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Review

Advances in the *Xoo*-rice pathosystem interaction and its exploitation in disease management

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Bacterial blight caused by *Xanthomonas oryzae* pv. *oryzae* (*Xoo*), is one of the devastating diseases of rice worldwide. The pathogen reported to cause 70% crop loss in some of the susceptible genotypes under disease favoring environments, viz., temperature ranging between 25 to 34°C and relative humidity more than 70%. In *Xoo*, about 245 genes govern the pathogenicity and host specificity. The hypersensitive response and pathogenicity (*hrp*) genes responsible for disease occurrence were clustered in the pathogenicity island of 31.3 Kb. The protein secreted through type three secretory system and type one secretory system mediates infection and establishment of the pathogen inside the host. However, elicitor molecules from *Xoo* triggered the resistant response in rice against the pathogen. An array of resistant genes (*R* genes) was known to be invoked by the host to combat the bacterial infection. To date, of the 45 *Xa* genes in rice, nine were cloned and characterized. The evolution of new races has made the task of developing resistant rice genotypes more challenging as it demands a comprehensive breeding strategy involving the best use of *R* genes from the existing gene pool. Thus, to combat the infection from the existing races and to slow down the emergence of new *Xoo* races, pyramiding two or more *R* genes was found to be effective against bacterial blight disease. In India, the successfully commercialized example includes the development of rice genotypes, viz., Improved Pusa Basmati-1, Improved Samba Mahsuri, PR106, Type 3 Basmati, and Mahsuri with selected *R* genes, viz., *xa5*, *Xa4*, *xa13* and *Xa21* against bacterial blight resistance. This review primarily portray *Xoo*-rice interactions and provides opportunities for its effective management through sustainable technologies.

Keywords. Bacterial blight; etiology; pathogenicity genes; pyramiding; *R* genes; rice; *Xa* genes

Abbreviations: *avr*, avirulence; EBE, effector binding elements; ETI, effector-triggered immunity; *hrp*, hypersensitive response and pathogenicity; LRR-RLK, leucine-rich repeat receptor-like kinase; MAMP, microbe-associated molecular patterns; NBS-LRR, nucleotide binding site leucine-rich repeat; *OryR*, orphan receptor; *OsSWEET*, *Oryza sativa* sugar will eventually be exported transporter gene; PAMP, pathogen associated molecular patterns; PIP, plant inducible promoter; PTI, PAMP triggered immunity; *R* genes, resistance genes; RTX, repeats in toxin; *S* genes, susceptible genes; T3E, type-three effectors; T3SS, type-three secretory system; TAL, transcription activator-like; $UPT_{AvrXa27}$, up-regulated by transcription activator-like effector of *AvrXa27*; *Xoo*, *Xanthomonas oryzae* pv. *oryzae*; *Xop*, *Xanthomonas* outer protein.

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1. Introduction

Rice being the third-largest food crop behind wheat and corn, is widely cultivated in the world. Of the 499.2 million tonnes of rice produced globally, 158.76 million tonnes of rice was produced in India during 2016–17 (Indiastat 2019; FAO 2017). Bacterial blight caused by *Xanthomonas oryzae* pv. *oryzae* (*Xoo*) is the most devastating and harmful disease that accounts for 40% of the rice crop loss due to pests and diseases (Shaheen *et al.* 2019; Syed-Ab-Rahman *et al.* 2020). The tropical climatic conditions of Asian, south-east Asian, and African countries favor the disease spread. In southeast Asia and India, disease outbreak is likely to occur during monsoon season (June–September) than other periods (Nino-Liu *et al.* 2006) and can cause infection at any growth stage of the plant (Jabeen *et al.* 2012).

In rice, bacterial blight infection resulted in reduced straw weight (43–60%), reduced number of filled grains (23–40%), less filled grain weight (24–45%), increased chaffiness (9.9–23%) (Adhikari *et al.* 1999) and the severity of loss depends on the crop stage. Nevertheless, the infection at the booting stage did not compromise the yield but lead to reduced grain quality resulting in a higher proportion of broken kernels (Basavaraj *et al.* 2010). Conversely, the loss due to kresek syndrome was very high (60–75%) and can even result in total crop loss (Rai 2007). Kresek syndrome was mainly observed in India, Philippines, Indonesia, and the infected crops recorded 56% grain chaffiness and 94% yield loss per hill (Srinivasan 1982; Nino-Liu *et al.* 2006). With the introduction of new high yielding varieties, the disease prominence has also increased (Ghasemie *et al.* 2008). Consequently, the yield loss recorded in India, Africa, Japan, Indonesia, and Pakistan were about 30–80%, 41–70%, 30–50%, 60–75%, and 57–75% respectively (Chaudhary *et al.* 2012; Awoderu *et al.* 1991; Sere *et al.* 2005; Khan *et al.* 2014; Arshad *et al.* 2015).

Given the above, the necessity to increase rice productivity *via* adoption of several high-yielding varieties, which are fertilizer responsive particularly to high doses of nitrogen and suitability to high-density planting is inevitable and therefore intensified the bacterial blight severity. Owing to its importance, international conference on rice bacterial leaf blight held in 2004 discussed the challenges and research progress made worldwide on the disease and its management strategies (Sundaram *et al.* 2014). In view of the gravity of the disease, the present review discusses the history, disease cycle, rice immunity, *Xoo*-rice pathosystem, and developments in management practices through molecular approaches.

