Chapter 10

Genetics and Genomics of Bacterial Blight Resistance in Rice

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Additional information is available at the end of the chapter

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Abstract

Rice is an important food crop for half the world's population and has been in cultivation for over 10,000 years. During the last few decades, rice has evolved intricate relationships with associated pathogens and pests, bacterial blight (BB) being one of the most important among them. Utilization of resistant varieties with agricultural management practices is a more effective way to control BB. Of the 42 different resistance (*R*) genes identified to confer BB resistance, 9 have been isolated and cloned, whereas a few of the avirulence genes and a large number of candidate pathogenicity genes have been isolated from *Xanthomonas oryzae* pv. *oryzae*. The complete genome sequences of two different rice subspecies *japonica* and *indica* and three different races of BB pathogen are available. Therefore, the interaction between rice-*Xoo* could be deciphered and pave a way to study the molecular aspects of bacterial pathogenesis and host counter measures like innate immunity and R gene–mediated immunity. Although several of the type III effectors of *Xoo* have been characterized and the host targets of a few of them identified, a relatively large number of candidate effectors remain to be studied and their functional analysis may provide key for developing broad spectrum and durable resistance to BB.

Keywords: *Xanthomonas oryzae* pv. *oryzae*, *Oryza sativa*, Genome structure, *Xa* genes, mapping

1. Introduction

Rice (*Oryza sativa* L.) is a staple food for a large part of the world's human population, especially in South and Southeast Asia and tropical Latin America, making it the second most consumed cereal grain. It accounts for 35–60% of the calories consumed by more than 3 billion Asians. To meet the growing demand of nearly 5.0 billion consumers, rice-growing countries

will have to produce 40% more rice by 2030 [1]. However, its production is being reduced severely by several rice diseases. Bacterial blight (BB) caused by *Xanthomonas oryzae* pv. *oryzae* (*Xoo*) is one of the major foliar diseases, causing yield losses by 20–30%. It was first reported by farmers in 1884 in the Southern region of Japan [2]. It is a vascular disease resulting in tannish grey to white lesions along the veins. The BB pathogen invades the host through natural openings in leaves, including hydathodes or wounds, and colonizes the xylem vessels. Under field conditions, symptoms are onset at the tillering stage and disease incidence increases with plant growth, which is maximum at the flowering stage. The kresek, a severe form of disease, occurs at the seedling stage resulting in a partial or total crop failure.

The incidence of BB has been widely reported in all rice-growing regions worldwide except North America [3]. The high degree of pathogenic variation in *Xoo* due to the evolution of new pathotypes often causes the breakdown of resistance. Genome structure of *Xoo* revealed the presence of effector (*avr*) genes and insertion sequences, which may be playing a major role in generating a high degree of genetic diversity and race differentiation [4]. However, with the evolution of new races, broader and more comprehensive resistance must be pursued in order to prevent super-races of *Xoo*, which can overcome all genetic sources of resistance, from becoming prevalent. Therefore, it is of immense interest to diversify the germplasm, scouting of resistance genes from wild species and pyramiding two or more effective resistance genes in developing rice cultivars with durable BB resistance to *Xoo*. For successful deployment of stable resistance (R) genes, their characterization and availability of tightly linked markers will greatly facilitate the development of new versions of cultivars. To date, more than 40 different resistance genes conferring host resistance to BB have been identified in rice so far and some of those have been characterized [5].

The rice-*Xoo* system is distinguished from *Arabidopsis*—*Pseudomonas syringae*, tomato—*Cladosporium fulvum*, and rice—*Magnaporthe grisea*, with respect to that about one-third of the *R* genes for *Xoo* resistance are recessive in nature [6]. Secondly, diverse types of proteins are encoded by the *R* genes such as *Xa3*/*Xa26* and *Xa21* encode leucine-rich repeat (LRR) receptor kinase-type proteins [7–9] that mediate race-specific resistance. *Xa1* encodes a nucleotide-binding LRR protein [10]. The recessive gene *xa5* encodes the γ-subunit of transcription factor IIA [11, 12]. *Xa27* encodes a novel protein [13]. The recessive gene *xa13* encodes a novel plasma membrane protein [14]. Thus, it is clear that *Xoo* has the diverse interaction mechanism within the host species. Hence, the isolation and characterization of BB resistance genes from diverse germplasm will insight depth knowledge of both pathogen and host system, which will further lead to the development of BB resistant varieties with broad spectrum and durable resistance.

2. Genetics of BB resistance

Genetic analysis of many plant-pathogen interactions has demonstrated that plants often contain a single locus that confers resistance against a complementary avirulence gene [15]. The genetic basis of host resistance to BB has been studied in depth. The genetics of resistance to

bacterial blight was first carried out by Japan and IRRI, subsequently, followed by Sri Lanka, India, China, and so on. As there is diversity of *Xoo* strains in different countries, scientists found that it was difficult to characterize and distinguish the resistance genes. In order to compare the identified genes, the identical differential standard was set up [16]. About 42 BB resistance (R) genes, designated from *Xa1* to *Xa42* [5, 17, 18], conferring resistance against various strains of *Xoo*, have been identified from cultivated, mutant population, and wild rice species (**Table 1**). These genes have been mapped on six of the twelve rice chromosomes. A total of 14 recessive genes (*xa5*, *xa8*, *xa13*, *xa15*, *xa19*, *xa20*, *xa24*, *xa25*, *xa26b*, *xa28*, *xa31*, *xa32*, *xa33*, and *xa34*) in the series from *Xa1* to *Xa42* have been identified. Of these *R* genes, nine of the *R* genes have been cloned and characterized (*Xa1*, *Xa3*/*Xa26*, *xa5*, *xa13*, *Xa10*, *Xa21*, *Xa23*, *xa25*, and *Xa27*) encoding different types of proteins, suggesting multiple mechanisms of *R*-gene–mediated *Xoo* resistance [7, 8, 10, 11, 13, 14, 19–21]. Most of these genes provide complete and race-specific resistance to *Xoo* and have been used singly or in combination in rice breeding for BB resistance [22–32]. Since the bacterial races tend to evolve continually, influenced by the artificial and natural selection of genes resistant to BB, it is required to explore critically the new resistant resources to combat the evolved races.

The *Xa1* gene was identified by Sakaguchi [33] from rice cultivars Kogyoku and Java14. It conferred a high level of specific resistance to Japanese *Xoo* race 1. Since then, *Xa1* has been extensively used in Japanese rice breeding as *Xoo* race 1 is the most prevalent in Japan. Later on, a high-resolution genetic map was developed for *Xa1* using F_2 population and RFLP markers [34]. Three markers (XNpb235, XNpb264, and C600) on chromosome 4 were found to be tightly linked to *Xa1*, while another RFLP marker $U08_{750}$ was at 1.5 cM distance from *Xa1*. Screening of the YAC library with three linked markers resulted in identification of ten contiguous YAC clones. Out of these clones, Y5212 clone hybridized with all the three markers and was confirmed to possess the *Xa1* locus. Sakaguchi [33] also identified another BB resistance gene, *Xa2*, in rice cultivar Tetep. It conferred specific resistance to T7147 (Japanese *Xoo* race 2). Earlier, *Xa2* was mapped on chromosome 4, linked to *Xa1* with a recombination frequency of 2–16% [35]. He et al. [36] reported the fine mapping of *Xa2* using an F_2 population of ZZA (Zhengzhui Ai) X IRBB2 and *Xa*2 was localized between two markers spanning approximately 190 kb region. Sequence analysis of this region revealed the presence of a homologous sequence of leucine-rich repeat (LRR) kinase, which is the product of a cloned BB resistance gene, *Xa*21 [7].

The *Xa3* gene was identified in a *japonica* variety Wase Aikoku 3 [37]. It was mapped to the long arm of chromosome 11 and was found to be tightly linked to another BB resistance gene *Xa*4 [38, 39]. *Xa3* was later known to be genetically linked to *Xa26*, another BB resistance gene. For characterization of *Xa3*, the gene was fine mapped using a population segregating for only a single resistance gene and markers developed from *Xa26* gene family. Genetic analysis showed that *Xa3* co-segregated with the *Xa26* gene marker but segregated from the markers of other members of *Xa26* gene family.

DNA fingerprinting also revealed that IRBB3 carrying *Xa3* had the same copy numbers of *Xa26* gene family members as were present in Minghui 63 carrying *Xa26*. The putative coding products of *Xa3*/*Xa26* and the susceptible allele *xa3*/*xa26* shared 92% homology with difference in the LRR domains, proving that *Xa3* is the same as *Xa26* [9].

