

Review

Wheat blast research: Status and imperatives

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Wheat blast is relatively a new disease of wheat that first appeared in Brazil in 1985. It did spread to some other neighbouring countries in the following years and owing to its predisposing factors, was feared to be capable of moving across continental boundaries. The disease has now been reported from Bangladesh. Wheat blast at best can be described as poorly understood as both pathogen and its pathogenicity as well as host and its ability to resist need to be investigated before breeders could confidently field varieties with sufficient levels of genetic resistance. In the meantime, chemical protectants and management strategies need to be worked out to tackle this menace that has already been important in rice.

Key words: Wheat, blast, *Magnaporthe*, disease, research.

INTRODUCTION

Literally speaking, blast means explosion. Wheat blast directly strikes wheat ear and renders grains shrunken, shrivelled and deformed within a week of initial symptoms giving no time to farmers to react. Climatic conditions viz., hot and humid climate play a crucial role in disease development. The blast pathogen shows various infection abilities and is known to infect many grasses like rice, wheat, barley, etc. In fact, rice blast has been one of the most important and damaging rice diseases, whereas wheat blast is of relatively recent occurrence (Maciel, 2016). First sighted in 1985 in Brazil, it soon spread to other iso-climatic neighbouring countries of South America. It is now a serious production constraint in the tropics and sub tropic regions, including Brazil, Argentina, Bolivia and Paraguay causing yield losses of up to 100% (Peng et al., 2011). Most current wheat varieties are blast susceptible, pathogen is highly variable, epidemiology as

well as genetics of resistance is poorly understood. All this makes wheat blast a formidable wheat enemy. Since wheat blast requires concurrent heat and humidity to develop, experts had earlier warned about a possible movement of blast from Latin America to similar regions of Africa and Asia. The detection of blast in early 2016 in Bangladesh (Callaway, 2016) confirmed the fear. The blast in Bangladesh was most likely caused by a wheat-infecting South American lineage of the blast pathogen, *Magnaporthe oryzae* (Islam et al., 2016). If blast fungus continues to show similar migratory capacity, it could soon spread to other hot and humid wheat growing regions in South Asia and beyond. The situation perhaps is even more demanding as fungicides at best offer only a partial defence (CIMMYT, 2016). A spread of wheat blast in South Asia could jeopardize food security of 300 million inhabitants of this region as they consume over

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100 million tonnes of wheat each year. It is already reported that blast affected 16000 hectares of wheat crop in Bangladesh and consequent poor harvest led to Bangladesh importing extra 400,000 tonnes of wheat as compared to previous year (New Age, 2016). This article attempts to review the available information on wheat blast research and also identify gaps to be addressed.

PATHOGEN, DISEASE DEVELOPMENT AND HOST VARIATION

Literature now accepts *M. oryzae* pathotype *Triticum* as the correct name for wheat blast pathogen (Maciel, 2016; Castroagudin et al., 2015; Perello et al., 2015; Maciel et al., 2014) although recently, a new species named *Pyricularia graminis-tritici* was proposed to cause wheat blast by Castroagudin et al. (2016). Ever since its first report, blast pathogen was variously named by researchers for example, *Pyricularia oryzae* (Araujo et al., 2016; Oliveira et al., 2015; Cruz et al., 2015a; Silva et al., 2015), *Pyricularia grisea* (Filha et al., 2011; Kohli et al., 2011; Rocha et al., 2014), *Magnaporthe grisea* (Urashima and Kato, 1994; Peng et al., 2011; Pagani et al., 2014) and *M. oryzae Triticum* (Cruz et al., 2015a). Blast pathogen has shown capability to evolve fast to adapt to new climates. Peng et al. (2011) reported that isolates of blast pathogen from different species displayed differential infection abilities and host parasite specificity between wheat cultivars and pathogen isolates was observed. Triticale (*Secale X triticum*) and barley (*Hordeum vulgare*) have also been reported to be infected by *M. grisea* (Urashimae et al., 2004). Cross infectivity studies among hosts revealed that blast pathogen from triticale and barley could infect triticale, barley, wheat, oat and rye but not rice, sorghum, maize, common millet, sugarcane and *Brachiaria brizantha* (Urishama et al., 2004). Ever since its detection in 1985, blast had been observed only on wheat and black oats in Brazil. The disease did spread to some other *Gramineae* species but white oat remained resistant till 2012 when cultivar IAC 7 was severely attacked in Sao Paulo (Marangoni et al., 2013). It is worth mentioning that first detection of blast in US in 2011 was reported to be a case of host jumping by blast pathogen (Tosa et al., 2016). Urashima and Kato (1994) screened 43 wheat lines from Brazil, Japan, USA, Bulgaria, seven *Triticum* spp., and 18 *Aegilops* lines against *M. grisea* inoculation under greenhouse conditions. They found only two *Aegilops* accessions resistant and all others screened were susceptible. Attempting to differentiate between young and adult stage resistance, Cruz et al. (2010) challenged 70 wheat genotypes in young stage to 18 isolates of blast pathogen. They found BRS229, BRS179, CNT8, BRS120 and BRS Buriti with better resistance levels. 12 of the 70 genotypes were inoculated at adult stage and they found CNT8, NE 20156-B, PF 844001, PF