2. History

Japanese farmers first reported the disease in the Fukuoka area (1884) and were considered to be caused by the physiological factor, acidic dew. In 1909, the actual causal organism, the bacterium isolated from the acidic dew drops (Ishiyama 1922) was later named as *Xanthomonas oryzae* pv. *oryzae* in 1990 (Swings *et al.* 1990). Henceforth bacterial blight occurrence has been reported from Africa, Bangladesh, Australia, Cambodia, South Korea, India, Indonesia, and many other countries (Singh *et al.* 2013; Wang *et al.* 2017; Naqvi 2019). In India, bacterial blight incidence was reported since 1951 and was wrongly considered as a nutritional deficiency. It was Srinivasan and his co-workers to first report the rice bacterial blight disease from Koloba district of Maharashtra during 1959 and was of minor importance until the epidemic outbreak in Shahabad (Bhojpur) district of Bihar in 1963 (Srivastava and Rao 1966). Now the disease has widely spread in almost all rice-growing states of India, viz., Andhra Pradesh, Assam, Delhi, Bihar, Goa, Karnataka, Tamil Nadu, Gujarat, West Bengal, Punjab, Haryana, Uttar Pradesh, Orissa, Maharashtra, Jammu, and Kashmir, Andaman and Nicobar Island (Mondal *et al.* 2014; Saha *et al.* 2015; Midha *et al.* 2017; Yugander *et al.* 2017) and reported in all the places where high-yielding varieties were grown (Ghasemie *et al.* 2008; Figure 1). India had recorded bacterial blight epidemics during 1963–64 in Bihar; 1965–66, 2010, 2013 in Andhra Pradesh; 1980–81 in Punjab, Haryana, Western Uttar Pradesh; 1998 in Kerala; 2014 in Tamil Nadu (Gnanamanickam *et al.* 1999; Laha *et al.* 2014; Yugander *et al.* 2017) and highly virulent *Xoo* pathotypes were isolated from north-western, eastern, north, north-eastern, southern and western India. Among the Indian states, Punjab recorded the most diverse *Xoo* pathotypes followed by Tamil Nadu, Andhra Pradesh; and isolates from Bihar were genetically distant to isolates from other states in India (Yugander *et al.* 2017). Disease survey data of past 34 years from several rice-growing regions of India revealed an increase in disease intensity with widespread geographical distribution of the pathogen, and the extent of yield loss was about 50% or more (Yugander *et al.* 2018).

Figure 1. Bacterial blight disease incidence across the state of India (red circles indicate the disease incidence in various locations across the Indian states). Source: CABI compendium records (CABI 2020).

3. Etiology

X. oryzae pv. *oryzae*, a non-spore-forming, rod-shaped, motile, single flagellate bacteria (Swings *et al.* 1990) caused bacterial blight in rice. The infection spread was severe during rainfall, high humidity, warm temperature (25–30°C), and deep-water system (Sharma *et al.* 2017). The pathogen enters the rice plant through the natural openings, viz., hydathodes present in the leaves or *via* wounds causing grey to white lesions in the leaf (Malik 2013; Cerutti *et al.* 2017). The bacterial inoculums in leaf guttation fluid swim into the intercellular space, multiply later enter the xylem vessel, and spreads through the vascular system systemically (Shen and Ronald 2002; Syed-Ab-Rahman *et al.* 2020). The bacterium utilizes chemotaxis for its directional movement to the plant openings (Kumar-Verma *et al.* 2018). Under severe disease conditions, bacterial exudates can be seen on the leaf surface (Nino-Liu *et al.* 2006). As the pathogen being Gram-negative, the virulence factor injected through the type three secretory system (TTSS) is necessary for pathogenesis (Furutani *et al.* 2009). The disease severity is determined by the virulence of the race, which manifests in the form of either blight (water-soaked yellow lesions with irregular margin) or kresek symptom (wilt phase-leaf roll, drop, turn yellow or grey and wither away) (Singh *et al.* 2013). During offseason, the bacteria survive on rice stubbles, weed hosts, wild rice, and for a shorter period in seed and soil (Mew *et al.* 1992).

4. Disease physiology/cycle

Infected rice seeds are the source of primary inoculum for the spread of bacterial blight besides soil, plant debris, and weeds. The rain/irrigation water, insects, and contact between healthy and infected seedlings act as the secondary source of inoculum and hasten the spread of the disease (Singh 1972). The bacterium can survive for 15–38 days in soil and water (Singh 1971a, b), 34– months in crop debris, and 78– months in seeds (Reddy 1983). Weeds associated with rice ecosystem, viz., *Oryza sativa* f. *spontanea*, *Cyperus rotundus*, *C. defformis*, *Leersia hexandra* act as a host for *Xoo* and harbor the bacteria in their roots (Singh 1972). The bacterium from the roots or infected stubbles or seeds reaches nursery beds and further spreads through irrigation water (Naqvi 2019). After reaching the rice seedlings, the bacteria accumulate on the root surfaces, which later penetrate through the root splits or wounds caused by insects or mechanical means and thereafter, multiply inside the plant by utilizing their metabolites (Dath and Deevadath 1983; Yasmin *et al.* 2017). Upon multiplication, the bacteria ooze out through root or leaf tip, causing further infection. The pathogen can re-enter quickly through stomata, hydathode, or wounds, and sometimes by insects, *i.e.*, rice bug *Leptocorsia acuta* (Mohiuddin *et al.* 1976). It causes linear yellow-colored stripe symptom with wavy outline along the leaf margins from tip to downward in seedling and early tillering stages; later the lesion expands and become greyish brown in later stages (Singh and Saksena 1968; Kini *et al.* 2017). Thus, wherever rice is grown as a single crop, the self-sown seeds; and in double-cropped areas, the Kharif crop will serve as primary inoculum for a possible epidemic (Singh 1972).

5. Rice innate immunity

Rice, like other crops, is threatened by several pests and diseases, and bacterial bight is one of the destructive diseases of rice (Jiang *et al.* 2020). Generally, in response to infection, plants confer different types of resistance, viz., qualitative resistance, and quantitative resistance. Qualitative resistance conferred by a single gene is pathogen race-specific, while

