

Innate Immunity in Rice: A Defense Against Bacterial Leaf Blight

ABSTRACT

Rice (*Oryza sativa* L.) is one of the major staple food crops of the world and sustainable rice production is important for ensuring global food security. Throughout the growing season, a variety of pathogens including fungi, bacteria, viruses, and nematodes, infect different parts of the crop resulting in yield loss. Bacterial leaf blight caused by *Xanthomonas oryzaepv.oryzae*(Xoo) is one of the major limiting factors in rice production. Being a vascular pathogen, *X.oryzaepv. oryzae* interferes with a range of physiological and biochemical exchange process in rice. The earlier the pathogen infection, more will be the loss and grain quality is also severely affected due to infection. The spread of the pathogen is too rapid in the favourable climatic conditions. The disease results in 20 to 30 per cent annual loss in rice production and under severe conditions, the yield loss goes up to 80 per cent. Plant pathogens employ different ways to attack host plants and impair plant growth and reproduction. Unlike vertebrates, plants lack mobile immune cells and an adaptive immune system. Plants mainly rely on two interconnected tiers of the innate immune system to perceive and respond to pathogen infection. This innate immune system or basal resistance mediated by a repertoire of Resistance or R genes in plants acts as the first line of pre-formed and inducible defenses that protect the host plants from large number of pathogens. Over the years, extensive investigation on the molecular interactions between rice and *Xanthomonas oryzaepv.oryzae* has made impressive progress in understanding the molecular basis of rice innate immunity against bacterial leaf blight. Improving plant immunity has been considered as one of the best choices available for achieving economical and sustainable management of bacterial leaf blight in a durable manner. In this review, we summarize the molecular basis of two tiered innate immune system including PAMP triggered immunity (PTI) and Effector triggered immunity as well as R genes involved in rice - *Xanthomonas oryzaepv.oryzae* interaction.

*Key words: Xanthomonas oryzae*pv. *oryzae*, *Bacterial leaf blight*, *R genes*, *Innate immune system*, *Defense*

INTRODUCTION

Rice is the most important cereal crop in the world feeding more than 50 per cent of the world population. It is a rich source of carbohydrates and energy and provides 20 per cent of the world calories requirement [1]. Demand for rice is increasing year after year with the increase in population. But rice production is limited by various biotic and abiotic factors. Among them, vulnerability of the crop to diseases results in drastic crop loss and yield reduction. A large number of fungi, bacteria and virus infect rice crop. Bacterial leaf blight (BLB) of rice is one of the most devastating and economically important diseases in rice. It is caused by the bacterial pathogen *Xanthomonas oryzae* pv. *oryzae*, which has been ranked as the fourth most important plant pathogenic bacteria worldwide [2]. The disease causes yield loss in the range of 20 to 30 per cent, but under severe infection, yield loss up to 80 per cent has been observed [3]. Several control measures such as chemical and biological methods are used to control the spread of the disease. Unfortunately, these measures could not effectively control the disease. The use of pesticides is expensive and are not eco-friendly and biological control alone cannot completely control the disease. Therefore, one of the best, economic and safe ways to manage this disease is by boosting the innate resistance of the crop against pathogen attack[4].

Microbes live in nearly every environment and even in nutrient limited conditions in soil and water. Eukaryotes provide an ideal habitat for various microbes. Even though plants and animals are surrounded by millions of microorganisms, they remain healthy in most of the situations. Plants are attacked by large number of microbes, but only few succeed in causing disease. Others are detected by multiple layers of sophisticated surveillance mechanisms that recognize potentially dangerous pathogens and rapidly respond before those organisms have a chance to cause serious damage. This is known as the innate immunity or basal resistance of the plants and it is mediated by a repertoire of Resistance or R genes in plants. By using this innate immunity, plants encounter majority of the pathogens which come in their way. Hence, one of the best ways to manage BLB is

by enhancing the innate immune system of rice by breeding for resistance varieties[5].

