Comparison of Two Wheat Powdery Mildew Differential Sets in Seedling Tests

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Abstract

Two wheat powdery mildew differential sets were tested in the seedling stage in the 2001/2002 season using 192 monoisolates. The data of genotypes carrying the same *Pm* gene in different genetic backgrounds were compared. Both varieties with gene *Pm8* (Salzmünde14/44 and Disponent) were infected by all the isolates. Less than 10% of the isolates gave different responses on varieties with genes *Pm2* and *Pm3c* (6 and 16, respectively). It is doubtful whether the degree of infection of genotypes carrying genes *Pm1, Pm4b, Pm5, Pm6* or *Pm7* can be compared, while it is completely impossible to compare the data for varieties from the old and new sets carrying genes *Pm3a, Pm3b* and *Pm4a*.

Keywords: powdery mildew; wheat; differential set; virulence

INTRODUCTION

Powdery mildew is one of the most frequent diseases of wheat all over the world, so of all pathosystems, this host plant-pathogen relationship is one of the most widely studied. After the demonstration of physiological specialisation within the wheat powdery mildew species, the identification and cataloguing of the races was begun in Germany in the second half of the 1930s (NOVER 1957). In the course of this work tests were made on an empirically compiled collection of test varieties carrying different resistance genes, which served for a long period for the identification of powdery mildew races. During this period the races were numbered in the order in which they were discovered. Based on Nover's system, FRAUENSTEIN *et al.* (1979) listed all the powdery races identified in various countries to date and described their reactions on the varieties in the test collection. Since then many new resistance genes have been discovered. Up till now 30 *Pm* genes have been registered (MCINTOSH *et al.* 2002), and their number is increasing year by year. However, since the test collection was compiled, the genetic background of powdery mildew resistance in cultivated wheat varieties has completely changed, so the original varieties used for differentiation are no longer suitable for the detection of changes in the virulence of the natural pathogen population and in the dominance of different pathotypes. For the above reasons it has become necessary to include new *Pm* resistance genes in powdery mildew population tests (FRAUENSTEIN *et al.* 1983; PERSAUD & LIPPS 1995; SZUNICS *et al.* 2000). Unfortunately, as different varieties are used for differentiation it is difficult to compare the results achieved. Within the framework of the COST Action 817 several laboratories cooperated to compile a Core Differential Set consisting of genotypes carrying the resistance genes occurring most frequently in the wheat varieties grown in Europe, with the help of which virulence surveys can again be made uniform and comparable (CLARKSON 2000).

In Hungary investigations on the physiological specialisation and virulence of wheat powdery mildew have been underway since 1965. Changes in the pathogen population have been monitored in Martonvásár since 1970. The differential set designed by NOVER (1957) was used in the early years to identify pathotypes isolated from widely grown varieties (SZUNICS & SZUNICS 1972). Over the last three decades the number of genotypes used for testing has increased to nineteen and these were used to determine the frequency and virulence of the pathotypes to be found in the powdery mildew population (SZUNICS *et al.* 2000). Since the varieties used in the Martonvásár Institute

differ from those recommended by the COST Action 817, it was necessary to compare the two differential sets to determine whether the earlier data could be converted to the new system.

MATERIALS AND METHODS

In the 2001/2002 season 192 wheat powdery mildew (*Erysiphe graminis* DC. f.sp. *tritici* Ém. Marchal) monoisolates were collected in seedling tests in and around Martonvásár. The powdery mildew pustules were collected from winter wheat varieties grown on large areas now or in earlier periods. The pathogens were multiplied on a susceptible variety (Bezostaya 1) grown in pots under a glass bell. Thirty wheat genotypes carrying 18 different powdery mildew resistance genes or combinations of these (Table 1), grown in boxes in the greenhouse containing a mixture of soil, sand and mineral fertiliser, were used to identify the pathotypes and to determine their virulence. The boxes were covered with glass bells to prevent accidental infection.

The plants were inoculated 9–10 days after sowing, in the 1–2-leaf stage, by shaking the conidia, obtained by multiplying the isolates on susceptible plants, on to the leaf surface of the test plants. During the experiments the temperature in the greenhouse was 16–22°C, with a relative air humidity of 50–90%. Depending on the amount of sunshine, additional artificial illumination was provided for 6–12 hours a day with an intensity of 9–12 kLux. Ten days after inoculation the type of infection was determined using the method recommended by NOVER (1957). Genotypes given a score of 0–2 were regarded as resistant and those with scores of 3–4 as susceptible.

RESULTS

The virulence of 192 isolates was determined on wheat varieties or lines carrying 18 different powdery mildew resistance genes or combinations of these. The results are summarised in Table 1.

All the pathotypes caused infection in the susceptible control (Carsten V). Among the wheat genotypes carrying *Pm* powdery mildew resistance genes, Chul *(Pm3b)*, Khapli/8*CC *(Pm4a)*, Rektor *(Pm5),* Salzmünde 14/44 and Disponent (both carrying the *Pm8* gene) were susceptible to all the isolates. A high proportion of the isolates (>80%) proved virulent to host plants carrying the *Pm2, Pm3c, Pm3f* and *Pm4b*

Table 1. Number and ratio of pathotypes virulent to wheat genotypes carrying various powdery mildew resistance genes (Martonvásár, 2001/2002)

