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RESEARCH ARTICLE / *ARTIGO*

Pyricularia blast on white oats - a new threat to wheat cultivation

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ABSTRACT

A newly emerged disease, the Pyricularia blast caused by *Pyricularia grisea*, is commonly observed on wheat and black oats, since its discovery in 1985. Within a few years the disease spread to other species of Gramineae, but white oat cultivars remained resistant and were the only ones among cultivated cereals resistant to this disease. In 2012, one commercial field of white oat cultivar IAC 7 was found severely attacked by Pyricularia blast in Assis, in the state of São Paulo. The objective of the present investigation was to find out whether other white oat cultivars are also susceptible to the new *P. grisea* isolate from cv. IAC 7 and to determine the pathogenic variability, if any, among *P. grisea* isolates from black oats. Thirty-three white oat cultivars, along with some cultivars of black oat, barley, wheat, triticale, and rye were evaluated against the IAC 7 isolate. Results of disease severity analysis indicated that four white oat cultivars were resistant, seven were moderately resistant and the remaining were susceptible or highly susceptible. Five wheat cultivars were also resistant. Pathogenic variation among *P. grisea* isolates from black oats was observed.
Key words: *Avena sativa*, *A. stigosa*, *Magnaporthe grisea*, *Pyricularia grisea*.

INTRODUCTION

Black oats (*Avena strigosa* L.) are being widely used in crop rotation for winter for the past 30 years especially in south of Brazil. The use of oats in crop rotation is of fundamental importance to the conservation tillage system because they are one of the best options for winter. Oats are preferred either for mulch or for fodder because of their capacity to produce high quantity of dry matter (5.5-7.1 $t.ha^{-1}$).

In recent years, the newly emerged disease, the Pyricularia blast caused by *Pyricularia grisea* (Cooke) Sacc. [telemorph *Magnaporthe grisea* (Herbert) Barr], is commonly observed on wheat in Brazil and in some neighboring countries, since its discovery in 1985, causing yield losses between 40 and 100% (Igarashi et al., 1986; Picininn & Fernandes, 1990; Cunfer et al., 1993; Dos Anjos et al., 1996; Mehta 1993; Kohli et al., 2011). Couch & Kohn (2002) suggested *Magnaporthe oryzae* as the correct name for the isolates associated with rice blast and grey leaf spot but not for the wheat and oat blast fungus. At present, the disease is not adequately controlled through fungicidal applications although in some cases triazols combined with strobilurins have been used with little success (Kohli, 2011). The wide virulence diversity in the pathogen population makes breeding for resistance difficult.

Soon after its discovery in 1985, the disease spread to other cereals. Infections on triticale (*X. Triticosecale*) for example, were reported for the first time in 1998 (Mehta & Baier, 1998). These authors reported that under glasshouse conditions, isolates from triticale were aggressive on triticale and oats, less aggressive on wheat, not compatible with rice, and thus were of a different origin than the wheat isolates.

Later, blast infection in commercially grown black oats was also reported (Mehta et al., 2006). Black oats were considered resistant till the year 2004, when the commercial cultivation of this species was attacked by *P. grisea* throughout the State of Paraná. Severe infections of black oats in Brazil were observed in the State of Paraná, in some commercial fields in Londrina, Cornelio Procopio, Cascavel, Medianeira and Santa Terezinha. Exact data on yield or dry matter losses are not available but they were roughly estimated to be between 25 and 30% in the heavily infested fields.

Most of the white oat cultivars then were resistant to *P. grisea* (Mehta et al., 2006). However, in the year 2012, one commercial field of white oat cultivar IAC7 in the State of São Paulo, was found severely attacked for the first time by *P. grisea*. On oats the pathogen attacks the leaves, rachis at the base of the glumes. The glumes become bleached and may not form grains or form shriveled grains. On oat leaves, the symptoms are characterized as elliptical lesions with dark-brown margin and whitish centre. The base of the glume looks dry, strangulated, and dark brown to ash color (Figure 1). Abundant sporulation of the pathogen can be observed on these lesions. Heavily infected seeds fail to germinate.

