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ORIGINAL PAPER

Molecular characterization of a new powdery mildew resistance gene *Pm54* in soft red winter wheat

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Abstract
Key message **A new powdery mildew resistance gene *Pm54* was identified on chromosome 6BL in soft red winter wheat**.
Abstract Powdery mildew is causing increasing damage to wheat production in the southeastern USA. To combat the disease, a continuing need exists to discover new genes for powdery mildew resistance and to incorporate those genes into breeding programs. Pioneer® variety 26R61 (shortened as 26R61) and AGS 2000 have been used as checks in the Uniform Southern Soft Red Winter Wheat Nursery for a decade, and both have provided good resistance across regions during that time. In the present study, a genetic analysis of mildew resistance was conducted on a RIL population developed from a cross of 26R61 and AGS 2000. Phenotypic evaluation was conducted in the field at Plains, GA, and Raleigh, NC, in 2012 and 2013, a total of four environments. Three quantitative trait loci (QTL) with major effect were consistently detected on wheat chromosomes 2BL, 4A and 6BL. The 2BL QTL contributed by 26R61 was different from *Pm6*, a widely used gene in the southeastern USA. The other two QTL were identified from AGS 2000. The 6BL QTL was subsequently characterized

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as a simple Mendelian factor when the population was inoculated with a single *Blumeria graminis* f. sp. *tritici* (*Bgt*) isolate in controlled environments. Since there is no known powdery mildew resistance gene (*Pm*) on this particular location of common wheat, the gene was designated *Pm54*. The closely linked marker *Xbarc134* was highly polymorphic in a set of mildew differentials, indicating that the marker should be useful for pyramiding *Pm54* with other *Pm* genes by marker-assisted selection.

Introduction

Powdery mildew, caused by the obligate fungus *Blumeria graminis* f. sp *tritici* (*Bgt*), is one of the most damaging diseases of wheat (*Triticum aestivum* L.). The pathogen can attack all above-ground wheat parts including leaves, stems, and spikes. The disease commonly occurs at economically damaging levels in the southeastern USA, where the climate is maritime or semi-continental (Cowger et al. 2012). Yield losses from powdery mildew ranging from 17 to 34 % have been reported in these regions (Johnson et al. 1979; Leath and Bowen 1989). Although fungicides controlling the disease are available, deployment of resistant cultivars remains the most reliable, economical, and environmentally safe approach to control this disease (Bennett 1984).

To date, about 77 formally designated *Pm* genes have been cataloged at 49 loci (*Pm1–Pm53*, *Pm18* = *Pm1c*, *Pm22* = *Pm1e*, *Pm23* = *Pm4c*, *Pm31* = *Pm21*) with the loci *Pm1*, *Pm3*, *Pm4*, *Pm5* and *Pm24* having 5, 17, 4, 5 and 2 alleles, respectively (Hao et al. 2008; Hsam et al. 1998; McIntosh et al. 2013, 2014; Singrün et al. 2003; Xie et al. 2012a). In the soft red winter wheat (SRWW) growing region of the southeastern USA, cultivars having *Pm3a*, *Pm8* and *Pm17* are widely grown even though the effectiveness of these resistance genes is limited as virulent *Bgt* isolates are now common in the field (Cowger et al. 2009; Hao et al. 2012a). The gene *Pm6* that was transferred to common wheat lines 'CI 12632' and 'CI 12633' from tetraploid *T. timopheevii* (Allard and Shands 1954; Jørgensen and Jensen 1973) has also been widely deployed in these areas. The cultivars 'Arthur', 'Arthur 71', 'Abe' and other Arthur-type wheat or their derivatives were all supposed to possess *Pm6* or the combination *Pm2* + *Pm6* (Bennett 1984). The recessive gene *Pm5a*, originally derived from Yaroslav emmer via the hard spring wheat variety 'Hope' (Lebsock and Briggle 1974; McFadden 1930), was also common in the SRWW region, since Hope was used extensively as a parent in wheat breeding programs (Bennett 1984). In addition, cultivar 'Roane' was assumed to have inherited *Pm4a* from 'IN65309C1-18-2-3-2', a line developed by Purdue University (Griffey et al. 2001), and 'NC-Neuse' probably possessed *Pm1a* from CI 13868 (Murphy et al. 2004). The genes *Pm3b*, *Pm3e*, and *Pm3f* were also reported in SRWW based on amplification of *Pm3* allele-specific functional markers (Tommasini et al. 2006), but in low frequencies compared with the more common *Pm3a* allele (Uniform Eastern and Southern Soft Red Winter Wheat Nursery Reports, http://www.ars.usda.gov/Main/docs.htm?docid=21894). Apart from the formally named genes used in these regions, quantitative trait loci (QTL) located on chromosomes 1BL, 2AL and 2BL were also reported in the SRWW cultivar 'Massey' and its derivative 'USG 3209' (Liu et al. 2001; Tucker et al. 2007).

Bgt isolates in the southeastern USA usually have the highest levels of genetic diversity in the country, including isolates present in the states of North Carolina (NC) and Georgia (GA) (Parks et al. 2008). Some *Pm* genes start to lose effectiveness even before commercial deployment. For example, little or no virulence to *Pm12* was detected in this region in the early 1990s (Niewoehner and Leath 1998), but in 2005 a low frequency of *Pm12* virulence was observed among NC isolates, an intermediate level among Virginia isolates and a high level among GA isolates (Parks et al. 2008). To the best of our knowledge, *Pm12*, derived from *Aegilops speltoides* (Jia et al. 1996), has never been used in commercial wheat cultivars. To meet the challenges of pathogen diversity and increasingly complex virulent *Bgt* isolates, there is an urgent need to discover new genes or QTL for *Pm* resistance and promptly incorporate them into breeding materials.

