Chapter 1
Blast Disease: Historical Importance, Distribution, and Host Infectivity Across Cereal Crops

Rajashekara Hosahatti, B. Jeevan, K. K. Mishra, A. R. N. S. Subbanna, and Lakshmi Kant

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1.1 Rice Blast Symptoms

The blast pathogen infects all the growth stages and parts of rice plant including leaf blades, nodes, and neck region (Fig. 1.1); in severe cases, the infection can be seen on leaf sheaths, rachis, joints of the culm, and even on glume. Blast disease can be described in three types depending on symptoms like leaf blast, panicle/neck blast, and node blast.

R. Hosahatti (✉) · B. Jeevan · K. K. Mishra · A. R. N. S. Subbanna · L. Kant
ICAR-Vivekananda Parvatiya Krishi Anusandhan Sansthan, Almora, Uttarakhand, India

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Fig. 1.1 Symptoms of rice blast during different growth stages: (**a**) leaf blast, (**b**) panicle blast, (**c**) node blast

1.1.1 Leaf Blast

The initial infections can be seen in the form of minute and brown-colored lesions, specks, or spots which elongate over time to become spindle-shaped pointed lesions at both ends which measure several centimeters long and about 0.5–1.0 cm wide. The center region of spot is greenish gray often covered by a brownish margin. The lesion characteristics, viz., size, shape, and color, will vary with different climatic conditions and also depending on varietal response. With prevailing favorable conditions, the disease progresses on a susceptible cultivar which appears as larger and broader lesions which are more in number and finally coalesce, leading to complete drying of the entire leaf (Padmanabhan 1974; Manibhushanrao 1994).

1.1.2 Panicle Blast

The panicle blast starts appearing at the booting stage of the crop where the stem portion just below the ear becomes brown or black which is a characteristic symptom, the neck blast. The infection is mostly confined to the neck region or sometimes the individual branches of the panicle get infected and turn brown to black. The infected panicles and whole inflorescence often break and fall off at the rotten neck. The grains on the panicle of the infected neck are generally chaffy and look whitish by far distance.

1.1.3 Node Blast

The infection starts at the juncture of two nodes, mostly infected in lower nodes of the rice plants, and turns black at the point of infection. The infected rachis and glumes contain brown to black spots particularly where the disease occurs on

branches. Finally, the infection leads to breakdown of the entire tillers of plant at the juncture of node portion resulting in yield loss of affected crop. The incidence of node blast was observed in moderate severity during recent years in North-Western Himalayan region.

1.2 Finger Millet Blast Symptoms

Similar to rice blast, the disease symptoms on finger millet can be seen at all growth stages starting from seedling to grain maturity; depending on the crop growth stage and prevailing environmental conditions the disease can be classified into leaf blast, panicle/neck blast, and finger blast. The symptoms include formation of typical elliptical or diamond-shaped lesions on leaves with gray centers which are water soaked with a chlorotic halo surrounding the lesions. The blast lesions enlarge and coalesce and give burnt appearance depending on congenial conditions. The neck blast symptoms appear as elongated black color lesion usually 1 or 2 in. below the ear and in severe infection toppling of ear head can be observed. Finger blast symptom initially appears as brown spot at the tip and as the disease progresses it proceeds toward the base of the finger. Neck infection is the most destructive stage of the disease that causes major loss in grain yield by decreasing the number and individual grain weight and can also cause spikelet sterility (Fig. 1.2).

1.3 Pearl Millet Blast Symptoms

The initial blast symptoms appear as minute specks or lesions that broaden and turn necrotic, thereby causing widespread chlorosis and complete drying of young leaves (Fig. 1.3). The symptoms are usually referred to as gray leaf spot disease. Initially, the lesions start at the leaf tips or leaf margin or both and extend down along the outer edges. Young lesions are pale green to grayish green in appearance and at later stages they turn into yellow to gray along with maturity of plants. During humid weather conditions and with high plant density the disease becomes severe.

