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An Overview of Bacterial Leaf Blight Disease of Rice and Different Strategies for its Management

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A B S T R A C T

Rice crop accounts for nearly a third of the total area under food grains in India and is the staple food for a significant portion of the world's population. Widely varying factors influence the growth of rice in different rice growing areas and render the crop susceptible to various pathogenic and non-pathogenic diseases, resulting in extensive damage of grain and straw yield. Bacterial leaf blight disease of rice caused by *Xanthomonas oryzae* pv. *oryzae* (*Xoo*) is a serious menace to rice production in India, besides other rice producing countries. Elaborate studies have been conducted on the genetics of host-pathogen interaction of BLB and exploitation of host resistance to combat the disease. This article comprehensively reviews the etiology, symptomatology, pathogen biology, disease development, disease cycle, epidemics and epidemiology, geographical distribution and strategies for the disease management *viz.*, exploitation of host plant resistance, cultural, physical, chemical and biological control.

Introduction

Rice (*Oryza sativa* L), is consider as queen of the cereal crops because of its importance as staple food of about half of the world's population (Qudsia *et al.*, 2017). The bacterial leaf blight disease of rice caused by *Xanthomonas oryzae* pv. *oryzae* and huge losses in form of quantitative and qualitative of rice. In world, due to this disease yield loss was estimated approx 50% (Shekhar and Kumar, 2020) and in India 81.3% (Prasad *et al.*, 2018; Swati *et al.*, 2015). Several biotic and abiotic factors are the main constraint for reducing the production and productivity of rice.

Among the biotic factors, diseases (26%), insects (20%) and weeds (23%) are affecting large amount of yield loss, both in terms of quality and quantity. Rice crop is affected by more than 36 fungal, 21 viral and 6 bacterial diseases. The major diseases affect the production and productivity of rice crop

worldwide due to infection of various fungi, bacteria and viruses (Jena and Mackill, 2008).

Among the different diseases of rice, brown leaf spot (*Helminthosporium oryzae*), blast (*Pyricularia grisea*), sheath blight (*Rhizoctonia solani*), sheath rot (*Sarocladium oryzae*), bacterial leaf blight (*Xanthomonas oryzae* pv. *oryzae*), rice tungro virus (Virus), false smut (*Ustilaginoidea virens*), leaf scald (*Rhynchosoprium oryzae*), bakanae disease (*Fusarium moniliforme*), grain discoloration (*Drechslera oryzae*, *Fusarium spp*.) are more prevalant (Mustafa *et al.,* 2013). Among different diseases affecting rice, bacterial leaf blight (BLB) caused by *Xanthomonas oryzae* pv. *oryzae* highly destructive in nature.

Historical background

Bacterial leaf blight disease caused by *Xanthomonas oryzae* pv. *oryzae* is one of most destructive and oldest diseases of rice recorded in the world. It was first time reported farmers of fukuoka area of Japan in 1884-1885 (Yamanuki *et al.,* 1962: Tagami and Mizukami, 1962). In early studies showed that the bacterial leaf blight disease was a physiological problem due to the acidic soils in rice growing areas (Yoshida and Muko, 1987). According to Takaishi, (1908) masses of bacteria were isolated from the (acidic) turbid dew drops on infected rice leaves, were acidic in reaction and disease was reproduced by inoculating healthy leaves with these drops (Nino liu *et al.,* 2006). The causal agent was first isolated by Hori and Bokura, in 1911 and named as *Bacillus oryzae* and Iyeda and Ishiyama renamed as *Pseudomonas oryzae* (Gnanamanickam *et al.,* 1999, Chauhan and Vaishnav, 1980) and later *Xanthomonas oryzae*. In 1978, it was reclassified as *Xanthomonas campestris* pv. *oryzae*. Southern China, disease was again characterized as distinct from bacterial leaf blight and called bacterial leaf streak in 1957. The bacterial leaf blight was named as *Xanthomonas oryzicola* and it was renamed *Xanthomonas translucens* f. sp. *oryzae* by Goto in 1964. In 1990, it was named as *Xanthomonas oryzae* pv. *oryzae* (Nino liu *et al.,* 2006).

