Plant Pathology Journal

ISSN 1812-5387

science alert

ANSInet
an open access publisher
http://ansinet.com

Plant Pathology Journal 6 (2): 164-168, 2007
ISSN 1812-5387

Inheritance and Chromosomal Location of Powdery Mildew Resistance Gene in Wild Wheat *Triticum turgidum* Var. *dicoccoides*

[1]Hadi Ahmadi and [2]K. Moore
[1]Agricultural Faculty, University of Lorestan, P.O. Box 465, Khoramabad, Iran
[2]Department of Agricultural and Environment Science, The University,
Newcastle Upon Tyne NEl 7RU, UK

Abstract: Powdery mildew of wheat is caused by *Erysiphe graminis* f. sp. *tritici* and it is a major disease of wheat (*Triticum aestivum* L.). Therefore there is a need for more research to find new genes for resistance. To study the inheritance of gene/s for resistance to powdery mildew three susceptible *T. durum* varieties were crossed with three resistant *T. dicoccoides* accessions. The segregation results in F_1, F_2, F_3 and reciprocal BC_1 progenies confirmed that *T. dicoccoides* accessions TA1055 and TA1150 possess one similar dominant gene for resistance to *Erysiphe graminis* f. sp. *tritici* To determine chromosomal location of gene for resistance to powdery mildew 14 monosomic lines of Chinese Spring were used (1A-7A and 1B-7B) to cross as female with the resistant accessions TA1055 and TA1150. The F_1 hybrid seeds were germinated to obtain F_2 seeds. Analysis of obtained data revealed that one major dominant gene conferring resistance is located on chromosome 2A of *T. dicoccoides* accession TA1055 which is different from 33 current known genes for resistance to wheat powdery mildew and should be designated *Pmtd1055*.

Key words: Chromosomal location, Inheritance monopentaploid, Powdery mildew, *T. dicoccoides*, *T. durum*

INTRODUCTION

Powdery mildew is caused by *Erysiphe graminis* f. sp. *tritici* and it is one of the most important disease of wheat in arias with maritime or semicontinental climate (Bennett, 1984). Up to now, 51 *Pm* alleles at 33 loci have been identified for wheat powdery mildew resistance (McIntosh *et al*., 2005). Thirty alleles at 24 loci from *Pm1* to *Pm25*, their locations on chromosomes and their sources have been reviewed, other *Pm* genes, *Pm26, Pm27* and *Pm28*, *Pm29, Pm30, Pm31* and *Pm32* have also been reported (Shi *et al*., 1998; Jarve *et al*., 2000; Peusha *et al*., 2000; Rong *et al*., 2000; Zeller *et al*., 2002; Liu *et al*., 2002; Xie *et al*., 2003; Hsam *et al*., 2003).

Pm1, *Pm2*, *Pm3 (a-f)*, *Pm9*, *Pm18*, *Pm22, Pm28* and *Pm29* were found in the hexaploid common wheat (*Triticum aestivum*). *Pm19* was derived from *Triticum tauschii* Coss (2n = 14, DD); *Pm4a* and *Pm5* from *T. dicoccum* (2n = 28,AABB); *Pm4b* from *T. carthlicum* (2n = 28, AABB); *Pm6* and *Pm27* from *T. timopheevii* (2n = 28, AAGG); *Pm7*, *Pm8*, *Pm17* and *Pm20* from *Secale cereale* (2n = 14, RR), *Pm12* and *Pm32* from *T. speltoides* (2n = 14, S^bS^b); *Pm13* from *T. longissimum* (2n = 14, S^pS^p). *Pm16 Pm26* and *Pm31* from *T. turgidum* var. *dicoccoides*; *Pm21* from *Dasypyrum villosum* (2n = 14, VV) and *Pm25* from *T. monococcum* ssp. *aegilopoides* (2n = 14, AA). *Pm10* and *Pm11* were detected in *T. spelta* ssp. *duhamelianum*, *Pm14* in Norin 10 and *Pm15* in *T. macha* ssp. (McIntosh *et al*., 1998).

