Theor Appl Genet (2009) 119:1489–1495
DOI 10.1007/s00122-009-1150-y

ORIGINAL PAPER

MlAG12: a *Triticum timopheevii*-derived powdery mildew resistance gene in common wheat on chromosome 7AL

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Received: 14 March 2009 / Accepted: 30 August 2009 / Published online: 18 September 2009

Abstract Wheat powdery mildew is an economically important disease in cool and humid environments. Powdery mildew causes yield losses as high as 48% through a reduction in tiller survival, kernels per head, and kernel size. Race-specific host resistance is the most consistent, environmentally friendly and, economical method of control. The wheat (*Triticum aestivum* L.) germplasm line NC06BGTAG12 possesses genetic resistance to powdery mildew introgressed from the AAGG tetraploid genome *Triticum timopheevii* subsp. *armeniacum*. Phenotypic evaluation of F_3 families derived from the cross NC06BGTAG12/'Jagger' and phenotypic evaluation of an F_2 population from the cross NC06BGTAG12/'Saluda' indicated that resistance to the 'Yuma' isolate of powdery mildew was controlled by a single dominant gene in NC06BGTAG12. Bulk segregant analysis (BSA) revealed simple sequence repeat (SSR) markers specific for chromosome 7AL segregating with the resistance gene. The SSR markers *Xwmc273* and *Xwmc346* mapped 8.3 cM distal and 6.6 cM proximal, respectively, in NC06BGTAG12/Jagger. The multiallelic *Pm1* locus maps to this region of chromosome 7AL. No susceptible phenotypes were observed in an evaluation of 967 F_2 individuals in the cross NC06BGTAG12/'Axminster' (*Pm1a*) which indicated that the NC06BGTAG12 resistance gene was allelic or in close linkage with the *Pm1* locus. A detached leaf test with ten differential powdery mildew isolates indicated the resistance in NC06BGTAG12 was different from all designated alleles at the *Pm1* locus. Further linkage and allelism tests with five other temporarily designated genes in this very complex region will be required before giving a permanent designation to this gene. At this time the gene is given the temporary gene designation *MlAG12*.

Communicated by B. Keller.

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Introduction

Powdery mildew, caused by *Blumeria graminis* (syn. *Erysiphe graminis*) DC. f. sp. *tritici* (*Bgt*) Em. Marchal, is an economically important disease of wheat in growing regions with cool and humid environments. This biotrophic fungus reduces grain yields by as much as 48% on susceptible cultivars during severe epidemics (Everts and Leath 1992). Powdery mildew infections have negative impacts on end-use quality parameters of wheat caused by the depletion of carbohydrate reserves available for grain filling (Shtienberg 1992). The disease appears on susceptible cultivars from the seedling stage through head

emergence and causes a decrease in number and survival of tillers, kernels per head, and kernel weight (Leath and Bowen 1989).

Host resistance in the form of race-specific, monogenic, or qualitative resistance is a common type of control for powdery mildew in wheat. Race-specific host resistance is the most cost effective, environmentally sound, and consistently used method of control (Leath and Bowen 1989; Hardwick et al. 1994). Currently 57 powdery mildew resistance (*Pm*) genes at 40 loci have been formally designated (Huang and Röder 2004; McIntosh et al. 2008). Nevertheless, the effectiveness of widely deployed *Pm* genes can be ephemeral due to new mutations for virulence in the pathogen and introduction or increase in frequencies of previously rare variants. (Niewoehner and Leath 1998; Parks et al. 2008).

Breeding for powdery mildew resistance has relied heavily on the primary and secondary wheat gene pools for new sources of resistance. *T. timopheevii* (Zhuk.) Zhuk. subsp. *timopheevii*, and subsp. *armeniacum* (Jakubz.) MacKey ($2n = 4x = 28$) are valuable resources for powdery mildew resistance. Three resistance genes, *Pm6* (Jorgensen and Jensen 1973), *Pm27* (Peusha et al. 2000), and *Pm37* (Perugini et al. 2008), have been transferred from *T. timopheevii* into common hexaploid wheat.