In India, during 1951, a very devastating and widespread disease of rice was found in different areas of Maharashtra state which was first reported caused due to bacterial leaf blight by Srinivasan *et al.,* 1959. The disease was initially recorded in Maharashtra state but occurrence of epidemic in the Shahabad district of Bihar in 1963 established the destructiveness of the disease all over India (Srivastava, 1967).

Geographical distribution

In Japan, this disease has been referred to as "white withering disease" since 1881 where it was previously recorded in various localities of southern Japan. It was first reported by farmers of Fukuoka area of Japan in 1884. Later on, it was reported from all over the world *viz*. Japan, Africa, South America, Korea, Indonesia, Taiwan, China, Mexico, Thailand, India (Maharashtra) Sri Lanka, Vietnam, Philippines, Bangladesh, Australia, Malaysia, Cambodia and Latin America which signifies the wide occurrence of this disease around the world (Srinivasan *et al.,* 1959, Ou, 1977).

Bhapkar *et al.,* (1960) referred *Xanthomonas oryzae* pv. *oryzae* (*Xoo*) is one of the most catastrophic diseases of rice in Asia. In India, during 1951 a very important disease of rice was found on widespread basis in different areas of Maharashtra state which was first reported by Srinivasan *et al.,* in 1959. Most important rice growing states *viz*. Andhra Pradesh, Punjab, Haryana and western Uttar Pradesh states of India, major epidemic of this disease occurred during 1979 -1980.

This disease occurred in epidemic form in the Shahabad district of Bihar in 1963 (Srivastava, 1967). According to Mew 1987 the extent of yield loss depends on locality, season, weather, cultivar and application of high amount nitrogen fertilizer that tends to yield loss about 60%.

Economic importance of bacterial leaf blight

Bacterial leaf blight of rice has been epiphytotic in many places of the world and yield loss was estimated approx 50% (Kulkarni and Jahagirdar, 2011). According to Patel *et al.,* 2009, it is one of most devastating disease in both tropical and sub-tropical region in the world. In tropical countries *viz.* India, Philippines, Indonesia, it is more destructive because of resulting kresek syndrome of bacterial leaf blight, which affects 3-4 weeks of transplanted seedling of rice and yield loss reached upto 60-75%. This disease affects grain quality by interfering with maturation depends on weather, location and varieties. Crop losses of 10-20 per cent in moderate conditions or severe losses of up to 50 per cent in highly conducive conditions have been recorded in several Asian and Southeast Asian countries (Sharma *et al.,* 2017; Moria *et al.,* 2017).

Yield loss depends on virulence of pathogen, host pathogen relationship and different environment factors and reported that yield loss from 20-74% (Singh *et al.,* 2015). The yield loss due to this disease is 6-60% in terms of quantity and quality. About 22,000 - 1,10,000 MT losses were reported in Japan in 1954 due to the bacterial leaf blight disease of rice. In Philippines, losses were reported in resistant crop up to 9.5% in wet condition whereas for susceptible crop, losses were found 22.5% in wet and 7.2% in dry condition. In West African countries, the disease incidence ranged from 70-85% and yield losses ranged from 50-90% in the severely infected fields (Kumar *et al.,* 2017). According to Khan *et al.,* (2015) in case of severe infection, 50% yield reduction and about 10-12% yield reduction in mild infection. Yield losses as high as 81.3% have been reported due to this disease in India (Prasad *et al.,* 2018; Swati *et al.,* 2015).

This disease is endemic because of high nitrogen level, close plant spacing and high moisture condition (Mandal *et al.,* 2017; Parthasarathy *et al.,* 2014). Rafi *et al.,* (2013) reported that BLB disease of rice affect filling of the grains and emergence of panicles, about 28-30% yield reduction was observed in susceptible cultivars. This disease may weaken the seedling and in older plants the loss of grain may be 4.5-29.1%. Mubassir *et al.,* (2016) revealed that yield loss was up to 80% and depend on the crop stage, environmental condition and susceptibility.