The most resistance genes/alleles are currently in use in Europe (Zeller *et al*., 1993; Peusha *et al*., 1996) however, in recent years most of the established genes for resistance have been ineffective (Bennett, 1984; Brown *et al*., 1990). Therefore additional effective resistance genes or alleles need to be identified to enable the continued diversification of gene combinations in breeding new cultivars. In the present study a new gene for resistance to mildew is described in the wild emmer *T. dicoccoides* accessions TA1055 and TA1150.

MATERIALS AND METHODS

Plant materials and isolate of *Erysiphe graminis* f. sp. *tritici*: Three *T. durum* varieties (IR4 = Tokhmi Siah; IR5 = Siah Daas and IR10 Chahar Tokhmi) from Iran. Three resistant accessions of *T .dicoccoides* TA64, TA1055 and TA1150 (Ahmadi firouzabad, 2001) were obtained from Kansas State University.

The *Erysiphe graminis* f. sp. *tritici* isolate Nor2 used for the inheritance and location of gene for resistance

Corresponding Author: Hadi Ahmadi, Faculty of Agricultural, University of Lorestan, P.O. Box 465, Khoramabad, Iran

to powdery mildew was provided as described by Ahmadi firouzabad (2001). This isolate contains genes for virulence against *Pm1*, *Pm2*, *Pm3a*, *Pm3c*, *Pm4a*, *Pm4b*, *Pm5*, *Pm9*, *Pm11*, *Pm15*, *Mld* and Mlk .

Resistance analysis: The susceptible *T. durum* varieties were crossed with three resistant *T. dicoccoides* accessions in 2000 at department of field station, University of Newcastle Upon Tyne. The F_1 hybrids were grown to produce F_2 populations. Backcrosses to IR10 and crosses between TA1055 X TA1150 were made to confirm the number of genes for resistance present in these accessions. The methods of inoculation and conditions of incubation and disease assessment were used according to Hsam and Zeller (1997).

Chromosomal location: In this study the source of resistance *T. dicoccoides* is a tetraploid wheat, and thus 14 monosomic lines of Chinese Spring were used (1A-7A and 1B-7B) to cross as female with the resistant accessions TA1055 and TA1150. The F_1 monopentaploid lines were grown to produce F_2 populations. The F_2 populations were inoculated with isolate Nor2. Twenty four hours prior to inoculation the isolate was tapped to dislodge the old conidia, and the two-week-old seedlings of parents and F_2 plants were inoculated with the Nor2 isolate. After two weeks the numbers of plants with either resistant or susceptible reactions to mildew were recorded.

Cytological analysis: Meiosis in F_1 hybrid plants was studied to identify monopentaploid hybrids. For the meiotic study, spikes of the F_1 plants were fixed in acetic acid: ethanol (1:3) solution and anthers were selected for meiotic metaphase (MI) by carmine staining and stored in 70% alcohol at 4°C.

In a F_1 monopentaploid, it is expected that 13 or 14 bivalents (genomes A and B) and 7 or 8 univalent (7 from genome D and one from genome A or B) are observed at metaphase I. In the case of F_1 plants (2n = 34 or 2n = 35) at metaphase I, the number of univalents will be odd or even. The even ones are monopentaploid because 7 univalents belong to the D genome and one univalent is the homologous *T. dicoccoides* chromosome of the monosomic A or B genome chromosomes.

At early anaphase I the number of chromosomes were counted and plants with 2n = 34 and 2n = 35 chromosomes were identified.

RESULTS AND DISCUSSION

Inheritance of gene for resistance to powdery mildew: The F_1 hybrids from all crosses were resistant when tested at the seedling stage and exhibited slight symptoms on their leaves, with scattered colonies beginning to necrose before much sporulation had occurred. By the second tiller stage, symptoms of infection had progressed to a characteristic chlorosis spreading from the tip of the oldest leaves (infection type 1-2). The leaf blades and spikes of all of the F_1 plants were resistant at the heading stage and as at the seedling stage a few necrotic flecks were observed on leaf blades. However, the F_1 plants displayed more obvious symptoms of susceptibility with moderate, sometimes heavy infection on leaf sheaths (IT 3-4), some sporulation and no apparent necrosis around colonies.

