Molecular Approaches for Disease Resistance in Rice

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Abstract Rice production needs to be sustained in the coming decades, with changing climatic conditions becoming more conducive to the prevalence of disease outbreaks. Major rice diseases collectively cause enormous economic damage and yield instability. Breeding for disease-resistant rice varieties could be one of the best options to counter these disease outbreaks. Disease-screening protocols and newer technologies are essential for effective phenotyping and would aid in gene discovery and function. Understanding the genetics of disease mechanisms and stacking of broad-spectrum disease-resistance genes could lead to faster development of rice varieties with multiple disease resistance. New molecular breeding approaches are discussed for the development of these varieties. The molecular biology of disease resistance is now better understood and could be well manipulated for improved resilience. Transgenic approaches for disease resistance are discussed. Genome-editing tools for the development of disease-resistant rice varieties are thoroughly discussed. The use of bioinformatics tools to speed up the process and to obtain a better understanding of molecular genetics mechanisms of disease resistance is explained.

Keywords Rice · Biotic diseases · Phenotypic screening · QTLs and genes · Breeding strategies · Genome editing

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1 Introduction

Rice (*Oryza sativa* L.) is a staple and the most crucial food security crop in the world. It plays a vital role in the human diet and feeds more than 50% of the world's population (Rathna Priya et al. 2019). By 2050, global demand for rice is projected to rise more than 40% to feed the rapidly growing world population (Milovanovic and Smutka 2017). Despite impressive global increases in production from 289 million tons in 1968 to 782 million tons in 2018, this quantum jump still has to keep pace with demand for rice from the rising population (FAOSTAT 2020). At present, rice cultivation throughout South Asia and in ASEAN countries is facing significant threats because of a few major biotic stresses (Yugander et al. 2017). Approximately 52% of the global productivity of rice grain yield is severely damaged by biotic factors, of which nearly 31% is due to various diseases such as bacterial blight (caused by *Xanthomonas oryzae*), blast (caused by *Magnaporthe grisea*), sheath blight (caused by *Rhizoctonia solani*), and tungro disease (tungro bacilliform virus and tungro spherical virus) (Park et al. 2008). Detailed information about the symptoms caused by these major diseases, along with the favorable conditions required by these pathogens and yield losses incurred, is presented in Table 1. The severity of biotic stresses in rice production is increasing at a startling pace of late because of rapid changes in climate (Jamaloddin et al. 2020). Changing climatic conditions are contributing to the emergence of new virulent races and the occurrence of diseases in new localities. Many diseases considered as minor thus far have become economically significant in many rice-cultivating areas and are exacerbating their impact (Anderson et al. 2004). According to Zhang et al. (2009), rice crops are affected by around 70 pathogens, especially viruses, bacteria, fungi, and nematodes. Estimated yield loss because of pathogens globally and as per hotspot range for rice is 30% (24.6–40.9%) (Savary et al. 2019). Over the past 150 years, many rice diseases have caused outbreaks and spread rapidly in different parts of the world. Rice diseases were observed for the first time in different locations, such as bacterial blight and sheath blight in Japan during 1884–1885 and 1910, respectively; false smut in the United States during 1906; blast in Africa during 1922; rice tungro in the Philippines during 1940; rice brown spot in India during 1942; bacterial leaf streak in India during 1963; and rice yellow mottle disease in Kenya during 1966. These diseases, along with a few newly emerging epidemics, are becoming a significant threat to rice production.

Despite so many alternatives for crop protection, plant pathogens still pose a challenge to agriculture. Several management practices have been adopted to decrease their impact, such as chemical control, biological control, optimum fertilizer application, appropriate planting dates, and disease forecasting. However, not all of these methods are environment-friendly and alone are not enough to control the diseases completely. The present situation thus requires environment-friendly and cost-effective modern technologies such as the development and cultivation of disease-resistant cultivars. The development of these varieties using only conventional breeding methods consumes a lot of time, land, and labor. In this context,

Table 1 Key features of major diseases in rice

Disease	Pathogen	Symptoms	Favorable conditions	Yield loss	Reference
Blast	Caused by the fungus *Magnaporthe oryzae* (*Mo*)	Early-stage symptoms appear as white to gray-green lesions with dark green specks. These soon enlarge and spindle-shaped lesions appear with a gray center and dark brown margin.	Prolonged period free from moisture. High humidity conditions. Gentle or no wind at night. Night temperatures from 17 to 22 °C. High rate of fertilizer.	70–80%	Jamaloddin et al. (2020)
Bacterial blight (BB)	Caused by bacterium *Xanthomonas oryzae* pv. *oryzae* (*Xoo*)	Normally, disease appears at heading stage, but can occur early in severe conditions. Infected plants' young leaves change from pale green to gray-green and roll up. As the disease progresses, the entire leaf may eventually be affected, becoming whitish or grayish and then dying.	Suitable temperature is 25–30 °C. High humidity (above 70%), rain, and deep water. Severe winds, which cause wounds. High rate of fertilizer.	Up to 50%	Liu et al. (2014)
Bacterial leaf streak (BLS)	Caused by bacterium *Xanthomonas oryzae* pv. *oryzicola* (*Xoc*)	Plants can be affected from maximum tillering to panicle initiation. Symptoms appear on leaf blades as narrow, dark greenish water-soaked interveinal streaks of various lengths. Later, these streaks become light brown to yellowish gray.	There is a higher probability of developing it in areas having weeds and stubbles harboring infection. Temperatures from 25 to 34 °C with relative humidity >70% are more congenial.	8–32%	Liu et al. (2014)

(continued)

Table 1 (continued)

Disease	Pathogen	Symptoms	Favorable conditions	Yield loss	Reference
Tungro	Caused by Rice tungro bacilliform virus (RTBV) and rice tungro spherical virus (RTSV)	Infection can occur during all growth stages but mostly during the vegetative phase. The tillering stage is the most vulnerable. Leaves of infected plants become yellow or orange-yellow and may also have rust-colored spots. Most of the panicles are entirely or partially sterile with ill-filled grains.	Viruses are transmitted by leafhoppers that feed on tungro-infected plants. Leafhoppers are capable of transmitting viruses to other plants within 5–7 days.	Up to 100%	Bunawan et al. (2014)
False smut	Caused by the fungus *Villosiclava virens* (anamorph: *Ustilaginoidea virens*)	False smut can infect individual rice grains. Only a few panicle grains are usually infected, and the remaining grains are normal. A smut ball appears at first and grows gradually up to 1 cm. As fungi growth intensifies, the smut balls burst and become orange and then later yellowish green/ greenish black in color.	The disease can occur in areas with high relative humidity (>90%) and temperature ranging from 25 to 35 °C. Rain and soils with high nitrogen content also favor false smut. Wind can spread the fungal spores from plant to plant.	In severe cases, tillers will be affected 85–100%.	Huang et al. (2019)

(continued)

Table 1 (continued)

Disease	Pathogen	Symptoms	Favorable conditions	Yield loss	Reference
Sheath blight	Caused by the fungus *Rhizoctonia solani*	The fungus attacks the plants from tillering to heading stage. Initial symptoms appear on leaf sheaths near the water line in the form of oval or irregular greenish gray lesions. Later, lesions extend to the upper parts of the plants and rapidly coalesce, covering entire tillers from the water line to the flag leaf.	Temperature from 28 to 32 °C, high rates of N fertilizer, high seed rate or low spacing, dense canopy, inoculum in soil or floating on the water, and continuous cultivation of high-yielding varieties favor disease development. The crop is more vulnerable during the rainy season.	20–60%	Molla et al. (2020)
Sheath rot	Caused by fungus *Sarocladium oryzae*	The sheath rot lesion starts at the uppermost leaf sheath consisting of young panicles within. Early symptoms are oblong to irregular lesions on the leaves with dark reddish brown margins and brownish gray throughout. The disease can cause partial emergence of panicles present in the infected sheaths. The unmerged panicles rot and turn dark brown with a whitish powdery growth inside the sheaths. Infected panicles and grains look sterile, ill-filled, shriveled, and discolored.	More prevalent during wet seasons than dry seasons. High relative humidity and temperatures from 20 to 28 °C from heading to crop maturity. High rates of N fertilizer application. Plant injuries and wounds caused by insects such as stem borers at the panicle initiation stage.	20–85%	Peeters et al. (2020)

molecular markers come to the rescue of plant breeders by helping them decrease the time between breeding and achieving the desired product. The discovery of DNA markers led to a new tool in plant breeding called marker-assisted selection (MAS), which is one of the widely used components of a discipline called molecular breeding. The application of DNA markers to plant breeding significantly increased its efficiency and precision. MAS is now one of the most advanced methodologies on hand for the transfer of one or more desired genes/genomic regions into elite rice varieties in more durable combinations. Deploying a single *R*-gene often leads to resistance breakdown in a short period as the pathogen evolves and makes itself resistant to the action of the gene. Therefore, pyramiding of multiple *R*-genes imparting resistance against different races of a pathogen through MAS is an efficient way to attain long-term and broad-spectrum resistance. Although this is an advanced method, it has some disadvantages. The main drawback of this approach is that one parent, or even both parents, used in the breeding program may carry quantitative trait loci (QTL) alleles that are either similar or exact to the ones present in the elite germplasm accessions used in other breeding programs. In such a situation, the QTL being introgressed may contribute only partially to the trait improvement. In other cases, the impact of a QTL may differ based on the genetic background as a result of interactions with other loci or epistasis (Holland 2001). Moreover, there are many more important traits for which no genes have been reported so far. In such situations in which a gene is not available in the gene pool, researchers are forced to look outside the gene pool toward other genera or sometimes toward another kingdom to find the desired gene.

Genetic modification (GM) technology has been developed to make changes to an organism's genes to give it new traits that would not occur in nature or to eliminate undesirable characteristics. GM technology using recombinant DNA technology is useful for developing disease-resistant varieties but still has not reached farmers because of a lack of public acceptance and political issues in many countries. Under these situations, researchers are left with an option to create mutations in the gene pool with an expectation to generate variation for a trait not naturally present in the gene pool.

Mutation breeding is helpful in creating novel mutants with genetic variations for plant breeding and functional genomics. It could be used for rice crop improvement programs. Mutation induction can be of advantage to produce cultivars with desired characteristics within defined germplasm pools. Normally, gamma-rays (γ-rays) and ethyl methane sulfonate (EMS) have been used extensively to develop rice mutants. In rice, there have been reports of some important mutant collections developed to carry out functional studies and Hirochika et al. (2004) made available a list of the mutant libraries. Madamba et al. (2009) found a gamma-ray-induced IR64 mutant, G978, that gave enhanced resistance to blast and bacterial blight. The resistance was found to be quantitative and nonrace-specific against bacterial and fungal pathogens. The mutation was shown to be inherited as a single recessive gene, *Bsdr*1, and it caused a shorter stature relative to IR64 and was mapped as a QTL to a 3.8-Mb region on chromosome 12. Comparison of the gene expression profiles of the mutant and wild type showed the candidate gene to encode a U-box

domain-containing protein. The disrupted gene exhibited a loss of expression in the mutant and cosegregated with the mutant phenotype (Madamba et al. 2009). These techniques of causing mutations have a problem of creating more undesirable than desirable phenotypes. In other words, these techniques result in random mutations in the genome. The frequency of variations can be controlled but not in the genomic region where they are desired to occur. To achieve a desirable outcome from these experiments, a large population of mutants has to be screened, and this requires a lot of time, space, and resources.

Ultimately, the new era of genome engineering technologies offers vast potential for crop improvement as they allow site-specific modifications of DNA sequences to be executed under laboratory conditions. The accessibility to vast genomic resources and an easy-to-handle genome size make rice more amenable for GM technologies. Advances in genomics and the development of various genome-editing technologies using engineered site-specific nucleases (SSNs) have made the application of genome engineering to crops easy. Among various SSNs, the CRISPR/Cas9 system is commonly applied because of its simplicity, robustness, and high efficiency (Wang et al. 2018). In comparison with other genome-editing tools such as zinc-finger nucleases and transcription activator-like effector nucleases (TALENs), this technique is versatile and simple (Ma et al. 2015b). This technology has been applied to agricultural crop plants with the aim of crop improvement. Oliva et al. (2019) used the CRISPR-Cas9 system to introduce mutations in three *SWEET* gene promoters to make robust and broad-spectrum bacterial blight-resistant lines. There is still much scope for its use and application.

In the future, the challenge for scientists is not only to develop rice varieties for specific diseases but also to select for horizontal resistance without altering other desirable traits of elite rice varieties. A systematically designed experiment involving highly efficient molecular tools would make it possible to achieve this outcome. Hence, the current chapter amalgamates details on the present status of the key diseases that affect rice production, various molecular strategies for attaining disease resistance, and prospects of molecular breeding for disease resistance in rice.

2 Phenotypic Screening Techniques for Major Diseases of Rice: Pathogen Inoculum, Plant Infection Assays, and Disease Scoring

2.1 Bacterial Blight

Bacterial blight (BB), caused by *Xanthomonas oryzae* pv. *oryzae* (*Xoo*), targets the seedling stage of rice, resulting in leaves turning grayish green and rolling up. Usually, BB inoculation can be done in two ways, either in the field or in the greenhouse. Many techniques are available to infect the plant with inoculum such as clipping, needle prick, paint-brush, and spray methods. But the most preferable,

efficient, and feasible for inoculation is the clipping method (Jabeen et al. 2011). Individually, collected *Xoo* strains are multiplied and stored on modified Wakimoto'sagar (Sundaram et al. 2009) and the selected rice plants at 45-days-old stage are clip-inoculated with a freshly prepared bacterial suspension (~$10^{8–9}$ cfu/mL) by the following method given by Kauffman et al. (1973). In this method, 1–2-cm tips of five leaves are clipped with scissors, after they were already dipped in bacterial suspension culture, and disease score is recorded 2 weeks post-inoculation both by visual scoring and by measuring the lesion length (LL) as per the Standard Evaluation System (SES) scale of the International Rice Research Institute (IRRI 1996) (0–3 = resistant, 3–5 = moderately resistant (MR), 5–7 = moderately susceptible (MS), and 7–9 = susceptible).