BACTERIAL LEAF BLIGHT OF RICE

Bacterial blight is a seed borne disease caused by the gram-negative bacterium, *X. oryzaepv.oryzae*. It is a severe threat to rice production. The disease was first observed by the farmers of the Fukuoka area of Japan, in 1884 [6]. In India, bacterial leaf blight disease incidence was first reported in Maharashtra [7]. Earlier, the disease was considered to be of minor importance in India until it broke out in an epidemic form in Shahabad district of Bihar in 1963. This disease can affect rice plants at any plant growth stages. Bacterial leaf blight generally causes yield loss ranging from 20 to 30 per cent. In case of severe infection, disease cause yield loss up to 80 per cent besides severely affecting the grain quality [3].

PATHOGEN MODE OF INFECTION AND SYMTOMATOLOGY

X. oryzaepv.oryzae enters the rice leaf typically through hydathodes at the leaf tip and leaf margin [8]. Subsequently, it multiplies and flourishes in the intercellular spaces (apoplast) beneath the epithelial cells and later disseminates to other parts of plants through the xylem vessels [9]. Within a few days of infection, bacterial cells and Exopolysaccharides (EPS) fill the xylem vessels and ooze out from hydathodes, forming beads or strands of exudate on the leaf surface, a characteristic sign of the disease and a source of secondary inoculum [10].

The symptom of bacterial leaf blight is first observed as water-soaked spots at the leaf tips and margins. The spots expand along the veins, merge, and become chlorotic and then necrotic, forming opaque, white to grey coloured lesions that extend from the leaf tip down along the leaf veins and margins. Symptoms are often evident at the tillering stage and become severest at the flowering stage. In the tropics, susceptible rice plants show a seedling blight disease called Kresek, which is a more destructive manifestation of the disease in which the entire foliage becomes pale yellow and wilts, resulting in a partial or total failure of the crop [11].

INNATE IMMUNE SYSTEM OF PLANTS

Immune system is essential for the survival of all living organisms. Without an immune system, both plants and animals would be open to attack to many microbes

such as bacteria, viruses, fungi and protozoa. Plants and animals should avoid becoming a meal to microbes, which vastly outnumber eukaryotic life in both quantity and diversity. It is the immune system that keeps the plants and animals safe and healthy against a sea of pathogens. Both plants and animals have an innate immune system, which protect them from the majority of microbes they encounter during their lifetime [12] [13].

Plants have multiple layers of sophisticated surveillance mechanisms that recognize dangerous pathogens and rapidly respond before those organisms which have a chance to cause serious damage. These surveillance mechanisms are linked to some specific pre-programmed defense responses and this is known as the innate immunity or the basal resistance of the plant. It is the first line of pre-formed and inducible defense that protect against entire groups of pathogens. In short, the innate ability to defend against pathogen attack is known as innate immunity of the host [14].

Plants have developed a two-layered innate immune system that includes pathogen-associated molecular pattern (PAMP) triggered immunity (PTI) and effector triggered immunity (ETI). PTI is the first line of defense, which is governed by pattern recognition receptors (PRRs) that recognize highly conserved PAMPs to trigger a relatively weak immune response that restricts colonization by invading organisms [15]. In contrast, ETI, the second line of defence, is a rapid and robust response, usually associated with a hypersensitive reaction (HR) in plants. ETI is initiated by R proteins that directly or indirectly recognize highly variable pathogen produced molecules called avirulence (Avr) effectors [16]. The innate immune system of plants operates through three stages Viz., PAMP triggered immunity (PTI), Effector triggered susceptibility (ETS) and Effector triggered immunity (ETI) (Fig. 1).

PAMP-TRIGGERED IMMUNITY (PTI)

PTI involves the recognition of conserved, indispensable microbial elicitors called microbe/pathogen associated molecular patterns by a class of plasma membrane bound extracellular receptors called PRRs [17]. Activation of PRRs leads to intracellular signaling, transcriptional reprogramming, and biosynthesis of a

complex output response that limits colonization, which ultimately contribute to halt infection before the pathogen gains a hold in the plant [18].

PAMPs essential structures or components that are conserved throughout the whole classes of pathogens. This includes oligogalacturonides, ergosterol, bacterial flagellin, pep-13, xylanase, cold-shock proteins, lipopolysaccharides (LPS) *etc.* Many plant pathogens produce lytic enzymes to break the structural barriers of plant tissues. The products formed as a result of these enzymatic degradation such as cell wall fragments and peptides, can function as endogenous elicitors called damage-associated molecular patterns (DAMPs). These DAMPs emerge in the apoplast and serve as danger signals to induce innate immunity similar to MAMPs [19].