quantitative resistance mediated by several genes is pathogen race non-specific (Zhang and Wang 2013). When microbes invade the plant, the characteristic patterns from microbes, viz., flagellin, bacterial peptidoglycan, fungal chitin act as molecular signatures [pathogen associated molecular patterns (PAMP)/microbe associated molecular patterns (MAMP)]. These molecules enable the plant to identify the invaders using pattern recognition receptors on the plant cell surface and activate the plant defense response called PTI (PAMP/MAMP-triggered immunity; figure 2a). PAMP/MAMP recognition invokes a cascade of events such as; activation of mitogen-activated protein kinases, calcium signalling, callose deposition, phytoalexin production, burst in ROS and transcriptional reprogramming that ultimately leads to PTI (Malik *et al.* 2020). During the process of evolution, the pathogen has evolved effector molecules that are injected into host plants through type three secretory system (T3SS) to suppress the plant immune response. In turn, plants have evolved the second line of defense called effector-triggered immunity (ETI) by sensing the effector molecule through cytosolic nucleotide-binding site leucine-rich repeat (NBS-LRR) encoded by R-genes (figure 2b; Albert 2013; Liu *et al.* 2020). ETI evokes a strong and rapid hypersensitive response (HR) in resistant plants with systemic signalling (Jiang *et al.* 2020). On the other hand, PTI confers broad-spectrum resistance against pathogens, and the well studied molecular signature from *Xoo* is XA21 mediated resistance in rice. The MAMP, axY^{S}22 from *Xoo* was perceived by XA21, which is an LRR-RLK in rice plants and confer pathogen race non-specific resistance against rice bacterial blight and blast diseases (Song *et al.* 1995; Lee *et al.* 2009; Figure 2a). On the other hand, the transcription activator-like (TAL) effector, *AvrXa27* from *Xoo*, bind to the UPT$_{AvrXa27}$ box of *Xa27* promoter region in rice genome which encodes for an apoplast protein and triggers a hypersensitive response, a race-specific ETI (Gu *et al.* 2005; Figure 2b).

6. *Xanthomonas oryzae* pv. *oryzae* pathogenesis on rice

Advances in high throughput next-generation sequencing (NGS) and single-nucleotide polymorphism (SNP) technique had made genome-wide association studies (GWAS) towards understanding the

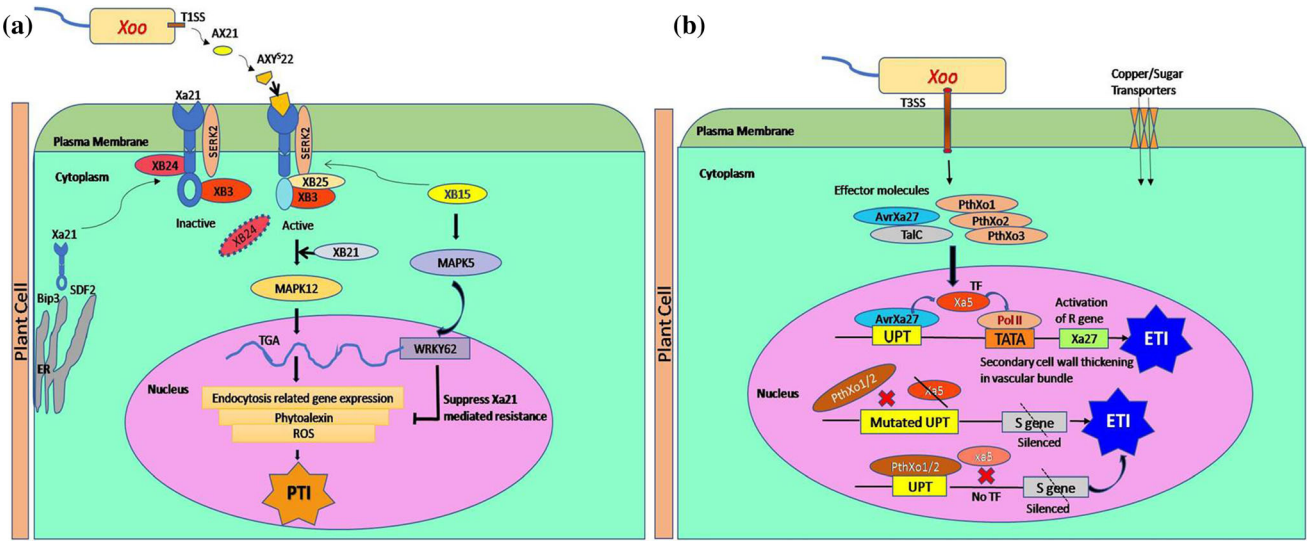

Figure 2. Rice immune response to *Xanthomonas oryzae* pv. *oryzae* infection. (**a**) Pathogen Triggered Immunity: RLK protein XA21 synthesized and processed at endoplasmic reticulum was transported to plasma membrane and association of an ATPase, XA21 binding protein 24 (XB24) to the juxtamembrane domain autophosphorylates XA21 to remain inactive. Upon recognition of sulfated Ax21from *Xoo*, the XB24 disassociates thereby activating the XA21 and the downstream defense signals viz., MAPKs, WRKY, XB3, XB21 gets triggered. MAPK5, WRKY62, XB15 (PP2C phosphatase) attenuates the XA21 mediated resistance while MAPK12, XB3 (E_3 ubiquitin ligase), XB21 (auxlin like protein) positively promotes resistance. (**b**) Effector Triggered Immunity: Binding of effector to the promoter of *R* gene activates *R* gene expression (*Xa27*) or mutated *S* gene upstream sequence prevents effector binding and *S* gene expression (*OsSWEET*) or *S* gene expression is silenced due to presence of recessive rice TFs (*xa5*) even after proper binding of effector, leads to resistance against *Xoo* infection).

