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ORIGINAL PAPER

Identification and mapping of *PmG16*, a powdery mildew resistance gene derived from wild emmer wheat

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Abstract The gene-pool of wild emmer wheat, *Triticum turgidum* ssp. *dicoccoides*, harbors a rich allelic repertoire for disease resistance. In the current study, we made use of tetraploid wheat mapping populations derived from a cross between durum wheat (cv. Langdon) and wild emmer (accession G18-16) to identify and map a new powdery mildew resistance gene derived from wild emmer wheat. Initially, the two parental lines were screened with a collection of 42 isolates of *Blumeria graminis* f. sp. *tritici* (*Bgt*) from Israel and 5 isolates from Switzerland. While G18-16 was resistant to 34 isolates, Langdon was resistant only to 5 isolates and susceptible to 42 isolates. Isolate *Bgt*#15 was selected to differentiate between the disease reactions of the two genotypes. Segregation ratio of $F_{2\text{-}3}$ and recombinant inbreed line (F_7) populations to inoculation with isolate *Bgt*#15 indicated the role of a single dominant gene in conferring resistance to *Bgt*#15. This gene, temporarily designated *PmG16,* was located on the distal region of chromosome arm 7AL. Genetic map of *Pm*G16 region was assembled with 32 simple sequence repeat (SSR), sequence tag site (STS), Diversity array technology (DArT) and cleaved amplified polymorphic sequence (CAPS) markers and assigned to the 7AL physical bin map (7AL-16). Using four DNA markers we established colinearity between the genomic region spanning the *PmG16* locus within the distal region of chromosome arm 7AL and the genomic regions on rice chromosome 6 and *Brachypodium* Bd1. A comparative analysis was carried out between *PmG16* and other known *Pm* genes located on chromosome arm 7AL. The identified *PmG16* may facilitate the use of wild alleles for improvement of powdery mildew resistance in elite wheat cultivars via marker-assisted selection.

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Introduction

Wheat (*Triticum* spp.) is a major staple food crop in many parts of the world in terms of cultivated area and food source. To date, with ~620 million tons produced annually worldwide, wheat provides about one-fifth of the calories consumed by humans (FAO stat 2008). Powdery mildew, caused by the biotrophic pathogen *Blumeria graminis* (DC.) EO Speer f. sp. *tritici* Em. Marchal (syn. *Erysiphe graminis* f. sp. *tritici*), is a foliar disease of wheat, which causes severe yield reduction in areas with cool or maritime

climates (e.g., Smith and Smith 1974; Bennett 1984; Hsam and Zeller 2002). In recent years, agro-technical improvements in irrigation and fertilization techniques have increased the risk of disease spreading. Yield losses ranged from 5 to 34% and in severe cases to 45% or more (Griffey et al. 1993; Hsam and Zeller 2002; Yao et al. 2007). Therefore, developing new cultivars with improved powdery mildew resistance is economically and environmentally sound approach to reduce yield losses. This approach, however, requires comprehensive exploration of potential genetic resources and an in-depth understanding of their resistance mechanisms.

It is well established that the genetic diversity of crop plants has been eroded relative to their wild relatives as a result of the genetic bottleneck associated with the domestication process (i.e., founder effect; Ladizinsky 1985) and subsequent modern breeding processes (Tanksley and McCouch 1997; Ladizinsky 1998). This genetic erosion had far-reaching agronomic consequences limiting our ability to protect crop plants from biotic and abiotic stress factors and to meet future global challenges (e.g., Harlan 1972; Zamir 2001). Using crosses between domesticated and wild species of inbreeding plants, alleles that were "left behind" during domestication may be reintroduced into the domesticated gene-pool (McCouch 2004).

Wild emmer wheat [*T. turgidum* ssp. *dicoccoides* (Körn.) Thell., also known as *T. dicoccoides*], is the allotetraploid ($2n = 4x = 28$; BBAA) progenitor of both domesticated tetraploid durum wheat [*T. turgidum* ssp. *durum* (Desf.) MacKey] and hexaploid ($2n = 6x = 42$; BBAADD) bread wheat (*T. aestivum* L.) (Feldman 2001). Wild emmer wheat germplasm harbors a rich allelic repertoire for improving agronomically important traits (e.g., Feldman and Sears 1981; Nevo et al. 2002; Peleg et al. 2007) including disease resistance (Fahima et al. 1998; Hsam and Zeller 2002). The earliest finding of wild emmer showing symptoms of powdery mildew was reported by Reichert (1940), decades after wild emmer had been discovered by A. Aaronsohn in 1906 (Aaronsohn and Schweinfurh 1906). Later on, the wild emmer gene-pool was shown to contain a particularly promising allelic reservoir for powdery mildew resistance (e.g., Gerechter-Amitai and van Silfhout 1984; Moseman et al. 1984). Sixty-six powdery mildew resistance genes/alleles have been reported so far in 43 loci (*Pm1–Pm43*) and located onto 18 different chromosomes of hexaploid wheat. Eight of these genes originated from wild emmer germplasm: *Pm16* (Reader and Miller 1991), *Pm26* (Rong et al. 2000), *Pm30* (Liu et al. 2002), *Pm36* (Blanco et al. 2008), *MlZec1* (Mohler et al. 2005), *MlIW72* (Ji et al. 2008), *Pm41* (Li et al. 2009) and *Pm42* (Hua et al. 2009).

The overall objective of this study was to explore the genetic bases of powdery mildew resistance in wild emmer wheat as a source for improving domesticated wheat. In the current study we report on (1) the identification and genetic mapping of a novel powdery mildew resistance gene, designated *PmG16*, derived from wild emmer wheat; (2) comparative genetic mapping of *PmG16* and other known *Pm* genes located on the same chromosome; and (3) the establishment of colinearity between the genomic region spanning the *PmG16* locus within the distal region of chromosome arm 7AL and the genomic regions on rice chromosome 6 and *Brachypodium* chromosome Bd1.