Xa gene	Resistance to Xoo race	Donor cultivar	Chromosome	Reference
Xa1	Japanese race-I	Kogyoku, IRBB1	4	[10, 33, 34]
Xa2	Japanese race-II	IRBB2	4	[33, 36]
Xa3/Xa26	Chinese, Philippine, and Japanese races	Wase Aikoku 3, Minghui 63, IRBB3	11	[9, 37]
Xa4	Philippine race-I	TKM6, IRBB4	11	[39, 40, 45]
xa5	Philippine races I, II, III	IRBB5	5	[11, 40, 46]
Xa6	Philippine race 1	Zenith	11	[47]
Xa7	Philippine races	DZ78	6	[48–51]
xa8	Philippine races	PI231128	7	[52]
xa9	Philippine races	Khao Lay Nhay and Sateng	11	[53]
Xa10	Philippine and Japanese races	Cas 209	11	[54, 55, 57]
Xa11	Japanese races IB, II, IIIA, V	IR8	3	[58, 59]
Xa12	Indonesian race V	Kogyoku, Java14	4	[60]
xa13	Philippine race 6	BJ1, IRBB13	8	[14, 61–64]
Xa14	Philippine race 5	TN1	4	[65–67]
xa15	Japanese races	M41 mutant	–	[69]
Xa16	Japanese races	Tetep	–	[70]
Xa17	Japanese races	Asominori	–	[71]
Xa18	Burmese races	IR24, Miyang23, Toyonishiki	–	[58]
xa19	Japanese races	XM5 (mutant of IR24)	–	[72]
xa20	Japanese races	XM6 (mutant of IR24)	–	[73]
Xa21	Philippine and Japanese races	*O. longistaminata*, IRBB21	11	[7, 76, 77]
Xa22	Chinese races	Zhachanglong	11	[79, 80]
Xa23	Indonesian races	*O. rufipogon* (CBB23)	11	[24]
xa24(t)	Philippine and Chinese races	DV86	2	[81, 82]
xa25/ Xa25(t) Xa25	Chinese and Philippine races	Minghui 63, HX-3 (somaclonal mutant of Minghui 63)	12	[85 86]
xa26(t)	Philippine races	Nep Bha Bong	–	[86]
Xa27	Chinese strains and Philippine race 2 to 6	*O. minuta* IRGC 101141, IRBB27	6	[26, 85]
xa28(t)	Philippine race2	Lota sail	–	[86]
Xa29(t)	Chinese races	*O. officinalis*(B5)	1	[87]
Xa30(t)	Indonesian races	*O. rufipogon* (Y238)	11	[88]

Xa gene	Resistance to Xoo race	Donor cultivar	Chromosome	Reference
xa31(t)	Chinese races	Zhachanglong	4	[89]
Xa32(t)	Philippine races	*Oryza australiensis* (introgression line C4064)	11	[90]
xa33(t)	Thai races	Ba7	6	[91]
Xa33(t)		*O. nivara*		[92]
Xa34(t)	Thai races	Pin Kaset	–	[91]
Xa34(t)		*O. brachyantha*		[93]
Xa35(t)	Philippine races	*Oryza minuta* (Acc. No.101133)	11	[94]
Xa36(t)	Philippine races	C4059	–	[95]
Xa38	Indian Punjab races	*O. nivara* IRGC81825	–	[17, 96]
Xa39	Chinese and Philippines races	FF329	11	[97]
Xa40(t)	Korean BB races	IR65482-7-216-1-2	11	[5]
xa41(t)	Various *Xoo* strains	Rice germplasm	–	[98]
xa42	Japanese *Xoo* races	XM14, a mutant of IR24	3	[18]

Revised and updated from Ref. [147].

Table 1. Summary of resistance genes to bacterial blight in rice.

The *Xa4* and *xa5* genes were identified by Petpisit et al. [40]. The dominant gene *Xa4* confers resistance to Philippine race 1 of *Xoo* and was identified in rice cultivars TKM6, IR20, IR22, and IR72. The recessive gene *xa5* confers resistance to Philippine *Xoo* races 1, 2, 3, and 5 [41]. Yoshimura et al. [39] reported the mapping of *Xa4* and mapped an RFLP marker G181 on one flanking side of *Xa4* locus on chromosome 11. However, no marker was identified on the other flanking side of *Xa4* locus. The *Xa4* locus was further mapped between two RFLP markers, RZ536 and L457 [41]. Wang et al. [42] localized the *Xa4* locus between G181 and L1044 at a distance of 4.4 and 3.8 cM, respectively. However, using other rice molecular linkage maps as references [43, 44], Sun et al. [45] deduced that *Xa4* locus reported earlier [41, 42] was not in the same position. Using an F_2 population from IR24 X IRBB4 cross, *Xa4* gene was genetically mapped to a region less than 1 cM. A contig map was constructed consisting of six nonredundant BAC clones, spanning approximately 500 kb length. Analysis of recombination events located *Xa4* locus to one BAC "3H8" and assay of recombinants using subclones of "3H8" along with sequence analysis, further narrowed the *Xa4* locus to a 47 kb fragment.

A high-resolution genetic map of the chromosomal region harboring *xa5* was developed and mapped to a 0.5 cM interval between markers RS7 and RM611, which spanned an interval of 70 kb and contained 11 open reading frames. The potential candidate gene products of *xa5* may be basal transcription factor IIA (TFIIA), an ABC transporter, a tRNA synthase, a MAP kinase and a cysteine protease, as well as four unknown, hypothetical or putative proteins [46].

While studying the inheritance of resistance to *Xoo* isolate PXO61 in five rice cultivars, Sidhu and Khush [47] observed that the same gene conferred resistance in all the cultivars. The pattern of segregation indicated a monogenic recessive factor when plants were inoculated at booting stage, but monogenic dominant when plants were inoculated during flowering. This reversal of dominance was caused by dosage effect of the resistance gene at a specific stage of growth of the plant when exposed to the bacterial inoculum. Apparently, the gene could express itself at booting stage only when present in two dosages. Heterozygous plants with one dose of resistance allele were susceptible during booting. However, at flowering, one dose was enough for expression of resistance. The new gene identified as *Xa6* and was linked to *Xa4* with a crossover value of 26%. Later studies on the genetics of resistance to *Xoo* in 74 cultivars of *O. sativa* using P*XO*61 isolate from the Philippines identified an additional dominant gene, *Xa7*, that conferred resistance in DZ78 [48]. A recombination value of 8.8% between *Xa7* and G1091 located at 107.5 cM on chromosome 6 was determined [49]. Porter et al. [50] performed the AFLP analysis of a segregating, near-isogenic F_3 population of IR24 X IRBB7, which revealed one polymorphic fragment, M1, mapped to position 107.3 cM on the rice map. Sequence comparisons of resistant and susceptible lines near M1 were used to generate additional markers linked to the rice BB resistance gene *Xa7*. A sequence-tagged site (STS) named M2 was mapped proximal to M1 and farther from *Xa7*, indicating that *Xa7* lay distal to M1. Two SSRs, M3 and M4, were mapped at a distance of 0.5 and 1.8 cM from *Xa7*, respectively. The pattern of recombinants and the map distances indicated that *Xa7* was located in the region corresponding to the ends of physically mapped Clemson University Genomics Institute (CUGI) BACs 96 and 143. A complex repeat was identified in the DNA sequence from *O. sativa* cultivars 93-11 and Nipponbare that matched the end of contig 96 and an EST marker (C52865S). Amplification of the repeat revealed the presence and absence of the repeat in IR24 and IRBB7, respectively. However, no recombinants were identified between *Xa7* and the polymorphic repeat (M5). Comparison of the physical and genetic maps of rice in that region indicated that *Xa7* lay within 40 kb of M5, a distance suitable for MAS and cloning of *Xa7*. A high-resolution genetic map of the chromosomal region surrounding *Xa7* gene was constructed using SSR markers from the Gramene database (www.gramene.org/db) based on the *Xa7* gene initial mapping on chromosome 6 [51]. Primary analysis of F_2 population from the cross of IRBB7 and IR24, located *Xa7* in approximately 0.28 cM region. To walk closer to the target gene, recombinant F_2 individuals were tested using STMS markers and the gene was finally mapped to a 0.21 cM region between markers GDSSR02 and RM20593. A contig map corresponding to *Xa7* gene was constructed using reference sequence of cultivar Nipponbare through bioinformatics analysis. The target gene was assumed to span an interval of approximately 118.5 kb, containing a total of fourteen genes. Candidate gene analysis showed that the fourteen genes encoded novel domains that had no amino acid sequence similar to other cloned *Xa* (*xa*) genes.

A single recessive gene, *xa8*, conditioned resistance in rice germplasm accession PI 231129. Vikal et al. [52] mapped the gene between two consecutive SSR markers, RM21044 and RM21045 at 7.0 and 9.9 cM, respectively, on chromosome 7 and the physical distance between markers was 9.5 kb. This region harbors three intact genes, which codes for putative expressed

proteins *viz*., Zinc finger A20 and AN1 domain-containing protein (LOC_Os07g07400), oxidoreductase, 2OG-Fe oxygenase family protein (LOC_Os07g07410), and Gibberellin 20 oxidase 1-B protein (LOC_Os07g07420). All the three candidate genes are responsible for plant response to various stresses.

The BB resistance in 'Khao Lay Nhay' and 'Sateng' was recessive in nature and were allelic to each other but nonallelic to and segregated independently of *Xa3, Xa4*, *xa5, Xa7*, and *xa8* [53]. This new recessive gene was linked to *Xa6* with a crossover value of 5.9%. This new gene for resistance was designated as *xa9*. The *Xa10* was identified from rice cultivar Cas 209 [54, 55]. There is an extensive polymorphism on chromosome 11L between IRBB10 and IR24 and also a great distortion in *Xa10* inheritance. Rice lines that carry *Xa10* gene confer race-specific resistance to *Xoo* strains harboring avirulence (Avr) gene *avrXa10*. The *Xa10* locus was roughly integrated to a large region between RFLP markers RG103 (~83 cM) and RG1109 (~91.4 cM) on the long arm of rice chromosome 11 [56]. High-resolution mapping by Gu et al. [57] narrowed down *Xa10* to a 74 kb region between RFLP markers M491 and M419. Out of the 7 identified genes in this region, 6 are considered possible candidate genes for *Xa10*. Ogawa and Yamamoto [58] identified another BB resistance gene *Xa11*. *Xa11* confers specific resistance to strains T7156, T7147, T7133, and H75304 (Japanese *Xoo* races IB, II, IIIA, and V, respectively). *Xa11* was mapped between the loci of RM347 (2.0 cM) and KUX11 (1.0 cM) on the long arm of chromosome 3 [59]. It was reported that a single dominant gene, *Xa12*, controlled the resistance in Kogyoku and Java14 to BB race V of Indonesia [60].

