### Pyricularia Blast - a Threat to Wheat Cultivation

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Abstract: Wheat blast disease caused by Pyricularia grisea (telemorph Magnaporthe grisea) has become a serious restriction on increasing the area and production of the crop, especially in the tropical parts of the Southern Cone Region of South America. First identified in 1985 in the State of Paraná in Brazil, it has become an endemic disease in the low lying Santa Cruz region of Bolivia, south and south-eastern Paraguay, and central and southern Brazil in recent years. Severe infections have also been observed in the summer planted wheat crop in north-eastern Argentina. So far, only sporadic infections have been seen in Uruguay, especially during the wet and warm years. Spike infection (often confused with Fusarium head blight infection) is the most notable symptom of the disease and capable of causing over 40% production losses. However, under severe infection, the loss of production can be almost complete in susceptible varieties. Wheat blast is mainly a spike disease but can also produce lesions on all the above ground parts of the plant under certain conditions. Depending upon the point of the infection on the rachis, the disease can kill the spike partially or fully. The infected portion of the spike dries out without producing any grain which can be visibly distinguished from the healthy portion. While virulence diversity in the fungus has been reported in the literature and is under further exploration, genetic resistance in the host species has been more difficult to identify. Earlier, Brazilian cultivars such as BH 1146, CNT 8, several IAC and OCEPAR selections were credited as demonstrating different levels of field resistance, but this was not confirmed under artificial inoculation studies. However, other cultivars such as BR18, IPR 85, CD 113, have shown moderate levels of resistance over the years in many locations. Recently, several cultivars and advanced lines derived from the CIMMYT line, Milan, have been observed to carry a high level of resistance to blast disease throughout the endemic region. However, to date, the genetic basis of this resistance is not very clear due to extreme variation in the pathogen. Cultivars showing complete resistance against a few isolates under controlled conditions in the glasshouse, may or may not show field resistance in commercial cultivation. Due to an increase of the area under Milan based resistant wheat cultivars in Bolivia, Brazil and Paraguay, it needs to be combined with other sources of resistance urgently to prevent the selection of a virulent pathotype in the fungus. Besides genetic resistance, avoidance of early dates of seeding and chemical control can reduce the disease severity. Fungicides combining triazols with strobilurins can, under some situations, be effective in disease control at the heading stage. Even when all components of integrated disease management of wheat blast are not in place yet, it is seen as an essential strategy to reduce production losses in this region. Given the threat that the blast disease may pose to world wheat growing areas in the future, more research efforts are deemed urgent and necessary.

Keywords: blast; disease; non traditional regions; Pyricularia; Triticum; wheat

Pyricularia blast disease, first identified in 1985 in the State of Parana, Brazil (IGARASHI *et al.* 1986) has become a serious threat to the cultivation

and spread of wheat production in tropical and subtropical areas of the Southern Cone region of South America. Spike infection caused by blast can be easily confused with Fusarium head blight infection. Rather than attacking individual spikelets, wheat blast attacks the rachis. The portion of the spike above the point of infection becomes bleached and no grains are formed, whereas the portion below remains healthy and produces normal grains (Figure 1).

#### Geographical distribution

At present wheat blast disease is restricted to the tropical regions of South America (north-eastern Argentina, lowlands of Bolivia, central and south-central Brazil and Paraguay). However, climatic changes associated with the global warming could trigger its spread to other parts of the world, where wheat and rice crops are grown together. Any eventual move of the disease to the Asian continent is likely to cause devastating effects unless disease control research programs are put in place.

#### Disease spread and production losses

The production losses caused by *Pyricularia* blast can vary from very low to almost 100%. The disease can attack all above ground parts of the plant, but severe spike infection can be observed with very little infection on the leaves or culms. Highest losses occur when the fungus attacks the rachis at the base of the spike thereby limiting the development of the grains and killing the spike completely.



Figure 1. Typical symptoms of wheat blast infection on the spikes

In 1987, the weather was dominated by the El Niño climatic phenomenon, and yield losses incurred by the three Brazilian wheat producing states (Parana, Matto Grosso do Sul and Sao Paulo) varied between 10.5 and 53% (Goulart & Paiva 1992). The same year, the disease also appeared in the traditional wheat region of Rio Grande dol Sul causing variable losses (Picinini & Fernandes 1990).