Since several white oat cultivars were found resistant to *P. grisea* (Mehta et al., 2006) and since there is no report of *P. grisea* infection on other white oats, it is possible

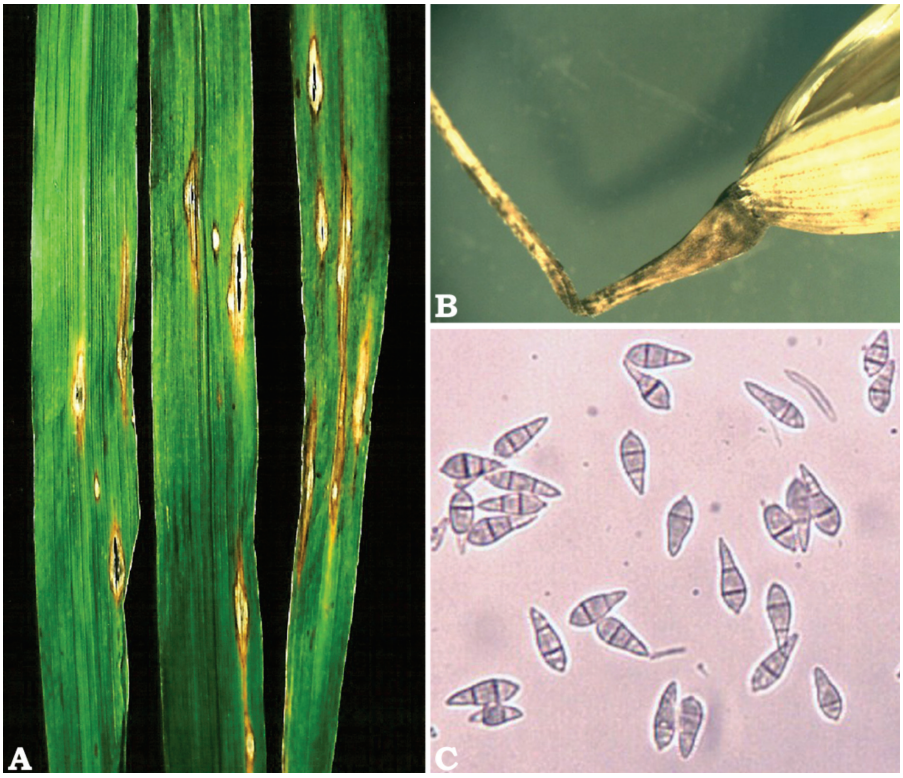

FIGURE 1 – *P. grisea* on white oat. **A.** Symptoms on leaves; **B.** Symptoms on base of the glume; **C.** Conidia.

that some white oat cultivars could still be resistant. The objective of the present investigation was to find out if other white oat cultivars are also susceptible to the new isolate of *P. grisea* from white oats cv. IAC 7, and to comprehend the pathogenic variability among the isolates of *P. grisea* from black oats.

MATERIAL AND METHODS

The pathogen was isolated (isolate no. 15837) directly from the sporulating lesions on infected leaves of cv. IAC 7 received from the field, on water agar containing 0.25 mg/L of streptomycin and later was multiplied on plates containing oat-meal agar. Sporulation was obtained by flattening 12 days old colony with an L-shaped glass rod and incubating under fluorescent light with lids open for three days. Sporulating plates were flooded with distilled water and the spores were removed by a paint brush. Spore suspension was adjusted to 2.5 x 104 spores per milliliter. Twenty-five days old plants grown in the glasshouse were inoculated by a hand sprayer and incubated for 24h in a humid chamber at 20oC. Plants were later transferred to the glasshouse bench. Disease severity was recorded 15 days after inoculation on the leaf showing maximum severity on each plant, using a visual scale between 0 and 1 where: 0 = no visual infection or small brownish-white spots; 0.25 = small elliptical spots with ash color or whitish center, capable of sporulation, spread over the leaf covering <5% leaf area infected (LAI); 0.5 = typical elliptical lesions capable of sporulation covering approximately 25% LAI; 0.75 = typical elliptical lesions covering 25-50% LAI; 1.0 = typical elliptical lesions covering >50% LAI. Three experiments were conducted using identical inoculation and evaluation procedures. In Exp. 1, ten wheat genotypes showing some degree of field resistance to *P. grisea* were used. In this experiment leaf reaction of the wheat genotypes to a mixture of five randomly selected *P. grisea* isolate from black oat was evaluated. In Exp. 2, pathogenic variation of 19 *P. grisea* isolates from black oats on some wheat and oat cultivars was studied. Two wheat cultivars showing field resistance over several years were used along with one susceptible black oat cultivar and one resistant white oat cultivar. Leaf reactions were classified in different groups using Scott & Knott (1974) analysis. Disease severity rating between 0 and 0.08 is considered resistant; between 0.11 and 0.19 were considered moderately resistant; and between 0.21 and more were arbritorily considered as susceptible or highly susceptible (Prabhu et al., 1992; Zadoks, 1972). In Exp. 3, 33 white oat cultivars along with some cultivars of black oat, wheat, triticale, rye and barley were evaluated for their reaction to white oat isolate no. 15837.

