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ORIGINAL PAPER

Identification and genetic mapping of *pm42*, a new recessive wheat powdery mildew resistance gene derived from wild emmer (*Triticum turgidum var. dicoccoides*)

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Abstract Powdery mildew, caused by *Blumeria graminis* f. sp. *tritici*, is one of the most important wheat diseases worldwide in areas with cool or maritime climates. Wild emmer (*Triticum turgidum* var. *dicoccoides*) is an important potential donor of disease resistances and other traits for common wheat improvement. A powdery mildew resistance gene was transferred from wild emmer accession G-303-1M to susceptible common wheat by crossing and backcrossing, resulting in inbred line P63 (Yanda1817/G-303-1 M//3*Jing411, BC_2F_6). Genetic analysis of an F_2 population and the $F_{2:3}$ families developed from a cross of P63 and a susceptible common wheat line Xuezao showed that the powdery mildew resistance in P63 was controlled by a single recessive gene. Molecular markers and bulked segregant analysis were used to characterize and map the powdery mildew resistance gene. Nine genomic SSR markers (*Xbarc7*, *Xbarc55*, *Xgwm148*, *Xgwm257*, *Xwmc35*, *Xwmc154*, *Xwmc257*, *Xwmc382*, *Xwmc477*), five AFLP-derived SCAR markers (*XcauG3*, *XcauG6*, *XcauG10*, *XcauG20*, *XcauG22*), three EST–STS markers (*BQ160080*, *BQ160588*, *BF146221*) and one RFLP-derived STS marker (*Xcau516*) were linked to the resistance gene, designated *pm42*, in P63. *pm42* was physically mapped on chromosome 2BS bin 0.75–0.84 using Chinese Spring nullisomic-tetrasomic, ditelosomic and deletion lines, and was estimated to be more than 30 cM proximal to *Xcau516,* a RFLP-derived STS marker that co-segregated with the wild emmer-derived *Pm26* which should be physically located in 2BS distal bin 0.84–1.00. *pm42* was highly effective against 18 of 21 differential Chinese isolates of *B. graminis* f. sp. *tritici*. The closely linked molecular markers will enable the rapid transfer of *pm42* to wheat breeding populations thus adding to their genetic diversity.

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Introduction

Powdery mildew, caused by *Blumeria graminis* f. sp. *tritici* (*Bgt*), is a serious fugal disease in many wheat-growing areas with cool or maritime climates. Due to more intensive wheat cultivation systems, including the use of semi-dwarf cultivars and improved irrigation and fertilization conditions, powdery mildew severities have increased in drier and warmer areas as well. The use of resistant cultivars is the most economical and environmentally safe approach to control the disease. However, since some powdery mildew resistance genes are rapidly overcome by new and/or virulent races of the pathogen, it is necessary to search for new sources of resistance and to use available genes in combinations that will prolong the period of effectiveness of such genes. Some of the 41 designated powdery mildew resistance loci (*Pm1–Pm43*; McIntosh et al. 2008; McIntosh personal communication) were derived from diploid and tetraploid cultivated and wild wheats.

Wild emmer, *Triticum turgidum* var. *dicoccoides* ($2n = 4x = 28$; genome AABB), is a progenitor of both cultivated tetraploid and hexaploid wheats. It carries many agronomically important traits that can be exploited for wheat improvement, including those conferring quality attributes and disease resistance (Moseman et al. 1985; Nevo et al. 1991, 2002). Such traits are easily transferred to cultivated wheat by traditional breeding methods. Several powdery mildew resistance genes, including *Pm16*, *Pm26*, *Pm30* and *MlZec1*, were transferred from wild emmer to hexaploid wheat (Reader and Miller 1991; Rong et al. 2000; Liu et al. 2002; Mohler et al. 2005), and *MlIW72* and *Pm36* were introduced to durum wheat (Ji et al. 2007; Blanco et al. 2008).