Pm gene	Pedigree	Virulent pathotypes		*Pm* gene	Pedigree	Virulent pathotypes	
		No.	(%)			No.	(%)
–	Carsten V (susc. check)	192	100.00	*Pm4b*	Weihenstephan M1	184	95.83
Pm1	Axminster	91	47.40	*Pm4b*	**Ronos**	156	81.25
Pm1	**Axminster/8*CC**	85	44.27	*Pm5*	Hope	141	73.44
Pm2	Red Fern	170	88.54	*Pm5*	**Rektor**	192	100.00
Pm2	**Ulka/8*CC**	176	91.67	*Pm6*	TP114/StarkeB	184	95.83
Pm3a	Asosan	187	97.40	*Pm6*	**NK-747**	147	76.56
Pm3a	**Asosan/8*CC**	87	45.31	*Pm7*	Transec	162	84.38
Pm3b	Chul	192	100.00	*Pm7*	**Transfed**	119	61.98
Pm3b	**Chul/8*CC**	34	17.71	*Pm8*	Salzmünde 14/44	192	100.00
Pm3c	Sonora	191	99.48	*Pm8*	**Disponent**	192	100.00
Pm3c	**Sonora/8*CC**	167	86.98	*Pm17*	**Amigo**	129	67.19
Pm3d	**Ralle**	24	12.50	*Pm2,6*	**Maris Huntsman**	109	56.77
Pm3f	Michigan Amber/CC*8	190	98.96	*Pm2+Mld*	Halle 13471	53	27.60
Pm4a+	Khapli	0	0.00	*Pm1,2,9*	**Normandi**	13	6.77
Pm4a	**Khapli/8*CC**	192	100.00	*Pm2,4b,8*	**Apollo**	126	65.63

The COST Action 817 Core Differential Set is in bold

Table 2. Virulence frequencies determined for varieties carrying the same *Pm* genes; number and proportion of isolates having the same virulence (Martonvásár, 2001/2002)

Pm gene	Origin		Isolates with identical virulence	
	Martonvásár	COST817	No.	(%)
Pm1	47.40	44.27	169	88.02
Pm2	88.54	91.67	186	96.88
Pm3a	97.40	45.31	93	48.44
Pm3b	100.00	17.71	35	18.23
Pm3c	99.48	86.98	176	91.67
Pm4a	0.00	100.00	0	0.00
Pm4b	95.83	81.25	157	81.77
Pm5	73.44	100.00	141	73.44
Pm6	95.83	76.56	155	80.73
Pm7	84.38	61.98	137	71.35
Pm8	100.00	100.00	192	100.00

genes. A small, but detectable number of isolates were found to be virulent to the *Pm3d* gene (Ralle) and to the gene combination *Pm1,2,9* (Normandi), while none of the isolates were capable of infecting the variety Khapli, which contained gene *Pm4a* plus two unidentified genes.

As can be seen from Table 1, in many cases genotypes carrying the same *Pm* gene but originating from different differential sets responded differently when inoculated with the same isolate. The data for genotypes from the two differential sets carrying the same genes are compared in Table 2, which gives the number and ratio of isolates showing the same type of virulence.

Among the differential varieties carrying the same *Pm* genes, the virulence of the isolates only concurred completely in the case of *Pm8*, where both varieties (Salzmünde 14/44 and Disponent) were infected by all 192 isolates. For varieties carrying genes *Pm2* and *Pm3c* more than 90% of the isolates (86.88 and 91.67%, respectively) exhibited the same type of infection, while genotypes carrying resistance genes *Pm1, Pm4b, Pm5, Pm6* and *Pm7* gave the same response to 71.4–88.0% of the isolates. A weak correlation could be demonstrated between the differential varieties in the case of *Pm3a* and *Pm3b*. Among the two varieties carrying *Pm4a*, Khapli was not infected by any of the isolates, while the near-isogenic line Khapli/8*CC was infected by all 192 isolates.

DISCUSSION

The virulence data determined using the differential set now used for many years in Martonvásár are well correlated with earlier data (SZUNICS *et al.* 2000). Even in 1985 the ratio of pathotypes virulent to the *Pm8* resistance gene was over 80% and since 1990 it has continually approached or reached 100%. Since the tests were begun there has been a constant increase in the proportion of isolates in the powdery mildew population capable of infecting varieties carrying the *Pm2, Pm3b, Pm4b, Pm5, Pm6* and *Pm7* genes. Virulence frequencies similar to those recorded in Martonvásár have been found for these genes in several European countries (CLARKSON 2000). A high proportion of pathotypes virulent to the *Pm17* (Amigo) gene could be demonstrated in the powdery mildew population even when the tests were first begun. In this case, however, it should be noted that the tests are carried out in the seedling stage, while the data indicate that this gene provides more efficient protection in the adult stage. In some cases, where the two differential sets contain different genotypes with the same resistance genes, the difference in the extent of infection was greater than expected. The greatest difference (100%) was observed for Khapli and Khapli/8*CC, both carrying the *Pm4a* gene. According to MOSEMAN *et al.* (1980) the reason for the discrepancy is the presence of two further resistance genes in Khapli in addition to *Pm4a*, while the near-isogenic line produced by backcrossing to Chancellor has only two resistance genes. It is difficult to explain the difference in virulence frequency between the genotypes carrying the *Pm3b* gene (Chul and Chul/8*CC), since only 18% of the isolates can be regarded as identical, and the original variety was infected by all the isolates while the near-isogenic line was one of the most resistant genotypes. A substantial difference of a similar type was also observed for another allele of the *Pm3* gene, *Pm3a*.

The results of these tests indicate that further joint tests on the two differential sets will be required for the full appraisal of the powdery mildew population, since the virulence frequencies determined using the two sets differed considerably for some genes. The varieties recommended by the COST Action 817 need to be used in order to achieve uniformity and data convertibility, but changes in the virulence levels determined with the old test collection over several decades and the dynamics of change in the powdery mildew population can only be studied using data from the original test collection.

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