964009 and PF 804002 having less leaf and head area affected by blast. Blast pathogen has also evolved to acquire resistance to fungicides extensively used to manage the disease. Oliveira et al. (2015) compared resistance presented by two groups of *Pyricularia oryzae* isolates from wheat to two fungicides viz., azoxystrobin and pyraclostrobin, both of which are quinone oxidoreductase inhibitors or QoI fungicides. They concluded that high level of resistance to QoI fungicides may be the result of high selection pressure exerted by consecutive years of strobilurin application for the management of wheat diseases in Brazil.

Blast is still spreading in South America and now covers large geographic regions. Maciel et al. (2014) found no subdivision among isolates collected from wheat fields of central-western, southwestern and southern Brazil, indicating high level of gene flow across a large geographic expanse. They proposed that populations of wheat blast pathogen exhibited a mixed reproductive system in which sexual reproduction is followed by local dispersal of clones. Based on seedling virulence assays with local wheat cultivars, they reported 14 pathotypes in the current population; however, detached head virulence assays differentiated only eight virulence groups on the same set of wheat cultivars, and there was no correlation between seedling and head reactions.

Epidemiology, distribution and quantification

First detected in Brazil in 1985, wheat blast soon spread to other neighbouring countries like Bolivia, Paraguay (Kohli et al., 2011) and was detected in Argentina in 2012 (Perello et al., 2015). The blast causing fungal pathogen *M. oryzae* can spread through seed and can also survive on crop residues. The blast mainly affects grains; however, leaf lesions are also observed. The leaf lesions and/or sporulation on leaves does not precede spike blast and therefore importance of inoculum originating from leaves in severely affected fields is disputed, even though it was observed that conidia production coincided with spike emergence under both green house and field conditions (Cruz et al., 2015b). However, Goulart et al. (1995) reported that infected rachis (black point infection in rachis) did pass on pathogen to harvested seed. They found BH 1146 with least infection index and consequently no BH 1146 seed carried infection. On the contrary, variety Anahuac had highest infection index (99.5%) and 26.7% of its seeds carried wheat blast pathogen. They reported a significant positive correlation between field incidence of wheat blast and percentage of seed with wheat blast pathogen across varieties. Cytological investigations revealed that in case of compatible reactions (host resistance) viz., rice blast pathogen on rice (r_b_p_r) or wheat blast pathogen on wheat (w_b_p_w) fungal hyphae penetrated and colonized

the epidermal cells and also invaded many neighbouring cells. On the other hand, in the case of incompatible reactions (non-host resistance) of the type *w_b_p_r* and *r_b_p_w* fungal hyphae were not able to neither penetrate nor colonize the epidermal cells. Interestingly, in the case of non-host resistance if penetration did occur, the hyphae remained restricted to the first invaded epidermal cell (Araujo et al., 2016). Additionally, unsuccessful penetration occurred with high frequency in incompatible interactions as compared to compatible ones. Correct and uniform scoring of disease symptoms is a critical prerequisite to comparative studies, understanding and reporting. Visual scoring like that for glume blotch and fusarium head blight can be an efficient and useful tool. Maciel et al. (2013) employed a software 'ImageJ' to propose a diagrammatic scale to record varying severity of the disease symptoms on wheat spikes. Similarly, Rios et al. (2013) developed a standard area diagram sets (SADs) to quantify wheat blast severity on wheat leaves. Severity estimates were more reliable even with inexperienced scorers when SADs were employed.