From the early 1990s, molecular markers and linkage mapping have become powerful tools in refining the accuracy and efficiency of genetic mapping in wheat. Molecular markers linked to more than 30 wheat powdery mildew resistance genes/alleles have been reported (Huang and Röder 2004; McIntosh et al. 2008) and some of these markers have been used in marker-assisted selection (MAS) and for pyramiding resistance genes. Based on fine mapping with molecular markers, *Pm3b* was cloned and characterized from hexaploid wheat (Yahiaoui et al. 2004).

Molecular markers used to map powdery mildew resistance genes in wheat have included restriction fragment length polymorphisms (RFLP), amplified fragment length polymorphisms (AFLP) and microsatellites (or simple sequence repeats, SSR) (see Huang and Röder (2004) for review). RFLP and AFLP are laborious, time-consuming and costly, and these features limit their use for routine genotyping in breeding. Consequently, some RFLP and AFLP markers were converted into PCR-based sequence tagged site (STS) and sequence-characterized amplified region (SCAR) markers. For example, the STS marker *Xmag2185* linked to powdery mildew resistance genes *Mlm2033* and *Mlm80* was derived from RFLP marker *PSR680* (Yao et al. 2007) and the SCAR marker *SCM08* linked to seed coat color in *Brassica juncea* was derived from AFLP marker *AFLP8* (Negi et al. 2000). To date, no SCAR marker in wheat has been derived from an AFLP marker linked to a powdery mildew resistance gene.

The increasing availability of expressed sequence tags (EST) and genomic sequences in wheat are now providing valuable resources for marker enrichment. Currently, there are more than 17,000 STS primers (forward and reverse) designed according to EST and genomic sequence information (http://wheat.pw.usda.gov/wEST/).

The present paper reports genetic and linkage analyses involving the identification and transfer of a new recessive powdery mildew resistance gene on chromosome 2BS from wild emmer accession G-303-1M to hexaploid wheat.

Materials and methods

Plant materials

Wild emmer accession G-303-1M was kindly provided by Dr. ZK Gerechter-Amitai, Agricultural Research Organization, The Volcani Centre, Israel. G-303-1M was highly resistant, with infection type (IT) 0, to *Bgt* isolates E09, E11, and E15, three prevailing pathotypes in the Beijing area from 1993 to 2008 (unpublished data). Yanda 1817, Jing 411 and Xuezao were locally adapted common wheat lines highly susceptible to *Bgt* isolate E09, E11, and E15, with infection type (IT) 4. Yanda 1817 and Jing 411 were used as parental lines for crossing and backcrossing to introduce resistance gene from G-303-1M to common wheat, resulting in the BC_2F_6 powdery mildew resistance inbred line P63 (Yanda 1817/G-303-1 M//3*Jing 411). This line was crossed with Xuezao for genetic analysis and molecular mapping. Cultivars/lines with known powdery mildew resistance genes used for comparisons of powdery mildew responses with that of line P63 are listed in Table 1.

Chinese Spring (CS) and selected nullisomic-tetrasomic lines (N2AT2B, N2AT2D, N2BT2A, N2BT2D, N2DT2A and N2DT2B), ditelosomic lines (Dt2AS, Dt2AL, Dt2BL, Dt2DS and Dt2DL) and deletion lines (d2AS-5; d2AL-1; d2BS-1; d2BS-3; d2BS-4, 2BL-4, 3DL-2; d2BL-6; d2DS-5; d2DL-9) of homoeologous group 2 were used for chromosomal arm assignment and bin mapping of molecular markers.