The long arm of chromosome 7A is a key genomic region for powdery mildew resistance. The *Pm1* locus with five designated alleles (a–e) is located on chromosome 7AL. Alleles at the *Pm1* locus trace to *T. monococcum*, *T. spelta*, and *T. aestivum* (Hsam et al. 1998; Singrün et al. 2003). Powdery mildew genes *Mlm2033* and *Mlm80*, identified in *T. monococcum* (Yao et al. 2007), and powdery mildew gene *MlIW72T,* identified in *T. dicoccoides* (Ji et al. 2008), also mapped to the *Pm1* region. Additional resistance genes mapped close to the *Pm1* locus include *Pm9*, *mlRD30*, and *PmU* (Schneider et al. 1991; Singrün et al. 2004; Qiu et al. 2005).

Molecular markers closely linked to powdery mildew resistance genes are reported in the literature (Huang and Röder 2004; Miranda et al. 2006, 2007; Perugini et al. 2008). Molecular markers facilitate the combining of resistance genes into inbred lines or cultivars to increase the durability of the race-specific resistance (Liu et al. 2002; Hsam and Zeller 2002). Marker-assisted selection (MAS) can decrease the breeding population size and increase the frequency of favorable alleles at the target loci under selection (Bonnett et al. 2005). Many different marker systems have been employed on powdery mildew resistance genes (see Huang and Röder 2004 for review), but microsatellites or simple sequence repeats (SSR) are the most polymorphic and cost effective marker system available so far in wheat.

The identification of new and novel sources of powdery mildew resistance in adapted germplasm is beneficial to wheat breeders. Further identification of molecular markers linked to each new source of resistance is advantageous for MAS in cultivar development. The objectives of this study were to determine the inheritance, chromosomal location, and SSR markers linked to powdery mildew resistance in wheat germplasm line NC06BGTAG12.

Materials and methods

Plant materials

Powdery mildew resistant wheat germplasm line NC06BGTAG12 (NC-AG12) was crossed with the susceptible winter wheat cultivars Jagger (PI 593688) and Saluda (PI 480474). NC-AG12 (PI 642416) is a BC_2F_7-derived line with pedigree Saluda*3/PI 538457 (Murphy et al. 2007). PI 538457 is a powdery mildew resistant *T. timopheevii* subsp. *armeniacum* accession collected in Iraq. Saluda is a soft red winter wheat released by Virginia Polytechnic Institute and State University (Starling et al. 1986). Saluda possesses the powdery mildew resistance gene *Pm3a*, which is ineffective against the naturally occurring powdery mildew population in North Carolina (Parks et al. 2008). Jagger is a hard red winter wheat developed and released by Kansas State University (Sears et al. 1997). One hundred twenty-eight F_3 families were developed from NC-AG12/Jagger. F_2 populations derived from the crosses NC-AG12/Saluda, and NC-AG12/Axminster were also developed. The NC-AG12/Saluda population was used to determine the gene action of the powdery mildew resistance gene in NC-AG12. Axminster (PI 228307) is a powdery mildew resistant *T. aestivum* cultivar that possesses *Pm1a*. The NC-AG12/Axminster population was used to determine the linkage relationship of the resistance gene in NC-AG12 to the *Pm1* locus.

Phenotypic evaluations

Detached leaf evaluations

Lines possessing *Pm1a*, *Pm1b*, *Pm1c*, *Pm1d*, and *Pm1e* along with NC-AG12, Saluda, Jagger, and the powdery mildew susceptible control Chancellor (CI 12333) were evaluated in a detached leaf test as described by Perugini et al. (2008). Ten *Blumeria graminis f.sp. tritici* (*Bgt)* isolates collected from wheat fields in North Carolina were used for the evaluation. The isolates were chosen because of their ability to differentiate between the *Pm1* alleles and the germplasm line NC-AG12. Disease reactions were based on a resistant (R), intermediate (I), or susceptible (S) scale, in which resistant phenotypes had no visible signs of infection to small necrotic lesions, intermediate phenotypes had decreasing necrotic lesion area and conidial production

increased from slight to moderate, susceptible phenotypes had increasing amounts, size, and density of conidia to a fully compatible reaction.

Whole plant evaluations

All F_3 families, from the NC-AG12/Jagger population as well as parental lines were evaluated for their reactions to powdery mildew in the greenhouse. An experimental unit was two 10-cm pots each containing five seedlings per genotype under evaluation. The parental lines NC-AG12 and Jagger were included at 10-pot intervals as controls. Parental seeds were derived from selfed progenies of the plants used to produce the original cross. A randomized complete block design with two replications over time was utilized. The greenhouse was maintained at 24°C/20°C (day/night), and provided plentiful natural light supplemented with artificial high intensity discharge 1,000 W lights.

