

Wheat Blast: A Fearsome Threat to Food Security

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Wheat blast caused by *Pyricularia graminis-tritici* is a devastating disease that was first reported in Brazil in 1985. The disease was restricted to South America until 2016, when a series of grain imports from Brazil led to a wheat blast outbreak in Bangladesh. There is the need to strengthen quarantine and biosafety regulations to avoid additional spread of the pathogen to disease-free countries along with collaborated breeding efforts to develop resistant wheat varieties.

Emergence and Spread of Wheat Blast

Wheat blast was not a widespread disease in the South American countries before the first epidemic in 1985, though earlier reports indicate the pathogen's presence in the Brazilian agroecosystem. After emerging in Brazil, the wheat blast pathogen spread to the neighbouring states. *Pyricularia graminis-tritici* (Pygt) is now firmly established in South America. Starting from its origin in the pathogen spread into warmer regions supposedly via wind, but seed borne inoculum is might also have facilitated the long-distance dispersal of the pathogen to Southeast Asia. Brazilian bread wheat and durum wheat seed have been repeatedly imported for sowing by nearly 65 countries during the past decade. In 2016, wheat blast emerged in a destructive form in Bangladesh. Weather suitable for wheat blast coupled with susceptible cultivars may prompt severe outbreaks of the disease in South-east Asia.

The Pathogen: *Pyricularia graminis-tritici*

Pygt is a highly diverse species with a broad host range. Similar to the rice blast pathogen (*Pyricularia oryzae*) a multiname species confusion exists for the wheat blast pathogen. Phylogenomic analyses including some housekeeping loci for several isolates of *Pyricularia* spp. sampled from populations of wheat, rice and other grasses growing in or near wheat fields led to the identification of the new species *P. graminis-tritici* as the blast pathogen on wheat and several other hosts. The new species clearly distinguished from the rice-infecting isolates. Pygt is capable of causing blast on many important crop species, including wheat, barley and oats as well as grasses (weeds) in commercial wheat fields (e.g., *Cynodon* spp., *Digitaria insularis*, *Equinochloa crusgalli*, *Panicum maximum*, *Sorghum sudanense*). The confusing nomenclature and a lack of knowledge of the most recent findings, was a plausible cause for the quarantine breach that led to the introduction of Pygt into Bangladesh.

Disease Epidemiology

Wild grass hosts in wheat fields, may function as a major reservoir of wheat blast inoculum as evident from extensive gene flow between Pygt populations sampled from wheat and other grasses. Thus, non-wheat hosts probably play an important role in the epidemiology of wheat blast. Studies on wheat leaf infection process have revealed that Pygt conidia after landing on the adaxial leaf surface begins to germinate six hours after inoculation (hai) forming a dome-shaped appressorium at 12 hai. The first leaf blast symptoms were observed at 48 hai. Airborne Pygt conidia produced on and released from infected wheat seedlings provide source of inoculum for spikes infection. Fungal hyphae grows both inter- and intracellularly in the rachis tissues breaching the cell wall and destroying the spike completely.

Symptoms and Physiological Changes on Wheat Plants During Infection

The wheat blast pathogen affects the physiology of wheat plants by changing metabolic processes such as carbon assimilation and stomatal function. Development of leaf blast symptoms decreases CO₂ assimilation and cause stomatal closure damaging the photosynthetic machinery. Wheat leaf infection by Pygt triggers a series of photochemical malfunctions that affect the ability of leaves to intercept light. The concentration of photosynthetic pigments also decreases due to intense leaf chlorosis. Biochemical changes in wheat grains resulted in 25–45% reductions in soluble sugars (e.g., fructose, glucose, and sucrose) and starch

content. Increase in the breakdown of starch in the infected grains substantially diminished the synthesis of endosperm, resulting in smaller and shrunken grains thus grain yield reduction.



Reddish brown to dark grey spots appears on mature leaves followed by eye-shaped lesions with light grey centres on the leaves. The inflorescence blight and necrosis are observed. Light dark brown lesions with grey centres develop on stalks of wheat spikes. Damaged spikes show discoloration. Blackening of the rachis, lower nodes along with discoloration and shrivelling of grains is also observed. Optimum weather conditions for the development of wheat blast include the simultaneous occurrence of high temperatures (25–30°C) and long and frequent leaf wetness periods (24-40 hrs.).

Management Strategies

The first major global concern is to prevent additional spread of the pathogen to disease-free countries. Since the pathogen can be spread long distances on infected seeds, it is mandatory to strengthen quarantine and biosafety regulations. In the endemic areas management strategies for wheat blast must be based on knowledge of Pygt biology and wheat blast epidemiology, including the pathogen's disease cycle, survival strategy, means of spread, host range and the most conducive weather conditions for disease development.

The adoption of IDM strategies should be coordinated. Pygt can survive as perithecia on crop residues between wheat-cropping seasons. Invasive grasses may also play an important role as sources of secondary inoculum. Therefore, sanitary practices should be adopted. Deep ploughing of crop stubble can be used for reduction in the amount of initial Pygt inoculum. The destruction of crop residues (especially the debris of barley, oats, millets, and wheat) and eradication of invasive grasses is recommended. Crop rotation with non-host crops, like soybean and jute is also a viable option.

Periods of high temperature, rain, and high relative humidity are the most conducive for blast incidence. Therefore, adjusting the sowing date to escape the occurrence of flowering or grain filling during the conducive period can effectively reduce disease incidence and grain yield loss. The wheat blast pathogen is seedborne and seeds are considered the primary sources for long-distance dispersal of Pygt. Pygt conidia can survive and remain infectious for almost two years on or inside seeds, and even healthy-looking seeds from infested fields might carry fungal spores. Therefore, the use of certified, pathogen-free or fungicide-treated wheat seeds should be mandatory. Seed treatment with benomyl and carbendazim is highly effective. The triazole difenoconazole is also recommended for managing Pygt.

Leaves and ears sprays of mixtures of triazole and quinone outside inhibitor (QoI) fungicides at early heading and early grain-filling stages were found moderately effective in disease control and reduced crop losses. Recent recommendations to manage the disease in Asia include spraying of triazoles combined with strobilurins (e.g., tebuconazole and trifloxystrobin). To decrease the risk of fungicide resistance in Pygt populations, only mixtures or coformulations with lowrisk fungicides (such as the dithiocarbamate mancozeb and chlorothalonil) should be deployed to manage wheat blast.

Biofortification with nonfungicidal chemicals like Potassium phosphate, potassium silicate and application of silicon to wheat leaves have been assessed and were found to limit Pygt colonization through the

flavonoid biosynthetic pathway and the deposition of phenolic compounds. biocontrol. Biological control agents have been shown as alternatives for managing wheat blast. For example, *Bacillus methylotrophicus*, *Trichoderma harzianum* and *Chaetomium globosum* have potential for biological control of blast on wheat.

References

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