2.2 Blast Disease

The causal organism for blast disease is a fungus, *Magnaporthe oryzae* (*Mo*). Symptoms of blast can appear during any developmental stage and on all parts of the rice plant, including leaves, leaf collars, necks, panicles, pedicels, and seeds. Standard screening protocols of rice varieties for susceptibility to rice blast are usually carried out by spraying the plant with conidial suspensions under greenhouse and field conditions using local isolates of the pathogen (Takahashi et al. 2009). However, for screening against exotic strains, quarantine restrictions are frequently applied to control any escape of the pathogen into the surrounding environment (Jia et al. 2003). In field conditions, artificial leaf blast disease screening usually takes place in a Uniform Blast Nursery (UBN) (Jamaloddin et al. 2020). Applying an excess rate of nitrogen fertilizer (150 kg N/ha) makes rice more vulnerable to spreading blast infection. Artificial inoculation is done with a highly virulent blast race (fungal conidial suspension at a concentration of 1×10^5 spores/mL) by spraying on UBN beds 25–30 days after sowing (DAS). Later, the beds are covered with polythene sheets during the night to create humid conditions for disease development. The disease score is collected 10–15 days after infection, depending on the severity of the infection on the susceptible check using the SES (IRRI 1996). In in vitro conditions, spot inoculation and filter paper inoculation methods are used for inoculation at the vegetative and reproductive stages of rice plants (Jia et al. 2003; Takahashi et al. 2009).

2.3 Sheath Blight

Sheath blight (ShB) disease is caused by a fungus, *Rhizoctonia solani*. The fungus attacks the rice plant from tillering to heading stage. The early symptoms of sheath blight involve oval circles on leaves just above the waterline. Various screening methods have been developed to screen for ShB in greenhouse and field conditions.

Eizenga et al. (2002) delineated a growth-chamber screening technique for sheath blight on *Oryza* spp. Later, Jia et al. (2003) developed the detached-leaf method. For screening ShB under greenhouse conditions, three inoculation methods have been described: liquid-cultured mycelia ball, mycelia suspension, and agar block. Out of these, the liquid culture mycelia ball is a more efficient and better method for successful inoculation (Park et al. 2008). Field screening at the reproductive stage is the most commonly used method. But field trials require a lot of labor and a large amount of seed material, inoculum, and high-humidity conditions for up to 3–5 months to complete the evaluations (Jia et al. 2007). Normally, screening of selected material for ShB tolerance/susceptibility is done using a highly virulent isolate of *Rhizoctonia solani*. Initially, ShB isolate is maintained on a potato dextrose agar (PDA, extract from 200 g/L of potato, 20 g/L of dextrose, and 20 g/L of agar) plate and incubated at 28 °C in darkness. For plant inoculations, *Typha* stem pieces (3–4 cm) are cut and autoclaved in plastic covers. This sterile *Typha* is inoculated with a 5-mm mycelial plug of *R. solani* from a 3-day-old PDA plate and incubated in the dark for 10 days at 28 °C. The colonized *Typha* pieces will be used for inoculating the rice plants at a rate of three to four pieces per hill.

Disease phenotype will be scored 2 weeks after inoculation by measuring the relative lesion height (RLH) as per the following formula:

$$\mathrm{RLH}(\%) = (\text{Lesion length} / \text{Plant height}) \times 100$$

The IRRI (1996) phenotype scale is used to classify the plants based on their disease severity index from 0 to 9.

2.4 Sheath Rot of Rice

Sheath rot (ShR) is a symptom that is observed in rice plants when infected by any of the following pathogens: *Sarocladium oryzae*, *Fusarium* sp., *Pseudomonas* sp., and *Cochliobolus lunatus*. Other pathogens have been reported to cause similar symptoms in rice (Bigirimana et al. 2015). Multiple screening techniques that are being used for sheath rot disease resistance in rice include the mealybug inoculation method, rice grain/hull inoculum, leaf piece inoculum, cotton swabbing of conidial spores, spraying or injecting conidial suspension on the sheath, and detached tiller–based assays (Mahadevaiah et al. 2015; Samiyappan et al. 2003). The established screening methods differ depending on the causal agent of ShR as well as the growth stage of the plant. The pathogen is cultured on PDA plates for up to 14 days at 28 °C (Panda and Mishra 2019). A study by Mahadevaiah et al. (2015) compared multiple inoculation methods for *Sarocladium oryzae* during different growth stages and observed that seed inoculation is a suitable screening method for screening for disease resistance in young plants or early infection. In this method, the seeds are soaked overnight in conidial suspension (10^5 spores/mL) and then germinated. The number of germinated plants and lesion lengths 14 days after inoculation are scored.

For screening plants in peak vegetative to booting stage, foliar inoculation methods were able to provide reliable and conclusive results 15 days after infection. For a faster in vitro screening, the detached-tiller assay is recommended, in which the tillers are cut and placed on moist paper and inoculated with mycelial mats. Visible lesions are observed as early as 3 days postinoculation (Samiyappan et al. 2003; Mahadevaiah et al. 2017). Disease severity is estimated by measuring the area of the sheath and/or leaf affected (Mahadevaiah et al. 2017). *Pseudomonas fuscovaginae* also causes ShR in rice (Bigirimana et al. 2015). Adorada et al. (2013) reported and recommended multiple screening techniques for screening ShR caused by *P. fuscovaginae*. The bacteria are cultured using King's medium B initially for about 24 h. For plant inoculations, the following methods were found to be effective: (1) pinpricking the upper leaf sheath using a needle dipped in bacterial suspension (10^7 cfu/mL) and measuring disease severity 14 days postinoculation in plants at the booting stage; (2) spraying the inoculum was found effective and is recommended for mass screening for ShR resistance in plants at the booting stage; (3) for early-stage resistance, soaking seeds in bacterial inoculum before germination is recommended, followed by measuring the decrease in seedling height 10 days later (Adorada et al. 2013).

2.5 False Smut

The fungus *Ustilaginoidea virens* causes false smut of rice. This fungus attacks the developing panicles and leads to the formation of smutted balls (cottony flakes around the grains). The fungus is generally cultured on potato sucrose agar plates or potato sucrose broth for mass production of conidial suspension (Panguluri and Kumar 2013). Screening for false smut is done during the booting stage of the plants through the following methods. Spraying conidial suspension (5×10^4 spores/mL) at the booting stage is one of the recommended ways for screening for false smut (Kaur and Singh 2017). Another method involves injecting the conidial suspension into the boot (Panguluri and Kumar 2013; Kaur 2014). It has been observed that spraying spores has produced a higher disease incidence and this is suitable for screening for resistant varieties (Kaur 2014). Disease severity is scored by calculating the percentages of infected tillers and infected grains per panicle and a score is assigned as recommended by Rice SES (IRRI 2013; Chaudhari et al. 2019).

2.6 Tungro Disease of Rice

Rice tungro is caused by two viruses, RTBV (rice tungro bacilliform virus) and RTSV (rice tungro spherical virus), and is transmitted by green leafhopper (GLH: *Nephotettix virescens* (Dist.)). The viral infection is manifested by the stunted growth of rice plants and yellowing of leaves (Anjaneyulu et al. 1982; Panguluri and

Kumar 2013). Nursery screening for tungro resistance in rice is carried out by letting three to five viruliferous GLH per plant (20–30 days old) feed in a closed environment and scoring the disease symptoms 14 days later as recommended by Rice SES (Anjaneyulu et al. 1982; Sebastian et al. 1996).

2.7 Bacterial Leaf Streak

Bacterial leaf streak (BLS) of rice is caused by the bacterium *Xanthomonas oryzae* pv. *oryzicola* (*Xoc*). BLS is manifested as water-soaked lesions on the leaf surface, which can result in decreased photosynthesis and hence diminished yield (He et al. 2012). Screening for BLS resistance is mainly performed using either of two methods. For screening seedlings, bacteria are initially grown in peptone sucrose broth, and a bacterial suspension from 10^8 to 10^9 cfu/mL is used for infiltrating the expanded leaves using a needleless syringe. The disease symptoms are scored 14 days postinoculation (Ju et al. 2017). For field screening or screening older plants, matured leaves are pin-pricked with needles that are dipped in bacterial inoculum on either side of the leaves. The lesions caused are measured 20 days postinoculation (Tang et al. 2000; Chen et al. 2006a, b; He et al. 2012). Disease severity is scored as per Rice SES.

3 Genetics of Disease Resistance

Deployment of genes conferring host-plant resistance provides an economical, durable, effective, and environmentally safe approach to combat plant diseases and decrease yield losses (Fig. 1). Major resistance genes from different resistance donors have been reported for various rice diseases. So far, more than 44 resistance genes have been identified against bacterial blight (Kim and Reinke 2019). More than 100 distinctive blast-resistance genes have been reported on different rice chromosomes and, out of these, 21 genes have been cloned (Devi et al. 2020). Two major sheath blight QTLs (*qShB9-2* and *qSBR11-1*) have been reported (Channamallikarjuna et al. 2010). But, thus far, genetic diversity for high resistance to/tolerance of ShB has not been reported in either cultivated rice or its wild relatives; thus, cloning of genes for ShB resistance is straggling (Bonman 1992). For bacterial leaf streak (BLS), no major resistance genes (*R*-genes) have been identified and only a few QTLs have been mapped. Out of these, *qXO-2-1*, *qXO-4-1*, and *qXO-11-2* were showing resistance to more than nine *Xoc* and *Xoo* strains (Bossa-Castro et al. 2018). In the case of tungro disease, a resistance QTL was found in Indian landrace ARC 11554 and was localized on chromosome 4 (Wang et al. 2016). False smut resistance in several rice cultivars has been identified as a quantitative trait controlled by multiple genes (Andargie et al. 2018; Han et al. 2020). But, to date, no rice variety has been identified to show complete resistance to false smut, whereas many cultivars exhibit

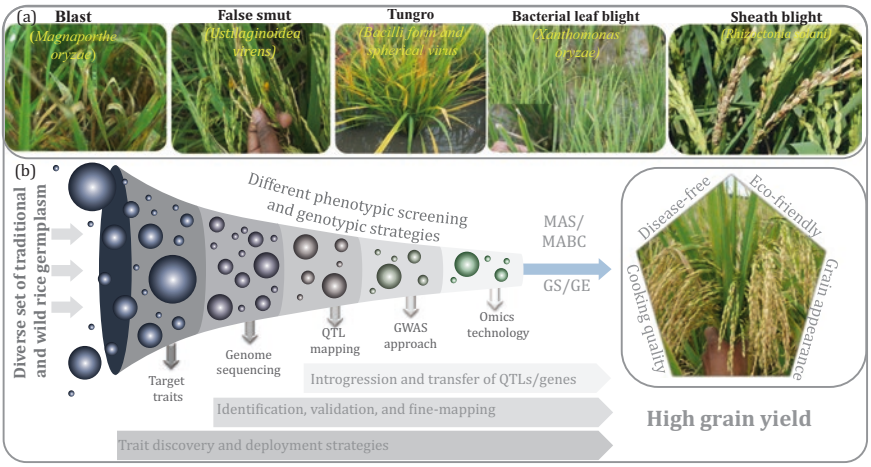

Fig. 1 (**a**) An illustration of the different biotic diseases in rice and (**b**) a funnel diagram representing the sources of valuable traits (tolerance of biotic diseases, yield components, and superior grain quality) that exist in traditional and wild rice germplasm. Using various phenotypic screening techniques and genome sequencing technologies can enable us to understand the molecular genetics and physiological mechanisms of stress tolerance. The identified genomic regions of QTLs and genes associated with the key traits play a vital role in understanding the interactions and further improving disease tolerance and superior grain quality traits with the help of marker-assisted selection and genomic selection approaches for crop improvement

considerable differences in quantitative field resistance to the pathogen (Huang et al. 2019). According to previous studies, the genetics of sheath rot disease resistance was dissected by studying the segregating pattern in an F_2 population (Rajashekara et al. 2014; Mvuyekure et al. 2017) and recombinant inbred lines (Graichen et al. 2010; Mahadevaiah et al. 2017). Some of these *R*-genes or loci have been extensively used in MAS breeding programs, and some of them have been fine-mapped and are undergoing cloning efforts. Detailed information on resistance genes/QTLs for economically important rice diseases (i.e., bacterial blight, blast, and sheath blight) appears in Tables 2, 3, and 4, respectively.