Table 1 Differential reactions of 30 wheat cultivars/lines possessing known powdery mildew resistance genes after inoculation with 21 isolates of *Blumeria graminis* f. sp. *tritici*

Cultivar/line	*Blumeria graminis tritici* isolate																				Pm
	E01	E02	E03	E05	E06	E07	E09	E11	E13	E15	E16	E17	E18	E20	E21	E23	E25	E26	E30	E31	E32	
Chancellor	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	–
Axminister/8*Cc[a]	S	S	S	S	S	S	S	R	R	S	S	S	S	S	S	S	S	S	S	S	S	*1*
Ulka/8*Cc	R	R	R	R	R	R	R	R	R	R	R	S	S	S	S	R	R	R	R	R	S	*2*
Asosan/8*Cc	S	S	R	S	S	S	R	S	S	R	R	S	S	S	S	S	S	S	S	S	S	*3a*
Chul/8*Cc	S	S	S	S	S	R	S	R	R	R	S	R	S	S	S	S	S	R	S	S	S	*3b*
Sonora/8*Cc	S	S	S	S	S	S	S	S	S	R	S	S	S	S	S	S	S	S	S	S	S	*3c*
Kolibri	S	S	R	S	S	S	R	S	R	R	S	S	S	S	S	S	S	S	S	S	S	*3d*
W150	S	S	S	S	R	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	*3e*
Michigen Amber/8*Cc	R	S	R	R	S	R	R	R	R	S	S	S	S	S	S	R	R	R	R	R	R	*3f*
Khapli/8*Cc	R	S	R	R	R	R	R	R	R	S	S	R	S	S	S	R	R	R	R	R	R	*4a*
Armada	R	S	R	R	R	R	R	R	R	R	S	R	S	S	S	R	R	R	R	R	R	*4b*
Hope/8*Cc	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	*5a*
Aquila	R	R	R	R	R	R	R	R	R	R	R	S	S	S	S	R	R	R	R	R	S	*5b*
Coker747	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	*6*
CI14189	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	*7*
Kavkaz	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	R	S	R	*8*
Wembley	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	*12*
R4A	R	R	R	R	R	R	R	R	R	S	R	R	R	R	R	R	R	R	R	R	R	*13*
Brigand	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	*16*
Amigo	S	S	R	S	S	S	S	R	R	R	S	S	S	S	S	R	S	R	R	R	R	*17*
XX186	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	*19*
Yangmai5/Sub.6v	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	*21*
81-7241	R	R	R	R	R	R	R	R	R	R	R	R	S	S	S	R	R	R	R	R	R	*23*
Chiyacao	R	R	R	S	S	R	R	R	S	R	R	R	S	R	R	R	R	R	R	R	S	*24*
5P27	R	R	R	R	R	R	R	R	R	R	S	R	S	S	S	R	R	R	R	R	R	*30*
Normandie	R	R	R	R	S	S	R	S	S	R	R	R	R	R	S	S	R	R	R	R	R	*1+2+9*
Maris Huntsman	R	R	R	R	R	R	R	R	R	R	R	S	S	S	S	R	R	R	R	R	S	*2+6*
Coker 983	S	S	R	S	S	S	S	S	S	R	S	S	S	S	S	S	S	S	S	R	S	*5+6*
Mission	R	R	R	R	R	R	R	R	R	R	S	R	S	S	S	R	R	R	R	R	R	*4b+5b*
P63	R	S	R	R	R	R	R	R	R	R	R	R	R	R	S	R	R	R	R	R	S	*42*

R resistant, *S* susceptible

[a] Seven times backcrossed to cv. Chancellor

Powdery mildew evaluations

Twenty-one Chinese *Bgt* isolates were used to compare the reactions of P63 and 29 accessions with known powdery mildew resistance genes (Table 1). Isolate E09, virulent to *Pm1*, *Pm3a*, *Pm3b*, *Pm3c*, *Pm3d*, *Pm3e*, *Pm3f*, *Pm5*, *Pm6*, *Pm7*, *Pm8*, *Pm17* and *Pm19*, but avirulent to line P63 and its derivatives (Table 1), was used to inoculate parental lines P63 and Xuezao, and the F_1, F_2 and $F_{2:3}$ progenies of P63/Xuezao. The $F_{2:3}$ families (25 seedlings of each $F_{2:3}$ family) were tested to confirm the phenotypes and to establish the resistance genotypes of each F_2 plant. Under the controlled greenhouse conditions seedlings were inoculated when the first leaf was fully expanded. Inoculations were performed by brushing conidia from neighboring sporulating susceptible seedlings of Xuezao. Infection types (IT) were scored 15 days after inoculation when Xuezao was heavily infected, on a scale of 0, 0; and 1, 2, 3, 4, with 0 for no visible symptoms; 0 for necrotic flecks; and 1, 2, 3, 4 for highly resistant, resistant, susceptible and

