Abstract: Rice is a global food grain crop for more than one-third of the human population and a source for food and nutritional security. Rice production is subjected to various stresses; blast disease caused by *Magnaporthe oryzae* is one of the major biotic stresses that has the potential to destroy total crop under severe conditions. In the present review, we discuss the importance of rice and blast disease in the present and future global context, genomics and molecular biology of blast pathogen and rice, and the molecular interplay between rice–*M. oryzae* interaction governed by different gene interaction models. We also elaborated in detail on *M. oryzae* effector and *Avr* genes, and the role of noncoding RNAs in disease development. Further, rice blast resistance QTLs; resistance (*R*) genes; and alleles identified, cloned, and characterized are discussed. We also discuss the utilization of QTLs and *R* genes for blast resistance through conventional breeding and transgenic approaches. Finally, we review the demonstrated examples and potential applications of the latest genome-editing tools in understanding and managing blast disease in rice.

Keywords: rice; *Magnaporthe*; resistance; *R*-genes; QTLs; resistance-breeding; CRISPR/Cas

1. Introduction

Rice, being the major staple food and one of the main sources of income and employment, is an important crop all over the world. Almost 90% of the global production and consumption of rice is reported from Asia, where a considerably large part of the world’s population resides (www.fao.org; accessed on 20 January 2022).

Since 2000, global rice consumption has exceeded its production and the annual shortage of rice is estimated to increase from 400,000 tons in 2016 to 800,000 tons by 2030 [1,2]. As the global population is expected to reach 9.77 billion by 2050, the rice production needs to be doubled from the present levels to ensure both global food and nutritional security. The total rice production can be enhanced by either increasing area...
of cultivation, productivity, or by avoiding the yield losses. However, the global analysis shows no scope for expanding the area due to the unavailability of arable land [3]. Therefore, avoiding the losses due to adverse environmental factors and post-harvest losses is the only feasible way to enhance the productivity and overall production.

Rice crop is challenged by a number of biotic and abiotic stresses in the various rice-growing regions of the world. Among these, blast disease caused by M. oryzae is considered as the most serious disease of rice [4]. The average losses due to this disease range from 10 to 30%, with up to 100% loss having been reported under severe conditions. In this article, we presented a critical analysis of the literature on the recent developments in understanding the rice–M. oryzae interaction, and understanding the complex mechanism of host resistance. Further, we elaborated on different approaches for the development of blast-resistant rice lines to counteract the imminent threat posed by the emergence of new M. oryzae races.

2. Global and National Significance of Rice Blast in Present and Future Context

Among the biotic stresses, rice blast is the most serious constraint that restricts the global rice production [4,5]. The major blast epidemics covering vast areas occur on a regular basis, resulting in 10–30% crop losses annually which represents a yield loss of about 157 million tons worldwide. The disease was reported for the first time in China in 1637 and was recognized as rice fever disease. Further, it was described as imochibyo in Japan in 1704 and as brusone in Italy in 1828. Now this disease covers almost 85 nations, posing a major threat to food security and farmers’ revenue in the regions of South Asia and Africa [6,7]. M. oryzae causes blast in more than 50 grass species [8], and among agriculturally important crop species, it infects rice, wheat, rye, barley, pearl millet, and finger millet.

Several blast epidemics have been recorded in rice. In the epidemic year 1953, an estimated loss of about 800,000 tons of rice was recorded in Japan. In India, blast disease was first reported in 1913, and in the year 1919 an epiphytotic was reported from the erstwhile Madras state [4]. Further, seven epidemics of rice blast were recorded in Himachal Pradesh, Andhra Pradesh, Tamil Nadu, and Haryana between 1980 and 1987 [4]. Even though huge yield losses have resulted due to rice blast epidemics, proper information on yield loss data during the last 30 years is not available for India. Frequent outbreaks have been recorded in hilly areas of Uttarakhand, Himachal Pradesh, and Jammu and Kashmir, where about 65% yield is lost to blast disease due to the prevailing blast-favorable condition during the kharif season [9]. Further, eastern India experiences frequent occurrences of the blast due to the favorable climatic conditions for the growth and development of the pathogen. About 564,000 tons of rice is lost yearly due to blast in eastern India alone, nearly 50% of which is lost under the upland ecosystem [9]. Blast disease incidence has also been reported in the rice-growing areas of peninsular and plain regions of India.

3. Molecular Interplay between Rice and M. oryzae

3.1. The Pathogen: M. oryzae

Magnaporthe oryzae, the causal agent of blast disease in rice, is a hemibiotrophic pathogen belonging to the Magnaporthaceae family. This fungus attacks rice plants at all developmental stages and infects leaves, stems, nodes, panicles, and roots. The process of infection begins with the landing and attachment of a conidium to the rice leaf cuticle. An adhesive from the germinating conidium helps to stick to the cuticle. The conidium under favourable conditions germinates to produce germ-tube, which further differentiates into appressorium. The appressorium has a differentiated cell wall and a distinct melanin layer between its cell wall and the cell membrane. This layer helps in the generation of turgor pressure, which is later translated into mechanical force through the penetration peg and helps in penetration through the leaf cuticle. Once inside the cell, hyphae multiply rapidly, leading to disease development and visible blast symptoms. A graphical representation of the infection cycle of M. oryzae in rice is given in Figure 1. For detailed information about
the infection process of *M. oryzae* in rice, readers can refer to the article by Wilson and Talbot [10].

![Figure 1](image_url)

**Figure 1.** Schematic representation of the life cycle of the *M. oryzae* in rice.

### 3.1.1. *Magnaporthe oryzae* Genomics

To develop durable and effective blast resistance in rice crop against *M. oryzae*, a comprehensive understanding of the molecular mechanism underlying the blast pathogenesis and host resistance in rice required. The developments in the field of genomics, associated with various molecular biology techniques, are enabling researchers to dissect out the molecular mechanism of rice–*M. oryzae* interaction.

Among the phytopathogenic fungi, *M. oryzae* is the first one to have its genome sequenced [11]. This fungus has emerged as a model system for studying the host–pathogen interactions and understanding the pathogenicity mechanisms of plant pathogens. *M. oryzae* displays an exceptional genomic plasticity due to frequent occurrences of repeat elements in its genome. Such genome instability leads to a rapid evolution of new races in the population. Genome resequencing of *M. oryzae* provides an opportunity to investigate and understand the host–pathogen interaction processes of a particular strain at the molecular level for effectively managing the rice blast disease [12]. Consequently, genomes of more than 74 strains of *M. oryzae* have been sequenced (Table 1). On average, the genome size of *M. oryzae* is 40.12 Mb and contains 12,684 genes (Table 1). However, the estimated genome size with a gap was reported to be 40.3 Mb [11]. Repeat elements distribution throughout its genome is the main factor that determines the genome plasticity and several studies have reported the influence of repeat elements in the genomic features [12,13]. Many avirulence and effector molecules have been characterized in this pathogen and were found to have association with the repeat element for their functional activities [12]. The repeat elements are known to play a vital role in *M. oryzae* genome variation and genome evolution, and largely impact the virulence spectrum at the individual strain level [13–15]. Interestingly, every sequenced *M. oryzae* strain had isolate-specific genomic regions as well as genes. Such isolate-specific genes and genomic regions determine racial evolution, environmental adaptation, chromosomal variability, variation in repeat element distribution, and host range specificity during the course of evolution [14,16]. It is also hypothesized that the isolate-specific genes might be an event of the gene gained or lost during the evolution process [13]. The resequencing of different *M. oryzae* genomes assists in constructing a pan-genome that describes a consensus genome sequence derived from multiple or individual genomes of different strains, species, or genera, and could be utilized as a complete reference sequence. Pan-genome has many prospective applications and helps to analyze multi-omics data. It could be used for genome-wide association study, metagenomics, population genetics, phylogenomics, etc. Pan-genome also provides information about the presence and absence of variation, core genes, dispensable genes, etc. Using the pan-
genome approach in *M. oryzae*, Singh et al. [12] identified a retro-transposable element that displayed a significant difference in copy number and distribution between virulent and avirulent strains. The knowledge gathered through different genomics resources by applying bioinformatics approaches such as comparative genome analysis, pan-genome analysis, and metagenomics analysis can shed light on the mechanisms of frequent emergence of new races of *M. oryzae*, and advances our understanding about the host–pathogen interactions for the effective management of blast disease.

