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Genetic variability of *Puccinia triticina* Eriks. in Brazil

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ABSTRACT

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Studies on the genetic variability of *Puccinia triticina* in inoculum collected in Brazil started in 1941 with Vallega (20). The pioneering work in Brazil dates from 1949 (16) at "Instituto Agronômico do Sul", Ministry of Agriculture (MA), in Pelotas, Rio Grande do Sul State (RS), and continued after 1975 at Embrapa Wheat in Passo Fundo, RS. In 2002, analyses for the identification of *P. triticina* races continued at OR Seed breeding, simultaneously to Embrapa's program, both in Passo Fundo. The investigators involved in the identification of races in Brazil were Ady Raul da Silva in Pelotas (MA), Eliza Coelho in Pelotas (MA) and in Passo Fundo (Embrapa), Amarilis Labes Barcellos in Pelotas (MA) and in Passo Fundo (Embrapa and OR), Camila Turra in Passo Fundo (OR) and Marcia Chaves in Passo Fundo (Embrapa). From 1979 to 2010 growing season, 59 races were determined, according to the differentiation based on the expression of each *Lr* resistance gene. On average, one to three new races are detected per year. Research has focused on the use of vertical resistance; however, lately some institutes have searched more durable resistance, of the adult-plant type (horizontal, less race-specific). The uninterrupted monitoring of the **wheat rust** pathogenic population **in Brazil** during so many decades allowed the understanding of the evolution and virulence of races. The use of international nomenclature adopted by some programs has allowed the comparison of the fungus variability in Brazil with that in other countries, especially where frontiers are not barriers for spore transportation, confirmed by the occurrence of the same races all over one region.

Additional keywords: Leaf rust, *Triticum aestivum*, races.

RESUMO

Bianchin, V.; Barcellos, A.L.; Reis, E.M.; Turra, Camila. Variabilidade genética de *Puccinia triticina* Eriks. no Brasil. ***Summa Phytopathologica,*** v.38, n.2, p.113-118, 2012.

Os estudos da variabilidade genética de *Puccinia triticina* em inóculo coletado no Brasil começaram em 1941 por Vallega. O trabalho pioneiro no Brasil teve início em 1949 no Instituto Agronômico do Sul, Ministério da Agricultura (MA) em Pelotas, RSs, depois de 1975 na Embrapa Trigo em Passo Fundo, RS. Em 2002 prosseguiram as análises de identificação de raças na OR Melhoramento de Sementes, paralelamente ao programa da Embrapa, ambos em Passo Fundo. Trabalharam na identificação de raças no Brasil, Ady Raul da Silva em Pelotas (MA), Eliza Coelho em (Pelotas (MA) e em Passo Fundo (Embrapa), Amarilis Labes Barcellos em Pelotas (MA) e em Passo Fundo (Embrapa e OR), Camila Turra em (Passo Fundo (OR) e Márcia Chaves em (Passo Fundo (Embrapa). Até a safra 2010, 59 raças já foram determinadas, considerando-se a partir de 1979, de acordo com a diferenciação pela expressão de cada gene de resistência *Lr*. Na média surge uma a três raças por ano. A pesquisa tem se concentrado no uso da resistência vertical, porém, ultimamente, algumas instituições têm buscado a resistência mais durável, do tipo planta adulta (horizontal, menos específica a raças). A não interrupção do monitoramento da população patogênica da ferrugem da folha do trigo no Brasil durante tantas décadas possibilitou conhecer a evolução das raças e da virulência. Embora tenha havido esforço de alguns programas O uso da nomenclatura internacional adotado por alguns programas tem permitido a comparação da variabilidade do fungo ocorrida no Brasil com a de outros países e especialmente com aqueles onde as fronteiras não são barreiras para o transporte dos esporos, como confirmado pela ocorrência de mesmas raças em toda a região.

Palavras-chave adicionais: Ferrugem da folha**,** *Triticum aestivum*, raças.