genetic basis of disease resistance and pathogen evolution (Singh *et al.* 2018). Rice and *Xoo* pathosystem was widely used to study the host-plant interaction since the whole genome sequence of both rice and *Xoo* were available (Jha and Sonti 2009). More than 30 *Xoo* pathotypes were reported from all around the world and had evolved over a period of years, with varying levels of virulence (Gautham *et al.* 2014). Complete genome sequence analysis of 100 *Xoo* isolates revealed five clonal lineages (LI to LV), of which the lineage LI was predominant, and LIII marked the most virulent strains. The switching of *Xa* gene resistance breakdown had occurred twice in *Xoo* pathotypes of L1 and LIII (Midha *et al.* 2017). Twenty-two different *Xoo* pathotypes have been recorded from different geographical locations of India and were categorized into IXoPt-1 to IXoPt-22 chronologically based on the increasing order of virulence. The pathotypes from IXoPt-18 to 22 were highly virulent (Yugander *et al.* 2017), and their virulence level was governed by the virulence factor called TAL effectors that are injected into rice cells through T3SS (Sundaram *et al.* 2014). The *hrp* and effector gene expression was transcriptionally regulated by *hrpX*, which in turn was regulated by a response regulator gene, *hrpG* (Wengelnik *et al.* 1996). The 4941 kb genome of *Xoo* has six GC-rich insertion sequence (IS) elements and 245 species-specific genes involved in pathogenesis and host specificity. The signal molecule known as diffusible signal factor (DSF), a cis-unsaturated fatty acid molecule mediates quorum sensing (QS) system and directs expression of virulence factors in plant pathogenic *Xanthomonas* spp., which is an essential initial step during pathogenesis (Lee *et al.* 2005; Li *et al.* 2019). The orphan receptor *OryR* in *Xoo* can sense the small molecules from rice and regulate the virulence factors leading to infection (Sundaram *et al.* 2014). *Xoo* contains 14 copies of plant inducible promoter (PIP) box (TTCGN16TTCGn) in the promoter of *hrp* gene cluster (4), *avr* gene (1), *PopC* (1) and 8 copies were dispersed in their genome that regulates the formation of T3SS and injection of effector proteins into the rice cytoplasm. *Xoo* has a unique extracellular polysaccharide (EPS) production controlled by a *gum* operon (16 Kb) consisting of 13 genes, namely, *gum BCDEFGHIJKLMN*. EPS production is transcriptionally regulated by *rpf* (regulation of pathogenicity factor) genes, viz., *ABFCGDIE*, of which *rpfC and rpfI* has a major effect on virulence determination (Chatterjee and Sonti 2002; Patil *et al.* 2007). Genes for O-antigen synthesis, type IV fimbriae, glycine-rich outer membrane protein (*Xanthomonas* adhesin like protein XadA, XadB) and EPS production were present in *Xoo* genome and were found to play a significant role in pathogen adhesion and rice colonization (Ray *et al.* 2002; Lee *et al.* 2005; Das *et al.* 2009).

Rice immune responses and basal defenses initiated in response to *Xoo* attack can be suppressed by T3Es like *XopQ*, *XopR*, *XopN*, *XopX*, *XopZ* (Sinha *et al.* 2013; Sundaram *et al.* 2014; Gupta *et al.* 2015; Midha *et al.* 2017). Recently *XopU*, *XopV*, *XopP*, *XopG*, and *AvrBs2* effectors that can suppress *XopQ-XopX* induced immune responses in rice were identified (Deb *et al.* 2020). Extracellular enzymes, viz., cellulase (7 types), xylanase (4 types), xylosidases (6 types), protease (6 types), pectin esterase, pectate lyase (2 types), polygalacturonase and 1,4-β-cellobiosidase secreted through type II secretory system disintegrated rice cells and used as food by the pathogen (Xu and Gonzalez 1989; Ray *et al.* 2000; Jha *et al.* 2005; Aparna *et al.* 2009; Ryan *et al.* 2011). Similarly, reduced pathogenicity by xylanase mutant *Xoo* strains on rice plants suggested that mutation of genes involved in cell wall degrading enzymes (cellobiosidase, xylanase), T3E (*AvrBs2*) had affected the virulence and pathogenicity of *Xoo* (Kearney and Staskawicz 1990; Ray *et al.* 2000; Rajeshwari *et al.* 2005; Jha *et al.* 2007). Repeats in toxins, RTX (*rtxA* and *rtxC*), 3-methylthio-propionic acid, phenylacetic acid (PAA), and trans-3-methyl-thio-acrylic acid (MTAA) produced from *Xoo* will later lead to wilting and chlorosis in rice after colonization (Lee *et al.* 2005; Noda *et al.* 1989). Lee *et al.* (2017) reported that the transcription activators like effectors (TALE), EF-Tu, genes related to protein synthesis, and transposase were up-regulated in *Xoo* during rice-*Xoo* interaction *in planta*. Over-expression of *HrpF* (essential for T3SS), proteases (to cleave the host defense proteins), and *TonB* (involved in nutrient uptake) genes, among other genes, suggests their importance in pathogenesis.

7. Molecular mechanism of rice-*Xoo* interaction

The proteins responsible for pathogenesis from *Xoo*, secreted through type three secretory system (T3SS) was assembled by the structural proteins encoded by the hypersensitive response and pathogenicity (*hrp*) genes found in the pathogenicity island (PAI) of 31.3 Kb. Compared to *Xac* and *Xcc*, the *Xoo* has larger PAI, due to the insertion of four transposase gene (6 Kb) between *hpaB* and *hrpF* genes (Lee *et al.* 2005). Besides structural proteins, the *hrp* genes also encode the T3SS regulatory proteins called effector proteins

and extracellular accessory proteins (Choi *et al.* 2013). Type three effectors (T3Es) play an essential role in disease development. Effectors can be of TAL effectors or non-TAL effectors (Midha *et al.* 2017). TAL effector has a nuclear localization signal at C-terminus, a transcriptional activation domain, and a central repeat domain of 33–35 amino acids (Xu *et al.* 2019). The repeat domain recognizes the specific sequence in the promoter region of the host genome called effector binding elements (EBE) and induces the expression of susceptibility (*S*) gene that favor the disease development (Boch *et al.* 2014). In rice, sugar transporter genes, viz., *OsSWEET11*, *OsSWEET13*, and *OsSWEET14* were induced by the effectors *PthXo1*, *PthXo2*, *PthXo3*, *TalC*, *Tal5*, *AvrXa7* respectively to favor the pathogen growth and multiplication (Xu *et al.* 2019).

In due course of interaction, rice plants had evolved *R* gene-mediated resistance, which includes recessive *S* gene, TAL effector, or non-TAL effector based dominant *R* genes (Zhang *et al.* 2015). Specific interaction between *R* genes and its cognate *Avr* gene products evoke defense response against the pathogen (Praveen *et al.* 2019). An open database, RiceMetaSysB furnish information on bacterial blight responsive genes based on global microarray data from rice (Sureshkumar *et al.* 2019). Till now, 45 *R* genes involved in bacterial blight resistance of rice have been discovered. Of which 18 were recessive, and nine were cloned (Kesh and Kaushik 2020). The list of *R* genes deployed by rice and their characteristics are given in table 1. Based on the proteins encoded, the *R* genes were classified into four groups, specifically receptor-like kinase (RLK) genes, sugar will eventually be exported transporter (SWEET) genes, executer (E) genes and other genes (Jiang *et al.* 2020).

