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Review Paper

Occurrence of wheat blast in Bangladesh and its implications for South Asian wheat production

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Abstract

The first recorded occurrence in Asia of wheat blast caused by *Magnaporthe oryzae*, pathotype *Triticum* (synonym *Pyricularia oryzae*) occurred in Bangladesh in March 2016. Crop losses of up to 90% have been reported, with late-sown wheat suffering particularly badly. The emergence of this disease has raised concern in neighboring countries where wheat represents a significant crop, most notably in India and Nepal. The existence of effective genetic resistance is in doubt, so for the moment the sole means of control is via the application of fungicides and adoption of beneficial cultural practices. The disease has been endemic in parts of South America for the last 30 years, so only a coordinated program of research and development has the potential to deliver rapid progress in combating the disease. In addition to evaluating and deploying genetic resistance and applying fungicides on an occasional basis, some control could be made possible by altering current crop rotation practice and/or manipulating the sowing time to promote disease escape.

Key words: *Triticum aestivum, Magnaporthe oryzae* pathotype *Triticum,* resistance, Bangladesh, South Asia

Introduction

The announcement by the Government of Bangladesh on March 27, 2016 that wheat (*Triticum aestivum*) crop in the trans-Ganges region (southern and south-western Bangladesh) had been infected by blast caused by *Magnaporthe oryzae* pathotype *Triticum* represents the first report of this devastating disease in Asia (Malaker et al. 2016). The reported extent of yield loss ranged from 20 to 90% (Fig. 1). For many years the disease has been endemic to parts of Latin America, but has not, until now, moved out of this area. Its emergence in Bangladesh emphasizes the potential of this disease to spread unless effective control measures are put in place in a timely fashion.

What is wheat blast?

The causative pathogen of wheat blast is the ascomycete fungus *Magnaporthe oryzae* B.C. Couch and L.M. Kohn (synonym *Pyricularia oryzae*). The fungus reproduces sexually (Maciel et al. 2014). The pathogen consists of a number of host-specific pathotypes, including forms able to colonize only one of rice, wheat, ryegrass, finger millet, foxtail millet and several other grass species (Farman 2002; Kato et al. 2000; Tosa et al. 2004; Khanna et al. 2015). Most isolates have only a limited ability to infect an alternative host (Mehta and Baier 1998; Urashima and Kato 1998). *Triticum* pathotypes are thought to be closely related to those which attack rice, foxtail millet or finger millet (Urashima et al. 1993; Kato et al. 2000; Murakami et al. 2000; Tosa et al. 2006). Nevertheless, isolates attacking wheat can be distinguished from other subgroups based on host range (Urashima et al. 1993) and DNA fingerprint (Urashima et al. 1999, 2005).

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Rice pathotypes cannot, by and large, recognize wheat as a host, or *vice versa* (Prabhu et al. 1992; Tosa et al. 2004), but wheat pathotypes can infect both triticale (*X Triticosecale*) and barley (*Hordeum sativum*) (Urashima et al. 2004). There is a growing belief that the wheat pathotype evolved from fungal populations colonizing a tropical grass species in South America (Duveiller et al. 2016).

Occurrence of wheat blast

The disease was first recorded in 1985 in the Brazilian state of Paraná (Igarashi et al. 1986), from where it has gradually spread across South America, remaining however, until now, confined to that continent (Fig. 2). Up to 3 mha of wheat crops were infected in the 90's, and the disease is recognized as being a significant production constraint in Brazil, Bolivia and Paraguay. A particularly severe outbreak occurred in Brazil in 2009. In Argentina, the disease was first detected in 2007 in a limited area of the country's northern region bordering Brazil (Cabrera and Gutierrez, 2007), but has been detected in one experimental plot in Buenos Aires in 2012 underlining the potential threat in other wheat production areas (Perelló et al. 2015). Currently the most strongly affected regions include central and southern Brazil, the lowland Santa Cruz region of Bolivia, southern and south-eastern Paraguay and north-eastern Argentina (Kohli et al. 2011). Yield losses incurred as a result of infection have been estimated to range from 10-100% (Goulart et al. 2007; Goulart and Paiva 2000; Urashima et al. 2009; Duveiller et al. 2011).

What does the disease look like?