Fig. 1.2 Symptoms of finger millet blast during different growth stages: (**a**) leaf blast, (**b**) neck blast, (**c**) finger blast

Fig. 1.3 Symptoms of pearl millet blast: (**a**) initial lesions, (**b**) complete drying, (**c**) Setaria leaf blast

1.4 Foxtail Millet Blast Symptoms

The disease is characterized by the presence of diamond- or eye-shaped lesions with gray centers bordered by yellow halo. Over time, these spots enlarge and coalesce to give blasted appearance. Although the symptom appears similar to that of finger millet blast (see Fig. 1.3), the neck and finger infections are almost missing.

1.5 Wheat Blast Symptoms

The symptoms occur on all aboveground parts of the plant. Spindle-shaped lesions, which are water soaked and with gray-green color having dark brown to reddish-brown margin, often have yellow halos, and appear on leaves. These lesions have gray centers during sporulation and white to tan centers after sporulation. As the disease advances these lesions coalesce resulting in complete death of the infected tissue (Rios et al. 2013). The most deleterious stage of disease is seen, if infection during flowering or early grain formation leads to bleaching of the spike. Depending on the susceptibility levels of cultivar, and timing and point of infection, the disease can prevent seed setting or can induce spike sterility. Infections on the rachis or peduncle can also kill the upper parts of the spike which causes highest yield losses (Goulart et al. 2007). Yield loss is in correlation with the extent of spike damage.

1.6 Historical Perspective

The first record of rice blast disease was in China by Soong ying-shin in 1637 in his book on utilization of natural resources (Manibhushanrao 1994). In Japan, it was first reported by Tsuchiya in 1704 (Goto 1955). The causal organism, *Pyricularia oryzae*, was named by Cavara in Italy (Cavara 1892) and subsequently in Japan (Shirai 1896). In Asia, the disease was first reported more than three centuries ago and distributed throughout the rice-growing ecosystems of continents. The patho-

gen is a complex species, is heterothallic, and is with continuous development of new races and isolates that are diverse in phenotypic virulence (Tharreau et al. 2009). The wide geographic distribution, rapid race evolution, high yield losses, and additional costs incurred in disease management make this disease a serious concern to rice cultivation with an estimated yield loss of US $55 million every year in South and Southeast Asia. The losses are even higher in East Asia and other more temperate rice-growing regions of the world (Herdt 1991). The disease is estimated to cause production loss to an extent of 70–80% (Ou 1985) when predisposition factors (high mean temperature values, relative humidity higher than 85–89%, presence of dew, and excessive nitrogen fertilizer application) favor epidemic development (Piotti et al. 2005). The understanding on biology of rice blast disease is therefore of particular importance, because it promises development of novel and robust disease control strategies (Skamnioti and Gurr 2009).

In India, the disease was first recorded in Thanjavur (Tanjore) delta of South India by Mc Rae in 1918. The disease is causing damage to rice production (Sundararaman 1927; Thomas 1930) in India with its recurrence in every season. But it attracted the attention only when a devastating epidemic occurred in 1919 in the Tanjore delta of erstwhile Madras state (Padmanabhan 1965). Seven epidemics of blast disease occurred incessantly between 1980 and 1987 in the states of Himachal Pradesh, Andhra Pradesh, Tamil Nadu, and Haryana imparting severe yield losses (Sharma et al. 2012).