7.1 *RLK genes*

The RLK genes (*Xa21*, *Xa3*/*Xa26*, *Xa4*) encode proteins of LRR receptor kinase predicted structure and could trigger a strong race-specific resistance. RLK encompasses an extracellular domain, transmembrane domain, and an intracellular kinase domain. The widely studied RLK gene, *Xa21* was the first cloned *R* gene from *Oryza longistaminata* and was reported to be developmentally regulated acquiring full resistance adult stage (Song *et al.* 1995; Wang *et al.* 1996). The tyrosine-sulfated *RaxX* secreted through type I secretory system acts as a ligand and directly binds to the XA21 extracellular domain with high affinity (Luu *et al.* 2019). Upon pathogen recognition, disassociation of XA21 kinase from XB24 and dephosphorylation of XA21 induce the XA21 mediated resistance (Park *et al.* 2008; Chen *et al.* 2010; Figure 2a). The second LRR-RLK gene, *Xa3* from *japonica* variety, was found identical to *Xa26* gene, identified from *indica* variety. *Xa3*/*Xa26* encodes an extracellular domain comprising 26 imperfect LRRs made of 24 amino acids each, a membrane-spanning region, and a serine/threonine kinase domain in the cytoplasm (Sun *et al.* 2004). Recognition of the cognate avirulence gene *AvrXa3 via* Xa3/XA26 mediate OsSERK2 and OSTPI1.1 interaction leading to resistance both in juvenile and adult plants constitutively (Liu *et al.* 2018; table 1). In China, nearly all *indica* hybrid rice cultivars contain *Xa4*, a cell wall-associated kinase encoding a protein and confers race-specific resistance at all stages of growth through cell wall reinforcement and phytoalexin (sakuranetin and momilactone A) accumulation (Leach *et al.* 2001; Sun *et al.* 2003; Hu *et al.* 2017). The *Xa4* corresponding *Avr* gene and the mechanism of *Xa3*/*Xa26* mediated disease resistance remain undiscovered (Jiang *et al.* 2020). A novel rice receptor *WALL-ASSOCIATED KINASE-LIKE 21* (*OsWAKL21.2*), identified by transcription analysis was found to be up-regulated upon *Xoo* infection and confer disease resistance by activation of defense-related genes (Malukani *et al.* 2019).

7.2 *SWEET genes*

SWEET genes code for a unique family of sugar efflux transporters actively involved in various biological processes (Chen *et al.* 2012; Streubel *et al.* 2013). Rice genome contains more than 20 SWEET genes reported to have an essential role in pollen nutrition, seed filling, senescence, and plant-pathogen interaction (Yang *et al.* 2006; Antony *et al.* 2010; Chen *et al.* 2010). The three genes, viz., *Xa13*, *Xa25*, and *Xa41(t)* encode clade III SWEET proteins that export sugar into the apoplast, and *Xoo* TAL effectors had hijacked the SWEET genes for its growth and virulence inside the host (Eom *et al.* 2015). The *Avr* genes that target SWEET genes are listed in table 1, and those TALEs were considered important due to their strong virulence effect (Oliva *et al.* 2019). Recessive genes *Xa13*, *Xa25*, and *Xa41(t)* with effector binding elements mutation were identified in rice varieties against *OsSWEET11*, *OsSWEET13*, and *OsSWEET14* susceptible genes, respectively and confer resistance against different *Xoo* races (Romer *et al.* 2010; Hutin *et al.* 2015; Xu *et al.* 2019;

Table 1. Bacterial blight resistant *Xa* genes identified in rice

R-gene	Nature	Chromosome	Marker linked	Function	Identified	Cognate *Avr* genes
Xa1	Dominant, Cloned	4	XNpb235,XNpb264, C600, $U08_{750}$	Resistant against Japanese race I NBS-LRR class	Kogyoku and Java14 cultivars	*PthXo1*/ *Tal4*/ *Tal9d*
Xa2	Dominant	4	HZR950-5, HZR970-4 XNpb235, XNbp197	Resistant against Japanese race II	Rantai Emas2	–
Xa3/ Xa26	Dominant, Cloned	11	XNpb181, XNbp78	Resistant against bacteria at seedling and adult stage	WaseAikoku	*AvrXa3*
Xa4	Dominant	11	RM224, RM144 XNpb181, XNbp78	Codes for wall associated kinase (WAK) protein	TKM6, IR20, IR22, IR72 and IR1529-680-3	–
xa5	Recessive, Cloned	5	RM13, RG136, RG556	Provides broad spectrum resistance by inhibiting TALE function	IR1545-284 and RP291-7DV85, DV86 and DZ78	*AvrXa5*/ *PthXo7*
Xa6/ xa3	Dominant	11	-	Resistant to Philippine race 1	Zenith, Malagkit Sungsong	–
Xa7	Dominant	6	GDSSR02, RM20593	Resistant to Philippine races	DV85, DV86, DZ78	–
xa8	Recessive	7	RM21044, RM21045	Resistant to Philippine races	P1231129	–
xa9	Recessive	11	-	Resistant to Philippine races	Khao Lay Nhay, Sateng	–
Xa10	Dominant, Cloned	11	M491, M419, $OP07_{2000}$	126aa that mediate Ca^{2+} depletion and initiate PCD	Cas209	*AvrXa10*
Xa11	Dominant	3	RM347, KUX11	Resistant to Japanese races IB, II, IIIA,V	IR944-102-2-3	–
Xa12	Dominant	4	–	Resistant to Indonesian race V	Kogyoku and Java14	–
xa13	Recessive, Cloned	8	RG136,R2027,RG556, RM122,RZ390	Encodes a novel plasma membrane protein involved in sugar transport	BJ1	*PthXo1*
Xa14	Dominant	4	HZR970-8, HZR988-1	Resistant to Philippine race 5	Taichung Native 1	–
xa15	Recessive	–	–	Resistant to Japanese races	M41 mutant	–
Xa16	Dominant	–	–	Resistant to Japanese races	Tetep	–
Xa17	Dominant	–	–	Resistant to Japanese races	Asominori	–
Xa18	Dominant	–	–	Resistant to Burmese races	Toyonishiki, Milyang 23, IR24	–
xa19	Recessive	–	–	Resistant to Japanese races	XM5	–
xa20	Recessive	–	–	Resistant to Japanese races	XM6	–
Xa21	Dominant Cloned	11	Pta818 Pta248 RG103	Broad spectrum resistance against all *Xoo* races in India and 6 from Philippines RLK class	*O. longistaminata*	*RaxX*
Xa22	Dominant	11	R1506	Resistant to Chinese races	Zhachanglong	–
Xa23(t)	Dominant Cloned	11	Lj138, A83B4	113aa with HR activity	*O. rufipogon*	*AvrXa23*