Materials and methods

Plant material and powdery mildew isolates

Two mapping populations previously obtained from a cross between *T. durum* wheat cultivar (cv. Langdon; LDN hereafter) and wild emmer wheat (*T. turgidum* ssp. *dicoccoides* accession #G18-16) (Peleg et al. 2008) were used in the current study. The first population consisted of 93 F_2-derived F_3 families (20 plants for each F_3 family). Additionally, a population of 152 F_7 RILs was developed from the same original cross by single-seed descent procedure. The two parental lines, LDN and G18-16, were tested for resistance to 47 isolates of powdery mildew (*Blumeria graminis* f. sp. *tritici*; *Bgt* hereafter). Forty-two *Bgt* isolates were collected from various wheat species (wild and domesticated) or cultivars in various locations across Israel by Dr. N. Eshed and A. Dinoor. In addition, five *Bgt* isolates were provided by the Federal Research Station for Agriculture, Forschungsanstalt Agroscope Reckenholz-Tänikon, Zürich, Switzerland (Table 1).

Isolate *Bgt*#15 (collected from durum wheat cultivar Inbar at Yavor, Western Galilee, Israel, E35°35′ N32°47′) clearly differentiated between the reactions of the two parental lines. This isolate was therefore selected for further characterization using a set of differential wheat accessions carrying known *Pm* resistance genes that were obtained from the National Small Grain Collection (Aberdeen, Idaho, USA) and from Prof. F.J. Zeller (Technische Universität München, Institut für Pflanzenbau und Pflanzenzüchtung, Germany). Phenotypic performance of *Bgt*#15 was tested on two independent progeny sets, a segregating population of 93 F_2- derived F_3 families and 152 F_7 RILs.

Reactions to powdery mildew

Host seedlings were grown at 21°C, 12 h photoperiod. The tests for mildew resistance were conducted on 10 to 14-day-old primary leaf segments maintained on 6 g/l of agar supplemented with 50 mg/l of benzimidazole in polistyren boxes. Conditions of incubation, inoculation and disease assessment were according to Hsam and Zeller (1997).

Table 1 Phenotypic characterization of the two parental lines Langdon and G18-16 for powdery mildew resistance to 47 *Bgt* isolates

Isolate #	Collection site		Wheat species collected from	Wheat cultivar	Infection type	
	Country	Site			G18-16	Langdon
1	Israel	Hula Valley	*T. aestivum*	Dariel	R	S
4	Israel	Hula Valley	*T. aestivum*	Dganit	S	S
6	Israel	Hula Valley	*T. aestivum*	Atir	R	S
7	Israel	Hula Valley	*T. aestivum*	Shapir	S	S
8	Israel	Hula Valley	*T. durum*	Bareket	R	S
9	Israel	Hula Valley	*T. aestivum*	652	S	S
13	Israel	Hula Valley	*T. aestivum*	519	R	S
15	Israel	Yavor	*T. durum*	Inbar	R	S
16	Israel	Nahal Oz	*T. durum*	Inbar	R	S
20	Israel	Ein Hanatziv	*T. durum*	Inbar	R	S
25	Israel	Bet Dagan	*T. durum*	Inbar	R	R
29	Israel	Ein Hanatziv	*T. aestivum*	Shapir	R	S
30	Israel	Talmei Yafe	*T. aestivum*	Shapir	R	S
33	Israel	Erez	*T. aestivum*	Dganit	S	S
36	Israel	Lahav	*T. aestivum*	Dganit	R	S
37	Israel	Nahal Oz	*T. aestivum*	Dganit	R	S
43	Israel	Yesodot	*T. aestivum*	Bet Hashita	R	S
44	Israel	Negev	*T. aestivum*	Miriam	R	S
47	Israel	Sa'ad	*T. aestivum*	Bet Lehem	R	R
50	Israel	Nahal Oz	*T. aestivum*	Barkai	R	S
52	Israel	Dir El Balakh	–	–	R	S
58	Israel	Ammiad	*T. dicoccoides*	58	S	S
61	Israel	Tabigha	*T. durum*[a]	ZB	S	S
63	Israel	Gilbboa	*T. dicoccoides*	–	S	S
64	Israel	Karei Deshe	*T. dicoccoides*	–	S	S
66	Israel	Ammiad	*T. dicoccoides*	–	S	R
67	Israel	Lahav	*T. durum*	Inbar	R	S
68	Israel	Bet Dagan	*T. aestivum*	Bet- Hashita	S	S
70	Israel	Beeri	*T. aestivum*	Dganit	R	S
91	Israel	Sde Eliahu	*T. durum*	Inbar	R	S
92	Israel	Tel Aviv University	*T. dicoccoides*	–	R	S
94	Israel	Ein Hanatziv	*T. aestivum*	Dganit	R	S
95	Israel	Ein Hanatziv	*T. durum*	Bareket	S	S
96	Israel	Negba	*T. aestivum*	M50	R	S
97	Israel	Negba	*T. durum*	M	S	S
101	Israel	Nahal Oz	*T. aestivum*	Dariel	R	S
103	Israel	Ammiad	*T. dicoccoides*	–	R	R
106	Israel	Nahal Oz	*T. aestivum*	Atir	R	S
107	Israel	Nahal Oz	*T. aestivum*	Shapir	R	R
108	Israel	Nahal Oz	*T. aestivum*	Bareket	R	S
109	Israel	Tel Aviv University	*T. dicoccoides*	–	S	S
113	Israel	Ammiad?	*T. dicoccoides*	–	R	S
96229	Switzerland	Ellighausen-Kloten Rd.	*T. aestivum*[a]	–	R	S
96224	Switzerland	Winterthur-Kloten Rd.	*T. aestivum*[a]	–	R	S
96236	Switzerland	Oensingen-Schoetz Rd.	*T. aestivum*[a]	–	R	S
96244	Switzerland	Coppet-Yverdon Rd.	*T. aestivum*[a]	–	R	S
96275	Switzerland	Nyon-Cheseaux Rd.	*T. aestivum*[a]	–	R	S