In the F_2 generations, at the seedling stage, the F_2 plants segregated in a ratio 1:2:1 with two groups resistant (IT 0-1) and (IT 2) and one susceptible (IT 3-4) (Table 1). This segregation can be explained in terms of one partially dominant gene present in the *T. dicoccoides* accessions. At heading stage, the F_2 plants were classified into 3 classes: 'Class A' in which the leaf blade (IT 0-1), leaf sheath (IT 0-1) and spikes were resistant; 'Class B' in which the leaf blades (IT 2) and spikes were resistant, but the leaf sheath was susceptible (IT 3); and 'Class C' in which the leaf blade (IT 3-4), leaf sheath (IT 3) and spikes were susceptible. The F_2 segregations of crosses of IR10 with both *T. dicoccoides* accessions fit a 1 class A : 2 class B : 1 class C ratio (Table 2). In order to confirm the F_2 segregation results, twenty F_3 selfed seed was germinated from each F_2 plant (5 to 7 F_2 plants from each of the classes A, B and C) from the crosses IR10 x TA1055 and IR10 x TA1150. The results are summarized in Table 3. The offspring of class C F_2 plants were all susceptible (IT 3-4) like their parents, which were obviously homozygous recessive for the gene for resistance to powdery mildew. The F_2 resistant plants could be divided into segregating F_3 families (class B) and families in which all individuals were fully resistant (class A). The latter class A F_2 plants must have been homozygous dominant because all of the plants had a reaction IT 0-1 and the segregating F_3 families (class B) obviously originated from F_2 plants heterozygous for the gene for resistance since this group segregated plants with IT 0-1, IT 2 and IT 3-4. The ratio between F_2 plants with segregating offspring (12 individuals) and with only resistant offspring (9 individuals) was expected to be 2:1. Despite the low numbers a chi-square value was calculated, indicating a good fit to the expectation (x^2 = 0.86, P = 0.30-0.50). Thus it was concluded that resistance in *T. dicoccoides* accessions TA64, TA1055 and TA1150 is controlled by a single partially dominant gene.

The BC_1 segregation of crosses of IR10 with both *T. dicoccoides* TA1055 and TA1150 fit a 1:1 ratio

Table 1: Segregation for resistance to *Erysiphe graminis* f. sp. *tritici* in three F_2, reciprocal BC_1 populations and their parents at the seedling stage

Cross	Parents and generation	No. of plants 0-1†	2	3-4	Expected ratio	χ^2	χ^2 probability
IR10	P_1	0	0	10	-	-	-
TA1055	P_2	10	0	0	-	-	
IR10 x TA1055	F_2	23	48	29	1:2:1	0.88	0.50
F1 x IR10	BC_1	0	12	9	1:1	0.43	0.50
F1 x TA1055	BC_1	6	8	0	-	-	
TA 1150	P_3	10	0	0	-	-	
IR10 x TA1150	F_2	27	49	24	1:2:1	0.22	0.80
F1 x IR10	BC_1	0	9	7	1:1	0.25	0.50
F1 x TA1150	BC_1	4	4	0	-	-	
TA64	P_1	10	0	0	-	-	
IR4 x TA64	F_2	19	38	22	1:2:1	0.34	0.80
IR5 x TA64	F_2	9	14	6	1:2:1	0.66	0.70
TA1055 x TA1150	F_2	100	0	0			
TA1150 x TA1055	F_2	100	0	0	-	-	

†Infection Type: 0-1 = Resistant, 2 = Moderately resistant, 3-4 = Susceptible

Table 2: Segregation for resistance to *Erysiphe graminis* f. sp. *tritici* in three F_2, reciprocal BC_1 populations and their parents at heading stage

Cross	Generation	No. of plants Class A	Class B	Class C	Expected ratio	χ^2	χ^2 probability
IR10 x TA1055	F_2	23	48	29	1:2:1	0.88	0.50-0.70
IR10 x TA1150	F_2	27	49	24	1:2:1	0.22	0.80-90

