Chapter

Rice Blast Disease in India: Present Status and Future Challenges

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Abstract

Rice (Oryza sativa L.) is the staple food of the majority of Indians, and India is both the major producer and consumer of rice. Rice cultivation in India is confronted with diverse agro-climatic conditions, varying soil types, and several biotic and abiotic constraints. Among major fungal diseases of Rice in India, the blast caused by Magnaporthe oryzae is the most devastating disease, with the neck blast being the most destructive form. Most of the blast epidemic areas in India have been identified with a mixture of races blast fungus resulting in the resistance breakdown in a short period. At present, a more significant number of the rice varieties cultivated in India were bred by conventional breeding methods with blast resistance conferred by a single resistance gene. Therefore, the blast disease in India is predominantly addressed by the use of ecologically toxic fungicides. In line with the rest of the world, the Indian scientific community has proven its role by identifying several blast resistance genes and successfully pyramiding multiple blast resistance genes. Despite the wealth of information on resistance genes and the availability of biotechnology tools, not a great number of rice varieties in India harbor multiple resistance genes. In the recent past, a shift in the management of blast disease in India has been witnessed with a greater focus on basic research and modern breeding tools such as marker-assisted selection, marker-assisted backcross breeding, and gene pyramiding.

Keywords: Magnaporthe oryzae, blast, resistance breeding, Marker Assisted Selection (MAS), Pyramiding, disease management, Marker-assisted backcross breeding (MABB)

1. Introduction

As the theme “Rice is life” reflects, Rice (Oryza sativa L.) is the single most important staple food crop for more than one-third of the world’s population and more than half of the population India. Rice is grown in a wide range of
agro-ecological conditions in India. Rice provides 21% of global human per capita energy and 15% per capita protein [1]. Amongst the important rice-producing nations in the world, India ranks second in terms of area and production. Out of 782 million tons (mt) of global rice production from 167.1 million hectares (m ha), India produced 116.42 m t in 44.5 m ha (rainy season: 102.13 m t from 39.27 m ha) [2]. For food insecurity to recede, agricultural production on currently cultivated land will increase by 70% globally and 100% in the developing countries by 2050 [3]. Of the various biotic factors limiting rice production and productivity, diseases continue to be an enigmatic problem in several rice-growing ecosystems of the world’s tropical and temperate regions. The annual losses due to rice diseases are estimated to be 10–15% on an average basis worldwide. Rice blast fungus infects host plants at various crop growth stages, including leaf, stem, neck, collar, node, and root. The biggest challenge for rice breeders is the breakdown of resistance in existing rice varieties over the years. Therefore, breeding durable and broad-spectrum resistant cultivars is again a challenging task. The broad host range, continuous genetic variation, evolution, and host shifts are the main reasons behind the emergence of virulent pathotypes of \textit{Magnaporthe}, which make blast management a daunting task. Hence, the Rice-\textit{Magnaporthe} interaction pathosystem emerged as a model system to study host-pathogen interaction for several reasons, including the economic importance of blast disease in rice production and human diet.

2. Blast disease of rice and its economic importance

The blast disease affects almost all parts of the rice plant and occurs in different crop growth stages, starting from nursery to harvesting. The symptoms at different stages are called by different names, \textit{viz.}, nursery blast, leaf blast, node blast, neck blast, and panicle blast (Figure 1, panels a-e). The disease was first reported as “rice fever” in China by Soong Ying-shin in 1637, and later, it was reported from Japan by Imochi-byo during 1704. It is presently found in approximately 85 countries in the world and India. It was first recorded in 1913, and the first devastating epidemic was reported in 1919 in the Tanjore delta of erstwhile Madras state [5]. Later, the disease

\begin{center}
\textbf{Figure 1.}
(a) Blast disease symptoms at nursery stage (b) typical leaf blast symptoms under field condition (c) typical node blast symptoms (Photo Courtesy: http://www.knowledgebank.irri.org) (d) Neck blast infection leading to the choppiness and breakage of panicle (e) Panicle blast where symptoms appears on grains (f) life cycle of rice blast fungi [4].
\end{center}
has been reported to occur in different regions of India [6, 7]. Blast disease occurs in all rice ecosystems. However, it is more damaging in upland rice than in irrigated ecosystem of rice cultivation. It is the major contributor to the yield gap. It causes more losses, especially in the humid rice-growing areas of India, including the cool season crop in Karnataka, Andhra Pradesh, Tamil Nadu, and Kerala. With the introduction and spread of semi-dwarf high-yielding varieties in the 1960s, its incidence became almost insignificant, especially in plains of North India during the Kharif season. The relative losses from this disease vary in different production zones depending on the physical environment, crop management, and pathogen population dynamics. The upland rice, grown in about 6.3 M ha in Eastern India and hill rice, is more prone to blast disease, and in many cases, the disease is left uncontrolled due to non-remunerative management options. Severe epidemics of the blast have occurred between 1980 and 1987 in Himachal Pradesh, Andhra Pradesh, Tamil Nadu, and Haryana, resulting in huge financial losses. As per estimation, the extent of annual yield reduction caused by rice blast disease is sufficient to provide food to around 60 million people [8]. Among the different stages of the disease, drastic yield reductions are reported in neck and panicle blast, reducing the grain weight, the percentage of ripe spikelets, and the percentage of fully mature grains [9]. The infection of the panicle base (Neck blast) by the blast pathogen until 20 days after heading was found to cause more than 50 percent yield loss [10]. In India, yield losses due to blast could be as high as 50% when the disease attains an epidemic proportion [11]. During natural epidemics of blast disease in the wet season, disease incidence ranged from 14 to 27% (above the economic threshold), resulting in yield loss of about 27–35 percent [12].

3. Rice blast fungus: *Magnaporthe oryzae*

The fungus causes rice blast disease *Pyricularia oryzae* Cavara [synonym *P. grisea* Sacc, teleomorph *Magnaporthe oryzae* [(Hebert) Barr]. The genus *Magnaporthe*, which consists of five species (*M. grisea, M. oryzae, M. salvinii, M. poae,* and *M. rhizophila*), has shared morphological traits such as three-septate fusiform ascospores and black non-stromatic perithecia (ascocarp) with long hairy necks [13]. However, due to the limited host range of the individual isolates, all *Magnaporthe* sps were regarded as the *M. grisea* species complex (Mg complex) [14, 15]. Blast disease of rice and other gramineous species is caused by the members of the Mg complex [15]. Recently, based on phylogenetic analyses and mating tests, isolates from crabgrass were separated from the Mg complex and named *M. grisea*, and other isolates from grasses, including rice, were named as *M. oryzae* [16–19]. One hundred thirty-seven family members, including rice, are affected by *M. oryzae* where it causes blast disease [20, 21].

3.1 Disease cycle and epidemiology

The pathogen perpetuates as mycelium and conidia on diseased straw, seed, rice ratoons, volunteer rice plants, and weed hosts. The initiation of the primary infection process begins with the attachment of the conidium of *M. oryzae* to the leaf cuticle. Later stages of the infection process include the formation of appressorium, which assists in pathogen penetration, generation of turgor, formation of penetration peg, and finally penetration into host tissue (Figure 1, panel f) [4, 22, 23]. After penetration, hyphae grow through the plant tissue, resulting in the disease lesions and typical rice blast symptoms. Under congenial weather conditions (high relative humidity and low night temperature), the fungus produces an enormous number of conidia which brings the secondary spread and infection to other healthy plants nearby and spreads rapidly to adjacent fields by wind leading to field epidemic [4, 20, 24]. New blast
lesions appear within 4–5 days after landing spores at the optimum temperature on the leaf surface. New conidia are produced under warm and wet weather conditions on the disease lesion within few hours of lesion appearance. The sporulation continues for several days and provides the inoculum (secondary conidia) for secondary infection.