Bacterial leaf blight is a typical vascular disease, systemic in nature (Sharma *et al.,* 2017). This bacteria infected at all stages of the rice and manifested by either Kresek or leaf blight symptom. This infection due to this disease occurs after 3-4 weeks of transplanting and later at booting or heading stage and also bacterium can infect at flowering stage (Bala *et al.,* 2017). Infection of this disease in rice is also found at the tillering stage, resulted sometimes 100% yield losses (Shahbaz *et al.,* 2016; Parthasarathy *et al.,* 2014).

The disease

Bacteria leaf blight (BLB) is vascular disease resulting in systemic infection. *Xanthomonas oryzae* pv. *oryzae* enters either through wounds or hydathodes, multiplies in the epitheme cell and move to the xylem vessel. The symptoms of this disease include Kresek (wilting) and blight of the leaves (Nino liu *et*

al., 2006). Kresek symptoms are more destructive resulted of systemic infection that commonly occurs in tropics in young plants and during the tillering stage of susceptible cultivars. Plants less than 21 days old are most susceptible and 28-34°C temperature favor Kresek development (Gnanamanickam *et al.,* 1999; Kumar *et al.,* 2009; Mew *et al.,* 1969). In kresek phase due to blockage of nutrient transfer from root to different parts of plant resulting in pale yellow symptoms appears and finally wilting of the plant. Surviving plants look like stunted and yellowing (Mew, 1987, Chauhan and Vaishnav, 1980).

Leaf blight symptoms generally occur from maximum tillering stage and onwards. Damage due to bacterial leaf blight, ranges from 20-30% and as high as 50% (Nino liu *et al.,* 2006; Habarurema *et al.,* 2013). Leaf blight symptoms favoured by warm temperatures 25-30°C, high humidity, rain and deep water. Leaf blight symptoms are characterized by wavy elongated lesion, which develop along the leaf margins (Khan *et al.,* 2015). They start as small water soaked stripes from the tips where water pores are found and rapidly enlarge in length and width, forming a yellow lesion with a wavy margin along the edge. Later on, diseased areas turn white to grey. These lesions can develop on one or both sides of the leaf and occasionally along the midribs (Qudsia *et al.,* 2017; Mew, 1987) (Fig. 1 and 2).

The sign of *Xanthomonas oryzae* pv. *oryzae* is bacterial ooze and can be seen on the margins or veins of the freshly infected leaf under moist conditions in morning hours and a source of secondary inoculums (Mew *et al.,* 1993; Nino liu *et al.,* 2006).

Fig.1 Oozing of *Xanthomonas oryzae* pv. *oryzae* in rice plant.

Fig.2 Symptoms of Bacterial leaf blight in rice plant.

Causal bacterium: *xanthomonas oryzae* pv. *oryzae*

Kingdom	:	Prokaryote
Phylum	:	Proteobacteria
Class	:	Gammaproteobacteria
Order	:	Xanthomonadales
Family	:	Xanthomonadaceae
Genus	:	*Xanthomonas*
Species	:	*oryzae.*

Morphology and life cycle of *xanthomonas oryzae* pv. *oryzae*

The *Xanthomonas oryzae* pv. *oryzae* is obligate aerobic, non-spore forming, rod shape, gram negative, round ended, motile with single polar flagellum, slime-producing. Individual cells vary in length from approximately 0.7 µm to 2.0 µm and width from 0.4 µm to 0.7 µm. Optimum temperature for *Xanthomonas* growth is between 25-30°C (Gnanamanickam *et al.,* 1999). It is catalase-

positive, unable to reduce nitrate and a weak producer of acids from carbohydrates. *Xanthomonas* colonies are formed on solid media containing glucose are round, convex, mucoid and yellow colour is due to production of the pigment xanthomonadin. *Xanthomonas oryzae* cells produce copious capsular extracellular polysaccharide (EPS). This EPS is important for the formation of droplets or stands of bacterial exudates from infected leaves, producing protection from dessication and resulted wind and rain borne dispersal (Nino liu *et al.*, 2006).