Table 3: Analysis of F_3 families from F_2 plants with resistant and susceptible reactions, from crosses between *T. durum* and two *T. dicoccoides* genotypes at the seedling stage

Cross	: F_2	No. of families	No. of plants	No. of plants 0-1†	2	3-4	Chi-square 1:2:1	χ^2probability
IR10 x TA1055	R‡ (class B)	6	103	28	56	19	2.36	0.30-0.50
	R (class A)	4	93	93	0	0	-	-
	S§ (class C)	7	109	0	0	109	-	-
IR10 x TA1150	R (class B)	6	96	24	45	27	0.56	0.56
	R (class A)	5	69	69	0	0	-	-
	S (class C)5	87	0	0	87	-	-	-

†Infection types: 0-1 = Resistant, 2 = Moderately resistant, 3-4 = Susceptible, ‡ = Resistant, § = Suseptible

(Table 2). This lead to confirm one dominant gene for resistance to powdery mildew is present in each *T. dicoccoides* accession. When the *T. dicoccoides* accessions were used as the recurrent parent, all of the BC_1 progenies were resistant (Table 2). The F_2 populations of reciprocal crosses between resistant TA1055 and TA1150 after inoculation with Nor2 isolate were all resistant and did not segregated (Table 2). This indicated that both *T. dicoccoides* accessions carry the same gene for resistance to powdery. The segregation results in F_1, F_2, F_3 and reciprocal BC_1 progenies confirmed that *T. dicoccoides* accessions TA1055 and TA1150 possess one similar dominant gene for resistance to *Erysiphe graminis* f.sp. *tritici* isolate Nor2.

Many sources of resistance to *Erysiphe gramins* f.sp. *tritici* have been identified in *T. dicoccoides* (Moseman *et al*., 1984). Thus *T. dicoccoides* can be a valuable genetic resource to broaden the genetic base of both cultivated species. Wild emmer population contain high frequencies of disease resistant genes. Gerechter-Amitai and Stubbs (1970) studied *T. dicoccoides* accessions collected from 32 sites, and found seedlings from 17 of the 55 accessions were resistant to *Puccinia striiformis* West. in the first leaf stage. Gerechter-Amitai and Grama (1974) showed that the resistance of selection G-25 of *T. dicoccoides* had one dominant gene conditioning its resistance to *P. striiformis*.

Moseman *et al.* (1984) tested the reaction of 233 *T. dicoccoides* accessions to infection with cultures of *E. graminis* f. sp. *tritici*. The reactions indicated that one hundred and fourteen or 49% of the accessions were resistant and 23 of the accessions were moderately resistant to infection with four cultures of *E. graminis* f. sp. *tritici* which possessed the virulence genes (*Pm1*, *Pm2*, *Pm3a*, *Pm3c*, *Pm4*).

Self seed-set: The emergence rate of F_1 monopentaploid hybrid seed was 97.5 and 98.2% for crosses CS/TA1055 and CS/TA1150, respectively. This indicates that the majority of the monopentaploid seeds were viable. No selfed seed was produced by the F_1 hybrid plants from crosses CS/TA1150. The F_1 plantsof monopentaploid

Table 4: Segregation for seedling reaction to mildew isolate Nor2 in 14 monopentaploid F_2 populations from crosses of 14 Chinese Spring monosomics with *T. diccoccoides* accession TA1055

	No. of plants				
Cross	0-1†	2	4	χ^2 1:2:1	χ^2 probability
1A	6	11	3	1.1	0.50-0.70
2A	11	35	3	11.61**	p<0.01
3A	8	19	9	0.17	0.90-0.95
4A	11	21	14	0.74	0.50-0.70
5A	13	22	13	0.33	0.80-0.90
6A	5	12	6	0.13	0.90-0.95
7A	6	13	9	0.79	0.50-0.70
1B	8	17	5	1.13	0.50-0.70
2B	12	26	9	0.91	0.50-0.70
3B	6	13	5	0.25	0.80-0.90
4B	12	18	8	0.95	0.50-0.70
5B	10	18	12	0.60	0.70-0.80
6B	16	25	13	0.63	0.70-0.80
7B	14	28	16	0.21	0.80-0.90
Total‡	127	243	122	0.17	0.90-0.95