Although blast disease is distributed across all the parts of India, some parts of the country have been identified as hotspots of blast disease. Sub-Himalayan regions of Jammu and Kashmir, Himachal Pradesh, hill districts of Uttaranchal, and West Bengal are often associated with the northern part of India repeated epidemics of blast disease. In the eastern part of India, the blast is in its destructive form in upland rice-growing areas of Arunachal Pradesh, Manipur, Mizoram, Meghalaya, Assam, Chotanagpur belt, and Jaypore tract of Orissa. While blast is of much importance in the Konkan region of Maharashtra and Gujarat in the west, the disease is frequently reported from Andhra Pradesh, Telangana, Tamilnadu, and Coorg region of Karnataka in peninsular India. From several blast disease incidence reports and surveys, blast disease occurs in different agroclimatic conditions in the country. In North and North-Eastern India, the blast disease occurs in June to September in high rainfall areas with 20–24°C. In medium rainfall areas (1000 mm per annum) and temperatures ranging between 24 and 34°C in Western and Central India, blast occurrence is reported from August to October. However, the disease is associated with Andhra Pradesh, Telangana, Karnataka, Tamilnadu, and Kerala states in dry periods with cooler nights (18–22°C).

3.2 Pathogen variability

One of the main strengths of a blast pathogen in its interaction with the host and overpowering of the host defense system is the existence of several races. The Indian subcontinent is a center of origin and diversity for the *Magnaporthe* species complex. The pathogen is highly variable and evolves into new pathotypes within a short period. There is a nationally coordinated system (All India Coordinated Rice Improvement Programme) for regular monitoring of virulence pattern of blast disease using twenty-five rice cultivars that include international blast differentials, recombinant inbred lines, donors, and commercial cultivars. Cluster analysis of the *M. oryzae* reaction on these cultivars in different rice growing ecosystems revealed that the pathogen population could be clustered into four separate groups.

Further, there was a considerable variation within the groups, also suggesting the significant variability in the virulence of the *M. oryzae* population of India [25]. Efforts were made during the 1970s, where race profiling of Indian isolates of *M. oryzae* was carried out, and a new race group IJ was identified [6]. During the 1970s, race IC3 and ID 1 were predominantly distributed in India [6]. In another report, five pathogenic race groups, ID-1, ID-2, IB-4, IC-17, and IC-25, were identified from India and group ID-17 to be predominantly distributed in the Indian paddy ecosystem [26]. A total of 72 isolates of *M. oryzae* from rice in different districts of Karnataka were examined for identifying sexual mating alleles MAT1, MAT2, and understanding the genetic diversity based on the DNA fingerprint of pot2 an inverted repeat transposon. Among 72 isolates, 44 isolates belonged to MAT1 type (male fertile), and 28 isolates were of MAT2 (female fertile), and there were no hermaphrodite isolates[27]. Multi-marker systems including Simple Sequence Repeats (SSRs), repetitive DNA-based markers (Pot2), pathogenicity genes were used to study genetic variability of *Magnaporthe* species in rice and finger millet ecosystems from southern India. Data from multiple markers revealed high genetic diversity and clustering based on geographical location and host species [28]. Interestingly, major cluster I is dominated by Indian isolates whereas cluster II is dominated by isolates from different rice growing region of the world. Similarly, the blast isolates from the same geographical location did not belong to the same sub-cluster while genetically
similar isolates from different geographical location were grouped together. Same authors grouped most of Indian isolates in one group whereas blast isolates from other parts of the world in other group might be due to presence of distinct strain in India than rest of the world [29]. Despite few studies, the race distribution of the rice blast fungus is poorly understood in India. It demands enormous attention in the context of deploying suitable resistance genes to confront the pathogen.

4. Pathogenomics

4.1 Sequencing of rice blast fungus

The whole genome of *M. oryzae* strain 70–15 was the first to be sequenced among plant pathogenic fungi using the Sanger sequencing method [30]. Subsequently, several field isolates of the blast have been sequenced using next-generation sequencing (NGS). While Field isolates from Japan (Ina168 and P131) and China (Y34) [31, 32] were sequenced using the 454 sequencing platform, more recently, two field isolates, FJ81278 and HN19311 from China, have been sequenced using Illumina technology [33]. A highly diverse *Magnaporthe* species complex and multiple field isolates of *Magnaporthe* infecting different hosts such as rice (leaf and neck), finger millet (leaf and neck), foxtail millet (leaf), and buffelgrass (leaf) have recently been sequenced from India using Illumina sequencing technology [34, 35]. The majority of these isolates included virulent field isolates from southern India and a commonly used virulent reference strain B157 isolated from rice [36]. The genomes were extensively analyzed to compare the variability in gene content, repeat element distribution, candidate effectors, genes involved in carbohydrate metabolism, and single nucleotide polymorphisms (SNPs). This study has shed light on genomic factors contributing to genome variation, pathogenic strain evolution, and host-specificity. It was the first to compare blast fungal isolates from different hosts and different host tissues in India at the genome level.

Interestingly, whole-genome sequencing of multiple isolates has revealed large chunks of novel genomic regions and multiple novel genes. In another report, the whole-genome assembly of *M. oryzae* RMg-D1 yielded 34.82 Mb genome sequence by PacBio single-molecule and Illumina HiSeq2500 sequencing, which aids in better understanding the genetic determinants of host range, host jump, survival, pathogenicity, and virulence factors of *M. oryzae* [37]. The genomic variation was attributed to race evolution over a period of time by geographical separation, chromosomal variation, and variability in repetitive elements [30, 31, 33]. The availability of pathogen genomes will undoubtedly be helpful to breeders and researchers to understand *Magnaporthe* virulence spectrum and improve blast resistance in rice and other important food crops affected by blast disease.

4.2 Rice genome sequencing

Pathogen and host are the two faces of a coin in the context of host-pathogen interactions and disease management. Hence, characterizing the rice genotypes for novel resistant genes (R) should be done parallel with that of the pathogen as host and pathogen evolve simultaneously for their survival in nature. The discovery of novel `R` genes and understanding their mutation in evolving novel alleles/genes is an important step in resistance breeding. Allele discovery/mining could be made using high throughput technologies like whole genome sequencing using next-generation sequencing (NGS) technologies. Rice is a model cereal crop, and several rice cultivars have been sequenced at the genome level, with Nipponbare
as the first rice cultivar to be sequenced and published in 2002 [38]. Further, the indica cultivar 93-11 was also sequenced and published in the same year [39]. These initial efforts laid the foundation for the genomic era in rice. Subsequently, several whole-genome sequencing efforts of rice cultivars like IR-64 [40], Kasalath [41], and HR-12 [42] also added quantum of genomic information to the existing genomic resources. HR-12 genome was assembled using a combination of Illumina short reads and PacBio long reads. This was the first report in the world to sequence rice genome using third-generation sequencing technology. The power of long-read technologies helped in repeat resolution compared to second-generation technologies. Large-scale discovery of novel alleles by resequencing of 3000 rice germplasm accessions belonging to 89 countries contributed significantly to the rice genomic resources [43]. Exploiting natural variation existing among rice landraces is an ideal method to map R genes. Mapping of R genes based on avirulent (Avr) genes pattern in the rice-growing areas is the best strategy to mitigate *Magnaporthe* via exploiting host plant resistance. The product of the avirulence (Avr) gene of *Magnaporthe* can be detected by the corresponding resistance (R) gene of rice and activates immunity to rice mediated by the R gene. The high degree of variability of *M. oryzae* isolates in pathogenicity makes the control of rice blast difficult. That resistance of the R gene in rice has been lost ascribed to the instability of the Avr gene in *M. oryzae*. Further study on the variation of the Avr genes in *M. oryzae* field isolates may yield valuable information on the durable and effective deployment of R genes in rice production areas. *AvrPiz-t* and *Piz-t* are a pair of valuable genes in the Rice-*Magnaporthe* pathosystem. *AvrPiz-t* is detectable by *Piz-t* and determines the effectiveness of *Piz-t* [44]. Rice SNP-seek database developed based on 3000 rice genomes, possessing a large-scale single base level variation across three geographical rice ecotypes (*japonica*, *indica*, and *javanica*) been made available to the public [45]. These variants could be harnessed to study the genetic diversity and development of subspecies-specific rice cultivars. Also, rice breeders can focus on allele mining for corresponding R genes and pyramiding these genes in commonly grown cultivars in a given location to help develop resistant rice varieties.