highly susceptible reactions, respectively (Liu et al. 2002). Phenotypes were pooled into two groups, resistant (R, IT = 0–2) and susceptible (S, IT = 3, 4).

Marker analysis

Genomic DNA was extracted from the uninfected seedling leaves of each F_2 plant by the CTAB method (Saghai-Maroof et al. 1984). For bulked segregant analysis, separate DNA bulks were assembled by using equal amounts of DNA from ten homozygous resistant and ten homozygous susceptible F_2 plants. Wheat microsatellite markers (*Xgwm, Xwmc, Xbarc, Xcfa*, and *Xcfd* series) mapped on A and B genome chromosomes were chosen for marker analyses. Information of these markers is available online at http://wheat.pw.usda.gov. The primer sequences of RFLP-derived STS marker *Xcau516* (CAU516F: 5′-AAGAAGA AGCAAAGCGT-3′, CAU516R: 5′-AGAGAAGAGAAA GAGATAGC-3′) were designed by end-sequencing the RFLP probe WG516. To saturate the genomic region carrying the powdery mildew resistance gene with additional markers, 31 AFLP-derived SCAR markers (unpublished data) were tested, for which the sequence information of 5 polymorphic SCAR markers relevant to this study are listed in Table 2. Forty-seven STS primers developed from wheat ESTs (http://wheat.pw.usda.gov/SNP/primers/contig_primer_list.xls) mapped to chromosome 2BS were also screened for polymorphism.

PCR were conducted in total volumes of 10 µl containing 10 mM Tris–HCl, pH 8.3, 50 mM KCl, 1.5 mM $MgCl_2$, 0.2 mM dNTPs, 25 ng of each primer, 50 ng genomic DNA and 0.75U Taq DNA polymerase. Amplifications were performed at 94°C for 5 min, followed by 40 cycles at 94°C for 45 s, 50–60°C (depending on specific primers) for 45 s, and 72°C for 1 min, with a final extension at 72°C for 10 min. Three µl of PCR products mixed with 2 µl loading buffer were separated on 8% non-denaturing polyacrylamide gels (39 acrylamide:1 bisacrylamide). Gels were silver stained and photographed.

Data analysis

Chi-squared (χ^2) tests for goodness-of-fit were used to evaluate deviations of observed data from theoretically expected segregation ratios. Linkages between markers and the resistance gene were determined using Mapmaker 3.0, with a LOD score threshold of 3.0.

Chromosome arm assignment and physical bin mapping

Polymorphic markers linked to the resistance gene were located using a set of Chinese Spring homoeologous group 2 nullisomic-tetrasomics, ditelosomics and deletion lines. Markers located to chromosome bins by determining the largest deletion line possessing them.

Results

Differential reactions of P63 and wheat accessions with known genes for *Bgt* resistance

Differential reactions of P63 and 29 wheat accessions possessing known powdery mildew resistance genes to 21 *Bgt* isolates are listed in Table 1. P63 was highly resistant to 18 isolates and gave a unique pattern of responses indicating that it was different from the other lines by at least one resistance allele.

