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Chromosomal location of genes for resistance to powdery mildew in common wheat (*Triticum aestivum* L. em Thell.) 6. Alleles at the *Pm5* locus

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Abstract Genetic characterization of powdery mildew resistance genes were conducted in common wheat cultivars Hope and Selpek possessing resistance gene *Pm5*, cvs. Ibis and Kormoran expressing resistance gene *Mli*, a backcross-derived line IGV 1–455 and a *Triticum sphaerococcum* var. *rotundatum* Perc. line Kolandi. Monosomic analyses revealed that one major recessive gene is located on chromosome 7B in the lines IGV 1–455 and Kolandi. Allelism tests of the F_2 and F_3 populations involving the tested resistant lines crossed with either cv. Hope or Selpek indicated that their resistance genes are alleles at the *Pm5* locus. The alleles are now designated *Pm5a* in Hope and Selpek, *Pm5b* in Ibis and Kormoran, *Pm5c* in *T. sphaerococcum* var. *rotundatum* line Kolandi, and *Pm5d* in backcross-derived line IGV 1–455, respectively.

Keywords *Triticum aestivum* · Powdery mildew resistance · Monosomic analysis · Gene location · Recessive gene · Allelism

Introduction

Powdery mildew, caused by *Erysiphe graminis* (*Blumeria graminis*) DC. f. sp. *tritici*, is a destructive foliar disease of common wheat in areas with cool or maritime climates. Deployment of resistance genes is the most economical and environmentally safe method by reducing the application of fungicides to combat this disease. Because of the co-evolution of host and pathogen race-specific resistance genes can be overcome by new races of the pathogen possessing corresponding virulence genes. Hence, it is beneficial to search for new sources of resistance to this disease. Twenty-five gene loci for resistance to powdery mildew (*Pm1-Pm25*) have been assigned to specific chromosomes (Szunics and Szunics 1999; McIntosh et al. 1998). In addition, resistance alleles are present at the *Pm1* (Hsam et al. 1998), *Pm3* (Zeller et al. 1993), *Pm4* (Baier et al. 1973; The et al. 1979) and *Pm8* (Hsam and Zeller 1997) loci. Among the 25 powdery mildew resistance genes, the gene *Pm5* derived from Yaroslav Emmer (*T. dicoccum* Schübl) and initially introgressed into common wheat cultivar Hope is the only powdery mildew resistance gene in *Triticum aestivum* exhibiting a recessive mode of inheritance.

Powdery mildew resistance of cv Hope was first described by Mains (1933). The recessive gene was subsequently located on chromosomal arm 7BL (Law and Wolfe 1966; McIntosh et al. 1967) and designated as *Pm5* (Lebsock and Briggle 1974). Bennett (1984) described a mildew resistance gene temporary symbolized as *Mli* in cvs. Ibis, Aquila, Flanders and Rothwell Perdix. Heun and Fischbeck (1987), on the basis of tests with 6 *Erysiphe graminis tritici* (*Egt*) isolates, reported that the disease reaction of Aquila, Flanders and other cultivars carrying *Mli* was very similar to that of Hope possessing *Pm5*. However, the evidence shown was not conclusive as both *Mli* and *Pm5* showed either intermediate or susceptible disease reponses to the isolated used. Hovmøller (1989) and Winzeler et al. (1991) obtained similar results between *Pm5* and *Mli*. Pedigree analyses of the German wheat cultivars carrying the resistance gene *Mli* indicated that this source of resistance originates from the Hindukush material collected in the early 1930s (Nover 1942). Although preliminary studies revealed that cultivars and lines possessing either *Mli* or *Pm5* exhibited similar disease reactions to some *Egt* isolates (Lutz, unpublished data), their disease reactions could be distinguished clearly by the use of additional *Egt* isolates. Hence, further tests were conducted on

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cultivars and lines showing similar disease responses to a set of 11 differential *Egt* isolates routinely used in Weihenstephan (Zeller et al. 1998) and those possessing a recessive mode of monogenic inheritance.