While Cunfer et al. (1993) observed the disease in the border region of Brazil/Paraguay in 1987, the first epidemic in Paraguay occurred in 2002, causing production losses of more than 70% in the early seeded fields (Viedma & Morel 2002). Most of the harvested grain did not meet commercial standards for test weight, and had to be used for animal consumption.

The first severe infections of wheat blast in Bolivia were observed in the lowland Santa Cruz region in 1996 and resulted in the loss of almost 80% of the production (Barea & Toledo 1996). In 1997 (another Niño year), the disease was devastating in the early seeded fields, causing 100% loss, and responsible for the severe decline in the wheat area in subsequent years.

Argentina reported its first blast infections and associated losses in a summer seeded wheat experimental crop in the north-eastern state of Chaco in 2007/08 (Alberione *et al.* 2008).

#### Disease causal organism

Pyricularia grisea (Cooke) Sacc. [telemorph Magnaporthe grisea (Herbert) Barr]

## Climatic conditions favourable to disease development

Although exact weather conditions required for a field epidemic are not clear, most severe blast years have coincided with wet years (a prevalence of the El Niño phenomenon). These are characterized by several days of continuous rains and average temperatures between 18–25°C during the flowering stage of the crop followed by sunny, hot and humid days. Under controlled growth chamber conditions, Cardoso *et al.* (2008) observed highest blast intensity at 30°C which increased with the duration of the wetting period, and lowest at 25°C with a wetting period of less

than 10 h. However, at 25°C and 40 h of wetting, blast intensity exceeded 85%.

Seed and secondary hosts as source of primary inoculum

Seed transmission of the wheat fungus has been shown by GOULART and PAIVA (1990). However, seed infection seems to play only a minor role in the epidemiology of the disease because spike infection comes from the air-borne conidia mainly from several secondary hosts (Ркавни et al. 1992; Urashima et al. 1993). Several grass weeds (Cenchrus echinatus, Eleusine indica, Digitaria sanguinalis, Brachiaria plantaginea, Echinocloa crusgalli, Pennisetum setosum, Hyparrhenia rufa and Rhynchelytrum roseum) occur commonly in wheat and rice fields of Brazil and are secondary hosts of Pyricularia, but their role in the epidemiology of wheat blast is not well understood (Prabhu et al. 1992). In Bolivia, Eleusine indica, Digitaria sanguinalis and Rottboelia exaltata have been identified with blast symptoms (HURTADO & TOLEDO 2005). First infections on triticales were reported by MEHTA and BAIER (1998), and recently, blast infection in commercially grown black oats (Avena strigosa) has been added to this list (Мента *et al.* 2006).

Black oats and foxtail millet (*Setaria italica*) are widely used in the crop rotation system in the

Southern Cone region, and their susceptibility to wheat blast may well make them candidates for the primary source of infection.

#### Host pathogen interaction

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 and 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 to the wheat and the rice pathotypes. The leaf reaction of some wheat cultivars to a mixture of *P. grisea* isolates from black oats is presented in Table 1.

#### Sources of resistance to wheat blast

In the face of wide virulence diversity in the fungus, genetic resistance in wheat has been more difficult to identify, URASHIMA *et al.* (2004). While some Brazilian cultivars (BR 18, IPR 85 and CD 113) have shown moderate levels of resistance over the years, new Bolivian cultivars such as Paragua CIAT

Table 1. Differential response of wheat cultivars to infection caused by a mixture of five *P. grisea* isolates from black oats, under glasshouse conditions, IAPAR, Londrina, Brazil, 2006

| 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                    | С                                      |
| BRS 220  | 0.4073                    | С                                      |
| BRS 193  | 0.2367                    | d                                      |

<sup>\*</sup>Average of six plants per cultivar; severity scale between 0–1 (ZADOKS 1972); source: Nunes and Mehta (2006, unpublished data)