### Table 1. List of *Magnaporthe oryzae* genome sequenced.

| Strain      | Genome Size (Mb) | N 50 Value | No. of Genes | % of Repeats in Genome | Reference |
|-------------|------------------|------------|--------------|------------------------|-----------|
| 70-15       | 38.8             | 1.6 Mb     | 11,109       | 9.7                    | [11]      |
| Ina168      | 38.0             | 28.4 Kb    | NA           | NA                     | [16]      |
| P131        | 37.95            | 12.3 Kb    | 12,714       | 3.15                   | [13]      |
| Y34         | 38.87            | 11.6 Kb    | 12,862       | 3.41                   | [13]      |
| FJ81278     | 37.3             | 151.7 Kb   | 10,453       | 2.73                   | [14]      |
| HN19311     | 37.1             | 147.4 Kb   | 10,256       | 2.83                   | [14]      |
| 98-06       | 42.1             | 88.6 Kb    | 14,019       | 9.3                    | [17]      |
| B157        | 41               | 92.4 Kb    | 12,535       | 10.4                   | [15]      |
| MG01        | 43               | 54.6 Kb    | 13,135       | 10.39                  | [15]      |
| GF51-7-2    | 39.1             | 88.3 Kb    | 12,468       | NA                     | [16]      |
| Br48        | 40.7             | 97.5 Kb    | 12,671       | NA                     | [16]      |
| Br58        | 40.2             | 91.1 Kb    | 12,626       | NA                     | [16]      |
| Z2-1        | 39.5             | 64.1 Kb    | 12,383       | NA                     | [16]      |
| Dig41       | 41.3             | 24.3 Kb    | 11,457       | NA                     | [16]      |
| RML-29      | 42.2             | 10.4 Kb    | 12,746       | 11.78                  | [12]      |
| RP-2421     | 44.85            | 35.35 Kb   | 12,957       | 12.28                  | [12]      |
| 2539        | 38.08            | 107 Kb     | 12,116       | NA                     | [18]      |
| RMg_Dl      | 42.42            | 524.2 Kb   | 10,555       | NA                     | [19]      |
| FR13        | 46.45            | 5.39 Mb    | 14,322       | 13.23                  | [20]      |
| US71        | 45.61            | 2.81 Mb    | 14,348       | 13.23                  | [20]      |
| CD156       | 43.39            | 5.53 Mb    | 14,304       | 6.21                   | [20]      |
| BR32        | 41.85            | 5.09 Mb    | NA           | 6.26                   | [20]      |
| QJ08-2006   | 38.41            | 127.4 Kb   | 10,432       | 2.28                   | [21]      |
| QJ10-10     | 38.28            | 105.1 Kb   | 10,418       | 2.22                   | [21]      |
| QJ10-3001   | 38.40            | 133.1 Kb   | 10,401       | 2.28                   | [21]      |
| RMg-Dl      | 34.82            | 45.894 Kb  | 12,747       | NA                     | [22]      |
| 70-15       | 40.90            | NA         | 12,991       | 11.1                   | [23]      |
| FR13        | 42.40            | 0.104 Mb   | 14,384       | 1.56                   | [23]      |
| GY11        | 39.00            | 0.226 Mb   | 14,781       | 1.00                   | [23]      |
| PH14        | 40.00            | 0.757 Mb   | 13,816       | 1.16                   | [23]      |
| TH12        | 40.10            | 0.716 Mb   | 14,026       | 1.46                   | [23]      |
| TH16        | 39.10            | 0.939 Mb   | 13,571       | 1.60                   | [23]      |
| US71        | 41.20            | 0.814 Mb   | 13,803       | 2.40                   | [23]      |
| BR32        | 41.90            | 1.760 Mb   | 14,336       | 2.00                   | [23]      |
Table 1. Cont.

| Strain   | Genome Size (Mb) | N 50 Value | No. of Genes | % of Repeats in Genome | Reference |
|----------|------------------|------------|--------------|------------------------|-----------|
| CD156    | 42.70            | 1.066 Mb   | 14,067       | 1.47                   | [23]      |
| BR29     | 40.90            | 0.955 Mb   | 12,283       | 1.60                   | [23]      |
| V86010   | 38.9             | 93.4 Kb    | 11,857       | 5.1                    | [24]      |
| 76_3     | 38.35            | 0.159 Mb   | NA           | NA                     | [25]      |
| 82_0835  | 40.07            | 0.136 Mb   | NA           | NA                     | [25]      |
| 90_4_1   | 39.92            | 0.151 Mb   | NA           | NA                     | [25]      |
| BF17     | 39.72            | 0.138 Mb   | NA           | NA                     | [25]      |
| BF32     | 40.18            | 0.120 Mb   | NA           | NA                     | [25]      |
| BF48     | 40.01            | 0.144 Mb   | NA           | NA                     | [25]      |
| BF5      | 40.96            | 0.122 Mb   | NA           | NA                     | [25]      |
| BN0293   | 38.14            | 0.178 Mb   | NA           | NA                     | [25]      |
| EG308    | 41.56            | 0.149 Mb   | NA           | NA                     | [25]      |
| Glhn3    | 39.39            | 0.134 Mb   | NA           | NA                     | [25]      |
| Glhn4    | 39.52            | 0.134 Mb   | NA           | NA                     | [25]      |
| JUM1     | 40.50            | 0.127 Mb   | NA           | NA                     | [25]      |
| KE002    | 40.25            | 0.147 Mb   | NA           | NA                     | [25]      |
| KE016    | 40.27            | 0.156 Mb   | NA           | NA                     | [25]      |
| KE017    | 40.10            | 0.141 Mb   | NA           | NA                     | [25]      |
| KE019    | 39.37            | 0.176 Mb   | NA           | NA                     | [25]      |
| KE021    | 40.07            | 0.152 Mb   | NA           | NA                     | [25]      |
| KE029    | 41.03            | 0.154 Mb   | NA           | NA                     | [25]      |
| KE041    | 39.00            | 0.146 Mb   | NA           | NA                     | [25]      |
| KE210    | 38.85            | 15.86 Kb   | NA           | NA                     | [25]      |
| KE255    | 39.83            | 0.119 Mb   | NA           | NA                     | [25]      |
| KE332    | 41.00            | 35.0 Kb    | NA           | NA                     | [25]      |
| KE415    | 39.45            | 33.91 Kb   | NA           | NA                     | [25]      |
| KE443    | 40.88            | 36.41 Kb   | NA           | NA                     | [25]      |
| KE473    | 40.61            | 37.27 Kb   | NA           | NA                     | [25]      |
| KE491    | 40.05            | 34.50 Kb   | NA           | NA                     | [25]      |
| NG0110   | 39.60            | 0.115 Mb   | NA           | NA                     | [25]      |
| NG0135   | 39.85            | 0.109 Mb   | NA           | NA                     | [25]      |
| NG0153   | 39.80            | 0.130 Mb   | NA           | NA                     | [25]      |
| NGO104   | 38.94            | 0.128 Mb   | NA           | NA                     | [25]      |
| TG004    | 39.96            | 0.125 Mb   | NA           | NA                     | [25]      |
| TH3      | 37.30            | 22.62 Kb   | NA           | NA                     | [25]      |
| TZ090    | 38.95            | 0.127 Mb   | NA           | NA                     | [25]      |
| UG08     | 39.18            | 0.122 Mb   | NA           | NA                     | [25]      |
| V0104    | 40.34            | 0.126 Mb   | NA           | NA                     | [25]      |
| V0108    | 39.90            | 0.153 Mb   | NA           | NA                     | [25]      |
| V0113    | 39.75            | 0.151 Mb   | NA           | NA                     | [25]      |

NA: Not available
3.1.2. Pathogenicity Related Factors of *M. oryzae*

i. Analysis of Avr and effector genes. Pathogen-associated molecular patterns (PAMPs) are recognized by host cell surface-localized pattern-recognition receptors (PRRs) to activate plant immunity. PAMP-triggered immunity (PTI) constitutes the first layer of plant immunity that restricts pathogen proliferation. To establish a successful infection, the *M. oryzae* has to overcome the PTI. Therefore, a successful isolate switches to the deployment of effector proteins, leading to a response known as effector-triggered susceptibility (ETS) [26]. In ETS, the effector plays with host defense regulators, such as the Secreted LysM protein 1 (Slp1) with two LysM domains; acts as a competitive inhibitor for the binding of chitin with host chitin elicitor-binding protein (CEBiP); and thereby subverts the PTI [16]. A recent study demonstrated a similar mechanism, where chitinase 1 (MoChia1) competitively inhibits the binding of host tetratricopeptide repeat protein (OsTPR1) with PAMP, chitin, and compromises PTI [27]. More recently, effectors such as MoHTR1 and MoHTR2 were found to be directly targeted into the rice nucleus to undermine PTI [28]. Till-date, 26 Avr/effector genes have been mapped in *M. oryzae*, and 14 of them, including two unmapped Avrs, MoHTR1 and MoHTR2, have been cloned and characterized (Table 2).