Xanthomonas can survive in the soil for 1-3 months depending on the soil moisture and acidity. In tropical region, due to high temperature, humidity and an abundance of host plants, *Xanthomonas* persists throughout the year. This disease is more likely to occur during the monsoon season of the south-east Asian and Indian oceans particularly from June to September. Kumar *et al.*, (2017) reported that the primary inoculum of disease in rice crop occurs due to *Xanthomonas oryzae* pv. *oryzae* (*Xoo*) infected rice planting seed, stem and plant parts left out after harvest.

Xoo enters through hydrathodes and wounds of the leaf tip and leaf margin of the rice leaves. Cells on the leaves surface may become suspended in guttation fluid as it exudes at night and enter the plant by swimming, or passively as the fluid is with drawn into the leaf in the morning (Nino liu *et al.*, 2006; Mouria *et al.*, 2017).

The *Xanthomonas oryzae* pv. *oryzae* is seed borne transmitted pathogen, although, transmitted through the seed has been not reported yet (Gnanamanickam *et al.*, 1999). The wind, rain splash, birds, insects and human activities disperse the *Xanthomonas* from one place to other place. The *Xanthomonas oryzae* pv. *oryzae* survive on different hosts *viz. Brachiaria mutica, Cenchus ciliaris, Cyperus difformis, C. rotundus, Cynodon dactylon, Echinochloa crusgalli, Leersia spp* (*Leersia hexandra, L. oryzoides*), *Leptochloa chinensis, Panicum maximum, Zizania aquatica, Z. palustris, Leersia. Oryzoides* and *Zizania latifolia* in temperate region and *Laptochloa spp*. and *Cyperus spp*. in tropical region. In temperate regions, *Xanthomonas* can survive in the rhizosphere of weeds of the genera *Leersia* and *Zizania* as well as in the base of the stem and the roots of rice stubble in winter season (Nino liu *et al.*, 2006).

Bacterium multiplies inside the vascular system and moves systemically to different plant parts. *Xanthomonas oryzae* pv. *oryzae* grows in plant and infected plant veins as well as the xylem that leads to blockage and wilting. Most favorable temperatures for *Xoo* growth ranges from 26-30°C and 20°C is ideal temperature for initial multiplication and growth. *Xoo* can tolerate pH ranges from 4 to 8.8 and optimum pH observed for its survival was reported 6-6.5 (Kumar *et al.*, 2017).

Management of bacterial leaf blight disease of rice

At present, cultural, physical, chemical and biological control have been used to manage Bacterial leaf blight disease of rice.

Cultural management

Tabei (1960, 1967) reported that for management of the disease normally use of healthy seeds, infected stubbles, straws, leaves and weeds were removed from rice fields. Using suitable plant spacing, application of appropriate nitrogenous fertilizer (NPK), using modern irrigation system and by making the drainage systems as better as recommended.

Physical management

Srivastava and Rao (1963, 1964) described that 95-100% eradication of *Xanthomonas oryzae* pv. *oryzae*, rice seeds was dipped into 0.07% solution of agrimycin by 12 hours and move out these seeds into water bath at 54°C for 30 minutes that reduces the bacterial leaf blight incidence in the rice field. Jain (1970) reported that effective control of bacterial leaf blight disease of rice, hot water treatment of rice seeds for about 30 minutes at 52ºC proceeded by 8-10 hour of presoaking in water which was found to be the most effective against the bacterial leaf blight of rice. Zhang *et al.,* (1996) described that 2-3 times washing of rice seeds in distilled water after that washing by the brine solution also minimized the disease and increased the germination percentage. Presoaking of rice seed in 50 ppm suspension of zhonheshengmycin also minimized bacterial leaf blight disease incidence.

Chemical management

Elaborate studies have been conducted to determine the efficiency of combinations of different chemicals to manage the disease and reduce yield loss. Different combinations of antibiotics at varied concentrations have been recommended for disease inhibition by different workers over the years.

Singh *et al.,* (2015) reported a combination of Streptomycin + Copper oxychloride @ 4% was highly effective under *in vitro* condition. Other recommendations like Copper oxychloride (0.25%) + Streptomycin sulphate 200 ppm (Kumar *et al.,* 2009); Streptomycin @ 0.03% and @ 0.05% were also found effective (Prasad *et al.,* 2018). In studies conducted by Patil *et al.,* (2017) Streptocycline (streptomycin sulphate + tetracycline hydrochloride, 9:1) + Copper sulphate ($CuSO_4$) @ 1000 ppm was more effective than Streptocycline @ 1000 ppm alone. Similar results were stated by Thimmegowda *et al.,* (2012).