The study reported here was a phytopathological and genetic analysis of gene *Pm5* in Hope and Selpek, gene *Mli* in Ibis and Kormoran as well as resistance genes localized on chromosome 7B in *T. sphaerococcum* var. *rodundatum* line Kolandi and backcrossed-derived lines IGV 1–455 and IGV 2–556, respectively, and the results demonstrate that the genes are either alleles at the *Pm5* locus or else a closely linked cluster of genes.

Materials and methods

German wheat cultivars and lines previously described to possess either *Pm5* or *Mli* (Heun and Fischbeck 1987) were obtained from the respective breeders at the time of cultivar registration and maintained at Weihenstephan. The backcrossed-derived wheat lines IGV 1–455 and IGV 2–566 were provided by J. Mac Key, Uppsala, Sweden. *T. sphaerococcum* var. *rotundatum* (ATRI 2996/74) line Kolandi originated from India and was provided by Gene Bank Gatersleben, Germany. Cultivars Hope and Redman were provided by H. Bockelman, USDA, Aberdeen, Idaho; cv. Wattiness by R. Herzog, Templeuve, France; Çucurova -86 and Kirkpinar-79 by R. Ercan, Diskapi-Ankara, Turkey; the Russian cultivars Kutuluskaya and Zolotistaya by H. Peusha, Tallinn, Estonia; Ilona and Regina from P. Bartoš, Prague, Czech Republic; Sicco from the National Institute of Agricultural Botany, Cambridge, England; Nadadores and Siete Cerros from CIMMYT, Mexico, and Una from S. Borojevic, Novi Sad, Yugoslavia.

The Chinese Spring (CS) monsomics used for locating the resistance genes were originally obtained from E.R. Sears, USA. All 21 Chinese Spring monosomic lines were used as the female parents in the cross with IGV 1–455 and IGV 2–556. Kolandi showed a disease reaction pattern similar to *Pm5* in a preliminary test with the standard 11 *Egt* differential isolates routinely used in Weihenstephan. The presence of *Pm5* was postulated, and hence for Kolandi crosses were made only between certain CS monosomic lines, which included among others CS monosomic 7B. Cytologically confirmed monosomic F_1 plants (2n=41) were raised to maturity in the greenhouse. Somatic chromosome counts were conducted employing the standard Feulgen method. Allelic crosses were made between representative lines and cultivars carrying either *Pm5* or *Mli* such as Hope, Selpek, Ibis and Kormoran. The lines Kolandi, IGV 1–455 and IGV 2–556 were also crossed with Hope and Selpek, after it was known that the location of the resistance gene in these lines was on chromosome 7B.

The *Egt* isolates used to postulate and differentiate the resistance genes were selected from single-spore progenies collected in Europe (Felsenstein et al. 1991), and classified under Weihenstephan accession numbers and maintained at the Institut für Pflanzenbau und Pflanzenzüchtung, Weihenstephan. The tests for mildew disease resistance were conducted on primary leaves from 10- to 15-day-old seedlings cultured on 6 g/l of agar and 35 mg/l benzimidazole in 12-chambered plastic boxes. The expression of resistance in backcrossed-derived lines IGV 1–455 and IGV 2–556 was scored at the 10-day stage, whereas in the other cultivars and lines, resistance was analysed at the 15-day-old seedlings. The methods of inoculation and the conditions of incubation and disease assessment were according to the detached leaf segment method described by Hsam and Zeller (1997). In the screening of lines using differential *Egt* isolates, three main classes of host reactions were distinguished: r=resistant (0–20% infection relative to susceptible cv. Kanzler); i=intermediate (30–50% infection); s=susceptible (>50% infection). In a combined classification, r,i means that among the 18–20 seedlings analysed, more than two-thirds showed a resistance response and the rest an intermediate reaction. In monosomic analysis and allelic tests, a 0, 1 to 4 infection-type scale of Stakman et al. (1962) was additionally used to clearly separate the tested individuals into two categories, either resistant or susceptible. Chi-square tests for goodness of fit were used to test for deviation of the observed data from the theoretical expected segregation.