RESULTS AND DISCUSSION

In Exp. 1, all the wheat genotypes showed susceptible reaction to a mixture of five isolates from black oats indicating that the black oat isolates had a different origin than the wheat isolates (Table 1). In Exp. 2, pathogenic

variation between the 19 *P. grisea* isolates from black oats was observed (Table 2). With one exception, wheat cv. CD 103 showed susceptible reaction to all the isolates. Cultivar BR 18 showed resistance to 14 isolates and susceptibility to five isolates. White oat cultivar IPR 126 was resistant to 12 isolates and susceptible to seven isolates. These results indicate the presence of high pathogenic variability among the *P. grisea* isolates from black oats. *P. grisea* as a very variable pathogen was also reported earlier (Mehta, 1993; Valent & Chumley, 1991; Urashima et al., 1994a; Urashima et al., 2004b).

In Exp. 3, results of disease severity caused by white oat isolate no. 15837 indicated that out of 33 white oat cultivars four were resistant, seven were moderately resistant and the 22 were susceptible to highly susceptible. Most of the white oat cultivars, one rye cultivar IPR 89 and one white oat cultivar IPR Afrodite were resistant or moderately resistant (Table 3). Interestingly, some wheat genotypes like Milan CD 116, Milan3/Atila/Cimmyt 3, BRS 210 and BRS 229 showed resistance to wheat as well as to oat isolates.

The breakdown of resistance of white oat cultivars is considered serious since among the cereals white oats were the only resistant hosts of *P. grisea*, offering one of the options for crop rotation for winter. The systematically breaking down of resistance of different plant species and their cultivars demonstrates the aggressiveness and wide adaptability of the pathogen.

Prabhu et al. (1992) reported that all of the *P. grisea* isolates from rice, wheat and grass weeds were pathogenic on the wheat cultivars and barley, but none of the 10 wheat and seven grass isolates infected any of the 30 rice cultivars. Similarly, Mehta & Baier (1998) reported variation for virulence and host specificity among *P. grisea* isolates from triticale. In this study, isolates from triticale were aggressive on triticale and oats, less aggressive on wheat, and not compatible with rice, thereby making them to be of a different origin than the wheat and the rice pathotypes. The wheat cv. BRS 229 has been showing reasonable level of resistance under field conditions for several years, but its susceptibility to mixture of black oat isolates, suggests a different origin for the black oat isolates (Table 1). Thus, the constant change in the pathogen population raises doubts about the longevity of the remaining resistant white oat cultivars which could be used either for crop rotation or for grain production.

A threat to wheat cultivation due to the expansion of the disease in the southern cone region of Latin America was reported earlier (Kohli et al., 2011). Although the primary infection may come from several secondary hosts (Prabhu et al., 1992; Urashima et al, 2004a, 2004b), the fast spread of Pyricularia blast from wheat to several gramineous hosts like triticale, foxtail millet (*Setaria italica*), black oats, and white oats, in a relatively short span of time is a matter of concern. It poses a new threat to wheat and oat cultivation in the region. Besides limiting the option for crop rotation in winter, the susceptibility of white oats would add inoculum of *P. grisea* in the atmosphere and would further complicate the disease management strategies in Brazil as well as in the neighboring countries.