Table 1 (continued)

R-gene	Nature	Chromosome	Marker linked	Function	Identified	Cognate *Avr* genes
xa24(t)	Recessive	2	RM14222 RM14226	Resistant to Philippine and Chinese races	DV86	–
xa25(t)	Recessive Cloned	12	–	Race specific resistance Mutation in UPT $_{PthXo2}$ box prevent *S* gene (*OsSWEET13*) regulation by TAL	Minghui63, HX3	*PthXo2*
xa26(t)	Recessive	–	–	RLK class	Nep Bha Bhong To	–
Xa27(t)	Dominant Cloned	6	–	113aa cytoplasmic membrane protein	Arai Raj	*AvrXa27*
xa28(t)	Recessive	–	–	Resistant to Philippine race 2	Lota Sail	–
Xa29(t)	Dominant	1	–	Resistant to Chinese races	*O. ofiicinalis*	–
Xa30(t)	Dominant	11	RM1341, V88, C189,O3STS	Resistant to Indonesian races	*O. rufipogon*	–
xa31(t)	Recessive	4	G235, C600	Resistant to Chinese races	ZCL	–
Xa32(t)	Dominant	11	RM2064, RM6293	Resistant to Philippine races	*Oryza australiensis*	–
Xa33(t)	Dominant	7	RMWR7.6	Resistant to Thai races	*O. nivara* IRGC 105710	–
xa33(t)	Recessive	6	RM21004 RM21177	Resistant to Thai races	Ba7	–
xa34(t)	Recessive	1	–	–	BG122	–
Xa35(t)	Dominant	11	RM144, RM6293, RM7654	Resistant to Philippine races	*O. minuta*	–
Xa36(t)	Dominant	11	RM224, RM2136	Resistant to Philippine races	C4059	–
Xa38	Dominant	4	os04g53050-1	Broad spectrum resistance against Indian races and NBS-LRR type	*O. nivara*	–
Xa39	Dominant	11	RM21, RM206 RM26985, DM13	Resistant to 7 Chinese pathotype and 14 Philippines races	FF329 introgression line	–
Xa40(t)	Dominant	11	RM27320 ID55.WA18-5	Resistant against all Korean races	IR65482-7-216-1-2	–
xa41(t)	Recessive	–	–	EBE mutational allele of *OsSWEET14*	African wild cultivated species of *O. Barthii* and *O. glaberrima*	*Tal5*/ *TalC*/ *AvrXa7*/ *PthXo3*
xa42	Recessive	3	KGC3_16.1, RM15189	Resistant against all Japanese races	XM14 mutant (Developed from IR24 by mutational breeding)	–
Xa43(t)	Dominant	11	IBb27os11_14, S_BB11.ssr_9	Broad spectrum resistance to Korean races	Colombia XXI(P8)	–
xa44(t)	Recessive	11	–	Resistant against all Korean races	IR73571-3B-11-3-K3 (P6)	–
xa45(t)	Recessive	8	C8.26737175 C8.26818765 Os0842410	Resistant against *Xoo* pathotypes from North India	*O. glaberrima* accession IRGC 102600B	–

*Source: Chukwu *et al.* 2019; Kim and Reinke 2019; Jiang *et al.* 2020; An *et al.* 2020; Kesh and Kaushik 2020

Figure 2b). Later it was found that XA13 protein cooperates with COPT1 and COPT5 transporters in copper redistribution, and TAL effectors of *Xoo* employed XA13, copper transporters to remove copper from xylem vessels favoring pathogen multiplication and spread systemically (Yuan *et al.* 2010). The *xa13*, *xa25* conferred race-specific resistance, while *xa41(t)* showed broad-spectrum resistance against *Xoo* isolates from Asia and Africa (Jiang *et al.* 2020). The *OsSWEET13*, *OsSWEET14* promoter polymorphism analysis in rice germplasm revealed novel variation in EBE that can be exploited for resistant cultivar development (Zaka *et al.* 2018).

7.3 *Executor genes*

The transcriptional activation of executor *R* genes by TAL effectors from *Xoo* triggers a defense response in rice (Wang *et al.* 2015). Three executor *R* genes *Xa27*, *Xa10*, *Xa23* reported from rice were activated by the following TAL effectors *AvrXa27*, *AvrXa10*, *AvrXa23* respectively (table 1) conferring broad-spectrum resistance against *Xoo* strains from different countries (Wu *et al.* 2008; Tian *et al.* 2014; Wang *et al.* 2014). The *Xa27*, *Xa23*, and *Xa10* were dominant *R* genes, and binding of *Xoo* TAL effectors to *R* gene promoter, trigger a hypersensitive response (Xu *et al.* 2017). Following the elicitation of *Xa27* gene (originated from *O. minuta*) by *AvrXa27* resulted in the induction and secretion of protein to apoplast based on the signal-anchor-like sequence at N-terminal leading to inhibition of *Xoo* and secondary cell wall thickening in vascular bundles (Wu *et al.* 2008; Figure 2b). Similarly, the XA10 protein forms hexamers, located in rice cell ER membrane leading to ER disruption, cellular Ca^{2+} homeostasis, and programmed cell death (Tian *et al.* 2014). The *AvrXa23* was prevalent in *Xoo* strains, and expression of XA23 protein triggers a hypersensitive response and bacterial blight resistance constitutively (Wang *et al.* 2014; Wang *et al.* 2015).