Finger millet is an important nutri-cereal food crop of rainfed and marginal lands of arid and semiarid regions. It is mostly grown in East Africa, India, and other Asian countries including Sri Lanka and China (Fakrudin et al. 2004). In recent past, overall production and productivity of the crop have been declining majorly due to several biotic and abiotic constraints. Its production is adversely affected by a number of diseases among the blast caused by *M. grisea* (anamorph-*Pyricularia grisea* (Cooke) Sacc.), which is a major problem in India and Africa causing substantial yield losses. The blast on finger millet is known to occur in India (Mc Rae 1920), Sri Lanka (Park 1932), Nepal (Thompson 1941), Malaya (Burnett 1949), Tanzania (Kuwite and Shao 1992), Somalia (Mohamed 1980), Zambia (Muyanga and Danial 1995), Ethiopia, Kenya, and Uganda (Dunbar 1969; Adipala 1992). In India, the blast disease was first recorded from the Tanjore delta of Tamil Nadu by Mc Rae in 1920 with an estimated yield loss of 50% (Venkatarayan 1946). The average grain yield loss was reported to be around 28–36% (Vishwanath et al. 1986; Nagaraja 2007), and in the endemic areas it could be as high as 80–90% (Vishwanath et al. 1986; Bisht 1987; Rao 1990). In finger millet, the host plant resistance to blast disease is often confirmed at the seedling stage, which is not in correlation with neck and finger blast which are economically more damaging. Therefore, both neck and finger are considered as most important parameters of resistance (Nagaraja 2007) which in finger millet can be identified only by screening under natural field conditions (Nagaraja 2007; Nagaraja et al. 2010; Babu et al. 2013). The pure cultures of *M. grisea* are established from infected leaves, necks, and panicles and tested for their pathogenicity and organ specificity toward the other plant parts of finger millet (Puri and Kumar 2012). The cross-infectivity and host range studies with other cereal hosts and weed hosts were carried out with *M. grisea* infection (Shanmugapackiam and Raguchander 2018).

Pearl millet blast is a destructive disease in southern coasts of the USA (Wilson and Gates 1993). For the first time blast disease in pearl millet was reported during 1952 at Government Research Farm, Kanpur, Uttar Pradesh, by Mehta et al. (1953). In India, blast on pearl millet has emerged as a serious threat (Lukose et al. 2007; Anonymous 2009), which becomes more destructive during humid weather conditions, especially when more plants per unit area are established. Earlier it was considered as a minor disease in India; presently pearl millet blast incidence has increased in several states of India and most predominantly on new commercial hybrids (Thakur et al. 2009). Blast disease has been occurring in all the states of major pearl millet-growing states of India since 1970 and its high incidence was observed recently in all the pearl millet-growing states like Gujarat, Madhya Pradesh, Uttar Pradesh, Delhi, Maharashtra, Rajasthan, and Karnataka (Nayaka et al. 2017).

Pyricularia setariae is the most destructive pathogen, which causes blast disease of foxtail millet. Under favorable conditions, the pathogen has the potential to cause yield losses up to 40% (Nagaraja 2007). It was first officially identified in Japan by Nishikado during 1917. In India it was reported from Tamil Nadu in 1919 (Mc Rae 1920). During 2016 blast disease outbreak on foxtail millet was observed for the first time in Mazandaran province of Iran (Pordel et al. 2018).

1.7 Disease Emergence and Spread of Wheat Blast

Recently, wheat blast disease is the most destructive disease caused by *M. oryzae Triticum* pathotype (MoT) and has the ability to cause complete crop failure under favorable weather conditions. The disease was first noticed in 1985 from Brazilian state of Parana (Igarashi et al. 1986). Later, it was reported from Santa Cruz Department of Bolivia in 1996 (Barea and Toledo 1996). The pathotype is distinct from the pathotypes infecting rice (the *Oryza* pathotype, MoO); finger millet (the *Eleusine* pathotype); Italian or foxtail millet (the *Setaria* pathotype); and turf grasses (the *Lolium* pathotype, MoL). During 2016, the disease has spread to Bangladesh which impacted around 15% of total wheat-growing area with an average yield loss of 51% in affected fields (Cruz and Valent 2017). This large-scale incidence outside South America has been a concern for the potential spread to other wheat-producing areas in Bangladesh, South Asia, and beyond. Alarmed with this the Indian Government has declared wheat holiday in the neighboring districts of West Bengal.