Breeders and pathologists need to work faster to develop resistant cultivars by biotechnological as well as conventional means to prevent selection of more and more

TABLE 1 - Differential response of wheat cultivars to infection caused by mixture of five *P. grisea* isolates from black oats, under glasshouse conditions

Cultivar	Average disease severity**	Grouping category (Scott & Knott 1974)
BRS 229*	0.9163	a
ONIX	0.89	a
BRS 249	0.89	a
BR 18	0.743	b
IPR 118	0.6868	b
CD 114	0.612	b
CD 108	0.612	b
BRS 248	0.4073	c
BRS 220	0.4073	c
BRS 193	0.2367	d

*This cultivar always showed good level of field resistance. **Average of two replications of six plants each; severity scale between 0-1 (Zadoks, 1972).

TABLE 2 - Pathogenic variation between the *Pyricularia grisea* isolates from black oats *(Avena strigosa)* on some wheat and oat cultivars

Isolates	*P. grisea* severity seven days after inoculation*			
	Wheat cultivar		Oat cultivar	
	CD 103	BR 18	Black oat IAPAR 61	White oat IPR 126
15700	0.08	0.08	0.75	0.08
15701	0.58	0	0.33	0
15714	0.33	0	0.5	0
15715	0.83	0	0.33	0
15718	0.58	0	0.33	0.08
15720	0.58	0	1	0
15721	0.5	0	1	0.42
15722	0.67	0.08	0.92	0
15723	0.08	0	0.58	0
15724	0.33	0.08	1	0.25
15725	0.58	0	0.92	0
15726	0.17	0.42	1	0.67
15728	0.25	0.25	0.42	0.08
15730	0.58	0.58	1	0.25
15732	0.92	0	1	0.17
15741	0	0	0.25	0.17
15742	0.75	0.25	0.83	0.08
15749	0.75	0.58	0.75	0
15756	0.58	0	1	0.58

*Average of two replications of six plants each. See text for disease severity scale 0-1 (Zadoks, 1972).

TABLE 3 - Reaction of white oat and some other cereal cultivars to *Pyricularia grisea* under glasshouse conditions

