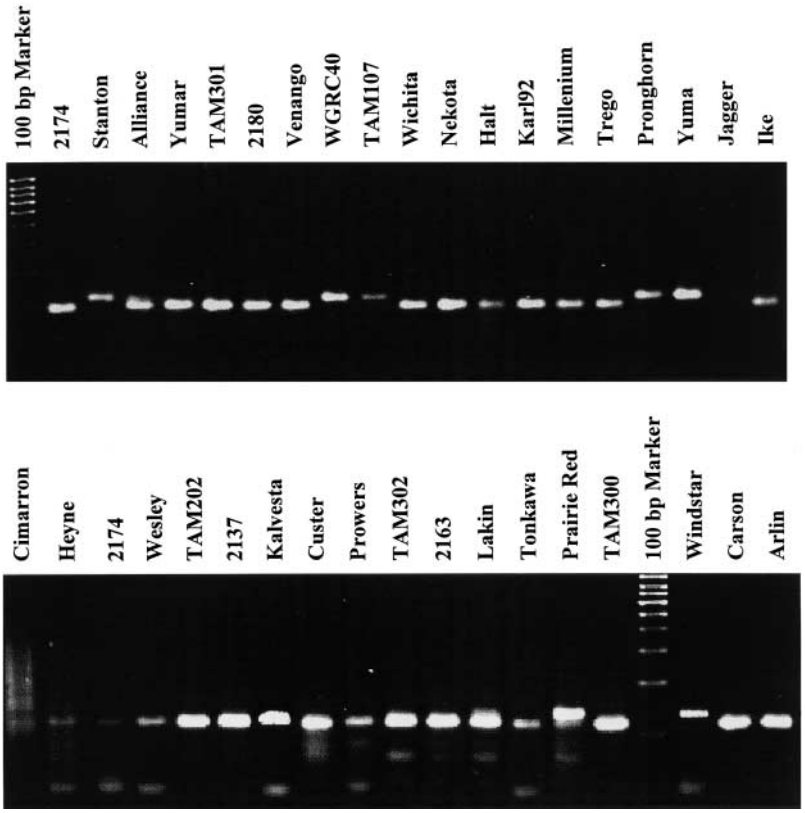

Fig. 4. Polymorphism detected with a closely linked microsatellite marker GDM141 in hard winter wheat cultivars.

sistance to leaf rust (*Lr41*), Septoria leaf blotch (caused by *Septoria tritici* Roberge in Desmaz.), and Stagnospora leaf blotch [caused by *Phaeosphaeria avenaria* (G.F. Weber) O. Eriksson f. sp. *triticea* T. Johnson] in addition to *Cmc4* (Cox et al., 1999). However, only a small fragment of the terminal portion of wheat chromosome 6DS from TA 2397 was transferred to KS96WGRC40. Deletion mapping of the microsatellites GWM469 and GDM141 showed their presence within the interval 0.99–1.00 (Malik et al., unpublished). The *XksuG8* and *XksuG48* loci we mapped between *Xgdm141* and *Xgwm469* were previously physically mapped in the same deletion interval (Boyko et al., 1999; Weng et al., 2000). This deletion interval on chromosome 6D accounts for the terminal 1% of the short arm and contains the *Cmc4* gene.

Traditional breeding methods can be enhanced by marker-assisted selection, especially in pyramiding genes for resistance. On the basis of recombination frequencies, the probability that *Cmc4* will be selected by retaining the markers *Xgdm141* or *XksuG8* individually is 0.96 or 0.94, respectively. If there is no interference in the region of interest during introgression, then the probability of selecting a resistant genotype by means of both flanking markers is 0.995. Although the *Xgdm141* fragment linked to *Cmc4* in KS96WGRC40 was derived from TAM 107, this marker will be useful in transferring *Cmc4* to wheat cultivars adapted to the central plains of the USA that are not closely related to TAM 107.

The use of microsatellite marker *Xwms904* can further increase the efficiency of the marker-assisted selection of *Cmc4*. Locus *Xwms904* was located between *Cmc4* and *XksuG8*, although the null allele present in KS96WGRC40 did not allow an accurate estimate of linkage to *Cmc4*. The WMS904 microsatellite amplified a fragment in 31 of the 34 wheat cultivars tested (data not shown) and would thus be polymorphic between KS96WGRC40 and most hard winter wheat lines. Haley et al. (1994) showed that selections based on markers linked in repulsion provided a greater number of plants (81.8 versus 26.3%) homozygous for resistance to bean common mosaic virus as opposed to selection based on only markers in the coupling phase. This improvement in selection efficiency resulted from a reduction in the number of heterozygote and homozygote susceptible classes.

Several *Cmc* genes provide resistance to WCM colonization. However, *Cmc4* is the first gene mapped in common wheat that provides resistance to diverse populations of the WCM. The flanking markers *Xgdm141*, *Xwms904*, and *XksuG8* should be useful in molecular marker-based introgression of *Cmc4* into wheat cultivars. In addition, the *Xscm09* microsatellite marker can be used as a tag for the *Cmc3* gene and the T1AL•1RS chromosome. These markers can be used in combination for pyramiding WCM resistance genes.

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