Jadhav *et al.,* (2018) studied the *in vitro* effect of Carbendazim @ 500 ppm, Mancozeb @ 500 ppm and Streptocycline @ 250 ppm against *Xanthomonas axonopodis* pv. *citri* causing bacterial canker of kagzi lime and found Mancozeb @ 500 ppm to be highly effective followed by Streptocycline @ 250 ppm. Comparable results have been reported against *Xanthomonas campestris* pv. *viticola* causing bacterial leaf spot of grapes and bacterial leaf blight of rice (Gangwar, 2013). Inhibition by Mancozeb @ 500 ppm and Streptocycline @ 100 ppm was found to be at par (Kamble *et al.,* 2017). Gangwar and Sinha (2012) found that 6% inhibition of *Xanthomonas* in Carbendazim treatment as compared to control in laboratory condition.

Several strobilurins have been evaluated and found effective against BLB. Bala *et al.,* (2017) reported the efficiency of Trifloxystrobin 25% + tebuconazole 50% @ 50 ppm against *Xoo* under *in vitro* condition. Azoxystrobin 25 SC (Amistar) @ 1 ml/l water performed better than Carbendazim 50 WP @ 1gm/l water against bacterial leaf blight disease of rice (Swati *et al.,* 2015).

Different recommendations of chemicals have been reported by workers against BLB in rice field. Treatments that resulted in minimum disease severity, least economic loss and maximum overall yield have been considered superior to others. Patil *et al.,* (2017) tested different chemicals and found that the application of Streptocycline @ 0.5% + copper oxychloride @ 2.5% was most effective against bacterial leaf blight disease of rice. Other recommendations like Copper oxychloride @ 0.30%, Streptomycin sulphate @ 0.05%, Copper oxychloride 2.5 gm/lit, Streptomycin sulphate 0.05 gm/lit + Copper

oxychloride 0.5gm/lit, Streptocycline @ 0.025% + Copper oxychloride @ 0.1%, Streptomycin sulphate @ 0.03% + copper hydroxide @ 0.25% and Streptocycline @ 0.03% + copper hydroxide @ 0.25% were also found competent in disease control (Singh *et al.,* 1980; Khan *et al.,* 2005; Patel *et al.,* 2009; Kulkarni and Jahagirdar, 2011; Thimmegowda *et al.,* 2012; Prasad *et al.,* 2018).

Jagtap *et al.,* (2012) reported that disease control efficiency of Copper oxychloride (0.25%) + Streptocycline 100 ppm was at par with Carbendazim @ 0.1% + Streptocycline @ 100 ppm against *Xanthomonas axonopodis* pv. *malvacearum* causing bacterial leaf blight disease of cotton. Combination of Blitox (0.3%) + streptocycline (250 ppm) was found effective against *Xanthomonas axonopodis* pv. *punicae* causing bacterial leaf blight disease of pomegranate (Ashish *et al.,* 2016). Copper oxychloride (1000 ppm) + Streptocycline (250 ppm) have also been reported against *Xanthomonas axonopodis* pv. *punicae spp*. (Meena *et al.,* 2017). Jarial *et al.,* (2015) reported that Streptocycline @ 0.01% was most effective in reducing bacterial spot of bottle gourd caused by *Xanthomonas cucurbitae*.