Resistance mechanism

Interaction studies of 27 wheat cultivars with two ear pathogens viz., *Magnaporthe* wheat blast (WB) and *Fusarium* head blight (FHB) revealed that most of the 27 cultivars displayed inverse disease response to two diseases. The cultivar 'Milan' displayed resistance (R) to blast and susceptible (S) reaction to FHB. The reactions were reversed when cultivar 'Sumai 3' was inoculated with these two pathogens. Microscopic studies revealed that MWB similarly colonized spikelets in both the cultivars and FHB infected anthers of the susceptible cultivar earlier. Interestingly, both the pathogens grew much faster in the rachilla of the susceptible cultivars indicating that resistance mainly expressed in this part connecting spikelet with rachis (Ha et al., 2016). Gene expression patterns confirmed differential disease phenotypes, fungal spread in the rachis and colonization patterns. The differential response of resistant and susceptible cultivars rules out availability of common resistance genes at least in the material investigated. Cellular investigations revealed that resistance to non-adapted *Magnaporthe* isolates was due to formation of appositions beneath pathogen penetration sites that adapted virulent isolates were able to breach (Tufan et al., 2009). They also reported differential transcription post infection between adapted and non-adapted isolates. Five major genes for wheat blast resistance viz., *Rmg1*, *Rmg2*, *Rmg3*, *Rmg4* and *Rmg5* have been reported (Peng et al., 2011). It is interesting however, to note that wheat leaf rust resistance gene *Lr34* confers resistance to blast in rice (Krattinger et al., 2016). A word of caution on resistance that even complete resistance

may break down due to nitrogen induced susceptibility (NIS). Ballini et al. (2013) reported that NIS is a general phenomenon affecting resistance to blast fungus in both wheat and rice.

BLAST MANAGEMENT

Peng et al. (2011) reported absence of effective method for control of wheat blast and emphasized that efforts should be focused to prevent pathogen dispersal to protect wheat production, a warning that came true when blast was detected in Bangladesh (Callaway, 2016). Blast pathogen has also evolved to acquire resistance to fungicides extensively used to manage the disease (Oliveira et al., 2015). Similar findings of widespread distribution of Qol (group of fungicides used for controlling blast) resistance in *M. oryzae* populations sampled from wheat fields and Poaceous hosts across central and southern Brazil were reported by Castroagudin et al. (2015). This resistance is a result of mutation of G143A which led to evolution of cytochrome b gene. Since strobilurins are widely used to manage wheat blast in Brazil, there has been a surge in frequency of the G143A mutation in the wheat infecting population of *M. oryzae* from 36% in 2005 to 90% in 2012 (Castroagudin et al., 2015).

Pagani et al. (2014) advocated integrating several options for efficient management of wheat blast. In a two year study, they found that phosphite treated plots increased yield by 9 to 80%, silicon (Si) treatment by 26 to 92% and synthetic fungicides by 90 to 121%. Rocha et al. (2014) however, concluded that control of wheat blast by means of fungicides application was effective for flag leaves but not for ears. Positive contribution of Si in augmenting the resistance to blast was confirmed by Cruz et al. (2015a). They reported limited colonization of +Si plants by pathogen and that this was associated with the deposition of phenolic compounds. They also observed that expression of all defence related genes was significantly increased on infection but expression level was two to three times higher for +Si plants as compared to -Si counterparts. Similar results of increased Si concentration causing reduced fungal growth were reported by Silva et al. (2015). They found that at histochemical level, Si is involved in the potentiation of the biosynthetic pathway of flavonoids that increases wheat resistance to blast. Silicon application reduced area under blast progress curve by 31% in an experiment reported by Filha et al. (2011). Several substances like jasmonic acid (JA), deacetylated chitosan (DC), potassium silicate (PS), potassium phosphate (PP), tebuconazole (TE) etc. have been experimented to manage wheat blast (Cruz et al., 2011). They found that PP was the best treatment that most reduced severity in the three cultivars tested. TE and PS when added to the culture medium gave lowest values for mycelial growth.