Table 2. Evaluation of wheat blast severity under field conditions, Quirusilla, Bolivia, 2003/2004

| Cutivar       | Average leaf infection<br>(FL + FL-1) | Spike infection (%) |             |  |
|---------------|---------------------------------------|---------------------|-------------|--|
|               |                                       | evaluation          |             | J., 41. 1:                             |
|               |                                       | February 12         | February 19 | area under the disease progress curve* |
| Azubi CIAT    | $0.96^{a}$                            | 15.1                | 90.2        | $368.4^{a}$                            |
| Surutu CIAT   | $0.85^{a}$                            | 5.1                 | 50.5        | 194.7 <sup>b</sup>                     |
| BR 18         | $0.27^{\rm b}$                        | 5.5                 | 23.8        | $102.7^{c}$                            |
| Parapeti CIAT | $0.08^{\rm c}$                        | 6.1                 | 15.2        | 74.5 <sup>d</sup>                      |
| Paragua CIAT  | $0.04^{\rm c}$                        | 0.9                 | 4           | 17.3 <sup>e</sup>                      |

<sup>\*</sup>Area under the disease progress curve based on two evaluations; source: CIAT 2004 (unpublished data)

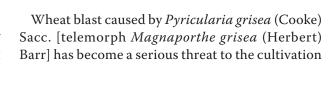
and Parapeti CIAT demonstrate a higher level of resistance (Table 2). Recently, several cultivars and advanced lines derived from the CIMMYT line, Milan, have been observed to carry a very high level of resistance to blast disease in the endemic region. While the genetic basis of resistance in Milan has yet to be studied, MEHTA *et al.* (2001) reported leaf resistance of the OR1cultivar to be based on a single recessive gene. The increased area under Milan-based resistant wheat cultivars such as Sausal CIAT, CD 116 and Caninde 1, released in Bolivia, Brazil and Paraguay, respectively, need to be combined with other sources urgently to prevent the selection of a virulent pathotype in the fungus.

# sulted in good control of the disease and hence are not cost-effective.

from the air-borne spores.

#### Integrated control measures

Besides genetic resistance, the avoidance of early dates of seeding and chemical control at heading



**CONCLUSIONS** 

stage can help reduce the disease severity (Mehta

et al. 1992). Fungicidal seed treatments help in

eliminating the seed-borne infection but do not

protect the plant from spike infection. This is

because the pathogen is not of a systemic nature,

and also because spike infection mainly comes

Fungicides combining triazols with strobilurins

have been used effectively at the heading stage to

control the disease, especially in the moderately

resistant varieties (Figure 2). However, fungicide

applications in susceptible varieties have not re-

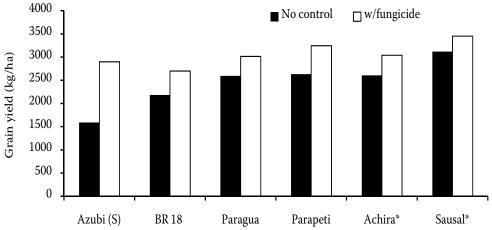


Figure 2. Chemical control of wheat blast disease, Bolivia, 2004–2006 (\*data 2007–2008)

and spread of wheat production in the tropical and subtropical regions of South America. Under favorable climatic conditions, wheat blast can be devastating and cause 100 percent losses to production. Besides wheat, the fungus can survive on triticale, barley, black oats, foxtail millet and many grassy weeds, in and around the crop. Given the extreme variability found in the fungus, it has been difficult to find good sources of resistance so far. Integrated management of the disease including some level of host resistance, avoidance of early seeding and chemical control, have been successfully applied to assure production in the endemic region. Given the threat blast disease may pose to world wheat production in the future, more research efforts are deemed urgent and necessary.