Based on field trials conducted for two consecutive years, Chaudhary *et al.,* (2012) reported that Bordeaux mixture alone and in combination with oxytetracycline and streptomycin was significantly effective against BLB in rice. Singh *et al.,* (2015) found minimum disease incidence and maximum yield in plots treated with Oxytetracycline @ 75 gm/ha + Copper oxychloride @ 500 gm/ha which was as effective as Streptocycine @ 15 gm/ha + Copper oxychloride 500 gm/ha against bacterial leaf blight disease of rice. Biswas *et al.,* (2009) observed that seed treatment with Streptocycline (100 ppm) and three foliar application of Streptocycline in combination with Copper oxychloride was most effective in minimizing bacterial leaf blight disease incidence. Seedling root dip with streptocycline (0.01%) + spraying of Streptocycline (0.01%) + Copper oxychloride (0.2%) was recommende by Mandal *et al.,* (2017). Shahbaz *et al.,* (2016) found treatment with copper oxychloride @ 1235 gm/ha to be at par with Trifloxystrobon + Tubeconazol @ 160 g/ha in disease control. Application of newer chemicals like Amistar @ 0.1%, Nativo 75 WG @ 0.65% have been found effective against bacterial leaf blight disease of rice by several workers in the recent years (Mustafa *et al.,* 2013; Razu and Hossain, 2016; Qudsia *et al.,* 2017).

Biological management

The biological control of plant pathogens by antagonistic microorganisms is known to be a cheap, effective and eco-friendly method for the management of crop diseases (Cook and Baker, 1983). *Pseudomonas* and *Trichoderma* have been known for their potential to reduce the plant disease caused by fungal and bacterial pathogens (Pant and Mukhopadhyay, 2001). The bacterial antagonist *Pseudomonas fluorescens* strain 7-14, suppress both blast and sheath blight of rice (Gnanamanickam and Mew, 1992; Chatterjee *et al.,* 1996; Krishnamurthy and Gnanamanickam, 1998). The use of antagonistic bacteria *viz*. *Bacillus* sp. for suppression of BLB of rice has been documented (Vasudevan, 2002).

Ark *et al.,* (1986) reported that the antibacterial and antifungal activities of alcoholic plant extracts of barley and wheat seeds which significantly checked the growth of the tested bacterium. Rode *et al.,* (1989) investigated that potential botanical extracts in different solvents of *Allium ampeloprasum* and *A. sativum* under *in vitro* conditions against bacteria and fungi, which showed

significantly higher results as compared to the un-treated control as these extracts. Kazmi *et al.,* (1991) tested different plant extracts for controlling the growth of *Rhizoctonia solani* and found aqueous extracts of neem seeds, 30% neem and garlic bulb 4% showed antifungal activity whereas neem oil at 0.1% was found more effective against tested fungi than benomil.

Resistance and susceptibility in rice to *Xanthomonas oryzae* pv. *oryzae*

The Bacterial leaf blight of rice caused by *Xanthomonas oryzae* pv. *oryzae* (*Xoo*) is known to be controlled by genetic resistance with over 40 resistance (R) genes identified to control the disease (Verdier *et al.,* 2011). Representatives of a major class of resistance genes (R genes) against the disease that are prominent in rice are given the prefix *Xa* for *Xanthomonas*. The first R gene for bacterial blight as well as the first R gene of the receptor kinase (RLK) class to be cloned is *Xa21* which was introgressed into rice from the related species *Oryza longistaminata* at Central Rice Research Institute, Cuttack, India (Song *et al.,* 1995). *Xa21* confers broad spectrum resistance to several strains of *Xoo* (Ronald *et al.,* 1992; Wang *et al.,* 1996). RLKs have been shown to play a key role in a number of signaling pathways in plants, including innate immunity (Morillo and Tax, 2006).