7.4 *Other R genes*

In rice-*Xoo* interaction, the NBS-LRR (NLR) genes from rice were hijacked by *Xoo* TAL effectors by physical binding and regulate the host *S* or *R* gene transcription (Jiang *et al.* 2020). The first isolated NLR gene was *Xa1* from *japonica* cultivar and conferred resistance by recognition of TALEs. In order to suppress this immunity during evolution, the *Xoo* produced truncated interfering TALEs (iTALEs) to block TALE recognition (Ji *et al.* 2016). The most widely used recessive *R* gene in bacterial blight resistant rice development is *xa5* that confers broad-spectrum resistance to *Xoo* and is a natural allele of *Xa5* for the TFIIA gamma subunit 5 (TFIIAγ5) with the valine (V) mutated to glutamine (E) at 39th residue (V39E) (Jiang *et al.* 2006). Besides the regulation of *S* genes (*OsSWEET11*, *OsSWEET14*), *Xa5* induce the TALE based activation of *R* genes (*Xa27*, *Xa23*) (Figure 2b; Yuan *et al.* 2016). Another *R* gene with durable resistance is *Xa7*, effective even at high temperatures, whereas other *R* genes are not (Webb *et al.* 2010).

8. Molecular breeding for developing bacterial blight-resistant rice

With the dawn of the molecular biology era, the genes involved in host-pathogen (rice-*Xoo*) interaction revealed a few *R* genes deployed by rice plants for resistance against *Xoo* (table 1). Among these 45 genes, 3 recessive genes and 6 dominant genes, viz., *Xa1*, *Xa3/Xa26*, *xa5*, *Xa10*, *xa13*, *Xa21*, *Xa23*, *xa25(t)*, *Xa27(t)* were cloned and characterized (Kesh and Kaushik 2020). The *R* gene containing QTLs from wild/resistant cultivars were introgressed into the cultivated rice lines employing marker-assisted breeding (Rao *et al.* 2002). Introgression of bacterial blight-resistant genes from non-Basmati cultivars into Basmati cultivar resulted in the loss of aroma and grain quality (Singh *et al.* 2011). This has been overcome by screening, identification, and introgression of bacterial blight-resistant genes from Basmati landraces into Basmati cultivars (Ullah *et al.* 2012). Moreover, it was found that the rice cultivars harboring a single *R* gene was found to be susceptible in due course owing to pathogen evolution and prevalence of more *Xoo* races (Singh *et al.* 2015; Saha *et al.* 2015). The predominant *Xoo* pathotype IXoPt-19 (except for *xa13*) and IXoPt-22 were virulent on rice lines harboring a single bacterial blight resistance gene (Yugander *et al.* 2017). Furthermore, compared to African isolates, iTALE genes (suppress TALE recognition) are common in Asian *Xoo* isolates. Hitherto, no single *Xa* gene was reported to be effective against all Indian pathotypes (Saha *et al.* 2015). This was overcome by stacking two or more genes into the same rice cultivar by marker-assisted pyramiding (Rajpurohit *et al.* 2010). The development of molecular markers linked to *Xa* genes facilitated the successful pyramiding of several *Xa*

genes into single rice cultivar (table 1, Chukwu *et al.* 2019). In 1997, Huang and his co-workers successfully pyramided two dominant (*Xa4*, *Xa21*) and two recessive genes (*xa5*, *xa13*) into the cultivated rice variety IR24 and found the pyramided lines (IRBB60) had a higher level of resistance against the six races of *Xoo* compared to single or two gene containing lines (Huang *et al.* 1997). Likewise, bacterial blight resistance genes (*Xa21*, *xa13*, *xa5*) from SS1113 donor line was pyramided into an elite *indica* rice variety Samba Mahsuri (BPT5204) by backcross breeding programme and the three-gene pyramided line displayed high level of resistance and yield besides retaining the grain and cooking quality of Samba Mahsuri (Sundaram *et al.* 2008). Similarly, *xa5*, *xa13*, and *Xa21* genes were pyramided into two popular high yielding bacterial blight susceptible *indica* rice cultivars, viz., ADT43, ASD16, and the pyramided lines exhibited impressive resistance against two predominant south Indian *Xoo* isolates (Perumalsamy *et al.* 2010; Bharani *et al.* 2010). Besides bacterial blight resistance genes, pyramiding blast and phosphorus starvation tolerance (*OsPSTOL1*) gene into ADT43 and ASD16 cultivars trade-off the disease resistance and phosphorus uptake leading to varietal improvement (Chithrameenal *et al.* 2018). Furthermore, combination of *Xa+Xa21*, *xa5+Xa21*, *Xa4+xa5+Xa21*, *xa5+xa13+Xa21*, *xa5+Xa4*, *Xa4+Xa7*, *xa13+Xa21*, *Xa4+xa5+xa13+Xa21*, *Xa21+Xa33*, *Xa21+xa13* containing rice lines were developed (Sundaram *et al.* 2008; Ramalingam *et al.* 2017; Rao *et al.* 2017; Gao *et al.* 2018; Balachiranjeevi *et al.* 2018; Chukwu *et al.* 2019; Raina *et al.* 2019). The stacking of recessive *xa5* genes along with *Xa10*/*Xa23*/*Xa27* showed attenuated resistance to bacterial blight compared to the lines having these genes individually. It has been demonstrated that *Xa5* is the transcriptional activator of *Xa*10/*Xa23*/*Xa27* genes and recessive *xa5* in pyramided lines resulted in inactivation or loss of *Xa10*/*Xa23*/*Xa27* function leading to disease susceptibility (Gao *et al.* 2018).