### Table 2. List of *Magnaporthe* Avr/effector genes cloned.

| Avr Gene        | Protein Size (aa) | Chromosome | Effector Type * | Cognate R Gene | Reference |
|-----------------|-------------------|------------|-----------------|----------------|-----------|
| PWL1            | 147               | 2          | Glycine-rich    | Unknown        | [29]      |
| PWL2            | 145               | 2          | Glycine-rich    | Unknown        | [30]      |
| AVR1-CO39       | 89                | 1          | ToxB like      | Pi-CO39        | [31]      |
| AVR-Pita        | 224               | 3          | Zinc metalloprotease | Pi-ta | [32]      |
| ACE1            | 4035              | 1          | PKS/NRPS       | Pi33 (not cloned) | [33]    |
| AVR-Pia         | 85                | 5 or 7 **  | ToxB like      | Pia            | [16,34]  |
| AVR-Pii         | 70                | 7          | Unknown        | Pii            | [16]      |
| AVR-Pik/km/kp; (AVR-Pikh) | 113 (5 alleles) | 1          | ToxB like      | Pik/Pik-m/Pik-p; Pik-h | [16,35] |
| AvrPiz-t        | 108               | 7          | ToxB like      | Piz-t          | [36]      |
| AVR-Pi9         | 91                | 7          | Six cysteine   | Pi9            | [37]      |
| AVR-Pib         | 75                | 3          | Unknown        | Pib            | [38]      |
| AVR-Pi54        | 153               | 4          | ToxB like      | Pi54, Pi54rh, Pi54of | [39] |
| MoHTR1          | Unknown           | Unknown    | zinc-finger TF | Unknown        | [28]      |
| MoHTR2          | Unknown           | Unknown    | zinc-finger TF | Unknown        | [28]      |

PKS, polyketide synthase; NRPS, non-ribosomal peptide synthetase. *Magnaporthe Avis and ToxB like (MAX)-effectors are classified based on protein 3-D models [38–40]. **AVR-Pia is located on chromosome 5 (isolates Ina168 and Y93-165g-1; [34,41]) and chromosome 7 (isolate JS153; [42]).

The first discovered Pwl effectors (Pwl1–Pwl4) belong to a small, glycine-rich rapidly evolving effector family that provides avirulence on weeping lovegrass and finger millet, but has no effect on rice. Except for cell death-induction/suppression or interacting with resistance proteins features, the identification of candidate effector proteins is a difficult task due to their unique sequence features. Recent structural studies have shown that despite sequence divergence, several effector proteins can share structural similarities. An NMR-based study on diverse ascomycetous effectors from *M. oryzae* and *Pyrenophora tritici-repentis* revealed these proteins to possess conserved 6 β-sandwich structures stabilized by conserved cysteines. A detailed study showed that previously characterized effectors such as Avr Piz-t and ToxB also possess those folds, forming a conserved MAX effector family (*Magnaporthe* Avis and ToxB like). Additionally, 5–10% of the effectors expressed in the biotrophic phase of *M. oryzae* possess MAX, and more than 50% of cloned effectors also
belong to the MAX effector class. The computation prediction of the effector proved to be a useful method to highlight potential candidates with conserved folds.

Among the 26 reported Avr-genes, 15 were mapped near the chromosome ends, and 5 of the cloned Avr genes were flanked by transposons. These transposons are active companions of the Avr genes and play a role in the loss and gain of these genes. The molecular interaction studies of the reported seven R-Avr pairs showed that five of them, namely, Pi-ta/AVR-Pita, Pik/AVR-Pik, Pia/AVR-Pia, Pi-CO39/AVR1-CO39, and Pi54/AVR-Pi54 interact directly, whereas Piz-t/AvrPiz-t and Pi1/AVR-Pi1 have indirect interaction. Besides the one-to-one interactions, two other types of interactions were also reported. One, where two R proteins (homologs) interact with one Avr protein, such as in Pik-1 and Pik-2 with AVR-Pik [43], and a similar mechanism is predicted in the case of recently cloned Avr MoHTR1 and MoHTR2, which are predicted to interact with the same target protein [28]. In another interaction, two different Avr proteins could be recognized by a single R protein complex, such as two NLR proteins, RGA4 and RGA5 hetero-dimers interacting with AVR-Pia or AVR1-CO39 [44,45].

ii. Small non-coding RNAs in pathogenesis. Eukaryotic organisms produce small RNAs (sRNAs) that include microRNAs (miRNAs) and short interfering RNAs (siRNAs) of approximately 18–25 base pairs. These sRNAs modulate the diverse cellular activities through a process known as RNA interference (RNAi) [46]. These RNAs also take part in the mechanisms to subvert the host immune system during infection [47]. Most of the sRNAs produced by the organisms’ function endogenously, however, there are reports suggesting that these sRNAs can travel beyond the organismal boundaries and regulate the genes in interacting organisms through a mechanism commonly known as “trans-kingdom RNAi” [46,48]. This bidirectional movement of sRNAs has been reported between animal/plant hosts and microorganisms interacting with them [48–53].

Magnaporthe oryzae possess functional RNAi pathway genes such as Dicer, Argonaute, and RNA-dependent RNA polymerase. These genes are required for the biogenesis of sRNAs that are involved in the regulation of fungal growth, virulence, and stress tolerance [54,55]. Nunes et al. [56] characterized the sRNA repertoire in this pathogen by using the next-generation sequencing approach. They reported tissue-specific enrichment of sRNAs from mycelia and appressoria-specific small RNA libraries. Raman et al. [54] analyzed the expression sRNAs in M. oryzae by subjecting it to different in-vitro stresses and observed two distinct peaks of sRNAs of 24 nt and 26 nt during mycelial and in-planta growth, respectively [55]. The majority of sRNAs produced by this pathogen were aligned to intergenic (54%) and repeat regions (41%) and only 4% of the total sRNAs matched to protein-coding genes. Although the numbers of sRNAs mapped to intergenic and repeat regions was high, the proportion of uniquely mapped sRNAs was higher in protein-coding regions [54]. The sRNA expression profiles of the pathogen indicated differential preference for 5’ nucleotide; the most abundant nucleotide at the start of sRNAs was Uracil followed by Guanine and Adenine in in-planta libraries. By employing a knockout mutant of Argonaute (Δmoago3) and RNA-dependent RNA polymerase (Δmordrp1), Raman et al. [55] demonstrated that sRNAs produced by the pathogen are involved in the regulation of pathogenesis-related genes such as MGG_01662 (4-aminobutyrate aminotransferase), MGG_02329 (isotrichodermin C-15 hydroxylase), and MGG_02378 (glutamate decarboxylase). M. oryzae mutants lacking moago3 and mordrp1 failed to infect barley during wound assays, and both mutants showed reduced production conidia. The deletion of a single Dicer (Δmodcl1) or double knockout for Dicer genes (Δmodcl1+Δmodcl2) upregulated many genes involved in pathogenicity such as MGG_10932 coding for C2H2 zinc-finger transcription factor involved in the formation of appressorium; MGG_14068, encoding a putative FAD oxidoreductase; MGG_02065 encoding kinesin light chain; and MGG_10027 encoding a calcium-transporting ATPase 1.