The *xa13* gene was first discovered in the rice variety BJ1 and this gene specifically confers resistance to the Philippine *Xoo* race 6, the most virulent race and one not overcome by most reported *R* genes [61–63]. Sanchez et al. [63] reported the genetic and physical mapping of *xa13* gene using two F_2 populations of the cross between IR24 and IRBB13. The gene was fine mapped to a region less than 4 cM on the long arm of chromosome 8 between two RFLP markers, RG136 and R2027. Sequence analysis of the 14.8 kb fragment carrying *xa13* gene indicated only two apparently intact candidate genes—an extensin-like gene and a homologue of nodulin (MtN3) and the 5′ end of a predicted hypothetical protein [14]. The expression of the rice gene *Os8N3*, a member of the MtN3 gene family from plants and animals, was elevated upon infection by *Xoo* strain PXO99A and depended on type III effector gene PbXo-1. *Os8N3* resides near *xa13* and could not be induced in rice lines with *Xa13*. Inhibitory RNA silencing of *Os8N3* produced PX099A resistant plants [64].

Xa14 identified from Taichung Native 1 is a dominant gene against resistance to PXO112, BB race 5 of the Philippines [65]. *Xa14* was located on chromosome 4 between marker RG620 and G282, with 20.1 and 19.1 cM, respectively by Tan et al. [66]. The gene was fine mapped by using two F_2 populations. Combining recombination frequencies for the two populations together, the gene *Xa14* was mapped to 3 BAC clones spanned approximately 300 kb in length between SSR markers HZR970-8 and HZR988-1 [67].

A series of nine genes (*xa15*, *Xa16*, *Xa17*, *Xa18*, *xa19*, *xa20*, *xa26*, *Xa27*, and *xa28*) have been obtained from mutagenesis which had different resistance levels and resistance spectrums [58, 68–73]. A mutant rice line, XM5, was resistant to bacterial blight [72]. Allelic relationship

studies with the known recessive genes, *xa5*, *xa8*, and *xa13* revealed that the gene was not allelic to them and was a new gene. It was designated as *xa19*.

Devadath [74] identified a strain of *Oryza barthii* which was resistant to all the races of *Xoo* in India. Khush et al. [75] multiplied the strain at IRRI and studied its resistance to Philippine *Xoo* races. It was found that the strain was akin to *Oryza longistaminata* which was resistant to all the six Philippine *Xoo* races [76]. F_1s of the cross of the strain with IR24 were resistant showing that the resistance was conferred by a dominant gene. The gene was different from the known BB resistance genes and was designated as *Xa21*(t). Genetic and physical analysis of *Xa21* localized it in an 8.3 cM interval on chromosome 11 and the physical size of the region containing *Xa21* gene was estimated to be about 800 kb [77].

Genomic DNA gel-blot analysis revealed that *Xa21* belonged to a multigene family containing at least eight members. Most of these members were mapped to a single locus on chromosome 11, which is linked to at least nine other major resistance genes and one QTL for resistance [7, 77]. Wang et al. [78] studied the inheritance patterns and resistance spectra of transgenic plants carrying six *Xa21* gene family members and observed that one member, designated *Xa21D*, conferred only partial resistance. Analysis of *Xa21D* sequence showed that *Xa21D* transcript terminated shortly after the stop codon introduced by retrotransposon. Sequence comparison of *Xa21* and *Xa21D* provided evidence of adaptive selection. *Xa21D* encodes a receptor-like protein carrying LRR motifs in the presumed extracellular domain. Both functional and evolutionary evidence indicated that *Xa21D* LRR domain controlled race-specific pathogen interaction.

Another BB resistance gene, *Xa22* was identified from landrace Zhachanglong (ZCL) of Yunnan province in Southwest China. It showed high level of resistance to 16 of the 17 BB strains tested [79]. The gene was nonallelic to *Xa1*, *Xa2*, *Xa4*, and *Xa14* but was found to be linked to *Xa4*. *Xa22* gene was mapped on short arm of chromosome 11, but according to new molecular map developed by Harushima et al. [43], it was present on the long arm. *Xa22* provides resistance to a broad spectrum of *Xoo* isolates. The gene was localized to a small 100 kb fragment delimited by R1506 and a subclone from M3H8 BAC clone by a combination of genetic recombination analysis and physical mapping [80].

An interspecific cross was made between RBB16 and Jiagang30 (JG30), and the F_1 plants showing highest resistance to BB were anther cultured to obtain doubled haploids for resistance. These anther-cultured progenies were inoculated with three strains of *Xoo*, and lines showing high resistance and improved plant type in H_2 generation were advanced to H_4 and collectively designated as WBB1. Zhang et al. [24] crossed WBB1 with two susceptible Chinese varieties, JG30 (*indica*) and 02428 (*japonica*), and IRBB21 (*Xa21*). Inoculation results with PXO99 at seedling stage suggested that a single dominant gene from *Oryza rufipogon* conferred resistance and the gene was nonallelic but probably linked to *Xa21*. PCR analysis of WBB1 with *Xa21* specific primers also indicated that the gene was different from *Xa21*. The new gene, designated as *Xa23*(t), was mapped within a 0.4 cM region between markers Lj138 and A83B4, and the corresponding physical distance between these markers is 49.8 kb. Six *Xa23* candidate genes have been annotated, including four candidate genes encoding hypothetical proteins, and the other two encoding a putative ADP-ribosylation factor protein

and a putative PPR protein. Nine varieties (AC19-1-1, Aus 274, Chinsurah Boro II, Kalimekri 77-5, Tepa I, Long grain, Aus 295, DV85, and DV86) were crossed with BJ1 (carrying *xa13*) to confirm whether the resistance was due to *xa13* or a new gene. The results indicated that the recessive gene identified in DV86 for resistance to race 6 was nonallelic to and independent of *xa13*. DV85, DV86, and Aus 295 had the same recessive resistance gene and it was designated as *xa24*(t) [81].

The *xa24* resistance gene was fine mapped to a 71 kb DNA fragment in the long arm of chromosome 2 using PCR-based markers. It mediates resistance to the Philippine *Xoo* races 4, 6, and 10, and Chinese *Xoo* strains Zhe 173, JL691, and KS-1-21. The analysis of F_2 mapping population, developed from the cross between a resistance line DV86 (*Oryza sativa* ssp. *indica*) carrying the target gene and a sensitive line IR24 (*O. sativa* ssp. *indica*), first mapped the gene to terminal region of long arm of chromosome 2 at 8.0 and 0.9 cM from RM482 and RM138, respectively. Further, fine mapping placed the *xa24* gene between RM14222 and RM 14226 at a distance of 0.07 cM from both markers [82].

Xa25(t), a new dominant resistance gene, was identified in Minghui 63, a restorer line for a number of rice hybrids cultivated widely in China [83]. The gene conferred resistance to Philippine race 9 (PXO330) at both seedling and adult plant stages. Gao et al. [84] identified another dominant gene in a somaclonal mutant HX-3 providing resistance to BB strain Zhe173 and was also designated as *Xa25*(t). Bulked segregant analysis carried out on the DH population located the gene at the terminal region of the long arm of chromosome 4 between the two SSR markers RM6748 and RM1153 at a map distance of 9.3 and 3.0 cM, respectively.

In order to broaden the germplasm pool for breeding of disease resistance to *Xoo*, a wide hybridization project to transfer *R* genes from wild species *Oryza minuta* was initiated at IRRI in the late 1980s. A novel resistance (*R*) locus was identified from a progeny of interspecific hybrids of *O. sativa* cv. IR31917-45-3-2 and *O. minuta* acc. 101141 and was tentatively designated as *Xa27*(t) [85]. The genetic analysis of 21 rice cultivars for BB resistance divided the cultivars into two groups (group 1 and group 2) based on their resistance to Philippine *Xoo* races 1 and 2. Group 1 was resistant to race 1 and group 2 to race 2. When crossed with TN1 and evaluated for resistance with races 1 and 2, all cultivars showed monogenic inheritance of resistance. Allelic relationships of the genes were investigated by crossing these cultivars with different testers having single genes for resistance. Out of all the cultivars, three had new, undescribed genes. Nep Bha Bong had a recessive gene for moderate resistance to races 1, 2, and 3 and resistance to race 5 and was also designated as *xa26*(t). Arai Raj had a dominant gene for resistance to race 2 which segregated independent of *Xa11* and was designated *Xa27*(t). Lota Sail had a recessive gene for resistance to race 2 which segregated independently of *Xa10* and was designated as *xa28*(t) [86].

Gu et al. [26] reported the fine genetic mapping of the *Xa27*(t) gene by performing disease evaluation of an *Xa27*(t) NIL, IRBB27, with 35 *Xoo* strains from 11 countries. Resistance of *Xa27*(t) gene was developmentally regulated in IRBB27 and showed semidominant or dosage effect in the cultivar CO39 genetic background. Three markers, M336, M1081, and M1059, were closely linked to *Xa27*(t). The *Xa27*(t) locus was confirmed by chromosome landing of

M1081 and M1059 markers on rice genome. Markers derived from the genomic sequence of *O. sativa* cv. Nipponbare finally located the *Xa27*(t) gene within a genetic interval of 0.052 cM on the long arm of chromosome 6 between markers M964 and M1197, and co-segregated with M631, M1230, and M449.