The SRWW cultivars, 'AGS 2000' and 'Pioneer® variety 26R61' (shortened as 26R61), were both released in 2000 and have been used as checks in the Uniform Southern Soft Red Winter Wheat Nursery (USSRWWN) for more than a decade. Both cultivars have generally exhibited good resistance to *Bgt* in the field across locations and years. In seedling tests conducted in 2001–2002, AGS 2000 was resistant or moderately resistant to 27 of 30 *Bgt* isolates, and 26R61 was resistant or moderately resistant to 28 of the same isolates (USSRWWN Reports, http://www.ars.usda.gov/Main/docs.htm?docid=21894). However, information on the genetic basis of powdery mildew resistance in the two cultivars is limited. Hao et al. (2012a) reported that both 26R61 and AGS 2000 possessed the 1BL.1RS translocation and therefore *Pm8*; AGS 2000 also has *Pm3a*. As *Pm3a* has lost its effectiveness in southeastern USA and *Pm8* is becoming less effective in the states of GA and NC (Parks et al. 2008), other *Pm* genes must be conferring resistance in these cultivars. The primary objective of the present study was to determine the unknown factors for mildew resistance in the two cultivars using a RIL population with good genome coverage of molecular markers.

Materials and methods

Plant materials

Two SRWW cultivars, 26R61 (PI 612153) and AGS 2000 (PI 612956), were crossed and 178 $F_{7:8}$ recombinant inbred lines (RILs) were developed by single-seed descent (Hao et al. 2011). Cultivar 26R61 (Omega 78/S76/Arthur 71/3/Stadler//Redcoat/Wisconsin 1/5/Coker 747/6/PIO2555 sib) was developed by Pioneer Hi-Bred, and AGS 2000 (PIO2555/PF84301//Florida 302) was developed and released jointly by the University of Georgia and University of Florida (Johnson et al. 2002). The population name was abbreviated as PR61/A2000. Three lines (42, 149 and 172) were omitted from mapping and QTL analysis due to high percentages of missing molecular data.

The cultivars Arthur, Arthur 71, Coker 747 and Coker 68-15 were included in the study. Coker 747 (Arthur/Coker 68-15) is a *Pm6* differential line used in the USDA Powdery Mildew Differential Collection and Resistance Nursery at Raleigh, NC. *Pm6* was presumably inherited from Arthur. Coker 68-15 was used as a negative check, and was assumed to have no *Pm* gene (Shi et al. 1998). Arthur 71 (thought to have *Pm6*) was a five-time backcross derivative of Arthur and a parent of the cultivar 26R61.

A set of *Pm* differentials including 39 formally named *Pm* genes, 5 temporarily named genes, and 2 susceptible checks (Chancellor and Coker 68-15) was also included to test the polymorphism of markers closely linked with the newly identified gene. All the germplasm are maintained by the USDA-ARS Plant Science Research Unit, North Carolina State University, Raleigh, NC, USA.

Evaluation of powdery mildew response in the field

The PR61/A2000 RILs and parents were evaluated for mildew response under natural infection in the field at Plains, GA, and Raleigh, NC, in 2011–2012 and 2012–2013, hereafter referred to as 2012 and 2013. Plants were grown in randomized complete blocks with two replications in Plains and three replications in Raleigh. Both parents were randomly interspersed with the population five to ten times each depending upon environment.

Powdery mildew response was assessed at the adult-plant stage when the most susceptible checks (Saluda or Chancellor) approached maximum disease severities. A numeric 0–9 scale was adopted based on disease severity (DS), where 0 indicated immunity, DS = 0; 1, 0 < DS ≤ 10 %; 2, 10 % < DS ≤ 20 %; 3, 20 % < DS ≤ 30 %; 4, 30 % < DS ≤ 40 %; 5, intermediate type, 40 % < DS < 60 %; 6, 60 % ≤ DS < 70 %; 7, 70 % ≤ DS < 80 %; 8, 80 % ≤ DS < 90 %; 9, full susceptibility, DS ≥ 90 %. This method provides a fast and repeatable way of scoring powdery mildew response (Bennett and Westcott 1982), and has been routinely used in regional screening nurseries. For each environment, mean response values over the replications were used for QTL analysis.

Single conidium isolation

In the spring of 2012, a severe powdery mildew epidemic occurred in the field at Plains, GA. Cultivar AGS 2000 was nearly immune to the disease and 26R61 was moderately resistant. Leaves with fresh conidiospores were collected from 26R61 on April 13, 2012, and cultured in a growth chamber maintained at 17 °C, relative humidity at least 70 %, and a 12:12 h (light:dark) photoperiod. Conidiospores were isolated three times from a single conidium according to the protocol described by Namuco et al. (1987) with minor modifications. Briefly, one pot of 26R61 covered with a plastic bag was placed in a *Bgt*-free growth chamber. The plants were inoculated at the three-leaf stage with the inoculum collected in the field. After about 10 days, when new conidiospores were visible, a small leaf segment with a single-colony conidium was cut out, and the conidiospores were shaken onto 26R61 seedlings in another pot and covered with a plastic bag. The isolation was repeated twice to increase the probability of avoiding an isolate mixture. The resulting single-conidial isolate, designated PL-12 (Plains in 2012), was avirulent to AGS 2000 and virulent to 26R61. It was increased and maintained on 26R61 seedlings and used to inoculate the entire PR61/A2000 population.

Evaluation of powdery mildew response in growth chamber

Inoculation of isolate PL-12 onto the RIL population was conducted in two separate growth chambers, with two replicates of the population in each chamber. About ten seeds of each line were planted in a 15 cm pot. The pots in each replicate were randomly distributed in the growth chamber. AGS 2000 and 26R61 were included at 20 pot intervals as resistant and susceptible controls, respectively. The growth chamber was programmed the same as the chamber used in the single conidium isolation. Plants were inoculated at the three-leaf stage by shaking conidiospores from susceptible 26R61 plants onto the test seedlings.

Reactions were scored 15–20 days after inoculation when the susceptible parent 26R61 was heavily infected, and then repeated once a week later. Five major classes of infection types (ITs) were categorized: 0 (resistant), no visible symptoms or a few flecks; 1 (moderately resistant), necrosis with low to medium sporulation; 2 (segregating), both resistant and susceptible plants observed; 3 (moderately susceptible), no necrosis with medium to high

sporulation; and 4 (susceptible), no necrosis with full sporulation. In later mapping of the resistance gene, IT 0 and 1 classes were pooled as resistant; those with 3 and 4 as susceptible; and those with 2 as segregating.

Data analysis and QTL mapping

The SAS 9.1 statistical package was used for statistical analysis and output of histograms (SAS Institute, Cary, NC, USA). The genetic linkage maps used for QTL analysis were described by Hao et al. (2012b) with updates of QTL target regions in the present study. Two SNP markers from a 9 k iSelect Beadchip Assay were also added near the major QTL region on 6BL (Cavanagh et al. 2013). The maps included 972 loci on 24 linkage groups, with gaps for chromosomes 2A, 4D, and 7D, and spanned 2,757 cM, with 1,125, 916, and 716 cM in the A, B, and D genomes, respectively.