Infection type: *R* resistant (IT = 0–2), *S* susceptible (IT = 3–4)

[a] Approximation, i.e., the species powdery mildew was collected from is uncertain. In order to obtain a set of Swiss representative random mildew samples, a car, with a spore living trap mounted on its roof, was driven through the main wheat growing areas of Switzerland. Those areas are dominated by bread wheat cultivars

Inoculum was first produced on leaf segments of the susceptible wheat cv. Chinese Spring and dispersed in a settling tower over the plant material at densities of ~500 spores cm^{-2}. The boxes were incubated at 15°C with 12 h photoperiod of white fluorescent light (photosynthetic photon flux density of 55–65 µmol m^{-2} s^{-1}). Results were scored about 12 days after inoculation and once again 2–5 days later. Each box included segregating genotypes along with three replicates of a susceptible genotype (cv. Chinese spring) and the two parental lines as controls. The infection types (IT) of powdery mildew were recorded for symptoms on a scale of 0–4, with 0 representing no visible symptoms and with values of 1, 2, 3 and 4 representing highly resistant, resistant, susceptible and highly susceptible reactions, respectively (Mains and Dietz 1930). Score of 0–2 was recorded as resistant and 3–4 as susceptible.

Analysis of SSR and STS markers

PCR reactions of simple sequence repeat (SSR) and sequence tag site (STS) markers were carried out in a 20 µl reaction volume under the following conditions: one denaturation cycle at 94°C for 5 min, followed by 35 cycles of 94°C for 60 s, 50–65°C (depending on the primer) for 60 s, and 72°C for 90 s, followed by an elongation step of 72°C for 7 min. Fragment analysis was carried out on an automated laser fluorescence (ALF) sequencer using the computer program Fragment Analyzer ver. 1.02 (Amersham Biosciences, USA) comparing with internal size standards (Röder et al. 1995). The bread wheat cultivar Chinese Spring was used as a reference in each run to ensure size accuracy and avoid run-to-run and gel-to-gel variations. Genotyping of the SSR and STS markers was also preformed on 1–3% MetaPhor® Agarose (Cambrex, Rockland, ME, USA) stained with GelRed™ (Biotium, Hayward, CA, USA).

Developing CAPS marker $XBE442572_{GCTAGC}$

Wheat expressed sequence tags (ESTs) and genome-specific primers (GSPs) to chromosome arm 7AL distal region were obtained from the wheat single nucleotide polymorphism (SNP) database (http://wheat.pw.usda.gov/SNP/new/index.shtml). Cleavage amplified polymorphic sequence (CAPS) marker $XBE442572_{GCTAGC}$ was developed as described by Distelfeld et al. (2006). Comparison of 980 bp BE442572 PCR product of LDN with G18-16 sequence revealed the presence of six SNPs, one of which was used to develop a CAPS marker. Digestion of the amplification product with restriction enzyme *NheI* yielded fragments of 500 and 480 bp in LDN while leaving the 980-bp G18-16 product uncut (Supplementary material: Fig S1, Table S1). This 7AL locus was designated $XBE442572_{GCTAGC}$. The GenBank accession number of this EST provides a unique locus identifier together with the subscript indicating the base pair in the EST that carries the SNP.

Genetic mapping

A genetic linkage map of 2,317 cM was previously developed using the same 152 RIL mapping population based on 197 SSR and 493 DArT markers (Peleg et al. 2008). Chromosome 7A of the above genetic map included 38 markers. In the current study, we added 16 markers to this map in order to increase the marker saturation in the target region. For each segregating marker, a χ^2 analysis was performed to test for deviation from the 1:1 expected segregation ratio in the RIL population. Linkage analysis and map construction were performed based on the evolutionary strategy algorithm included in the MultiPoint package (Mester et al. 2003), as described in Peleg et al. (2008). The wheat EST sequences of STS and CAPS markers were aligned by BLASTN to the rice (*Oryza sativa* L.) genome sequence (using both http://blast.jcvi.org/euk-blast/index.cgi?project=osa1 and http://rice.plantbiology.msu.edu) and to the *Brachypodium distachyon* L. genome sequence (JGI 8X Brachy sequence, http://blast.brachybase.org/). In addition, ten of the DArT marker sequences, kindly provided by Diversity Arrays Technology Pty Ltd (Yarralumla, Australia), were compared in silico to search for colinearity with the *Brachypodium* and rice genomes.

Results

Reactions of the two parental lines to powdery mildew isolates

The reactions of the two parental lines (LDN and G18-16) to a set of 42 Israeli and 5 Swiss powdery mildew isolates are listed in Table 1. The cultivated durum line (LDN) was susceptible to most (37) Israeli *Bgt* isolates and to the 5 Swiss isolates. The wild emmer accession (G18-16) was resistant to most (29) Israeli *Bgt* isolates and to the Swiss isolates and susceptible to 13 Israeli isolates (Table 1). The Israeli isolate *Bgt*#15 was selected for further genetic mapping studies and was used to test a set of *Pm* differential wheat accessions (Table 2). *Bgt*#15 was virulent on lines carrying *Pm1a*, *Pm1e*, *Pm2*, *Pm3a*, *Pm3c*, *Pm3f*, *Pm3g*, *Pm4b*, *Pm5a*, *Pm5b*, *Pm6* and *Pm7* and avirulent on *Pm1b*, *Pm3b*, *Pm3d*, *Pm4a*, *Pm17* and *Pm9* (this last avirulence is based on the susceptibility of the line Normandie, which includes the *Pm* combination of *Pm1a* + *Pm2* + *Pm9*). The inoculation of differential lines *Pm8* and *Pm3e* resulted in an intermediate reaction, IT = 2–3 (Table 2).