1.8 Distribution Pattern of Blast Disease

The severity and extent of damage caused by blast disease vary every year and from place to place depending on the weather conditions and early inception of disease. However, the detailed information collected on the blast endemic districts/rice-growing areas of the country is given in Table 1.1. The finger millet blast-occurring

Table 1.1 Occurrence of blast disease during rice cropping season in India

State	Rice ecology	Endemic districts/rice-growing area	Conducive period
Andhra Pradesh	Irrigated	Srikakulam, Vishakhapatnam, Guntur, Nellore, Chittoor, East and West Godavari	September–February
Assam	Irrigated	Karimganj, Tinsukia, Nowgong, Kamrup, Goalpara, North Lakhimpur	August–October
Bihar	Irrigated	Rohtas, Samastipur, Madhubani	August–October
Chhattisgarh	Irrigated	Jagdalpur, Northern hill regions	September–October
Gujarat	Irrigated	Kheda, Nawagam, Navasari	September–October
Haryana	Irrigated	Hissar, Karnal, Panipat, Sonipat, Kaithal	August–October
Himachal Pradesh	Irrigated/ Upland	Kangra Valley (Malan, Palampur), Kulu, Mandi	August–October
Jammu and Kashmir	Irrigated/ Upland	Hill zones of Anantnag, Rajouri, Jammu, Udhampur, Larnoo	July–September
Jharkhand	Irrigated	Ranchi, Hazaribagh	August–October
Karnataka	Irrigated	Mandya, Kodagu, Shimoga, Dharwad, Gangavathi	September–October
Kerala	Irrigated	Palghat, Kuttanad, Pattambi	September–February
Madhya Pradesh	Irrigated	Bastar region, Rewa, Bilaspur	September–October
Maharashtra	Irrigated	Pune, Ratnagiri, Kolaba, Parbhani, Kolhapur, Karjat	September–October
Manipur	Irrigated/ Upland	Wangbal, Manipur Central valley	July–October
Meghalaya	Irrigated/ Upland	West Khasi hills, Umiam, Upper Shillong	July–October
Mizoram	Irrigated/ Upland	Mizoram	August–October
Odisha	Irrigated	Cuttack, Ganjam, Koraput	July–August
Punjab	Irrigated	Amritsar, Bhatinda, Patiala, Ferozpur, Ropar, Hoshiarpur	August–October
Sikkim	Irrigated/ Upland	Gangtok	July–September
Tamil Nadu	Irrigated	Thanjavur, Coimbatore, Chengalpattu, South and North Arcot, Periyar, Madurai, Pudukkottai, Tirunelveli	October–February
Telangana	Irrigated	Rajendra Nagar, Jagtial, Ranga Reddy, Nizamabad, Medak, Mahbubnagar	September–February
Tripura	Irrigated/ Upland	West and South Tripura	July–October
Uttarakhand	Irrigated/ Upland	Almora, Bhageshwar, Dehradun, Nainital and other hill areas	August–October
Uttar Pradesh	Irrigated	Faizabad, Balia, Mathura, Meerut	August–October
West Bengal	Irrigated/ Upland	Darjeeling, Cooch Behar, Bankura	August–September

Table modified from source: Forewarning Rice Blast in India, Technical Bulletin No. 9, 2004–2005