Genetic analysis of resistance

P63 was highly resistant to isolate E09 (IT 0), whereas Xuezao was highly susceptible (IT 4). Thirty-five F_1 seedlings plants of P63/Xuezao all produced IT 4 indicating recessiveness of resistance. The F_2 population segregated 57 resistants:141 susceptibles fitting the 3:1 ratio ($\chi^2_{3:1} = 1.37$, $P > 0.2$). When 198 F_2 plants were transplanted to the field, only 124 survived to produce F_3 seeds. Progenies of these plants were classified 29 homozygous resistant:76 segregating:19 homozygous susceptible, a poor fit to an expected 1:2:1 ratio ($\chi^2_{1:2:1} = 7.94$, $P < 0.05$), which may be a result of lower survival rate of highly susceptible F_2 plants during transplantation. We concluded that the powdery mildew resistance in P63 was controlled by a single recessive gene, which we designated *pm42*.

Table 2 AFLP-derived SCAR markers linked to powdery mildew resistance gene *pm42*

SCAR marker	AFLP primer pairs	Forward primer (5′–3′)	Reverse primer (5′–3′)	Product size (bp)
XcauG3	*PgagMcaa*	GCAGGAGGCGAAACAACT	TAACAACCATTTGTGCCAATG	278
XcauG6	*PaatMcac*	CTATGGTATTGTCCGCTAG	GCAGAATTTTCACAGTTGC	293
XcauG10	*PaagMcac*	CTGCAGAAGAAGTAGGCT	CACTACTATGCTTTTCTCCT	293
XcauG20	*PgatMcat*	TTAACATGCCACGTTGAC	TTAGCTGATGGTTGTTGC	387
XcauG22	*PgttMcat*	CAGGTTGTTGGTGGAGC	ATGCTTCAACCTCGCTGT	325

Identification of molecular markers linked to *pm42*

Initially, 552 wheat SSR markers (*Xgwm*, *Xwmc,* *Xbarc*, *Xcfa*, and *Xcfd*) mapped to A and B genomes were screened for polymorphism between the resistant and susceptible F_2 DNA bulks. Nine genomic SSR markers, *Xbarc7*, *Xbarc55*, *Xgwm148*, *Xgwm257*, *Xwmc35*, *Xwmc154*, *Xwmc257*, *Xwmc382*, and *Xwmc477*, were linked to the powdery mildew resistance gene in P63 after testing the 124 individual DNA samples of the surviving F_2 plants (Fig. 1). All nine SSR markers were located on chromosome 2BS, and a linkage map was constructed (Fig. 2a). Among the nine SSR markers, seven were co-dominant and *Xwmc257* and *Xwmc382* were dominant markers in coupling with the resistance allele.

To saturate the genomic region harbouring *pm42*, 31 AFLP-derived SCAR markers mapped on chromosome 2BS were tested on the resistant and susceptible DNA bulks. Five, *XcauG3*, *XcauG6*, *XcauG10*, *XcauG20*, *XcauG22,* were polymorphic and linked to *pm42* (Fig. 2a) as dominant markers, among which *XcauG3*, *XcauG20*, *XcauG22* were in coupling and *XcauG6* and *XcauG10* (Fig. 1b) were in repulsion with *pm42*.

Of 47 2BS EST–STS primer pairs screened, 3, BQ160080, BQ160588, BF146221, generated polymorphic bands between the resistant and susceptible DNA bulks (Fig. 1c). *BF146221* mapped 0.9 cM proximal to *pm42* (Fig. 2a).

Powdery mildew resistance gene *Pm26* also originated from wild emmer, and co-segregated with RFLP marker *Xwg516* (Rong et al. 2000). To clarify the relationship between *pm42* and *Pm26*, the RFLP probe WG516 was end-sequenced and STS marker *Xcau516* was developed. When this marker was genotyped on the P63/Xuezao F_2 population, it was estimated to be 36.8 cM distal to *pm42* (Figs. 1d, 2a). Thus *Pm26* and *pm42* appear to be located at different loci.