Table 2 Phenotypic response of wheat differential lines and genotypes possessing known *Pm* genes for resistance to the powdery mildew isolate *Bgt*#15

Cultivar/line	*Pm* gene	Infection type
Axminister/8*Cc	*Pm1a*	S
MocZlatka	*Pm1b*	R
Virest	*Pm1e*	S
Ulka/8*Cc	*Pm2*	S
CItr14120	*Pm3a*	S
CItr14121	*Pm3b*	R
Sonora/8*Cc	*Pm3c*	S
Kolibri	*Pm3d*	R
W150	*Pm3e*	M
Michigan	*Pm3f*	S
Amber/8*Chancellor	*Pm3g*	S
Khapli/8*Cc	*Pm4a*	R
Weihenstephan	*Pm4b*	S
Hope	*Pm5a*	S
Ibis	*Pm5b*	S
TP114/2*Starke	*Pm6*	S
Transec	*Pm7*	S
Disponent	*Pm8*	M
Amigo	*Pm17*	R
Normandie	*Pm1a* + *Pm2* + *Pm9*	R
TA2033	*Mlm2033*	R

R resistant (IT = 0–2), *S* susceptible (IT = 3–4), *M* intermediate reaction (IT = 2–3)

Inheritance of powdery mildew resistance derived from wild emmer G18-16

The wild parent (G18-16) was highly resistant to the powdery mildew isolate *Bgt*#15, whereas LDN was highly susceptible to this isolate (IT scores 0 and 4, respectively; Fig. 1). The phenotypic test of the F_{2-3} population (93 F_{2-3} families) showed the following segregation: 17 resistant, 53 segregating and 23 susceptible families. This segregation pattern fits the theoretical expected ratio of 1:2:1 for F_{2-3} families (Table 3). In the second stage, 152 F_7 RILs developed from the original F_2 population were inoculated with *Bgt*#15. The results showed a segregation ratio of 68 resistant and 75 susceptible RILs (9 RILs were excluded due to missing phenotypic data). This segregation pattern fits the expected theoretical ratio of 1:1 for an F_7 RIL population (Table 3), thus confirming the involvement of a single dominant gene in conferring resistance to *Bgt*. The newly identified gene was temporarily designated *PmG16*.

Genetic and physical mapping of PmG16

To map the *PmG16* gene, we used the genetic linkage map of 2,317 cM, previously developed using the 152 RIL population (LDN × G18-16) based on 197 SSR and 493 DArT markers (Peleg et al. 2008). *PmG16* was located to the distal end of chromosome arm 7AL. To saturate the map in the *PmG16* region, 16 additional markers, all but $XBE442572_{GCTAGC}$ linked to known *Pm* genes on 7AL, were analyzed. These included the SSR markers *Xwmc809* (Somers et al. 2004), *Xcfa2019*, *Xcfa2040*, *Xcfa2257*, *Xcfa2240* (Sourdille et al. 2004), *Xbarc275* (Song et al. 2005) and *Xgwm1066* (Ganal and Röder 2007), and the STS markers *Xsts*638 (Hu et al. 1997), *XstsBE406627*, *XstsBE445653* (Perugini et al. 2008), *Xmag1705*, *Xmag1759*, *Xmag1986*, *Xmag2185* (Yao et al. 2007) and *YP7A* (He et al. 2008). A new genetic linkage map was then constructed for chromosome arm 7AL using 46 markers. Markers interfering with map stability were removed and the 7A linkage group was re-analyzed to construct a stabilized map, as recommended by Mester et al. (2003). As a result, a map consisting of 32 SSR, DArT, STS and CAPS markers and the *PmG16* gene provided a basic map for calculating recombination frequencies and centiMorgan (cM)

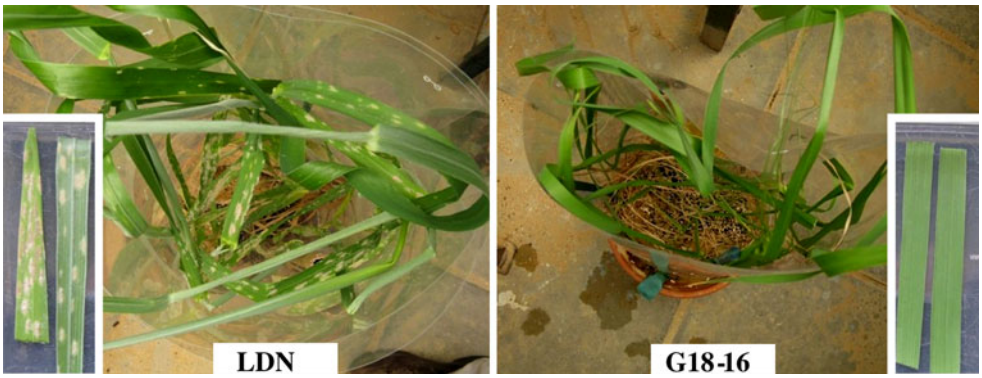

Fig. 1 Phenotypic response of the two parental lines of the mapping population, the durum wheat cv. Langdon and wild emmer wheat acc. G18-16, at adult stage after inoculation with isolate *Bgt*#15

Table 3 Segregation ratio in F_{2-3} and RIL mapping populations of the cross between the durum wheat cv. Langdon and wild emmer G18-16 in response to inoculation with isolate *Bgt*#15

Population type	Size	Segregation						
		Type	Expected ratio	R	S	H	χ^2	*P* value
F_{2-3}	93[a]	Dominant	A:H:B = 1:2:1	17	23	53	2.6	0.27
RIL	143	Dominant	A:B = 1:1	68	75	–	0.35	0.55

[a] 20 plants for each family

distances. Five SSR (*Xwmc809, Xcfa2019*, *Xcfa2040*, *Xcfa2257* and *Xgwm1066*), two STS (*Xmag2185 and XstsBE445653*) and one EST-based markers (*XBE 442572*$_{GCTAGC}$; see Supplementary Material) were added to the final version of the genetic map presented in the current study. The *PmG16* gene was genetically mapped to a 3.6 cM interval between the markers *Xgwm344* and *wPt-9217* (Fig. 2). The two DArT markers, *wPt-1424* and *wPt-6019*, were located on the same map interval.