#### References

- Alberione E., Bainotti C., Cettour I., Salines J. (2008): Evaluación de enfermedades en trigos en siembra de verano en el NEA argentino-Campaña 2007/2008. In: 7 Congreso Nacional de Trigo. Santa Rosa, La Pampa.
- BAREA G., TOLEDO J. (1996): Identificación y zonificación de piricularia o bruzone (*Pirycylaria oryzae*) en el cultivo del trigo en el dpto. de Santa Cruz. In: CIAT. Informe Técnico. Proyecto de Investigación Trigo, Santa Cruz, 76–86.
- CARDOSO C.A. DE A., REIS E.M., MOEIRA E.N. (2008): Desenvolvimento de sistema de previsão de brusone causada por *Pyricularia grisea*. Summa Phytopathologica, **34**: 216–221.
- Cunfer B.M., Yorinori T., Igarashi S. (1993): Wheat Blast. In: Mathur S.B., Cunfer B.M. (eds): Seed Borne Diseases and Seed Health Testing of Wheat. Danish Government Institute of Seed Pathology for Developing Countries. Copenhagen.
- GOULART A.C.P., PAIVA F. DE A. (1990): Transmissão de *Pyricularia oryzae* através de sementes de trigo (*Triticum aestivum*). Fitopatología Brasileira, **15**: 359–362.
- GOULART A.C.P., PAIVA F. DE A. (1992): Wheat yield losses due to *Pyricularia oryzae* in the 1988–91 periods in Mato Grosso do Sul (Abstr.). Fitopatología Brasileira, **17**: 171.

- Hurtado J., Toledo J. (2005): Malezas hospederas de Piricularia, *Pyricularia grisea*. Invierno 2004. Informe CIAT, 2005.
- IGARASHI S., UTIAMADA C.M., KASUMA A.H., LÓPEZ R.S. (1986): *Pyricularia* sp em trigo. 1. Ocurrencia de *Pyricularia* sp no Estado do Paraná. Fitopatología Brasilera, **11**: 351–352.
- Mента Y.R., Ваієк А. (1998): Variação patogênica entre isolados de *Magnaporthe grisea* atacando triticale e trigo no estado do Paraná. Summa Phytopathologica, **24**: 119–125.
- MEHTA Y.R., ARIAS C.A.A., TOLEDO J.F.F. (2001): Inheritance of resistance to *Magnaporthe grisea* in wheat. Summa Phytopathologica, **2**: 300–304.
- Mehta Y.R., Riedi C.R., Campos L.A.C., Kohli M.M. (1992): Integrated management of major wheat diseases in Brazil: an example for the S. Cone Region of Latin America. Crop Protection, 11: 517–524.
- Mehta Y.R., Nunes M.P., Oliveira J.C. (2006): Occurrência de brusone em aveia no Estado do Paraná. In: Resultados Experimentais. XXVI Reunião da Comissão Brasileira de Pesquisa de Aveia, 4–6 de abril, FAPA, Guarapuava, Paraná, 55–57.
- PICININI E.C., FERNANDES J.M.C. (1990): Ocorrência da brusone (*Pyricularia oryzae*) em lavouras comerciais de trigo (*Triticum aestivum*) no estado do Rio Grande do Sul. Fitopatología Brasilera, **15**: 83–84.
- Prabhu A.S., Filippi M.C., Castro N. (1992): Pathogenic variation among isolates of *Pyricularia grisea* infecting rice, wheat, and grasses in Brazil. Tropical Pest Management, **38**: 367–371.
- SCOTT A.J., KNOTT M. (1974): Cluster analysis method for grouping means in the analysis of variance. Biometrics, **30**: 507–512.
- URASHIMA A.S., IGARASHI S., KATO H. (1993): Host range, mating type, and fertility of *Pyricularia grisea* from wheat in Brazil. Plant Disease, **12**: 11–16.
- URASHIMA A.S., LAVORENT N.A., GOULART C.P., MEHTA Y.R. (2004): Resistance spectra of wheat cultivars and virulence diversity of *Magnaporthe grisea* isolates in Brazil. Fitopathologia Brasileira, **29**: 511–518.
- VIEDMA L.Q., MOREL W. (2002): Añublo o Piricularia del Trigo. Díptico. MAG/DIA/CRIA. Programa de Investigación de Trigo, CRIA, Capitán Miranda, Itapúa.
- ZADOKS J.C. (1972): Methodology of epidemiological research. Annual Review of Phytopathology, **10**: 253–276.