5. Resistance genes and QTLs for rice blast disease

The resistance for blast disease is two types: i) qualitative or complete resistance governed by a major R gene, and ii) quantitative or partial resistance governed by many quantitative trait loci [46]. While qualitative resistance confers resistance against a specific race of blast pathogen, the quantitative resistance is non-race specific. To date, 109 major blast resistance genes have been identified in rice. Out of these, 25 R genes have been successfully cloned and characterized, with Pi9 being the first cloned R gene (Table 1). Japan and China have lead the race in identification of major R genes by identifying 34 and 27 blast resistance genes, respectively. Followed by these, a significant contribution has also been made by USA, France, Philippines and India. To date eight R genes have been mapped in India that include Pi10, Pi157f, Pi38, Pi42(t), Pikh (Pi54) [129], Pitp [117], Pi54rh [127], and Pi54of [128]. The details of the genes mapped in India and the rice varieties in which they were identified are provided in Table 1. Majority of the genes identified in India and rest of the world encode proteins with NBS-LRR (Nucleotide Binding Site and Leucine Rich Repeats) and Zinc finger domains that confer disease resistance. Among all the genes that were mapped in India, pi54 is of great importance as it a major blast resistance gene and provides durable resistance against Indian races of blast fungus. These qualitative and major R genes have been extensively used in blast resistance breeding programs worldwide (Table 2). For instance,
| Sl. No | Gene  | Chr. No | Position Location (bp) | Source | Country   | References |
|--------|-------|---------|-------------------------|--------|-----------|------------|
| 1      | Mpiz  | 11      | 4073204-1673079         | Zenith (I) | Japan |   |
| 2      | Pb1   | 11      | 2171457-2587368        | Medan (I) | Japan | [48, 49] |
| 3      | PBR   | 11      | -                       | Sr-No. 1 (I) | Japan |   |
| 4      | P17   | 4       | 2648854-2873448        | LAC23 (I) | Philippines |   |
| 5      | P1   | 11      | 14521809-18054-805     | Teng1 (I) | India |   |
| 6      | P11   | 8       | 698220-1524064         | Zhai-Ya-Quing8 (I) | China |   |
| 7      | P17   | 12      | 2171457-2587368        | LAC23 (I) | Philippines |   |
| 8      | P21   | 6       | 22250443-24995083      | Suweon365 (J) | Korea |   |
| 9      | P19   | 11      | 26796917-2873693       | O. miniatum (W), Kasalath (I), Maowang1 (I) | China | [55-57] |
| 10     | P10   | 2       | 1-6725831              | Aichi Asahi (J) | Japan |   |
| 11     | P12   | 12      | 882655-13417087        | Aichi Asahi (J) | Japan |   |
| 12     | P13   | 2       | 34360810-37725160      | IR64 (I) | France | [68] |
| 13     | P15   | 7       | 22250443-24995083      | Suweon365 (J) | Korea |   |
| 14     | P16   | 12      | 882655-13417087        | Aichi Asahi (J) | Japan |   |
| 15     | P18   | 11      | 26796917-2873693       | Owaraihame (I) | Japan | [67] |
| 16     | P20   | 4       | 497044-6024472         | Aichi Asahi (J) | Japan |   |
| 17     | P22   | 5       | 1075867-1975845        | Aichi Asahi (J) | Japan | [67] |
| 18     | P23   | 6       | 1075867-1975845        | Aichi Asahi (J) | Japan | [67] |
| 19     | P24   | 1       | 524254-556378          | Sowon365 (I) | Korea |   |
| 20     | P25   | 2       | 346048-10275410        | Gumi 2 (I) | Korea | [69] |
| 21     | P25*  | 2       | 346048-10275410        | Gumi 2 (I) | Korea | [69] |
| 22     | P26   | 2       | 346048-10275410        | Gumi 2 (I) | Korea | [69] |
| 23     | P25   | 2       | 346048-10275410        | Gumi 2 (I) | Korea | [69] |
| Sl. No | Gene | Chr. No. | Position Location (bp) | Source | Country | References |
|--------|------|----------|------------------------|--------|---------|------------|
| 24     | Pi26 | 6        | 8751256-11676579       | Gumei 2 (I) | China   | [70]       |
| 25     | Pi26(t) | 5      | 2069318-2760202        | Azucena (J) | France  | [68]       |
| 26     | Pi27 | 1        | 5556378-744329        | Q14 (I) | France  | [68]       |
| 27     | Pi27(t) | 6      | 6230045-6976491       | IR64 (I) | France  | [68]       |
| 28     | Pi28(t) | 10     | 19565132-22667948     | IR64 (I) | France  | [68]       |
| 29     | Pi29(t) | 8      | 9664057-16241105      | IR64 (I) | France  | [68]       |
| 30     | Pi3(t) | 6        | –                     | Pai-kan-tao (J) | Philippines | [71]       |
| 31     | Pi30(t) | 11     | 441392-657875         | IR64 (I) | France  | [68]       |
| 32     | Pi31(t) | 12     | 7731471-11915469      | IR64 (I) | France  | [68]       |
| 33     | Pi32(t) | 12     | 13103039-18867450     | IR64 (I) | France  | [68]       |
| 34     | Pi33 | 8        | 5915858-6152906       | IR64 (I) | France  | [68]       |
| 35     | Pi34 | 11       | 19423000-19490000     | Chubu32 (J) | Japan   | [72]       |
| 36     | Pi35(t)* | 1      | –                     | Hokkai 188 (J) | Japan | [73]       |
| 37     | Pi36* | 8        | 2870061-2884353       | Q61 (I) | China   | [74]       |
| 38     | Pi37* | 1        | 33110281-33489931     | St-No 1 (J) | China | [75, 76]   |
| 39     | Pi38 | 11       | 19137900-21979485     | Tudukan (I) | India | [77]       |
| 40     | Pi39(t) | 4, 12   | –                     | Chubu111 (J) Q15(I) | China | [78]       |
| 41     | Pi40(t) | 6      | 16274830-17531111     | O. australiensis (W) | Philippines | [79]       |
| 42     | Pi41 | 12       | 33110281-34005652     | 93-11 (I) | China   | [80]       |
| 43     | Pi42(t) | 12     | 19565132-22667948     | DHR9 (I) | India   | [81]       |
| 44     | Pi44 | 11       | 20549800-26004823     | Moroberekan (J) | USA | [82]       |
| 45     | Pi47 | 11       | –                     | Xiangzi 3150 (I) | China | [83]       |
| 46     | Pi48 | 12       | –                     | Xiangzi 3150 (I) | China | [83]       |
| Sl. No | Gene   | Chr. No | Position Location (bp)   | Source                  | Country   | References |
|--------|--------|---------|--------------------------|-------------------------|-----------|------------|
| 47     | Pi5(t) | 9       | –                        | Moroberekan (J)         | Philippines | [84]       |
| 48     | Pi6(t) | 12      | 4053339-18867450         | Apura (I)               | USA       | [85]       |