Disease symptoms form mainly on the spike, which becomes partially or completely bleached (Fig. 3). Above the point of infection on the rachis, which is signaled by the formation of black spots (Igarashi 1990), the spikelets appeared bleached. Under heavy inoculum pressure, disease symptoms can also develop on the leaf (Igarashi 1990; Urashima et al. 2009; Maciel 2008, 2014), in the form of gray-green, water-soaked lesions with dark green borders; as the lesions expand, their color changes to a light tan, and their borders become necrotic (Xavier-Filha et al. 2011). The grains set in a blast-infected spike are typically shrunken and deformed; severely infected plants are sterile above the point of infection on the rachis. The symptoms can be confused with those induced by an infection of Fusarium head blight mainly caused by *F. graminearum*; however, the latter disease is readily distinguished by the salmon/pink coloration affecting the glumes.

Why wheat blast is considered dangerous?

Wheat blast is a particularly dangerous disease as it develops very rapidly, leaving the farmer no time to take preventative measures. The most vulnerable growth stage in terms of yield reduction is between anthesis and early grain development (Igarashi 1990). Some other features which contribute to the level of danger of the disease are that: (i) the pathogen survives on a number of alternate grass species, so crop rotation may represent an ineffective prophylactic measure, (ii) chemical control is unreliable, because, given the rapidity of the infection progress, there is typically too little warning time available, (iii) little research investment has yet been made to aid the discovery and genetic characterization of effective sources of resistance, (iv) breeding for resistance is difficult given the sporadic occurrence of the disease, and (v) most current wheat varieties are susceptible (www.cimmyt.org).

Environmental conditions favoring disease development

The most severe field infections occur in seasons when the period around anthesis features continuous rainfall and an average temperature of 18-25^{o}C, followed by a period of sunny, hot and humid weather (Kohli et al. 2011). Climate-controlled experiments have confirmed that conidia form most readily under conditions of high humidity (>90%) and a temperature of around 28^{o}C (Alves and Fernandes 2006). According to Cardoso et al. (2008), the rate of infection is highest when plants remain wet for a period of at least ten hours during which the temperature is maintained in the range 25-30^{o}C, while Ha et al. (2012) claim that a longer wetting period (24 h) and a similar temperature range (26-32^{o}C) are optimal for infection. The 2016 outbreak in Bangladesh occurred following a particular rainy February (Fig. 4). Disease severity is enhanced when excessive nitrogen fertilizer is given to the crop, whether the host be perennial ryegrass (Williams et al. 2001), rice (Skamnioti and Gurr 2009) or wheat (Ballini et al. 2013). Late-sown wheat crops appeared more prone to be infected than conventionally timed-sown ones in the Bangladesh epidemic (P. Malaker, personal communication) although this may not compare with observations in South America where early sowing has to be avoided to esacape rainfall (Mehta et al. 1992).

Epidemiology

The factors which govern the wide-ranging, almost synchronous development of bleached wheat spikes in a large commercial crop are unknown (Cruz et al. 2015). The most important source of initial inoculum is considered by some to be infected grain (Maciel et al. 2014), but others discount this on the grounds that the disease is initiated by an infection of the spike, presumably from air-borne conidia (Prabhu et al. 1992; Urashima et al. 1993; Urashima et al. 2007). The infection observed over a large area of wheat plants in Paraná in 2009 implies a massive infection by airborne inoculum coinciding with the host plants having reached the most susceptible phenological stage. According to our observation, the blast inoculum may have blown into southern Bangladesh (Fig. 1) during the crop season (Fig. 5). However, an initial source of inoculum prior to heading in those hot spots may not be ruled out.Unlike rice blast, the disease in wheat tends to affect older rather than younger leaves (Ghatak et al. 2013; Cruz et al. 2015). Under optimum infection conditions, a single gram of dry basal diseased leaf taken from a highly susceptible cultivar can release >1.5 x 10^6 conidia (Cruz et al. 2015). Under laboratory conditions, exposing a spike of a susceptible cultivar to a population of 24,000 spores generates a severe blast infection (Cruz et al. 2012). The lower canopy of the wheat crop is believed to contribute massively to disease development through the release of inoculum (Cruz et al. 2015). The sexual stage of the fungus has yet to be found in nature, but some isolates can be induced to undergo sexual reproduction under experimental conditions (Bruno and Urashima 2001; Urashima et al. 1993; Tosa et al. 2004). As ascospores are generated without difficulty, it has been suggested that under natural conditions, sexual spores are likely to play a significant role in the pathogen's diversification (Urashima et al. 1993). Indeed, Maciel et al. (2014) have proposed that the production of ascospores is the driver of genetic diversity in wheat isolates. Several grass species have been suggested to serve as alternative hosts for wheat blast (Prabhu et al. 1992; Urashima and Kato 1998; Kohli et al. 2011). These could include other cereal crops, such as barley, rye, triticale, pearl millet, oats, maize and sorghum, which are often planted as part of the local rotation system (Urashima et al. 2005).