Wheat leaf rust caused by *Puccinia triticina* Eriks. is one of the most important diseases of the crop worldwide. Many years ago, Silva et al. (17) considers the adult plant resistance of the Brazilian cultivar Frontana adequate protection, as well as for many of its cultivated derivatives. The low incidence of wheat leaf rust was considered by breeders a secondary problem. Consequently, the

Secretariat of Agriculture of Rio Grande do Sul State decided not to study the basic principles, while "IPEAS", Ministry of Agriculture – (MA) continued the surveys on wheat leaf rust and evaluations at seedling stage, as supplemental information. This guideline resulted in regression and leaf rust became one of the most important wheat diseases.

This is one of the most studied diseases and has worldwide importance. The dynamic nature of the fungus, the annual occurrence of the disease and the cultivation of genotypes with specific resistance favor the emergence of new races. The search for genotypes with durable resistance (adult plant resistance, horizontal resistance or non-specific resistance), which allows the maintenance of resistance effectiveness over the years, has been a major challenge for plant breeding programs.

The pathogen co-evolves to adapt to new resistant genotypes introduced by the breeding programs as the area of new cultivars increases. This co-evolution is noticed by the interaction of pathogen genes with the host genes, demonstrating the fungus capability to adapt.

The change in the pathogen population results in new physiological races, not morphologically but physiologically different and having the ability to infect different wheat cultivars (12).

The aim of this study was to gather the information in the literature and consult wheat leaf rust experts regarding the variability of the fungus *Puccinia triticina* Eriks. in Brazil. Most data were not published or are difficult to be found.

Methodology for race identification

Initially, a literature survey on the early work on the identification of *P. triticina* races in Brazil was conducted to recover the races found until 2008. For the identification of races occurring in 2008 and 2009, infected wheat leaves were collected and the races were identified.

Sampling was done in wheat fields over all growing areas of Brazil. The rust-infected leaves were collected and sealed in wax paper bags and brought to the rust laboratory of OR Seed Breeding Ltd., located in Passo Fundo – RS, where they were stored in refrigerator at 5°C.

Inoculum isolation and multiplication were done in living plants due to the condition of the biotrophic parasite. Thus, the cultivars susceptible to all races (Devoid of *Lr* seedling genes), PG1 and Morocco, were used. The inoculation was done at seedling stage 11 (First expanded leaf) (17), initially removing plant serosity by moistening the leaves and passing hand fingers. Uredospores were scraped from the leaves and transferred to the plants by using a spatula. Then, inoculated plants were sprayed with water + Tween 20 (Polyoxiethylenesorbitane monolaurate - Synth - four drops per liter of water). The incubation period was 20 to 24 hours in the dark at 20°C and 100% relative humidity. Once the inoculum had multiplied, the spores were collected and stored in gelatin capsules, using suitable collectors coupled to a vacuum pump.

To identify the race, a suspension of uredospores in Soltrol mineral oil was sprayed on a differential set of wheat isogenic lines (Table 1) using a proper sprayer adapted to a compressed air pump. Inoculation was separately done with each individual rust sample.

The races are identified based on the symptoms that each gene *Lr* can express (10) and grouped according to the nomenclature system for North America (9) and the Brazilian correspondent (Brazilian nomenclature for race classification) for the response avirulence/virulence genes, concerning 19 known *Lr* (Leaf rust) genes that were previously inserted in isogenic lines of Thatcher cultivar: *Lr1, Lr2a, Lr2c, Lr3, Lr3ka, Lr9, Lr10, Lr11, Lr14a, Lr14b, Lr16, Lr17, Lr18, Lr20, Lr21, Lr23, Lr24, Lr26* and *Lr30* (Table 1). The following differentials were added to the main series: *Lr3bg, Lr19*, *Lr27+Lr31* in addition to a gene not yet cataloged and important to distinguish the actual predominant race.