| 49     | Pi62(t) | 12     | 2426648-18050026         | Yashiro-mochi (J)       | Japan     | [68]       |
| 50     | Pi67   | –       | –                        | Tsuyuake                | Philippines | [68]       |
| 51     | Pi8    | 6       | 6230045-8751256          | Kasalath (I)            | Japan     | [57]       |
| 52     | P9*    | 6       | 10386510-10389466        | O. minuta (W)           | China     | [86]       |
| 53     | Piα*   | 11      | 4073024-8078510          | Aichi Ashi (J)          | Japan     | [87, 88]   |
| 54     | Piβ*   | 2       | 35107768-35112900        | Tohoku IL9 (J)          | Japan     | [89, 90]   |
| 55     | Piβ2   | 11      | 26796917-28376959        | Lemont (J)              | Philippines | [91]       |
| 56     | PiCO39(t) | 11 | 6304007-6888870        | CO39 (1)                | USA       | [92]       |
| 57     | Pi(t)1 | 2       | 20143072-22595831        | Digu (I)                | China     | [93]       |
| 58     | Piδ2*  | 6       | 17159337-17163868        | Digu (I)                | China     | [94]       |
| 59     | Pf     | 11      | 24695583-28462103        | Chugoku 31-1 (J)        | Japan     | [95]       |
| 60     | Pig(t) | 2       | 34346727-35135783        | Guangchangzhan (I)      | China     | [96]       |
| 61     | PiGD1  | 8       | –                        | Sanhuangzhan 2 (I)      | China     | [97]       |
| 62     | PiGD-2 | 10      | –                        | Sanhuangzhan 2 (I)      | China     | [98]       |
| 63     | PiGD3  | 12      | –                        | Sanhuangzhan 2 (I)      | China     | [98]       |
| 64     | Pigm(t)* | 6 | 10367751-10421545      | Gumei4 (1)              | China     | [99]       |
| 65     | Pi1    | 9       | 2291804-28431560         | Ishikari Shiroke (J) Fujisaka 5 (J) | Japan | [95, 100] |
| 66     | PiII   | 6       | 2291804-28431560         | Fujisaka 5 (J)          | Japan     | [56, 57]   |
| 67     | Pi2    | 9       | 1022662-7222779          | Ishikari Shiroke (J)    | Japan     | [101]      |
| 68     | Piis1  | 11      | 2840211-19029573         | Imochi Shiraz (J)       | Japan     | [102]      |
| 69     | Piis2  | –       | –                        | Imochi Shiraz (J)       | Japan     | [102]      |
| Sl. No | Gene   | Chr. No | Position Location (bp) | Source      | Country  | References |
|--------|--------|---------|-------------------------|-------------|----------|------------|
| 70     | Piis3  | –       | –                       | Imochi Shiraz (J) | –        | [102]      |
| 71     | Pik*   | 11      | 77314916-27532928       | Kusabue (I)  | China    | [103, 104] |
| 72     | Pikg   | 11      | 77314916-27532928       | GA20 (J)     | Japan    | [56]       |
| 73     | Pikh (P54)* | 11    | 24761902-24762922       | Tetep (I)    | India    | [105]      |
| 74     | Pikem* | 11      | 77314916-27532928       | Tsuyuake (J) | China    | [106, 107] |
| 75     | Pikp*  | 11      | 77314916-27532928       | HR22 (I)     | China    | [108]      |
| 76     | Piks   | 11      | 77314916-27532928       | Shin 2 (J)   | Japan    | [109]      |
| 77     | Pikar1 | 4       | 24611955-33558479       | Kuroka (J)   | Japan    | [102]      |
| 78     | Pikar2 | 11      | 2840211-18372685        | Kuroka (J)   | Japan    | [110]      |
| 79     | Pilm2  | 11      | 13635033-28377565       | Lemont (J)   | USA      | [111]      |
| 80     | Pir2-3(t) | 2     | –                       | IR64 (I)     | Indonesia| [112]      |
| 81     | Pirf2-1(t) | 2      | –                       | O. rufipogon (W) | Indonesia| [112]      |
| 82     | Pis   | 11      | 57406-42-16730739       | Sensho (J)   | Japan    | [102]      |
| 83     | Pis2  | –       | –                       | Sensho (J)   | Japan    | [102]      |
| 84     | Pis3  | –       | –                       | Sensho (J)   | Japan    | [102]      |
| 85     | Pik*  | 1       | 33381385-35283446       | Shin 2 (J)   | Japan    | [65]       |
| 86     | Pir-h | 11      | 33381385-35283446       | Nipponbure (J) | Japan    | [65]       |
| 87     | Pit*  | 1       | 2270216-3043185         | Tjahaja (I), K59 (J) | Japan    | [108, 113]|
| 88     | Pita* | 12      | 10603772-10609330       | Tadukan (I), | USA      | [114]      |
| 89     | Pita2 | 12      | 10078620-13211331       | Shimokita (J) | Japan    | [115, 116]|
| 90     | Pitp(t) | 1      | 2513540-28667306        | Tetep (I)    | India    | [117]      |
| 91     | Pitq1 | 6       | 28599181-30327854       | Teqing (I)   | USA      | [111]      |
| 92     | Pitq2 | 2       | –                       | Teqing (I)   | USA      | [91]       |
| Sl. No | Gene | Chr. No. | Position Location (bp) | Source | Country | References |
|-------|------|----------|------------------------|--------|---------|------------|
| 93    | Pitq3 | 3        | –                      | Teqing (I) | USA | [91]        |
| 94    | Pitq4 | 4        | –                      | Teqing (I) | USA | [91]        |
| 95    | Pi-tq5 | 2        | 34614264-35662091      | Teqing (I) | USA | [111]       |
| 96    | Pitq6 | 12       | 5758663-7731471        | Teqing (I) | USA | [111]       |
| 97    | Pi1(t) | 2        | –                      | Yanxian No 1 (I) | China | [118]       |
| 98    | Pi2(t) | 2        | –                      | Yanxian No1 (I) | China | [118]       |
| 99    | Piz   | 6        | 10155975-10517612      | Zenith (J), Tadukan (I), Toride 1 (J), Fukunishiki (J) | Japan | [119]       |
| 100   | Pizh  | 8        | 4372113-21012219       | Zhai-Ya-Qing8 (I) | China | [51]        |
| 101   | Pi2*  | 6        | 10076481-10204423      | Jefferson | China | [120]       |
| 102   | Pil3*  | 6        | 13055253-13058027      | 93-11(I), Nipponbare (J) | China | [121]       |
| 103   | Pi4   | 12       | –                      | Pai-kan-tao | Philippines | [122]       |
| 104   | Piz*  | 6        | –                      | Wild rice | Japan | [123]       |
| 105   | Piz*  | 9        | S04G03-C1454 (Map position) | RIL260 | Korea | [124]       |
| 106   | Pki*  | 11       | Os11g0597700           | Kanto 209, Koshihikari Achi | Japan | [125]       |
| 107   | NLS1*  | 11       | AC134922 (Accession number) | Wild rice | China | [126]       |
| 108   | Pi54rh*  | 11       | –                      | Oryza rhizomatis (Wild rice) | India | [127]       |
| 109   | Pi54of*  | 11       | HES89448 (Accession number) | Oryza officinalis | India | [128]       |