PRRs are plasma membrane localised proteins capable of recognizing PAMPs and activating PTI. The PRR family includes receptor-like kinases (RLK) and receptor-like proteins (RLP) [20].

EFFECTOR TRIGGERED SUSCEPTIBILITY (ETS)

Successful pathogens have many evolved strategies to challenge the PTI and promote pathogenesis by injecting a set of effector proteins across the plant cell wall into the cytoplasm through the type III secretion system (TTSS) and this is known as effector triggered susceptibility [21].

EFFECTOR TRIGGERED IMMUNITY (ETI)

Effector triggered immunity (ETI) is activated by the effectors produced by the pathogens. Receptors on the plant recognize the pathogen effectors and activate ETI. ETI immune response is depended on R genes, and is activated by specific pathogen strains. Plants ETI often cause an apoptotic hypersensitive response [22].

RESISTANCE GENES (R GENES)

Plants have evolved R genes whose products allow recognition of specific pathogen effectors, either through direct binding or by recognizing effector's alteration of host proteins [17]. Plants encode many types of extracellular and intracellular R proteins. Many R genes encode NB-LRR type proteins (nucleotide-

binding/leucine-rich repeat domains, also known as NLR proteins or STAND proteins). R gene products control a broad set of disease resistance responses whose induction is often sufficient to stop further spread or growth of the pathogen. Most of the plant immune systems carry a repertoire of 100-600 different R genes that mediate resistance to various virus, bacteria, fungus, oomycete, nematodes and insects. R genes are generally very specific to particular pathogen strains [12].

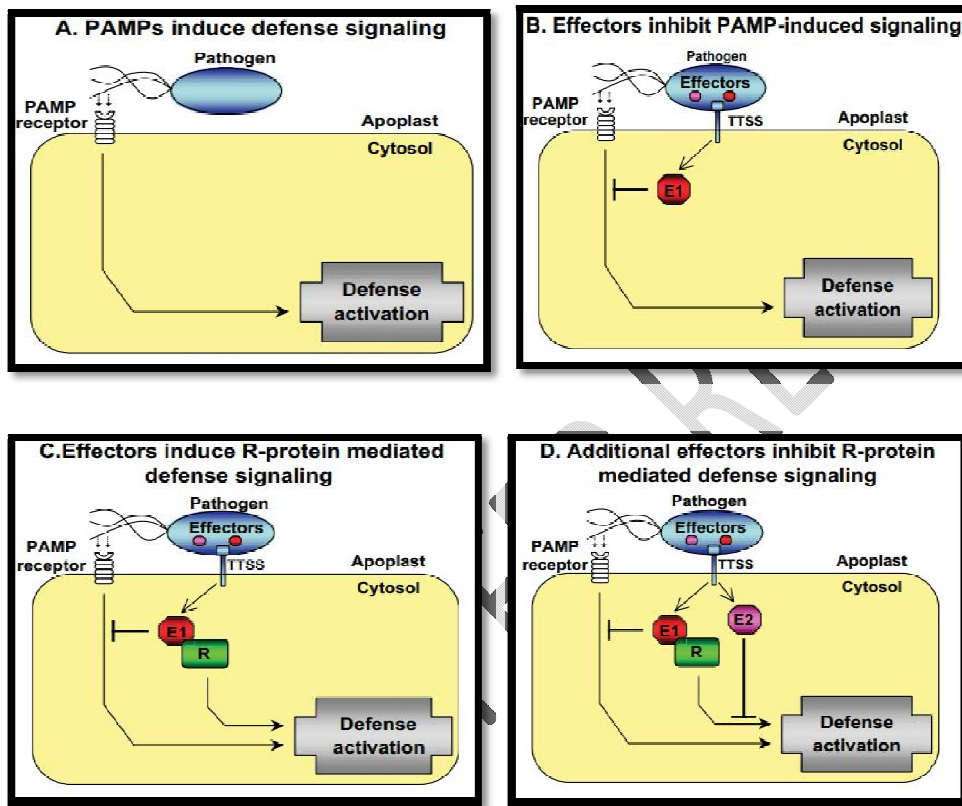


Fig. 1 Innate immune response in plants (a) PTI (b) ETS (c) ETI (d) ETS (da Cunha *et al.*, 2006) [19]