They concluded that PP and TE increased the potentiation of wheat resistance to blast which was also dependent on the inherent level of resistance of the cultivar. According to Urashima and Kato (1994), probenazole and tricyclazole gave good control of blast, except at heading stage. They also reported that new products containing blacin and acetamide gave good protection of the wheat head. Some combinations of earlier reported fungicides viz., tricyclazole and tebuconazole were reported (Goulart and Paiva, 1993) to give best yield increase if followed by thiophanate-methyl+ mancozeb. However, Goulart et al. (1996) reported only mancozeb application to be economically viable. Even though the disease is new and is a subject of detailed investigations worldwide, it is a disease of serious consequences and therefore early warning or disease forecasting can be of great help for farmers and administrators. Development of wheat blast requires simultaneous occurrence of both temperature and spike-wetness. Cardoso et al. (2008) reported highest blast intensity at 30°C which increased with duration of wetting period, while the lowest severity was at 25°C with 10 h of spike wetness. Irrespective of temperature, a wetting period of less than 10 h caused no disease, whereas at 25°C and 40 h wetting period, intensity exceeded 85%. Authors developed a model that shows blast intensity as a function of temperature and spike wetness. The model has then been used to prepare tables to predict blast. Rios et al. (2016) recommended combining both genetic resistance and fungicide treatment for most effective blast management. With 70 and 90% control of final incidence and severity, they found that effect of resistance and fungicide was additive of incidence as well as severity control.

There have been reports on agronomic management (Oliveira et al., 2016) and biological control (Singh et al., 2012; Gnanamanickam and Mew, 1992) of rice blast having potential implications for integrated management of wheat blast. Sowing date significantly affected disease incidence and yield of 14 wheat varieties in Brazil (Oliveira et al., 2016). The strain F0142 of *Chaetomium globosum* isolated from barnyard grass showed potent disease control efficacy against *M. grisea* and also wheat leaf rust (Park et al., 2005). The methanol extract from stems of a tree of Chinese origin, *Catalpa ovata* exhibited potent antifungal activity against several fungal pathogens including *M. grisea* (Cho et al., 2006). The fungus, *Trichoderma harzianum* (Singh et al., 2012) and bacteria viz., *Pseudomonas* spp. and *Bacillus* spp. (Gnanamanickam and Mew, 1992) were also observed to control rice blast. The results hold promise and warrant further investigations to integrate agents of biological origin in a wheat blast management strategy.

CONCLUSION

Wheat blast is a poorly understood emerging threat with

potential to be of catastrophic magnitude. There is need to investigate all the parties involved viz., pathogen, host and predisposing factors that would enable stakeholders to manage the disease. Research imperatives on pathogen side include pathogen range, evolution, patterns of variation, effect of climatic factors, epidemiology, virulence patterns, etc. There is need to study variation in pathogen vis a vis, its geographical spread to deploy genetic resistance accordingly. Some research in South America has identified resistance sources; however, this needs to be undertaken in all wheat growing areas where blast favouring conditions prevail. Establishing distinctness of resistance and any relationship with growth stage and/or environmental factors need to be investigated. Since a commercial product needs to have all the features including yield, therefore resistance to blast has to be an integral feature of breeding programmes targeting regions with blast favouring climate. Last but not the least, predisposing factors, chemical protectants, agronomic manipulations and biological agents need to be studied so as to devise management strategies till usable genetic resistance is available in commercial cultivars.

Conflict of Interests

The authors have not declared any conflict of interests.

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