The above studies clearly demonstrated that the RNAi pathway genes and sRNAs play an important role in the regulation of the pathogenicity during rice–M. oryzae interaction. Besides several proteinaceous and non-proteinaceous effector molecules [57], many fungal
pathogens have developed novel virulence mechanisms and deliver sRNAs as effector molecules to overcome immunity during interaction with the host plant [52,58,59]. In *M. oryzae*, the comparative expression profiling of sRNAs during vegetative phase and rice infection revealed a significant upregulation of 366 *M. oryzae* sRNAs during infection. Out of these differentially expressed sRNAs, 14 were identified as potential candidates, which may act as effectors during rice–*M. oryzae* interaction [60]. In another study, Zhao et al. [46] computationally created a regulatory network of *M. oryzae* sRNAs during rice–*M. oryzae* interaction by utilizing the available transcriptomics and proteomics resources. These researchers identified 22 sRNAs and 77 secretory proteins of *M. oryzae* that may participate in the infection process. Though both the above studies predicted *M. oryzae* sRNAs as possible mediators of rice–*M. oryzae* interaction, the accuracy of in-silico findings needs to be further verified through extensive laboratory studies.

3.1.3. Editing Pathogenicity Genes in *M. oryzae*

CRISPR-Cas systems have been extensively used in editing the genomes of diverse organisms for a wide range of applications [61,62]. For a wide range of filamentous fungus, including various plant pathogens, CRISPR/Cas-based genome-editing strategies have already been demonstrated [61,62]. They are usually based on Cas9 and gRNA transgenic expression. Cas9 and gRNA coding plasmid can be transformed to fungal cells; however, sometimes they might prove cytotoxic. To avoid cytotoxicity, Cas9 and gRNA can also be complexed into a functional ribonucleoprotein (RNP) *in vitro* and then introduced through transformation, as established in *M. oryzae* [62,63]. gRNA molecules may be synthesized in vitro and co-transformed alongside Cas9-encoding cassettes. In microalgae, *in vitro* synthesized and characterized gRNAs were supplied as RNPs along with Cas proteins [64–67]. In the rice blast fungus *M. oryzae*, concurrent double editing was accomplished using this approach [63]. Similarly, Cas12a RNP was successfully used for *M. oryzae* genome editing in a recent report [68]. However, there are no reports on CRISPR/Cas-based transformation of *M. oryzae* for changing pathogenicity or creating novel variants of the pathogen.

3.1.4. Magnaporthe Host-Shifting

*Magnaporthe oryzae* is known to infect many crops including rice, wheat, several millets, and grasses among the others. The rice blast pathogen (*M. oryzae* pathotype *Oryzae*: MoO) is ranked top in the list of 10 most destructive fungal plant pathogens [69], whereas wheat blast (WB) pathogen *M. oryzae* pathotype *Triticum* (MoT) was a lesser known disease with its confinement to parts of Africa. The recent wheat blast epidemics in Bangladesh and Zambia [70,71], however, put the MoT on the global map for having a serious consequence on the world wheat production. Besides other factors, the prevailing weather conditions during the wheat-growing season seem to have played a major role in wheat blast epidemics in Bangladesh [72,73]. Though initially it was assumed that WB is an event of host-shift of MoO from rice to wheat, recent molecular studies have confirmed that MoT from Bangladesh and South America have highly similar genetic content [74], and they are also distinct from other *Magnaporthe* pathotypes [75–78]. Interestingly, among the *Magnaporthe* pathotypes, only *Triticum* (MoT) infects other hosts than its main host, wheat [79,80]. These findings highlight the potential host-shift/jumping of *Magnaporthe* pathotypes and the associated threat to the crop production, specifically to cereals and millets.

3.2. The Host: Rice

3.2.1. Genomes Sequenced

The genome *Oryza* is the smallest cereal crop genome with an estimated size of 400–430 Mb [81]. *Oryza sativa japonica* cultivar *Nipponbare* was the first rice genome sequenced [81]. However, the *O. sativa indica* rice, which further has *indica* and *aus* subpopulations, is the most widely planted rice globally [82]. Several draft genomes of *indica* rice have been assembled [83]. A highly contiguous and near-complete *indica* rice genome reported
is for the cultivar Shuhui 498 (R498) [84]. The developments in the field of next-generation sequencing (NGS) technologies have revolutionized the field of genome sequencing in plants [85]. In a major effort, a core collection of 3,000 rice accessions from 89 countries were sequenced with an average sequencing depth of 14 X, average genome coverage of 94.0%, and average mapping rates of 92.5% [86]. After aligning these genomes to the reference genome, i.e., Nipponbare, a total of 18.9 million single nucleotide polymorphisms (SNPs) were discovered. SNP data were used for the phylogenetic analyses to differentiate O. sativa gene pool into five varietal groups (indica, aus/boro, basmati/sadri, tropical japonica, and temperate japonica). Besides O. sativa, genomes of different wild species of rice have been sequenced. The list of wild rices with their genomes sequenced include, O. longistaminata (2014; ID: 11285); O. glaberrima, an African wild rice (2010; ID:458); O. minuta, a perennial wild rice from southeast Asia (2014; ID: 10965); O. meridionalis, a wild rice from Australia (2012; ID: 11319); O. coarctata, a wild rice from Bangladesh (2019; ID: 11313); O. australiensis, an Australian wild rice variety (2021; ID: 10966); O. officinalis, a tropical and sub-tropical wild rice (2014; ID: 10964); O. punctata, an African wild rice (2014; ID: 10963); O. nivara, a wild rice from India (2014; ID: 2841); O. rufipogon, a wild rice species from tropical and subtropical regions of Australia and Asia (2014; ID: 457); O. meyeriana var. granulata, a wild rice from Thailand (2012; ID: 11287); and O. glumipatula (2013; ID: 11318) [72].

With the availability of a large number of genome sequences within rice, such as in 3K database [87], efforts are being made to study the rice genome at the pan-genome level. Zhao et al. [88] constructed a pan-genome dataset of the O. sativa–O. rufipogon species complex through sequencing and de novo assembly in 66 diverse accessions. Most of the stress response-related genes, including those coding for NBS-LRR proteins, were detected only in a subset of accessions, thus suggesting the existence of a diverse repertoire of biotic stress-resistance genes in the species studied. Therefore, pan-genome studies in rice hold a greater potential for the identification of new blast resistance genes.

3.2.2. Resistance Genes as Solo Protectors

Resistance (R) genes are an integral part of the plant defensome complex, and R and defense response (DR) genes contribute to broad-spectrum blast resistance in rice [89,90]. The immune responses governed by most of these R genes have intertwined networks and they mostly regulate downstream general defense pathways [91]. To date, more than 100 blast R genes have been mapped in rice and 38 of them have been cloned and characterized [Tables 3 and 4]. As a result of comprehensive genetic studies in rice, vis-à-vis its pathogen, M. oryzae, the rice–M. oryzae interaction has emerged as a premier model system for understanding the plant–fungal pathogen interactions [4,92]. The narrow race-specificity of R-genes and the ability of the pathogen to quickly evolve new races compatible with resistance genes are major hurdles in achieving long-lasting protection against the blast disease [93]. The identification and utilization of R genes that confer broad-spectrum resistance against a large number of pathogen races is the most effective approach to manage the disease [91]. Interestingly, among these 38 blast R genes, eight, namely Pi9 [94], Pi54 [95], pi21 [96], Pi50 [97], Pi7 [98], Pi57 [99], Pigm [100], and Ptr [101], are reported to provide broad-spectrum blast resistance, and only three, Pi5-1, Pi63, and Pb1 are pathogen-inducible, and the rest are expressed constitutively. Recently, Wang et al. [102] performed genome-wide analysis of NBS-LRR genes in a broad-spectrum resistant rice genotype Tetep, and functionally validated the role of 90 NBS-LRR genes in blast resistance. Since these genes have not been assigned designations as per the standard conventions for naming blast resistance genes in rice, these genes have not been included in the list of cloned R genes provided in Table 4.