INNATE IMMUNITY IN RICE AGAINST *X. ORYZAE* PV. *ORYZAE*

Like other plants, rice also has evolved a two-layered innate immune system which includes PTI and ETI. R genes form the basis of this innate immune system of rice plant. To date 45 genes conferring BLB resistance have been reported in rice plant and these R genes are designated in a series from *Xa1* to *Xa45*[23]. Out of these, 17

genes viz., *xa5*, *xa8*, *xa13*, *xa15*, *xa19*, *xa20*, *xa24*, *xa25*, *xa26*, *xa28*, *xa31*, *xa32*, *xa33*, *xa34*, *xa41(t)*, *xa42* and *xa44(t)* [74] are recessive and remaining genes are dominant. Out of these 45 resistance genes in rice, *Xa1*, *Xa3/26*, *Xa5*, *Xa10*, *Xa13*, *Xa21*, *Xa23*, *Xa25*, *Xa4*, *Xa41* and *Xa27* have been cloned successfully and *Xa2*, *Xa4*, *Xa7*, *Xa22*, *Xa30*, *xa31*, *xa33*, *xa34*, *Xa38*, *Xa39*, *Xa40*, *xa42* have been fine mapped. All these resistance genes follow Mendelian pattern of gene inheritance and express resistance to a diverse strains of *X.oryzaepv.oryzae* [24]. These R genes identified in rice can be categorized into four groups based on the encoded proteins such as receptor kinases, SWEET genes, Executer genes and other genes [25].

RECEPTOR KINASES

PRRs, which can recognize diverse groups of PAMPs form the key component of the innate immune system of the plant. All the known plant PRRs falls into mainly two groups such as transmembrane receptor like kinases (RLKs) or transmembrane receptor like proteins (RLPs). There are over 1100 identified RLKs/RLPs in rice genome. RLKs contain an extracellular domain, a transmembrane domain, and an intracellular kinase domain, whereas RLPs lack the kinase domain. Leucine rich repeat receptor like kinases (LRR-RLKs) are the largest subfamily of plant RLKs [26].

The rice genome encodes more than 290 LRR-RLKs. *Xa21* and *Xa3/Xa26* are the well-characterized members as well as representatives of non-RD (non-arginine-aspartate) receptor kinases and both confer broad-spectrum resistance to *X.oryzaepv.oryzae* strains. *Xa4* gene is a wall associated kinase belonging to receptor kinase family [27].

Xa21 was the first cloned R gene in rice and it was originated from the wild rice species *Oryza longistaminata* [28]. These proteins have an extracellular LRR domain, a transmembrane motif, a juxta membrane domain and a cytoplasmic kinase domain [26]. *XA21*-mediated immunity is activated upon recognition of a 194-amino acid protein, Ax21 (activator of *XA21*-mediated innate immunity) [29]. *Xa21* confer broad-spectrum resistance to *X.oryzaepv.oryzae*. *Xa21* recognizes, a sulfated 17-amino acid peptide derived from the amino terminus of Ax21, a protein secreted through type 1 secretion system of *X. oryzaepv.oryzae* and thus activates PTI [30].

XA21 MEDIATED DEFENSE AGAINST X.ORYZAE PV. ORYZAE

Ax21 protein is a sulfated peptide secreted by *X. oryzaepv.oryzae* through the type I secretion system. This protein triggers XA21-mediated resistance by binding to the LRR domain of XA21. Because this peptide is conserved in many *Xanthomonas* species and even outside the *Xanthomonas* genus, it is considered as a PAMP molecule and XA21 as a PRR protein [29].