Table 1. Reaction code of 19 genes of a differential cultivar set to *Puccinia triticina* (Pt), separated into five groups of four genes

		Type of symptoms produced by the pathogen in the isogenic lines with different *Lr* genes			
	Group 1	1	2a	2c	3
	Group 2	9	16	24	26
	Group 3	3ka	11	17	30
	Group 4	10	18	21	23
Pt code	Group 5	14a	14b	10 + 26	20
B		A	A	A	A
C		A	A	A	V
D		A	A	V	A
F		A	A	V	V
G		A	V	A	A
H		A	V	A	V
J		A	V	V	A
K		A	V	V	V
L		V	A	A	A
M		V	A	A	V
N		V	A	V	A
P		V	A	V	V
Q		V	V	A	A
R		V	V	A	V
S		V	V	V	A
T		V	V	V	V

The result of group 1 will lead to the first letter of the race code; group 2, the second letter; and group 3, the third letter. Similarly, for groups 4 and 5, the fourth and fifth letters, respectively.
A = avirulent; V = virulent.
Adapted from Long & Kolmer (9).

To achieve the classification proposed by Long & Kolmer (9), one must consider the scale adapted by Roelfs and Martens (14), which determines whether or not the pathogen is virulent (Figure 1 and Table 2).

The reaction pattern of the virulence set of ineffective *Lr* genes for resistance, the determination of the North American code and the search in the literature allows learning whether or not the pathogen is a new physiologic race.

Studies on the physiologic specialization of *P. triticina* in Brazil started in 1941 by Vallega (20). During that period, the races were not named as they are today, using the letter "B" and the corresponding number. They were simply referred to as the corresponding number in identification order, e.g. races 19, 64 and 105 (1).

Using the international differential series in the period 1949-1952, 26 races were identified. Races 20, 77 and 31 were most common (16). After 1952, a new set of cultivars were applied as differentials in naming the races IAS, also maintaining temporarily the international set. The cultivars used to distinguish races were: Bagé, Rio Negro, Sinvalocho, Klein Lucero, Lee, Gabo, Buck Tandil and Timstein. Twenty-eight races and ten sub-races were determined. Race determination was based on the differential set described by Silva et

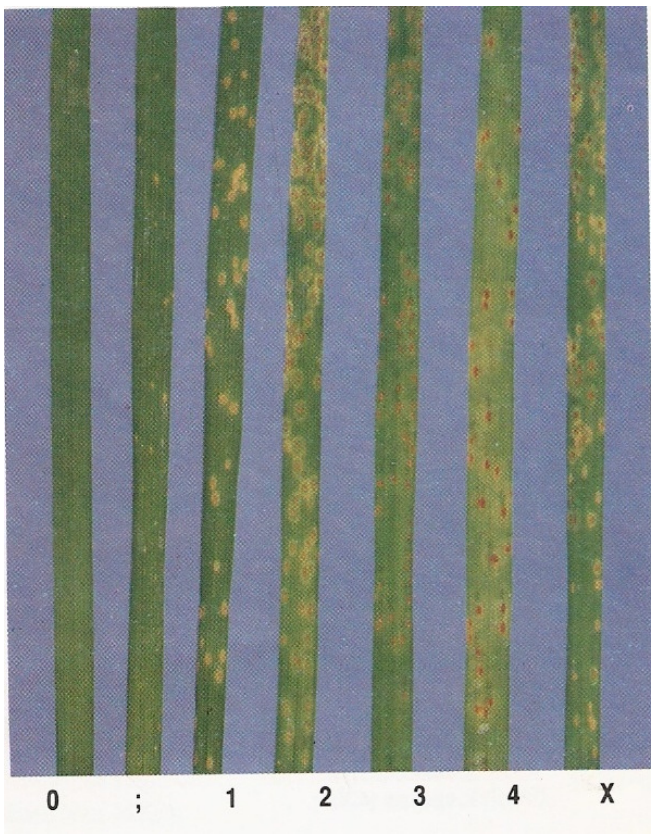

Figure 1. Scale to assess the reaction of close isogenic seedling lines to *Puccinia triticina* (Photo: Singh, R.P.).