A novel insight into *Xa21* mediated immunity in rice against *Xanthomonas* suggested that a sulfated peptide called ax YS22, derived from *Xanthomonas oryzae* secreted protein Ax21. This Ax21 being present in most *Xanthomonas* species was considered a PAMP and *Xa21* a PRR that elicits *Xa21* mediated resistance by binding to the LRR domain of the *Xa21* protein (Lee *et al.,* 2009). Extensive studies on *Xa21* mediated signaling network have revealed important role of five *Xa21* binding proteins (XBs), including an ATPase (XB24), an E3 ubiquitin ligase (XB3), a PP2C phosphatase (XB15), a WRKY62 transcription factor (TF) (XB10) and an ankyrin-repeat protein (XB25), in regulating the rice defense response against *Xanthomonas oryzae* pv. *oryzae* (Chen *et al.,* 2010; Jiang *et al.,* 2013; Park *et al.,* 2008; Peng *et al.,* 2008; Wang *et al.,* 2006). XB24 catalyzes the auto phosphorylation of serine and threonine residues on *Xa21* and this modification is essential to keep *Xa21* in an inactive form. *Xa21* kinase disassociates from XB24 and it activates upon recognition of pathogen invasion (Chen *et al.,* 2010). This activation triggers numerous downstream events that elicit the defense response. The N-terminal portion of XB25 physically interacts with the transmembrane domain of *Xa21* through the binding to transmembrane and positively charged domain (BTMP) repeats of XB25 (Jiang *et al.,* 2013). Down regulation of Xb25 reduces the levels of *Xa21* thus compromising the *Xa21* mediated resistance. Thus, several diverse proteins are involved in the activation of *Xa21* and signaling after pathogen recognition in rice (White and Yang, 2009).

Other members of the RLK family include *Xa26* also conferring broad resistance to a rather different strain profile (Sun *et al.,* 2004). *FLS2* and *EFR* genes have been identified as components of the pathogen-associated molecular pattern triggered immunity (PTI) response (Gomez-Gomez and Boller, 2002; Zipfel *et al.,* 2004). *Xa1* represents another major class of R genes, the nucleotide-binding site (NBS)-LRR group (Yoshimura *et al.,* 1998). The expression of *Xa1* is regulated upon bacterial infection and where in R gene regulation is coordinated with other pathways for defense responses. *Xa1* was found to be effective against some *Xoo* isolates in Japan but ineffective against most strains from the Philippines (Yoshimura

et al., 1998; Zhang and Gassmann, 2007). *Xa27* is another gene that confers broad resistance and is representative of are class of dominant R genes in rice (Gu *et al.,* 2004), whose specificity is based on differential gene expression (Gu *et al.,* 2005). *Xa4* was primarily derived from an Indian cultivar TKM6 and convene resistance in IR20 and other IR varieties, developed at IRRI (Khush *et al.,* 1990; Zhang *et al.,* 1998). Cao *et al.,* (2007) reported substantial resistance against the disease imparted by *Xa3*/ *Xa26* gene in rice at both seedling and adult stage (Liu *et al.,* 2020).

Although it has been well known that most plant R genes are dominant, recessive genes have also been recognized in many host-pathogen interactions. Several recessive R genes have been characterized from rice (Ogawa and Khush, 1988). Of these, 3 (*xa5*, *xa8* and *xa13*) occur naturally and confer race-specific resistance. Another 3 (*xa15*, *xa19* and *xa20*) have been induced by mutagenesis and each confers a wide spectrum of resistance (Iyer and Mc Couch, 2004; Jiang *et al.,* 2006).

The recessive resistance gene, *xa13*, has been identified by map-based cloning (Chu *et al.,* 2005; Chu *et al.,* 2006). The dominant allele *Xa13* was also named Os8N3 due to the location on rice chromosome 8 and the similarity to MtN3 [nodulin 3 (N3) protein of *Medicago truncatula*] (Gamas *et al.,* 1996; Yang *et al.,* 2006). It was recognized that the critical difference between resistant (*xa13*/*xa13*) and susceptible plants lies in the elevated expression of Os8N3 during bacterial infection in susceptible plant genotypes and the absence of Os8N3 induction during infection in the resistant genotypes (Yang *et al.,* 2006; Yuan *et al.,* 2009). Thus, Os8N3 is a gene for susceptibility that is exploited by *Xoo*, and the *xa13* allele occurs naturally for resistance (Yang *et al.,* 2006).

There have been evidences in support of the strategy of pyramiding appropriate resistant genes to develop resistant cultivars. Gene combination *Xa4 + xa5, xa5 + xa2l* and *xa4 + xa5+ xa2l* confers broad spectrum resistance to numerous *Xoo* isolates (Saha *et al.,* 2015). Marker assisted breeding strategy has also been found advantage in enhancing the resistance of elite cultivars (*viz.* Swarna and IR 64) to bacterial leaf blight by pyramiding few specific resistance genes (*xa5, xa13* and *Xa21*) through backcrossing. The lines thus developed exhibited a wider and higher level of resistance as compared to lines with only a single gene (Huang *et al.,* 1997; Sridhar *et al.,* 2001; Saha *et al.,* 2015).