Chromosome bin map of molecular markers linked to *pm42*

Chinese Spring homoeologous group 2 nullisomic-tetrasomics, ditelosomics and deletion lines were used to bin map the *pm42*-linked molecular markers. *Xwmc382, Xwmc154*, *Xgwm257*, *Xwmc257*, and *BQ160080* mapped to the distal bin 0.84–1.00; *BQ160588* and *BF146221* mapped

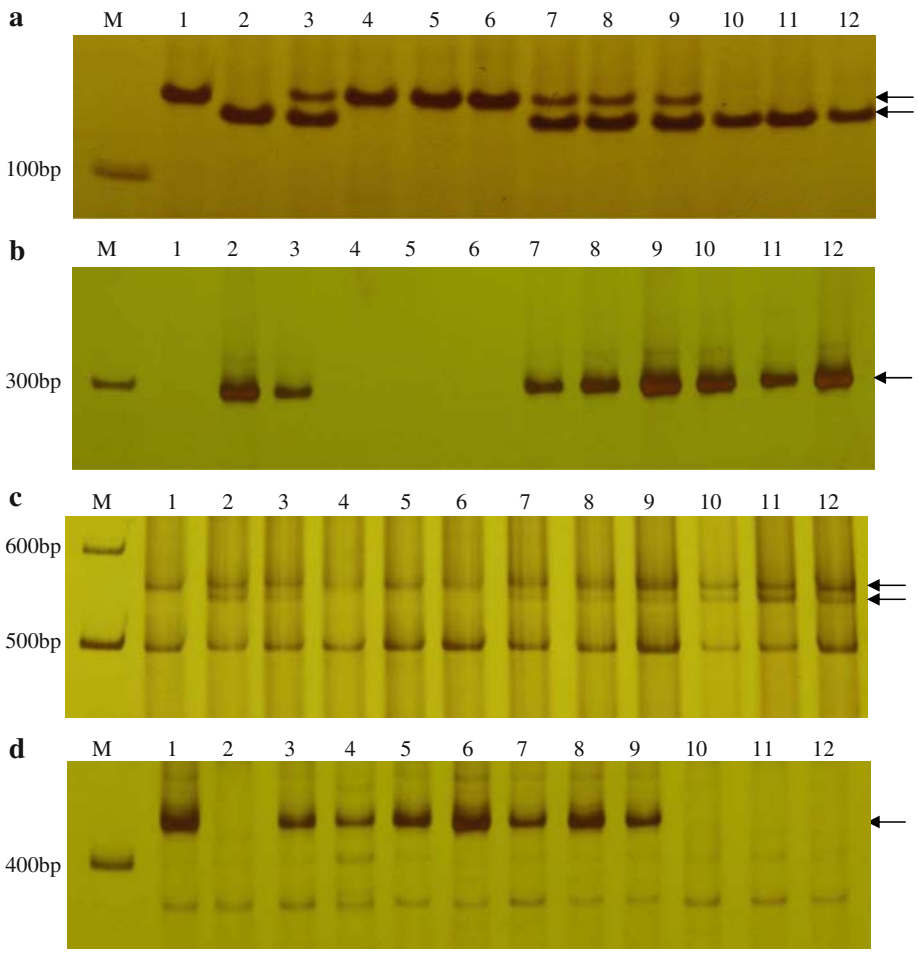

Fig. 1 Examples of amplification patterns of *pm42*-linked polymorphic primers BARC55 (**a**), CAUG10 (**b**), BF146221 (**c**) and CAU516 (**d**) detected in 8% non-denatured polyacrylamide gels. The arrows on the right side indicate the polymorphic bands. *M* 1 Kb DNA ladder, and numbers to the *left* are band size. *Lanes* 1, 2 and 3 are Xuezao, P63 and P63/Xuezao F_1, respectively. *Lanes* 4–6 represent homozygous susceptible plants, *lanes* 7–9 represent heterozygous susceptible plants and *lanes* 10–12 represent homozygous resistant plants