The *Xa* genes pyramided popular rice varieties; namely, Improved Pusa Basmati-1 (*xa13+Xa21*), Improved Samba Mahsuri (*xa5+xa13+Xa21*), PR106 (*xa5+xa13+Xa21*), Type 3 Basmati (*xa13+Xa21+sd-1*) and Mahsuri (*Xa4+xa5+xa13+Xa21*) were released for commercial cultivation in India against bacterial blight resistance (Chukwu *et al.* 2019; Kesh and Kaushik 2020). Due to the appearance of new virulent *Xoo* races, the durability of resistance in Improved Pusa Basmati-1 and Improved Samba Mahsuri is threatened. Hence continuous efforts to identify novel *Xa* genes and pyramid those genes into the pyramided lines are being made. Recently, a novel NBS-LRR type gene *Xa38* was pyramided into the Improved Samba Mahsuri background, and the pyramided lines had durable resistance to *Xoo* races that were virulent on Improved Samba Mahsuri (Yugander *et al.* 2018). Similarly, a combination of *Xa21* and *Xa33* in the DRR17B line exhibited resistance comparable to the three *Xa* gene pyramided line Improved Samba Mahsuri (Balachiranjeevi *et al.* 2018). Likewise, *Xa33* and *Xa38* were deployed along with *xa13+Xa21* into Basmati cultivars for diversified disease resistance. Recently it was demonstrated that pyramiding the 5 *Xa* genes (*Xa4*, *xa5*, *Xa7*, *xa13*, and *Xa21*) into the *japonica* cultivar (TNG82) contributed a higher level of resistance against bacterial blight disease, besides improving the grain yield, quality, and palatability (Hsu *et al.* 2020). Forbye, the results of Sakthivel and his co-workers suggest the rice genetic background determines the effectiveness of pyramided gene expression (Sakthivel *et al.* 2017). Apart from molecular breeding, the *Xa21* gene was transformed into 5 Chinese rice varieties through transgenics, and the transformed plant showed a broad spectrum of resistance against bacterial blight (Zhai *et al.* 2000; Lifen *et al.* 2013). In another study, the exogenous application of salicylic acid, jasmonic acid, and ethylene in *OsPR1b* over-expressing transgenic rice lines defended the bacterial blight disease by up-regulation of PR1b proteins (Luan and Zhou 2015). Correspondingly, transgenic rice lines over-expressing *MoSM1* showed improved resistance against *Xoo* and *Magnaporthe oryzae* by regulating JA/SA signalling pathways (Hong *et al.* 2017). Amidst the genome editing tools, viz., ZFNs (Zinc finger nucleases), MegaN (mega nuclease), TALENs (transcription activator-like effector nucleases) and CRISPR/Cas9 (clustered regularly interspaced short palindromic repeat/CRISPRs-associated protein 9); CRISPR/Cas9 system is considered the most important because of its high simplicity, efficiency and seemly for rice genome editing (Zhang *et al.* 2013; Jiang *et al.* 2013). However, recently TALENs and CRISPR/Cas9 were efficiently exercised to disrupt the effector binding elements in the rice genome to prevent the susceptible gene expression that favors disease development (Singh *et al.* 2018). The role of *OsSWEET14* in disease development was studied using TALEN technology (Blanvillain-Baufume *et al.* 2017). Disruption of *PthXo3*, *AvrXa7* and *PthXo3* binding elements in the promoter of *OsSWEET14/OsSWEET13* gene employing TALEN displayed strong bacterial blight resistance in rice (Li *et al.* 2012; Zhou *et al.* 2015; An *et al.* 2020). Likewise, CRISPR-Cas9 based disruption of *PthXo2* binding

elements of *OsSWEET13* gene in *indica* rice cultivars conferred bacterial blight resistance (Zhou *et al.* 2015). Furthermore, targeted disruption of EBEs in *OsSWEET14* and *OsSWEET11* promoters using this system showed decreased bacterial leaf blight symptoms with normal plant development (Jiang *et al.* 2013). Five mutations in the promoter of SWEET genes (*OsSWEET11*, *OsSWEET13*, *OsSWEET14)* conferred broad-spectrum resistance to Asian, and African strains. The introduction of larger polymorphisms in the effector binding elements of SWEET genes is preferred than an SNP. Simultaneously editing all EBEs are possible using multiplex targeting CRISPR–Cas9 system, but the limitation in possible edits and editing complex delivery approach needs to be addressed (Oliva *et al.* 2019). CRISPER-Cas9 based genome editing can be successfully used to create a wide range of resistance against different *Xoo* strains (Xu *et al.* 2019). Recently an Asian *Xoo* strain capable of colonizing without induction of clade III SWEET gene was reported (Carpenter *et al.* 2018). The *Xoo* populations continuously evolve with some degree of selection to target alternative *S* genes and break the host resistance. Hence it is important to continuously monitor the *Xoo* population by sequencing the complete genome to develop varieties with a broad spectrum of disease resistance (Mondal *et al.* 2014; Midha *et al.* 2017). Eom and his co-workers developed SWEET kit towards the visualization of SWEET protein accumulation to identify the bacterial blight-resistant lines for farmers, and the group can extend the use of this kit to track newly emerging *Xoo* variants in future (Eom *et al.* 2019). A recent study on *Xanthomonas* effectors suggests their complex interplay in the suppression of both PTI and ETI to promote virulence on rice (Deb *et al.* 2020). With the advances in NGS, the genomic resource of the *Xoo* isolates were made available and is invaluable in *Xoo* surveillance through the development of PCR based diagnostic tools (Midha *et al.* 2017). A clear understanding of the emergence of new virulent *Xoo* strains by continuous monitoring; exploration of new *R* genes and engineering promoter variants to suppress the evolving new TAL effectors will improve the bacterial blight resistance in rice.

9. Conclusion and the way forward

Rice bacterial blight, the deadly bacterial disease, was reported to cause significant crop loss in rice-growing countries, especially in tropics. Cognizance on the molecular basis of rice-*Xoo* interaction succored in bacterial blight-resistant rice lines development. Identification of *Xa* genes and associated markers has made the development of bacterial blight-resistant rice varieties easier. Due to the evolution of more virulent *Xoo* strains, it is mandatory to identify novel *Xa* genes with durability and to stack suitable combinations of *Xa* genes in local cultivars for utility. Further insight into the molecular mechanism behind the pathogen infection process and defense response in rice plants was derived from the complete genome information of rice and *Xoo* through genome wide association studies. The pathogen virulence relies strongly on bacterial proteins that function as transcriptional activators inside the plant cell. Gene editing technologies, viz., TALEN, and CRISPR-Cas9 had proved that precise editing of *Xoo* effector binding sequence and susceptible gene in rice genome reduced the disease incidence. Furthermore, advances in high throughput next-generation sequencing devised surveillance of newer virulent *Xoo* strains and development of PCR based diagnostic kits *ensue*. With the functions known for several *R* genes and its cognate *Avr* gene synergized by the advancements in molecular breeding, genetic engineering and gene editing technologies, development of rice genotypes to confer robust resistance against *X. oryzae* pv. *oryzae* appears promising.

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