Variability of the Wheat Powdery Mildew Pathogen *Blumeria* graminis f. sp. tritici in the 2003 Crop Season

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ABSTRACT

Wheat (*Triticum aestivum*) powdery mildew, caused by the biotrophic fungus *Blumeria graminis* f. sp. *tritici*, is one of the most severe foliar diseases attacking this crop, reducing grain yields by 10% to 62% in Brazil. The disease can be controlled by genetic resistance of the host, but the pathogen has physiological specialization, which enables it to infect wheat cultivars that have remained resistant for years. The objective of this work was to evaluate the variability of pathogenic strains of *B. graminis* f. sp. *tritici* collected in Brazil and the effectiveness of wheat resistance genes to powdery mildew in the 2003 crop season. Plants of a differential series were inoculated with each monopustular isolate. Thirty-one combinations of effective and ineffective resistance genes were identified. Only the gene Pm4a+... remained totally effective to all isolates, and gene Pm6 was highly effective (below 10% of susceptibility), whereas genes Pm3a and Pm8 were totally ineffective (susceptible to all isolates). Genes Pm3c, D1, and D2 showed low effectiveness (above 50% of susceptibility), and genes Pm1, Pm1,

Additional keywords: Erisyphe graminis f. sp. tritici, Triticum aestivum, genetic of populations, physiological specialization.

RESUMO

Variabilidade do agente causal de oídio de trigo, Blumeria graminis f. sp. tritici, na safra 2003

Oídio de trigo (*Triticum aestivum*), causado pelo fungo biotrófico *Blumeria graminis* f. sp. *tritici*, é uma das principais doenças desta cultura, levando a danos entre 10% e 62% no rendimento de grãos, no Brasil. A doença pode ser controlada por meio de resistência genética, porém o patógeno apresenta especialização fisiológica, o que o torna capaz de infetar cultivares de trigo resistentes em anos anteriores. Este trabalho teve como objetivo avaliar a variabilidade de populações patogênicas de *B. graminis* f. sp. *tritici* coletadas no Brasil e a efetividade de genes de resistência de trigo a oídio, na safra 2003. Plantas de trigo da série diferencial para raças foram inoculadas com cada isolado monopustular. Foram identificadas 31 combinações de genes efetivos e inefetivos para resistência. Para as amostras da população de oídio estudada, o gene de resistência de trigo *Pm4a+...* permaneceu totalmente efetivo para todos os isolados, e o gene *Pm6* foi altamente efetivo (abaixo de 10% de suscetibilidade), enquanto os genes *Pm3a* e *Pm8* foram totalmente inefetivos (suscetíveis a todos os isolados). Os genes *Pm1*, 2, 4a, 1+? e 2+Mld foram medianamente efetivos para a maioria dos isolados (entre 10% e 49% de suscetibilidade), e *Pm3c*, *D1* e *D2* mostraram baixa efetividade (acima de 50% de suscetibilidade). A fórmula de virulência *Pm1*, 3c, 4a, 6, 1+?, 2+Mld, 4a+..., D2 (genes efetivos) / 2, 3a, 8, D1 (genes inefetivos) foi a mais freqüentemente encontrada, respondendo por 15% das ocorrências. O número mais freqüente de genes inefetivos foi sete, variando entre três e dez.

Palavras-chave adicionais: Erisyphe graminis f. sp. tritici, Triticum aestivum, genética de polulações, especialização fisiológica.

Wheat (*Triticum aestivum* L.) powdery mildew, caused by the biotrophic fungus *Blumeria graminis* (DC) E.O. Speer f. sp. *tritici* Em. Marchal, is one of the most severe foliar diseases attacking this crop. In susceptible wheat cultivars and under favorable weather conditions (temperatures relatively cool and humid) the disease can cover the plant surface completely, mostly on the upper leaf surface, withering and weakening the plants. In Brazil, it is found in the Southern Region and under irrigated cropping system in Center West and Southeast regions. In the State of Rio Grande do Sul, yield reduction ranges from 10% to 62% (Fernandes

et al. 1988; Linhares, 1988; Reis et al., 1997). Szunics et al. (2001) reported damages from 5% to 8%, in years regarded as normal disease occurrence.