Table 2. Description of infection type and symptoms caused by *Puccinia triticina* in whet leaves

Infection type	Low /High	Symptoms
0	Low	No uredinia or other macroscopic sign of infectiton
0;	Low	Few faint flecks
;	Low	No uredinia, but hypersensitive necrotic or chlorotic flecks present
1	Low	Small uredinia often surrounded by necrosis
2	Low	Small-to-medium uredinia often surrounded by chlorosis
Y	Low	Ordered distribution of variable-sized uredinia, largest at the leaf tip
X	Low	Random distribution of variable-sized uredinia
3	High	Medium-sized uredinia without chlorosis or necrosis
4	High	Large uredinia without chlorosis or necrosis

The infection types are often refined by modifying characters as follows	
(=)	uredinia at the lower size limit for the infection type
(-)	uredinia somewhat smaller than normal for the infection type
(+)	uredinia somewhat larger than normal for the infection type
(++)	uredinia at the upper size limit for the infection type
C	more chlorosis than normal for the infection type
N	more necrosis than normal for the infection type

Adapted from Roelfs & Martens (1988).

al. (15) and Cenoz (5).

Races 25 and 26 were determined for the first time in 1961, race 27 in 1962, and race 28 in 1970. Among the races identified between 1958 and 1974, the most important occurring races were 4 and 19. In 1975 and 1976, the pathogen population has changed and race 19 lost its importance (1).

Since 1977, *P. triticina* races have been identified according to the virulence combination obtained by the reaction of lines carrying the *Lr* resistance genes. In 1978, virulence formulae were named with the letter "B" (Brazil) followed by the number in order of identification (1). From races B1 to B9, the differential set was different from that used in the subsequent years; therefore, it was not possible to compare these races with B10 to B58 (Last race with Brazilian nomenclature) (Table 3 and 4). Races identified by the early 80s have information of effective and ineffective *Lr* genes; however, there are difficulties in determining the old races using the North American current code because not all genes that are considered today were part of the differential set (Table 3).

From 1978 to 1980, races B12, B10, B14 and B11 were most expressive, respectively (1). In 1984 and 1985, the most important race was B25 (2).

From 1990 to 1993, seventeen races were identified. In 1990, the most important race was B32; in 1991, B35; in 1992, B38; and in 1993, B25. In terms of importance during this period, races B25 and B35 can be highlighted (11).

From 1999 to 2002, the predominant races were B40 and B48, corresponding in 2002 to the frequency of 41% and 12%, respectively (6). In 2004, race B55 M(DF)T-M(RT) (*Lr* virulence: *Lr1, Lr3, Lr3ka, Lr10, Lr11, Lr14a, Lr14b, Lr17, Lr20, Lr23, Lr24, Lr26, Lr30*) was first identified and, from 2005, predominated, causing damage in all wheat growing areas. In 2007, this race underwent a modification and was named B55 4002S, M(DF)T-M(RT) 4002S (*Lr* virulence: *Lr1, Lr3, Lr3ka, Lr10, Lr11, Lr14a, Lr14b, Lr17, Lr20, Lr23, Lr24, Lr26, Lr30,* 4002S, due to the virulence in the line OR 4002, while all genes of the current differential series express the same avirulence/virulence default). Race B55 4002S has dominated the leaf rust population from 2005 to the last still partial 2010 survey (Personal information, data OR Seed breeding).

In 2008 growing season, three new races were identified: TDP-MR, TPT-HT and TDP-HR (Table 4). From the samples handled in the Rust Laboratory of OR – Seed breeding, 102 isolates were obtained, 58% of these were identified as race B55 4002S, 16.5% as B57, 6% as B58, and 19.5% as other races. The predominant race in 2008 was B55 4002S.

The environmental conditions were not favorable to rust epidemics in 2009. During this season, 40 isolates were obtained, of which 75% were race B55 4002S. A new race was identified, probably a variant of race B58 identified in 2005. The new race showed the same susceptibility to wheat line 4002 [unknown gene(s)], thus B58 4002S, while 15% of the isolates were identified as B58 4002S.

