Chapter 8
Blast Disease of Rice: Evolution and Adaptation in Context of Changing Climate

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8.1 Introduction

Food security is becoming a global challenge in the era of elevating climate change scenario, in the context of rice, which has driven an increased focus on developed and improved technologies of crop protection to cope up biotic and abiotic stresses. Rice (*Oryza sativa*) is the primary and staple food consumed daily by more than 50% of the world's population. Extreme weather events in climate change, combined with increased air temperature and atmospheric CO_2 concentration, are anticipated to spread diseases of rice in fresh neighbourhood (Anderson et al. 2004). Biotic stresses, viz. fungi, bacteria, viruses and nematodes, can infect more or less in the cropping season of rice and cause significant biological yield losses. Among the various biotic stresses of rice, rice blast or rotten neck blast is considered as a major yield-influencing fungal disease of the rice-growing countries of the world.

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The disease causes significant economic yield losses annually, and each year it is estimated to wipe out a huge amount of rice which is quite enough to feed more than 60 million people of the world. The blast disease was first documented in 1637 in China, and then it spread from China to Japan in 1704, and then after that it was reported in almost all the rice-growing countries gradually, in Italy (1828), the USA (1886) and India (1913), by Veeraraghavan and Padmanabhan (1965). In India, blast disease was more or less consistently recorded in major rice-growing areas. The fungus is now known to be prevalent in more than 85 countries worldwide. Rice blast is caused by *Magnaporthe oryzae*, which was differentiated from *Magnaporthe grisea* based on multilocus gene sequence-based phylogenetic analysis (Couch and Kohn 2002). Better understanding of rice blast pathosystem which consists of two inter-related subsystems, leaf blast and neck blast pathosystem, is needed for effective management of the disease (Teng et al. 1991; Teng 1994; Savary et al. 2006, 2012). Within the subsystem, vertical and horizontal host resistance governs the host resistance against future infection. The understanding of infection is very important in non-host and host plants (rice) that will be helpful in rice blast forecasting and disease management (Padmanabhan 1965). In most of the infection in subsystem it is thought to occur with rich inoculums from rice plants in their immediate vicinity, which have been successfully infected, or from the pathogen of non-hosts. Once infection has been established with an initial amount of disease, then further disease severity increases through secondary spread.

8.2 Evolution of *Pyricularia*

Pyricularia, a genus of pathogen, has a very high evolutionary potential in the aberrant climatic condition. The evolutionary potential of a pathogen population reflects its ecology and biology, and its population genetic structure (McDonald and Linde 2002). Knowledge about the evolutionary potential of *Pygt* populations is needed to predict the durability of genetic resistance to wheat blast. An intense search for blast resistance began with the first report of the disease more than 30 years ago but breeding success has been erratic and inconsistent. The average durability of resistant wheat varieties has been only 2–3 years. Furthermore, wheat genotypes behaved differently in different regions, indicating genotype-by-environment interactions or region-specific distribution of virulence groups. Reports indicated that *Pygt* is present in all Brazilian wheat-growing areas; it is likely that both the incidence and severity of wheat blast are affected by the virulence groups that predominate in each region. In fact, the occurrence of virulence groups in *Pygt* populations was already described, but information about the virulence composition and genetic structure of contemporary populations of the wheat blast pathogen remains limited.

8.3 Adaptation

That any organism has adapted to its habitat means that it has evolved diverse molecular mechanisms that allow it to grow optimally spatio-temporally with altering physico-chemical conditions and their environment (Katsantonis et al. 2017). Each organism runs after their optimum fitness in the changing environments; there are delicate differences in fitness between individuals (due to genomic plasticity or metabolic flexibility) and phylogenetic complexity (numbers and diversity of species within a given community) which can lead to the diversification of species or the extinction of less fitted genotypes over time (Kassen 2009). Aaron et al. (2010) had given emphasis on the environmental adaptation of the microorganism on three evolutionary perspectives, i.e. (1) acclimation of the existing cellular machinery to operate optimally in a new environmental niche, (2) acquisition of entirely new capabilities through horizontal gene transfer or neo-functionalisation of gene duplications and (3) reorganisation of network dynamics to appropriately adjust existing physiological processes to match dynamic environmental changes. An environment is extremely heterogeneous at microscales, and microorganisms are challenged by fluctuating biotic and abiotic stresses and parameter mixed up in changes in pH (Hughes et al. 2007), in inter- and intraspecific competition and in nutrient and resource availability (Chesson 2000). An important illustration was made by Mitchell et al. (2009) in the study of *Escherichia coli* in the digestive tract of mammals that went through a succession of carbon sources such as lactose and maltose and a succession of stresses such as increasing temperature and decreasing oxygen levels. These changes occur in a definite time and space and vary infrequently, for instance within the time frame of a single generation. Likewise adaptations can occur via several mechanisms, such as an increase in measured gene quantity, neo-functionalisation and sub-functionalisation. In all these mechanisms, mutational changes in coding regions, changes in gene expression or a grouping of both drives adaptation. Reports in *Pseudomonas fluorescens* SBW25 have also smartly demonstrated the evolution of novel phenotypes in vitro (Beaumont et al. 2009).