4 Breeding for Disease Resistance

Rice breeders have come up with many disease-resistant cultivars adapted to different rice-growing regions worldwide by applying conventional breeding approaches. Because of the dominance and epistasis effects of genes conferring resistance to a few diseases, gene pyramiding through conventional breeding methods becomes a challenge. Also, genes having similar responses to two or more races of a pathogen are difficult to recognize and transfer by conventional approaches (Joseph et al. 2004; Sundaram et al. 2009; Rajpurohit et al. 2011). The exercise of breeding for

Table 2 List of bacterial blight-resistance genes/QTLs

S. No.	Genes/ QTLs	Chr.	Position (bp)	Donor parent	Inheritance	Origin	Resistance to *Xoo* race	Linked marker	Marker type	References
1	*Xa-1*	4	31,638,099–31,644,795	*Kogyoku*, *IRBB1*	Dominant, cloned, and characterized	Japan	Japanese race-I	Npb235	RFLP	Yoshimura et al. (1998)
2	*Xa-2*	4		*IRBB2*	Dominant	Vietnam	Japanese race-II	HZR950-5	SSR	Kurata and Yamazaki (2006)
3	*Xa-3/ Xa-26*	11	28,399,360–28,402,773	*WaseAikoku 3*, *Minghui 63*, *IRBB3*	Dominant, cloned, and characterized	Japan	Chinese, Philippine, and Japanese races	C481S	RFLP	Xiang et al. (2006)
4	*Xa-4*	11	–	*TKM6*, *IRBB4*	Dominant	India	Philippine race-I	Npb181 and RM224	RFLP and SSR	Wang et al. (2001)
5	*xa-5*	5	437,010–443,270	*IRBB5*	Recessive, cloned, and characterized	Bangladesh	Philippine races I, II, and III	RG556 and RM122	CAPS and SSR	Petpisit et al. (1977)
6	*Xa-6/ xa-3*	11	–	*Zenith*	Dominant	U.S.	Philippine race-I	Y68SSRA	RFLP	Sidhu et al. (1978)
7	*Xa-7*	6	–	*DZ78*	Dominant	Bangladesh	Philippine races	G1091, RM205S2	RFLP, SSR	Chen et al. (2008)
8	*xa-8*	7	–	*PI231128*	Recessive	U.S.	Philippine races	RM500, RM533	SSR	Vikal et al. (2014)
9	*Xa-9*	11	–	*Khao Lay Nhay* and *Sateng*	Dominant	Laos	Philippine races	C4S1S	RFLP	Ogawa (1988)
10	*Xa-10*	11	22,203,734–22,204,676	*Cas 209*	Dominant, cloned, and characterized	Senegal	Philippine and Japanese races	M491/M419	RFLP, GAPS	Kurata and Yamazaki (2006)

(continued)

Table 2 (continued)

S. No.	Genes/ QTLs	Chr.	Position (bp)	Donor parent	Inheritance	Origin	Resistance to *Xoo* race	Linked marker	Marker type	References
11	*Xa-11*	3	–	*IR8*	Dominant	Philippines	Japanese races IB, II, IIIA, and V	–	–	Kurata and Yamazaki (2006)
12	*Xa-12*	4	–	*Kogyoku*, *Java14*	Dominant	Japan	Indonesian race-V	–	–	Ogawa et al. (1987)
13	*xa-13*	8	–	*BJ1*, *IRBB13*	Recessive, cloned, and characterized	India	Philippine race-6	*RG136*, *xal3p*	STS, SSR	Kurata and Yamazaki (2006)
14	*Xa-14*	4	–	*TN1*	Dominant	Taiwan	Philippine race 5	VAZ190B/ RG163	RFLP	Kurata and Yamazaki (2006)
15	*xa-15*		–	*M41 mutant*	Recessive	ND	Japanese races	–	–	Ogawa (2008)
16	*Xa-16*		–	*Tetep*	Dominant	Vietnam	Japanese races	–	–	Kurata and Yamazaki (2006)
17	*Xa-17*		–	*Asominori*	Dominant	South Korea	Japanese races	–	–	Kurata and Yamazaki (2006)
18	*Xa-18*		–	*IR24*, *Miyang23*, *Toyonishiki*	Dominant	Philippines, Japan	Burmese races	–	–	Kurata and Yamazaki (2006)
19	*Xa-19*	3	–	*XM5* (mutant of *IR24*)	Recessive	–	Japanese races	–	–	Kurata and Yamazaki (2006)

S. No.	Genes/ QTLs	Chr.	Position (bp)	Donor parent	Inheritance	Origin	Resistance to *Xoo* race	Linked marker	Marker type	References
20	*Xa-20*		–	*XM6* (mutant of *IR24*)	Recessive	–	Japanese races	–	–	Kurata and Yamazaki (2006)
21	*Xa-21*	11	20,802,924–20,806,518	*O. longistaminata*, *IRBB21*	Dominant, cloned, and characterized	Africa, Mali	Philippine and Japanese races	pTA248	STS	Song et al. (1995)
22	*Xa-22(t)*	11		*Zhach anglong*	Dominant	China	Chinese races	L363B/P143	RFLP	Kurata and Yamazaki (2006)
23	*Xa-23*	11	22,203,734–22,204,676	*O. ruipogon (CBB23)*	Dominant, cloned, and characterized	China/ Cambodia	Indonesian races	–	–	Zhang et al. (2001)
24	*xa-24*	2		*DV86*	Recessive	Bangladesh	Philippine and Chinese races	–	–	Khush and Angeles (1999)
25	*xa-25/ Xa25(t)*	12	17,302,073–17,305,326	*Minghui 63*, *HX-3* (somaclonal mutant of *Minghui 63*)	Recessive, cloned, and characterized	China	Chinese and Philippine races	–	–	Liu et al. (2011)
26	*xa-26(t)*	11	–	*Nep Bha Bong*	Recessive	China	Philippine races	C4S1S/ Y6855RA	RFLP	Lee et al. (2003)
27	*Xa-27(t)*	6	–	*O. minuta IRGC 101141*, *IRBB27*	Dominant, cloned, and characterized	Philippines	Chinese strains and Philippine races 2–6	M10S1, M1095	RFLP	Gu et al. (2005)
28	*xa-28(t)*	11	–	*Lota sail*	Recessive	Bangladesh	Philippine race 2	–	–	Lee et al. (2003)
29	*Xa-29(t)*	1	–	*O. officinalis (B5)*	Dominant	–	Chinese races	–	–	Tan et al. (2004)

(continued)

Table 2 (continued)

S. No.	Genes/ QTLs	Chr.	Position (bp)	Donor parent	Inheritance	Origin	Resistance to *Xoo* race	Linked marker	Marker type	References
30	*Xa-30(t)*	11	–	*O. ruipogon (Y238)*	Dominant	India	Indonesian races	–	–	Cheema et al. (2008)
31	*xa-31(t)*	4	–	*Zhach anglong*	Recessive	China	Chinese races	–	–	Wang et al. (2009)
32	*xa-32(t)*	11	–	*O. australiensis* (introgression line C4064)	Recessive	–	Philippine races	RM27256, 27274	SSRs	Zheng et al. (2009)
33	*Xa-33*	7	–	*Ba7 O. nivara*	Dominant	Thailand	Thai races	RMWR7.1 and 7.6	SSRs	Hari et al. (2013)
34	*Xa-33(t)*	6	–	*Ba7O. nivara*	Dominant	Thailand	Thai race	RM20590	SSRs	Korinsak et al. (2009)
35	*xa-34(t)*	1	–	*Pin Kaset O. brachyantha*	Recessive	Sri Lanka	Thai race	RM493, RM446, RM10927, RM10591	SSRs	Chen et al. (2011b)
36	*Xa-35(t)*	11	–	*Oryza minuta* (Acc. No. 101133)	Dominant	Philippines	Philippine races	–	–	Guo et al. (2010)
37	*Xa-36(t)*	11	–	*C4059*	Dominant	China	Philippine races	–	–	Miao et al. (2010)
38	*Xa-38*	4	–	*O. nivara IRGC81825*	Dominant	–	Indian Punjab races	RM17499, RM459, RM317	STS/ SSR	Bhasin et al. (2012)
39	*Xa39*	11	–	*FF329*	Dominant	–	Chinese and Philippine races	RM21, RM206	STS/ SSR	Zhang et al. (2015)
40	*Xa40(t)*	11	–	*IR65482-7-216-1-2*	Dominant	–	Korean BB races	RM27320, ID55, WA18-5	STS/ SSR	Kim et al. (2015)

S. No.	Genes/ QTLs	Chr.	Position (bp)	Donor parent	Inheritance	Origin	Resistance to *Xoo* race	Linked marker	Marker type	References
41	*xa41(t)*	–	–	Rice germplasm	Dominant	–	Various *Xoo* strains	–	–	Hutin et al. (2015)
42	*xa42*	3	–	*XM14*, a mutant of *IR24*	Dominant	–	Japanese *Xoo* races	RM15189	SSR	Busungu et al. (2016)
43	*Xa-43 (t)*	11	11,92,907–11,943,779	*IR36(P8)*	Dominant	–	Korean BB races	–	–	Kim and Reinke (2019)
44	*Xa-44 (t)*	11	11,964,077–11,985,463	*R73571-3B-11-3-K3(P6)*	Dominant	–	Korean BB races	–	–	Kim (2018)
45	*Xa-45(t)*	8	26,737,175–26,818,765	*O. glaberrima IRGC 102600B*	Recessive	–	Indian Punjab races	xa13 *prom*	STS	(Neelam et al. 2020)
46	*Xa46(t)*	11	–	Mutant line *H 120*	Dominant	China	Chinese *Xoo* races	RM26981–RM26984	SSR	Chen et al. (2020)

Source: Revised and updated from Kou and Wang (2013)

Table 3 List of blast resistance genes/QTLs

S. No.	Gene/ QTL	Chr.	Position (bp)	Position (cM)	Donor rice variety	Method of identification	References
1	*Pit*	1	2,270,216–3,043,185	9.08–12.17	Tjahaja	Cloned	Hayashi and Yoshida (2009)
2	*Pi27(t)*	1	6,230,045–6,976,491	24.29–27.90	IR64 (I)	Mapped within 21.6 cM	Sallaud et al. (2003)
3	*Pi24(t)*	1	5,242,654–5,556,378	20.97–22.22	Azuenca (J)	QTL mapping	Zhuang et al. (2002)
4	*Pitp(t)*	1	25,135,400–28,667,306	100.54–117.49	Tetep	Cosegregation marker was identified	Barman et al. (2004)
5	*Pi35(t)*	1	33,000,000–34,150,000	132.0–136.6	Hokkai 188 (J)	Cloned	Xu et al. (2014)
6	*Pi37*	1	33,110,281–33,489,931	132.44–133.95	St. No. 1 (J)	Cloned	Lin et al. (2007)
7	*Pi64*	1	–	–	Yangmaogu (J)	Cloned	Ma et al. (2015a)
8	*Pid1(t)*	2	21,875,000–22,475,000	87.5–89.9	Digu	Mapped within 11.8 cM	Chen et al. (2004)
9	*Pig(t)*	2	34,346,727–35,135,783	137.38–140.54	Guangchang zhan (I)	Mapped within 11.8 cM	Zhou et al. (2004)
10	*Pitq5*	2	37,625,000–39,475,000	150.5–157.9	Teqing	QTL mapping	Tabien et al. (2002)
11	*Piy1(t)*	2	38,300,000–38,525,000	153.2–154.1	Yanxian No. 1	Mapped within 1.6 cM	Lei et al. (2005)
12	*Piy2(t)*	2	38,300,000–38,525,001	153.2–154.1	Yanxian No. 1	Mapped within 3.0 cM	Lei et al. (2005)
13	*Pib*	2	38,300,000–38,525,000	153.2–154.1	Tohoku IL9	Cloned	Wang et al. (1999)
14	*Pi25(t)*	2	34,360,810–37,725,160	137.44–150.90	IR64 (I)	QTL mapping	Wu and Tanksley (1993)
15	*Pi14(t)*	2	1–6,725,831	1.00–26.90	Maowangu	Linkage analysis using isozyme markers	Pan et al. (1996)
16	*Pi16(t)*	2	1–6,725,831	1.00–26.91	Aus373 (I)	Linkage analysis using isozyme markers	Pan and Tanisaka (1997)

(continued)

Table 3 (continued)

S. No.	Gene/ QTL	Chr.	Position (bp)	Position (cM)	Donor rice variety	Method of identification	References
17	*Pi68(t)*	3	14,738–14,761	9.30–9.70	*INGR15002*	QTL mapping	Devi et al. (2020)
18	*Pi63/ Pikahei-1(t)*	4	–	–	Kahei	Cloned	Xu et al. (2014)
19	*pi21*	4	5,242,654–5,556,378	20.97–22.22	Owarihatamochi	Cloned	Fukuoka et al. (2009)
20	*Pikur1*	4	24,611,955–33,558,479	98.44–134.23	Kuroka (J)	Linkage analysis using phenotypic marker	Goto (1988)
21	*Pi39(t)*	4	26,850,000–27,050,000	107.4–108.2	Chubu 111 (J)	Mapped within 0.3 cM	Liu et al. (2007)
22	*Pi(t)*	4	2,270,216–3,043,185	9.08–12.17	Tjahaja	Linkage analysis using phenotypic marker	Causse et al. (1994)
23	*Pi26(t)*	5	8,751,256–11,676,579	35.00–46.70	Gumei 2 (I)	QTL mapping	Wu and Tanksley (1993)
24	*Pi23(t)*	5	10,755,867–19,175,845	43.02–76.70	Sweon 365	QTL mapping	Ahn et al. (1997)
25	*Pi10*	5	14,521,809–18,854,305	58.08–75.41	Tongil	Mapped within 6.7 cM	Naqvi et al. (1995)
26	*Pi2*	6	–	–	C101A51	Cloned	Zhou et al. (2006)
27	*Pi22(t)*	6	4,897,048–6,023,472	19.50–24.09	Suweon365 (J)	QTL mapping	Ahn et al. (1997)
28	*Pi26(t)*	6	8,751,256–11,676,579	35.00–46.70	Azucena (J)	QTL mapping	Wu et al. (2005)
29	*Pi27(t)*	6	5,556,378–744,329	22.22–2.97	IR64 (I)	Mapped within 21.6 cM	Sallaud et al. (2003)
30	*Pi40(t)*	6	16,274,830–17,531,111	65.09–70.12	*O. australiensis* (W)	Mapped within 1.8 cM	Jeung et al. (2007)
31	*Piz*	6	10,155,975–10,517,612	40.60–42.07	Zenith (J)	Mapped within 0.43 cM	Ahn et al. (1996)
32	*Piz-t*	6	14,675,000	58.70	Toride 1	Cloned	Hayashi et al. (2006)
33	*Pi9*	6	10,386,510–10,389,466	41.50–41.55	*O. minuta* (W)	Cloned	Qu et al. (2006)

(continued)

Table 3 (continued)