Breeding as a control strategy

The best strategy to control wheat leaf rust is the development of resistant cultivars. Vertical resistance has no long-term duration, is easily defeated by the pathogen specialization, is easily obtained and widely used in breeding programs. On the other side, the adult plant resistance is long-lasting but more difficult to achieve and remains a major challenge for breeders.

Most of the effective resistance found for *P. triticina* in cultivated wheat varieties is given by the combination of major genes present in

Table 3. Early physiologic races of *Puccinia triticina* in Brazil according to the Brazilian nomenclature and identified by the virulence formulae

Races	Avirulence/virulence formulae	
	Effective *Lr* genes	Ineffective *Lr* genes
B1	2a, 2c, 2d, 9, 10, 16, 17, 18, 21	1, 3, 3ka, 14a
B2	2a, 2c, 2d, 9, 16, 17, 18, 21	1, 3, 3ka, 10, 14a
B3	2a, 3, 3ka, 9, 10, 16, 17, 18	1, 2c, 2d, 14a, 21
B4	2a, 3, 3ka, 9, 10, 16, 17, 18, 21	1, 2c, 2d, 14a
B5	2a, 3, 3ka, 9, 16, 17, 18	1, 2c, 2d, 10, 14a, 21
B6	2a, 3, 3ka, 9, 16, 17, 18, 21	1, 2c, 2d, 10, 14a
B7	2a, 3, 9, 16, 17, 18, 21	1, 2c, 2d, 3ka, 10, 14a
B8	3, 3ka, 9, 16	1, 2a, 2c, 2d, 10, 14a, 17, 18, 21
B9	3, 3ka, 9, 16, 21	1, 2a, 2c, 2d, 10, 14a, 17, 18
B10	2a, 3, 3ka, 9, 10, 16, 17, 18, 21 , 23, 24, 26	1, 2c, 2d, 14a, 14b
B11	2a, 3, 3ka, 9, 10, 16, 17, 18, 21 , 24, 26	1, 2c, 2d, 14a, 14b, 23
B12	2a, 3, 3ka, 9, 10, 16, 17, 18, 21 , 23, 24, 26	1, 2c, 2d, 10, 14a, 14b
B13	2a, 3, 9, 16, 17, 18, 21 , 23	1, 2c, 2d, 3ka, 10, 14a, 14b
B14	3, 3ka, 9, 16, 21 , 23, 24, 26	1, 2a, 2c, 2d, 10, 14a, 14b, 17, 18,
B15	1, 2a, 3, 3ka, 9, 10, 16,17, 18, 21, 23, 24, 26	2c, 2d, 14a,14b
B16	2a, 2c, 2d, 3, 3ka, 9, 10, 16,17, 18, 21, 24, 26	1, 14a, 14b, 23
B17	3, 3ka, 9, 10, 14a, 16, 21, 23, 24, 26	1, 2a, 2d, 17, 18
B18	2a, 2d, 9, 10, 16,17, 18, 21, 24, 26	1, 3, 3ka, 14a, 14b, 23
B19	1, 2a, 3, 3ka, 9, 10, 16, 17, 18, 21, 24, 26	2d, 14a, 14b, 23
B20	1, 2a, 3, 3ka, 9, 10, 16, 17, 18, 21, 23, 26	2d, 14a, 14b, 24
B21	2a, 2d, 3, 3ka, 9, 16, 17, 18, 21, 24, 26	1, 10, 14a, 14b, 23
B22	2a, 3, 3ka, 9, 16, 17, 18, 21, 24, 26	1, 2d, 10, 14a, 14b, 23
B23	2a, 2d, 3, 9, 16, 17, 18, 21, 24, 26	1, 3ka, 10, 14a, 14b, 23
B24	2a, 3, 3ka, 9, 10, 16, 17, 18, 21 , 23, 24, 26	1, 2d, 14a, 14b

the germplasm of the Southern Cone of South America region. The most common resistance genes in the Brazilian germplasm published in 2002 were *Lr10, Lr23, Lr24* and *Lr26* (21). For adult plants, the resistance genes *Lr*13 and *Lr*34 are present in Brazilian cultivars (21). Adult plant resistance can be effective by two other genes described in Toropi, *Trp*1 and *Trp*2 (3), and according to Brammer et al. (4), there is a third unidentified gene in BR35 cultivar.