Several markers that mapped in the *Pm*G16 region were previously linked to the physical deletion bin map of chromosome arm 7AL (Fig. 2). Markers *Xgwm282*, *Xgwm332*, *Xcfa2019* and *Xcfa2040*, located proximally to *PmG16*, were previously assigned to chromosome deletion bin 7AL-16 0.86-0.90 (Sourdille et al. 2004). The marker *Xcfa2257*, mapped distally to *PmG16,* is the only marker which was physically mapped to a proximal bin (7AL-1 0.39-0.71) (Sourdille et al. 2004). The EST-based marker *XBE4 42572*$_{GCTAGC}$ served as an additional anchor to the physical deletion bin map of wheat and was assigned to chromosome bin 7AL-21 074-0.86 (http://wheat.pw.usda.gov/SNP/new/index.shtml; Fig. 2).

Analysis of DArT marker sequences revealed two incidences of high intra similarity between markers mapped by Peleg et al. (2008) to the same interval. High sequence identity (>92%) was found between *wPt-1424*, *wPt-1023*

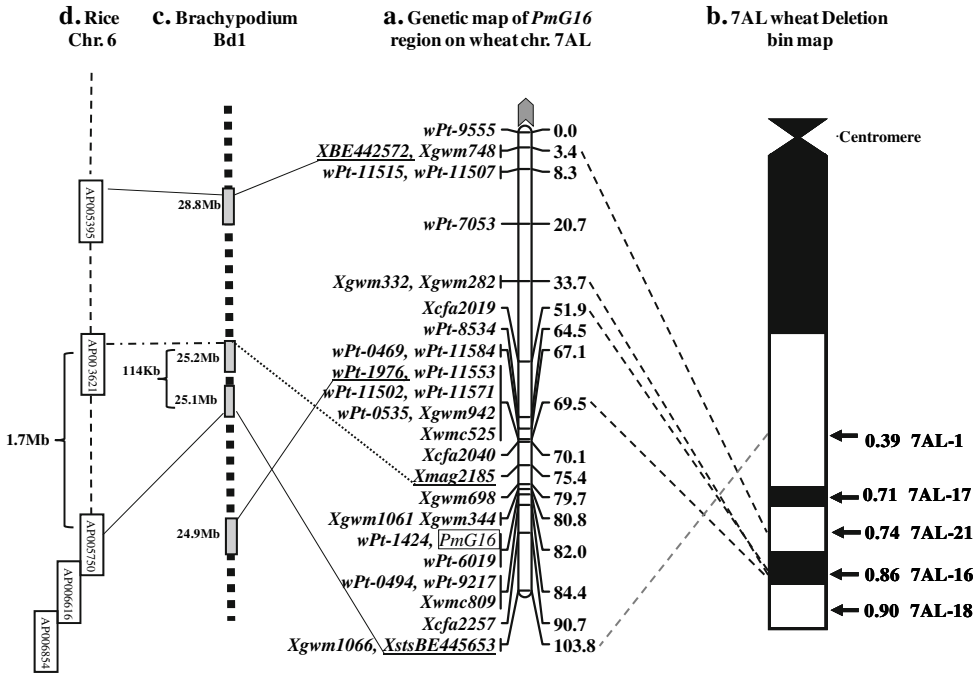

Fig. 2 Genetic map of chromosome arm 7AL of wheat containing powdery mildew resistance gene *PmG16* and anchored markers for the physical map of the chromosome arm 7AL and sequence of *Brachypodium* chromosome Bd1 and rice chromosome 6. **a** Genetic map of the *PmG16* gene region on wheat chromosome arm 7AL. Markers are shown on the *left* with map distances on the *right*. Molecular markers that were previously assigned to the 7A wheat deletion bin map (**b**) are connected to the physical map with *dashed lines*. The *PmG16* locus is framed with a *black line*. The four markers which served as anchors, establishing colinearity between the *PmG16* genetic map and the sequences of *Brachypodium* and rice, are *underlined*. **c** The homologous regions on *Brachypodium* Bd1 are marked with *gray bars*. Approximate locations (bp) along Bd1 sequence are shown on the *left*. The *round dot line* linking the *Xmag2185* anchor marker to the *Brachypodium* Bd1 indicates intermediate homology values (*E* value = 2.00E-12) to the *Brachypodium* gene Bradi1g29670.1. The *dashed dot line* connects this gene to the rice chromosome 6 BAC AP003621 sequence through homology to the rice locus LOC_Os06g49390. **d** The homologous regions on rice chromosome 6 BACs are marked with *white bars* (BAC No. included). AP00575, AP006616 and AP006854 are BACs representing the telomeric region of rice chromosome arm 6L. In both **c** and **d** the colinear genomic regions spanning the *PmG16* locus between markers *Xmag2185* and XstsBE4456531, 114 kb in *Brachypodium* and 1.7 Mb in rice, are marked with *brace*. In both **c** and **d** the length of the *bars* is not drawn to scale