Results

Disease responses of wheat lines to *E. graminis tritici*

Preliminary tests with a standard set of 11 *Egt* isolates, nos. 2, 5, 6, 9, 10, 12, 13, 14, 15, 16, and 17, with known virulences revealed that cvs. Hope. Selpek, Ibis, Kormoran and Kolandi showed a resistance response to *Egt* isolates 10 and 14, respectively, and susceptible responses to the others. This disease response pattern has been shown to be indicative of gene *Pm5* (Zeller et al. 1993). However, further tests with a collection of 104 *Egt* isolates maintained at Weihenstephan revealed that these cultivars possessed different response patterns against various race-specific *Egt* isolates. Consequently, a set of 12 *Egt* isolates was selected (Table 1) and employed to

Table 1 Differential response patterns of 8 wheat cultivars/lines possessing powdery mildew resistance alleles at the gene locus *Pm5* after inoculation with 12 isolates of *Erysiphe graminis* f. sp. *tritici*

Cultivar/line	*Erysiphe graminis tritici* isolates												Current *Pm*-genes	Proposed *Pm*-genes
	2	5	9	10	12	14	55	76	77	114	116	J2/2		
Hope	s[e]	s	s	r	s	r	i	r	s	r	r	r	*Pm5*	*Pm5a*
CS (Hope 7B)[a]	s	s	s	r	s	r	i	r	s	r	r	r	*Pm5*	*Pm5a*
Selpek	s	s	s	r	s	r	i	r	s	r	r	r	*Pm5*	*Pm5a*
Ibis	s	i, r	s, i	r	s	r	s	s	r, i	i	r	s	*Mli*	*Pm5b*
Kormoran	s	i, r	s, i	r	s	r	s	s	r, i	i	r	s	*Mli*	*Pm5b*
Kolandi[b]	i, s	s	s	r	s	r	s	i	s	s	r	i	—	*Pm5c*
IGV 1–455[c]	r	r	r	r	r	r	r	r	r	r	r	r	—	*Pm5d*
IGV 2–556[d]	r	r	r	r	r	r	r	r	r	r	r	r	–	*Pm5d*

[a] Chinese Spring (Hope 7B) substitution line
[b] *T. sphaerococcum* var. *rotundatum*
[c] Accession CI 10904, six times backcrossed to Prins
[d] Accession CI 10904, six times backcrossed to Starke
[e] r, Resistant; s, susceptible; i, intermediate

Table 2 Wheat cultivars/lines exhibiting differential disease response patterns of *Pm5* alleles

Cultivar/Line	Country	Alleles
Hope	USA	*Pm5a*
Redman	Canada	*Pm5a*
Kutulukskaya, Zolotistaya	Russia	*Pm5a*
Sicco	The Netherlands	*Pm5a*
Regina	Czech Republic	*Pm5a*
Navid	Iran	*Pm5a*
Galaxie, Tarasque	France	*Pm5a*
Lambros, Pagode, Selpek	Germany	*Pm5a*
Ilona	Czech Republic	*Pm5b*
Wattiness	France	*Pm5b*
ATRI-7584/74,Carimulti, Cariplus, Dolomit, Frühprobst, Ibis, Kontrast Kormoran, Rektor, Urban	Germany	*Pm5b*
Aquila	Great Britain	*Pm5b*
Nadadores, Siete Cerros	Mexico	*Pm5b*
Çucurova-86, Kirkpinar-79	Turkey	*Pm5b*
Una	Yugoslavia	*Pm5b*