† Infection Types: 0-1 = no symptoms or very slight chlorotic flecking on the leaves; IT 2 = small pustules surrounded by necrosis or chlorosis, no sporulation; IT 4 = large pustules with massive sporulation, no chlorossis ‡ Excluding mono-2A, **p<0.01

families had a range of 1.9 to 9.2 mean percent seed-set per plant. Eight monopentaploid F_1 hybrid lines, 1A, 3A, 4A, 6A, 7A, 3B, 4B and 5B, showed a significant reduction in self seed per plant, indicating that at least eight chromosomes may have factors which influence self seed-set.

Chromosomal location: The F_2 progeny from crosses of the 14 Chinese Spring (CS) monosomic lines with TA1055 were inoculated with the Nor.2 isolate. Thirteen of the F_2 progeny representing each hybrid combination and the pooled data (excluding mono-2A hybrid) segregated in a ratio of 3:1 (Table 4) for the three infection types. The more excess of type 2 of F2 plants in mono-2A population can be explained as follows. When a monosomic plant is allowed to produce selfed-seed, it is expected that the majority of the F_2 plants to be monosomic, because the frequency of monosomic plants in a F_2 population has been predicted about 73%. In this study the frequency of plants with type 2 which can be monosomic for chromosome 2A was 72.9% which is most closely to the expected frequency. This indicated that the gene for resistance to wheat powdery mildew is present and expressed in monopentaploid hybrids. The F_2 segregation from the monopentaploid hybrid CS mono-2A x TA1055 deviated significantly ($p<0.01$) from the expected 1:2:1 ratio. The results revealed that the gene conferring resistance to powdery mildew is located on chromosome 2A.

The reaction of *T. dicoccoides* accessions was different from the wheat mildew resistance genes *Pm4a* and *Pm4b* which are known to be located on chromosome 2A (McIntosh *et al*., 1998). Because Nor 2 was virulent to *Pm4b, Pm4a* and avirulent on the *T. dicoccoides* accessions. Resistance to powdery mildew in Td1055 and Td1150 is controlled by a gene other than *Pm16* and *Pm26* already transferred to common wheat from *T. dicoccoides*. The gene *Pm16* is located on chromosome 4A (Reader and Miller, 1991) and the gene *Pm26* is located on chromosome 2B (Rong *et al*., 2000) but the gene for resistance present in Td1055 is located on chromosome 2A. It is proposed that this new resistance gene in TA1055 which is different from 33 current known genes for resistance to wheat powdery mildew, should be temporarily designated *Mltd1055*.

REFERENCES

Ahmadi-Firouzabad, A., 2001. Introgression of resistance to powdery mildew (*Erysiphe graminis* f.sp. *tritici*) from alien species into bread wheat (*Triticum aestivum*). Ph.D., University of Newcastle, Newcastle upon Tyne.

Bennett, F.G.A., 1984. Resistance to powdery mildew in wheat: a review of its use in agriculture and breeding programmes. Plant Pathol., 33: 279-300.

Brown, J.K.M., S.E. Slater and P.M. Howe, 1990. Mildew of Wheat U.K. Cereal Pathogen Virulence Survey: 1989 Annual Report, N.I.A.B., Cambridge, pp: 1-7-10.

Gerechter-Amitai, Z.K. and R.W. Stubbs, 1970. A valuable source of yellow rust resistance in Israel populations of wild emmer, *Triticum dicoccoides* Koern. Euphytica, 19: 12-21.

Gerechter-Amitai, Z.K. and R.W. Grama, 1974. Inheritance of resistance to stripe rust (*Puccinia striiformis*) in crosses between wild emmer (*Triticum dicoccoides*) and cultivated tetraploid and hexaploid wheat. I. *Triticum durum*. Euphytica, 23: 387-392.