XA21 mediates a high level of PTI but with race specificity to *X. oryzaepv.oryzae*[31]. Several XA21 binding (XB) proteins have been reported to be involved in the rice defense response against *X. oryzaepv.oryzae*. XB24/ATPase promotes autophosphorylation of XA21 at ser/thr kinase (Ser 686, thr688, and Ser689) domains in the JM which maintains an inactive status for XA21 protein. Upon recognition of Ax21, XB24 dissociates from XA21, leading to its activation. XB3/E3 ubiquitin ligase is a substrate of XA21 kinase activity and is required for XA21-mediated resistance against BLB. XA21 transphosphorylates XB3, and activate MAPK cascade. Once the signal has been replayed, XA21 binds with the XB15 phosphates, which attenuates the immune response by dephosphorylation of aminoacids required for XA21 function. XB15/protein phosphatase 2C can dephosphorylate XA21, which results in the inactivation of XA21. The transcriptional regulator XB10/WRKY62 negatively regulates Xa21-mediated disease resistance. Cleavage of XA21 and translocalization of its kinase domain to the nucleus, where it interacts with WRKY62, is required for the Xa21-initiated defense response [32].

XA3/XA26 MEDIATED DEFENSE

Another LRR-RLK gene is Xa26 and it was originally identified from indica variety Minghui 63, an elite restorer line of hybrid rice in China. Further studies demonstrated that, Xa3 gene identified in a japonica variety WaseAaikoku 3, is the same gene as that of Xa26 gene. Xa3/Xa26 mediates a race-specific resistance to many strains of *X. oryzaepv.oryzae* but with a resistance spectrum different from Xa21 gene mediated defense. Xa3/Xa26 locus confers a durable resistance to strains of *X. oryzaepv.oryzae*. A gene-dosage effect exists in Xa3/Xa26-mediated defense. Higher expression level of Xa3/Xa26 confers a stronger, wider spectrum, and whole-growth-stage resistance to pathogen [33].

XA4 MEDIATED DEFENSE

Xa4 gene encodes a cell wall-associated kinase and confers a race-specific resistance to *X. oryzaepv.oryzae* at all stages of rice growth. Wall associated kinases (WAKs) are also a subfamily of RLKs that physically link the cell wall with the plasma membrane and transmit extracellular signals to the cytoplasm. In the early 1970s, *Xa4* was first introgressed into commercial rice varieties. It is one of the most widely employed resistance genes in breeding programmes in rice. Nearly all the *indica* hybrid rice cultivars in China carry the *Xa4* gene. *Xa4* prevents the invasion of *X. oryzaepv.oryzae* through reinforcing the plant cell wall. The *Xa4* mediated resistance is associated with the accumulation of two phytoalexins, viz., sakuranetin and momilactone A, which are likely to suppress *X. oryzaepv.oryzae* in plants. In addition to conferring durable resistance to *X. oryzaepv.oryzae*, *Xa4* also increases the mechanical strength of the culm and reduces the plant height slightly, and thus may enhance the lodging resistance. The multiple favourable agronomic traits related with *Xa4* explain why it is widely used in breeding programmes to develop resistant varieties [34].

Xa4 mediated defense operates through cell wall reinforcement, secretion of phytoalexins and by enhanced synthesis of jasmonic acid isoleucine (JA-Ile). Cellulose is mainly synthesized by cellulose synthase (CESA) family enzymes. Rice *CesA4*, *CesA7* and *CesA9* genes are responsible for the secondary cell wall synthesis. The expression of *CesA7* and *CesA9* genes was enhanced during *Xa4* mediated defense. Accumulation of phytoalexins such as sakuranetin and momilactone A, along with the increase in JA-Ile abundance occurs during *Xa4* mediated defense. *Xa4*-mediated resistance may require production of the bioactive JA-Ile, which in turn stimulates production of phytoalexins against *X. oryzaepv.oryzae* [35].

SWEET GENES

SWEET genes encode sugar transporter proteins, which are involved in exporting sugar across the membrane into the apoplast. During infection, these SWEET genes will be hijacked by transcription activator like effectors (TALE) of the pathogen, which is essential for growth and virulence of the pathogen. These SWEET proteins transport sugar across the membrane to the apoplast area which is essential for the survival of the pathogen. But any mutation in these genes confers

resistance to pathogen attack. Three recessive R genes such as *xa13*, *xa25* and *xa41* represent well studied examples of SWEET genes [36].