*Cloned and characterized blast resistance genes in rice.

Note: Chr No. = Chromosome number; I = Indica; J = Japonica; − = Not Known.

Table 1.

Blast resistance genes identified so far in different rice cultivars.
| Sl. No | Gene/ QTL | Marker type | Technique | Trait | Application | Reference |
|-------|-----------|-------------|-----------|-------|-------------|-----------|
| 1.    | Pi1, Piz-5, Pita | RFLP | MAS | Blast resistance | Pyramiding of three near isogenic lines (C101LAC, C101A51 and C101PKT) for blast resistance into a single cultivar CO39, each carrying the major genes Pi1, Piz-5 and Pita, respectively | [130] |
| 2.    | Pi1      | SSR, ISSR  | MABB      | Blast resistance | Applied for backcross breeding of variety | [131] |
| 3.    | Xa21, Piz | SSR        | MAS       | Bacterial blight and blast resistance | Pyramiding of target traits | [132] |
| 4.    | Pid1, Pib, Pita, Pi2 | SSR | MAS | Blast resistance | Pid1, Pib and Pita genes were introduced into G46B, while Pi2 was introduced into Zhenshan 97B | [93] |
| 5.    | Piz      | SSR        | MAS       | Blast resistance | Successfully used for selection of blast resistance in a wide array of rice germplasm | [133] |
| 6.    | Xa13, Xa21, P54, qSBR11 | SSR | MAS | Blast, Bacterial blight and Sheath blight resistance | Transfer genes conferring the resistances toward three different diseases in rice | [134] |
| 7.    | Pita     | Gene specific | MAS | Blast resistance | Existence of Pita gene in 141 rice germplasms was determined, but the results were more articulated when Pita gene was introduced through advanced breeding lines | [135] |
| 8.    | Pi1, Pi2, Pi33 | SSR | MABB | Blast resistance | Introgessed into Jin23B | [136] |
| 9.    | Pi1, Pi2, Xa23 | SSR | MABB | Blast resistance and bacterial blight | Successfully applied for breeding variety Rongfeng B | [137] |
| Sl. No | Gene/QTL | Marker type | Technique | Trait | Application | Reference |
|-------|-----------|-------------|-----------|-------|-------------|-----------|
| 10.   | *Piz-5*, *Pi54* | SSR         | MABB      | Blast resistance | Transfer blast resistance genes from donor lines (C101A51 and Tetep) into PRR78 to develop Pusa 1602 (PRR78 + *Piz-5*) and Pusa 1603 (PRR78 + *Pi54*), respectively | [138] |
| 11.   | *Pi9*     | Gene specific | MABB     | Blast resistance | Applied to introgress the cultivar Luhui 17 | [139] |
| 12.   | *Pi1*, *Pie* | SSR         | MABB      | Blast resistance | Pyramiding of *PiII* and *Pie-5* genes into introduced PRR78 | [140] |
| 13.   | *Py39*    | InDel       | MABB      | Blast resistance | Introgressed into Chinese cultivar Q15 | [141] |
| 14.   | *P2*, *Xa21*, *Xa33* | SSR         | MABB      | Blast and Bacterial blight resistance | Introgressed into RPHR-1005 | [142] |
| 15.   | *Pi40*    | SSR         | MABB      | Blast resistance | Introgressed into elite cultivars Turkish, Osmancik-97 and Hallibey | [143] |
| 16.   | *Pi1*, *Pi2* | SSR         | MABB      | Blast resistance | Introgressed into Intan variety and BPT5204 | [144] |
| 17.   | *Pi46*, *Pita* | SSR         | MABB      | Blast resistance | Introgressed into Hang hui 179 (HH179) | [145] |
| 18.   | *P2*, *P9* | SNP         | MABB      | Blast resistance | Introgressed into R179 | [146] |
| 19.   | *Piz*, *P2*, *Pigm*, *Pi40*, *Pi9*, *Piz* | SSR         | MAS       | Blast resistance | Introgressed into Yangdao 6 | [147] |
| 20.   | *Pi1*, *Pi2*, *P33* | SSR         | MAS       | Blast resistance | Introgressed into Russian rice varieties | [148] |
| 21.   | *P9*, *Piz*, *P54* | SNP         | MABB      | Blast resistance | Introgressed into japonica rice 07GY31 | [145] |
| 22.   | *Pi-b*, *Pik-h* | SSR         | MAS       | Blast resistance | Introgressed into MR219 | [149] |
| 23.   | *Pi-ar*   | RAPD        | MAS       | Blast resistance | Double haploid technique was used for the introgression of *Pi-ar* gene | [150] |
| Sl. No | Gene/ QTL | Marker type | Technique | Trait | Application | Reference |
|-------|-----------|-------------|-----------|-------|-------------|-----------|
| 24.   | Pi2, Pi54, xa13 and Xa21 | SSR | MABB | Blast and Bacterial blight resistance | Improvement of Basmati rice varieties | [151] |
| 25.   | Piz-5 and PS54 | SSR | MABB | Blast resistance | Incorporation of blast resistance into "PRR78", an elite Basmati rice restorer line | [152] |
| 26.   | Pi1, Pi2 and Pi33 | SSR | MABB | Blast resistance | Improve blast resistance in Indian rice (*Oryza sativa*) variety ADT43 | [153] |
| 27.   | Pi-2 and Pi-54 | SSR | MABB | Blast resistance | Introgression of blast resistance genes into the genetic background of elite, bacterial blight resistant indica rice variety, Improved Samba Mahsuri | [154] |
| 28.   | Xa21, xa13 and PS54 | Gene specific | MABB | Blast and Bacterial blight resistance | Marker-Assisted Pyramiding of Genes Conferring Resistance Against Bacterial Blight and Blast Diseases into Indian Rice Variety MTU1010 | [155] |
| 29.   | Xa 5 and 4 blast QTLs | SSR | MAS | Blast and Bacterial blight resistance | Pyramiding of blast and bacterial leaf blight resistance genes into rice cultivar RD6 | [156] |
| 30.   | PS54 and Pi1 | SSR | MAS | Blast resistance | Introgression of blast resistance genes into cold tolerant variety Tellahamsa | [157] |
| 31.   | Pi46 and Pita | SSR | MABB | Blast resistance | Blast resistance genes were introgressed into an elite restorer line Hang-Hui-179 (HH179) | [145] |
| 32.   | Pi2 and Xa23 | SSR | MAS | Blast and Bacterial blight resistance | Pyramiding of broad-spectrum disease resistance genes into GZ63-4S elite thermo-sensitive genic male-sterile line in rice | [158] |
the improved rice lines carrying Pi9 and Pi2 were highly resistant to 43 isolates collected from 13 countries and 455 isolates collected from different parts of the Philippines, and 792 isolates from several regions of China, respectively [86, 164]. Because of their high importance, there are continuing efforts to identify additional major blast resistance genes, especially in wild rice species, and transfer them into elite varieties. For example, Pi9 present in indica rice line 75-1-127 [131] was introgressed from O. minuta [55]. Amongst the molecularly characterized major leaf-blast R genes, 22 were; namely, Pi37, Pit, Pi-sh, Pi64, Pi-b, Pi63, Pi9, Pi-2, Piz-t, Pid3, Pigm, Pi25, Pi36, Pi5, Pi-54, Pik-m, Pik, Pik-p, Pik-e, Pi-a, Pi1, and Pita, belong to the largest class of plant R genes that encode proteins with the nucleotide-binding site (NBS). Leucine-rich repeat (LRR) domains whereas one, Pid2, encodes serine–threonine–kinase membrane-spanning protein [165]. Rice blast resistance gene, Pi54 provides broad-spectrum resistance against different strains of M. oryzae. Understanding the cellular localization of Pi54 protein is an essential step towards deciphering its interaction with the cognate Avr-gene. A study was
conducted to investigate the subcellular localization of Pi54 with Green Fluorescent Protein (GFP) as a molecular tag. This is the first detailed report, which emphasizes the cellular and subcellular distribution of the broad-spectrum blast resistance gene Pi54 in rice and the impact of its constitutive expression towards resistance against other fungal and bacterial pathogens of rice [166]. These R genes function in a gene-for-gene fashion, meaning that for every R gene in the host, there is an Avr gene in the pathogen. Therefore, the pathogen can easily break down the host resistance by modifying or deleting its corresponding Avr gene and rendering the resistant variety susceptible after a few years [167]. The quantitative or partial resistance is more suited to low-risk areas as it cannot suppress *M. oryzae* when the environments are conducive for its growth. The quantitative trait loci (QTL), which in the context of disease resistance also referred to as quantitative resistance loci (QRLs) [168], are thought to play an important role in sustainable food production in the years ahead by manifesting durable resistance against many races of the blast fungus [169]. Chromosomal locations of leaf-blast R genes and quantitative trait loci (QTLs) for neck-blast resistance in rice are illustrated in Figure 2. Recently, QTL analysis of introgression line (INGR15002) derived from *O. glumaepatula* led to the identification of two major QTL - qBL3 contributing about 34% and 32%
phenotypic variance towards leaf and neck blast resistance, respectively, and qBL7 contributing about 25% of phenotypic variance for leaf blast [170]. Hence, there are several and continuous attempts to identify QTLs for blast resistance in rice. However, the results of a meta-analysis of QTLs have indicated that the use of QTLs does not offer durable and broad-spectrum resistance compared to that offered by the major genes [171]. Hence, care has to be taken in future breeding programs to effectively combine the major genes and QTLs to achieve durable and long-lasting resistance against several races of the blast fungus.