In South America, the epidemiological importance of cleistothecia is yet to be determined, since ascospore maturation takes place after wheat harvest (Mehta, 1993; Cunfer, 2002). Primary inoculum mostly comes from voluntary wheat plants and/or from infected wheat crops (Mehta, 1993).

As the pathogen has physiological specialization, commercial wheat cultivars with few resistance genes can

cause selection pressure, changing the pathogen population genes frequency, which enables it to infect wheat cultivars that have remained resistant for many years (Bennett, 1984; Niewoehner & Leath, 1998). Therefore, periodic large scale surveys of the virulence frequency of B. graminis f. sp. tritici population are needed to identify effective resistance genes, in addition to detecting virulence changes, genetic diversity, and geographic patterns of pathogen population. These surveys also help to select resistance sources to be used in wheat breeding programs (Niewoehner & Leath, 1998). The virulence frequencies to powdery mildew resistance genes have been assessed intensively in Europe and in the United States of America, but this work is recent in Brazil. Costamilan & Linhares (2002), analyzing results obtained from powdery mildew isolates from Brazil and Chile over five wheat crop seasons, identified 90 different combinations of effective and ineffective resistance genes. During this period, Pm2 and Pm4a+... have remained totally effective for all isolates. In 2003, Costamilan (2004) found that only Pm4a+... remained totally effective for Brazilian strains of B. graminis f. sp. tritici, although Pm1, Pm2, Pm4a, and *Pm6* could still be considered moderately efficient.

Wheat powdery mildew isolates are collected annually in wheat growing areas of Brazil and are assessed at Embrapa Trigo, in order to gather information about virulence frequency on host resistance genes. The aim of this work was to evaluate the variability of *B. graminis* f. sp. *tritici* and the effectiveness of wheat resistance genes to powdery mildew, in the 2003 crop season.

Thirty-one samples composed of wheat leaves infected by *B. graminis* f. sp. *tritici* (one from Minas Gerais, two from São Paulo, 24 from Paraná, and four from Rio Grande do Sul) were received at Embrapa Trigo, in Passo Fundo, Brazil, in 2003. Tests were carried out in a greenhouse, under temperature ranging from 18 to 30 °C. Inoculum was collected from the original infected leaves with the aid of a

spatula and placed on the leaves of ten-day old plants of the susceptible cultivar IAS 54, in order to purify and increase the inoculum. Each IAS 54 inoculated plant was isolated inside a plastic chamber during the period of inoculum increasing. Three monopustular isolates were obtained from each sample, multiplied several times, and, then, screened on ten-day old plants from the differential host series to determine the virulence genes associated with each isolate. This was done by rubbing the infected leaves of the cultivar IAS 54 against the leaves of each cultivar. The differential host series consisted of twelve wheat cultivars, identified by their respective gene or gene combinations for resistance to powdery mildew: Axminster (Pm1), CI 12632/8*Cce (Pm2), Asosan/8*Cce (Pm3a), Sonora/8*Cce (Pm3c), Khapli/8*Cce (Pm4a), CI 13381/8*Prins (Pm6), Weique/8*Prins (Pm8), AsII (Pm1+?), Halle Stamm 13471 (Pm2+Mld), Khapli (Pm4a+...), CI 13374/8*Prins (PmD1) and PI 170913/ 8*Prins (*PmD2*), and the universal suscept, IAS 54. Reaction assessment was carried out after a period of seven to 14 days following inoculation, using a graduated scale varying from 0 to 5 (Costamilan, 2002). Genotypes with reaction up to grade 2+ were considered resistant.

Virulence formula of isolates of wheat powdery mildew found in 2003, with effective and ineffective Pm genes, according to the frequency of occurrence and isolate origin, is shown in Table 1. The virulence frequency per Pm gene in the samples assessed is provided in Figure 1. Only gene Pm4a+... remained totally effective to all isolates, and gene Pm6 was highly effective, whereas genes Pm3a and Pm8 were totally ineffective (susceptible to all isolates). Genes Pm3c, D1, and D2 showed low effectiveness, and genes Pm1, 2, 4a, 1+?, and 2+Mld had an intermediate effectiveness. Pm2 loss of effectiveness was observed, though this gene had been totally effective for many years (Costamilan & Linhares, 2002).