classify the disease response patterns in cultivars and lines previously reported to carry either *Pm5* or *Mli* (Table 2). It was found that *Egt* isolates nos. 10 (virulent to *Pm3d*, *Pm4a* and *Pm8* and avirulent to *Pm1*, *Pm2*), 14 (virulent to *Pm1*, *Pm2*, *Pm4a*, *Pm4b*, *Pm8*, *Pm 1+2+9*) and 116 (virulent to *Pm6* and *Pm22*) are avirulent to all the cultivars and lines tested possessing either gene *Pm5*, *Mli* or as yet undocumented resistance genes in the lines Kolandi, IGV 1–455 and IGV 2–556, whereas the differential disease response pattern of the cultivars and lines was evidenced from the virulence patterns of the other 9 *Egt* isolates. For example, *Egt* isolate nos. 76, 77 and J2/2 clearly separated cultivars possessing either *Pm5* or *Mli*. The cultivars Hope and Selpek carrying gene *Pm5* revealed identical disease patterns. Similarly, the Chinese Spring (Hope 7B) substitution line showed the same disease response as Hope, confirming the expression of gene *Pm5* located on chromosome 7B to the *Egt* isolates currently used. Likewise, Ibis and Kormoran, previously described to possess gene *Mli*, were found to have the same response pattern. *T. sphaerococcum* var. *rotundatum* line Kolandi showed a distinctive response pattern of its own. The backcrossed-derived lines IGV 1–455 and IGV 2–556 showed identical disease reactions and were differentiated from the other wheat cultivars by possessing a wider spectrum of resistance (Table 1). A list of 29 cultivars and lines from various origins (Table 2) could be distinguished by their differential disease response patterns using the 12 selected *Egt* isolates. Twelve cultivars showed the response pattern of cv. Hope (*Pm5*), and another 18 cultivars and lines possessed the response pattern of cv. Ibis (*Mli*). The response patterns of lines Kolandi, IGV 1–455 and IGV 2–556 were not detected in the commercial cultivars currently grown in Germany.

Chromosomal location of resistance genes in wheat lines Kolandi, IGV 1–455 and IGV 2–566

The disomic F_2 population from the cross Chinese Spring/Kolandi tested against *Egt* isolate nos. 14 and 116 segregated into individuals conforming to a ratio of 1 resistant: 3 susceptible (P=0.7–0.6), indicating the presence of a single recessive gene in Kolandi. Further analyses revealed that among the various F_2 populations between Chinese Spring monosomics and Kolandi crosses, the segregation of CS mono-7B/Kolandi F_1 progenies had more resistant plants and differed significantly (P<0.001) from the expected ratio of 1:3, indicating that the location of the resistance gene is on chromosome 7B (Table 3).

In the cross of Chinese Spring/IGV 1–455, the disomic F_2 population tested against *Egt* isolate no. 16 segregated into 33 resistant and 105 plants, satisfactorily fitting a genetic ratio of 1 resistant: 3 susceptible (P=0.8–0.7) and thereby conforming to a recessive monogenic inheritance. Similarly, a second test using *Egt* isolate no.14 also satisfactorily fit a 1 resistant: 3 susceptible ratio (Table 3). Among the 21 monosomic cross-combinations, 20 showed the expected 1:3 segregation. Only the F_2 population from the cross CS mono-7B/IGV 1–455 tested against *Egt* isolate nos. 14, 16 and 116 possessing different virulences segregated into more resistant than suceptible plants and deviated significantly (P<0.001) from the expected ratio of 1 resistant: 3 susceptible, indicating that the resistance gene analysed is on chromosome 7B (Table 3). The combined F_2 data from the other 20 CS monosomics/IGV 1–455 hybrids, with the exception of CS mono-7B/IGV 1–455, tested against *Egt* isolate no. 16, segregated into 698 resistant and 2036 susceptible plants, conforming to a 1:3 genetic ratio ($\chi^2_{(1:3)}$=0.41; P=0.7–0.5); this further confirmed the presence of a single recessive gene in line IGV 1–455, albeit one possessing a wider spectrum of resistance than that of Hope and Ibis. In test crosses of the line IGV 1–455 with each of the near-isogenic lines Axminister/8*Cc (*Pm1*) and Ulka/8*Cc (*Pm2*), respectively, using avirulent *Egt* isolate no. 10 the segregation of resistant to susceptible individuals conformed to a ratio of 13 resistant: 3 susceptible. The results revealed an independent segregation of the recessive resistance gene in IGV 1–455 and the dominant gene, either *Pm1* (64 resistant : 19 susceptible, $\chi^2_{(13:3)}$=0.94; P=0.5–0.4), or *Pm2* (59 resistant: 14 susceptible, $\chi^2_{(13:3)}$=0.009; P=0.95–0.9) in Axminster/8*Cc or Ulka/8*Cc, respectively. Hence, in line IGV 1–455 no evidence for the presence of any other powdery mildew resistance gene other than that located on chromosome 7B was detected.