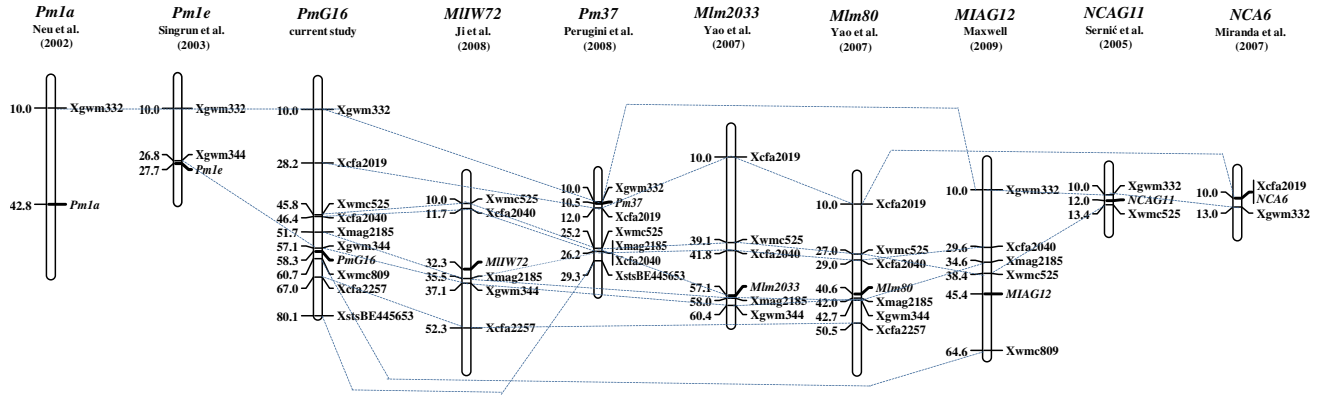

Fig. 3 Comparative view of the *PmG16* linkage map with other previously published *Pm* loci on chromosome arm 7AL. Map position of *Pm1a* is according to Neu et al. (2002), map position of *Pm1*e is according to Singrün et al. (2003), map position of *MlIW72* is according to Ji et al. (2008), map position of *Pm37* is according to Perugini et al. (2008), map position of *MIm2033* and *MIm80* is according to Yao et al. (2007), map position of *MIAG12* is according to Maxwell et al. (2009), map position of *NCAG11* is according to Srnić et al. (2005) and map position of *NCA6* is according to Miranda et al. (2007). Markers are shown on the *right* with map distances on the *left*. Common markers are connected with *dotted lines*

and *wPt-1259* and also between wPt-1976 and *wPt-3403*. Therefore, *wPt-1023*, *wPt-1259* and *wPt-3403*, which were included in the genetic map presented by Peleg et al. (2008), were omitted from the current genetic map.

Comparison of *PmG16* with other *Pm* genes on 7AL

The *PmG16* gene was compared to previously published *Pm* genes assigned to 7AL (Fig. 3). Five dominant alleles of *Pm1* were identified for this gene in *T. aestivum* (*Pm1a* and *Pm1e*), *T. monococcum* (*Pm1b* and *Pm1c*) and *T. aestivum* var. *spelta* (*Pm1d*) (Hsam et al. 1998; Singrün et al. 2003). Phenotypic test of a differential line carrying the *Pm1a* allele with *Bgt*#15 showed a susceptible reaction, thus differentiating *PmG16* from *Pm1a* (Table 2). The SSR marker *Xgwm344*, previously reported to be closely linked to *Pm1c* and *Pm1e* alleles (0.5 and 0.9 cM, respectively; Singrün et al. 2003; Stepien et al. 2004) was found here to be 1.2 cM proximal to *PmG16* (Figs. 2, 3). The STS marker *Xsts638* co-segregated with the *Pm1* locus (Hu et al. 1997) in addition to *XstsBE406627* and *XstsBE445653*, which were found to be closely linked to *Pm1* (Perugini et al. 2008). While the former two showed no polymorphism between the parental lines used in the current study (LDN and G18-16) the latter was mapped 21.8 cM distally from the *PmG16* on chromosome arm 7AL (Figs. 2, 3).

Four other *Pm* genes, *Mlm2033*, *Mlm80* (Yao et al. 2007), *MlIW72* (Ji et al. 2008) and *MlAG12* (Maxwell et al. 2009), were located in the *Pm1* genomic region. The two genes, *Mlm2033* and *Mlm80*, derived from *T. monococcum*, were mapped to the *Xcfa2040–Xmag2185* genetic interval (Figs. 2, 3). In addition, *MlIW72* gene derived from *T. dicoccoides* was mapped to the same genomic interval (Ji et al. 2008) as *Mlm2033* and *Mlm80*. In the current study, *PmG16* was located in a more distal interval defined by markers *Xgwm344–Xcfa2257* (Figs. 2, 3). Three other EST-based STS markers (*Xmag1705*, *Xmag1759* and *Xmag1986*) located in the same region (Yao et al. 2007) could not be mapped onto the current LDN X G18-16 map due to lack of polymorphism between the parental lines.

Several other *Pm* genes were also reported in the 7AL region, including two recessive *Pm* genes (*Pm9*, *mlRD30*), three genes derived from *T. monococcum* (*NCAG11* and *NCA4*, Srnić et al. 2005; and NCA6, Miranda et al. 2007) and one form *T. timopheevii* (*Pm37*, Perugini et al. 2008). These four dominant genes were mapped to a proximal segment relative to the *PmG16* gene (Fig. 3).

Colinearity between *PmG16* region and *Brachypodium* and rice genomes

CAPS marker $XBE442572_{GCTAGC}$ and STS marker *XstsBE445653* were mapped proximally and distally to the *PmG16* gene, respectively. $XBE442572_{GCTAGC}$ showed homology to *Brachypodium* Bd1 at physical position 28,892,870 bp (*E* value = 3.00E-40) and rice BAC AP003935 (*E* value = 1.30E-26) on chromosome 6. *XstsBE445653* showed homology to *Brachypodium* Bd1 at physical position 25,086,137 bp (*E* value = 1.00E-43) and to rice BAC AP005750 (*E* value = 2.9E-22) (Fig. 2). Two markers located in the *PmG16–*$XBE442572_{GCTAGC}$ interval, the STS marker *Xmag2185* and the DArT marker *wPt-1976*, served as additional anchors for comparative genomics. The sequence of STS marker *Xmag2185*, located 6.6 cM from *PmG16*, showed homology to *Brachypodium* Bd1 gene Bradi1g29670.1 (genomic location 25,200,121 bp, *E* value = 2.00E-12). The Bradi1g29670.1 sequence showed high homology to rice BAC AP003621 (*E* value = 2.80E-113) on chromosome 6 and therefore established a third anchor, hence confirming the wheat–*Brachypodium*–rice colinearity

in this genomic region (Fig. 2). The Bradi1g29670.1 sequence (5.6 kb long) showed also high homology to sequences located on other rice chromosomes, such as chromosomes 2, 5 and 1 (*E* value < 2.80E-135).