8.4 Changing Climate

Global population is increasing rapidly and the availability of natural resources for crop production continues to decline day by day which is escalating the challenge of global food security. An anticipated world's population of humans will be nine billion by 2050 and this is challenged by a shrinking of major land for rice (*Oryza sativa* L.) production, which is expected to decline by 18–51% in the tropics during the next century due to global warming (Godfray et al. 2010). Climate change directly or indirectly influences all the agricultural crops including cereal production through abiotic and biotic stresses, viz. heat stress, water stress along with

waterlogging, frost, disease and pest infestations (Porter et al. 2014). Challinor et al. (2014) predicted a decline of the yields of wheat, maize and rice in tropical and temperate regions. Baker (2004) reported the effect of elevated CO_2 (700 µmol mol^{-1}) under different temperature regimes at different temperatures, viz. 24, 28, 32, 36 and 40 °C, and found no increase in rice grains. On the contrary, Yang et al. (2006) showed that elevating the concentration of atmospheric CO_2 increased rice productivity. Goria et al. (2013) showed that the deleterious effect of elevated carbon dioxide concentration is likely to modify plant-pathogen interactions; when rice cultivars were exposed to elevated CO_2 (approximately 100–300 µmol mol^{-1} higher than ambient) in open-top chamber, the disease was more severe under high CO_2 concentration and area under disease progress curve was 35.43 under high CO_2 concentration and 17.48 for the normal concentration. Elevated CO_2 levels did not alter the occurrence of foliage-infecting pathogens, viz. *M. oryzae*, *Bipolaris oryzae*, *Phoma sorghina*, *Drechslera* spp., *Alternaria* spp. or *Microdochium oryzae*. Moreover, leaves of treated rice plants with CO_2 which contain less silicon have showed that leaves were more prone to foliar diseases (Goria et al. 2013). Severity of blast and sheath blight is associated with reduced silicon content in susceptible rice cultivars under elevated CO_2 (Kobayashi et al. 2006). Tonkaz et al. (2010) reported that elevated CO_2 levels also positively affected yield, grain number, leaf area and biomass. However, elevated CO_2 levels reduced harvest index and evapotranspiration but did not had any effect on flowering date, maturity and 1000 seed weight. Rodrigues and Datnoff (2005) already reported that rice cultivars which contain less silicon were more prone to foliar disease infestation, especially blast and brown leaf spot diseases, than high silicon-containing rice cultivars (Datnoff et al. 1991; Rodrigues and Datnoff 2005).

8.5 Symptoms, Pathogenesis and Management

Rice blast is a major foliage disease problem in tropical and temperate regions and is distributed in irrigated, lowland and upland rice-producing areas. The favourable conditions for rice blast include long periods of free moisture where leaf wetness and high humidity are required for infection. Spore germination, infection and lesion formation are at optimum levels at 25–27 °C while sporulation occurs in high relative humidity and temperature (25–27 °C). Severity of blast and sheath blight is associated with reduced silicon content in the leaves of susceptible rice varieties (Kobayashi et al. 2006). Additionally, increased leaf wax and epidermal thickness in rice are greater influence of physical susceptibility to pathogens along with better pathogen fecundity and changes in pathogen virulence and distribution (Plessl et al. 2005). Moreover, Matros et al. (2006) reported that elevated CO_2 modifies secondary metabolites which influence pathogen (potato virus Y) ingress in tobacco.