QTL detection was conducted in Windows QTL Cartographer 2.5 as follows: the composite interval mapping (CIM) method was used; walk speed was set as 1.0 cM and the control parameters were default; and the LOD (logarithm of odds) threshold was set as 3.0. To declare significance levels, LOD scores were calculated from 1,000 permutations for each trait at $P = 0.05$, 0.01 and/or 0.001, respectively (Wang et al. 2012). QTL designation referred to the guidelines for nomenclature of QTL in wheat (McIntosh et al. 2013). The function 'effectplot' in program R/qtl was used to create effect plots of phenotypes against genotypes at selected loci (Broman et al. 2003).

Molecular marker analysis

As a major QTL had been detected at a location near *Pm6* on chromosome 2BL, to clarify the relationship of the two resistance sources, genotyping was conducted on 26R61 and AGS 2000 with the *Pm6* diagnostic marker $NAU/STS_{BCD135\text{-}2}$ (Ji et al. 2008).

In addition, the simple sequence repeat (SSR) marker *Xbarc134*, which was closely linked with another major QTL on chromosome 6BL, was used to genotype a set of differential lines. PCR was performed using a touchdown program described by Hao et al. (2008), and amplified products were separated in a 6 % (w/v) polyacrylamide gel using a Mega-Gel High Throughput Vertical Unit, following the procedure reported by Wang et al. (2003).

Results

Phenotypic analysis

The parent AGS 2000 was highly resistant in the field in Plains in 2012 with an average score of 0.05 on the 0–9 scale. The mean score of parent 26R61 was 1.90. The difference between the two values was highly significant ($P < 0.001$), but there was no significant difference in the other three environments (Raleigh 2012, Plains 2013 and Raleigh 2013) (Fig. 1). For RILs at the adult-plant stage, the rating data were continuous and the distribution deviated significantly from a normal distribution ($P < 0.01$) in all environments except in Raleigh 2012 (Fig. 1), suggesting that both major and minor mildew resistance QTL might be involved. Transgressive segregation (Fig. 1) implied that both parents possessed favorable additive allele(s).

For the powdery mildew resistance in the seedling stage, AGS 2000 was uniformly resistant (IT = 0) to the *Bgt* isolate PL-12, and 26R61 was fully susceptible (IT = 4). Most of the RILs had ITs of 0 or 4; a few lines were 1 or 3; some segregating lines (IT = 2) were also observed and presumed to be heterozygous (Fig. 2).

QTL detection for powdery mildew resistance in the field

Three QTL of major effect located on chromosomes 2BL, 4A and 6BL were detected in all environments on the basis of whole-genome scanning and the CIM analysis (Supplementary Fig. S1). Together, these QTL explained 37–46 % of the phenotypic variation across locations (Table 1).

The 2BL QTL, with resistance contributed by 26R61, was designated *QPm.uga-2BL*; it was closely linked to marker *wPt-0694* (Fig. 3; Table 1). This QTL explained approximately 6 % of phenotypic variation in Plains, and 12–18 % in Raleigh; the peak LOD values were highly significant ($P < 0.001$) in Raleigh in both years, but only suggestive in Plains (Table 1).

The 4A QTL (*QPm.uga-4A*) explained 8–18 % of phenotypic variation and the 6BL QTL (*QPm.uga-6BL*) explained 7–30 %. Peak LOD values for both the 4A and 6BL QTL were significant in all environments (Table 1). *QPm.uga-4A* was closely linked with marker *wPt-3515* (Fig. 4a) and *QPm.uga-6BL* was closely linked with marker *Xbarc134* (Fig. 4b). The mean mildew severity for the lines with the *AA* genotype (26R61) was lower than those lines with the *BB* genotype (AGS 2000) at the *wPt-0694* locus on 2BL, but higher at the loci *wPt-3515* and *Xbarc134* on chromosomes 4A and 6BL, respectively, indicating that both the 4A and 6BL QTL were contributed by AGS 2000 (Fig. 5). In addition, two smaller QTL (*QPm.uga-4B* and *QPm.uga-5A*) were detected in Raleigh 2012 (Table 1).

Identification of a major gene for resistance to isolate PL-12 in AGS 2000

When seedlings of the RIL population were inoculated with *Bgt* isolate PL-12 in growth chambers, only