Another dominant *Xa* gene was identified from a rice line 'B5' derived from *Oryza officinalis* through introgression. It proved to be highly resistant to brown plant hopper, white-backed plant hopper and bacterial blight. The resistance gene identified in B5 was designated as *Xa29*(t) [87]. Bulked segregant analysis of RILs from a cross between 'B5' and 'Minghui63' located the resistance gene within a 1.3 cM region flanked by RFLP markers C904 and R596 on chromosome 1.

A new rice BB resistance germplasm (Y238) from the wild rice species *O. rufipogon* was identified and designated as *Xa30*(t) [88]. The gene was mapped on the long arm of rice chromosome 11. Linkage analysis revealed that four molecular markers RM1341, V88, C189, and 03STS were located on the same side of *Xa30*(t), with genetic distances of 11.4, 11.4, 4.4, and 2.0 cM to the candidate gene, respectively. Wang et al. [89] identified *Xa31*(t) gene for BB resistance in Zhachanglong (ZCL), a regional rice variety from Yunnan province in southwest China, which has a high level of resistance to a broad spectrum of *Xoo* isolates. Genetic linkage analysis and fine mapping localized *Xa31*(t) within a genetic distance of 0.2 cM between two RFLP markers G235 and C600 on the end of long arm of chromosome 4. The flanking markers were used to screen the MH63 BAC library and the *Xa31*(t) locus was limited to one BAC clone with a length of about 100 kb.

Another novel BB resistance gene from a wild rice (*Oryza australiensis*) introgression line, C4064, was found to be resistant to *Xoo* strains P*XO*61, P*XO*71, P*XO*99, P*XO*145, P*XO*280, P*XO*339, and KX085 but susceptible to P*XO*8 and P*XO*79. The gene was tentatively designated as *Xa32*(t). The chromosomal position of the gene was located using BSA with SSR and EST markers between two markers at the end side of long arm and four markers on centromere side. Finally, the gene was mapped within a 2.0 cM interval flanked by two SSR markers RM2064 and RM6293 on the long arm of chromosome 11 [90]. Another BB resistance gene, *xa33*(t) was identified from rice cultivar 'Ba7'. The gene was localized on long arm of chromosome 6, where two other dominant genes (*Xa7* and *Xa27*) have been reported. RM 20590 was identified as the closest linked marker. Although *Xa7* and *xa33* shared common markers, both showed different gene actions and were not growth stage-dependent genes [91]. Natarajkumar et al. [92] placed a new BB resistance gene, also designated as *Xa33*(t) from *Oryza nivara*, on chromosome 7 flanked by the markers RM21004 and RM21177 at a genetic distance of 2.0 and 4.5 cM, respectively.

Pin Kaset (PK), a Thai rice cultivar, had a high level of resistance to BB. The resistance gene in PK was identified using BC_2F_2 plants from a cross between Ba7 and PK. Phenotypic evaluation of TB0304 and genotypic analysis with SSR markers revealed that the gene was linked to RM224 on chromosome 11 [91]. The new gene was tentatively designated *Xa34*(t). Although *Xa34*(t) and other BB resistance genes are located in the same region and shared common linked markers, no evidence was obtained whether they shared the same genomic sequence

or were tightly linked to each other. Also, Ram et al. [93] screened 2 introgression lines (IR65483-118-25-31-7-1-5-B and IR65483-141-2-4-4-2-5-B) derived from IR56 X *O. brachyantha*, against a virulent isolate DXO44 and 21 different virulent isolates of BB from 11 states of India. The ILs showed resistance to 16 isolates, moderate resistance to one and susceptibility to 4 isolates, as against the differential reaction of IRBB lines to different isolates. This indicated that the introgressed gene is different from those in IRBB lines. It was designated as *Xa34*(t). Crosses of ILs with IR56 and susceptibility check BPT5204 gave all resistant F_1s and 3:1 ratio in F_2 revealing a single dominant gene in both cases. Allelic test was done by crossing ILs with IRBB21 which showed that *Xa34*(t) (resistance gene from *O. brachyantha*) and *Xa21* were nonallelic and inherited independently.

A new rice bacterial blight resistance gene *Xa35*(t) from the wild rice species *Oryza minuta* (Acc. No. IRGC101133) was identified and transferred into IR24 [94]. Through genetic analysis and identification of resistance spectrum, *Xa35*(t) showed a high level of resistance to PXO61, PXO112, and PXO339, but was susceptible to P*XO*86 and P*XO*99. The *Xa35*(t) locus was mapped to a 1.80 cM region and this locus was co-segregated with marker RM144, and was 0.7 cM from marker RM6293 on one side and 1.1 cM from marker RM7654 on the other side of the rice chromosome 11. Miao et al. [95] identified that the rice germplasm C4059 harbored a bacterial blight resistance gene and designated it as *Xa36*(t). The gene *Xa36*(t) was mapped on the long arm of rice chromosome 11 encompassing 4.5 cM region flanked by RM224 and RM2136.

An accession of *O. nivara* (IRGC 81825) was identified to be resistant to all the seven *Xoo* pathotypes prevalent in North India [96]. The F_2 population derived from a cross between PR114 and *O. nivara* acc. 81825 segregated in a 3:1 ratio of resistant: susceptible plants, indicating that the resistance was provided by a single dominant gene. The inheritance and mapping studies using F_2, BC_2F_2, BC_3F_1, and BC_3F_2 progenies of the cross *O. sativa* cv. PR114 × *O. nivara* acc. 81825 mapped the gene on chromosome 4L, spanning an approximate 38.4 kb region [17]. Since none of the known *Xa* genes mapped on chromosome 4L were effective against the *Xoo* pathotypes tested, the gene identified and transferred from *O. nivara* was considered novel and designated as *Xa38*.

A rice introgression line (IL), FF329, identified from a BC_1F_4 population derived from the cross between donor PSBRC66 (P66) and recipient Huang-Hua-Zhan (HHZ), exhibited a typical hypersensitive response (HR) with all 21 representative *Xoo* strains (14 Philippines races and seven Chinese pathotypes) [97]. By contrast, the parents were highly susceptible to 10 of the tested strains and resistant or moderately susceptible to 11 strains, but without HR symptoms. As FF329 showed broad-spectrum BB resistance, the gene identified was novel and was designated *Xa39*. Two SSR markers, RM21 and RM206, located on rice chromosome 11, were linked to the target gene by bulked segregant analysis of the F_2 population derived from the HHZ/FF329 cross. The fine mapping of *Xa39* locus placed the gene in the region of 97 · 4 kb interval flanked by markers RM26985 and DM13.

The *japonica* advanced backcross breeding lines derived from the *indica* line IR65482-7-216-1-2 in the background of cultivar Junam was resistant to all Korean BB races. Kim et al. [5]

using two F_2 populations derived from the crosses between 11325 (IR83261-3-7-23-6-2-1-1-2-1-2)/Anmi and 11325/Ilpum indicated that resistance (R) was controlled by a new resistance gene designated as *Xa40(t)*, which was co-segregated with the markers RM27320 and ID55. Thus, based on the physical map of *japonica* rice Nipponbare, the *Xa40(t)* gene was defined by RM27320 and ID55.WA18-5 located on the BAC clone OSJNBa0036K13 spanning approximately 80 kb region on chromosome 11.

Hutin et al. [98] screened a germplasm of 169 rice accessions for polymorphism in the promoter of the major bacterial blight susceptibility "S" gene OsSWEET14, which encodes a sugar transporter targeted by numerous strains of *Xoo*. They identified a single allele with a deletion of 18 bp overlapping with the binding sites targeted by several TAL effectors known to activate the gene. Further, they showed that this allele, which was designated as *xa41(*t), confers resistance against half of the tested *Xoo* strains, representative of various geographic origins and genetic lineages, highlighting the selective pressure on the pathogen to accommodate OsSWEET14 polymorphism. Analysis of *xa41*(t) demonstrated that the resistance through TAL effector-dependent loss of S-gene expression can be greatly fostered upon knowledge-based molecular screening of a large collection of host plants.

A new mutant named 'XM14' was obtained by treating IR24, which was resistant to all Japanese *Xoo* races. The gene identified in XM14 was designated as *xa42* [18]. The F_2 population from XM14 × IR24 clearly showed 1 resistant:3 susceptible segregation, suggesting control of resistance by a recessive gene. The *xa42* is located around the centromeric region of rice chromosome 3 in the chromosomal region encompassed by KGC3_16.1 and RM15189.

3. Determinants of pathogenicity

Xanthomonas oryzae pv. *oryzae* (*Xoo*) is a member of the c-subdivision of the Gram-negative proteobacteria. It continues to grow in the vascular system until the xylem vessels are clogged with bacterial cells and extracellular polysaccharide (EPS or xanthan). There are several races of *Xoo*, all of which secrete race-specific effectors into the xylem to trigger individualized response and cause infection. The bacteria also release factors which bind and activate transcription of genes that activate resistance response, known as resistance genes (*R* genes) [99]. The factors that activate *Xoo* resistance genes are known as avirulence factors that determine host specificity via gene-for-gene interactions, reducing the virulence of the pathogen as they are recognized by the host. Since each race of *Xoo* produces unique virulence and avirulence factors, *R* genes have evolved to provide resistance to individual races of *Xoo*. It has been known that the interactions of *Xoo* with plants are determined by hypersensitive response and pathogenicity (hrp) genes, which are required for pathogenicity in susceptible host plants and for the hypersensitive response (HR) in resistant and nonhost plants [3, 100]. A considerable effort has been made to identify genes involved in the pathogenesis of *Xoo* and to understand the roles of the gene products in the disease process. The knowledge of pathogenesis genes

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has become more clear and distinct from the genome sequence of *Xoo* strains KACC10331, MAFF311018, and PXO99A [4, 101, 102].