Hsam, S.L.K. and F.J. Zeller, 1997. Evidence of allelism between genes *Pm8* and *Pm17* and chromosomal location of powdery mildew and leaf rust resistance genes in the common wheat cultivar Amigo. Plant Breed., 116: 110-122.

Hsam, S.L.K., I.F. Lapochkina and F.J. Zeller, 2003. Chromosomal location of genes for powdery mildew resistance in common wheat (*Triticum aestivum* L. em Thell.). 8. Gene *Pm32* in a wheat-*Aegilops speltoides* translocation line. Euphytica, 133: 367-370.

Jarve, K., H.O. Peusha, J. Tsymbalova, S. Tamm, K. Devos and T. Enno, 2000. Chromosomal location of a *Triticum timopheevii* derived powdery mildew resistance gene transferred to common wheat. Genome, 43: 377-381.

Liu, Z.Y., Q.X. Sun, Z.F. Ni, E. Nevo and T.M. Yang, 2002. Molecular characterization of a novel powdery mildew resistance gene *Pm30* in wheat originating from wild emmer. Euphytica, 123: 21-29.

McIntosh, R.A., G.E. Hart, K.M. Devos, M.D. Gale and W.J. Rogers, 1998. Catalogue of gene symbols for wheat. In: Proc. 9th Int. Wheat Genet. Symp. Saskatchewan, Canada.

McIntosh, R.A., K.M. Devos, J. Dubcovsky and W.J. Rogers. 2005. http://shigen. lab. nig.ac.jp/ wheat /komugi/genes/macgene/2005/GeneSymbol.html.

Moseman, J.G., E. Nevo, M.A. Elmorshidy and D. Zohary, 1984. Resistance of *Triticum dicoccoides* to infection with *Erysiphe graminis* f. sp. *tritici*. Euphytica, 33: 41-47.

Peusha, H., S.L.K. Hsam and F.J. Zeller, 1996. Chromosomal location of powdery mildew resistance genes in common wheat (*Triticum aestivum* L. em. Thell.) 3. Gene *Pm22* in cultivar Virest. Euphytica, 91: 149-152.

Peusha, H., T. Enno and O. Priilinn, 2000. Chromosomal location of powdery mildew resistance genes and cytogenetic analysis of meiosis in common wheat cultivar Meri. Hereditas, 132: 29-34.

Reader, S. M. and T. E. Miller, 1991. The introduction into bread wheat of a major gene for resistance to powdery mildew from wild emmer wheat. Euphytica, 53: 57-60.

Rong, J.K., E. Millet, J. Manisterski and M. Feldman, 2000. A new powdery mildew resistance gene: Introgression from wild emmer into common wheat and RFLP-based mapping. Euphytica, 115: 121-126.

Shi, A.N., S. Leath and J.P. Murphy, 1998. A major gene for powdery mildew resistance transferred to common wheat from wild einkorn wheat. Phytopathology, 88: 144-147.

Xie, C.J., Q. Sun, Z. Ni, T. Yang, E. Nevo and T. Fahima, 2003. Chromosomal location of a *Triticum dicoccoides*-derived powdery mildew resistance gene in common wheat by using microsatellite markers. Theoretical Applied Genet., 106: 341-345

Zeller, F.J., U. Stephan and J. Lutz, 1993. Present Status of Wheat Powdery Muldew Resistance Genetics. In: Z.S. Li and Z.Y. Xin (Eds.), Proc. 8th Int. Wheat Genet. Symp., Beijing, China, pp: 929-931.

Zeller, F.J., L. Kong, L. Hartl, V. Mohler and S.L.K. Hsam, 2002. Chromosomal location of genes for resistance to powdery mildew in common wheat (*Triticum aestivum* L. em Thell.) 7. Gene Pm29 in line Pova. Euphytica, 123: 187-194.

Received 29 December 2006; Revised 14 February 2007; Accepted 1 March 2007; Available Online 29 May 2007.