The R genes generally act solo to induce the immune response in rice. However, the downstream pathways leading to resistance response encompass one or more DR proteins [91]. It is found that at least some of the reported R and DR gene combinations show positive association for conferring blast resistance in rice e.g., Pid2 with OsPUB15, Pik-H4 with OsBIHD1, and Pita with lesion mimic (sl) gene [103–105]. Besides R genes, five
DR genes *bsr-d1, bsr-k1, OsBBI1, spl11*, and *spl33* are reported to confer broad-spectrum blast resistance [69]. Among the DR genes identified for broad-spectrum blast resistance in rice [91], rice lesion mimic mutants (*lmm*) genes form a major section of these genes. These LMM mutants, through programmed cell death (PCD), mimic the blast symptoms and the natural HR response by inducing the resistance response. There are more than 30 *lmm* genes reported in rice for blast resistance. However, most of these genes have associated yield penalties while imparting the immune response. Hence, *lmm* genes are of limited use for rice blast-resistance breeding [91,106].

Table 3. Information on mapped rice blast resistance genes.

| Sl. No. | Gene ID | Chr. No. | Position (cM) | Source Cultivar | Reference |
|---------|---------|----------|---------------|-----------------|-----------|
| 1       | Pit     | 1        | 9.08–12.17    | Tjahaja         | [107]     |
| 2       | Pi27(t) | 1        | 24.29–27.90   | IR64 (Indica)   | [108]     |
| 3       | Pi24(t) | 1        | 20.97–22.22   | Azuenca (Japonica) | [109] |
| 4       | Pitp(t) | 1        | 100.54–108.43 | Tetep           | [110]     |
| 5       | Pi35(t) | 1        | 132.0–136.6   | Hokkai 188 (Japonica) | [111] |
| 6       | Pi37    | 1        | 132.44–133.95 | St. No. 1 (Japonica) | [112] |
| 7       | Pitsh   | 1        | 135.3–138.7   | Shin2 (Japonica) | [113]     |
| 8       | Pi11(t) | 2        | 87.5–89.9     | Digu            | [114]     |
| 9       | Pig(t)  | 2        | 137.38–140.54 | Guangchangzhan (Indica) | [115] |
| 10      | Pitq5   | 2        | 150.5–157.9   | Teqing          | [116]     |
| 11      | Pi11(t) | 2        | 153.2–154.1   | Yanxian No. 1   | [117]     |
| 12      | Pi21(t) | 2        | 153.2–154.1   | Yanxian No. 1   | [117]     |
| 13      | Pi(b)   | 2        | 153.2–154.1   | Tohoku II.9     | [118]     |
| 14      | Pi25(t) | 2        | 137.44–150.90 | IR64 (Indica)   | [119]     |
| 15      | Pi14(t) | 2        | 1–26.90       | Maowanggu       | [120]     |
| 16      | Pi(r-2-3(t)| 2 | 96.8–99.3 | IR64 (Indica) | [121] |
| 17      | Pitq2   | 2        | 109.6–112.2   | Teqing (Indica) | [122]     |
| 18      | Pirf2-1(t)| 2 | 109.6–112.2 | *O. rufipogon* (W) | [121] |
| 19      | Pi16(t) | 2        | 1–26.91       | Aus373 (Indica) | [123]     |
| 20      | Pitq3   | 3        | 6.8–9.7       | Teqing (Indica) | [122]     |
| 21      | Pi68    | 3        | 6.8–9.7       | *O. glumaepatula* (W) | [124] |
| 22      | pi21    | 4        | 20.97–22.22   | Owarihatamochi  | [125]     |
| 23      | Pikur1  | 4        | 98.44–134.23  | Kuroka (Japonica) | [126] |
| 24      | Pi39(t) | 4        | 107.4–108.2   | Chubu 111 (Japonica) | [127] |
| 25      | Pitq4   | 4        | 9.08–12.17    | Teqing (Indica) | [122]     |
| 26      | Pi(t)   | 4        | 9.08–12.17    | Tjahaja         | [128]     |
| 27      | Pi26(t) | 5        | 35.00–46.70   | Gumei 2 (Indica) | [119]     |
| 28      | Pi23(t) | 5        | 43.02–76.70   | Sweon 365       | [125]     |
| 29      | Pi10    | 5        | 58.08–75.41   | Tongil          | [129]     |
| 30      | Pi22(t) | 6        | 19.5–24.09    | Suweon365       | [125]     |
| 31      | Pi26(t) | 6        | 35.00–46.70   | Azucena (Japonica) | [130] |
| 32      | Pi27(t) | 6        | 22.22–2.97    | IR64 (Indica)   | [108]     |
| 33      | Pi40(t) | 6        | 65.09–70.12   | *O. australiensis* (W) | [131] |
| 34      | Piz     | 6        | 40.6–42.07    | Zenith (Japonica) | [132]     |
| Sl. No. | Gene ID | Chr. No. | Position (cM) | Source Cultivar | Reference |
|---------|---------|----------|---------------|----------------|-----------|
| 35      | Piz-t   | 6        | 58.7          | Toride 1       | [107]     |
| 36      | Pi9     | 6        | 41.5–41.55    | O. minuta (W)  | [133]     |
| 37      | Pi25(t) | 6        | 72.32–77.03   | Gumei 2        | [119]     |
| 38      | Pi8     | 6        | 19.5–24.09    | Kasalath (Indica) | [120] |
| 39      | Pi3(t)  | 6        |               | Pai-kan-tao (Japonica) | [134] |
| 40      | Pish1   | 6        | 92.6–98.2     | Teqing (Indica) | [135]     |
| 41      | Pi13(t) | 6        | 56.8–60.5     | Kasalath (Indica) | [136]     |
| 42      | Pi1     | 6        | 88.8–90.6     | Fujisaka 5 (Indica) | [120] |
| 43      | Pi2d    | 6        | 68.63–68.65   | Digu          | [137]     |
| 44      | Pigm(t) | 6        | 41.47–41.68   | Gumei 4       | [138]     |
| 45      | Pi17(t) | 7        | 89.00–99.9    | DJ 123        | [120]     |
| 46      | Pi36    | 8        | 11.48–11.53   | Q61 (Indica)  | [127]     |
| 47      | Pi33    | 8        | 23.66–24.61   | IR64 (Indica) | [134]     |
| 48      | Pizh    | 8        | 17.48–84.04   | Zhai-Ya-Quing8 (Indica) | [108] |
| 49      | Pi11    | 8        |               | Zhai-Ya-Quing8 (Indica) | [128] |
| 50      | Pi29(t) | 8        | 38.65–64.96   | IR64 (Indica) | [108]     |
| 51      | Pi2(t)  | 9        | 4.09–28.89    | Azucena       | [139]     |
| 52      | Pi5(t)  | 9        | 31.3–33.0     | RIL125, RIL249 and RIL260 (Moroberekan) | [140] |
| 53      | Pi3(t)  | 9        | 31.3–33.1     | Kan-Tao       | [128]     |
| 54      | Pi15    | 9        | 38.56–38.74   | GA25 (Japonica) | [120] |
| 55      | Pii     | 9        | 9.16–113.72   | Ishikari Shiroke (Japonica) | [141] |
| 56      | Pi28(t) | 10       | 78.26–90.67   | IR64 (Indica) | [108]     |
| 57      | Pia     | 11       | 1.01–2.09     | Aichi Asahi (Japonica) | [126] |
| 58      | PiCO39(t)| 11       | 25.21–27.55   | CO39 (Indica) | [142]     |
| 59      | Pilm2   | 11       | 54.54–113.5   | Lemont        | [116]     |
| 60      | Pi30(t) | 11       | 1.76–26.31    | IR64 (Indica) | [108]     |
| 61      | Pi7(t)  | 11       | 71.4–84.3     | RIL29 (Japonica) | [143] |
| 62      | Pi34    | 11       | 77.69–77.96   | Chubu32 (Japonica) | [144] |
| 63      | Pi38    | 11       | 76.55–87.91   | Tadukan (Indica) | [145] |
| 64      | PBR     | 11       | 80.5–120.3    | St. No. 1     | [146]     |
| 65      | Pi5     | 11       | 85.7–91.4     | Modan         | [147]     |
| 66      | Pi44(t) | 11       | 91.4–117.9    | RIL29 (Japonica) | [148] |
| 67      | Pik-h (Pi54) | 11       | 99.0–99.05    | Tetep         | [95]      |
| 68      | Pi1     | 11       | 105.99–113.49 | LAC23 (Japonica) | [149] |
| 69      | Pik-m   | 11       | 109.25–110.13 | Tsuyuake (Japonica) | [150] |
| 70      | Pi18(t) | 11       | 107.18–113.50 | Suweon365 (Japonica) | [132] |
| 71      | Pik     | 11       | 109.25–110.13 | Kusabue (Indica) | [151] |
| 72      | Pik-p   | 11       | 109.25–110.14 | HR22 (Indica) | [107]     |
| 73      | Pik-s   | 11       | 109.25–110.15 | Shin 2 (Japonica) | [152] |
| 74      | Pik-g   | 11       | 109.25–110.16 | GA20 (Japonica) | [120] |