3.1. Avirulence genes

Members of the *AvrBs3*/*PthA* family of transcription activator-like effectors play a major role in the virulence of *X. oryzae* pv. *oryzae*. The *avr* gene family consists of a repeat sequence in the central domain of the encoded protein with each repeat of 34 amino acids. The number of repeats in different gene families varies, but the amino acids in each repeat are conserved except amino acids at positions 12 and 13, which are referred to as variable regions. The arrangements of the variable regions of all characterized family members appear to be a critical feature for the race specificity of the proteins. The carboxyl terminus of *AvrXa7* and *AvrXa10* contains a domain that is structurally similar to the acidic activation domain of many eukaryotic transcription factors, in addition to three nuclear localization signal (NLS) sequences as with *AvrBs3* family members. It has been postulated that, two *raxP* and *raxQ* genes of *Xoo* are required for the activation of *AvrXa21*. Both *raxP* and *raxQ* resides in a genomic cluster of sulfur assimilation genes, which encodes an ATP sulfurylase and adenosine-5′-phosphosulfate (APS) kinase. These enzymes function together to produce activated forms of sulfate, APS, and 3′-phosphoadenosine-5′-phosphosulfate (PAPS). It has also been observed that the transcription activation domain and the NLS sequences in *AvrXa7* are required for the virulence activity, suggesting that the *avr* gene products act as virulence factors that enter the host nucleus and directly affect host gene transcription [103]. Adaptation of an avirulent bacterial race to host varieties containing a single dominant *R* gene often results from the loss of function of the corresponding *avr* gene. However, the fitness penalty associated with loss of the *avr* gene may prevent disease epidemics [104].

3.2. Type III effectors

The interaction of many Gram-negative plant and animal pathogenic bacteria with their hosts depends on a conserved type III protein secretion system (TTSS). The TTSS is encoded by *hrp* genes for eliciting either HR on nonhost/resistant host plants or pathogenesis on susceptible hosts. The *hrp* gene-encoded TTSSs are responsible for translocation of avirulence proteins into the host plant cells. In xanthomonads, the *hrp* gene cluster comprises six operons (*hrpA* to *hrpF*) and is positively regulated by *HrpG* and *HrpX* [105–107], repressed in nutrient-rich media, but induced in nutrient-limited media and inside the host [108–111]. *HrpG*, which is predicted to be a member of the *OmpR* response regulator family of two-component signal transduction systems, regulates the expression of *hrpX*, encoding an *AraC*-type transcriptional activator, which then activates the expression of other *hrp* operons. The highly conserved *hrp* genes named *hrc* encode the proteins of the apparatus of the type III secretion system, and are critical for pathogenicity and the initiation of disease [100, 112, 113]. A *hrp* gene cluster was identified in the *Xoo* genome harboring 26 genes inclusive of *hpa2* and *hrpF*. The *Xoo hrp PAI* (31.3 kb) was larger due to the presence of four transposase genes (about 6 kb) located between *hpaB* and *hrpF* genes than its counterparts of *Xac* (25.6 kb) and *Xcc* (23.1 kb). Strong amino acid identity was observed between several orthologous *hrp* genes of

Xoo and *Xanthomonas axonopodis* pv. *citri* (*Xac*), as *hpaF*, *hpaP*, *hrpD5*, and *hpaA* had identity of 74, 76, 79, and 82% respectively, whereas *hrpF* (68%), *hpa1* (65%), *hrpB5* (66%), and *hrpB7* (65%) exhibited relatively low similarity. The products of *hrpF* and *hpa1* are predicted to be exposed or secreted components of the type III secretion system, which is responsible for contributing to their diversity due to distinct selective pressures in the different hosts. It has been demonstrated that the transcriptional regulator for *hrp* (trh) and *phoP* genes in *Xoo* positively regulate expression of *hrpG*, but not known whether the regulation is direct or indirect [114, 115]. The *trh* gene encodes a putative transcriptional regulator [114] and *phoP* encodes a putative response regulator of two-component regulatory systems [115]. It has not been reported whether *trh* and *phoP* influence the expression of other *XrvA* targets such as *gum* and *rpf*. It will be of interest to further study the regulation of *hrpG* expression by deciphering the functional relationship (if any) between *xrvA*, *trh*, and *phoP* to better understand *hrp* regulatory mechanisms. A homolog to *hrpW*, a proposed pectate lyase, was not readily apparent in the *Xoo* genome but several candidate pectate lyase genes were identified that could function similarly to *hrpW*.

A review classified all known and candidate TTSS effectors from strains of *Xanthomonas* spp. into 39 groups based on sequence and structural differences, and similarities [116]. A class of T3 effectors called transcription activator-like effectors (TALE) was first identified due to their relatedness to the T3 effector *AvrBs3* from *Xanthomonas campestris* pv. *vesicatoria* [117]. Some of these TALEs have avirulence activity and three different TAL effectors *avrXa7*, *avrXa10*, and *avrXa27* from *Xoo* have been cloned [13, 118]. All TALEs have nuclear localization signals (NLS) and an activation domain rich in acidic amino acids (AAD) at their C terminus. Each TALE also contains a central region of multiple 34- to 35-amino acid direct repeats that are nearly identical except the 12th and 13th amino acid residues (so-called repeat variable diaminoacids, or RVD) [119]. The combination of repeat number and composition of RVDs of individual TALEs determine the specificity of the targeted genes [119, 120]. Five additional T3 effectors from *Xoo* have known contributions to virulence under the appropriate conditions. These genes are *pthXo1*, *pthXo2*, *pthXo3*, *pthXo6*, and *pthXo7*. *PthXo1* is the major TAL effector in many strains, including the common laboratory strain P*XO*99A, and induces the expression of host gene *Os8N3*, a member of *nodulin 3* (*N3*) gene family and encodes a predicted membrane protein [64]. In addition to *PthXo1*, two other TAL effectors of P*XO*99A also contribute to virulence by inducing the expression of two different host genes [121]. *PthXo6* and *PthXo7* elevate the transcription of host genes *OsTFX1* and *OsTFIIAγ1*, respectively. *OsTFX1* is a member of bZIP family of transcription factors, which are involved in the regulation of many developmental and physiological processes. Another TAL effector gene, named *pthXo8*, has been identified in P*XO*99A with quantitative effects similar to *pthXo6*. Preliminary evidence indicates that the effector is involved in manipulation of the small RNA pathways of the host.

By using custom-engineered TALEs to investigate the functionality of host target genes involved in *Xoo*/rice interaction, it has been demonstrated that *xa13* can be induced by *Xoo* and confer disease susceptibility, lending further evidence for *Os8N3* (*OsSWEET11* or *Xa13*) as an S gene. This approach facilitated to identify another *SWEET* gene (*OsSWEET12*) that can act as an S gene, provided that the *Xoo* pathogen contains a corresponding TALE. Further studies

revealed that the *xa27* allele can be activated and triggers resistance to the bacterium expressing a corresponding dTALE. Rice genome contains at least 21 *SWEET* (or *N3*) genes with a phylogenetic clade that harbors two known S genes (*OsSWEET11* and *OsSWEET14*) and three additional uncharacterized *SWEETs* [122, 123] generated seven artificial or designer TAL effectors based on the promoter sequences of six loci in rice and transformed them into *Xoo*. Gene expression analyses indicated all dTALEs were active in trans-activating target genes in rice achieving a 100% success rate in engineering active dTALEs. Gene activation of three alleles (*xa27*, *xa13*, and *OsSWEET12*) led to phenotypic changes in disease resistance or susceptibility in response to *Xoo* infection, suggesting the feasibility of this approach to the gene functional analysis.

The genomic sequences are available for three strains of *Xoo* [4, 101, 102]. A comparison of the repetitive regions of PXO99A and MAFF311018 indicates a high degree of rearrangements and shuffling of the genes at all of the loci, to the point where only three genes of 17 in MAFF311018 are identical [96]. The genome structure of *Xoo* MAFF 311018 was characterized by large numbers of effector (*avr*) genes of the *avrBs3/pth* family and insertion sequences (ISs). The high degree of genetic diversity and race differentiation characteristic of this pathogen is due to the presence of mobile elements, which leads to genome inversions and rearrangements. The large numbers of TAL effector genes in these species may reflect the evolutionary "investment" the strains have in utilizing the TAL effectors for virulence. The maintenance of high gene numbers may even be exacerbated by rice breeding and *R* gene deployment by farmers over the millennia. It could be inferred that *Xoo* targets different host genes to alter the host physiology and different TAL effectors to have qualitatively different effects on host susceptibility.