wPt-1976 showed homology to *Brachypodium* Bd1 at position 24,922,411 bp (*E* value = 7.00E-28), breaking the synteny along *Brachypodium* Bd1 and wheat chromosome arm 7AL. Furthermore, *wPt-1976* also showed homology to rice chromosome 11 (*E* value = 1.7E-25) and moderate homology to rice chromosome 4 (*E* value = 3.4E-15) and to the proximal region of chromosome 6 (*E* value = 1.2E-13), thereby disrupting the colinearity between wheat chromosome arm 7AL and rice chromosome arm 6L (Fig. 2). The marker *wPt-0494* showed only low homology to *Brachypodium* Bd1 and Bd4 (*E* value > 3.00E-09) and no homology to rice BAC sequences.

Discussion

Characterization and chromosomal location of *PmG16*

In the current study, a single dominant powdery mildew resistance gene derived from wild emmer was identified using the progeny of a cross between domesticated durum wheat and its wild progenitor. Isolate *Bgt*#15 was found virulent on a wide range of *Pm* genes (Table 2) and was previously reported as highly virulent on a large collection of domesticated and wild wheat germplasm (71% of the accessions were susceptible, Ben-David et al. 2008). The phenotypic reaction of the RIL population to this isolate was mapped to genomic region on wheat chromosome arm 7AL and was temporarily designated *PmG16*. Phenotypic response to *Bgt*#15 isolate at the adult plant stage of the two parental lines showed similar pattern of resistance in the wild parent G18-16 (Fig. 1) as found at the seedling stage.

Genome mapping of all 14 chromosomes of tetraploid wheat located the *PmG16* gene to the long arm of chromosome 7A. Further molecular zooming on this genomic region indicated that *PmG16* is located in a 3.6 cM interval between *Xgwm344* and *wPt-0494* (Fig. 2). Additionally, two DArT markers (*wPt-1424* and *wPt-6019*) co-segregated with *PmG16* on the same map interval. Previously mapped markers enabled us to assign *PmG16* to wheat chromosome deletion bin 7AL-16 0.86-0.90. Eight SSR and CAPS markers that physically mapped to chromosome arm 7AL were found to be linked to *PmG16* and served as anchors to the 7AL deletion bin map (Fig. 2). Notably, based on the markers *Xcfa2040* and *Xcfa2019*, the more distal marker *Xcfa2257*, assigned to chromosome bin 7AL-1 by Sourdille et al. (2004), was genetically mapped in the current study to a more distal chromosome interval, which corresponds to chromosome bin 7AL-16 (Fig. 2). Such discrepancies between genetic and physical maps have been previously reported (Sourdille et al. 2004). Nevertheless, the map order found in the current study is highly similar to previously published wheat maps (e.g. Somers et al. 2004; Ganal and Röder 2007) and linkage mapping of chromosome arm 7AL (e.g. Yao et al. 2007; Ji et al. 2008; Perugini et al. 2008).

Remarkable clustering of DArT markers was reported in gene-rich telomeric regions (e.g., Peleg et al. 2008; Akbari et al. 2006). In the current study, we noted high similarity between some of the DArT sequences that were mapped to the same cluster of markers on chromosome arm 7AL. These results may suggest that the observed clusters of DArT markers found by Peleg et al. (2008) are actually an outcome of a duplication of these DArT sequences during the process of marker development.

Colinearity of *PmG16* gene region to rice and *Brachypodium*

Four markers, $XBE442572_{GCTAGC}$, *wPt-1976*, *Xmag2185* and *XstsBE445653*, served as anchors between the *PmG16* region on chromosome arm 7AL and *Brachypodium* Bd1 chromosome. In addition, three out of these four markers (excluding *wPt-1976*) established colinearity to rice chromosome arm 6L. The two markers, *Xmag2185* and *XstsBE445653*, spanning the *PmG16* region in a 28.4 cM interval established colinearity with the currently available draft (8× depth) of *Brachypodium* genome on chromosome Bd1 and to the distal end of rice chromosome arm 6L. The physical stretch between those two markers in *Brachypodium* and rice includes 114 kb and 1.7 Mb (comprised of 10 rice BAC clones), respectively.

Some discrepancies in the colinearity between wheat and *Brachypodium* have been previously reported (e.g., Huo et al. 2009) and our data suggests that marker *wPt-1976* is creating such macro colinearity disruption (Fig. 2). Still the results obtained in the current study demonstrate the utilization of this relatively new tool of *Brachypodium* genome sequence for the fine mapping of wheat genes.

The firm colinearity found near the *PmG16* region of wheat and rice is in accordance with other studies. Yao et al. (2007) mapped two powdery mildew resistance genes (*Mlm80* and *Mlm2033*) on chromosome arm 7AL (interval *Xmag2185–Xcfa2040*) of diploid *T. monococcum* (genome AA) and found a distal anchor marker (*Xmag1986*) to BAC clone (AP006616), adjacent to the current distal anchor (*XstsBE445653*), on the annotated rice sequence. Quarrie et al. (2006) found high level of colinearity between wheat ESTs assigned to chromosome deletion bin 7AL-16 0.86-0.90 and rice chromosome arm 6L, which was also in agreement with the results of Hossain et al. (2004).