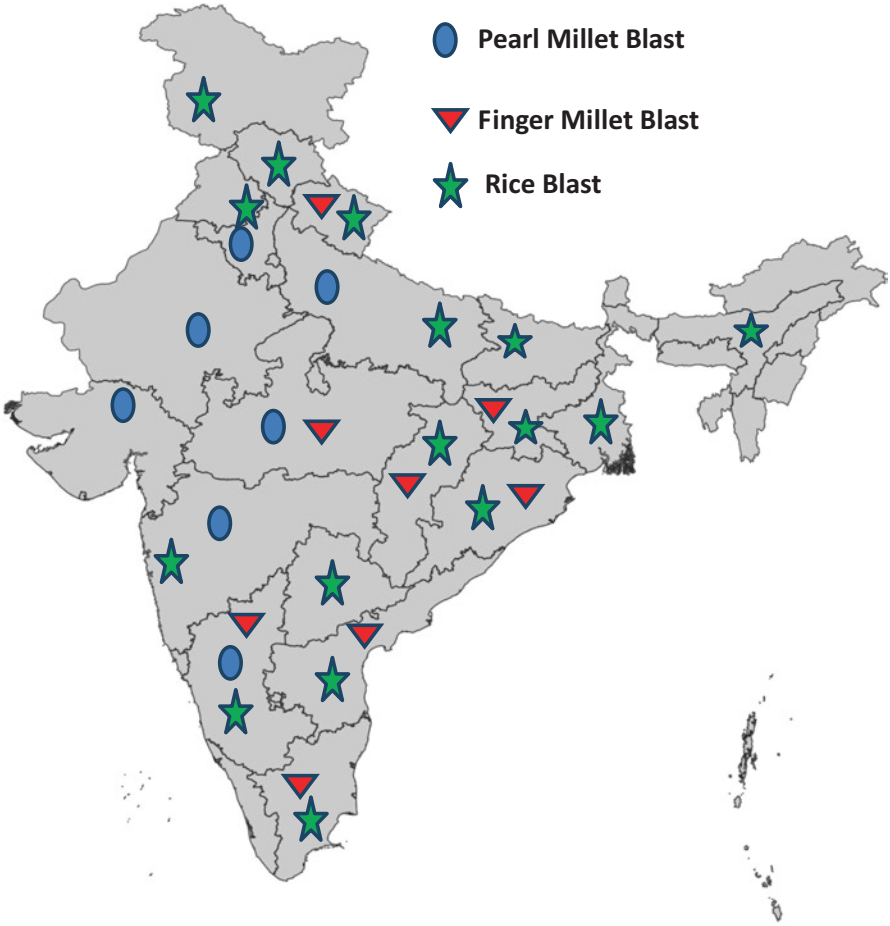

Fig. 1.4 Major blast hot spot locations of rice, pearl millet, and finger millet in India

states of India are Andhra Pradesh, Maharashtra, Karnataka, Tamil Nadu, Odisha, Uttarakhand, Madhya Pradesh, Bihar, and Chhattisgarh. The different pearl millet blast-occurring sates are also depicted (Fig. 1.4).

1.9 Establishment and Multiplication of Pathogen Isolates

Despite several decades of extensive studies on blast pathogen, the worldwide researchers always face difficulties in isolation of the pathogen from infected samples (leaf and neck) and its further establishment as monoconidial isolates (Jia 2009). Although several researchers proposed various isolation methods, the most commonly used was the slide moist chamber technique (Divya et al. 2013). In this

method, the clear blast lesions are surface sterilized by washing with 0.1% mercuric chloride and with sterile double-distilled water three times. The sterilized sample is placed over a clean glass slide which is kept inside a sterile Petri dish padded with moist cotton. After incubation, a single spore from the sporulating lesions is identified using a stereomicroscope transferred aseptically to potato dextrose agar (PDA) plates. Besides PDA, studies have also reported the use of synthetic medium like Richards agar (Ramakrishnan 1948) and natural media like autoclaved leaves and grains of different hosts for multiplication of the pathogen. However, superior growth of the pathogen was obtained on rice leaf decoction (Nishikado 1927). Recent studies also reported that prune agar and oatmeal agar supported maximum mycelial growth and sporulation of the isolates both from rice and finger millet (Khadka et al. 2012). Similarly, stem bits of 20-day-old maize, rice, and *Panicum repens* are employed in mass multiplication of blast pathogen (Divya et al. 2013). Rajashekara et al. (2016) reported efficient methods for isolation and mass multiplication of blast isolates using spore drop technique.

1.10 Host Range and Cross-Infectivity Among Major Cereal Crops

The blast pathogen *M. grisea* (Cooke) Sacc. (Rossman et al. 1990) belongs to ascomycetes group of fungi; it is a heterothallic, filamentous fungus, infecting important cereal crops like rice, wheat, barley, and millets. In addition, the host range of the pathogen spans to almost 50 plant species belonging to 30 genera of Poaceae family economically (Ou 1985). Rice blast, caused by *Magnaporthe oryzae* (Ana. *Pyricularia oryzae*), is one of the most widespread and destructive diseases. The pathogen also infects wheat and other small grain crops (Valent and Chumley 1991; Tablot 2003). In India, rice blast pathogen has been reported to infect weed hosts like crab grass (*Digitaria sanguinalis*), *Eleusine coracana*, and *E. indica* (Singh 1997). Different host range studies of *Magnaporthe* sp. are determined by mass multiplication and artificial inoculation of the pathogen on different host plants under epiphytotic conditions. Several pathogenicity tests for establishing cross-infectivity between rice and crabgrass isolates are neither consistent nor comprehensive (Choi et al. 2013).