The NC-AG12/Saluda and NC-AG12/Axminster F_2 populations were planted in Ray Leach Cone-tainers (Stuewe and Sons, Inc. Tangent, OR, USA) and grown under the same greenhouse conditions as described above. One plant was grown per container and ten plants of the parental lines were included as controls in the evaluations.

For all greenhouse evaluations, plants were inoculated at the 2–3 leaf stage by gently shaking conidia from leaves of infected Saluda plants onto the leaves of the F_2 plants or F_3 families and controls. The inoculation source was *Bgt* culture 'Yuma', which is maintained by the USDA-ARS plant pathology laboratory at North Carolina State University. Disease evaluations were made 10–15 days post inoculation, or when Saluda or Jagger showed intense and uniform powdery mildew infection across the entire experiment. Disease severity scores were on a scale from 0 to 9 as described by Srnić et al. (2005). Briefly, 0–3 = resistant, immune, no visible signs of infection to a detectable amount of conidial pustules, 4–6 = intermediate, pustule production increased from slight to moderate; 7–9 = susceptible, with increasing amount, size, and density of pustules to a fully compatible reaction. F_2 plants and F_3 families with similar disease reactions to NC-AG12 were classified as 'resistant' and those similar to Jagger or Saluda were classified as 'susceptible'. F_3 families with both resistant and susceptible plants were classified as 'segregating'. Chi-square tests were conducted to test the goodness of fit between observed and expected segregation ratios (Snedecor and Cochran 1956).

Marker analysis

Genomic DNA was extracted from leaf tissue of the F_2 plants that gave rise to the F_3 families in the NC-AG12/Jagger population by the CTAB procedure described by Stein et al. (2001). DNA tracing to 10 homozygous resistant and 10 homozygous susceptible families was utilized for a bulked segregant analysis (BSA) with chromosome and genome specific SSR markers (Michelmore et al. 1991; Röder et al.1998; Somers et al. 2004; Xue et al. 2008). Markers that were polymorphic between the resistant/susceptible classes, NC-AG12, Jagger, and Saluda were identified as tentatively linked to the resistance gene and were subsequently used to genotype the population. All the individuals in the population were genotyped with SSR markers to develop a linkage map around the resistance gene indentified in NC-AG12. Amplification and visualization of the SSR markers were conducted according to Miranda et al. (2006). Wheat SSR primers were synthesized according to sequences published in GrainGenes (http://wheat.pw.usda.gov), with all forward primers modified to include the M13 sequence (CACGACGTTG TAAAACGAC-) at the 5′ end for labeling purposes (Schuelke 2000).

Chi-squared analyses were used to test for expected segregation of markers and linkage analysis was conducted with MAPMAKER/EXP (version 3.0b) (Lincoln et al. 1993). The Kosambi mapping function was used to estimate centimorgan (cM) distances between the markers. Maximum likelihood estimates confirmed map orders of the wheat consensus map (Somers et al. 2004).

Results

Inheritance of powdery mildew resistance

NC-AG12 had a mean disease severity score of 0, whereas Jagger and Saluda had mean disease severity scores of 9 in the greenhouse evaluations. The NC-AG12/Jagger F_3 lines were classified as 32 homozygous resistant, 73 segregating, and 23 homozygous susceptible $\left(\chi^2_{1:2:1} = 3.8,\ p = 0.15\right)$. The NC-AG12/Saluda F_2 plants were classified as 99 resistant and 26 susceptible $\left(\chi^2_{3:1} = 1.67,\ p = 0.28\right)$ These results indicated that resistance to *Bgt* culture Yuma was conferred by a single dominant gene in NC-AG12. All 979 plants in the NC-AG12/Axminster population were resistant and no susceptible plants were observed. When no recombinants are observed between two loci linked in repulsion in an F_2 population, the maximum recombination value (r) between the loci is estimated from:

$$r^2 = 4\left(1 - \sqrt[n]{\alpha}\right)$$

where n is the number of F_2 plants evaluated and α is the Type1 error rate (Hanson 1959). Our results indicated that the linkage between the *Pm1* locus and the resistance gene in NC-AG12 was not more than 11.1 cM ($P = 0.05$).