The accumulated data on rice genome suggest that it has a limited benefit for map-based cloning of R-genes in cereals because of nonsyntenic map locations of R-genes between cereal species (Leister et al. 1998; Quraishi et al. 2009). Similar situation is probably true also for the use of *Brachypodium* genome as a model for positional cloning of R-genes in other cereals. Bossolini et al. (2007) found only partial colinearity between genes in the *Lr34* genomic region and the *Brachypodium* genomic sequence. Nevertheless, it seems that *Brachypodium* and rice genomes will continue to serve as models for genomic studies in large genomes, such as wheat and barley, until these genomes are fully sequenced. In the case of R-genes, even if the target genes are missing from the orthologous position in rice or *Brachypodium*, the flanking colinear genes can serve as potential markers in map-based cloning efforts of wheat and barley disease resistance genes (Quraishi et al. 2009).

Comparison of *PmG16* with other *Pm* genes on 7AL

Genomic regions located near telomeres are hot spots for chromosome evolution and recombination and are therefore associated with the emergence of novel genes and gene duplication (See et al. 2006; Akhunov et al. 2003). These processes may result in the formation of R-gene clusters. R loci may represent single genes with multiple alleles (Shepherd and Mayo 1972; e.g., *Pm1* locus with six known alleles; Hsam et al. 1998; Singrün et al. 2003) and on the other hand different genes within a single cluster can confer resistance to different pathogens (Michelmore and Meyers 1998).

Furthermore, the reaction to another powdery mildew isolate (*Bgt*#70, Table 1) was mapped to the same genetic interval (*Xgwm344–wPt-0494*) as *PmG16* gene (data not shown). This resistance may be conferred by the same locus/allele or even by a different locus of powdery mildew resistance situated in the same gene cluster. Several other R-genes, including resistance to powdery mildew (*Pm1*, *Pm9* and *mlRD30*; Schneider et al. 1991; Singrün et al. 2004), leaf rust (*Lr20*; Sears and Briggle 1969), stem rust (*Sr15*; Hu et al. 1997), *Fusarium* head blight (Shen and Ohm 2007) and common bunt (Fofana et al. 2008) were mapped to the same chromosome region as *PmG16*.

The *PmG16* gene was compared to previously published *Pm* genes on 7AL (Fig. 3). The *Pm1* locus was identified in the early 1950s (Pugsley and Carter 1953) and later on was located to chromosome arm 7AL (Sears and Briggle 1969). Our temporarily designated gene *PmG16* was genetically mapped to the same map interval as the *Pm1* locus (Fig. 3) and was distinguished phenotypically only from *Pm1a* allele based on allelic tests (Table 2). Recently, *MlAG12* gene derived from *T. timopheevii* was mapped to *Xwmc809–Xmag2185* region (Maxwell et al. 2009), a congruent genetic interval relative to the *PmG16* gene. Bearing this in mind, and although *PmG16* was mapped to a different interval than *Mlm2033*, *Mlm80* and *MlIW72*, additional studies are necessary to clearly differentiate between these four powdery mildew resistance genes and the new *PmG16* gene. Such additional studies could include testing the different resistant wheat genotypes with different sources of *Bgt* inoculums [e.g., *T. monococcum* accession TA2003 showed resistant response to *Bgt*#15 (Table 2)]. Moreover allelic tests of the *Pm* gene cluster located on 7AL could resolve the situation by implementing a more precise approach and determine the number of different genes or alleles of the same gene that are involved. However, the currently available list of differential lines covers only part of the gene\allelic variation in this gene cluster and therefore enabling only partial resolution of this problem. A more complete answer could be obtained through implementation of the map-based cloning approach (Lin et al. 2007) of one or more of the genes comprising this cluster.

The isolation of the fungal disease resistance genes *Lr10* (Feuillet et al. 2003), *Lr21* (Li et al. 2009) and *Pm3b* (Yahiaoui et al. 2004) and the recent cloning of *Yr36* and *Lr34* has shown that map-based cloning is now within reach, even in hexaploid wheat (Feuillet and Keller 2005; Fu et al. 2009; Krattinger et al. 2009). The advances in development of new genomic tools for wheat studies, together with the construction of the physical map of chromosome 7A, that is under way (http://www.wheatgenome.org/index.php), are making such an approach even more feasible. The currently ongoing map-based cloning project of *MlIW72* on the distal end of 7AL is the first step towards the exploration of such a genomics-based approach (Liu et al. 2010).

Conclusions and implementations for wheat improvement

The continuous increase in global human population poses huge challenges to world agriculture. Developing cultivars with good resistance to powdery mildew is the most economical and effective approach to reduce fungicide application and minimize grain-yield losses. The notion of using the wild emmer gene resources for wheat improvement has been repeatedly advocated since the discovery of the wild progenitor of cultivated wheats about a century ago (Aaronsohn 1910). In the current study, a cross between domesticated and wild wheat was used to map a newly discovered powdery mildew resistance gene. This single dominant gene, designated as *PmG16*, was derived from wild emmer wheat and mapped to chromosome arm 7AL. The colinearity between *PmG16* genomic region and the rice and *Brachypodium* genomes will advance future fine mapping and cloning of *PmG16* gene.

The resistance of the wild parent (G18-16) to a wide collection of *Bgt* isolates (Table 1) and the mapping of resistance to *Bgt*#70 to the same genetic interval as *PmG16* suggest a significant potential for further wheat breeding. The dense genetic map of *PmG16* region presented in the current study can promote its utilization via marker-assisted selection (MAS) in wheat breeding programs. Furthermore, simultaneous transfer of large DNA fragments containing multiple R-genes could act synergistically (Rommens and Kishore 2000). Therefore, transposing the tightly linked R-genes cluster found near the *PmG16* region could enhance the durability of *PmG16* resistance. Alternatively, the use of molecular markers for pyramiding of R-genes can allow the combination of several single R-genes together to increase resistance durability (Feuillet and Keller 2005).

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