Durability of genetic resistance

R genes stimulate a strong, usually race-specific, resistance response in host plant that results in development of small lesions, localized cell death or lack of susceptibility. The major constraint after deployment of these R genes is the fact that pathogen populations evolve rapidly in order to overcome the resistance. Durability of effective resistance is therefore, a continuing challenge for the control of bacterial leaf blight (Delorean, 2016).

In Effector triggered immunity (ETI) or the common R gene-mediated resistance, plant R proteins either directly recognize specific effectors or indirectly recognize the interference of the effectors through ‘guardee’ protein and this recognition leads to stimulation of resistance response.

This type of resistance is dependent on the pathogen’s effectors and is pathogen race-specific. In the *Xoo* rice pathosystem, resistance results from recognition of Transcription Activator Like (TAL) virulence effectors by host R gene (Rao *et al.,* 2002; Zhang and Wang, 2013). *Xoo* TAL effectors

are interesting proteins because of their unusual structure and functions that affect plant processes in unique ways (Schornack *et al.,* 2013). TAL effectors target host genes expression in order to manipulate their regulation in favor of pathogen development. In spite of intermittent differences, typically TALs are virulence effectors that activate host genes called susceptibility genes by binding to EBE in target gene promoters. Examples of susceptibility genes include copper and sugar transporters, transcription IIA subunits, and bZIP transcription factors (Iyer and McCouch, 2004; Sugio *et al.,* 2007; Yuan *et al.,* 2010; Streubel *et al.,* 2013; Hutin *et al.,* 2015).

In response, hosts too have evolved diverse resistance mechanisms to combat the action of TAL effectors. A majority of these include genetic mutations in the TAL target genes or their promoters. Mutations in the EBEs of susceptibility genes, for example, result in resistance because the mutations block activation of the genes. Recessive resistance genes *xa13* and *xa25*, the two well-characterized R genes to *Xoo* possess such EBE mutations (Chu *et al.,* 2006; Yuan *et al.,* 2009; Zhou *et al.,* 2015).

Some TALs directly activate resistance genes that regulate cell death for hypersensitive response. TAL effector Avr *Xa27*, for example, binds to an EBE in the promoter of the resistance gene *Xa27* and triggers a hypersensitive response (Gu *et al.,* 2005). The importance of TAL effectors in plant disease resistance is asserted by the cumulative understanding of TAL activity in host-pathogen interaction and the variations in mechanisms that evolved in plants to surpass TAL effectors.

This knowledge offers a basis for devising various strategies to overcome TAL effector-based virulence, exploiting TAL effector-triggered resistance and using TAL effector fusion proteins to engineer genomes with the motive of aborting or limiting pathogen infection (Schornack *et al.,* 2013).

Structural and biochemical mechanism of resistance

Besides, genetic resistance structural and biochemical resistance has also been suggested in rice against BLB (Khan, 2014; Saha *et al.,* 2015). Rice cultivars with few, short, narrow and erect leaves have been shown to be less prone to BLB infection as compared to those having luxuriant growth and spreading leaves (Kiryu and Mazuta, 1995). Retention of more bacterial inoculums was high in rice cultivars with hairy leaves and these showed maximum disease while the disease was very low in cultivars with glabrous leaves (Dath *et al.,* 1977). The stomatal index was usually lower in resistant varieties than susceptible ones (Shukla and Gangopadhyay, 1981). A negative correlation has been reported between BLB disease development and frequency of distribution of silicate cells in rice leaves (Kaul and Sharma, 1987). Resistant cultivars have a higher ratio of reducing sugars to total nitrogen, higher contents of polyphenols, lower contents of some free amino acids and produce some non-specific phytoalexins (Mahto *et al.,* 1987; Ou, 1985). The development of fibrillar material in rice plants has been reported to be a defense mechanism associated with host-pathogen interaction and causes immobilization of bacterial inoculum (Horino, 1981).

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