Thirty-one strains with different effective and

TABLE 1 - Virulence formulas of Blumeria graminis f. sp. tritici strains collected in Brazil in 2003

Pm gene		Origin ¹
Effective / Ineffective	- %	Origin
1, 3c, 4a, 6, 1+?, 2+Mld, 4a+, D2 / 2, 3a, 8, D1	15	RS, PR
1, 2, 4a, 6, 2+Mld, 4a+ / 3a, 3c, 8, 1+?, D1, D	2 11	RS, PR
1, 2, 6, 2+Mld, 4a+ / 3a, 3c, 4a, 8, 1+?, D1, D2	11	RS, PR
4a, 6, 1+?, 4a+, D2 / 1, 2, 3a, 3c, 8, 2+Mld, D1	9	SP, RS, PR
6, 1+?, 4a+ / 1, 2, 3a, 3c, 4a, 8, 2+Mld, D1, D2	5	PR.
1, 2, 4a, 6, 1+?, 2+Mld, 4a+ / 3a, 3c, 8, D1, D2	4	SP, PR
1, 4a, 6, 1+2, 2+Mld, 4a+, D2 / 2, 3a, 3c, 8, D1	3	PR
4a, 6, 1+?, 4a+ / 1, 2, 3a, 3c, 8, 2+Mld, D1, D2	3	PR
4a, 6, 4a+, D2 / 1, 2, 3a, 3c, 8, 1+?, 2+Mld, D1	3	MG, PR
4a, 6, 4a+ / 1, 2, 3a, 3c, 8, 1+?, 2+Mld, D1, D2	3	PR
4a, 6, 1+?, 2+Mld, 4a+, D2 / 1, 2, 3a, 3c, 8, D1	2	SP, RS
4a, 6, 1+?, 2+Mld, 4a+ / 1, 2, 3a, 3c, 8, D1, D2	2	PR
1, 2, 4a, 6, 2+Mld, 4a+, D2 / 3a, 3c, 8, 1+?, D1	2	PR
4a, 6, 2+Mld, 4a+, D2 / 1, 2, 3a, 3c, 8, 1+?, D1	2	MG
1, 4a, 6, 1+?, 2+Mld, 4a+ / 2, 3a, 3c, 8, D1, D2	2	PR
(other 16 different combinations)	1(E a	ch)RS, PR

¹Brazilian states: RS (Rio Grande do Sul); PR (Paraná); MG (Minas Gerais); SP (São Paulo).

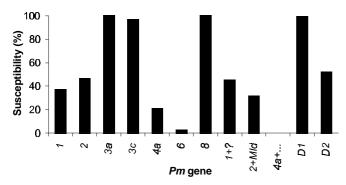


FIG. 1 - Virulence frequency of *Blumeria graminis* f. sp. *tritici* samples collected in Brazil in the 2003 crop season, on wheat (*Triticum aestivum*) resistance genes.

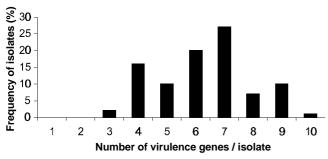


FIG. 2 - Number of virulence genes per isolate of *Blumeria* graminis f. sp. tritici in the 2003 population.

ineffective resistance gene combinations were identified in the populations collected in 2003, which confirms the wide variability of the pathogen (Table 1, showed only the most frequents, above 1%). The virulence formula Pm1, 3c, 4a, 6, 1+?, 2+Mld, 4a+..., D2 (effective genes) / 2, 3a, 8, D1 (ineffective genes) was the most frequently found, accounting for 15% of the occurrences. The most frequent number of ineffective genes was seven (Figure 2), ranging from three (obtained in Rio Grande do Sul and Paraná) to ten (from Paraná).

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