Genotype	Reaction (severity of infection)*	Cross/ Pedigree	CV %
IPR Suprema (WO)	0.00 a	Unknown	0.0
BRS 210 (Wheat)	0.00 a	CPAC 89118/3/BR 23//CEP 19/PF 85490	0.0
Milan CD 116 (Wheat)	0.00 a	MILAN/MUNIA	0.0
BRS 229 (Wheat)	0.00 a	EMB 27*3//BR 35/B.PONCHO	0.0
Milan 3/ Atila/ Cimmyt3 (Wheat)	0.00 a	MILAN /3/ATILA//FANG 69/CIMMYT 3	0.0
IPR 89 (Rye)	0.00 a	POPULAÇÃO (CENTEIOS AUSTRALIANOS/ CENTEIOS SUL-PARANAENSES)	0.0
IPR Esmeralda (WO)	0.04 a	Unknown	31.3
URS Estampa (WO)	0.04 a	UFRGS 995088-3 / UFRGS 006049	31.3
AL 0926 (WO)	0.08 a	UFRGS 940566-3/IAC 7	33.4
AL 0925 (WO)	0.11 b	UFRGS 940566-3/IAC 7	32.0
AL 0959 (WO)	0.12 b	ER 94152-4-1/UFRGS 93605	36.7
IPR Afrodite (WO)	0.16 b	CFT 2 x ER 88144-1	26.1
FAEM 5 - CHIARASUL (WO)	0.16 b	UFRGS 17 / UFRGS 10 // 90 SAT -28	26.1
UPFA Gaudéria (WO)	0.16 b	UPF 16 /CTC 5	38.5
AL 0924 (WO)	0.18 b	UFRGS 940566-3/IAC 7	29.6
UPF 18 (WO)	0.19 b	UPF 85S0238 x UPF 12	21.6
IPR 126 (WO)	0.21 c	Unknown	24.9
FAEM 4 - CARLASUL (WO)	0.25 c	UFRGS 10 / 90 SAT -285	0.0
URS 21 (WO)	0.25 c	UFRGS 10 x CTC 84B993	0.0
URS Corona (WO)	0.25 c	UFRGS 987016-1 / UFRGS 970497-1	0.0
UPFA 22 - Temprana (WO)	0.27 c	90SAT-37 (UFRGS10)/ 90SAT-28	9.5
URS Brava (WO)	0.27 c	UFRGS 995078-2 / URS 21	9.5
URS Guria (WO)	0.32 d	UFRGS 987015-2 / UFRGS 960195-2	13.7
FAPA 4 - Louise (WO)	0.39 d	UFRGS881920 x UFRGS 7	17.2
AL 0952 (WO)	0.39 d	ER 94152-4-1/UFRGS 93605	13.5
BRISASUL (WO)	0.41 d	OR 2 / UPF 18	16.2
FAEM 6 - DILMASUL (WO)	0.41 d	URFGS 10 / 90SAT-28	16.2
URS Taura (WO)	0.42 d	UFRGS 970216-2 / UFRGS 970461	12.3
SI 031AP09 (BO)	0.46 d	Unknown	19.5
CB 20007317 (Barley)	0.46 d	Unknown	16.5
BARBARASUL (WO)	0.50 d	CFT5/UPF 18	0.0
URS Torena (WO)	0.56 e	UFRGS 984111-4 / UFRGS 988109-1	15.5
CB 20004609 (Rye)	0.51 d	Unknown	16.5
URS/ FAPA Slava (WO)	0.59 e	UFRGS 984111-4 / UFRGS 988109-1	13.4
UPFPS Farroupilha (WO)	0.61 e	UPF 18 X OR2	13.4
IAPAR 61 (BO)	0.62 e	Unknown	9.2
URS Charrua (WO)	0.70 f	UFRGS 984126-1 / UFRGS 984109-7	7.0
URS Guará (WO)	0.72 f	UFRGS 987016-1 / UFRGS 970497-1	5.1
UPFA Ouro (WO)	0.75 f	UPF 16 /UFP 18	0.0
IAC 7 (WO)	0.75 f	V155,ML-IICV-77-CV78-R78-79CV-79-CV80 (México)	0.0
URS Guapa (WO)	0.77 f	UPF 17 RES.//GUAIBA SEL./CTC 84B993	4.3
URS Tarimba (WO)	0.82 f	UFRGS 987016-1 / UFRGS 19	9.4

BO= Black Oat; WO= White Oat; *Average of two replications of 10 plants each. Reaction was assessed using a visual severity scale of 0-1 (Zadoks, 1972). Genotypes were grouped in different categories of infection by Scott & Knott (1974) analysis.

virulent pathotypes of the fungus (Ralph et al., 2012). Simultaneously, transferring the resistance of some wheat genotypes like Milan CD 116, Milan3/Atila/Cimmyt 3, BRS 210 and BRS 229 to potential wheat cultivars against the *P. grisea* isolates from variable origin like wheat, triticale, white oats and foxtail millet should receive top priority. Although horizontal resistance is much preferred, so far no efforts have been made using this breeding approach. Transferring resistance from genotype Milan by conventional means has resulted in some success (Table 3). Integrated management of the disease especially by combining less susceptible cultivars, and avoidance of early seeding, have been successfully applied to assure wheat production in the endemic region (Mehta et al., 1992). Use of resistant white oat cultivars and avoidance of susceptible black oat cultivars in crop rotation would help managing the disease and reduce the inoculum of the pathogen in the air.

In recent years, emphasis is also being given to the disease forecasting computer models. Through such models

it is possible to provide estimates of disease likelihood and forecast outbreaks which in turn may improve the efficiency of fungicidal applications. Computer modeling thus in near future may become a part of the integrated disease management systems (Fernandes et al., 2005).

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