3.3. Type II secretion system

The bacterial type II secretion system mediates a two-step process. The proteins that are secreted through this system carry a secretion signal at their N termini and are transported into the periplasmic space through the inner membrane by either the general secretion pathway (GSP) or the twin arginine pathway (TWP) [124, 125]. Transport across the outer membrane is facilitated by the proteins of main terminal branch (MTB) of general secretion pathway (GSP). The *Xoo* genome encodes a single type II secretion system in contrast to two different type II systems in *Xac* and *Xcc* [126]. Mutations in the gene cluster *xps*, which is required for a functional type II system, result in strains defective in virulence [127]. Type II secreted proteins are mainly toxins and enzymes that target different components of the host cell, and some of these enzymes, including xylanase, cellulase, cysteine protease, cellobiosidase, and lipases, have been characterized as contributors to *Xoo* virulence. Experimental evidences indicate that rice plants perceive some type II secreted proteins and respond by hypersensitive responses (HRs), and these responses are suppressed by type III secreted effectors [125]. Other factors that contribute to the virulence of *Xoo* include EPS, the type II general secretion system and its secreted proteins, and regulation involving genes within the *rpf* cluster [3, 128–132]. The *rpfB*, *rpfC*, *rpfF*, and *rpfG* genes (regulation of pathogenicity factor) of *Xoo* have been shown to affect virulence in rice, EPS production, xylanase production, and motility [130–132]. The virulence of *Xanthomonas* also depends upon cell-to-cell signaling mediated by diffusible signal factor (DSF). Two of the genes in this cluster *rpfB* and *rpfF*

are involved in the synthesis of DSF. Knockout studies on *rpfF* indicate the role of DSF in the regulation of levels of extracellular enzymes and EPS [133]. The *RpfC*/*RpfG* two-component system couples the DSF sensing to intracellular regulatory networks through a second messenger, cyclic *diGMP*, and a global regulator, *Clp*. Protein-protein interaction between the DSF synthase *RpfF* and the sensor *RpfC* may act as a posttranslational mechanism to modulate the biosynthesis of DSF [134, 135]. Recently, an *X. oryzae* pv. *oryzae* flagellar operon region has been isolated from *Xoo*, which contains four ORFs. One of the ORFs, *flhF*, encodes a putative GTP-binding protein which is involved in chemotaxis. Mutation in *flhF* resulted in weak chemotaxis but did not show reduced virulence if inoculated on rice leaves with a scissors-clipping method, suggesting that chemotaxis is not required for virulence once the bacterial cells enter rice leaves [136]. EPS synthesis is directed by genes at multiple chromosomal loci; one of the loci is called the *gum* cluster [137]. The *Xoo gum* cluster in strain KACC10331 is composed of 14 ORFs arranged in a tandem array, expressed from a promoter located upstream of *gumB*, but internal promoters can also be found upstream of *gumG*, *gumH*, and *gumM*, respectively [101, 138]. Two different *Xoo* mutants, one with a transposon insertion in *gum G* and another with a spontaneous mutation due to the insertion of endogenous IS element in *gum M*, were incapable of EPS production and less virulent. In *Xoo*, EPS synthesis has been found to be controlled by the *rpfC* gene, which is part of a two-component system. Strains carrying a mutation in *rpfC* have greatly reduced EPS production and virulence but still attain maximum population levels in rice plants [130] indicating that EPS is a virulence determinant in *X. oryzae* pv. *oryzae*.

Recently, screening of a transposon mutant library of a Korean *Xoo* strain, KACC10331, in rice also showed that Tn5 insertion in the *xrvA* gene (*XOO*2744) led to reduced virulence; however, the mutant was not characterized in further detail [139]. The deduced protein encoded by *xrvA* possesses an H-NS domain. H-NS and H-NS–like proteins are modular proteins associated with the bacterial nucleoid. The discovery of *xrvA* as a regulator of virulence factor synthesis came from work aimed at identification of genes involved in EPS production of *Xoo*. Disruption of *xrvA* led to a significant reduction in virulence, a delay in HR elicitation, a decrease in EPS and DSF production, and an increase in glycogen accumulation.

3.4. Type I secretion systems

Type I secretion systems of Gram-negative bacteria are secretion systems that transport proteins directly to the extracellular environment from the bacterial cytoplasm through inner and outer bacterial membranes. Three highly conserved components of type I secretion systems are an ABC transporter, which forms a channel across the inner membrane, a membrane fusion protein (MFP), and an outer membrane protein called To1C [140]. P*XO*99A contains a type I secretion system that is involved in triggering resistance in rice cultivars which carry the *Xa21* resistance gene. Transposon-induced mutations in P*XO*99A and subsequent screening for mutants that lost *Xa21*-mediated avirulence activity identified eight genes *viz. raxA*, *raxB*, *raxC*, *raxST*, *raxQ*, *raxP*, *raxH*, and *raxR* [141]. *AvrXa21* activity requires the presence of *raxA*, *raxB*, and *raxC* genes, and these three genes encode the MFP, ABC transporter, and outer

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membrane protein. The *AvrXa21* pathogen-associated molecule is involved in quorum sensing and the expression of *raxST* is regulated by a two-component regulatory system encoded by *raxH* and *raxR* [142] that responds to *Xoo* cell population density, and may be conserved in most *Xanthomonas* spp. The *raxST* encodes a sulphotransferase enzyme, while *raxQ* and *raxP* are involved in the production of the sulfuryl donor phosphoadenosine phosphor sulfate (PAPS). The elicitor of *Xa21* immunity, *Ax21*, was characterized as a 194 amino acid sulfated protein, which is secreted into the extracellular environment [143]. The N-terminal 17 amino acid peptide of *Ax21* is sulfated at a tyrosine residue and is sufficient to trigger *Xa21*-mediated resistance.

4. Molecular mechanism of BB resistance in rice

Out of 42 *Xa* genes identified so far, nine genes (*Xa*1, *Xa3*/*Xa26*, *xa5*, *Xa10 xa13*, *Xa21*, *Xa23*, *xa25*, and *Xa27*) have been characterized at molecular level and these encode various types of proteins (**Table 2**). Based on these studies, the molecular mechanisms of BB resistance in rice seem to be largely different from the mechanisms of resistance to rice blast, although the mechanisms of rice disease resistance remain largely to be elucidated. Most of the characterized BB resistance genes are different from the most common R protein, nucleotide-binding site-leucine-rich repeat (NBS-LRR) protein [144]. Interestingly, except for the fact that Xa21 and Xa26 encode for similar receptor-like proteins, the products of the other genes are unique and not found in other plant species [145]. These features suggest that molecular mechanism of rice-*Xoo* system is more complicated and a unique pathosystem to study the interactions between hosts and pathogens. The molecular mechanism of BB resistance gene has also been discussed in other book chapters [146, 147]. The nine characterized BB resistance genes fall into six different classes of proteins and thus may give a wide scenario of understanding at molecular level.

4.1. BB resistance conferred by LRR receptor kinase protein

The LRR receptor kinase class of BB resistance is conferred by *Xa21* and *Xa3*/*Xa26* genes. The *Xa21* was the first rice BB resistance gene characterized [7] and was one of the most intensively studied genes at molecular level. It was originally identified in the wild species *O. longistaminata*, and isolated by map-based cloning strategy from the IRBB21, a near-isogenic line in the background of IR24. This gene confers a race-specific resistance to *Xoo*, and is the most widespread BB resistance gene in the rice cultivated area, thereby providing broad-spectrum resistance. The receptor kinase-like protein encoded by *Xa21* consists of putative extracellular domain containing LRR, a single pass transmembrane domain, and an intracellular domain containing serine/threonine kinase. This protein is unique in carrying the receptor domain LRR with hypothetical function in pathogen recognition, and the kinase domain that functions in subsequent signal transduction as compared to other cloned plant resistance genes [148]. The *Xa21*-mediated resistance is not expressed in the early developmental stages and gradually increases from the seedling stage to later stages,

S. No.	Gene	Encoded protein	Reference
1	*Xa*1	NBS-LRR	[10]
2	*Xa*3/*Xa*26	Leucine-rich repeat receptor-like kinase (LRR-RLK)	[8]
3	*xa*5	TFIIA Transcription factor	[11, 12]
4	*Xa10*	Executor R protein, encodes 126 AA, with four potential transmembrane helices	[20]
5	*xa*13	MtN3/saliva	[14]
6	*xa21*	Receptor-like kinase	[7]
7	*Xa23*	Executor R protein, encodes 113 AA, with four potential transmembrane helices	[21]
8	*xa25*	MtN3/saliva	[19]
9	*Xa27*	Apoplast (rice unique gene)	[13]

Table 2. The BB resistance genes characterized at molecular level.

with 100% resistance at the adult stage [149]. The gradual increase in expression of *Xa21* gene during rice development is associated with development-controlled *Xa21*-mediated resistance [150]. Ectopic expression of *Xa21* gene can generate rice plants with a high level of resistance to *Xoo* at both seedling and adult stages [150, 151]. The *Ax21* (avir*Xa21* protein; as called activator of *Xa21*) protein was secreted by *Xoo* through its type I secretion system switch on the *Xa21* gene present in the host [145]. A sulfated 17-amino acid synthetic peptide derived from the N-terminal region of *Ax21* is sufficient for its initiation. The *Ax21* is highly conserved in many *Xanthomonas* species including number of pathogens of plants and human across microbial genus. Thus, *Ax21* is considered a pathogen-associated molecular pattern, and thus, *Xa21* can be classified both as a plant pattern recognition receptor (PRR) and an R protein [145].