S. No.	Gene/ QTL	Chr.	Position (bp)	Position (cM)	Donor rice variety	Method of identification	References
34	*Pi25*	6	18,080,056–19,257,588	72.32–77.03	Gumei 2	Cloned	Chen et al. (2011a)
35	*Pid2*	6	17,159,337–17,163,868	68.63–68.65	Digu	Cloned	Chen et al. (2006b)
36	*Pigm(t)*	6	10,367,751–10,421,545	41.47–41.68	Gumei 4	Mapped within 70 kb	Deng et al. (2017)
37	*Pi50*	6	–	–	Er-Ba-zhan (EBZ)	Cloned	Su et al. (2015)
38	*Pid3-I1*	6	–	–	MC276	Cloned	Inukai et al. (2019)
39	*Pi17(t)*	7	22,250,443–24,995,083	89.00–99.90	DJ 123	Mapped within 1.8 cM	Pan et al. (1996)
40	*Pi36*	8	2,870,061–2,884,353	11.48–11.53	Q61 (I)	Cloned	Liu et al. (2005)
41	*Pi33*	8	5,915,858–6,152,906	23.66–24.61	IR64 (I)	Mapped within 1.6 cM	Berruyer et al. (2003)
42	*Pizh*	8	4,372,113–21,012,219	17.48–84.04	Zhai-Ya-Quing8 (I)	QTL mapping	Sallaud et al. (2003)
43	*Pi29(t)*	8	9,664,057–16,241,105	38.65–64.96	IR64 (I)	Mapped within 0.7 cM	Sallaud et al. (2003)
44	*Pii2(t)*	9	1,022,662–7,222,779	4.09–28.89	Azucena	Linkage analysis using phenotypic markers	Kinoshita and Kiyosawa (1997)
45	*Pi5*	9	7,825,000–8,250,000	31.30–33.00	RIL125, RIL249, RIL260 (Moroberekan)	Mapped within 170 kb	Lee et al. (2009)
46	*Pi3(t)*	9	7,825,000–8,250,001	31.3–33.1	Kan-Tao	Linkage analysis using RFLP markers	Causse et al. (1994)
47	*Pi15*	9	9,641,358–9,685,993	38.56–38.74	GA25 (J)	Mapped within 0.7 cM	Pan et al. (1996)
48	*Pii*	9	–	–	Hitomebore	Cloned	Takagi et al. (2013a)
49	*Pi28(t)*	10	19,565,132–22,667,948	78.26–90.67	IR64 (I)	QTL mapping	Sallaud et al. (2003)
50	*Pia*	11	–	–	Aichi Asahi (J)	Cloned	Okuyama et al. (2011)

(continued)

Table 3 (continued)

S. No.	Gene/ QTL	Chr.	Position (bp)	Position (cM)	Donor rice variety	Method of identification	References
51	*PiCO39(t)*	11	6,304,007–6,888,870	25.21–27.55	CO39 (I)	Cloned	Cesari et al. (2013)
52	*Pilm2*	11	13,635,033–28,377,565	54.54–113.50	Lemont	QTL mapping	Tabien et al. (2002)
53	*Pi30(t)*	11	441,392–6,578,785	1.76–26.31	IR64 (I)	QTL mapping	Sallaud et al. (2003)
54	*Pi7(t)*	11	17,850,000–21,075,000	71.40–84.30	RIL29 (Moroberekan)	QTL mapping	Wang et al. (1994)
55	*Pi34*	11	19,423,000–19,490,000	77.69–77.96	Chubu32 (J)	QTL mapping	Zenbayashi et al. (2002)
56	*Pi38*	11	19,137,900–21,979,485	76.55–87.91	Tadukan (I)	Mapped within 20 cM	Gowda et al. (2006)
57	*PBR*	11	20,125,000–30,075,000	80.5–120.3	St. No. 1	Mapped within 22.9 cM	Fujii et al. (1995)
58	*Pb1*	11	–	–	Modan	Cloned	Hayashi et al. (2010)
59	*Pi44(t)*	11	22,850,000–29,475,000	91.40–117.90	RIL29 (Moroberekan)	–	Chen et al. (1999)
60	*Pik-h/ Pi54*	11	24,761,902–24,762,922	99.0–99.05	Tetep	Cloned	Sharma et al. (2005b)
61	*Pi1*	11	26,498,854–28,374,448	105.99–113.49	LAC23 (J)	Mapped within 11.4 cM	Hua et al. (2012)
62	*Pik-m*	11	27,314,916–27,532,928	109.25–110.13	Tsuyuake (J)	Cloned	Ashikawa et al. (2008)
63	*Pi18(t)*	11	26,796,917–28,376,959	107.18–113.50	Suweon365 (J)	Mapped using RFLP markers	Ahn et al. (1996)
64	*Pik*	11	27,314,916–27,532,928	109.25–110.13	Kusabue (I)	Cloned	Zhai et al. (2011)
65	*Pik-p*	11			K60	Cloned	Yuan et al. (2011)
66	*Pik-s*	11	27,314,916–27,532,929	109.25–110.15	Shin 2 (J)	Mapped within 2.7 cM	Fjellstrom et al. (2004)

(continued)

Table 3 (continued)

S. No.	Gene/ QTL	Chr.	Position (bp)	Position (cM)	Donor rice variety	Method of identification	References
67	*Pik-g*	11	27,314,916–27,532,930	109.25–110.16	GA20 (J)	Linkage analysis to other resistance genes	Pan et al. (1996)
68	*Pise1*	11	5,740,642–16,730,739	22.96–66.92	Sensho	Linkage analysis using phenotypic markers	Goto (1970)
69	*Pif*	11	24,695,583–28,462,103	98.78–113.84	Chugoku 31-1 (St. No. 1)	QTL mapping	Shinoda et al. (1971)
70	*Mpiz*	11	4,073,024–16,730,739	16.29–66.92	Zenith (J)	Linkage analysis using phenotypic markers	Goto (1970)
71	*Pikur2*	11	2,840,211–18,372,685	11.36–73.49	Kuroka (J)	Linkage analysis using phenotypic markers	Goto (1988)
72	*Piisi*	11	2,840,211–19,029,573	11.36–76.11	Imochi Shirazu (J)	Linkage analysis using phenotypic markers	Goto (1970)
73	*Pike*	11			Xiangzao 143	Cloned	Chen et al. (2015)
74	*Pi24(t)*	12	5,242,654–5,556,378	20.97–22.22	Azuenca (J)	QTL mapping	Zhuang et al. (2002)
75	*Pi62(t)*	12	2,426,648–18,050,026	9.70–77.00	Yashiro-mochi (J), Tsuyuake	Mapped within 1.9 cM	Wu et al. (2008)
76	*Pitq6*	12	5,758,663–7,731,471	23.00–30.92	Tequing (I)	QTL mapping	Tabien et al. (2002)
77	*Pi6(t)*	12	1–6,725,831	1–1.68	Apura (I)	–	McCouch et al. (1994)
78	*Pi12*	12	6,988,220–15,120,464	27.95–60.48	Moroberekan (J)	Linkage analysis using RFLP markers	Inukai et al. (1996)
79	*Pi21(t)*	12	5,242,654–5,556,378	20.94–22.22	Owarihata mochi (J)	–	Ahn et al. (1997)
80	*Pi31(t)*	12	7,731,471–11,915,469	30.92–47.66	IR64 (I)	QTL mapping	Sallaud et al. (2003)

(continued)

Table 3 (continued)

S. No.	Gene/ QTL	Chr.	Position (bp)	Position (cM)	Donor rice variety	Method of identification	References
81	*Pi32(t)*	12	13,103,039–18,867,450	52.41–75.46	IR64 (I)	QTL mapping	Sallaud et al. (2003)
82	*Pi157*	12	12,375,000–15,550,000	49.5–62.2	Moroberekan	Mapped within 9.5 cM	Causse et al. (1994)
83	*Pita*	12	10,603,772–10,609,330	42.41–42.43	Tadukan (I)	Cloned	Hayashi et al. (2006)
84	*Pita-2*	12	10,078,620–13,211,331	40.31–52.84	Shimokita (J)	Mapped within 4.0 cM	Nakamura et al. (1997)
85	*Pi19(t)*	12	8,826,555–13,417,088	35.30–53.67	Aichi Asahi (J)	Linkage analysis to other resistance genes	Iwata (1996)
86	*Pi39(t)*	12	–	–	Chubu 111 (J),	Mapped within 37 kb	Liu et al. (2007)
87	*Pi20(t)*	12	12,875,000–12,950,000	51.50–51.80	IR24 (I)	Mapped within 0.6 cM	Liu et al. (2008)
88	*PiGD-3(t)*	12	13,950,000	55.80	Sanhuangzhan 2	QTL mapping	Liu et al. (2005)
89	*Ptr*	12			Katy	Cloned	Zhao et al. (2018)

Source: Revised and updated from Tanweer et al. (2015)

disease resistance could never obtain a break because of the emergence of new pathotypes, which could overcome the resistance. Advances in rice genomics provided tools such as molecular markers for plant breeders to effectively develop cultivars with resistance against various diseases, which is an environment-friendly alternative vis-à-vis the use of agrochemicals (Miah et al. 2013). Molecular markers can be used to map and introgress one or more desired genes for biotic and abiotic stress resistance from diverse gene pools (Suh et al. 2009). Marker-assisted selection for pyramiding desired genes without altering other quality characteristics of a rice cultivar is crucial in rice improvement (Sundaram et al. 2008; Suh et al. 2009; Shanti et al. 2010). As an added advantage, the availability of gene-linked molecular markers for the resistance genes eases the identification of plants harboring two or more *R*-genes at any growth stage without a bioassay (Sundaram et al. 2008; Shanti et al. 2010; Bainsla and Meena 2016).

Three bacterial blight-resistance genes (*xa5*, *xa13*, and *Xa21*) were pyramided into susceptible cultivar PR106 using MAS. The introgression lines were tested against 17 *Xoo* isolates under both glasshouse and field conditions. The trials suggested that the combination of genes provided broad-spectrum resistance against

Table 4 List of sheath blight-resistance genes/QTLs

S. No.	QTLs	Chr.	Linked markers	Mapping population	Type of marker	LOD	Associated character	Remarks	Reference
1	*qshb1.1*	1	RM151–RM12253	210 F_2 (ARC10531^I × BPT-5204^I)	SSR (70)	10.7	Percentage relative lesion height	32 candidate genes identified in the region *qShB9.2*	Yadav et al. (2015)
2	*qshb7.1*	7	RM81–RM615			8.8			
3	*qshb7.2*	7	RM10–RM2169			6.7			
4	*qshb8.1*	8	RM21792–RM310			4.2			
5	*qSBL7 (E2)*	7	D760–RM248	190 F_2 (Yangdao 4^I × Lemont^J)	SSR (52) and InDel (128)	3.12	DR (disease rating)	Sheath blight resistance is correlated with plant height	Wen et al. (2015)
6	*qSBPL-7 (E2)*	7	D760–RM248			5.07	LH (lesion height)		
7	*qSBD-12-2 (E1)*	12	RM1246–D1252			3.74	PL (percentage of lesion height), DR, LH		
8	*qHZaLH3*	3	RM143–RM514	116 DHs (TN1^I × CJ06^J)	SSR (214)			No correlation was found between LH and PH	Zeng et al. (2015)
10	*qHZaLH6*	6	WX–RM587						
11	*qHZaDR8*	8	RM1376–RM4085						
13	*qHZaDR9*	9	RM444–AGPSMA						
14	*qHZbDR5*	5	RM3321–RM3616						

S. No.	QTLs	Chr.	Linked markers	Mapping population	Type of marker	LOD	Associated character	Remarks	Reference
15	*qSB-9^{TQ}*	9	CY-85 and Y86	235CSSLs (BC_6F_3) (Teqing^{I} (TQ) × Lemont^{J})	InDel and CAPS (22)	–	ShB resistance	Fine-mapped (146 kb covering region), 12 genes were annotated	Zuo et al. (2014)
16	*qDR-4*	4	RM1155–RM 5757	155 RILs $F_{8:11}$ (RSB02 × HH1B)	SSR (163)	2.71	DR LL (lesion length)	Epistasis and QTL × environment (QE) interaction were studied	Liu et al. (2014)
17	*qRLL-4*	4	RM1155–RM5757			5.84	LH		
18	*qRLH-4*	4	RM1155–RM5757			4.77	Relative LL Relative LH		
19	*qSB-11^{LE}*	11	Z22-27C and Z23-33C	112CSSLs (Teqing^{I} × Lemont^{J} (LE)	STS and CAPS	–	ShB resistance	Fine-mapped (79 kb covering region), 11 genes were annotated	Zuo et al. (2013)
20	*qRTL3*	3	RM570	BILs (Jarjan^{I} × Koshihikari^{J})	SSR (151)	3.5	RTL (rate of tillers with lesions)		Taguchi-Shiobara et al. (2013)
21	*qRTL5*	5	RM5784			4.3			
22	*qRTL6*	6	RM1161			7.7			
23	*qRTL9*	9	RM6251			3.1			
24	*qRTL3*	3	RM16200			5.9			
25	*qRTL6*	6	RM2615			2.9			
26	*qRTL12*	12	RM7025			3.2			
27	*qRTL5*	5	RM3286			3.1			
28	*qRTL6*	6	RM6395			5.8			

(continued)

Table 4 (continued)

S. No.	QTLs	Chr.	Linked markers	Mapping population	Type of marker	LOD	Associated character	Remarks	Reference
29	*qRTL9*	9	RM3533			3.8			
30	*qShB2-1-*	2	RM279–RM71	216 RILs (Jasmine 85^{I} × $Lemont^{J}$)	SSR (199)	3.7	ShB resistance	The major QTL *qShB9-2* was reconfirmed based on the field data	Liu et al. (2013)
31	*ARqShB7-AR*	7	RM5711–RM2			4.0			
32	*qShB7 LA*	7	RM5711–RM2			6.0			
33	*qShB11-1-*	11	RM7203–RM536			3.2			
34	*TXqShB11-2-TX*	11	RM536–RM229			3.3			
35	*qShB6 (wild 1-field 2009)*	6	RM3431–RM3183	252 Wild-1 and 253 Wild-2 BC_2F_2	SSR (131)	7.8	ShB resistance	Colocalization of *qShB6* with *qDH1* and *qShB1* with *qPH1* revealed the influence of heading date and plant height on resistance	Eizenga et al. (2013)
36	*qShB6 (wild 2-field 2009)*	6	RM253–RM3431	(*Oryzanivara* × $Bengal^{J}$ (*O. sativa*))		21.2			
37	*qShB6 (wild2-field 2008)*	6	RM253–RM3431			11.1			
38	*qShB1 (wild 2-field 2008)*	1	RM431–RM1361			4.7			
39	*qShB6-mc (wild-1 microchamber)*	6	RM3183–RM541			3.3			

S. No.	QTLs	Chr.	Linked markers	Mapping population	Type of marker	LOD	Associated character	Remarks	Reference
40	*qsbr_2.1*	2	RM8254–RM8252	197 DHs (MCR10277[I] × Cocodrie[J])	SSR (111)	3.4–29.7	SBF (sheath blight disease		Nelson et al. (2012)
41	*qsbr_2.2*	2	RM3857–RM5404			2.9–37.8	severity in field), SBI (disease severity in microchamber)		
42	*qsbr_12.1*	12	RM3747–RM27608			49.1	SBM (disease severity in mist chamber)		
43	*qSBR1*	1	RM11229	217 core collection of USDA	SSR (154) and InDel (1)	9.5%	Sheath blight resistance		Jia et al. (2012)
44	*qSBR11*	11	RM7203			1.9%			
45	*qSBR1-1*, *qSBR2-1*	1, 2	RM5389–RM3825, RM5340–RM521	121 RIL (RSB03 × HH1B)	SSR (123)	3.2, 3.1	DR LL LH		Fu et al. (2011)
46	*qSBR2-2*	2	RM110–osr14			5.2	Relative LL		
47	*qSBR2-3*	2	RM7245–RM5303			3.3	Relative LH		
48	*qSBR4*	4	RM3288–RM7187			3.8			