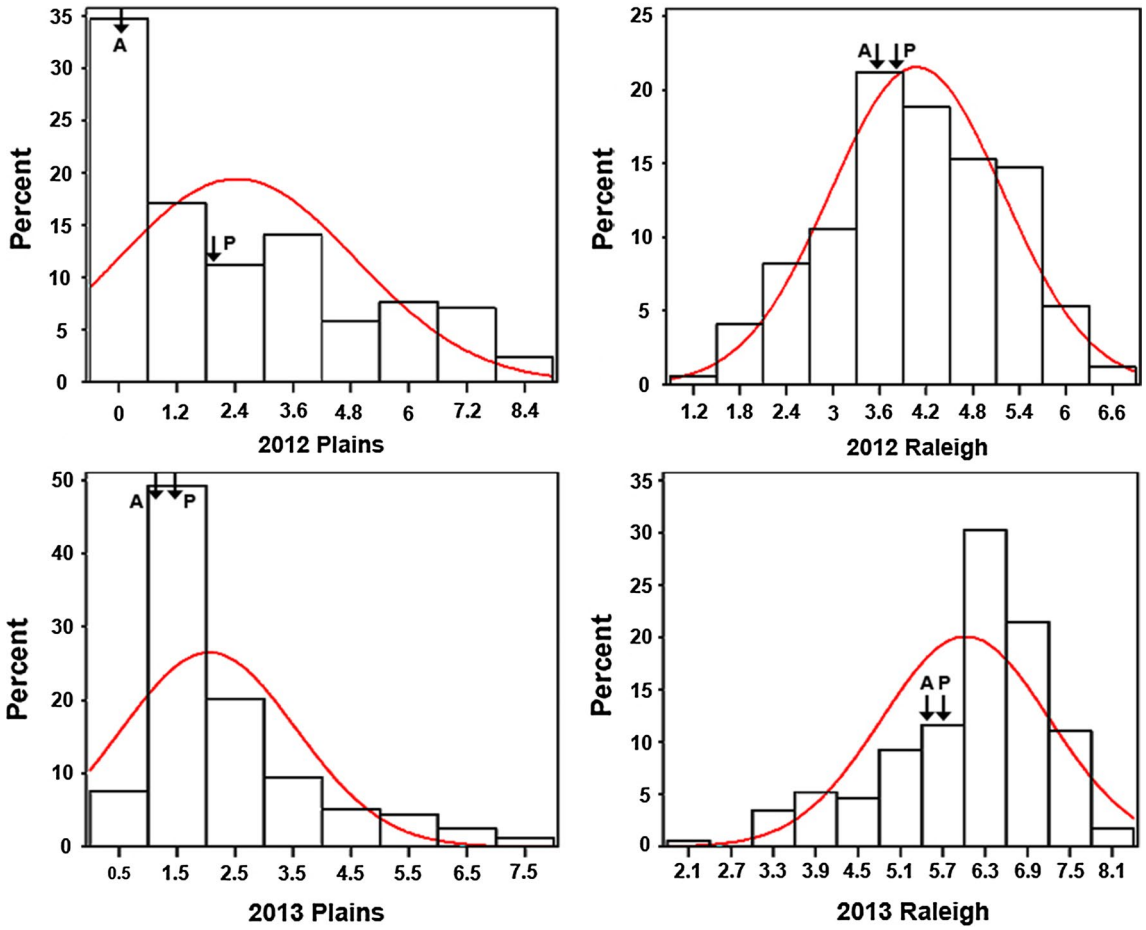

Fig. 1 Distribution of powdery mildew severity ratings on the PR61/A2000 wheat RIL population grown in four field environments (2012 Plains, 2012 Raleigh, 2013 Plains and 2013 Raleigh); *A*, AGS 2000; *P*, 26R61; the *curved lines* are the normal distribution curves

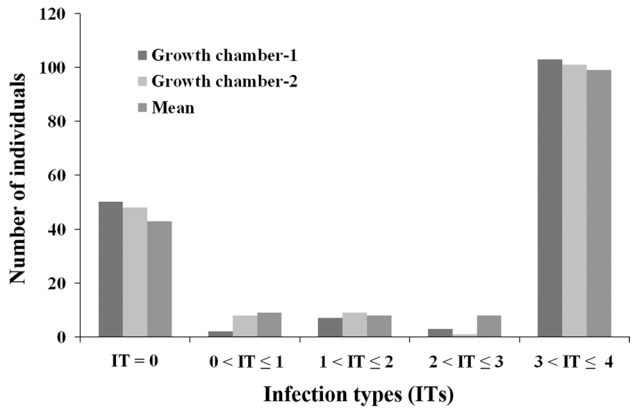

Fig. 2 Distribution of powdery mildew infection types (0–4 scale, with 0 being immune and 4 being fully susceptible) in growth chamber inoculations of the PR61/A2000 RIL population

one major QTL was identified based on whole-genome scanning (Supplementary Fig. S2). This QTL on 6BL was very closely linked with the marker *Xbarc134*, and was presumed to be the same as *QPm.uga-6BL* detected in field environments. The QTL explained up to 78 % of the total phenotypic variation in the controlled environment, with very high LOD values at the peak position (Supplementary Fig. S3, Table S1) suggesting the action of a single major gene. Following pooling of the IT 0 and 1 classes as resistant (AGS 2000 genotype), the IT 3 and 4 groups as susceptible (26R61 genotype), and those classified IT 2 as segregating, the resistance gene in AGS 2000 was definitively placed on the genetic map of 6BL (Fig. 6). This gene was temporarily designated *PmA2K*.

Relationship between *QPm.uga-2BL* and *Pm6*

As this study had identified a major QTL on 2BL close to *Pm6*, and *Pm6* is widespread in southeastern USA wheat germplasm, a need exists to clarify the relationship of the two resistance sources. The *Pm6* diagnostic marker $NAU/STS_{BCD135\text{-}2}$ produced the typical

Table 1 Position and effect of QTL for wheat powdery mildew resistance in field environments based on composite interval mapping analysis of a recombinant inbred line population from 26R61 × AGS 2000

Environment	QTL name[a]	Marker or marker interval	Position (cM)	Peak LOD	Peak position (cM)	R^2 (%)[b]	Additive effect[c]
2012 Plains	***QPm.uga-2BL***	*wPt-0694–Xbarc332*	76.1–76.6	4.5	76.5	6	−0.61
	QPm.uga-4A	*tPt-4753–wPt-3515*	0.5–7.6	6.3*	6.5	10	0.78
	QPm.uga-6BL	*Xbarc134–Ku_c28854_38769308*	191.4–196.7	17.2***	191.7	30	1.37
2012 Raleigh	***QPm.uga-2BL***	*wPt-0694*	76.1	10.5***	76.1	18	−0.57
	QPm.uga-4A	*tPt-4753–wPt-3515*	0.5–7.6	4.5**	4.1	8	0.38
	QPm.uga-4B	*Xgpw7272–Xgdm61*	36.5–39.8	3.8*	39.6	6	−0.35
	QPm.uga-5A	*wPt-3563–wPt-665622*	96.6–97.6	3.9*	96.7	6	0.33
	QPm.uga-6BL	*Xbarc134–Ku_c28854_38769308*	191.4–196.7	6.3**	192.5	15	0.51
2013 Plains	***QPm.uga-2BL***	*wPt-0694*	76.1	3.6	76.1	6	−0.39
	QPm.uga-4A	*tPt-4753–wPt-3515*	0.5–7.6	8.8**	5.5	18	0.66
	QPm.uga-6BL	*Xbarc134–Ku_c28854_38769308*	191.4–196.7	7.6**	192.5	15	0.61
2013 Raleigh	***QPm.uga-2BL***	*wPt-0694*	76.1	7.5***	76.1	12	−0.51
	QPm.uga-4A	*tPt-4753–wPt-3515*	0.5–7.6	9.5***	5.6	18	0.61
	QPm.uga-6BL	*Xbarc134–Ku_c28854_38769308*	191.4–196.7	3.9**	193.4	7	0.39

* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$

[a] Stable QTL identified in all environments are shown in bold

[b] Percent of phenotypic variation associated with the QTL

[c] Positive or negative values indicate that the alleles were inherited from AGS 2000 or 26R61, respectively

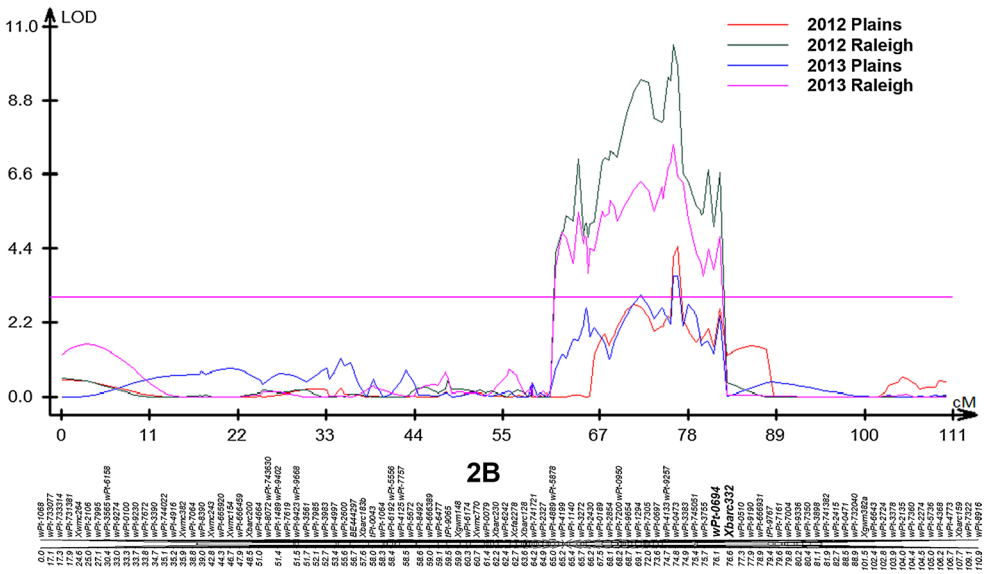

Fig. 3 A major-effect quantitative trait locus (QTL) for powdery mildew resistance was identified on chromosome 2BL of wheat cultivar 26R61 grown in four field environments (Plains, GA, and Raleigh, NC in 2012 and 2013). QTL flanking markers are in *bold*

amplification pattern shown in Fig. 7; *Pm6*-specific bands were amplified in the *Pm6* differential line Coker 747, but not in the susceptible check Coker 68-15. Cultivars Arthur and Arthur 71 yielded the same amplification as Coker 747, indicating that both possessed *Pm6*. The parents 26R61 and AGS 2000 were both negative for the marker, indicating that *Pm6* was not present in these cultivars, and that *QPm.uga-2BL* is different from *Pm6* (Fig. 7).

Genotyping differentials with marker *Xbarc134*

Marker *Xbarc134* tightly linked to the *PmA2K* was highly polymorphic between AGS 2000 (the source of *PmA2K*) and a set of *Pm* differentials (Supplementary Fig. S4, Table 2). No polymorphism was identified between AGS 2000 and the cultivars or lines 81–7241 (*Pm4c*), Coker 747 (*Pm6*), Amigo (*Pm17*), TAM W-104 (*Pm?*), L501 (*Pm32*), CH5025 (*Pm43*), and NC09BGTUM15 (*MlUM15*), or the

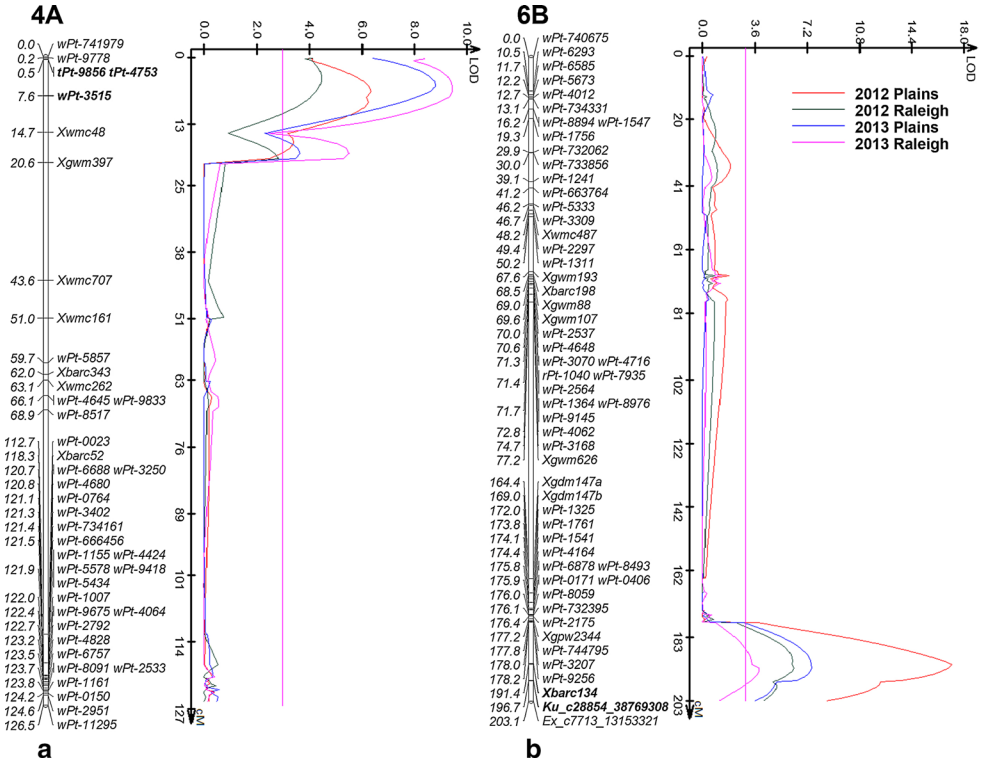

Fig. 4 Major-effect QTL for powdery mildew resistance were identified on **a** chromosome 4A and **b** chromosome 6BL of wheat cultivar AGS 2000 grown in four field environments (Plains, GA, and Raleigh, NC in 2012 and 2013). The QTL flanking markers are in *bold*