The *Xa*21 locus is a multigene family and several key components of *Xa21* protein such as E3 ubiquitin ligase/XB3, WRKY62/XB10, protein phosphatase 2C (PP2C)/XB15, ATPase/XB24, and Bip3 (also known as glucose-regulated protein), involved in defense signaling pathway have been identified. The E3 ubiquitin ligase interacts with the kinase domain of *Xa21* protein and acts as a substrate for the *Xa21* serine and threonine kinase activity, which is necessary for full accumulation of the *Xa*21 protein, thus *Xa21*-mediated immunity [152]. The *Xa21* protein binds to WRKY62 with catalytic activity of its juxtamembrane motif and serine/threonine kinase domain. The WRKY62 and WRKY76 (another WRKY transcription factor) function as a negative regulator of *Xa21*-mediated resistance [153, 154]. The PP2C component interacts with juxtamembrane motif and kinase domain of *Xa21* protein, and can dephosphorylate autophosphorylated *Xa21*, thus acting as negative regulator of *Xa21*-mediated resistance [155]. The XB24, an *Xa21* binding protein, was isolated using yeast two-hybrid screening and belongs to a class of ATPases. The ATPase/XB24 can enhance autophosphorylation of *Xa21* protein by its physical association *in vivo* with the juxtamembrane motif and kinase

domain, and downregulates the *Xa2*-mediated resistance *Xa21* protein by its enzymatic activity, because the activation of *Xa21* following interaction with pathogen-associated molecular pattern *Ax21* requires the dissociation of XB24 from *Xa21* or removal of autophosphorylation. The rice plants with reduced level of XB24 expression showed enhanced level of *Xa21*-mediated resistance, whereas *Xa21*-mediated resistance overexpressing the XB24/ATPase in rice plants showed lower level of *Xa21*-meditated resistance, as XB24 in this case cannot readily dissociate from *Xa21* and further binding of Ax21 with *Xa21* leads to conformational change in *Xa21* protein. The conformational change in *Xa21* protein exposes it to degradation in endogenous proteases leading to lower resistance 21 protein *a21* [156]. The endoplasmic reticulum chaperone Bip3 can interact with *Xa21* protein *in vivo*. Rice plants overexpressing Bip3 have decreased *Xa21* protein accumulation and inhibited *Xa21* protein processing, which results in compromised *Xa21*-mediated resistance [157].

The *Xa*3/Xa26 locus was isolated as *Xa26* from an *indica* rice cultivar Minghui 63 [8] and found to be similar to previously identified gene *Xa3*, therefore renamed as *Xa3*/*Xa26* [9]. It encodes a plasma membrane-localized LRR receptor kinase-type protein with an extracellular LRR domain, a transmembrane motif, and a cytoplasmic kinase domain [8]. It also confers a broad-spectrum resistance relative to *Xa21* and has been widely deployed in rice cultivars in China [158–160]. Two different alleles of *Xa3*/*Xa26* have been identified from "CC" genome of *O. officinalis* and *O. minuta* that can mediate a similar spectrum of resistance against *Xoo* [160]. This indicated the early origin and relatively conserved function of *Xa*3/*Xa*26 locus during evolution that can provide durable resistance against *Xoo* [160]. The MRKa gene, an ortholog of *Xa*3/*Xa*26 gene family in rice cultivar Mingui 63, can mediate partial resistance to *Xoo* when it is overexpressed [161]. The kinase domain is important for complete function of *Xa*3/*Xa*26 for resistance, as rice plants carrying truncated *Xa*3/*Xa*26 gene without kinase domain exhibits lower level of resistance. The kinase domain of MRKa protein can partially restore the function of the truncated *Xa*3/*Xa*26 gene in *Xoo* resistance, suggesting the partially conserved function of the orthologs of this family. This hypothesis is also supported by another study that NRKe gene from rice cultivar Nipponbare gets transcriptionally activated in response to raised temperature [162]. The kinase domain of *Xa3*/*Xa26* protein can replace the function of the kinase domain of NRKe protein in response to temperature change.

The genetic background and development stage of rice plant affect the *Xa*3/*Xa*26-mediated resistance. The higher level of resistance was observed in *japonica* as compared to *indica* background. The *Xa3*/*Xa26* expression gradually increases from seedling stage to adult stage and pathogen infection also differentially effects its expression in plants with different genetic backgrounds, *Xoo* strains at both seedling and adult stages, but has full resistance to other *Xoo* strains at adult stage [8, 163, 164]. Further resistance mediated by *Xa3*/*Xa26* increases with increase in its expression and its constitutive expression provides broad-spectrum resistance at both seedling and adult stage without affecting the agronomic performance [159].

Several members functioning downstream in the *Xa3*/*Xa26*-initiated defense signaling pathway have been identified, where the downstream function of these components can mediate a broad-spectrum resistance as compared to *Xa*3/*Xa*26 protein. Two WRKY-type transcription

factors namely WRKY13 and WRKY45-2 positively regulate rice resistance to *Xoo*, of which *M. oryzae* (causal organism of rice blast), with WRKY13 putatively functions upstream of WRKY45-2 in rice-*Xoo* interaction [165–167]. Two genes namely OsDR8 encoding a protein involved in thiamine biosynthesis and C3H12, a CCCH-type zinc finger nucleic acid-binding protein, also act as positive regulator of rice resistance to *Xoo* in *Xa3*/*Xa*26-initiated defense pathway [152, 168]. Of these, C3H12 functions upstream of WRKY45-2. A rice tribe-specific gene OsDR10 that probably functions upstream of WRKY13, negatively regulates resistance to *Xoo* in *Xa3*/*Xa26*-mediated resistance pathway [169].

Both *Xa*21 and *Xa3*/*Xa26* belong to multigene family; encode same type of proteins and have 53% sequence similarity [7, 8]. The only structural difference between two genes is the number of LRR, where *Xa26* encodes 26 LRR, whereas *Xa21* encodes 23 LRR [7]. However, the respective LRR domains of *Xa3*/*Xa26* and *Xa21* are the important determinants of race-specific recognition during rice-*Xoo* interactions as evidenced from experiment on domain swapping analyses, but a juxtamembrane motif of *Xa*3/*Xa*26 also seems to contribute in resistance specificity [150]. The kinase domain of *Xa*3/*Xa*26 can partially replace the function of the kinase domain of *Xa21*, or vice versa, in *Xoo* resistance, suggesting the partially conserved nature of this domain in defense signaling pathway [150]. Both *Xa3*/*Xa26* and *Xa21* genes are dose-dependent and their expression progressively increases with developmental stage. However, *Xa3*/*Xa26* has a higher expression level in the *japonica* background than in an *indica* background [150, 164]. Further study may also be required to determine, whether *Xa3*/*Xa26* is also a PRR in addition to being an R protein.

4.2. BB resistance conferred by MtN3/saliva class protein

The MtN3 (a homologue of nodulin protein) class of BB resistance is conferred by *xa*13, a fully recessive gene for BB resistance. The gene encodes a novel protein that has no sequence similarity with any known *R* proteins, but it shows 50% sequence identity and 68% sequence similarity to the product of a nodulin MtN3 gene in legumes [14]. The *xa*13 and its dominant allele *Xa13*, which is also named *Os8N3* and OsSWEET11 [64, 122], encode identical polytopic plasma membrane proteins of the MtN3/saliva family proteins, but have crucial sequence differences in their promoter regions. Promoter swapping analysis confirms that *Xa13* (*Os8N3* and OsSWEET11) is a susceptibility gene, which is induced by direct binding of the transcription activator-like (TAL) effector Pth*Xo*1 of *Xoo* strain P*XO*99 to the cis element, the UPT Pth*Xo*l box, on the *Xa13* promoter [64, 170, 171]. The *Xoo* strain P*XO*99 that secretes Pth*Xo*1 cannot induce recessive *xa13* due to the mutation of the UPT Pth*Xo*1 box in the *xa13* promoter. Further, P*XO*99 is more sensitive to copper (an essential micronutrient of plants, is also an important element for a number of pesticides in agriculture) than other *Xoo* strains. The *Xa13* protein interacts with two copper transporter-type proteins, COPT1 and COPT5, to promote removal of copper from xylem vessels, where *Xoo* multiplies and spreads to cause disease [172]. As P*XO*99 cannot induce recessive *xa13*, the copper levels in rice plants carrying the recessive *xa*13 gene can inhibit *Xoo* growth and thus plants show resistant reaction. Besides functioning against *Xoo* resistance, product of *xa13* gene also has an essential role in pollen development. This became evident from suppressing the function

of either the dominant or recessive allele of *xa13* in rice transgenic plants, where *xa13* not only enhanced the resistance but also caused male sterility [173]. The study showing the link between two unrelated biological processes further demands the detailed studies in the future on the functional overlap between pathogen-induced defense signaling and plant development.

The recessive *xa25* gene, also named *Xa25* (t), encodes a plasma membrane protein of the MtN3/saliva family similar to *xa13* and confers race-specific resistance to Philippine *Xoo* strain PXO339 [19, 83]. The protein encoded by *xa25* and its dominant allele differ in eight amino acids. The *Xoo* strain PXO339 can induce the expression of dominant *Xa25*, but not recessive *xa25*. The differences in proteins and expression pattern of *xa25* and its dominant allele *Xa25* in rice-P*XO*339 interaction suggest that the dominant *Xa25* may be a race-specific susceptible gene, whereas the recessive *xa25* has evolved as the mutant that cannot be induced by rice-*Xoo* interaction—similar to the recessive *xa13*. The developmental stage of rice plant also influences the *xa25*-mediated resistance. The *xa25* gene regulated by its native promoter, when transferred in the rice plants homozygous for *Xa25*, behaves in recessive nature. The rice plants homozygous for *xa25* showed resistant reaction on inoculation with P*XO*39. However, the rice plants heterozygous at the *xa25* locus were susceptible to P*XO*339 at the seedling stage, but became resistant to P*XO*339 at the adult stage. The dominance reversal characteristic of *xa25* may be because of suppression of P*XO*339-induced activation of dominant *Xa*25 at the adult stage [19]. The rice MtN3/saliva family contains more than 20 paralogs. Some MtN3/saliva proteins from different species act as glucose transporter [122, 174]. The *Xa13*, besides functioning as a susceptibility gene in race-specific rice-*Xoo* interaction, also acts as sucrose transporter. Similar role needs to be elucidated for *Xa25* gene along with rice-*Xoo* interaction.