XA13 MEDIATED DEFENSE

Rice plants carrying recessive *xa13* exhibit specific resistance to *Xoo* strain PXO99. [37]. This resistant gene is widely used for breeding programmes in South Asian countries [32]. The dominant allele of the of this recessive gene, *Xa13* (also named Os8N3 and OsSWEET11), is a susceptibility gene specific to PXO99 strain, which secretes the TAL effector PthXo1 during infection [38]. When rice plants are infected with *X. oryzae* pv. *oryzae*, TAL effectors are secreted into the plant cell. The TAL effector *pthXo1* injected into the nucleus directly target the effector binding element (EBEs) ($UPT_{PthXo1}box$) in the promoter of dominant *Xa13* gene and activates its expression. *Xa13* encode a plasma membrane located sugar transporter gene, which is involved in the release of sugar into the apoplast and xylem, where the *X. oryzae* pv. *oryzae* resides, thus providing nutrition for the *Xoo*. *Xa13* can also interact with two plasma membrane located copper transporter-type proteins, COPT1 and COPT5, to promote removal of copper from the xylem vessels. Copper is an essential micronutrient for plants but it inhibits *Xoo* growth, and PXO99 is more sensitive to copper than other *Xoo* strains. When TAL effector activates SWEET genes, these copper transporter proteins also get activated. Copper transporters facilitate the removal of Cu from the xylem vessels and apoplast thereby increasing the virulence of the bacteria [39]. Resistance of rice plants carrying recessive *xa13* gene is due to the mutation of $UPT_{PthXo1}box$ in *xa13* promoter [40]. The effectors secreted cannot bind to the EBE which results in PXO99 strain being unable to induce recessive *xa13* expression. The copper level in the xylem vessels of rice plants carrying recessive *xa13* can inhibit PXO99 growth and plants have passive resistance to *Xoo*. But when the gene is recessive effectors cannot bind to EBE of target genes, thereby imparting resistance [36].

EXECUTER GENES

Xa27, *Xa10* and *Xa23* are the three executor genes in rice with multiple potential transmembrane domains functioning as a promoter trap, which are transcriptionally activated by TAL effectors and trigger defense responses. Dominant *Xa27* mediated race specific resistance depends on its transcriptional activation by *AvrXa27*, a TAL effector of *Xoo*. Pathogen secreting TAL effector *AvrXa27*, would be directed in to the nucleus, where it interacts with the up regulated by transcription factor (UPT) box in the promoter region of the target *Xa27* gene and activates its expression leading to the resistance [41].

OTHER GENES

In rice genome, 480 nucleotide-binding domain and leucine-rich repeat (NLR) genes have been revealed, but only a single one, *Xa1*, conferring resistance to *X. oryzae*pv. *oryzae*, was isolated. *Xa1* was isolated from the japonica cultivar Kogyoku and its expression was induced by bacterial infection and wounding. This gene confers race-specific resistance to *Xoo* strain T7174 [42].

The recessive gene *xa5* confers broad resistance spectrum to *X. oryzae*pv. *oryzae*. The *xa5* is a recessive allele of the gene *Xa5*, which encodes transcription factor IIA gamma subunit 5 (TFIIA γ 5). TFIIA is a basal transcription factor of eukaryotes which is essential for polymerase II-dependent transcription. TFIIA γ 5 is hijacked by TAL effectors by direct physical interaction with a transcription factor binding (TFB) region of TALE during infection. But the recessive gene *xa5* contains a mutation in the 39th residue, in which the valine (V) residue is replaced with glutamine (E). The TAL effector cannot interact with recessive genes, leading to resistance [32].

MANAGEMENT OF BACTERIAL LEAF BLIGHT

Use of antibiotics such as **streptocyclin** for controlling BLB is not eco-friendly. Host plant resistance is generally the most favourable tactic to control diseases due to economic and environmental reasons. Marker-assisted selection (MAS) and genetic transformation are the two major approaches for R gene deployment in plant breeding programs. Pyramiding R genes resistant to different races of the pathogen through marker-assisted breeding strategies, is a very effective way to achieve durable and broad-spectrum resistance, while employment of a single R gene and adaption of the pathogen often lead to resistance breakdown in a short period. Based

on reports, xa5, Xa7, xa13, Xa21 and Xa23 are more frequently used by rice breeders due to the comparatively broader spectra of resistance [43].