Molecular mapping

Twelve wheat SSR markers that mapped to the A and B genomes were used in the bulked segregate analysis. One marker, *Xwmc525,* was polymorphic between the two bulks and the controls. *Xwmc525* mapped to the long arm of chromosome 7A on the wheat consensus map of Somers et al. (2004). An additional nine markers, specific to chromosome 7AL, were tested and found to be polymorphic between the contrasting classes and parental lines.

The nine markers were genotyped on all F_2 individuals from the NC-AG12/Jagger population. *Xwmc346*, *Xwmc525*, *Xcfa2040*, and *Xwmc116* were co-dominant markers (Fig. 1). *Xgwm332, Xwmc273,* and *Xwmc809* were dominant markers linked in repulsion to the resistance allele. *Xwmc790* and *Xmag2185* were dominant markers linked in coupling to the resistance allele. All the markers that were placed on the linkage map were tested on Saluda to determine the most likely interval for the *T. timopheevii* introgression. The markers *Xcfa2040, Xmag2185, Xwmc525*, *Xwmc346*, and *Xwmc273* were polymorphic between NCAG12 and Saluda, indicating that the introgression was delimited by *Xcfa2040* and *Xwmc273*.

Linkage analysis showed that the resistance gene in NC-AG12 was flanked by markers *Xwmc346* and *Xwmc273* at distances of 6.6 cM and 8.3 cM, respectively (Fig. 1). The flanking markers did not deviate from their expected segregation ratios (Table 1). All the markers used in this study were previously mapped to the terminal deletion bin (0.86–1.00) of chromosome 7AL (Sourdille et al. 2004; Ji et al. 2008; Perugini et al. 2008).

Detached leaf test

Differential reactions were observed between NC-AG12 and lines that possessed *Pm1a, Pm1b, Pm1c, Pm1d, Pm1e,* Jagger, Chancellor, and Saluda, when inoculated with ten *Bgt* isolates (Table 2). NC-AG12 resistance was differentiated from all five *Pm1* alleles.

Discussion

One dominant gene for resistance to *Bgt* culture Yuma was identified in NC-AG12. The resistance was derived from an accession of tetraploid *T. timopheevii* and introgressed into a hexaploid winter wheat background adapted to the southeastern USA. The *T. timopheevii* introgression carrying the resistance gene was delimited by the SSR markers *Xcfa2040* and *Xwmc273* on the chomosome arm 7AL. It is proposed that this *T. timopheevii*-derived resistance genes be given the temporary designation *MlAG12*. NC-AG12 has shown consistent resistance over six seasons in the field evaluations in North Carolina (data not shown) and should be a good parent for use in cultivar development.

We believe a single resistance gene was introgressed into NC-AG12 during the backcross and selfing generations (Murphy et al. 2007). Nevertheless, the data presented in Table 2 do not preclude the existence of a second introgressed gene that was responsible for resistance to some of the isolates because Yuma, the isolate used in phenotyping the population, did not distinguish *MlAG12* from *Pm1a, Pm1b, Pm1c,* and *Pm1d* by itself.

Wheat chromosome 7AL is a 'hotspot' for powdery mildew resistance genes. *Pm1*, *Pm9*, *Pm37*, *mlRD30*, *PmU*, *Mlm2033*, *Mlm80*, *MlW72*, as well as two unnamed genes derived from *T. monococcum* are located on 7AL. Of these

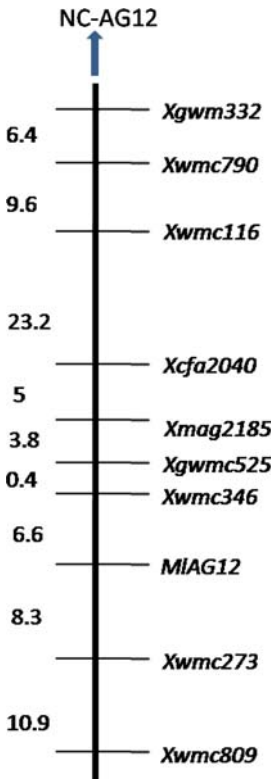

Fig. 1 Map positions of the wheat powdery mildew resistance gene *MlAG12* in NC-AG12 on chromosome 7AL. Marker names are at the *right* and the distances between markers in centimorgans (cM) are at the *left*

Table 1 Segregation ratios for SSR markers flanking the powdery mildew resistance gene *MlAG12* evaluated in NC-AG12/Jagger