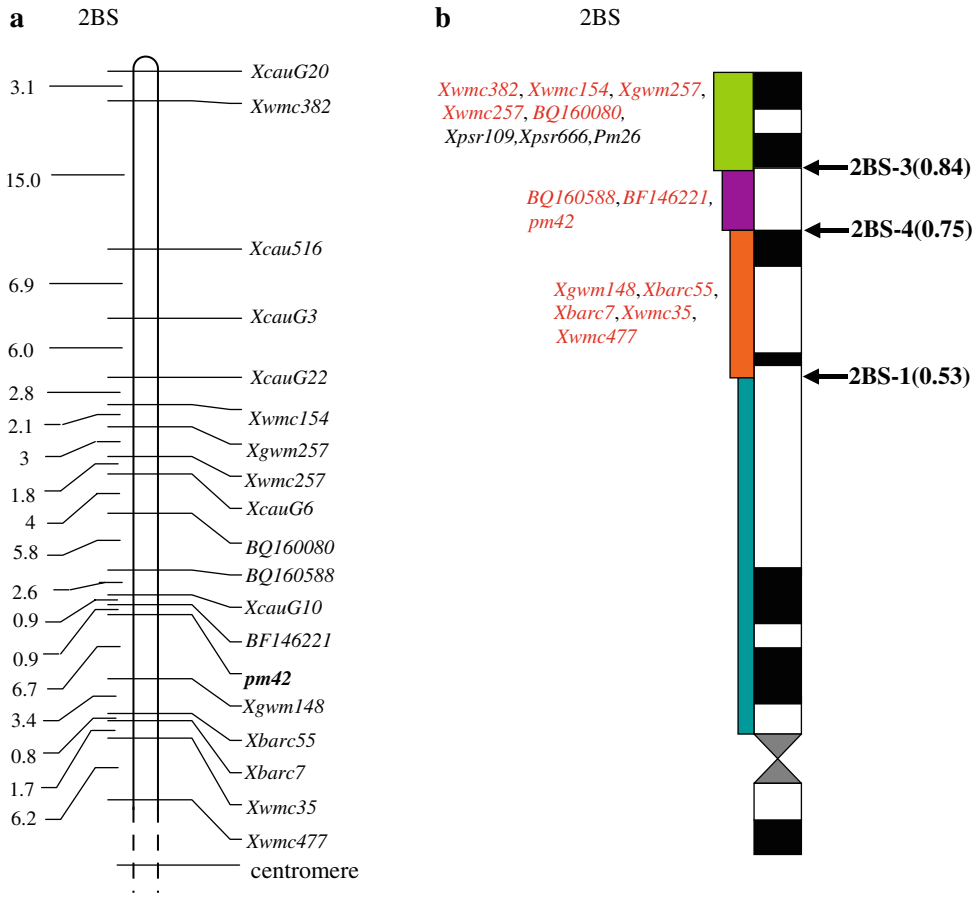

Fig. 2 Genetic linkage map with genetic distances in cM shown on the *left* (**a**) and physical bin map (**b**) of recessive powdery mildew resistance gene *pm42* on chromosome 2BS

to 2BS bin 0.75–0.84; and *Xgwm148, Xbarc55, Xbarc7*, *Xwmc35* and *Xwmc477* mapped 2BS bin 0.53–0.75 (Fig. 2b). The RFLP-derived STS marker *Xcau516* was null in Chinese Spring and could not be mapped. Deletion bin mapping thus indicated that *pm42* is most likely located in 2BS bin 0.75–0.84 (Fig. 2b).