Infectivity of blast pathogen is chiefly restricted to its host species (Ramakrishnan 1948; Todman et al. 1994), although the cross infection between the plant species is established under artificial inoculations. In Uganda, blast isolates from weed species are able to establish on finger millet seedlings. This indicates that weeds/wild grasses act as "green bridges" for finger millet blast pathogen (Ekwamu 1988). This also suggests that different weed hosts growing on field bunds can serve as chief sources of primary inoculum, thereby initiating the disease development (Mackill and Bonman 1986). Hamer et al. (1989) and Valent et al. (1986) reported that the blast pathogen is strongly delimited by host range although it is able to infect a wide range of hosts. Inoculations of rice seedlings under artificial epiphytotic conditions

with pathogen isolates of weeds resulted in successful (Mackill and Bonman 1986) and unsuccessful (Prabhu et al. 1992) cross-inoculations. Kulkarni and Govindu (1977) revealed interhost infection of blast pathogen between finger millet and foxtail, whereas both isolates did not infect rice seedlings. Viji et al. (2000) concluded that ten Indian isolates of blast pathogen from rice did not infect finger millet and vice versa, thereby confirming that the pathogen populations in India are distinct from each other. Similar findings are also reported by Kato et al. (1977) and Todman et al. (1994), whereas Kumar and Singh (1995) reported contradictory findings which may be due to the different environmental conditions prevailing during the time of experimentation and the status of nutrients in the soil (Asuyama 1965; Ou 1985). It is clear from different findings that the gene flow between rice and finger millet pathogen is majorly restricted to their respective host origin and they are considered as genetically distinct populations. The pathogenicity tests revealed that the isolates from infected weeds were pathogenic to finger millet and importantly some weed isolates are more aggressive than the finger millet isolates (Takan et al. 2004).

Pearl millet blast pathogen infects *Pennisetum glaucum*, *Pennisetum squamulatum*, *Pennisetum pedicellatum*, *Pennisetum macroforum* (Saikai et al. 1983), *Pennisetum purpureum* (Buckley and Allen 1951), and *Pennisetum ciliare* (Perrott and Chakraborty 1999). The pathogen also survives on other graminaceous hosts such as *Agrotis palustris*, *Brachiaria mutica*, *Cyperus rotundus*, *Eleusine indica*, *Eragrostis* sp., and *Panicum miliaceum* (Lanoiselet and Cother 2005). Isolates from *Panicum typhoides* did not infect rice, nor did *Pyricularia oryzae* (*Magnaporthe grisea*) attack bajra (Mehta et al. 1953).

Blast disease on wheat was first recorded in 1985 in the state of Parana, Brazil (Igarashi et al. 1986). Prabhu et al. (1992) showed that all the *P. grisea* isolates from rice, wheat, and grass weeds were pathogenic on wheat cultivars and barley in Brazil (Park et al. 2009). A special mechanism was reported to exist in these isolates so that the pathogen can infect Arabidopsis seedlings which are distinct from that of rice crop. Earlier, wheat blast outbreaks were limited and majorly reported in South America (Valent and Chumley 1991). Recently, wheat blast epidemics occurred in Bangladesh in the year 2016 (Cruz and Valent 2017).

1.11 Conclusions and Future Prospects

Blast disease of cereal crops is most widespread and destructive in nature and occurs virtually throughout the world. The pathogen is highly variable and it will vary from field to field and plant to plant during a particular season. The major findings of the studies showed that under artificial conditions mostly pathogen from one host could be able to infect another crop and *vice versa.* The continuous monitoring of pathogen shift from host to host is highly helpful for designing best management options for control of the disease.

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