Table 1 Cultivars improved for bacterial blight resistance through marker assisted breeding

Combination of gene	Variety/genotype	Country	Reference
<i>xa5</i> , <i>xa13</i> and <i>XA21</i>	PR106	India	Singh <i>et al.</i> (2001) [44]
<i>xa5</i> , <i>xa13</i> and <i>XA21</i>	Samba Mahsuri	India	Sundaram <i>et al.</i> (2008) [45]
<i>XA21</i> , <i>xa13</i> and <i>sd-1</i>	Type 3 Basmati	India	Rajpurohit <i>et al.</i> (2011) [46]
<i>Xa4</i> , <i>xa5</i> , <i>xa13</i> and <i>XA21</i>	Mahsuri	India	Guvvala <i>et al.</i> (2013) [47]
<i>Xa38</i> , <i>xa13</i> and <i>XA21</i>	PAU 201	India	Sundaram <i>et al.</i> (2014) [48]
<i>XA21</i> , <i>xa13</i> , <i>xa5</i> and <i>Xa4</i>	Lalat and Tapaswini	India	Sundaram <i>et al.</i> (2014) [48]
<i>XA21</i> , <i>xa13</i> and <i>xa5</i>	Swarna and IR64	India	Sundaram <i>et al.</i> (2014) [48]
<i>XA21</i> and <i>xa13</i>	Pusa Basmati 1	India	Sundaram <i>et al.</i> (2014) [48]
<i>XA21</i>	Zhongyou 1176	China	Cao <i>et al.</i> (2003) [49]
<i>XA21</i>	Zhongyou 6	China	Cao <i>et al.</i> (2003) [49]
<i>Xa4</i> and <i>Xa5</i>	Angke	Indonesia	Sattari <i>et al.</i> (2014) [50]
<i>Xa5</i> , <i>Xa13</i> and <i>Xa21</i>	Improved Pusa Basmati-1	India	Gopalakrishnan <i>et al.</i> (2008) [51]
<i>Xa21</i> , <i>xa13</i> , and <i>xa5</i>	Ranidhan	India	Mohapatra <i>et al.</i>

			(2023)[52]
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Tainung 82 (TNG82) is one of the most popular japonica varieties in Taiwan due to its relatively high yield and grain quality, however, TNG82 is susceptible to bacterial blight (BB) disease. The most economical and eco-friendly way to control BB disease in japonica is through the utilization of varieties that are resistant to the disease. In order to improve TNG82's resistance to BB disease, five bacterial blight resistance genes (*Xa4*, *xa5*, *Xa7*, *xa13* and *Xa21*) were derived from a donor parent, IRBB66 and transferred into TNG82 via marker-assisted backcrossing breeding. These individuals displayed a high level of resistance against the bacterial leaf blight. The five identified bacterial leaf blight gene pyramided lines exhibited yield levels and other desirable agronomic traits, including grain quality and palatability, consistent with TNG82. Bacterial blight resistant lines possessing the five identified leaf blight genes exhibited not only higher levels of resistance to the disease, but also greater yield levels and grain quality (Yu-Chia *et al.*, 2020).

CONCLUSION

Bacterial leaf blight disease caused by *X. oryzae*pv.*oryzae* is one of the most widespread and economically important diseases of rice. It is a threat to rice production in tropical and temperate countries due to its high epidemic potential. *X. oryzae*pv.*oryzae* has been ranked as one among the top ten economically important bacterial plant pathogens worldwide. Bacterial leaf blight is very difficult to manage and none of the methods could completely manage the problem. One of the best ways to manage the disease is by boosting the innate resistance of the host plant against the pathogen. It can be achieved by incorporating resistant genes in cultivars. It has been proven that adoption of resistance is the most effective, economic, and environment-friendly strategy to avoid yield loss caused by bacterial leaf blight disease. During the co evolution of rice with pathogen, successful defense systems with the core of R genes have been evolved by rice to resist the disease. These R genes can be utilized in breeding programmes for developing resistant varieties. Broad spectrum and durable resistance can be achieved by pyramiding of more than one resistance genes by marker assisted breeding programmes.

DISCLAIMER (ARTIFICIAL INTELLIGENCE)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc) and text-to-image generators have been used during writing or editing of this manuscript.

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