(continued)

Table 4 (continued)

S. No.	QTLs	Chr.	Linked markers	Mapping population	Type of marker	LOD	Associated character	Remarks	Reference
49	*qSBR5-2*	5	RM7446–RM3620			4.8			
50	*qSBR7*	7	RM1132–RM473			3.3			
51	*qSBR8*	8	RM8264–RM1109			4.2			
52	*qSBR9*	9	RM23869–RM3769			5.0			
53	*qShB1 (2007/2008)*	1	RM431–RM12017	251 DHs (Baiyeqiu[I] × Maybelle[J])	SSR (227)	5.18–8.03	Sheath blight resistance		Xu et al. (2011)
54	*qShB2 (2008)*	2	RM174–RM145			3.96			
55	*qShB3 (2007)*	3	RM135–RM186			3.42			
56	*qShB5 (2007)*	5	RM18872–RM421			4.35			
57	*qShB1-2 (2020)*	1		184 RILs (SH × DGWG[I]; BHA × DGWG[I])		5.71	Blast resistance		Goad et al. (2020)
58	*qShB4 (2020)*	4				4.50			
59	*qSBR3.2*	3	D311 or RM282	219 RILs (Lemont[J] × Yangdao4[I])		3.3	Sheath blight resistance		Yuan et al. (2019)
60	*qSBR7.1*	7	D709 or D715			3.7			
61	*qSBR8.1*	8	D804		SSR	1.8			

S. No.	QTLs	Chr.	Linked markers	Mapping population	Type of marker	LOD	Associated character	Remarks	Reference
62	*qSBR9.2*	9	D947 or D948			3.0			
63	*qSBR9.3*	9	D949			2.4			
64	*qSBR12.1*	12	D1211			2.3			
65	*qSBR12.2*	12	D1239 or D1246			3.6			

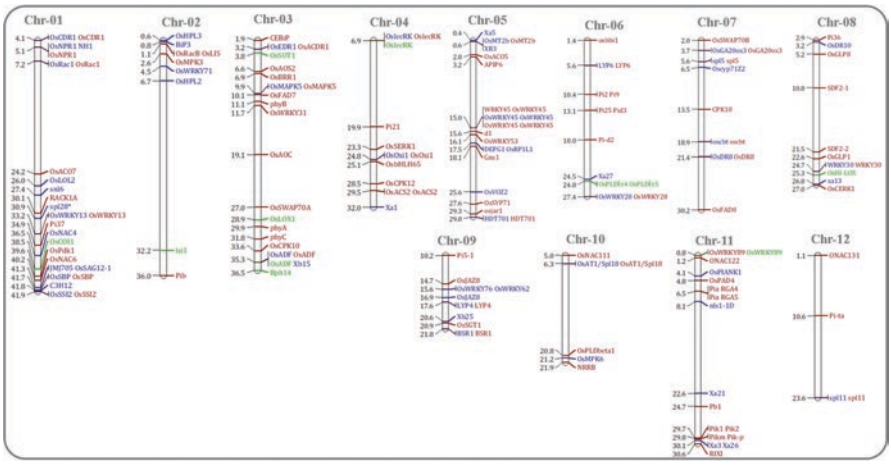

Fig. 2 Physical position of major biotic disease-resistance genes in rice. The chromosome left side indicates the location of genes and the right side shows the names of genes collected from the Q-TARO and Oryza base databases. The color indicates the genes related to various diseases such as bacterial leaf blight (red color) and blast (blue color), and green color indicates insect resistance in rice

the pathogen races predominant in the region (Singh et al. 2001). Recent advances in DNA sequencing have made fine-mapping and characterization of the mapped genes easier, thus contributing significantly to the use of MAS for the development of resistant cultivars. The complete list of cloned genes was collected from Q-TARO (http://qtaro.abr.affrc.go.jp/) and OryGenesDB (https://orygenesdb.cirad.fr/data.html) (Fig. 2). These genes were mainly associated with bacterial blight and blast resistance in rice. Interestingly, the regions on chromosomes 1, 4, and 5 were associated with multiple resistance genes, and these genes were colocalized in the same regions. These genomic regions play a major role in the resistance/tolerance mechanisms for diseases. To date, there are 46 BB *R*-genes mapped from different sources, out of which 29 are dominant, 12 are recessive, nine cloned, and nine fine-mapped (Chen et al. 2020). More than 100 *R*-genes (*Pi*) have been reported, and around 500 QTLs were associated with blast resistance. However, only 25 *Pi* genes were cloned and characterized (Sharma et al. 2012; Ashkani et al. 2015). Several *R* QTLs were reported against bacterial leaf streak, but their study was limited to inheritance analysis (He et al. 2012).

4.1 MAS/MABB Foreground/Background Selection

To address the limitations of conventional breeding, molecular breeding through MAS is among the most precise tools used to introgress multiple resistance genes into an elite varietal background at one time. Plant breeders were already successful

in using this tool in developing resistant rice cultivars by deploying broad-spectrum multiple-*R*-genes with the help of MAS (Huang et al. 1997; Sanchez et al. 2000; Sundaram et al. 2008; Hari et al. 2013; Hajira et al. 2016; Balachiranjeevi et al. 2018; Swathi et al. 2019; Jamaloddin et al. 2020). In marker-assisted backcross breeding (MABB), a combination of foreground selection and background selection followed by continuous backcrossing can recover up to 99% of the recurrent parent genome (RPG) (Tanksley et al. 1989). In foreground selection, gene-linked markers, or functional markers (SSRs, InDels, and SNPs), are applied to detect the associated *R*-genes in the target population at any stage of plant growth. In contrast, background selection applied by using polymorphic information (SSRs, SNPs) between the donor and recurrent parents can estimate RPG recovery in each backcross generation at any plant growth stage (Singh et al. 2001). Recently, the Green Super Rice (GSR) breeding strategy proved that one backcross followed by selfing could recover more than 90% of the recurrent parent genome (Balachiranjeevi et al. 2019).

4.2 Pyramiding Disease-Resistance Genes

Pyramiding of various biotic disease-resistance genes into a rice cultivar makes it a good candidate for breeders to introgress the resistance into locally adapted varieties that produce higher yield but are susceptible to diseases. The process of gene pyramiding through conventional breeding alone becomes difficult because the linkage between some undesirable traits is difficult to break even after repeated backcrossing (Tanksley et al. 1989). Pyramiding of two or more resistance genes renders the phenotypic assessment of rice genotypes ineffective as distinguishing the effect of each individual gene precisely becomes difficult since each gene imparts resistance to more than one race of the pathogen. Moreover, when a dominant and a recessive allele are present, the effect of the recessive gene is concealed. The availability of tightly linked markers for each of the resistance genes thus eases the recognition of plants with multiple genes. Initially, in rice, Huang et al. (1997) successfully introgressed four major BB resistance genes (*Xa4*, *Xa5*, *Xa13*, and *Xa21*) and developed breeding lines with combinations of two, three, and four genes. In an extension of this work, several research institutions in India and other countries have studied the effectiveness of the pyramided genes against BB disease, to which most of the popular varieties were susceptible. This research has opened the gates in India to address the susceptibility of popular rice varieties such as PR106 (Singh et al. 2001), Pusa Basmati-1 (Joseph et al. 2004), and Samba Mahsuri (Sundaram et al. 2008) by pyramiding the BB *R*-genes (*xa5*, *xa13*, and *Xa21*) in the initial phase of improvement. Later, the improvement of popular rice varieties and parental lines continued mainly against BB (*Xa21*, *Xa23*, *xa5*, *xa13*, *Xa4*, *Xa7*, *Xa33*, and *Xa38*) and blast disease (*Pi* genes *Pi2*, *Pi9*, *Pi40*, *Pi54*, *Piz*, and *Pi1*) separately or by combining *R*-genes for both diseases (Gopalakrishnan et al. 2008; Sundaram et al. 2009; Hari et al. 2013; Balachiranjeevi et al. 2015; Yugander et al.

2018; Rekha et al. 2018; Swathi et al. 2019; Jamaloddin et al. 2020). The possibility of recombination between the gene of interest and the linked marker has led to the selection of false-positive rice genotypes in the marker-assisted selection process, which could be overcome by using gene-specific functional markers (Ingvardsen et al. 2008). Many genetic markers, also called functional markers, have been identified for different disease-resistance genes in rice, such as BB-resistance genes *xa5* (Iyer-Pascuzzi and McCouch 2007), *xa13* (Chu et al. 2006), and *Xa21* (Song et al. 1995). The gene-pyramided lines enable the conducting of quantitative analysis to assess the effect of each gene and interactions between them and, most importantly, enhancing the performance, stability, and longevity of genetic resistance.

4.3 Varieties Improved and Developed

Highly accepted varieties and parental lines were improved against multiple diseases through MAS. For the first time, Huang et al. (1997) developed lines pyramided with two, three, and four genes through MAS and tested their resistance against BB. The resistance levels of introgressed lines showed an elevated resistance compared with lines containing a single gene. Later, Singh et al. (2001) improved Indian rice cultivar PR106 against BB through MAS by pyramiding *xa5*, *xa13*, and *Xa21* genes, followed by Joseph et al. (2004), who improved popular basmati variety Pusa Basmati-1, and Sundaram et al. (2008) improved popular variety Samba Mahsuri for BB and reported more than 95% RPG recovery through MABB. Through MAS, three blast genes (*Pi1*, *Pi2*, and *Pi33*) were introduced in the background of popular Russian rice variety Kuboyar. The improved lines of Kuboyar were used to develop blast-resistant hybrids by using them as hybrid parental lines. Similarly, Hari et al. (2011) improved restorer line KMR3R for resistance against BB by transferring the *Xa21* gene along with *Rf3* and *Rf4* (restorer of fertility) genes through MABB. Balachiranjeevi et al. (2015) imparted resistance to a maintainer line (DRR17B) by introgressing *Xa21* and *Pi54* genes against BB and blast disease, respectively.

4.4 Multiple Disease-Resistance Breeding Strategies

In breeding for disease resistance, multiple methodologies such as pedigree, modified bulk, single seed descent (SSD), doubled-haploid (DH), and MAB have been used to develop resistant rice varieties (Mackill et al. 1996; Khush 2005; Collard et al. 2013). In addition to these strategies, the GSR breeding program was one of the successful strategies that involved vigorous phenotypic screening at early backcross stages (BC_1F_2 to BC_1F_4) combined with three successive rounds of stringent selection for the best plant type to come up with climate-resilient rice varieties. This strategy could develop homozygous inbred cultivars within a short span of 4–5 years

vis-à-vis 9–10 years with a conventional breeding program (Yu et al. 2020). The GSR breeding strategy is carried out in three steps. The first is to develop early backcross BC_1F_2 populations by crossing a widely adapted recipient variety with a diverse set of donors. Second is to simultaneously do phenotypic screening of early backcross-derived lines of BC_1F_2, BC_1F_3, and BC_1F_4 generations under the different abiotic and biotic stress conditions in a rigorous manner to identify and select introgression lines (ILs) tolerant of different stresses as compared to the tolerant and susceptible checks. The third step is the mapping of genomic regions influenced by particular climate fluctuations and their characterization to decode the molecular and physiological basis of the identified genomic regions (Ali et al. 2017). Three rounds of screening of populations from BC_1F_2 to BC_1F_4 for different diseases simultaneously could help in the development of varieties with tolerance of multiple biotic stresses. The GSR breeding strategy led to successful mapping of the *Xa39* gene and deploying it in the background of Huang-Hua-Zhang (Zhang et al. 2015). Further, through the designed QTL pyramiding approach, one could combine selective ILs carrying different biotic and abiotic stress-tolerance genes/QTLs derived from different donors but having a common recipient parent. Similar to the GSR breeding program, breeders have simultaneously pyramided multiple disease-resistance genes (BB + blast) with different combinations such as *Xa21* + *Pi54*, *Xa21* + *Pi54* + *Pi2*, and *xa5* + *xa13* + *Xa21* + *Pi54* + *Pi2* into the background of an elite cultivar by employing MAS and MABB (Jiang et al. 2015; Jamaloddin et al. 2020). Recently, one of the successful breeding strategies (the GSR breeding program) revealed lots of hidden genetic diversity for disease resistance through MAS and also proved that RPG recovery could surpass 90% with one backcross followed by selfing (Balachiranjeevi et al. 2019). Furthermore, Feng et al. (2018) reported that pyramiding the detected QTLs effectively broadened the genetic base. Research is being extended to dissect the detected QTLs in order to identify candidate genes through functional validation using a map-based cloning approach.

5 Molecular Mechanisms of Disease Resistance

A wide variety of pathogens, including bacteria, fungi, and viruses, attacks crop plants. Either a pathogen can successfully invade, leading to the development of disease, or the plant can resist the pathogen using an active or passive form of resistance. Different strategies have been developed by various pathogens to enter, infect, and reproduce in plants. Pathogens are mainly classified as necrotrophs and biotrophs based on the method they use to invade, infect, and attack a plant (Oliver and Ipcho 2004). Necrotrophic pathogens kill the host-plant tissue soon after they establish infection and then develop and feed on the dead tissue. Unlike these, biotrophic pathogens require a live-host tissue for their growth and reproduction.

Specific defense mechanisms work effectively against biotrophs through a hypersensitive response developed by rapid local cell death surrounding infection, and this serves to hinder the growth and invasion of pathogens into other plant parts.