For references, please refer to the original publication.
### Table 3. Cont.

| Sl. No. | Gene ID | Chr. No. | Position (cM) | Source Cultivar | Reference |
|---------|---------|----------|---------------|-----------------|-----------|
| 75      | Pise1   | 11       | 22.96–66.92   | Sensho          | [153]     |
| 76      | Pif     | 11       | 98.78–113.84  | Chugoku 31-1 (St. No. 1) | [154]     |
| 77      | Mpiz    | 11       | 16.29–66.92   | Zenith (Japonica) | [155]     |
| 78      | Pikur2  | 11       | 11.36–73.49   | Kuroka (Japonica) | [125]     |
| 79      | Pish    | 11       | 110.3–111.8   | Nipponbare (Japonica) | [113]     |
| 80      | Pib2    | 11       | 105.99–113.49 | Lemont (Japonica) | [122]     |
| 81      | Pi44    | 11       | 85.7–89.7     | Moroberekan (Japonica) | [148]     |
| 82      | Pi47    | 11       |               | Xiangzi (Indica) | [116]     |
| 83      | Pise    | 11       | 22.96–66.92   | Sensho          | [153]     |
| 84      | Piis1   | 11       | 11.36–76.11   | Imochi Shirazu (Japonica) | [153]     |
| 85      | Pi24(t) | 12       | 20.97–22.22   | Azuenca (Japonica) | [156]     |
| 86      | Pi62(t) | 12       | 9.7–77        | Tsuysuke (Japonica) | [157]     |
| 87      | Pittq6  | 12       | 23.0–30.92    | Tequing (Indica) | [116]     |
| 88      | Pi6(t)  | 12       | 1–1.68        | Apura (Indica) | [158]     |
| 89      | Pi12    | 12       | 27.95–60.48   | Moroberekan (Japonica) | [159]     |
| 90      | Pi21(t) | 12       | 20.94–22.22   | Owarihatamochi (Japonica) | [125]     |
| 91      | Pi31(t) | 12       | 30.92–47.66   | IR64 (Indica) | [108]     |
| 92      | Pi32(t) | 12       | 52.41–75.46   | IR64 (Indica) | [108]     |
| 93      | Pi157   | 12       | 49.5–62.2     | Moroberekan (Indica) | [123]     |
| 94      | Pita    | 12       | 42.41–42.43   | Tadukan (Indica) | [107]     |
| 95      | Pita-2  | 12       | 40.31–52.84   | Shimokita (Japonica) | [160]     |
| 96      | Pi19(t) | 12       | 35.30–53.67   | Aichi Asahi (Japonica) | [161]     |
| 97      | Pi39(t) | 12       | -             | Chubu 111 (Japonica) | [127,162] |
| 98      | Pi20(t) | 12       | 51.5–51.8     | IR24 (Indica) | [163]     |
| 99      | Pi20    | 12       | 49.6–50.4     | IR24 (Indica) | [164]     |
| 100     | Pi42(t) | 12       | 58.9–56.7     | DHR9 (Indica) | [151]     |
| 101     | Pi48    | 12       |               | Xiangzi 3150 (Indica) | [116]     |
| 102     | PiGD-3(t) | 12  | 55.8       | Sanhuangzhan 2 | [138]     |

The blast R gene, *Pi54*, cloned from indica rice Tetep, is of particular interest as it is one of the smallest reported blast R genes. *Pi54* confers broad spectrum, durable resistance against *M. oryzae* infection [95,165]. The functional role of *Pi54* in blast resistance was validated using transgenic and RNAi-mediated approach [165–169]. Subsequently, orthologs of *Pi54* gene; *Pi54rh* and *Pi54of*, also show broad spectrum and overlapping patterns of resistance against multiple strains of *M. oryzae* [89,170,171]. We also cloned and characterized *Avr-Pi54*; this effector interacts directly with Pi54 protein through LRR and non-LRR domain [39]. The *in silico* analysis predicted that variations in LRR domain of *Pi54* and its orthologs alter their interaction with the counterpart *Avr-Pi54* [89,172]. The major domain of interaction of *Avr-Pi54* with Pi54 and its orthologs is the non-LRR domain. A schematic diagram depicting the molecular mechanism of *Pi54* locus-mediated resistance is given in Figure 2. The predicted model for the mechanism of action of *Pi54* consists of the inactive OsRac1 protein bound to Sti1. The OsRac1 protein is activated by GEF molecule by interacting with the RhoGEF domain. The *M. oryzae*-derived chitin, a major PAMP, induces the activation of GEF, which further triggers OsRac1. Further, the PAMP
The tug-of-war between the blast fungus *M. oryzae* and the host plant rice is an ongoing process involving various chemical molecules. In a broader sense, the general resistance response mediated by PAMP-triggered immunity and the more specific effector-triggered ETI comprise of various signaling pathways. However, recently scientists stated that this model has failed to capture the stochastic process linking the initial detection of the tug-of-war to the defense response.

**3.2.3. Chemical Modulators**

The tug-of-war between the blast fungus *M. oryzae* and the host plant rice is an ongoing process involving various chemical molecules. In a broader sense, the general resistance response mediated by PAMP-triggered immunity and the more specific effector-triggered ETI comprise of various signaling pathways. However, recently scientists stated that this model has failed to capture the stochastic process linking the initial detection of the tug-of-war to the defense response.
the pathogen and action of pathogen effectors, PAMPs, and damage-associated signals. The rationality of a conceptual division between PTI and ETI at a molecular or cellular level in plants has also been questioned [178]. In the view of the above observations, very recently, the famous Zig-Zag model was extended and an improved model was reported by Ngou et al. [179]. Subsequently, the new ‘Circular Model’ of plant–pathogen interaction was proposed by Yuan et al., [180], and it is also schematically represented in Figure 3. The pathways underlying the resistance response largely have a common set of overlapping defense-response mechanisms mediated by various chemical molecules. The very process of onset of resistance response, which is initiated immediately after the penetration of pathogen into rice cells, leads to atypical burst in the ROS, which is a hallmark of PTI. The ROS, which were once considered as harmful molecules for cells, are the essential components of the signaling process, leading to cell death and resistance response. Besides, ROS is also involved in the reinforcement of the cell wall during blast pathogen infection [181,182]. Other pathogen-inhibitory metabolites that have been shown to inhibit M. oryzae include, cyanides, Bayogenin 3-O-cellubioside, and phytoalexins. Cyanide is reported to contribute to restricting blast fungal growth [183], whereas saponin bayogenin 3-O-cellubioside confers cultivar-independent resistance against the pathogen [184]. Phytoalexins such as momilactone, oryzalexin, sakuranetin, and phytocassanes are reported to be induced in rice plants in response to fungal infection and have a greater activity against M. oryzae [185,186]. Diterpenoid gene cluster (DGC7) coding for diterpenoids, a major group of phytoalexins in rice, was characterized for its role in disease resistance [187]. The rice DR gene ethylene insensitive 2 (OsEIN2) possibly activates phytoalexin production after infection with M. oryzae to promote resistance [188].