8.5.1 Symptoms of Rice Blast

Disease symptoms are observed on all above ground parts of the rice plant. Blast pathogen produces lesions or spots on different parts of the rice plant such as leaf, leaf collar, panicle, culm and nodes. Initial symptoms are white to grey-green lesions/spots with darker borders produced on all infected shoots and leaves, while older lesions are elliptical/spindle shaped and whitish to grey with necrotic borders and these lesions may enlarge and coalesce to kill the entire leaf. Small specks originate on leaves—subsequently enlarge into spindle-shaped spots (0.5–1.5 cm length, 0.3–0.5 cm width) with ashy centre. Sometimes internodal infection of the culm gives banded pattern of lesions. Nodal infection causes the culm to break at the infected node (rotten neck); that is why the disease is popularly called 'rotten neck disease of rice'. As a result of the disease, the plant produces fewer seeds with dull and poor quality. Disease-causing pathogen can infect paddy at all stages of growth of rice from rice seedling to matured plants. It is well observed that the infection at three leaves and neck infections may cause severe yield loss than other stage of infection.

8.5.2 Pathogenesis

Disease-causing pathogens survive in the form of conidia on or inside the seed and perithecia on infected plant debris. Primary infection is caused by activated fungal mycelium or conidia and ascospores which germinate and cause primary infection when favourable environmental condition occurs (see Table 8.1) while secondary infection is caused by asexual spores, i.e. conidia (Fig. 8.1). The infection route requires an infection peg, called an appressorium, which uses a pressure-driven mechanism to break the tough cuticle of the rice plant and sticks firmly by means of an adhesive carried in the spore apex, generating turgor pressure of up to 8.0 MPa that ruptures the cuticle of the affected rice. Once inside the tissue, the fungus produces invasive hyphae that quickly colonise living host cells, secreting effector molecules to overpower host immunity and support infection. The effectors are transported into host cytoplasm by the aid of a biotrophic interfacial complex, a plant-derived membrane-rich structure in which effectors amass during transit to the host (Kankanala et al. 2007). The pathogen can replicate quickly and successively by mitosis, nuclear migration and death of conidia from which the infection originated, and produce appressoria capable of infecting aerial structures and hyphae capable of infecting roots of young and old rice plants. Autophagic cell death of conidia is connected to cell cycle control and produces conidiophores that are dispersed to other tissues and plants by wind and water splash to reinitiate the infection cycle by attachment of a spore that germinates and forms an appressorium. This allows the pathogen to infect epidermal cells with bulbous invasive hyphae that proliferate and grow from cell to cell, often through pit fields which invade neighbouring cells through

Table 8.1 Environmental factors which favour blast disease development in rice

Conditions	Stages	Range (°C)	Optimum (°C)
Leaf wetness	All stages	Always required	
Air temperature	Appressorium germination	10–33	25–28
	Appressorium formation	21–30	28
	Lesion formation (wet leaves)		4–5 days at 25–28
	Mycelium growth	8–37	28
	Mycelium survival for 18 months	−20 to −30	−30
	Sporulation	9–35	25–28
	Dispersal of conidia		20.5–21.8
	Host blast susceptibility	10–30	25–28
Soil temperature	Rice seedlings	20–30	
	Adult plants	18–24	
RH (air)	Mycelial growth	89–96	93%
	Conidial condition		93%
	Dispersal of conidia		90%
	Disease development		93–95%
Rainfall	All stages (direct effect)	Unclear	Unclear
Sunlight	Lesion formation		Night hours
Near-UV light	Germ tube length		
CO_2	Ambient +200–300 µmol mol^{-1}		

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plasmodesmata that requires mitogen-activated protein kinase signalling and manipulation of jasmonate signalling (Kankanala et al. 2007; Patkar et al. 2015). Appressorium penetration is a septin-dependent process and is linked to a burst of reactive oxygen species in the infected cell (Kankanala et al. 2007). Rice blast conidia can spread within 230 m from their source; dispersal is favoured in darkness and with high relative humidity and winds greater than 3.5 m s^{-1}. The primary source of inoculum is infected residue and seeds of rice, and in the tropics, airborne conidia are present throughout the year, enabling stable epidemics to occur year-round (Guerber and TeBeest 2006; Raveloson et al. 2018).