The F_2 populations from the cross of disomic CS/IGV 2–556 tested against *Egt* isolate no. 16 segregated into 47 resistant and 133 susceptible individuals, fitting a ratio of 1 resistant: 3 susceptible ($\chi^2_{(1:3)}$=0.12; P=0.8–0.7) and thereby indicating a recessive monogenic inheritance. Of the 231 F_2 plants from the cross CS mono-7B/IGV 2–556 an excess number of resistant individuals

Table 3 Segregation for seedling reaction to *Egt* isolates in critical monosomic F_2 populations in comparison with disomic Chinese Spring (CS)

Monosomic/ disomic hybrids	Chromosome no. of F_1 plant	*Egt* isolate no.[a]	Observed segregation		$\chi^2_{(1:3)}$
			Resistant	Susceptible	
CS/Kolandi	42	14	42	113	0.21
CS/Kolandi	42	116	41	114	0.17
CS mono-7B/Kolandi	41	14	51	16	93.37**
CS mono-7B/Kolandi	41	116	54	13	110.45**
CS/IGV 1–455	42	14	21	49	0.94
CS/IGV 1–455	42	16	33	105	0.09
CSmono7B/IGV 1–455	41	14	208	15	554.39**
CSmono7B/IGV 1–455	41	16	334	28	873.55**
CSmono7B/IGV 1–455	41	116	200	23	497.64**
CS/IGV 2–556	42	16	47	133	0.12
CSmono7B/IGV 2–556	41	16	202	29	480.41**

**$P<0.001$
[a] Avirulent *Egt* isolates for resistant lines in each cross/virulent for CS

Table 4 Tests between the powdery mildew resistance gene *Pm5* and other alleles at the *Pm5* locus in the F_2 population of hybrids between cultivars and lines

Hybrid	*Egt* isolate no.[a]	Observed segregation		$\chi^2_{(11:5)}$
		Resistant	Susceptible	
Kolandi/Selpek	14 and 116	145	0	65.88**
Hope/Kormoran	14 and 116	151	0	68.66**
Selpek/Kormoran	14 and 116	130	0	59.10**
Ibis/Hope	14 and 116	153	0	69.54**
Ibis/Selpek	14 and 116	115	0	52.28**
Hope/IGV 1–455	10 and 14	268	0	121.82**
IGV1–455/IGV2–556	10 and 14	265	0	120.45**

**$P<0.001$
[a] Avirulent *Egt* isolates for both parents in each cross

(202 plants) were observed, deviating from the expected ratio of 1: 3 ($P<0.001$) and revealing that the resistance gene in IGV 2–556 is also located on chromosome 7B (Table 3).