Genetic resistance: Sources and its mode of inheritance

The level of resistance to wheat blast is generally low. A number of commercial cultivars have been proposed as potential sources of resistance/tolerance, but mostly these have been proven to be unreliable or effective only in limited geographies (Urashima and Kato 1994; Kohli et al. 2011). Since the pathogen is actively evolving, lines classified as resistant may have become susceptible over time – for example, two cultivars (Overland and RonL) which expressed quite high levels of resistance against isolates collected some years ago, were completely susceptible to infection when challenged with current isolates (Cruz et al. 2016). Not a single member of a collection of lines selected as resistant proved to resistant against each of the 27 blast isolates tested by Maciel et al. (2014). Furthermore, when material selected under greenhouse or growth chamber conditions is field-tested, the level of resistance displayed seldom matches expectations (Kohli et al. 2011). Cruz et al. (2012) have identified the cultivars Postrock, JackPot, Overley, Jagalene, Jagger and Santa Fe as promising resistance sources, on the basis that they permitted only very low levels (<3% leaf area) of infection. Studies performed in collaboration with CIMMYT have confirmed an acceptable level of resistance in a different set of material (Table 1), in particular, the cultivar Milán (Kohli et al. 2011; Ha et al. 2012).

Table 1. Known sources of resistance against wheat blast according to CIMMYT and INIAF, Bolivia experiments

No.	Genotype	Pedigree
1	Milan	VS73.600/MIRLO/3/BOBWHITE//YOREME75/TRIFON
2	Reedling #1	Borlaug 100 F2014
3	Roelfs F2007	TACUPETO F2001*2/Kukuna
4	Sup152/Baj#1	Sup152/Baj#1
5	Misr3	Attila*2/PBW65//Kachu
6	Motacú	CROC-1/AE. SQUARROSA//OPATA/PASTOR
7	Urubó	MILÁN/MUNIA
8.	Cupesi-CIAT	ND643/2*WBLL1

To date, eight major resistance genes (*Rmg1-Rmg8*) have been identified at seedling stage (Table 2), but only four of these (*Rmg2*, *Rmg3*, *Rmg7* and *Rmg8*) determine an appreciable level of leaf resistance against the MoT isolates that cause wheat blast while *Rmg1*, *Rmg4* and *Rmg5*, and *Rmg* 6 are effective against *Avena*, *Digitaria* and *Lolium* isolates, respectively. Maciel et al. (2014) have concluded much of the resistance present in wheat germplasm is

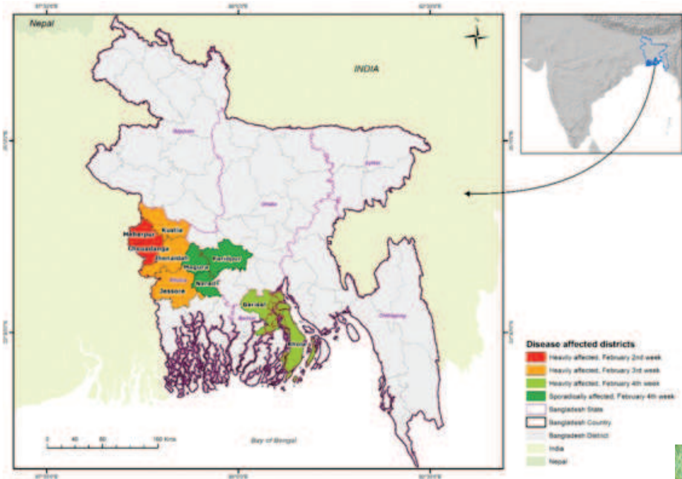

Fig. 1. Localities in Bangladesh where wheat blast was recorded during 2015-16. (Source: Amit Srivastava, Kai Sonder, CIMMYT and P. Malaker, WRC, Bangladesh)