Among the chemical modulators, phytohormones have their own significance when it comes to disease resistance in plants. These hormones, mainly salicylic acid (SA), jasmonic acid (JA), and ethylene (ET) have a clear dichotomy, while regulating the defense response. Against biotrophic pathogens, the regulation of immunity is mediated by SA, whereas JA and ET are the key regulators of immune response against necrotrophs and insect pests [189]. Intriguingly, rice plants challenged with M. oryzae and leaf blight pathogen Xanthomonas oryzae pv oryzae show no elevation in SA levels, however, the external application of SA does induce immune response [189]. Besides, at least a couple of studies indicate that M. oryzae

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**Figure 3.** Schematic representation of the ‘Circular Model’ of plant–pathogen interaction. (This figure was originally drawn by Prof. Jonathan Jones, Sainsbury Lab, Norwich, UK, and it is being produced here with his permission).
manipulates the rice JA-signaling pathway and interfere with SAR [190,191]. In one study, *M. oryzae* was found to suppress transcription factor gene OsTCP21, a positive regulator of blast resistance, through targeted induction of miR319 [190]. Hence, *M. oryzae* could reduce the biosynthesis of JA in rice via inducing rice miR319. Secondly, *M. oryzae* modifies the rice active JA molecule to an inactive 12-OH JA, thereby denting the host-resistance response [192]. In rice, infection with *M. oryzae* represses the auxin signaling pathway in the distal healthy leaves by repressing the auxin-responsive genes such as ARF1 and IAA9 and thereby inducing systemic acquired resistance (SAR) against this pathogen [193]. On the contrary, the accumulation of auxins leads to rice susceptibility to *M. oryzae* [194]. Similar to auxins, cytokinins (CKs) were also found to be induced upon *M. oryzae* infection. Defense response genes *OsPR1b* and *PBZ* were induced by the CKs accumulated post-*M. oryzae* infection, and this resistance response is in synergy with the SA signaling pathway [195].

Unlike auxins and CKs, ABA has an antagonistic effect on disease resistance through the suppression of SAR mediated by SA, JA, and ET signaling pathways [196]. It was observed that a reduction or disruption of ABA signaling enhanced blast resistance, whereas exogenous ABA application enhanced the susceptibility towards *M. oryzae* in rice [193,197]. A schematic diagram depicting the role of reported phytohormones in rice blast disease response is presented in Figure 4.

![Figure 4](image-url)  
*Figure 4. Representation of role of phytohormones in rice during *M. oryzae* infection. (A) Auxin: Upon infection with *M. oryzae*, rice reduces the production of auxin and thereby induces SAR. However, the pathogen secretes auxins to counteract this host-induced SAR. (B) Cytokinin: The host CKs at higher concentration induce SA-mediated SAR. (C) Jasmonic acid: JA through induction of *OsJAR1* induces resistance, whereas *M. oryzae* counteracts this through activation of miR319, which suppresses the expression of rice OsTCP21 and blocks the SAR-induced resistance. (D) Abscisic acid: ABA generally has antagonistic effects on blast disease resistance. *M. oryzae* induces the expression of rice NCED3 gene and thereby ABA biosynthesis and increased susceptibility to pathogens by inhibiting SAR.*

### 3.2.4. Modulation of Coding RNA (mRNA) of Rice upon *M. oryzae* Infection

The R gene-mediated resistance in rice blast is well established. So far, eight R genes (*Pi9*, *Pi54*, *Pigm*, *Pi50*, *pi21*, *Pi7*, *Pi57*, and *Ptr*) have shown to confer broad spectrum blast resistance. Defense response (DR) genes also generally mediate broad-spectrum resistance to pathogens. Till now, seven DR genes or factors have been identified that positively and negatively regulate blast resistance. These DR genes mainly belong to TFs
and kinases. R and DR genes work in concert to induce resistance against *M. oryzae* [91]. Several other co-operations may exist among R and DR genes but are still to be explored. The information on prevalent race flora of *M. oryzae* is required to stack the correct R genes in rice lines. In the absence of this information, stacking R genes together with DR genes may prove more useful for better disease management [91]. Transcriptome of rice upon *Magnaporthe* infection at different time points has been studied extensively in different rice genotypes [198]. These studies mainly reveal the expression levels of different coding transcripts and their probable functions in different temporal and spatial conditions. The compatible and incompatible interactions of *M. oryzae* with different rice lines (resistant or susceptible) lead to the expression of different sets of coding transcripts.

The majority of studies on rice transcriptomes upon *M. oryzae* infection provide a comparative picture of changes in expression level of DR genes in compatible and incompatible interactions. Transcriptome studies of rice NILs differing in a single-blast R gene give a clear picture to understand the mechanism of R–Avr and DR gene-mediated resistance. Comparative studies of NILs carrying different blast resistance genes (*Pi9*, *Pi54* and *PiI*) compared to susceptible control have shown that the transcripts related to cell wall structure (suberin biosynthesis) and secondary metabolite (JA biosynthesis, salicylate biosynthesis, 13-lox and 13-hpl, divinyl ether biosynthesis, and phenylpropanoid biosynthesis) were differentially expressed in all three NILs upon *M. oryzae* infection [199]. The study revealed that the NILs containing different blast resistance genes in a common background have some common set of functionally important transcript whose expression changes upon *M. oryzae* infection. Similarly, each NIL had a unique set of transcripts that change their expression upon blast pathogen infection. Further, at 24 hpi with *M. oryzae*, a significant change was recorded in the expression of genes related to biosynthesis of trehalose, flavonoids, aminopropanol, cellulose, UDP-D glucarate, xylose, serine and choline, cyclopropane FA and Cyclopropene FA, phaseic acid, and phytoctassane. Whereas at 72 hpi, a significant change in the expression levels of genes involved in triacylglycerol biosynthesis, flavonoids biosynthesis, GDP-mannose metabolism, trehalose biosynthesis-I, stachyose biosynthesis, mannose degradation, and nitrate reduction was recorded [199,200]. Transgenic lines help in understanding the basic mechanism of disease resistance by over expressing the genes responsible for disease resistance. During early stages of infection, rice cell wall offers the first level of a physical barrier to *M. oryzae* appressorium. So, genes related to cell wall modification show significant change in their expression upon pathogen infection. Callose (1, 3-β-glucan) and lignin are important components of the plant defense response and are involved in the blockage of plasmodesmata, thus posing a physical barrier for the penetration of fungal mycelia into nearby plant cells [198]. Two genes coding for callose biosynthesis were very highly upregulated in transgenic rice line TP-Pi54 in comparison to non-transgenic control lines upon challenge with *M. oryzae* [182]. In the case of *Pi54*, the role of callose in rice blast resistance has already been reported [165].