Powdery mildew resistance alleles at the *Pm5* locus

Results of allelism tests in the F_2 generation between the resistance genes in cvs. Hope, Selpek, Ibis, Kormoran and lines Kolandi, IGV 1–455 and IGV 2–556 are shown in Table 4. A total of seven cross-combinations involving cultivars and lines with differential disease response patterns as well as between lines IGV 1–455 and IGV 2–556 were performed. The *Egt* isolate no. 14, which is avirulent to all the tested lines involved in the crosses, was used for the anaylses. This *Egt* isolate had also been employed for the location of the resistance gene in monosomic segregation studies. Furthermore, *Egt* isolates nos. 10 and 116 possessing virulences differing from that of *Egt* isolate no. 14 were also used. The *T. sphaerococcum* line Kolandi/Selpek hybrid produced only resistant offsprings. A significant deviation was observed from the theoretical 11 resistance: 5 susceptible ratio for two unlinked recessive genes acting complementarily ($\chi^2=65.88$, $P<0.001$), indicating that the resistance gene in Kolandi is either allelic or tightly linked to gene *Pm5* in Selpek. Similarly, in four allellic crosses involving Hope/Kormoran, Selpek/Kormoran, Ibis/Hope and Ibis/Selpek, all possessing either resistance gene *Pm5* or *Mli*, no susceptible individual was observed from among 549 F_2 plants tested, which indicated that *Pm5* and *Mli* are either allelic or tightly linked (Table 4). Likewise, in the cross of Hope/IGV 1–455, the 268 F_2 plants tested were all resistant ($\chi^2_{(11:5)}=121.82$, $P<0.001$), indicating the resistance gene in IGV 1–455 is at the *Pm5* locus or else very tightly linked to *Pm5*. All 265 F_2 plants of the cross between IGV 1–455 and IGV 2–556 were resistant. It may be postulated that the backcrossed-derived lines IGV 1–455 and IGV 2–556 carry the same resistance gene as they exhibited identical disease response patterns (Table 1).

In crosses between two resistant lines, the lack of appearance of susceptible plants would indicate that the two lines are carrying resistance genes that are allelic or that the genes are very closely linked. Hence, for more clarity, F_2 plants from five cross-combinations tested for allelism with *Pm5* were analysed further in the F_3 generation by using common *Pm5* race-specific *Egt* isolate no. 14 in all the crosses. In addition, other race-specific *Egt* isolates were used to detect whether the resistance gene(s) assumed to be in the *Pm5* locus shared or produced a different disease resistance spectrum. The F_3 families from crosses between Kolandi/Selpek, Ibis/Hope and Hope/IGV 1–455 were inoculated with *Egt* isolate nos. 10, 14 or 116 possessing different virulences but avirulent to both parents of each cross; no susceptible offsprings were produced(Table 5). The same families segregated into three categories – homozygous resistant, segregating for resistance and susceptibility and homozygous susceptible – when tested with specific *Egt* isolates virulent to one of the parents in each cross. The F_3 families from the cross Kolandi/Selpek tested with *Egt* isolate no. 114 segregated into 9 resistant: 35 segregating: 12 susceptible families

Table 5 Allelism tests between wheat hybrids carrying resistance genes at the *Pm5* locus in the F_3 populations

Hybrid	*Egt* isol. no.	Number of F_3 families		
		Resistant	Segregating	Susceptible
Kolandi/Selpek	14[a]	56	0	0
	114[b]	9	35	12
Ibis/Hope	14 and 116[a]	76	0	0
	J2/2[c]	16	45	15
Hope/IGV 1–455	10 and 14[a]	60	0	0
	16[d]	11	31	18
Hope/Kormoran	14 and 116[a]	71	0	0
Selpek/Kormoran	14 and 116[a]	76	0	0

[a] Avirulent for both *Pm5* alleles in the cross
[b] Virulent for Kolandi and avirulent for Selpek
[c] Virulent for Ibis and avirulent for Hope
[d] Virulent for Hope and avirulent for IGV 1–455

($\chi^2_{(1:2:1,\ 2\ df),}$=3.83), fitting a 1:2:1 ratios as expected. Similarly, F_3 families from Ibis/Hope tested with *Egt* isolate no. J2/2 segregated into 16:45:15 families ($\chi^2_{(1:2:1,\ 2df),}$=2.60). As Hope, Selpek and the Chinese Spring (Hope 7B) substitution line expressed resistance to *Egt* isolate nos. 114 and J2/2, the non-segregating resistant F_3 families tested with these isolates carried the *Pm5* gene from either Hope or Selpek in the homozygous conditions, whereas the rest of the resistant families tested with *Egt* isolate no. 14 carried an allele from each of the respective parents involved in the cross. The cross Hope/IGV 1–455 tested with *Egt* isolate no. 16, which is virulent to Hope but avirulent to IGV 1–455 segregated into 11 resistant: 31 segregating: 18 susceptible families ($\chi^2_{(1:2:1,\ 2\ df),}$ =1.70). It is evident that in each of the above cross-combinations analysed, avirulent *Egt* isolates demonstrated allelism, and the virulent isolate to one parent showed a monogenic mode of inheritance for one of the parents at the *Pm5* locus. Furthermore, 71 F_3 families from the cross Hope/Kormoran and 76 F_3 families from the cross Selpek/Kormoran tested with *Egt* isolate nos. 14 and 116, avirulent to both parents, produced only resistant plants, supporting the result of the cross Ibis/Hope and indicating that *Pm5* and *Mli* are alleles at the *Pm5* locus.