This mechanism arises when the first level of the defense mechanism is breached by the pathogen (Zipfel and Felix 2005). Usually, most pathogens that infect plants, such as fungi, harbor secretory proteins, which disrupt these barriers (Serrano et al. 2014). After the entry of the pathogen into the host cell, it is recognized by special molecules called microbe-/pathogen-associated molecular patterns (MAMPs or PAMPs), which include ergosterol, peptidoglycan, lipopolysaccharide, and bacterial flagella in proteins. The innate immune system recognizes these proteins with the help of host plasma membrane-bound receptors called pattern recognition receptors (PRRs) to further obstruct the growth of infection, providing MAMP-triggered immunity (MTI). PRRs also detect molecules that become released in the host when the pathogens cause damage (damage-associated molecular patterns, DAMPs). The binding of these components also triggers pattern-triggered immunity (PTI) and downstream defense responses (Tena et al. 2011). Overall, the recognition of PAMP/MAMP or DAMP results in the activation of PTI, triggering the production of different reactive oxygen species (ROS), initiation of mitogen-activated protein (MAP) kinase activity, and various transcription factor activation, thus limiting the spread of pathogens completely (Nürnberger and Kemmerling 2009).

The widely accepted model of plant disease resistance is explained by a two-level innate immune system. The two levels include PTI, which is usually a weak, basal, and generic immune response, and the other is effector-triggered immunity (ETI), which is a potent response and is specific to the pathogen in question (Jones and Dangl 2006). PTI is mediated by the PRRs that recognize molecular patterns associated with the pathogens or the resulting damage products (PAMPs or DAMPs). On the other hand, ETI includes recognition of a pathogen-specific factor and results in a severe and rapid form of immune response leading to localized cell death (also known as a hypersensitive response or HR) to hinder the pathogen from spreading any further. ETI is achieved by a gene-to-gene interaction and is thus specific to the race of the pathogen. While PRRs mediate PTI, ETI is mediated by specific genes that belong to the nucleotide-binding-leucine-rich repeat (NB-LRR) domain-containing proteins, otherwise called resistance (*R*) genes. The recognition of cognate ligands results in activation of signaling events that in turn results in the generation of different forms of immune response such as callose and lignin deposition, production of antimicrobial compounds, induction of cell death, changes in primary and secondary metabolic flux, and synthesis of secondary metabolites depending on the type of elicitors. Other classifications of genes involved in disease resistance include major resistance (MR) genes and defense-related genes (DR), whose roles cannot be explained by the definition of PTI- and ETI-associated genes (Ke et al. 2017). PTI is considered to be quantitative in nature, that is, multiple genes function together to achieve immunity, also known as a QTL. ETI against a pathogen strain is controlled by a single gene and is specific only to those strains that contain the cognate avirulence (*Avr*) protein that the *R*-gene recognizes, thus leading to a qualitative resistance. Studies in the past few decades established a framework of how the resistance mechanisms act using model pathosystems. Along this line, rice resistance to its major pathogens such as *Xanthomonas oryzae* ssp., *Magnaporthe oryzae*, and *Rhizoctonia solani* has been studied to a reasonable

extent. However, rice resistance to other pathogens still needs more investigation to come to a consensus. This section summarizes the established mechanisms of disease resistance in rice.

Plant resistance is dictated by the type of resistance genes and a network of signaling pathways (Chisholm et al. 2006). Broadly, the plant defense system can be categorized into two classes: basal defense and specific defense. The basal defense system is much more effective against necrotrophic pathogens (Singh et al. 2018). Elicitors are molecules that induce a plant defense response at very low concentrations (Thakur and Sohal 2013). The role of the basal defense system is to check the entry of pathogens and provide immunity at the starting stage of infection. This defense response involves membrane permeability, activating ion fluxes (Ca^{2+}, K^{+}, H^{+}), generating ROS, producing nitric oxide (NO), and phosphorylation/dephosphorylation of proteins by protein kinases and phosphatases. It also includes the production of signaling molecules such as jasmonic acid (JA), salycilic acid (SA), and ethylene (ET). These proteins are characteristic players in the regulation of defense signal transduction cascades. These steps further trigger an array of signaling that leads to the regulation of the expression of defense-related genes and the stimulation of defense responses. These responses include cell-wall strengthening (callose and lignin deposition), phytoalexin synthesis, and activation of kinase cascades escorted by a hypersensitive response (Jones and Dangl 2006).

5.1 Resistance to Bacterial Blight

To date, 46 resistance genes have been identified to confer resistance to *Xoo* in rice. Among them, 11 genes were cloned and functionally characterized. Some of the resistance genes are quantitative in nature, whereas others confer qualitative resistance (Ke et al. 2017; Jiang et al. 2020; Chen et al. 2020). The 11 cloned genes fall under different classes of resistance genes: LRR-RLKs (leucine-rich repeat receptor-like kinases), NB-LRR, a wall-associated kinase, executor R proteins, *SWEET* (sugars will eventually be exported transporters) genes, and a transcription factor gamma subunit protein. Three of the cloned resistance genes, *Xa3/Xa26*, *Xa4*, and *Xa21*, code for kinases. *Xa4* is a wall-associated kinase (Ke et al. 2017; Jiang et al. 2020) that provides resistance to certain races of *Xoo* through cell-wall reinforcement. *Xa3/Xa26* and *Xa21* are LRR-RLKs that recognize *Xoo*-associated molecules AvrXa3 and sulphated RaxX, respectively. *Xa21*- and *Xa3/Xa26*-mediated resistance has been found to be positively regulated by *OsSERK2* (rice somatic embryogenesis receptor kinase 2). Nine genes were found to be regulating *Xa21*-mediated resistance positively or negatively. *Xa4*-mediated resistance leads to the accumulation of phytoalexins. *Xa1*, an NB-LRR, recognizes intact transcription activation-like effectors (TALEs) from *Xoo* and thus leads to resistance. *SWEET* genes code for sugar transporters and were identified to be targets of different *Xoo* TALEs, thereby acting as susceptibility factors. Natural polymorphisms were identified in the promoters of three SWEET genes, *OsSWEET11/Os8N3/xa13*, *OsSWEET13/*

xa25, and *OsSWEET14/Os11N3/xa41*, which promote their induction by cognate TALEs, thus providing recessive resistance. Genes, including *Xa10*, *Xa23*, and *Xa27*, are classified as executor *R*-genes as the expression of the respective resistance alleles is induced by *Xoo* TALEs. These genes are characterized by the presence of multiple potential transmembrane domains whose expression induction results in HR and thus resistance to *Xoo*. Another recessive resistance gene, *xa5*, codes for transcription factor IIA gamma subunit 5 (TFIIAγ5) with valine to glutamine mutation in the 39th position. The susceptible allele, *Xa5*, is hijacked by the TALEs to induce the expression of other host susceptibility genes. The mutation disrupts the ability of TALEs to bind to TFIIAγ5, thus leading to resistance (Ke et al. 2017; Jiang et al. 2020).

5.2 Resistance to Bacterial Leaf Streak

To date, no major BLS-resistance genes have been identified. However, the *xa5* gene was mapped to be a major resistance QTL for *Xoc* resistance. It was previously observed that TALEs from *Xoc* also hijack TFIIAγ5 for inducing host susceptibility genes. In another study, *Xo1*, a resistance locus in an American rice variety, was identified to be responsible for resistance to African *Xoc* strains but not to Asian strains. *Xa21* was identified to provide weak resistance to *Xoc* through the recognition of Ax21, a quorum-sensing molecule produced by *Xoc* (Jiang et al. 2020). Three major broad-spectrum resistance QTLs, *qXO-2-1*, *qXO-4-1*, and *qXO-11-2*, were identified to confer resistance to *Xoo* and *Xoc* (Bossa-Castro et al. 2018).

5.3 Resistance to Rice Blast

More than 100 resistance genes and 500 QTLs are known to be associated with blast resistance in rice. However, to date, only 25 genes have been cloned (Li et al. 2019b). These 25 cloned *R*-genes are called *Pi* genes. Of the 25 *Pi* genes, 22 encode NB-LRR family proteins. A majority of these *R*-genes trigger ETI, thus leading to qualitative or race-specific resistance. So far, seven *R*-genes have been identified to confer broad-spectrum resistance to blast: *Pi7*, *Pi9*, *Pi21*, *Pi50*, *Pi57*, *Pigm*, and *Ptr*. Apart from canonical *R*-genes, so far, five defense-related genes were also shown to confer resistance to blast: *bsr-d1*, *bsr-k1*, *spl11*, *spl33*, and *OsBBI1*, *Pi9*, *Pi50*, *Pigm*, *Ptr*, and *OsBBI1* are dominant resistance genes or positive regulators of blast resistance, whereas the rest of them are recessive resistance genes, in other words, their wild-type alleles negatively regulate blast resistance (Li et al. 2019b).

5.4 Resistance to Sheath Blight

Information on the mechanisms that govern ShB resistance in rice is just being uncovered. There are no reports on a single resistance gene that confers resistance to ShB. However, many QTLs have been identified to be associated with ShB resistance. Most of the QTLs were reported to provide a minor contribution to the resistance phenotype, whereas two QTLs (*qShB9-2* and *qShB11-1*) were found to contribute more than 10% to ShB resistance. Sequence analyses revealed the presence of various defense-associated genes in these QTLs. *qShB9-2* was identified in many rice varieties that exhibit resistance to ShB. It was observed that *qShB9-2* contains a β-1,3-glucanase, *OsWAK91*, and 12 other possible candidate genes. On the other hand, the *qShB11-1* interval was shown to have receptor-like kinases, a lipase, and a tandem array of 11 chitinase genes. Tens of minor QTLs were found to be associated with ShB resistance. Nevertheless, no information is available on the gene(s) responsible for the resistance. Studies using resistant cultivars shed light on the possible mechanisms by which rice fights *Rhizoctonia solani*. Various studies showed changes in metabolic pathways, including primary and secondary metabolites. Intermediates of glycolysis and tricarboxylic acid cycle were found to be accumulated in rice post *R.solani* infection, indicating the possible involvement of primary metabolism in response to the pathogen. Also, the accumulation of secondary metabolites such as phytoalexins, chlorogenic acid, polyphenols, and flavonoids was reported to be higher in the tolerant varieties than in the susceptible varieties postchitin treatment (Molla et al. 2020). ROS deregulation has been observed to delay pathogen colonization in resistant cultivars (Oreiro et al. 2019).

5.5 Broad-Spectrum Resistance Genes

From a breeder's point of view, a single locus/gene is more preferred as it would permit easier introgression. Many defense-related genes have been identified to provide broad-spectrum resistance to either multiple races of a pathogen (vertical resistance) or multiple pathogens altogether (horizontal resistance). Such responses are quantitative in nature and hence can be highly durable and practical to keep infectious diseases at bay. Several previous studies have been reported that the expression of defense-response genes (DR genes) such as rice germin-like proteins (*OsGLP*) or a class of DR genes present in a QTL along with *R* genes is also most probably associated with rice resistance, as knockdown of these genes escalated the susceptibility against two major rice fungal diseases, blast and sheath blight (Manosalva et al. 2009). *OsPAL4* is reported to impart broad-spectrum resistance to rice (Tonnessen et al. 2015). A LysM receptor-like kinase (RLS), *OsCERK1*, regulates cytoplasmic *OsRLCK176* and *OsRLCK185* recognizes chitin and peptidoglycans activating immune signaling pathways in rice against blast and bacterial blight diseases. *OsSERK1*, *OsWAK25*, *OsWRKY45-1*, *OsWRKY45-2*, *OsWRKY13*, *OsDR8*,

OsMPK6, *OsPAL4*, *OsNH1*, *OsLYP4*, *OsBSR1*, and *OSK35* have all been shown to regulate resistance to bacterial blight and rice blast positively. *OsPAD4* and *OsPAL4* positively regulate resistance to ShB, whereas *OsWAK25* negatively regulates ShB resistance (Ke et al. 2017). These genes, although identified in different studies, play a highly connected role in helping rice fight the invading pathogens. More comprehensive studies are needed to link the dots to construct a complete map of rice resistance to diseases.

6 Impact of Major Nutrient Fertilizers on Biotic Disease Resistance in Rice

The rapidly increasing world population requires a sustainable nutritional global food supply, which is a significant concern for crop production. Changing climatic scenarios and decreasing natural resources suggest that there is a need to intensify agricultural production using an efficient agronomic nutrient management (ANM) system. Following efficient ANM technologies can enable us to understand and mitigate the adverse impacts of stress, inadequate soil fertility status, pathogens, and pests (Dordas 2008).

Several efficient screening technologies exist, such as smart water irrigation systems, integrated fertilizer applications, and disease biocontrol strategies, that have been developed and adopted in different ecosystems to control various diseases in rice (Bargaz et al. 2018). Among these, the rate of fertilizer used, judicious and timely applications of nutrients, and availability of these nutrients play a crucial role in plant growth and also in developing defense mechanisms against various pests and diseases (Fageria et al. 2008; Sun et al. 2020). The management of nutrient statuses in the soil, especially nitrogen (N), phosphorus (P), and potassium (K), is an eco-friendly strategy to control different biotic stresses instead of frequent application of pesticides. Globally, the efficiency of fertilizer use by the crop and the correct rates of fertilizer applications are poorly studied. Earlier studies have indicated that only 30–35% of N, 10–25% of P, and 35–50% of K are taken up by plants. Particularly in China, the amount of fertilizer used has increased drastically from 270 to 350 kg/ha, which is more than 75% of the global average of fertilizer application. This excessive amount of N fertilizer leads to leaching, which is a significant cause of groundwater pollution and degradation of soil quality (Teng et al. 2016).