Discussion

Wild emmer is a useful potential donor of important agronomic traits, such as high grain protein content, disease resistance and abiotic stress tolerance, for wheat improvement (Nevo et al. 2002). It has diverse resistance to several pathogens, including stripe rust (Grama and Gerechter-Amitai 1974), stem rust (Nevo et al. 1991), leaf rust (Moseman et al. 1985) and powdery mildew (Moseman et al. 1984). The six cultivated wheat powdery mildew resistance genes derived from wild emmer, including *Pm16* (Reader and Miller 1991), *Pm26* (Rong et al. 2000), *Pm30* (Liu et al. 2002), *MlZec1* (Mohler et al. 2005), *MlIW72* (Ji et al. 2007) and *Pm36* (Blanco et al. 2008), are located on chromosomes 4A, 2BS, 5BS, 2BL, 7AL and 5BL, respectively. *Pm16* was initially placed on chromosome 4A by monosomic analysis (Reader and Miller 1991), but was later found to be linked to SSR marker *Xgwm159-5BS,* and therefore could be the same as *Pm30* (Chen et al. 2005). However, differential reactions to 4 *Bgt* isolates were observed between *Pm16* and *Pm*30 (Table 1), indicating they are likely different alleles. *MlIW72* was linked with SSR loci *Xgwm344* and *Xwmc525* on the long arm of chromosome 7A. Chromosome bin mapping indicated it is located in the most distal bin of chromosome 7AL and hence could be an allele to *Pm1* (Ji et al. 2007). *Pm36* was transferred to durum wheat and mapped close to the EST–SSR marker *BJ261635* on chromosome 5BL (Blanco et al. 2008). *MlZec1*, a dominant resistance gene, was mapped distally to SSR marker *Xwmc356* in terminal bin 2BL 0.89–1.00 (Mohler et al. 2005). *Pm26*, co-segregating with RFLP marker *Xwg516*, was the only recessive powdery mildew resistance allele derived from wild emmer prior to the present *pm42*. Interestingly, *Pm26* is also located on chromosome 2BS, but its position distal to RFLP marker loci *Xpsr109* and *Xpsr666* (Rong et al. 2000) which have been physically mapped on bin 2BS

0.84–1.00 (Delaney et al. 1995) appears to be significantly distal to that of *pm42*. Unfortunately, a test of allelism between *Pm26* and *pm42* was not possible as a genetic stock possessing *Pm26* is not publically available (Feldman, personal communication).

In addition to *Pm26* and *MlZec1*, another two powdery mildew resistance genes, *Pm6* and *Pm33*, have also been located on chromosome 2B. *Pm6* was originated from the 2G chromosome of *T. timopheevii* and introgressed into chromosome 2BL of common wheat (Tao et al. 2000). Zhu et al. (2005) transferred *Pm33* from *T. carthlicum* accession PS5 into common wheat and identified three closely linked SSR markers *Xwmc317*, *Xgwm111* and *Xgwm382* on chromosome 2BL. Thus, *pm42* is different from known powdery mildew resistance genes on wheat chromosome 2B and represents a new powdery mildew resistance locus.

Among known powdery mildew resistance genes, only five recessive loci, *Pm5* (7BL, Lebsock and Briggle 1974; Hsam et al. 2001; Huang et al. 2003), *Pm9* (7AL, Schneider et al. 1991), *Pm26* (2BS, Rong et al. 2000), *mlRD30* (7AL, Singrün et al. 2004), and *pm2026* (5A^{m}L, Xu et al. 2008), have been reported. *Pm5* complex locus contains five different alleles (*Pm5a–Pm5e*). *Pm5e* was found very common in Chinese landraces and highly effective in against prevalence isolate of *B. graminis* f.sp. *tritici* for quite a long period (Huang et al. 2003). Normandie, which possesses gene combination *Pm1+2+9*, was susceptible to four European Bgt isolates, indicating that *Pm9* is no longer effective (Singrün et al. 2004). *Pm26*, *mlRD30*, *pm2026* and *pm42* have been recently released to breeders in commercial wheat breeding program. However, the recessive nature and durability of these genes with respect to barley broad-spectrum powdery mildew resistance gene *mlo* remain unknown. Although *pm42* is highly effective against 18 out of 21 Chinese *Bgt* isolates, there is a possibility of its resistance to be overcome by virulent *Bgt* isolates if new varieties have been developed in and for monoculture. Pyramiding or combination of additional resistance genes to virulent pathotypes should be considered to provide durable resistance in breeding practice.

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