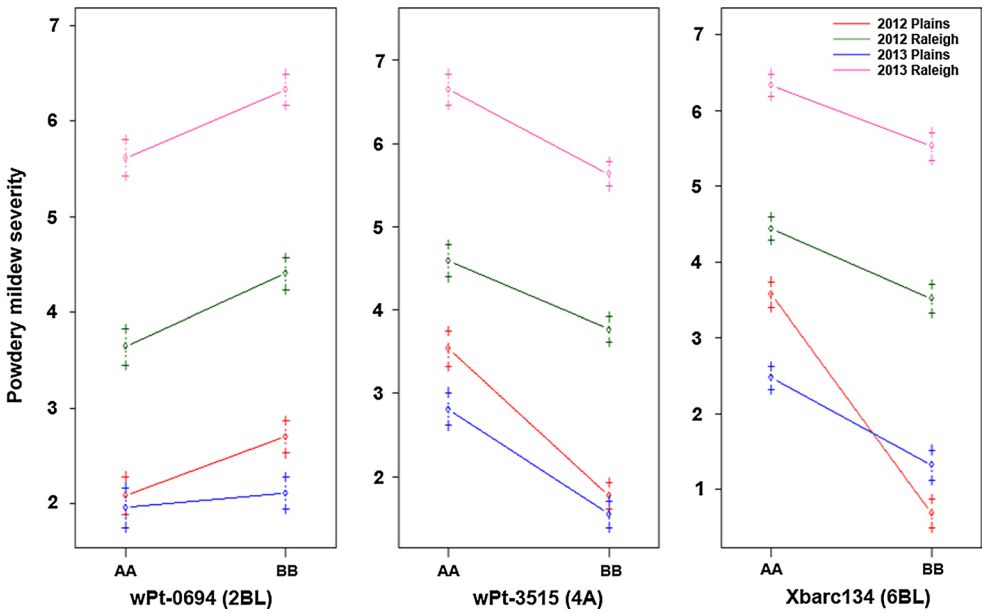

Fig. 5 Effect plots of three markers closely linked to QTL for powdery mildew resistance in wheat; *wPt-0694* is on chromosome 2BL, *wPt-3515* on chromosome 4A and *Xbarc134* on chromosome 6BL. *AA* represents the allele from 26R61, *BB* represents the allele from AGS 2000. Mildew severity ratings are on a 0–9 scale; 0 = immunity, 9 = maximum susceptibility. *Error bars* are ±1 SE

difference was too minor to detect using 6 % (w/v) polyacrylamide gels. In addition, Chancellor produced the same amplification pattern as CI 14114 (*Pm1a*), CI 14118 (*Pm2*), CI 14120 (*Pm3a*), CI 14121 (*Pm3b*), CI 14122 (*Pm3c*), CI 15888 (*Pm3f*), CI 14124 (*Pm4a*) and CI 14125 (*Pm5a*). This was expected due to the near-isogenic relationships of these differential lines and the differing locations of the respective *Pm* genes from 6BL. Similarly, because of the common recurrent parent Saluda, the same amplification pattern was found in differentials NC96BGTA5 (*Pm25*), NC97BGTD7 (*Pm34*), NC96BGTD3 (*Pm35*), NC99BGTAG11 (*Pm37*), NC96BGTA4 (*Pm-NCA4*) and NC96BGTA6 (*Pm-NCA6*), as well as Coker 68-15 (none), a parent of Saluda. However, NC06BGTAG12 (*MlAG12*), NC06BGTAG13 (*MlAG13*) and NC09BGTUM15 (*MlUM15*) had different patterns, indicating that during backcrossing,

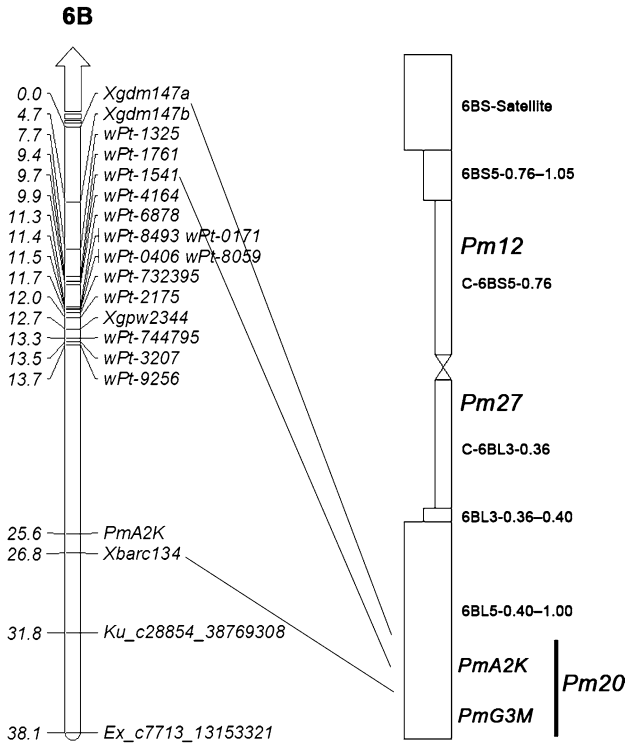

Fig. 6 Genetic map and physical location of powdery mildew resistance QTL *PmA2K* on the long arm of wheat chromosome 6B; deletion bins are indicated as intervals on the physical map (e.g. 0.40–1.00). *Pm20* from *Secale cereale* L. is on the distal third of the long arm of chromosome 6R in a T6BS.6RL translocation

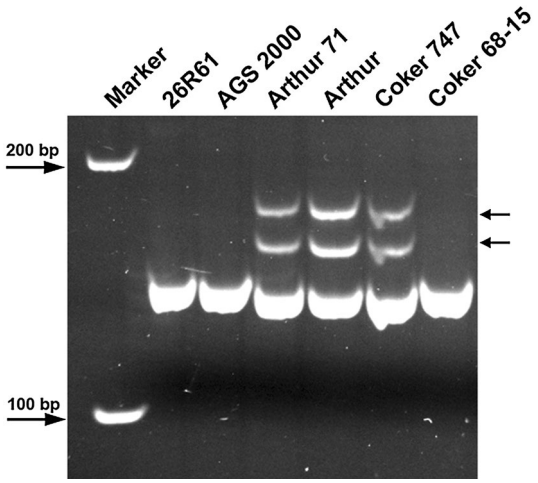

Fig. 7 PCR amplifications pattern of *Pm6* diagnostic marker *NAU/STS*$_{BCD135\text{-}2}$ for wheat RIL parents 26R61 and AGS 2000, as well as wheat cultivars Arthur 71, Arthur, Coker 747 and Coker 68-15

Saluda chromatin at the *Xbarc134* locus was replaced by that of the respective *Pm* donor parents (Supplementary Fig. S4, Table 2).