4.3. BB resistance conferred by TAL effector-dependent class protein

Three BB resistant genes *Xa27*, *Xa10*, and *Xa23* act as transcription activator-like (TAL) effector-dependent *R* genes [13, 20, 21]. The *Xa27* mediates race-specific resistance to diverse strains of *Xoo*, including Chinese and Philippine *Xoo* races [13]. This gene encodes an apoplast protein of 113 amino acids that has no distinguishable sequence similarity to proteins from organisms other than rice [175]. The resistant and susceptible alleles of *Xa27* encode an identical protein, but they differ from each other only in the promoter region [13]. The *Xa27*-mediated race-specific resistance to *Xoo* depends upon *AvrXa27*, a TAL effector of *Xoo* that induces resistance reaction by binding to the UPTAvr*Xa*27 box of the *Xa27* promoter [176]. However, the recessive MR gene *xa5* can attenuate the *Xa*27-mediated resistance in rice, which suggests that *Xoo* TAL effector could not use protein encoded by the recessive *xa5* as a transcription machinery for activation of *Xa27* [177]. The secondary cell wall thickening in vascular bundle elements is obviously associated with *Xa*27-mediated resistance [13]. According to the molecular models of plant innate immunity, pathogen-associated molecular pattern-triggered immunity (PTI) is induced by plasma membrane-localized plant pattern recognition receptors (PRRs) and effector-triggered immunity (ETI) is initiated by NBS-LRR–type R proteins present in the cytoplasm [178, 179]. The subcellular localization and sequence specificity of *Xa27* protein

suggest that it does not follow either PTI or ETI procedure [180], though its typical biochemical function in rice-*Xoo* interaction still remains to be elucidated.

Xa10, another TAL effector-dependent *R* gene for BB resistance contains a binding element for the TAL effector AvrXa10 (EBEAvrXa10) in its promoter, and AvrXa10 specifically induces *Xa10* expression [20]. The *Xa10*-encoded protein consists of 126 amino acid residues, which is predicted to have four potential transmembrane helices (M1–M4), among which, M2 contains charged amino acid residues. *Xa10* is an inducible protein that triggers programmed cell death by disruption of the endoplasmic reticulum (ER) and cellular Ca^{2+} homeostasis. The *Xa10* functions not only in rice but also in other species such as *Nicotiana benthamiana*, and in animal kingdom such as mammalian HeLa cells [20]. The *Xa10* protein localizes as hexamers in the ER and is associated with ER Ca^{2+} depletion in plant and HeLa cells. It shows hypersensitive response with faster cell death and shorter lesions in incompatible reactions. The variants of *Xa10* that abolish programmed cell death and ER Ca^{2+} depletion in *N. benthamiana* and HeLa cells, also abolish disease resistance in rice [20]. In one of the experiment, the modified *Xa10* gene designated as $XA10^{E5}$ was transferred into Nipponbare and 93-11 backgrounds to generate broad-spectrum resistance to most of the BB pathogens. The $XA10^{E5}$ contains five tandemly arranged effector binding elements (EBEs), each responding specifically to a corresponding virulent or avirulent TAL effector. The transgenic lines with $XA10^{E5}$ showed resistance to 27 of the 28 *Xoo* strains collected from 11 countries [181].

Xa23, another TAL effector associated executor *R* gene, was isolated from *O. rufipogon* using map-based cloning and TAL effector-based technology [21]. It encodes 113 amino acid long proteins (same number of amino acids as in *Xa27*) with 64% similarity only to *Xa10* protein, but nonsignificant similarity at genomic level. In addition, the predicted three transmembrane helices of *Xa23* largely overlap with transmembrane helices (M_2–M_4) of *Xa10* indicating that Xa23 could be a homolog of *Xa10* [21]. A paralog of *Xa23* has been found to exist in Nipponbare genome with unknown function. The AvrXa23 is responsible for activation of *Xa23* transcription by a TAL effector present in all examined *Xoo* isolates [21]. As compared to *Xa10*, *Xa23* is a broad-spectrum *R* gene and provides strong resistance to all natural *Xoo* strains tested so far possibly due to presence of avrXa23 in natural *Xoo* strains [182]. The susceptible *xa23* allele *Xa23* but differs in promoter region by lacking the TALE binding element (EBE) for AvrXa23. *Xa23* can trigger a strong hypersensitive response in rice, tobacco, and tomato [21].

4.4. BB resistance conferred by NBS-LRR class proteins

Xa1 is the only NBS-LRR–type BB resistance gene identified so far and is an inducible gene that means its expression is induced by wounding and pathogen infection [10]. However, out of number of uncharacterized BB resistance genes, some might be providing resistance through NBS-LRR class of proteins. One such gene is *Xa38*, identified from *O. nivara* seems to be of NBS-LRR type based on the putative candidate gene sequence analysis in the targeted bacterial artificial clone (BAC) [183]. Nucleotide-binding site-leucine-rich repeat (NBS-LRR) genes play important roles in plant disease resistance. They are generally believed to be

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responsible for the recognition of effectors delivered by pathogens during infection and the induction of downstream disease resistance reactions [184]. Plant NBS-LRRs can be divided into several types based on the variable N-terminus. In rice, most NBS-LRRs belong to the CC-NBS-LRR (CNL) subclass, in which there is a coiled-coil domain at the N-terminus [185, 186]. In the genome of rice (*O. sativa* cv. Nipponbare), more than 400 NBS-LRR genes have been identified [19], but although these genes are believed to be defense-related, the functions of most of them are unknown in rice genome. Recently, a novel NBS-LRR gene was isolated from rice and designated as *Oryza sativa* Rp1-like 1 (OsRP1L1) based on similarity to Rp1 locus of maize, an intensively studied NBS-LRR gene providing resistance to common leaf rust in maize [187]. Overexpression of OsRP1L1 moderately elevated the resistance of plants to *Xoo* strains P*XO*86 and P*XO*341 in a susceptible *japonica* cultivar [188]. Similarly, a large number of genes out of 400 NBS-LRR in the rice genome can be isolated that could be putative candidates for BB resistance.

4.5. BB resistance conferred by other class of protein

The recessive *xa5* that belongs to this class encodes a typical gamma subunit of transcription factor IIA (TFIIAγ), which is one of general transcription factors required for transcription by RNA polymerase II [11]. It mediates specific resistance to Japanese races and Philippine races 1, 2, 3, and 5 by restriction of bacterial movement, but not multiplication [11, 189]. This gene was cloned by a map-based cloning approach combined with allele sequence analysis [11], and further complementation testing confirmed this gene [12]. TFIIAγ is involved in the recruitment of the basal transcription machinery by eukaryotic transcription factors. The *xa5* allele contains a missense mutation that does not seem to influence its function in the recruitment of the basal transcription machinery [12]. It is speculated that *Xoo* TAL effectors usurp parts of plant basal transcription machinery to regulate rice gene expression; the missense mutation of *xa5* allele does not compromise its general function in transcription, but it may evade TAL virulence functions [119, 177]. The *Xoo avrXa5* is an avirulence gene, which encodes a TAL-type protein, corresponding to *xa5* [190]. The *xa5* showed a constitutive expression pattern in different tissues, and the resistance of *xa5* is not dose-dependent [11, 12].

5. Conclusion

Exploration, identification, and utilization of new resistant germplasms in rice breeding are the strategical steps to control the bacterial blight disease of rice. The *Xa21* gene has been successfully introgressed into several elite rice varieties and hybrid rice parental lines all over the world either singly or in combination with other major resistance genes such as *Xa*4, *xa*5, and *xa*13 [27–32, 191]. Traditionally, single recessive genes are overlooked in pyramiding plans since they have to be present in the homozygous condition, which is more difficult to achieve than a heterozygous- or homozygous-dominant genotype. But, in the last decades, rice genomic research has generated a wealth of information about gene function. These advances are now accessible for rice improvement, and have been applied in MAS and genetic engineering in breeding programs. Gene silencing also paves a way to utilize

these genes more efficiently. Artificial microRNA (amiRNA) technology has been developed to silence the dominant allele of *xa13*, allowing the recessive allele to be unmasked, thereby expressing the resistant phenotype, mimicking a homozygous state. This silencing conferred a higher degree of resistance to the rice line without affecting other essential traits, such as fertility [192]. Hummel et al. [99] concluded that genetic engineering of the *R* gene promoter can also effectively promote resistance towards *Xoo*. Many *R* genes are turned on in the presence of TALE released from the pathogen by binding to a specific effector binding element (EBE) and initiating the resistance response. So, by fusing new EBEs to the promoter of *Xa27*, gene transcription could be initiated by bacterial races other than the races that normally activate *Xa27*. Therefore, the *Xa27* gene will respond to a greater spectrum of pathogens, triggering resistance. Also, the addition of the EBEs resulted in resistance against a pathovar of *Xanthamonas* which is the causative agent of bacterial leaf streak, for which no *R* genes have yet been identified. This method creates a single gene with the expanded spectrum of traditionally pyramided lines but is less time-consuming and laborious than creating multigenic lines. Several EBEs could be added to a single promoter, greatly increasing the number of pathogens which initiate resistance. Thus, there are numerous factors impacting the resistance granted by different genes, and a complete understanding of the host-pathogen interaction dynamic would result in a better equipped scientific community.

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