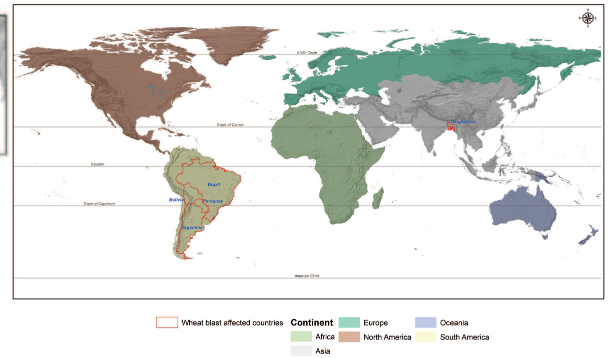

Fig. 2. The global distribution of wheat blast

Initial symptoms of wheat blast

Severe blast infection with silvery white spikes and green canopy

Fig. 3. Wheat fields affected by blast disease in southern Bangladesh in March 2016 (Image provided by P. Malaker, WRC, Bangladesh)

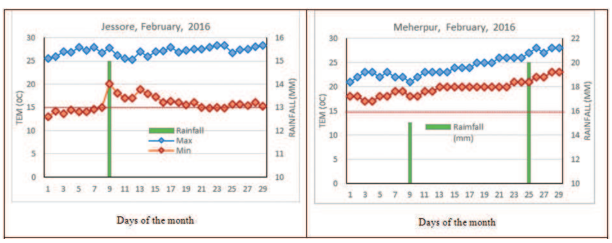

Fig. 4. Temperature and rainfall during February 2016 in the two blast-affected districts in Bangladesh. (Source: P. Malaker, WRC, Bangladesh)

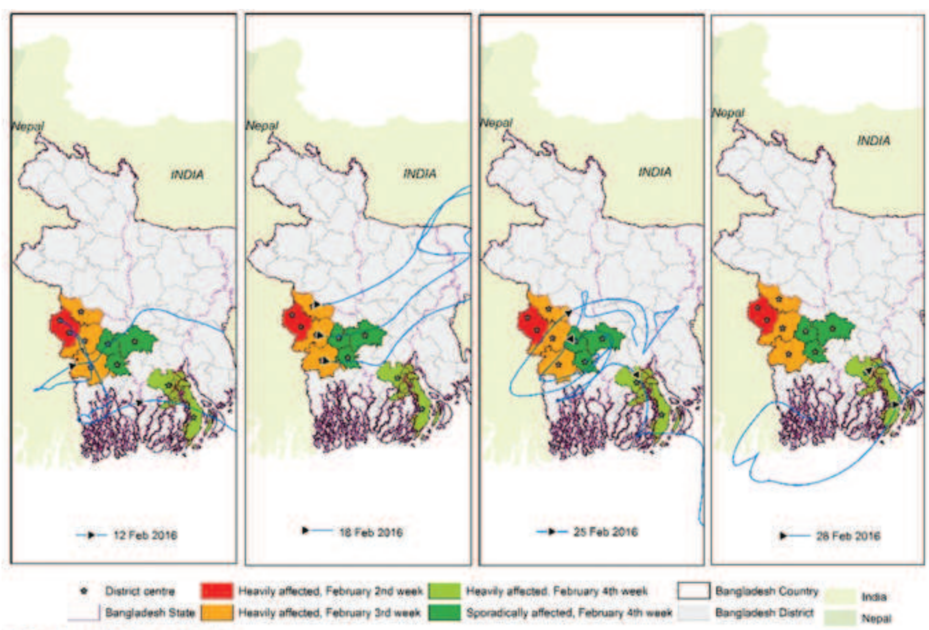

Fig. 5. Wind trajectories during Jan-March 2016 in Bangladesh. The wind trajectory was computed using HYSPLIT (Hybrid Single Particle Lagrangian Integrated Trajectory Model) model of Air Resource Laboratory (ARL), National Oceanic and Atmospheric Association (NOAA) (Stein et al 2015; Rolph 2016)