Developing sustainable agriculture is one of the major strategies to increase global rice production. Application of nutrient fertilizer at the right rate and stage and also microorganisms are the key factors in disease control. The essential nutrient elements can decrease disease severity but also increase the severity of disease incidence (Dordas 2008). Nitrogen is one of the key elements for plant growth and development, which are involved in the major physiological and metabolic pathways related to N assimilation (Bolton and Thomma 2008; Mur et al. 2017). Plenty of research has been conducted on the role of N and its interaction with disease

resistance and the results are inconsistent, with a poor understanding of the resistance mechanisms in physiological and metabolic pathways. These differences may be due to various stress signaling mechanisms caused by the different forms of N (NH_4^+ and NO_3^-), the type of pathogen specificity, and the stage of N application (Dordas 2008). However, several researchers have suggested that the correct time application of fertilizers has been significantly increasing disease resistance and decreasing the use of fungicides (Anderson 2002; Hervieux et al. 2002; Bhat et al. 2013). Recently, Sun et al. (2020) reviewed N applications and their critical role in the defense mechanisms in various diseases such as blast, downy mildew, stem rot, powdery mildew, leaf rust, stem rust, and rice blast diseases in plants. Balancing of these nutrients is imperative to understand the cellular structure and composition, which mainly affect plant defense mechanisms. For instance, high rates of N application lead to a significant impact on susceptibility by decreasing the thickness of cell-wall components (cellulose and lignin), whereas decreasing N applications lead to an increase in lodging resistance by changes in stem lignification and secondary cell-wall synthesis (Zhang et al. 2017b; Sun et al. 2018). Also, decreasing N fertilizer significantly increases the incidence of major insect pests, including brown planthopper, leaffolder, and stem borer, the key insect pests in the major rice-growing areas in Asian countries (Lu et al. 2007). Some reports have suggested that N applications significantly influence the size of leaf blast lesions (Matsuyama 1973; Kaur et al. 1979). Sime et al. (2017) studied the different rates of nutrient fertilizer application and their relation to blast disease. The combination of NPK (20-10-10) at a rate of 200 kg/ha has a remarkable impact on decreasing blast disease in all phases of plant growth. Similarly, Reddy et al. (1979) reported an optimal rate of N application (76 kg/ha) to maximize grain yield and also minimize disease. One of the major diseases is bacterial leaf blight of rice, caused by *Xanthomonas oryzae*, which increased significantly when a higher amount of N fertilizer (>100 kg/ha) was applied, and yield decreased. Begum et al. (2011) reported that a balanced application of nutrient fertilizers, including K, significantly decreased the percentage of BLB. The application of K fertilizer has dramatically decreased the intensity of various infectious diseases such as BLB, sheath blight, and stem rot in rice, and also in other cereal crops (Sharma et al. 2005a). Decreasing BLB severity by applying K topdressing is a viable approach just before disease-occurring stages and this makes it possible to maximize grain yield and have lesser disease development.

Using slag-based silicon (Si) fertilizer in rice fields is an alternative approach to control the major disease brown spot, which is caused by the fungus *Bipolaris oryzae*. This disease causes significant yield losses, mainly in tropical and subtropical areas, where the frequent occurrences of heavy rainfall and high temperature are the main factors in decreasing the Si content in highly weathered soils (Raven 2003). The major role of these Si applications is to mediate resistance mechanisms through the physiological and metabolic pathways that can lead to creating more pronounced cell silicification in rice leaves, and the strong leaf epidermal surface might increase the resistance to fungal penetration (Hayasaka et al. 2008; Sun et al. 2010; Ning et al. 2014). These Si fertilizers provided clear evidence showing the importance of increasing the thickness of the silicon layer in the epidermal cell walls that are

supposed to be the main site for conferring resistance to brown spot disease in rice. Interestingly, Wu et al. (2017) experimented with the transcriptional responses in two different nutrient fertilizers, Si and N concentrations, and their relation to BPH infestation. These two elements had a trade-off mechanism in terms of resistance. The interaction of these two elements clearly showed decreases in the expression of Si transporters such as OsLsi1 and OsLsi2 under high rates of N application, whereas, in the N transporters *OsNRT1:1*, *OsGS2*, *OsFd-GOGAT*, *OsNADH-GOGAT2*, and *OsGDH2*, expression increased under a high rate of Si fertilizer. This demonstrated that N and Si had antagonistic interactions in rice (Wu et al. 2017). Similarly, Robichaux (2001) identified a significant decrease in the major disease sheath blight, caused by the fungal pathogen *Rhizoctonia solani*, by adding calcium silicate in greenhouse and field conditions. Rice grain yield is increased by almost 13% from the use of a calcium silicate application rate of 3.3 mg/ha and also a significant decrease in ShB in different soil types. These results have proven that Si fertilizer can diminish fungal disease severity by increasing the Si concentration in rice leaves and boosting grain yield.

7 Genome-Editing Tools for Improving Disease Resistance

Diseases cause a considerable yield loss annually (Heinrichs and Muniappan 2017; Mushtaq et al. 2019). Breeding for disease resistance has been pursued for a long time. The traditional practice is to introgress disease resistance into elite cultivars through breeding techniques. Although a successful method, it has its downside (Zafar et al. 2020). The traditional way is time-, labor-, and resource-consuming (Romero and Gatica-Arias 2019). With the arrival of the genomics era, identifying disease-resistance genes has become highly efficient, and resistance alleles can be identified at a single base resolution. With such a massive potential in hand and constant improvement in various genome-editing (GE) tools such as site-specific mutagenesis (SSM), meganucleases (MNs), zinc-finger nucleases (ZFNs), transcription activator-like effector nucleases (TALENs), and clustered regularly interspaced short palindromic repeat (CRISPR)/CRISPR-associated protein (CRISPR/Cas), this has opened a novel path to achieve the improvement of disease resistance in various crops (Zhang et al. 2017a; Mishra et al. 2018; Zafar et al. 2020). Recently, several researchers have reviewed the various GE tool applications and their limitations in target gene specificity and accuracy (Abdallah et al. 2015; Mishra et al. 2018; Zafar et al. 2020). As compared with SSM, MNs, and ZFNs, the most widely used GE tools such as TALENs and the CRISPR/Cas system have a versatile, fast, and relatively efficient GE method. Over the past several years, these two methods have transformed the field of genome engineering, and they can easily edit and also recognize specific genomic regions (Gaj et al. 2016). These methods have a significant impact on the genomic revolution that has accelerated the discovery of novel sequence variations and breakthroughs in the scientific knowledge to demonstrate the power of these GE tools in establishing resistance to pathogens in various

diseases. The rapid progress in the CRISPR-Cas9 system makes it a highly accurate and efficient method that can edit in multiple genes at multiple locations using a single molecular construct (Cong et al. 2013).

7.1 Site-Specific Mutagenesis: The Path So Far

Site-specific mutagenesis is achieved by deploying a class of enzymes called "nucleases" fused with DNA-binding motifs to target specific sequences in the genome. The activity of nuclease results in double-strand breaks at the target site, which are then repaired by the host DNA repair mechanisms via nonhomologous end joining (NHEJ) or homologous recombination (Feng et al. 2013). During this process, small insertions or deletions occur in the genome, thus disrupting the gene sequence (Mishra et al. 2018). SSM is an evolving area of research with newer tools often emerging with improved precision and efficiency.

7.1.1 Meganucleases

Meganucleases (MNs) are endonucleases (enzymes that cut within a strand) that occur naturally and possess sequence-specific DNA-binding and nuclease activities. The application of MNs for site-targeted mutagenesis began in the 1980s. Owing to the recognition of long DNA sequences (18–40 bp), MNs were a good choice. On the other hand, the number of naturally occurring MNs was limited, thus diminishing their wider application. Moreover, custom modification of MNs is a viable but expensive option (Abdallah et al. 2015).

7.1.2 Zinc-Finger Nucleases and TALENs

Zinc-finger nucleases (ZFNs) kick-started the wider application of site-specific mutagenesis in 1996 (Kim et al. 1996). The zinc-finger motif is one of the most copious DNA-binding motifs present in eukaryotes (Klug and Schwabe 1995). Each ZF motif recognizes a specific 3-bp sequence in the major groove of DNA. Thus, tandemly placing multiple ZF motifs of different base specificity and fusing them to a nuclease can result in the generation of a molecular scissor that can precisely cut the target site. Modular assembly-based methods enabled the construction of ZFNs that can virtually target any DNA sequence (Gaj et al. 2013). However, limitations of using ZFNs exist. The modular assembly is a complex and expensive process that requires many optimizations. Off-target cleavage is another challenge that many SSM techniques face (Ramirez et al. 2008; Gupta and Musunuru 2014).

Transcription activator-like effectors (TALEs) are proteins that naturally occur in the genus *Xanthomonas*, which predominantly consists of phytopathogenic bacteria (Boch et al. 2009; Moscou and Bogdanove 2009). TALEs are employed by

Xanthomonas oryzae pv. *Oryzae* (*Xoo*) to target and activate the expression of specific host genes to increase the susceptibility of the host. The specific binding to DNA is achieved by a 33–35-amino-acid-long tandem repeat domain, each of which targets a specific base. The base specificity is conferred by the amino acids that are located in the 12th and 13th positions of the series. These positions are called repeat variable di-residues (RVDs) (Boch et al. 2009; Moscou and Bogdanove 2009; Gaj et al. 2013; Abdallah et al. 2015). Exploiting this brought in a revolution in the field of genome editing called TALENs. TALENs are TALE nucleases wherein the DNA-binding motif of a TALE is fused with a catalytic domain of a nuclease, thus allowing the domain to target and cleave a specific sequence in the genome. By modifying the RVDs, one could define the target site and thus assemble a custom TALEN to target any region of interest in the genome (Christian et al. 2010; Boch 2011). The design and delivery of TALENs, however, pose a setback for the technique owing to their large size (Abdallah et al. 2015).

7.1.3 CRISPR-Based Genome Editing

Clustered regularly interspaced short palindromic repeats (CRISPR) and CRISPR-associated (Cas), collectively called CRISPR-Cas, is a microbial adaptive immune strategy that works based on an RNA-guided nuclease complex to cleave foreign genetic elements. A CRISPR-Cas locus is a cluster of Cas genes, noncoding RNA, and an array of repetitive elements. The repeated elements are interspaced with protospacers (short variable repeats that are derived from foreign DNA targets). Together, the noncoding RNA and protospacers constitute the CRISPR RNA (crRNA). Each protospacer is associated with a protospacer adjacent motif (PAM) that differs between the types of CRISPR systems. Depending on the organization and composition of the nuclease genes, the CRISPR-Cas system is classified into Class I and Class II. Each class has three types of CRISPR-Cas system each. The Class I CRISPR system is less used owing to its limited knowledge and associated complexities. The Class II system, on the other hand, is a well-characterized and highly used genome-editing system. Class II is further subclassified into three types (types II, V, and VI) based on the specificities for nucleotide substrates, PAM, and the Cas genes that affect the substrate cleavage (Koonin and Makarova 2019; Moon et al. 2019). A brief overview is discussed below.

CRISPR-Cas9 from *Streptomyces pyogenes* is the founding system for CRISPR-based genome editing, which is economical, easier, and more efficient. Cas9 is an RNA-guided nuclease that causes double-strand breaks in the genomic region that is complementary to the crRNA provided that the 3′ of the DNA sequence is 5′-NGG-3′ (G-rich PAM). In addition, the CRISPR-Cas9 system needs a *trans*-acting crRNA (tracrRNA) to be functional (Deltcheva et al. 2011; Jinek et al. 2012). Cas9 proteins of different bacterial origins have different PAM specificities, spacer lengths, and sizes. Improvements in the techniques made it possible to multiplex genome editing with the use of polycistronic tRNA-gRNA (PTG), wherein the tRNA processing system is used to construct a tandem array of tRNA and gRNA

(attached to the spacer), which would be transcribed as a full primary transcript that is later processed and cleaved to release individual single-guide RNAs (sgRNAs), each of which targets a unique region in the genome (Zafar et al. 2020).

Cpf1, also known as Cas12a, belongs to the type V CRISPR-Cas system. Unlike Cas9, Cas12a does not need tracrRNA for the complex to be functional and it recognizes a 5′ T-rich PAM sequence (5′-TTTN-3′ or 5′-TTN-3′) (Zetsche et al. 2015). Also, Cpf1 exhibits RNase activity that can cleave pre-crRNAs to mature crRNA, thus enabling the possibility of including multiple crRNAs in a single cassette. Cpf1 allows the use of longer gRNAs of up to 100 nt (Zetsche et al. 2015; Mishra et al. 2018). This system is gaining usage because of its higher specificity and enhanced efficiency. Advances are made in terms of increasing the range of targets by engineering the complex to target other PAMs.

Base editing can be used to modify single bases in the genome, thus opening an avenue to increase the allelic diversity of the genes and also to create specific mutations to alter a gene function. The use of the CRISPR-Cas system achieves this in conjunction with base-modifying enzymes such as cytidine deaminase (to induce C:G to A:T mutations) or adenine deaminase (to induce A:T to G:C mutations). Base-editing techniques eliminate the need for double-strand breaks and thus the activation of DNA repair pathways (Lu and Zhu 2017; Hao et al. 2019).

Gene knock-in thus far required double-strand breaks and activation of homology-directed repair (HDR), which uses a donor template (carrying the gene copy to be knocked-in) to incorporate the new copy in the genome. This technique is extremely limited because of the less frequent and cell cycle stage-dependent nature of HDR. Also, the effective delivery of the donor template has posed a serious challenge. To overcome this, an elegant method was devised, called prime editing. Prime editing depends on a two-component system that includes (1) Cas9 nickase fused with a reverse transcriptase and (2) a prime editing guide RNA (pegRNA) that carries the desired edit(s) to be incorporated into the genome. Once delivered, the complex is guided to the target site by pegRNA and a nick is created in the genome. The nicked DNA serves as a primer that the reverse transcriptase uses to reverse transcribe the pegRNA, thus incorporating the edit into the genome (Anzalone et al. 2019; Lin et al. 2020). All the above-discussed techniques were successfully applied in rice and other plant species to edit various genes. The following few paragraphs will provide a glimpse of the application of genome editing in rice, focusing mainly on disease resistance.

7.2 Application of Genome Editing in Biotic Stress Tolerance in Rice

Conventional breeding has been successfully employed to date to develop disease-resistant rice varieties by introgressing resistance genes from wild rice varieties or landraces into elite cultivars. Although successful, it is a time-consuming

procedure, and also the wild germplasm does not contain genes/loci for all the economically important traits that are of concern for breeders. With the advent of genome editing, several research groups started testing the possibility of using genome editing in rice and were by and large successful.