Results obtained from the F_2 and F_3 populations indicated that resistance genes in lines Kolandi and IGV 1–455 and resistance gene *Mli* in cvs. Ibis and Kormoran are allelic to gene *Pm5* in cvs. Hope and Selpek. Moreover, these genes revealed differential disease response patterns to a selected set of 12 *Egt* isolates that distinguished them from one another. It is proposed that the powdery mildew resistance alleles at the *Pm5* locus be designated *Pm5a* in Hope and Selpek, *Pm5b* in Ibis and Kormoran, the former *Mli* cultivars, *Pm5c* in *T. sphaerococcum* line Kolandi and *Pm5d* in backcrossed-derived lines IGV 1–455 and IGV 2–556.

Discussion

Differential *Egt* isolates are very useful for identifying of resistance genes to powdery mildew disease. Resistance genes may be postulated from the disease response of distinct host lines to differential *Egt* isolates based on the assumption of Flor's classical gene-for-gene hypothesis (Flor 1955). In the present study, a set of 12 differential *Egt* isolates was selected from a total of 115 isolates, and these clearly distinguished the *Pm5* alleles encountered in the present study. The difference in disease response patterns of the lines tested in comparison to standard cultivars and lines with documented resistance to the same *Egt* isolates led to further genetic study to determine whether the resistance is located at a new locus or is an allele at an already known locus. The differentiation of the resistance genes or alleles would have been very difficult, if not impossible without an appropriate collection of pathogen isolates. In the present study, conventional monsomic analyses and allelism tests in combination with disease response patterns were employed to provide a basis for the classification of resistance alleles at the *Pm5* locus.

Hope was the first common wheat cultivar described to carry gene *Pm5*. The German spring wheat cultivar Selpek has also been reported to carry gene *Pm5* (Heun and Fischbeck 1987). The resistance gene in Selpek can be traced to the North American cultivar Selkirk, a derivative of a cross involving cv. Redman, possessing *Pm5* (McIntosh et al. 1967), which in turn was derived from H-44, a sister line of Hope originating from the same cross Marquis/Yaroslav Emmer (McFadden 1930; McIntosh et al. 1967). The first cross between the two lines was made in 1916 by McFadden, with the intention of transferring rust resistance from a *T. dicoccum* line selected in 1915 from Yaroslav (Russia) with Marquis. Hope was developed from the line H49–24 selected for rust resistance and was distributed in 1926 as a breeding line (McFadden 1930). The cultivar Sicco (Ring/Opal/Selkirk) from The Netherlands may have inherited *Pm5a* resistance from Selkirk. However, the donors of resistance in the other cultivars carrying *Pm5a* could not be deduced from their pedigrees.

A number of German cultivars possessing gene *Pm5b* (former *Mli*) can be traced to cvs. Kormoran and Ibis. The *Mli* gene in Ibis is probably derived from line DHE 516 (DHE=Deutsche Hindukusch Expedition), which originated from the German expedition to Hindukush in the early 1930s (von Rosenstiel 1938; Lange de la Camp 1939). According to the late K. Lein (personal communication from 1976 provided by K Brunckhorst, von

Lochow-Petkus GmbH Wetze, Northeim, Germany) 2 lines, namely winter wheat Heine 2167.50 and spring wheat Heine 2174.50 were developed from the line DHE 516. Line Heine 2167.50 in crosses with Heine VII and Merlin gave rise to winter wheat cultivars Ibis and Tadorna, the latter a parent line of Aquila and Heine 684.57. The latter is included as a parent of cv. Kormoran which passed its resistance further to many German cultivars, such as Dolomit (Ferto/Kormoran), Frühprobst (Pilot/Kormoran) and Rektor (Kormoran/ Monopol). The resistance in cvs. Carimulti (Ibis/Caribo) and Cariplus (Ibis/Caribo) traces to Ibis. On the other hand, the wheat line Heine 2174.50 is the donor of resistance gene *Pm3d* present in cv. Kolibri (Zeller et al. 1993). However, the number of resistance genes in the parent line DHE 516 could not be verified as the original line is no longer available.