6. Defence mechanism of rice against blast infection

Rice employs a twolayered innate immune system to defend itself against blast invasion. PAMP-triggered immunity (PTI) forms the first layer of immunity, and is boosted after PAMP recognition by membrane-associated PRR on the host cell membrane [172]. Chitin well-known type of PAMP capable of activating plant immune responses. Chitin from \textit{M. oryzae} is recognized by rice transmembrane LysM receptor-like proteins (LysM-RLPs), including two lysin motif-containing proteins, OsLYP4 and OsLYP6, and a chitin elicitor binding protein (CEBiP). When defending against \textit{M. oryzae}, rice forms a receptor complex called LysM-RLPsOsCERK1. Two rice Receptor like kinases (RLKs), Flagellin Sensing 2 (OsFLS2) and BRI1-Associated receptor Kinase 1 (OsBAK1), are also involved in PTI. To achieve successful infection, virulent \textit{M. oryzae} isolates have evolved a strategy to secrete effectors into the rice cell for subverting PTI, leading to effector-triggered susceptibility (ETS). To combat a blast fungus capable of subverting PTI, rice deploys nucleotide-binding site leucine-rich repeat (NLR) proteins to recognize the effectors named avirulence (AVR) proteins. Several AVR proteins have been cloned, including AVR-Pita, AVR-Pi9, and Avr-Pizt [173, 174]. Recognition of AVR by NLR promotes strong immune responses referred to as effector-triggered immunity (ETI), which arms rice with a second layer of protection in case of disabled PTI [172]. Defence regulators (DR) genes can activate various signaling pathways, such as MAPK cascades and the ubiquitination-mediated pathway, as well as hormonal signaling (Figure 3). Upon activation by extracellular stimuli, MAPKs transmit signals from the cell membrane to the nucleus, acting in defense against \textit{M. oryzae} [172]. Transcription factors (TFs) are also involved in defense against infection by \textit{M. oryzae}. of particular interest are broad-spectrum resistance Digu 1 (bsr-d1) and Ideal Plant Architecture 1 (IPA1). Lesion-mimic mutant (LMM) genes are the main DR genes capable of activating immune responses such as ROS bursts. Lesionmimic mutants, including spl30-1, spl33, spl35, lmm24 and spl-D usually show increased disease resistance [172]. Several other DR genes can confer similar blast resistance by initiating ROS bursts. For example, SPL11 cell-Death Suppressor 2 (SDS2) is a ubiquitination substrate of SPL11 (an E3 ubiquitin ligase comprising an armadillo repeat domain and a U-box domain). SDS2 interacts with OsRLCK118/176 and phosphorylates OsRbohB, and then induces a ROS burst, resulting in increased resistance to \textit{M. oryzae} [172]. Hormones are another class of regulators involved in rice blast defense response. Suppressor of Salicylic acid Insensitivity-2 (OsSSI2), OsSec3a (a principal subunit of the exocyst complex in rice), OsAAA-ATPase 1 all mediate resistance by modulating salicylic acid (SA) signaling [175]. JA-resistant 1 (OsJAR1) and JAresponsive MYB (OsJAMyb) are associated with jasmonic acid (JA) signaling, and determine rice blast disease resistance. In the early stage of infection by pathogens, rice accumulates antimicrobial compounds as a defense response. For example, cyanide contributes to rice resistance by restraining fungal growth [176]. Bayogenin 3-O-cellobioside confers cultivar-nonspecific defense against the rice blast fungus [177]. Diterpenoids are a major group of antimicrobial phytoalexins in rice, and their
role in rice disease resistance has been indicated by functional analysis of a diterpenoid gene cluster (DGC7) located on rice chromosome 7 [178]. Excessive or deficient supply of nutrients, such as nitrogen, phosphate, potassium, and silica, affects stress response and can potentially influence rice disease resistance. For instance, nitrogen partially breaks down rice blast resistance triggered by the Pi1 gene. Potassium is also associated with rice blast resistance. For example, *M. oryzae* can disrupt rice immune response by regulating host K$^+$ channels. Silicon nutrition can mitigate various biotic stresses [172]. On one hand, silicon acts as a physical barrier against plant disease. On the other hand, silicon boosts the plant’s defense by functioning as a biological inducer. Silicon-induced defense response and cell silicification of leaves both contribute to rice blast disease resistance. In *M. oryzae*-infected rice plants, silicon-enhanced blast disease resistance is also associated with an increase in photochemical efficiency and adjustment of mineral nutrient absorption.

7. Strategies for breeding blast resistance in Rice

Although few cultural practices such as nutrient and water management, planting time, spacing, and application of fungicides are employed in managing blast disease, it has not been possible for the farming community to effectively and efficiently offset the blast disease [179]. This is mainly because of the complex etiology of the pathogen *M. oryzae* that includes infection of almost all parts of the rice plant, at all stages starting from seedling stage to maturity. Hence, breeding
for durable resistance and resistant cultivars has been a proven ecologically viable and crucial option for addressing the infection by rice blast fungus [155, 180, 181]. Breeding for blast resistance in rice can be broadly categorized into four classes, including conventional breeding methods, marker-dependent breeding methods, breeding approaches requiring genetic transformation, and genome editing.

7.1 Conventional breeding approaches

The conventional breeding approaches, including the pedigree method, backcrossing, recurrent selection, and mutation breeding, have been widely used in developing blast-resistant varieties in rice [182]. The pedigree method of breeding is the most commonly used breeding method for improving pest and disease resistance in rice. The pedigree method is the quick method employed to develop the resistance for one or more pests or diseases governed by major R genes. Backcross breeding improves an agronomically superior and high-yielding elite variety for resistance against insect pests and disease-causing pathogens. The major advantage of backcross breeding is avoiding the undesired genes from the donor parent due to linkage drag [183]. Backcross breeding has been used in South and Southeast Asia to improve blast resistance of several rice varieties including, KDML 105, Basmati, and Manawthukha [184]. With the advantages of shorter breeding cycles, control of genetic gains, and developing a broad range of genetic diversity in breeding lines, the recurrent selection breeding method is another choice of conventional breeding method to improve disease resistance in rice [185]. Mutation breeding is the method of choice when all the alleles available in the germplasm are exhausted, and there is a need to develop novel alleles. Mutation breeding has been effectively used to complement the other conventional methods of breeding. Although there are no breakthroughs achieved using mutation breeding, several examples of the use of this method exist that include the development of blast-resistant varieties RD6, KDML 105, Ratna, and R917 [186, 187]. However, the major limitation of the mutation breeding is the low efficiency, generation of recessive alleles, tracking of the mutated gene, and exposure of the personnel for mutagenic agents.