Discussion

Since their release, the soft red winter wheat cultivars AGS 2000 and 26R61 have exhibited moderate to high levels of resistance to powdery mildew at locations in the southeastern USA. In the present study, the factors in 26R61 and AGS 2000 contributing to powdery mildew resistance in the field were revealed. Three QTL, one from 26R61 on chromosome 2BL and the other two from AGS 2000 on chromosomes 4A and 6BL, were stably detected across environments. The fact that both parents contributed resistance QTL probably accounted for the transgressive segregation in the RIL population. Based on the LOD values and percentages of phenotypic variation explained, *QPm.uga-2BL* played a key role in mildew resistance at Raleigh; *QPm.uga-6BL* contributed significantly at Plains, particularly in 2012; and *QPm.uga-4A* was a major determinant of resistance at both locations.

The chromosome 2BL QTL was closely linked with the marker *wPt-0694*, and flanked by markers *wPt-3755* and *wPt-0510*, both located in the distal 11 % of wheat chromosome 2BL in physical map (Wilkinson et al. 2012). *QPm.uga-2BL* was further assigned to deletion bin 2BL6-0.89–1.00. Within that deletion bin or nearby, three formally named *Pm* genes (*Pm6*, *Pm33* and *Pm51*, the last is unpublished) and three temporarily named powdery mildew resistance genes (*MlZec1*, *MlAB10* and *PmJM22*) have been identified, as well as QTL detected in Massey or its derivative USG 3209, 'RE9001', 'Fukuho-Komugi', 'Lumai 21' and 'Naxos' (Cowger et al. 2012; Lu et al. 2012; McIntosh et al. 2013, 2014). Erayman et al. (2004) suggested that the distal part of 2BL is a gene-rich region of the wheat genome which may be true in particular for *Pm* genes. According to the marker data (Fig. 7), *QPm.uga-2BL* should be different from *Pm6*, a commonly used gene in the southeastern USA, but its relationship to other resistance genes remains uncertain.

Similarly, several QTL have been reported on chromosome 4A, and most of them are of minor effect and only detected in certain environments, except *QPm.tut-4A*, the race non-specific resistance QTL from a *T. militinae* translocation (Jakobson et al. 2006, 2012). *QPm.tut-4A* and a minor QTL identified in US hard winter wheat cultivar '2174' are both closely linked to the marker *Xgwm160* and assigned to the most distal part of 4AL (Chen et al. 2009; Jakobson et al. 2012). They are presumed to be different from the QTL detected in the present study on chromosome 4AS or near the centromere (Fig. 4a). The present 4A QTL is about 68 and 180 cM, respectively, from two QTL detected in Swiss wheat cultivar 'Forno' on 4AL based on the common marker *Xgwm397* (Keller et al. 1999), indicating that they are also different. However, the present 4A

Table 2 Presence (+) or absence (−) of the AGS 2000 amplification pattern of SSR marker *Xbarc134*, closely linked to the resistance gene *PmA2K*, in a set of wheat powdery mildew differential lines

Cultivar/line	Pedigree	*Pm* genes	*Xbarc134*[a]
CI 14114	Axminster/8*Chancellor	*1a*	−
MocZlatka	ATRI1509/Slaska//ATRI3310/Slaska*3/3/Zlatka	*1b*	−
M1N	Weihenstephan M1 or M1/Blaukorn Weihenstephan	*1c*	−
TSD TRI2258	Unknown, collection in Gatersleben Gene Bank	*1d*	−
Virest	EST-39-12/Virgilio	*1e*	−
CI 14118	Ulka/8*Chancellor	*2*	−
CI 14120	Asosan/8*Chancellor	*3a*	−
CI 14121	Chul/8*Chancellor	*3b*	−
CI 14122	Sonora/8*Chancellor	*3c*	−
Ralle	Svenno/Parlo//2149.60	*3d*	−
W150	Yandilla King/Zaff	*3e*	−
CI 15888	Michigan Amber/8*Chancellor	*3f*	−
Aristide	Boulmiche//Mexique 50/B21	*3g*	−
CI 14124	Yuma/8*Chancellor	*4a*	−
Ronos	Graf/Kormoran//Kronjuwel	*4b*	−
81-7241	Fan7/Aurora//77S-3521	*4c*	+
CI 14125	Hope/8*Chancellor	*5a*	−
Kormoran	Cappelle Desprez/Vilmorin 27//Heine VI	*5b*	−
IGV 1-455	CI 10904/7*Prins	*5d*	−
Coker 747	Arthur/Coker 68-15	*6*	+
Transec	Chinese Spring/irradiated Cornell Sel. 82a1-2-4-7	*7*	−
Kavkaz	Lutescens 314H147/Bezostaja 1	*8*	−
N14	Normandie/Ares	*9*	−
Trans. line 31	*A. speltoides* 2140008/Wembley	*12*	−
CS trans. line	Chinese Spring/*A. longissima*	*13*	−
Norman rec. line	Norman/*T. dicoccoides*	*16*	−
Amigo	Teewon“S”/6/Gaucho/4/Tascosa/3/Wichita/Teewon/5/2*Teewon	*17*	+
XX 186	*T. durum* ‘Santa Marta’/*A. tauschii* ‘BGRC 1458’	*19*	−
TAM W-104[b]	Male sterile Sturdy/PI 252003 (rye) (=TX673)	*?*	+
DH2 (T6AL.6VS)	Yangmai 5/6V(6A)	*21*	−
Chiyacao	Unknown, landrace in Zhengzhou, China	*24a*	−
NC96BGTA5	Saluda*3/PI 427662, shorten as NCA5	*25*	−
Pova	*T. aestivum* cv. Poros-*A. ovata* alien addition line selection 42	*29*	−
C20 derivative	87-1/*T. dicoccoides* C20//2*8866	*30*	−
L501	Rodina *5/*A. speltoides* VIR	*32*	+
NC97BGTD7	Saluda*3/TA2492, shorten as NCD7	*34*	−
NC96BGTD3	Saluda*3/TA2377, shorten as NCD3	*35*	−
5BIL-29	*T. dicoccoides* MG29896/*T. durum* Latino BC_5F_5	*36*	−
NC99BGTAG11	Saluda*3/PI 427315, shorten as NCAG11	*37*	−
CH5025	76216-96/TAI7045//2*Jing 411	*43*	+
Chancellor	Carina/Mediterranean//Dietz/Carina/3/P-1068/3*Purplestraw	*Check*	−
Coker 68-15	Coker 57-6*2//Norin 33/R-485	*Check*	−
NC96BGTA4	Saluda*3/PI 221414, (NCA4)	*Pm-NCA4*	−
NC96BGTA6	Saluda*3/PI 427772, (NCA6)	*Pm-NCA6*	−
NC99BGTAG11[c]	Saluda*3/PI 427315, (NCAG11)	*Pm-NCAG11*	−
NC06BGTAG12	Saluda*3/PI 538457, (NCAG12)	*MlAG12*	−