Table 2. Major genes associated with resistance against the wheat blast pathogen

Resistance genes	Reference
Rmg1 (Rwt4) identified as the first known wheat gene for resistance to wheat blast	Takabayashi et al. 2002
Rmg1 (Rwt4) proved effective against Avena and Panicum isolates	Hau et al. 2007
Rmg1 (Rwt4) reported ineffective to Triticum isolates of M. oryzae	Zhan et al. 2008
Rmg2 and Rmg3, identified on 7A and 6B chromosomes in Thatcher	Zhan et al. 2008
Rmg4 and Rmg5 genes identified on chromosome 4A and 6D, respectively	Nga et al. 2009
Rmg6, was mapped on chromosome 1D	Vy et al. 2014
RmgTd(t), considered to be a hidden resistance gene, was identified. Cytological analysis revealed that the moderate resistance controlled by this gene was associated with a hypersensitive reaction of mesophyll cells	Cumagun et al. 2014
Rmg7, was identified in a tetraploid wheat accession, St24 (Triticum dicoccum, KU120)	Tagle et al. 2015
Rmg8, was identified in chromosome 2B in common wheat 'S-615'	Anh et al. 2015
2NS translocation from Aegilops ventricosa (Zhuk.) Chennav provides resistance	Cruz et al. 2016

quantitatively inherited. While both seedlings and adult plants can be infected, it is not clear whether resistance at the former stage is predictive of resistance at the latter stage (Cruz et al. 2012; Ha et al. 2012; Maciel et al. 2014). More recently, lines bearing a distal segment of chromosome arm 2NS from *Aegilops ventricosa* translocated to wheat chromosome arm 2AS have shown an improved level of resistance to wheat blast in the spike, although surprisingly not so on the leaf (Cruz et al. 2016).

Disease control measures

Deployment of genetic resistance

As for most plant diseases, the most effective and environmentally friendly way to control wheat blast would be to breed for resistance. As yet, however, there is no certainty that durable, wide spectrum resistance can be found in the wheat gene pool.

Fungicide treatment

Chemical control can be partially effective if fungicide can be applied in a timely and safe manner. Affordability for the farmer is another key issue, especially in the context of small-holder producers. The efficacy of a number of fungicides has been widely tested (Igarashi, 1990; Goulart and Paiva 1993; Urashima and Kato 1994; Urashima et al. 2009; Kohli et al. 2011; Pagani et al. 2014; Sussel et al. 2013; Oliveira et al. 2015), but the consensus is that none can provide sufficient protection under highly conducive conditions (Urashima et al. 2009; Kohli et al. 2011; Oliveira et al. 2015). The pathogen has been shown capable of evolving resistance against both strobilurin and quinone oxidase inhibitor compounds (Oliveira et al. 2015). Based on studies in controlled conditions various authors have claimed that supplying silicate to wheat plants can improve the level of resistance expressed by the leaf (Xavier-Filha et al. 2011; Debona et al. 2013; Rios et al. 2014; Perez et al. 2014). However, the treatment did not prove so effective when infection of the spike was studied (Pagani et al. 2014).

Crop management

A number of cultural practices have been suggested as providing a measure of protection (or escape) against blast infection: these include planting date, plowing depth, the removal of alternative hosts and appropriate crop rotation. In Brazil, for instance, the recommendation is to avoid planting before April 10, because crops sown earlier would reach anthesis exactly when the right environmental conditions for the pathogen to infect the crop are most likely (Mehta et al. 1992). Deep plowing and eliminating alternative hosts have both been promoted as a means to reduce the inoculum load (Igarashi 1990). The former may, however, be too costly, apart from going against the principles of conservation agriculture currently being promoted in many parts of the world. The elimination of alternative hosts may be rather impractical. Crop rotation has some potential, but it is important to note that many other cereal species are themselves susceptible to wheat blast (Urashima et al. 2005; Kohli et al. 2011).