In Argentina, the effective genes *Lr9, Lr10, Lr19, Lr20, Lr24, Lr26, Lr34, Lr37* and Lr47 were identified in 98 recent cultivars, alone or in combinations of two or three (7). In Uruguay, *Lr*3, *Lr10*, *Lr14b*, *Lr16*, *Lr17a*, *Lr24, Lr26* were found for seedling resistance and *Lr13* and *Lr34* for adult plant resistance (8).

There are other genes that express adult plant resistance, some less race-specific than others and consequently lasting longer and shorter. Genes *Lr35*, *Lr37* and *Lr22a* are race-specific for adult plant resistance. *Lr34* and *Lr46* are genes for adult plant resistance, non-race-specific.

Some additive minor genes confer resistance to adult plants and have been used in breeding programs (18). Some examples of materials distributed by CIMMYT with these minor additive genes are Parula (*Lr34, Lr46* + one or two minor genes), Chapio (*Lr34* + three or four minor genes), Amadina (four minor genes) among other lines with this type of resistance (19). These materials distributed by CIMMYT are used in crosses with adapted material in breeding programs in South America.

Adopting the methodology of 'One Backcross', divulged by Ravi Singh, from CIMMYT, the OR Seed breeding wheat leaf rust program developed lines in one same adapted OR cultivar, each of them with the following resistances: Amadina, Chapio, Frontana, Kukuna, Tukuru and Toropi.

The pyramiding of genes in order to obtain additive effect by them is a viable strategy. In a single cultivar, through breeding techniques, multiple genes are introduced in order to achieve greater efficiency and most enduring resistance to the fungus; however, the disadvantage is to expose many genes at once at risk of pathogen adaptation. Another strategy is the genetic control by means of cultivation of different combinations of genes in different epidemiological zones and the inclusion of gene rotation. However, this practice is difficult to implement as it requires commitment by all people involved in the cultivation (13).

Variety mixture and multiline involves the use of different or the same variety with the addition of distinct genes (through backcrossing) on separate lines forming a whole to further cultivation. Such strategy has the advantage of keeping the pathogen population below its capability level of causing epidemic. The disadvantage of using multiline is the risk of selecting a virulent super race able to overcome several genes at the same time and also the difficulty of obtaining lines resistant to leaf rust in conjunction with other diseases (13). It is also difficult due to the need of regularly selecting the genes for an efficient combination.

Table 4. Physiologic races of *Puccinia triticina* (Brazilian nomenclature) identified in Brazil, year of identification, United States code and corresponding genes of possible variations from the differential set