The *T. sphaeroccum* line Kolandi (ATRI 2996/74) possessing *Pm5c* was originally collected from India and maintained at the gene bank in Gatersleben, Germany. *T. sphaeroccum*, also known as 'Indian dwarf' is a cultivated wheat of northwestern India and parts of Iran (Miller 1987) and may have arisen in Pakistan (Kihara 1958). *T. sphaerococcum* accessions have already been reported to carry resistance genes to powdery mildew: *T. sphaerococcum* var. *spicatum* Cawnpore and *T. sphaerococcum* var. *rubiginosum* Hindukusch both carry resistance gene *Pm3c* (Zeller et al. 1993). Hence, it may be beneficial to screen further accessions of *T. sphaerococcum* for resistance to powdery mildew and other diseases.

The backcrossed derived lines IGV 1–455 (CI 10904/7* Prins) and IGV 2–556(CI 10904/7* Starke) apparently inherited the resistance gene *Pm5d* from CI 10904. The accession CI 10904 is a common wheat line introduced into USA from the University of Nanjing, Jiangsu, China, in 1929 (H.E. Bockelman, personal communication). Leijerstam (1972) was the first to find that CI 10904 was resistant to the powdery mildew pathogen. After carrying out six backcrosses with the susceptible Swedish spring wheat cultivar Prins and winter wheat cultivar Starke, Leijerstam achieved the transfer of the resistance in CI 10904 into the susceptible cultivars in the backcrossed derived lines IGV 1–455 and IGV 2–556, respectively.

The resistance gene *Pm5* is prevalent in commercial cultivars and landraces grown in the Mediterranean region (Zeller et al. 1998). It is known that *Pm5* occurs in tetraploid emmer wheat from which it was introduced into common wheat (Law and Wolfe 1966). It is not known whether *Pm5* is being transferred from tetraploid to hexaploid level in nature. It is apparent from the present study that new *Pm5* alleles are present in *T. sphaerococcum* in India as well as in the wheat landrace CI 10904 originating from China.

In the genomes of *Triticeae*, resistance genes are non-randomly distributed and form a cluster of genes at gene-rich regions. Multiple resistance genes may be clustered, as in the *Rp1* complex rust (*Puccinia sorghi*) resistance locus in maize (Hu and Hulbert 1996), or exist as an allelic series at a structurally simple locus. More than 30 alleles were identified at the *Mla* locus for resistance to powdery mildew (*Erysiphe graminis* f. sp. *hordei*) in barley (Kintzios et al.1995). In wheat, ten different alleles have been located at the *Pm3* locus on chromosome 1AS (Zeller et al. 1993; Zeller and Hsam 1998), four alleles at the *Pm1* locus on chromosome 7AL (Hsam et al. 1998) and two alleles at the *Pm8* locus on the 1RS arm of the T1BL·1RS translocation (Hsam and Zeller 1997). The *Pm5* locus is composed of four different alleles to date. However, the detail, nature and function of these alleles awaits further molecular characterization, as has been done, for example, for the maize *Rp1-D* rust resistance haplotype (Collins et al. 1999). Nevertheless, resistance gene *Pm5a* of cv. Hope in combination with other genes, such as *Pm1c* or *Pm17*, is very effective against the currently existing powdery mildew pathogenic races in Germany. In addition, up to now, virulent *Egt* pathotypes to resistance gene *Pm5d* have not yet been detected in Europe. Therefore, the introgression of these new alleles may be useful in future wheat breeding programmes for resistance to powdery mildew.

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