7.2.1 Resistance to Biotic Stress Factors

Genome editing has been successfully employed to generate rice plants resistant to various biotic stress factors, including bacterial blight, bacterial leaf streak, blast, and tungro virus. The application of genome-editing techniques for BB resistance began with modifying the promoter of a BB susceptibility gene, *OsSWEET14* (*Os11N3*), using TALENs (Li et al. 2012). *SWEET* genes are sugar transporters, and the expression of certain *SWEET* genes is induced upon *Xoo* infection by the action of TALEs. So far, the *SWEET* genes, including *OsSWEET11*, *OsSWEET12*, *OsSWEET13*, *OsSWEET14*, and *OsSWEET15*, have been shown to be induced by *Xoo* and could act as susceptibility factors (Streubel et al. 2013). TAL effectors bind to the effector-binding elements (EBEs) in the target promoter and activate the expression of the downstream gene, which tends to be a susceptibility factor in many cases. Different TAL effectors induce many such susceptibility genes, and their cognate EBEs were also deciphered. Li et al. (2012) have successfully employed TALENs to modify the EBEs of *OsSWEET14*. This study established the possibility of using genome-editing techniques to generate disease-resistant varieties as well as to understand the targets of different TAL effectors. Jiang et al. (2020) have conducted a proof-of-concept study to confirm the applicability of the CRISPR-Cas9 system in rice by editing the promoters of *OsSWEET11* and *OsSWEET14* genes (Jiang et al. 2013). In a study using CRISPR-Cas9, Zhou et al. (2015) created a null mutant of *OsSWEET13* to show that *PthXo2* (an *Xoo* TAL effector)-dependent disease occurrence needs intact *OsSWEET13*. Xu et al. (2019) used CRISPR-Cas9 to edit *OsSWEET11* and *OsSWEET14* to engineer broad-spectrum resistance to BB in rice variety Kitaake. In addition to that, they identified new EBEs in the promoter of *OsSWEET13* and successfully used CRISPR-Cas9 to disrupt the EBE, thus generating a rice line that was resistant to all the tested *Xoo* isolates ($n = 133$) (Xu et al. 2019). Oliva et al. (2019) generated five mutations in the promoters of *OsSWEET11*, *OsSWEET13*, and *OsSWEET14* in three rice lines, Kitaake, IR64, and Ciherang-Sub1. All the lines were reported to show robust and broad-spectrum resistance in the paddy trials (Oliva et al. 2019). Zhou et al. (2018) used CRISPR-Cas9 to create a knockout of a susceptibility gene called *BsrK-1* (broad-spectrum resistance Kitaake-1), which resulted in resistance to BB as well as blast. *BsrK-1* is a tetratricopeptide domain-containing protein that was shown to bind to the mRNAs of multiple *OsPAL* (phenylalanine ammonia-lyase) genes and promote their turnover. In *BsrK-1* knockouts, the accumulation of *OsPAL* mRNA was observed along with increased resistance to diseases (Zhou et al. 2018). The feasibility of the

transgene-free method of genome editing was tested by mutating *Os8N3/OsSWEET11* (Kim et al. 2019). Cai et al. (2017) demonstrated that a TAL effector (*Tal7*) from *X. oryzae* pv. *oryzicola* RS105 targets the promoter of rice *Cyclin-D4-1* and induces its expression. They have successfully applied TALEN-based genome editing to disrupt the EBE in the promoter of *Cyclin-D4-1*, which leads to resistance to RS105 infection.

The applicability of CRISPR-Cas9 for generating blast-resistant rice lines has been demonstrated by performing both single-site and multisite-targeted mutagenesis of *OsERF922*, a negative regulator of blast resistance, to produce knockouts. All the mutants showed blast resistance while not having any adverse effect on other agronomic traits (Wang et al. 2016). Rice tungro disease (RTD) is a disease caused by rice tungrospherical virus (RTSV) and rice tungrobacilliform virus (RTBV) and is transmitted by green leafhoppers. RTD results in yellowing of leaves, decreased tiller numbers, and stunted growth (Azzam and Chancellor 2002; Lee et al. 2010). Macovei et al. (2018) generated RTSV-resistant lines in the background of IR64 using the CRISPR-Cas9 system. In this study, the *eIF4G* gene was successfully mutated independently, using three different gRNAs, and the mutant plants showed heritable resistance to RTSV (Macovei et al. 2018).

7.2.2 Summary of Nonbiotic Stress-Related Phenotypes

The application of genome editing in rice is rising with time. Other than for biotic stress tolerance, genome editing has been successfully applied to edit several genes having various roles, including nutritional value, yield, and abiotic stress tolerance (Shan et al. 2015; Li et al. 2016; Sun et al. 2016; Shen et al. 2017; Tang et al. 2017; Abe et al. 2018; Endo et al. 2019; Romero and Gatica-Arias 2019).

7.3 Improvements in the Techniques

Currently, transgene-free methods are being tested and employed for genome editing wherein the mutant plants do not contain any of the CRISPR-Cas9 components. This is achieved in several ways, including using Cas9-gRNA ribonucleoproteins (RNPs). This RNP complex is directly delivered into plant cells by transfection or particle bombardment. The RNP complex can perform the editing and will be degraded by the cellular types of machinery. Another approach is to transiently express CRISPR-Cas9 from DNA or RNA in plants from regenerated calli. Both methods suffer from the possibility of component degradation, which might lead to less-efficient editing. To eliminate this disadvantage, He et al. (2018) came up with the suicide gene-based method of a transgene-free CRISPR-Cas9 approach in rice. In this method, a pair of suicidal genes, encoding toxic proteins that kill plant cells,

are incorporated into the CRISPR-Cas9 cassette. Therefore, no plant with a CRISPR-Cas9 construct will survive, thus eliminating the plants containing the transgenes. Among other surviving plants, the true mutants can be screened and identified using appropriate techniques. In addition to protein-coding genes, miRNAs are being targeted for editing owing to their involvement in various growth, development, and stress-response pathways. The use of the CRISPR-Cas system to edit miRNAs has been functionally validated in rice (Zhou et al. 2017; Mangrauthia et al. 2017).

8 Bioinformatics Tools for Disease Resistance and Management

Bioinformatics is an interdisciplinary field that uses computational tools to capture and interpret the function of various genes. The advent of genomics has revolutionized every aspect of life science. The availability of a large amount of data has necessitated better ways to analyze, interpret, and organize the results for the scientific community (Bayat 2002; Vassilev et al. 2005). Thousands of databases and repositories are available for various datasets such as for the genome, gene and protein sequences, expression and coexpression of genes, and genomic variations such as SNPs and InDels, to name a few (Garg and Jaiswal 2016). With time, sequencing platforms have seen an astounding revolution and are becoming more efficient and affordable day by day. Since the first report on the whole-genome sequence of rice in 2005, many varieties were further sequenced as a part of the 3000 Rice Genomes Project (Matsumoto et al. 2005; Li et al. 2014). The data obtained from the project resulted in establishing rice variation databases and these data have provided invaluable insights into rice evolution and domestication (Chen et al. 2019). Moreover, the readily available data can guide breeders to wisely choose varieties and markers for breeding various traits from one cultivar to another. Procedures to score the expression of genes have also undergone an overwhelming transformation from methods such as serial analysis of gene expression (SAGE) to microarrays to RNA-sequencing (Perez-de-Castro et al. 2012). As a result, other than genome databases, gene and protein expression databases play an important role in elucidating the various mechanisms that control a given trait, such as days to flowering, growth and development, abiotic stress tolerance, and disease resistance, among others. Multiple other tools and databases are available to study and acquire information on different aspects, including phylogenomics, protein-protein interaction, promoter analysis, gene and QTL information, marker-trait association, and metabolite profiles (Garg and Jaiswal 2016). This section aims to provide an overview of the application of bioinformatics in breeding for disease resistance in rice.

8.1 The Role of Bioinformatics in Mapping Genomic Loci

8.1.1 Mapping QTLs and Genes Associated with Disease Resistance

Mapping the loci responsible for a desired trait has been successfully carried out for years using a conventional method such as simple sequence repeat (SSR)-based genotyping of a mapping population. With the arrival of affordable sequencing techniques, QTLs and genes can now be mapped at a gene-level resolution. Methods such as MutMap and QTLseq have made it possible not only to identify the genomic locus responsible for a trait but also to pinpoint the causal variation within a gene that led to the phenotype (Abe et al. 2012; Takagi et al. 2013b). Thus, SNP markers that are highly associated with a trait can be identified and employed in breeding programmed for efficient introgression of the trait. In a proof-of-concept study, Takagi et al. (2013a) had identified a QTL conferring partial resistance to blast disease of rice. Following this attempt, multiple studies have successfully used this procedure to map QTLs for various traits in rice and other species. A rice blast resistance gene called *Pii* was mapped by another method called MutMap-Gap (Takagi et al. 2013b).

Sequencing data have been successfully used to compare the genomes of different cultivars and obtain the resistance alleles of cloned rice blast resistance genes (Mahesh et al. 2016). Genes with highly repetitive sequences pose a challenge in accurately characterizing them in context with short-read sequences. A recent study by Read et al. (2020) addressed this challenge by using a long-read sequencing approach called nanopore sequencing in combination with Illumina sequencing to assemble the genome of American rice variety Carolina Gold Select and identify 529 complete or partial NB-LRR domain-containing protein genes that are highly repetitive in nature. The study identified a major disease resistance locus called *Xo1* that confers resistance to *Xanthomonas oryzae* pv. *oryzae* (the causal agent of bacterial blight of rice) and *X. oryzae* pv. *oryzicola* (the causal agent of bacterial leaf streak of rice). Also, a blast resistance gene called *Pi63* at the *Xo1* locus was identified (Read et al. 2020). Another study compared the genomes of 13 domesticated and wild rice relatives and shed light on the complex phylogeny of the *Oryza* genus and identified many haplotypes of disease-resistance genes that can be of potential use for breeding (Stein et al. 2018). Using the genome sequence of rice variety Tetep, an extensive set of molecular markers was designed for breeding novel resistant varieties (Wang et al. 2019).

8.1.2 Genome-Wide Association Studies

Genome-wide association studies (GWAS) exploit the natural variation among different cultivars to identify trait-associated genes (Hu et al. 2018). This is one of the preferred methods for the identification of gene targets for breeding. The availability of genome sequences and phenotype data, along with powerful statistical and

bioinformatics tools, have made it possible to analyze hundreds or thousands of genomes in one go and identify genes and haplotypes that are associated with given traits. A GWAS on 373 *indica* rice sequences identified SNPs associated with 14 different agronomic characteristics (Huang et al. 2010). A GWAS with a panel of 584 rice accessions led to the identification of a gene called *PiPR1* that confers partial resistance to blast disease of rice (Liu et al. 2020). Other GWAS have identified tens of QTLs and new alleles of known blast-resistance genes (Li et al. 2019a).

8.1.3 Speeding Up Breeding

The main setback with conventional breeding methods is the time taken for developing a new variety. Also, for durable disease resistance and other complex traits, it is essential to efficiently identify minor-effect QTLs and use the associated markers in breeding programs. The existing methods, including QTL mapping from biparental crossing and GWAS, are not up to the mark to efficiently identify such minor-effect QTLs (Bhat et al. 2016). To address both of these concerns, genomic selection (GS) was proposed (Meuwissen et al. 2001). Unlike MAS, GS does not necessarily need QTL information before selection. GS uses reference population data containing phenotype and high-density marker data to predict breeding values for all the markers. Based on the predicted values, the breeding population data will be analyzed to select the individual that possesses the desirable phenotype (Perez-de-Castro et al. 2012; Hu et al. 2018). In this way, it is possible to introgress even minor-effect QTLs efficiently, as there are no biased marker effects, unlike with MAS. Studies on other plant species have shown the higher prediction accuracy of GS in genetic gains and a significant decrease in the time taken for breeding (Hu et al. 2018). Although proposed two decades ago, the implementation of GS in crop breeding has just begun, mainly because of the advent of high-throughput and affordable genotyping methods that produce dense marker information such as genotyping-by-sequencing and automated phenotyping (Bhat et al. 2016). Given the importance of disease-resistance breeding in rice, the application of GS could be of tremendous benefit.

8.1.4 Using Machine Learning and Artificial Intelligence

The field of computational biology is advancing at an unprecedented rate with the arrival of machine learning (ML) and artificial intelligence (AI). In ML, the machine gains experience by identifying patterns in given datasets and using that experience to interpret the data in question. ML has applications in various aspects of plant sciences, including phenotyping and increasing the accuracy of sequence analysis pipelines, such as differentiating true SNPs from spurious SNPs (Hu et al. 2018). ML was successfully used to phenotype and categorize foliar stresses in soybean

with high accuracy (Ghosal et al. 2018). Various parameters such as yield, developmental stage, weed status, crop quality, water and soil management, and disease occurrence were successfully predicted using ML (Liakos et al. 2018). In rice, bakanae disease (caused by *Fusarium fujikuroi*) was predicted with an accuracy of 87.9% using support vector machine classifiers, a popular ML tool that is often used to overcome classification and regression problems (Chung et al. 2016). Although few in number, these studies have put forth the applicability of advanced computational strategies to improve agriculture.

9 Future Prospects and Conclusions

The rice crop plays an essential role in ensuring global food security and providing nutritional security for the rapidly growing world population. Increasing grain yield is a significant target for plant breeders apart from identifying resistance to/tolerance of biotic and abiotic stresses. Enhancing genetic gain is also a primary concern to meet the food demand of the ever-increasing world population, especially with global climate change. In recent years, the innovations in rice breeding programs and advanced genomics technologies such as next-generation sequencing and high-throughput genotyping have been fully exploited to understand trait interactions and select promising rice genotypes for use in breeding programs. The genetic improvements in yield component traits and increasing yield significantly under biotic and abiotic stresses have not been achieved to a great extent due to the complex nature of these stresses. The knowledge of integrated genomics and high-throughput phenomics technologies has laid the foundation to understand these complex traits and also associated molecular genetics and physiological mechanisms that can enable breeders to find better rice genotypes and to move forward as knowledge-based rice breeding is the most acceptable approach in developing climate-smart stress-tolerant and high-yielding rice genotypes. This approach has advanced at a fast pace with low-cost, efficiency, and high resolution of genetic mapping for QTLs and genes and also haplotype blocks to find allelic variations for the target trait of interest. The current advances in CRISPR/Cas9 genome-editing tools have led to significant targeted changes in specific trait-associated genes and changes in single base levels that promise to accelerate crop improvement. These genomic-assisted breeding tools are breeder-friendly, and smart decisions in breeding programs can enhance the efficiency of the selection of rice genotypes in a short period.

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