Implications for wheat production in the Indian subcontinent

Wheat producers in India, Bangladesh, Nepal and Pakistan face a similar spectrum of crop disease constraints. Yellow rust (*Puccinia striiformis*), for example, is a major pathogen in the North Western Plains Zone of India, which borders both Pakistan and Nepal. Likewise, spot blotch (*Bipolaris sorokiniana*) is more prevalent in the Eastern Gangetic Plains (EGP) area, which spans India, Nepal and Bangladesh. Now that wheat blast has become established in Bangladesh, there is every reason to expect that it may move into adjoining areas of India and Nepal which share a similar climate, unless effective preventive measures are soon taken. The disease could also thrive in parts of Africa (particularly Ethiopia). Epidemiological studies have concluded that the disease only arises when high humidity and warm temperatures coincide with anthesis. This scenario is geographically rather restricted in the Indian subcontinent. Areas of India and Nepal which border the wheat production area in Bangladesh currently do not produce much wheat. The major point of concern is the pathogen's capacity to evolve rapidly especially with regards to gain in virulence as has been observed in South America. In future maybe the pathogen can, perhaps evolve more widely adapted pathotypes and climate change may increase geographies conducive for wheat blast.

A strategy to contain wheat blast

Containing wheat blast within Bangladesh will require a multi-pronged strategic approach. The first priority must be to minimize its occurrence in Bangladesh itself. For neighbouring countries, containing wheat blast would need constant disease monitoring, a better understanding of its epidemiology, the breeding and release of more resistant cultivars, the development of a scheme of integrated management based on both chemical interventions and improved crop management, revisiting seed quarantine measures to restrict the movement of grain, and promoting the production of other crops in the regions bordering Bangladesh. A particularly important priority will be to identify sources of resistance to wheat blast, an approach being intensively pursued in South America (Duveiller et al. 2011; Kohli et al. 2011). Since the expression of the disease depends strongly on environmental factors, screening for resistance will need the elaboration of robust, standardized procedures. Given the apparent rarity of resistance in wheat germplasm, a substantial investment will be required to screen large sets of lines under controlled conditions, a process already begun by CIMMYT. It would be desirable to mobilize the resources of the CIMMYT led “Global Wheat Blast Consortium”, a group of European and American institutions established in 2011. At present, several international nurseries developed at CIMMYT are being screened at several locations in Paraguay, Bolivia, Argentina and Brazil. Previously 1,500 lines under the Seeds of Discovery project were evaluated for blast reaction in Paraguay, Bolivia and Brazil with few promising lines being identified as potential sources of partial resistance. CIMMYT has also initiated projects to determine the allelic relationship (if any) between genes determining resistance and is currently engaged in mapping the resistance genes expressed by the cultivar Milán and other donors (Pawan Singh, personal communication). Advanced generations of a range of populations are being bred at CIMMYT with a view to their phenotypic evaluation in South America.

Containing the disease would be supported if two major changes were implemented in cropping activity carried out in the Indian region bordering Bangladesh. These are, firstly to alter the established crop rotation regime, and secondly to promote the early sowing of wheat. Farmers need to be discouraged from rotating wheat with other cereals, instead they should choose to grow pulses or jute, which would have the added benefit of improving soil fertility. Early sowing of the wheat crop is preferred as this avoids coinciding anthesis with the weather conditions highly conducive for blast infection. It has been understood for many years that many EGP producers plant their wheat crop too late (Nagarajan 2005; Joshi et al. 2007a). Although conservation agriculture practices (Joshi et al. 2007b) have brought forward sowing time somewhat, a lot still remains to be done. The threat of wheat blast may provide a strong incentive for farmers to bring forward sowing date. Apart from escaping blast, early sown crops should be more productive, since they also avoid the stress imposes by high temperatures during grain fill.

Concluding remarks

Over the last 30 years, wheat blast has become an increasingly serious constraint on productivity in South America. Now that the pathogen has reached the Indian subcontinent, there is an urgent priority for measures to be adopted to prevent the occurrence of wide-scale epidemics. At the moment, neither genetic resistance, nor fungicidal application or crop management measures can assure an appropriate level of control. There is urgent need to identify new sources of resistance, to refine crop management practices and to better understand the disease's epidemiology. An investment in reliable screening facilities at an adequate number of sites is needed to facilitate the testing of large sets of germplasm. Germplasm exchange and the sharing of dedicated nurseries would be very beneficial. The monitoring of the movement of the wheat blast pathogen through collaborations between national and international institutions will be an important activity.

Declaration

The authors declare no conflict of interest.

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