Table 2 continued

Cultivar/line	Pedigree	*Pm* genes	*Xbarc134*[a]
NC06BGTAG13	Saluda*3/PI 427442, (NCAG13)	*MlAG13*	−
NC09BGTUM15	Saluda*3/TTCC223 (*T. neglecta*) (NCUM15)	*MlUM15*	+

[a] For SSR marker *Xbarc134*, "+" indicates the same amplification pattern in the differential and AGS 2000, "−" indicates different patterns

[b] TAM W-104 is a wrong collection of *Pm20*

[c] *Pm-NCAG11* was formally designated *Pm37*, a duplicate collection

QTL is very near a minor QTL detected in French wheat cultivar 'Courtot' and also based on the marker *Xgwm397* (Bougot et al. 2006). Another minor QTL was also reported on 4A in French wheat line 'RE714', but it is difficult to compare their locations because the genetic map of 4A for RE714 only included RFLP (restriction fragment length polymorphism) markers and the chromosomal assignments of the markers were also uncertain (Chantret et al. 2001; Mingeot et al. 2002). In the present research, *QPm.uga-4A* was stably detected in all field environments and contributed similar major effects on phenotypic variation in both GA and NC. Since *Bgt* race frequencies in the two states differ (Parks et al. 2008), it is likely that the 4A QTL from AGS 2000 represents a race non-specific adult-plant resistance or horizontal resistance locus (Nelson 1978). To the authors' knowledge, this is the first QTL detected on chromosome 4A in southeastern USA wheat germplasm, and it should be valuable for breeding mildew resistant wheat cultivars in the SRWW growing regions.

Based on the positions of the closely linked marker *Xbarc134* and proximal markers *Xgdm147* and *wPt-1541*, *PmA2K* was physically mapped to deletion bin 6BL5-0.40–1.00 (Fig. 6). At least three formally named genes (*Pm12*, *Pm20* and *Pm27*) and one temporarily named gene (*PmG3M*) have been identified on chromosome 6B. *Pm12* was introgressed into line #31 from *A. speltoides*, and was located on the short arm of translocation chromosome 6BS-6SS.6SL (Jia et al. 1996). *Pm27* was introduced from *T. timopheevii* in line 146–155-T of common wheat, and located on a translocation segment near the centromeric region of 6B (Järve et al. 2000). Thus, *Pm12* and *Pm27* were located in different deletion bins from *PmA2K* (Fig. 6). However, two other genes, *PmG3M* and *Pm20*, were assigned to the same deletion bin as *PmA2K* (Fig. 6). *PmG3M* was 23.3 cM distal to the marker *Xbarc134* (Xie et al. 2012b), whereas *PmA2K* was proximal to the marker at 1.2 cM (Fig. 6); thus, the genetic distance between the two genes is about 24.5 cM. *PmG3M* was derived from wild emmer wheat (*T. dicoccoides*) whereas *PmA2K* came from common wheat (*T. aestivum*). It is thus concluded that *PmA2K* is probably different from *PmG3M*. Further, there are four lines of evidence that *PmA2K* is different from *Pm20*: (1) *Pm20*, derived from *Secale cereale* L. cv. 'Prolific', is located on the distal third of the 6RL segment in a T6BS.6RL translocation (Friebe et al. 1994), whereas *PmA2K* is not from rye; (2) normal crossing-over was observed between markers in the *PmA2K* region at the distal end of 6BL, supporting a common wheat origin for *PmA2K*; (3) *Pm20* has never been used in wheat production in the USA or elsewhere because of unfavorable linkage drag (B. Friebe, personal communication); and (4) 'KS93WGRC28', the germplasm containing *Pm20*, was developed in 1993 and released in 1995 (Friebe et al. 1995), later than the cross made in 1989 to develop AGS 2000 (experimental name GA89482E7) (Johnson et al. 2002), and AGS 2000 should not have KS93WGRC28 or its progenitors in its pedigree. In addition to the genes already discussed, one QTL was detected on chromosome 6BL in CIMMYT breedling line SHA3/CBRD (Lu et al. 2012), but its location was near the centromere and about 55 cM proximal to *PmA2K*, indicating they are also different. Thus, it is concluded that *PmA2K* is different from any named gene or QTL reported on this particular chromosome arm. The gene is formally designated *Pm54*.

For the 2BL and 4A QTL, the closely linked molecular markers were all diversity arrays technology (DArT) markers. Since there are limitations on their direct utilization in breeding programs, it would be advantageous to convert them to more easily used PCR-based markers according to their sequences (http://www.diversityarrays.com/). For the gene *Pm54* on 6BL, the most closely linked marker is an easily used SSR marker; genotyping the differentials with the marker *Xbarc134* demonstrates that the marker is suitable for marker-assisted selection in pyramiding *Pm54* with most of the differential genes (Supplementary Fig. S4, Table 2). However, knowing the polymorphism in a panel of SRWW cultivars would be more useful to aid the introgression of *Pm54* with molecular markers. Cautions should also be given for the appearing virulent *Bgt* isolates for this gene in SRWW growing regions. In conclusion, the QTL reported here and their corresponding closely linked molecular markers will help diversify the genetic sources of *Pm* resistance in SRWW and will facilitate the breeding process.

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Ethical standard The experiments reported in the manuscript are in accordance with the ethical standards in the country in which they were performed.

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