Further, associated markers have been effectively used to tag the mutated gene and follow them up in the subsequent generations [188]. The blast resistance genes that have been deployed in different rice varieties to address the incidence of the blast pathogen by using the above-mentioned conventional breeding methods include Pib, Pita, Pia, Pi1, Pikh, Pi2(t), and Pi4(t) [130]. Despite several rice varieties with high yield and grain quality in the previous few decades, the conventional breeding methods suffer several limitations, including high cost, labor-intensive, more time consuming, less reliability, difficulties in the appropriate genotypic selection, and linkage drag. Therefore, these limitations have necessitated the development of modern molecular breeding methods, which have overcome the limitations of conventional breeding methods.

7.2 Marker-based breeding methods

The main problem of traditional breeding methods is the selection of a genotype based on the phenotype. For instance, in disease resistance breeding, the resistant genotype is selected on their manifestation of resistance to the disease. However, a particular genotype without any R gene may be selected as resistant in the absence of a minimum level of disease pressure. Therefore, molecular markers associated with specific R genes have been widely employed to make the selection procedure more reliable, effective, and less time-consuming. Modern sequencing technologies have led to the identification of a large number of different DNA markers such as
simple sequence repeats (SSRs), single-nucleotide polymorphisms (SNPs), small insertions/deletions (InDels), amplified fragment length polymorphisms (AFLPs), random amplified polymorphic DNAs (RAPDs), restriction fragment length polymorphisms (RFLPs) associated with blast resistance genes that are effectively used in the selection of a genotype during handling of segregating generations [163, 179].

A panel of 80 released varieties from National Rice Research Institute, Cuttack, India, was genotyped with 36 molecular markers that were linked to 36 different blast resistance genes, to investigate the varietal genetic diversity and molecular marker-trait association with blast resistance. The cluster analysis and population structure categorized the 80 National Rice Research Institute released varieties (NRVs) into three major genetic groups. The principal co-ordinate analysis displays the distribution of resistant and moderately resistant NRVs into different groups. Analysis of molecular variance result demonstrated maximum (97%) diversity within populations and minimum (3%) diversity between populations. Among tested markers, two markers (RM7364 and pi21_79-3) corresponding to the blast resistance genes (Pi56(t) and pi21) were significantly associated and explained a phenotypic variance of 4.9 to 5.1% with the blast resistance [189]. In another research article, molecular analysis of variance of landraces originated from nine diverse rice ecologies of India showed maximum (93%) diversity within the population and least (7%) between populations. Five markers like K3957, Pikh, Pi2–i, RM212 and RM302 were strongly associated with blast disease with the phenotypic variance of 1.4% to 7.6% [190].

7.2.1 Marker Assisted Selection (MAS)

In MAS, the resistant phenotype of a variety is selected based on the presence of an R gene linked or R gene-based molecular marker. This selection method is more dependable and time-saving, does not require proper disease favoring environmental conditions, and selects the resistant genotypes even in the absence of the pathogen or disease. Hence, many present-day breeders are resorting to MAS in the developing blast-resistant varieties [163, 179, 191]. A set of well-characterized PCR-based markers such as SSR markers linked to blast R genes have been established currently used in the MAS programs worldwide. Similar to the rice breeders in the rest of the world, Indian rice breeders working on the improvement of blast resistance are not left behind in the use of MAS. The pioneering work of MAS in rice blast improvement began with Hittalmani et al. in 2000 [192]. Since then, several rice breeders in India have efficiently used MAS to incorporate different blast resistance genes, resulting in blast-resistant varieties. In China, rice lines were recently bred for blast resistance with four broad-spectrum resistance genes viz., *Pi9, Pi47, Pi48*, and *Pi49* [193]. A list of blast resistance breeding programs in rice using MAS in India is furnished in Table 2.

7.2.2 Marker-Assisted Backcross Breeding (MABB)

Like MAS, the MABB is also dependent upon DNA markers such as SSRs or SNPs. However, the main difference between the MAS and MABB lies in the type of the parent variety in which the improvement is sought and in recurrent parent genome recovery. While the MAS is used to introduce the blast resistance gene into any genotype, the MABB is employed to improve blast resistance in otherwise high-yielding elite varieties or genotypes. Therefore, the end product of the MABB is the same as the original rice variety except with improved blast resistance. Further, the ill effects of the unwanted genes from the donor are avoided by using a set of polymorphic markers for the recovery of the recurrent parent genome. Hence, MABB involves two stages of selection: foreground selection using markers linked
to blast resistance genes and background selection using polymorphic markers spread randomly throughout the rice genome. It is reported that short-grained landrace *Mushk Budji* was crossed to a triple-gene donor line, DHMAS 70Q 164-1b, and followed through marker-assisted foreground and background selection in first and second backcross generations that helped to incorporate blast resistance genes *Pi54, Pi1* and *Pita*. Marker-assisted background selection was carried out using 78 SSR and STS markers [194]. Several elite varieties such as MTU1010, IR-64, and Swarna have been improved for their blast resistance in India. A list of all other rice varieties improved for blast resistance in India following MABB is listed in Table 2.

### 7.2.3 Gene pyramiding

A major R gene confers the durable resistance to blast pathogen *M. oryzae* in rice. There are several major genes identified to govern the blast resistance in rice which were discussed earlier. Despite, presence of an R gene, a resistant rice variety becomes susceptible mainly due to the breakdown of the resistance by the evolution of new races of the blast pathogen. The evolution and breakdown of resistance are facilitated by the much longer life cycle of the crop than the quicker life cycle of the pathogen. Further, most of the paddy-growing regions of India are characterized by the presence of a mixture of races of *M. oryzae*. Because of these reasons, the mere presence of a single R gene is not enough for durable blast resistance over the long run. Hence, the deployment of more than one R gene into a single genotype, called gene pyramiding, is most essential. Therefore, gene pyramiding can be described as adding more than one desired gene into a single variety or cultivar. For example, in a recent study, gene pyramided lines were evaluated for key agro-morphological traits, quality, and resistance against blast at different hotspot locations. Two ICF3 genes, pyramided lines viz., TH-625-159, and TH-625-491 possessing *Pi54* and *Pi1* genes, exhibited a high level of resistance to blast [195]. Often gene pyramiding, ably supported by MAS, involves assembling more than one gene for different insect pests or diseases or a combination of both. This helps to achieve multiple goals in a single effort and reduces the duration of the crop improvement programs. Gene pyramiding has been successfully used for accumulating different blast resistance genes such as *Pi1, Pi2, and Pi33* [136], *Pib and Pita* [196], *Pish and Pib* [197], *Piz5 and Pi54* [138], *Piz and Pi5* [198] and *Pi21, Pi34 and Pi35* [199].