Population	*Xwmc346*[a]					*Xwmc273*[b]			
	AA	H	BB	$\chi^2_{1:2:1}$	*P* value	AA	H/BB	$\chi^2_{3:1}$	*P* value
NC-AG12/Jagger	29	78	32	2.21	0.33	31	101	0.16	0.69

[a] *AA* homozygous for the NC-AG12 allele, *H* heterozygous, and *BB* homozygous for the Jagger allele

[b] *AA* homozygous for the NC-AG12 allele, *H/BB* not homozygous for the NC-AG12 (dominant marker for the Jagger allele)

Table 2 Reactions of genotypes possessing *Pm1* alleles, NC-AG12, Saluda, Jagger, and Chancellor to ten *Bgt* isolates

Cultivar/line	Pm gene	*Bgt* isolate									
		Arapahoe	Asosan	$E_3$14	Flat 7-11	85063	101a2	#8	127	ABK	Yuma
Axminster	*Pm1a*	R[a]	R	S	R	R	S	I	S	S	R
MocZlatka	*Pm1b*	R	R	S	R	R	I	R	S	S	R
M1 N	*Pm1c*	S	S	R	S	S	S	S	I	I	R
T. spelta	*Pm1d*	R	R	S	R	R	I	R	I	I	R
Ovest	*Pm1e*	S	S	S	I	S	S	S	S	S	S
NC-AG12	*MlAG12*	R	R	R	R	R	R	R	R	R	R
Saluda	*Pm3a*	S	S	I	S	S	R	R	S	S	S
Jagger	None	S	S	S	S	S	S	S	S	S	S
Chancellor	None	S	S	S	S	S	S	S	S	S	S

The virulence/avirulence combinations for the *Bgt* isolates are

Arapahoe: *Pm 1c, 1e, 2, 3a, 3c, 3e, 3 g, 4a, 5a, 5b, 5d, 6, 7, 9, 20, 34/Pm 1a, 1b, 1d, 3b, 3d, 3f, 4b, 8, 12, 16, 17, 21, 25, 37*

Asosan: *Pm 1c, 1e, 3a, 3b, 3c,3d, 3e, 3 g, 4a, 5a, 5b,6, 7, 9, 17, 20, 34/Pm 1a, 1b, 1d, 2, 3f, 4b, 5d, 8, 12, 16, 21, 25, 37*

$E_3$14: *Pm 1a, 1b, 1d, 1e, 3a, 3b, 3c, 6, 12, 13, 16, 19, 20, 21, 25/Pm 1c, 4a, 4b, 5, 7, 8, 17*

Flat 7-11: *Pm 1c, 1e, 2, 3a, 3b, 3c, 3e, 3 g, 4a, 5a, 5b, 5d, 6, 7, 9, 17, 20, 34/Pm 1a, 1b, 1d, 3d, 3f, 4b, 8, 12, 16, 21, 25, 37*

85063: *Pm 1c, 1e, 3a, 3d, 3e, 3f, 3 g, 5b, 5d, 7, 8, 9/Pm1a, 1b, 1d, 2, 3b, 3c, 4a, 4b, 5a, 6, 12, 16, 17, 20, 21, 25, 34, 37*

101a2: *Pm 1a, 1b, 1c, 1d, 1e, 3b, 3c, 3d, 3e, 3f, 3 g, 4a, 4b, 5a, 5b, 6, 7, 8,9, 17, 20/Pm 2, 3a, 5d, 12, 16, 21, 25, 34,37*

#8: *Pm 1a, 1c, 1e, 2, 3b,12, 13, 16, 20, 21, 25/Pm 1b, 1d, 3a, 4a, 4b, 5, 6, 7, 17, 19*

127: *Pm 1a, 1b, 1c, 1d, 1e, 2, 3a, 3c, 3f, 4a, 4b, 5a, 6, 7, 8, 17/Pm 3b*

ABK: *Pm 1a, 1b, 1c, 1d, 1e, 2, 3a, 3b, 3c, 3f, 5a, 6, 7, 8/Pm 4a, 4b, 17*

Yuma: *Pm 1e, 2, 3a,3c,3f, 3 g, 5a, 5b, 6, 7, 8, 9, 20, 34/Pm 1a, 1b, 1c, 1d, 3b, 3d, 4a, 4b, 12, 16, 17, 21, 25,37*