8.5.3 Management

Integrated disease management strategies are required for effective successful management of rice blast by including all the available options of disease control like physical, chemical and biological agents; selection of advanced breeding lines and cultivars with resistance genes; disease forecasting; and mapping distribution of the disease. These available tactics should be integrated with agronomic practices including the removal of crop residues to decrease pathogen survival, collateral

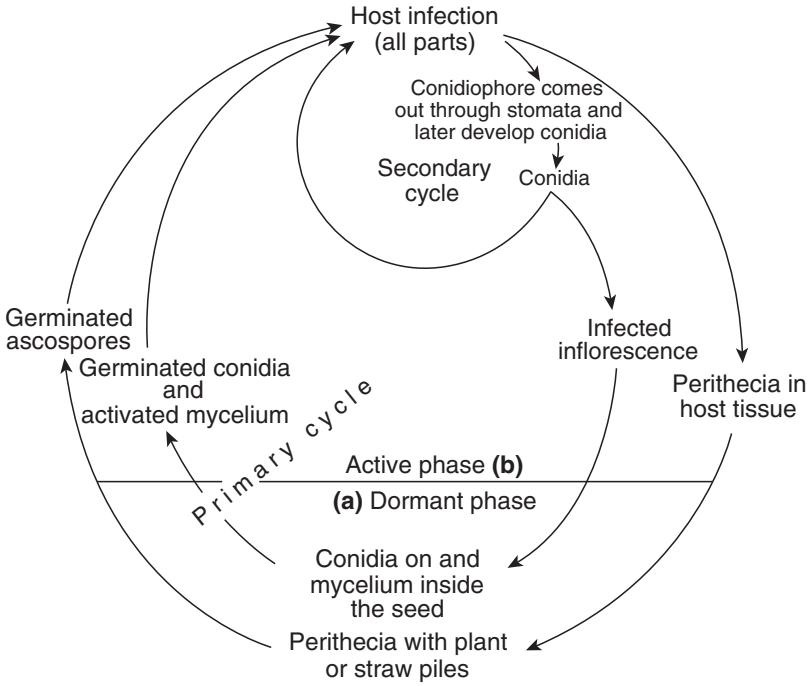

Fig. 8.1 Blast disease cycle (*Magnaporthe grisea*) (**a**). In dormant phase: in the form of conidia that survive on mycelium inside of the infected seeds or perithecia on infested plant debris or residues. (**b**). Active phase: dormant activated mycelia or conidia and ascospores germinate and cause primary infection while secondary infection causes conidia

host, adoption of crop and land rotations, avoiding of broadcast planting and double cropping, water management and balanced nutrient management (Asibi et al. 2019). Excessive use of nitrogen fertilisation as well as drought stress increases rice susceptibility to foliar disease-causing pathogens which leads to plant placed in a weakened position and its defences in weaker zone. There are two basic techniques that can be adopted for successful management of blast with the chemical fungicide strategy. In the first technique, seed treatment is used to prevent infection in seedlings after germination while in the second technique, fungicides are used to prevent infection of leaves and panicles during the growing season by making one or two foliar applications of fungicides to protect the panicles when they are emerging from the boot. This technique attempts to reduce the incidence of rice blast of seedlings, panicle necks and panicles. The most efficient way to control infection by *M. oryzae* is adopting of integrated disease management approaches. For example, eliminating crop residue could reduce the occurrence of overwintering and discourage inoculum load in subsequent seasons. Use resistant rice varieties to minimise yield losses. Knowledge of the pathogenicity of *M. grisea* and its need for free moisture suggests other control strategies such as regulated irrigation and a combination of chemical treatments with different modes of action. Managing the amount

of water supplied to the crops limits spore mobility, thus dampening the opportunity for infection. Cultural disease management practices were found highly satisfactory by removing collateral weed hosts from bunds. Use disease-free seedlings for transplanting; avoid excess nitrogen application which enhances the leaf area in per unit area which induces disease susceptibility. Application of nitrogen (N) in three split doses (50% N basal, 25% N in tillering stage and 25% N in panicle initiation stage) minimises the risk of disease. Moreover, foliar spray of chemical fungicides immediately after disease initiation/symptoms appears with tebuconazole 75 WG @ 500–750 g/ha, tricyclazole 75 WP @ 500 g/ha, or metominostrobin 20 SC @ 500 mL/ha (Groth 2006). Moreover, azoxystrobin 25 SC and propiconazole 25 EC @ 500 mL/ha were found highly effective as prophylactic as well as curative mode of management of blast disease of rice (Pak et al. 2017).

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