Races	Year	North American nomenclature	Possible variation in North American code
B10	1979	NBG-GR	
B12	1979	NB(B)-LR	NB(B/C/D/F/H/K/G/J)-LR
B14	;	SBJ-LQ	
B22		NB(K)-MR	NB(K/C/D/F/H/B/G/J)-MR
B25	1982	LC(B)-(M)S	LC(B/C/D/F/H/K/G/J)-(M/R)S
B26	1982	NC(B)-TR	NC(B/C/D/F/H/K/G/J)-TR
B27	1983	MB(R)-(M)(R)	MB(R/T)-(M/R)(R/Q)
B28	1983	MB(G)-ML	MB(G/H/J)-ML
B29	1984	TD(R)-(G)L	TD(R/T)-(G/B)L
B30	1983	SD(G)-QR	SD(G/H/J/K)-QR
B31	1985	CB(Q)-HQ	CB(Q/R/S/T)-HQ
B32	1986	LL(B)-HQ	LL(B/C/D/F/H/K/G/J)-HQ
B33	1986	TG(B)-BH	TG(B/C/D/F/H/K/G/J)-BH
B34	1989	MC(G)-(R)(L)	MC(G/Q/S/T/F/H/K/R/J)-(R/M/L/N/C/H)(L/N/S)
B35	1989	MC(R)-(C)(S)	MC(R/G/C/H/J/K/Q/R/T)-(C/H)(S/N)
B36	1989	MBR-RL	MBR-RL
B37	1988	SL(K)-HQ	SL(K/B/C/D/F/G/H/J)-HQ
B38	1991	TB(J)-(R)(R)	TB(J/B/C/H/D/F/G)-(R/Q/M)(R/Q)
B39	1993	CG(T)-CQ	CG(T/R)-CQ
B40	1994	MF(T)-(K)(S)	MF(T/G/H/J/K/L/Q/R/S/T)-(K/C/H)(S/M/L)
B41	1995	SB(B)-BR	SB(B/C/D/F/H/J/K/G)-BR
B42	1995	LF(G)-(M)S	LF(G/B/C/D/F/H/J)-(M/R)(S/Q)
B43	1995	TD(F)-(R)R	TD(F/C/D/F/G/H/J/K)-(R/Q/M)R
B44	1997	LP(G)-(R)S	LP(G/H/J/K)-(R/M)S
B45	1997	CHT-(C)Q	CHT-(C/G)Q
B46	1998	KDJ-QR	KDJ-QR
B47	1998	TDK-(B)L	TDK-(B/G)L
B48	1999	M(C)(N)-(M)T	M(C/H)(N/D/G/J/K/P/Q/R/S/T/C/F)-(M/P/R/L/Q/S)T
B49	2001	TF(T)-(C)S	TF(T/K)-(C/H)S
B50	2002	SPJ-RS	SPJ-RS
B51	2002	M(D)(T)-(C)(R)	M(D/F)(T/R)-(C/H)(R/T)
B52	2003	MF(J)-(M)N	MF(J/S)-(M/M)N
B53	2003	MHT-LS	MHT-LS
B54	2003	TF(T)-(C)T	TF(T/R/K)-(C/H)T
	2003	MBD-MR	MBD-MR
	2003	MCD-MN	MCD-MN
	2003	SNJ-RR	SNJ-RR
	2003	TDT-CR	TDT-CR
B55	2004	M(D)(T)-(M)(R)	M(D/F)(T/K/R/H)-(M/R)(R/T)
B56	2005	MFP-CT	MFP-CT
B55 4002 S	2007	M(D)(T)-(M)(R)	M(D/F)(T/K/R/H)-(M/R)(R/T)
B57	2007	TD(T)-MR	TD(T/R/K)-MR
B58	2005	MD(P)-MR	MD(P/M/F)-MR
	2007	M(F)(P)-H(T)	M(F/D)(P/K/H/R)-H(T/P)
	2008	TDP-MR	TDP-MR
	2008	TPT-HT	TPT-HT
	2008	TDP-H(R)	T(D/F)P-H(R/T)
B58 4002S	2009	MD(P)-MR	MD(P/M/F)-MR(T)

Data from the literature and Dr. Amarilis Labes Barcellos, personal information. Letters in brackets mean the possible race variations due to response instability of some genes on the basis of environmental conditions.
(-) years of identification not possible to recover.

Vertical resistance has been used by most Brazilian wheat breeding programs which subjected *P. triticina* to a high selecting pressure, resulting in a vicious cycle of new resistant cultivar – new virulent race.

Due to the variability of *P. triticina* in Brazil from 1977 (beginning of the use of the differential cultivar set with isolated resistance genes) to 2009, an average of two new races per year are identified.

In 2008, the predominant race was B55 4002S and three new races, TDP-MR, TPT-HT and TDP-HR, were identified.

In 2009, the prevalent race was again B55 4002S and as a new race, MDP MR-4002S, a possible change in race B58 (MDP-MR), was identified.

The use of the international methodology and nomenclature should be pursued for all South American countries with some adaptation.

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