[a] *R* resistant, *I* intermediate, and *S* susceptible

loci, *Pm1* is the most complex with multiple dominant alleles *Pm1a, Pm1b, Pm1c, Pm1d,* and *Pm1e* (Hsam et al. 1998). Of these, *Pm1a, Pm1c*, and *Pm1e* were identified in *T. aestivum* (Sears and Briggle 1969; Hsam et al. 1998; Singrün et al. 2003). The alleles *Pm1c* and *Pm1d* were introgressed from *T. monococcum* and *T. spelta* var. *duhamelianum*, respectively (Hsam et al. 1998). The *Pm1* locus mapped close to the SSR marker *Xgwm344*, placing it in the terminal deletion bin (0.86–1.00) of chromosome 7AL (Singrün et al. 2003; Ji et al. 2008; Perugini et al. 2008). *Pm37*, a *T. timopheevii*-derived gene, was located about 16.2 cM proximal to *Pm1* (Perugini et al. 2008). The resistance gene in NC-AG12 is unlikely to be an allele of the *Pm37* locus because the markers flanking *Pm37*, *Xwmc790*, and *Xgwm332,* were not polymorphic between NC-AG12 and Saluda. This indicated that the *T. timopheevii* introgression in NC-AG12 was distal to the introgression for *Pm37*. Moreover, *Xwmc790* mapped 48.6 cM proximal to *MlAG12* (Fig. 1).

The recessive resistance gene *Pm9* was identified in the spring wheat cultivar Normandie and mapped 8.5 cM distal to the *Pm1* locus (Schneider et al. 1991). The *Pm9* gene was ineffective in the field evaluations in North Carolina and cv. Normadie exhibited a susceptible response (data not shown). Another recessive resistance gene, *mlRD30*, identified in the common wheat line TA2682a mapped 1.8 cM distal to the *Pm1* locus or marker *Xgwm344* (Singrün et al. 2004). The *T. urartu*-derived resistance gene *PmU* mapped to a similar region on chromosome 7AL located 2.2 cM distal to the SSR marker *Xwmc273* (Qiu et al. 2005). Two other *T. monococcum*-derived resistance genes *Mlm2033* and *Mlm80* were fine-mapped and found to be tightly linked to marker *Xmag2185* at less than 1 cM (Yao et al. 2007). The marker *Xmag2185* mapped in the same region as the *Pm1* locus. Recently, Ji et al. (2008) found the *T. dicoccoides*-derived resistance gene *MlIW72* mapped 3.3 cM proximal to the marker *Xmag2185* and 4.9 cM proximal to *Xgwm344*. Taken together, these reports suggested that the chromosome region in the vicinity of the *Pm1* locus is rich in disease resistance genes. However, because of the close proximity of the markers, it remains unclear whether these genes represent a series of different loci, or are alleles at a single locus.

The identification and introgression of novel genes for powdery mildew resistance, and the identification of molecular markers linked to them, are key to the future genetic control of wheat powdery mildew. Virulence surveys in the eastern US have shown that the pathogenic structure of the *Bgt* population continues to evolve. More complex pathogen virulence patterns have been identified that

overcome widely deployed resistance genes (Niewoehner and Leath 1998; Parks et al. 2008). Host gene pyramiding is a common breeding strategy that should result in more effective genetic control. Marker-assisted selection is a key component of this approach, because it is effective in identification of genotypes with multiple genes, especially when virulences for the individual resistance genes are not present. Marker-assisted selection will not only facilitate the gene pyramiding, but it can also reduce the amount of non-target genetic background (Bonnett et al. 2005). This study details a new source of powdery mildew resistance in an adapted background ready for use in wheat breeding programs in the southeastern US. The gene reported herein has the potential to be pyramided with powdery mildew genes at other loci, such as *Pm34, Pm35*, and *Pm37,* which are available in similar, adapted, genetic backgrounds (Miranda et al. 2006, 2007; Perugini et al. 2008).

Future prospects for breeding powdery mildew resistant wheat appear excellent, given the diverse approaches that can be employed based on recent research. Besides the numerous qualitative resistance genes that have been mapped, several QTL controlling quantitative adult plant resistances have been identified also (Keller et al. 1999; Tucker et al. 2007; Lillemo et al. 2008). Cultivars with quantitative resistance alone or quantitative plus qualitative resistance for environments where seedling resistance is important can be readily developed.

Acknowledgments This research was supported, in part, by the North Carolina Small Grain Growers Association. We thank Dr. R. A. McIntosh for his excellent insights and suggestions during the final draft of this manuscript.

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