### 7.3 Genome editing approaches

During evolution, plants have acquired the ability to recognize the invading pathogen and fight against it. In general, the surface proteins of the plants are called pattern recognition receptors to recognize the pathogen-associated molecular patterns (PAMPs) and trigger an array of defense reactions. While this way of recognizing the pathogens is the first step in the plant defense system, several downstream proteins and plant hormones also play an important role in mediating the plant’s fight against the pathogen. Like any gene regulatory system, these mediators of plant defense system may impact the defense system. Recently, the availability of sequence-specific nucleases (SSNs) based genome editing tools, including zinc finger nucleases (ZFNs), transcription activator-like effector nucleases (TALENs), and clustered regularly interspaced short palindromic repeats (CRISPR)/CRISPR-associated (Cas) 9 (CRISPR/Cas9) have made the possibilities of genome editing to regulate gene expression without any genetic modification. Hence, these SSNs-based genome editing technologies may be used in the years ahead to alter the expression of the genes involved in the plant immune system and achieve resistance against the invading pathogen. In a recent study involving...
CRISPR/Cas9, resistance against *M. oryzae* was achieved by knocking out the expression of OsERF922, a plant ethylene-responsive factors (ERF) gene and a key negative regulator of plant immunity [200]. Similarly, *OsSEC3A* gene has been disrupted using CRISPR/Cas9 SSN to explore its role in plant immunity [201]. Proline-rich motif of *Pi21* was edited to induce resistance against *M. oryzae* [202]. Invention of CRISPR/Cas9 technology in Indian scenario to edit genes mapped or identified against *M. oryzae* is in infancy stage. However, couple of researchers have begun to edit negative regulators of blast resistance genes and other defense related genes identified through RNA sequencing [203]. Rice and other crops genome editing using TALENs produced disease resistance against diverse pathogens [204, 205]. CRISPR-Cas9 is generally limited to perform genome editing at sites with canonical NGG PAMs. Much effort has focused on overcoming this restriction. Numerous Cas9 orthologs have been developed with altered PAM specificities, such as *Staphylococcus aureus* Cas9 (SaCas9) and Cas9-VQR (D1135V/R1335Q/T1337R). The CRISPR-SaCas9 toolset was recently re-optimized by introducing three key mutations, and its activity was analyzed in rice. The newly optimized system performed genome editing with a mutagenesis efficiency of up to 77%. Other versions of Cas9 have also been tested in rice, including expanded PAM SpCas9 (xCas9) and Cas9 that can recognize relaxed NG PAMs (Cas9-NG) [206]. Comparison of conventional breeding, genetic engineering, and genome editing is illustrated in Figure 4.

**Figure 4.** Developing disease-resistant rice: Comparison of conventional breeding, genetic engineering and genome editing [194].

8. Future perspectives and strategies

In the recent past, rapid development in biotechnology and genomics has aided deep understanding of both host and pathogen. In this view, there are a handful of innovative tools and strategies available for developing rice varieties with effective and durable resistance against several races.

8.1Allele mining

Allele mining is the most widely used method in identifying naturally occurring novel alleles or allelic variants of a gene in a set of germplasm. The allele mining
mainly involves two different approaches, i.e., EcoTilling and sequence-based or PCR-based allele mining. Compared to EcoTilling, sequence-based allele mining is reported to be simple and cheaper [163, 207]. The sequence-based allele mining involves PCR amplification of a particular gene and sequencing the PCR product to look for different gene versions. Hence, the host’s sequencing information is an essential prerequisite of any allele mining program. The alleles arise in a population due to natural mutations such as transition, transversion, and InDels. Hence, allele mining has to be regularly performed to identify any valuable alleles originating in the germplasm. In a recent report, sequence-based allele mining was performed to amplify and sequence the allelic variants of the major rice blast resistance genes at the \textit{Pi2/Pi9} locus of chromosome 6 from the 361 blast-resistant varieties. Thirteen novel \textit{Pi9} alleles (named \textit{Pi9-Type1} to \textit{Pi9-Type13}) were identified in these 107 varieties. These could potentially serve as a genetic resource for molecular breeding resistance to rice blast [176].

8.2 Identification of SNPs for fast-tracking of MAS

Owing to the evolution of next-generation sequencing technology, genome-wide association mapping (GWAS) has found its way as an efficient tool for mapping genes. Using this method, several QTLs and loci have been identified to be associated with a set of different traits of agronomical importance [208–210]. Similarly, GWAS can identify functional SNPs associated with resistance to rice blast fungus, and MAS can be made much faster and robust. Further, GWAS can also be used to fast-track the background selection of a MAS program by collecting SNPs distributed evenly on the whole genome. The use of these high-density markers and high-resolution genome scans can identify the genomic content contributed by each parent in a breeding program involving multiple parents [181].

8.3 Host induced gene silencing

Induction of host resistance to several pathogenic fungi following the expression of the fungal genes in the host plant has been demonstrated in several cases [211]. Similarly, genes encoding a set of proteins that are very crucial in the initial establishment of \textit{M. oryzae} in rice can be silenced by their expression in the host system. This approach holds considerable potential in breeding the next-generation rice varieties and seeks more research. However, one of the main drawbacks of this approach is that it requires genetic transformation and expression of the foreign genes in the plants.

8.4 Modification of host genes targeted by blast pathogen

The infection of the host by the rice blast pathogen requires recognition of some host proteins for establishing the infection. Hence, articulating the host target proteins by genome editing technologies fails the pathogen to recognize the host targets, limiting the infection. This approach is novel and different as the focus is on articulating the host susceptibility genes rather than R genes. A susceptibility gene refers to genes that render the host susceptible to a pathogen. This approach is now facilitated and made practical with the availability of biotechnology tools such as TALENs and CRISPR/Cas9 technologies. In this direction, the proof of concept has been demonstrated by modifying a specific target gene recognized by \textit{Xanthomonas oryzae pv. oryzae} using TALEN technology [205].

Here, we report the identification and functional characterization of a new member of the miR812 family in rice (named as miR812w) involved in disease
Rice resistance. miR812w is present in cultivated Oryza species, both japonica and indica subspecies, and wild rice species within the Oryza genus, but not in dicotyledonous species. miR812w is a 24 nt-long that requires DCL3 for its biogenesis and is loaded into AGO4 proteins. Whereas overexpression of miR812w increased resistance to infection by the rice blast fungus Magnaporthe oryzae, CRISPR/Cas9-mediated MIR812w editing enhances disease susceptibility, supporting that miR812w plays a role in blast resistance.

One recent report showed the identification and functional characterization of a new member of the miR812 family in rice (named as miR812w) involved in disease resistance. miR812w is present in cultivated Oryza species, both japonica and indica subspecies. miR812w is a 24 nt-long that requires DCL3 for its biogenesis and is loaded into AGO4 proteins. Overexpression of miR812w in rice increased resistance to infection by M. oryzae, CRISPR/Cas9-mediated MIR812w editing enhances disease susceptibility, supporting that miR812w plays a role in blast resistance [212].

8.5 Race dependent deployment of R genes

The success of any resistance breeding program mainly depends on the precise identification of the Avr genes prevailing in a particular location. This challenge can be met by identification and characterization of different races of M. oryzae of a location. While the irrational deployment of R genes to address blast disease incidence will not lead to the expected outcome, it adds additional burden to the host in expressing a specific R gene for which the Avr gene is absent and finally results in yield penalty. Furthermore, this exercise has to be continued due to the shift of the avirulence composition in M. oryzae populations. To date, a significant number of Avr genes have been identified and cloned. Hence, a simple PCR can be used to ascertain the frequencies of Avr genes and further planning of the breeding program.

9. Conclusion

The management of rice blast fungus is complex due to the continuous evolution of new pathotypes worldwide and India. Although several fungicides, cultural and biological control measures of blast disease are employed at the field level, the use of durable host plant resistance has shown great potential. In addition to being cost-effective, resistance breeding is environmentally friendly and demands less attention and intervention by illiterate farmers. Most of the resistance breeding programs in India were primarily based on single-gene resistance through conventional breeding approaches. However, blast pathogen has successfully overcome the single-gene resistance in a short period and rendered several varieties susceptible to blast, which was otherwise intended to be resistant. Some of the blast endemic areas of India are characterized by the existence of a mixture of more than one race of the blast pathogen, making the situation more challenging. However, the recent technological advancements, including genomics, gene editing, and pyramiding of more than one resistance gene assisted by genetic markers, hold huge promise in counteracting M. grisea. Hence, future resistance breeding programs should exploit the modern biotechnology tools and conventional breeding approaches in developing durable blast resistance varieties harboring multiple R genes. The harmonious blending of the bio-control approaches, cultural management practices, and modern breeding methods is the key to successfully addressing blast disease in rice cultivation ecosystems. Further, the effectiveness of the blast-resistant varieties developed for a location can only be achieved when the gene deployed is based on
the Avr genes prevalent in that area. Therefore, more efforts are needed to conduct the basic research pertaining to a specific location.

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Conflict of interest

The authors have no conflict of interest associated with this work.

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