During the early stages of infection, enzymes such as beta-glucanase and chitinase enhance the resistance against fungus by catalyzing the hydrolytic cleavage of glycosidic linkages in β-glucans present in the fungal cell wall [201]. In rice, it was reported that beta-glucanase is highly induced in response to *M. oryzae* infection [202,203]. In the incompatible interactions [174,182], class III peroxidase gene was found to be highly up-regulated after blast pathogen infection [182,200]. Peroxidases are found to be involved in lignin biosynthesis [204] and cross-linking of cell-wall components, thus fortifying the cell wall against pathogen infection. Redoxin, glutathioredoxin, peroxidase, and catalase are major ROS-scavenging enzymes that fine tune ROS signaling and restrict ROS-dependent damage [205]. In both compatible and incompatible interactions, changes in the expression level of genes involved in cell wall biosynthesis, cell wall modulation, and cell wall degradation have been observed with a number of upregulated genes being higher in incompatible interaction compared to compatible interaction [198]. The studies suggest that during the initial phase of interaction with *M. oryzae*, rice plants enforce the defense mechanism by cell wall
fortification in both resistant and susceptible rice lines [198]. Once the primary defense mediated by cell wall reinforcement is breached, the next level of defense response is induced. Transgenic rice line Pi21-RNAi showed changes in the expression levels of 43 receptor kinases (belonging to WAK, L-LEC, LRR, CAMK, MEKK, LRK10L-2, SD, DUF26, RLCK, and CR4L subfamilies) associated with PAMP recognition and calcium ion influx [206]. These kinases are involved in perceiving internal and external signals. As a result, a more robust PTI was observed in the Pi21-RNAi line compared to susceptible control, and 53 TF genes (WRKY, NAC, DOF, and ERF families) were observed to be differentially expressed in the Pi21-RNAi line. Cell wall-associated kinases participate both positively and negatively in basal defense against rice blast fungus [207]. The BAK1 is a coreceptor of receptor-like kinase RLKs [208] and acts as a central regulator in PTI [209]. During the later stages of fungal infection, MAPK (mitogen-activated protein kinase) cascades play an important role in downstream signaling processes leading to ETI. Receptor kinase and MAPK act as signaling molecules in both PTI and ETI [198–200]. As a part of ETI, several TF genes are induced. WRKY genes are one of the major families among them, and many of them such as WRKY76, WRKY47, WRKY45, WRKY55, WRKY53, WRKY62, and WRKY71 are induced in rice upon M. oryzae infection [201,210,211]. Similarly, differential expression of 53 TFs such as WRKY, NAC, DOF, and ERF families, and 62 PR genes was recorded between Pi21-RNAi and Nipponbare [51]. The role of WRKY genes in the activation of several PR genes was also reported [210,212]. A higher number of WRKY genes is reported to be upregulated in blast-resistant genotype GV (WRKY 45, WRKY79) and near-isogenic line-carrying blast-resistance gene Pi9 compared to susceptible control [213,214]. Phytohormones play a significant role during compatible and incompatible rice–M. oryzae interactions. JA–ET and SA pathways act synergistically in pathogen-triggered immunity (PTI) response, while ETI uses the JA-ET pathway when SA signaling is not present in the plant [215]. Several reports confirm that changes in JA expression act as a powerful mediator of resistance against M. oryzae [216,217]. A high upregulation of genes involved in ET and JA metabolism has been reported in incompatible rice–M. oryzae interactions [6,199]. A common upregulation of transcripts involved in JA biosynthesis was observed in NILs carrying blast-resistance gene Pi9, Pi54, and Pi1 following M. oryzae inoculation [199]. A higher level of upregulation of lipoxygenase (Lox), a gene involved in JA biosynthesis, was reported in resistant lines compared to susceptible control [51,199,200]. Similarly, an upregulation of genes related with ethylene, salicylic acid (SA), and jasmonic acid (JA) signaling was reported in incompatible interactions involving M. oryzae [203]. Genes governing secondary metabolism are another set of genes that are upregulated in resistant and susceptible rice lines after M. oryzae infection. A large number of enzymes that are part of phenylalanine and shikimate biosynthesis as well as downstream phenylpropanoid biosynthesis show upregulation in resistant Pi9 NILs [199,214] and transgenic lines carrying the Pi54 gene [182]. The phenylpropanoid pathway plays an important role in the rice–M. oryzae interaction, because phenylpropanoids are important antimicrobial compounds. This pathway is involved in synthesizing lignin and phytoalexins that prevent pathogen invasion of the host cells. Rice plants accumulate diterpene, phytoalexins, phytocassanes, oryzalexins, and momilactones to counteract M. oryzae invasion [218,219]. The important genes involved in the biosynthesis of diterpene phytoalexin, momilactone, and phytocassanes are reported to be upregulated in the blast-resistant genotype after M. oryzae infection [213]. The diterpene phytoalexin biosynthetic (DPB) gene OsKSL8 (LOC_Os11g28530; oryzalexin S synthesis) shows higher expression in the resistant than in the susceptible genotype after M. oryzae infection [213]. A higher accumulation of phytoalexins Momilactone A and Sakuranetin has been observed in incompatible compared to compatible interactions [213]. These phytoalexins inhibit germination of M. oryzae spores in infected leaves [220–222], thus suggesting the importance of genes of diterpene phytoalexin biosynthesis in resistance to M. oryzae.

A comparison of the rice–M. oryzae interaction transcriptome from our laboratory with previous six studies indicated that the cytochrome P450 gene was commonly upregulated
in all these studies. We also observed that the transcriptome of rice varies quantitatively rather than qualitatively during incompatible and compatible interactions with functional categories of differentially expressed genes remaining similar in both the cases [198].

3.2.5. Small and Long Non-Coding RNA Play a Regulatory Role in Rice upon *M. oryzae* Infection

Small non-coding RNA include microRNA (miRNA), small interfering RNA, piwi-interacting RNAs, trans-acting siRNAs, and natural antisense transcript siRNAs. In rice, miRNAs are well known to control many important agronomic traits such as tiller development, flowering time, panicle establishment, grain formation, and yield production, and also regulate a plant’s response to both abiotic and biotic stresses [223,224]. Few conserved novel miRNAs and miRNAs families have been found to be responsive to blast fungal elicitors, which suggests their possible involvement in rice–*M. oryzae* interaction [225]. Among eight rice DCL genes, till now only OsDCL1a has been studied and was found to negatively regulate PTI against *M. oryzae*, while the knock-down of OsDCL1a enhanced resistance to the blast disease [226]. Four miRNA families, namely miR160, miR166, miR398, and miR7695 have been reported to act as positive regulators of rice immunity against *M. oryzae* [227]. OsDCL1s is known to act as a key molecule in the miRNA signaling pathway that mediates cross talk between rice-*M. oryzae* interaction and miRNA network. Our group recently deciphered the role of miRNAs in Pi54 mediated incompatible interaction with blast pathogen. Upregulated expression levels of miR164c, miR164e, miR1849, miR1854-3p, miR2925, miR936c-5p, miR936c-3p, and miR812f have been found in the resistant line in comparison to susceptible control [182].

3.3. Interplay between Rice-*M. oryzae*: A Classical Example for Plant-Pathogen Interactions

Antagonistic interactions between rice plants and the fungus, *M. oryzae*, result in ‘arms races’. While the plant attempts to recognize the pathogen and subvert its growth and spread, the pathogen tries to subvert recognition and suppress the host responses [228]. Several hypothesis and models were proposed to explain the race that underpins disease resistance. We discuss here three models: Gene-for-gene (GFGM) model, Guard model (GM), and Decoy model (DM), with diagrammatic representation (Figure 5).

![Figure 5](image_url)  
**Figure 5.** Simplified diagrammatic representation of models on host disease-resistance mechanism. Gene for gene model (GFGM): Physical interaction of pathogen-derived PAMP (pathogen associated...
molecular pattern) with DR protein (pattern recognition receptor, PRR), resulting in PAMP-triggered immunity (PTI); physical interaction of Avr (effector) with R protein (NBS-LRR), resulting in effector-triggered immunity (ETI); Guard Model (GM): Interaction of effector with guardee triggers effector-triggered immunity (ETI) by their interaction with R protein (NBS-LRR). However, in the absence of R protein, binding of guardee with effector enhances the susceptibility/fitness of the pathogen (ETS); Decoy Model (DM): Interaction of effector with decoy triggers effector-triggered immunity (ETI) with the interaction of R protein (NBS-LRR), however, in the absence of R protein, there is no increase in the virulence/fitness of the pathogen.

3.3.1. Gene-for-Gene Model

The gene-for-gene concept, initially discovered in the flax-rust pathosystem by Harold Henry Flor [229], essentially describes an interaction between a matching gene in a host (called resistance \( R \) gene) and pathogen (called avirulence or \( Avr \) gene) that interact physically in a receptor–ligand mode to initiate the defense response. \( R \)-genes and their structure were described earlier [230,231]. The majority of the \( R \)-genes harbor signature nucleotide binding site—leucine rich repeat (NBS-LRR) or pattern recognition receptors (PRR). \( Avr \), on the other hand, can be effectors, the pathogen-secreted proteins that manipulate host cell functions, or molecular motifs conserved across many pathogen species known as pathogen-associated molecular patterns (PAMPs). LRRs (approximately 24 amino acid motifs) are principally involved in protein–protein (R–Avr) interaction, and the greatest variation in this class emanates from the LRR domain. The classic examples of R–Avr interaction include pairs of R–Avr proteins such as Pi54 and AvrPi54 [39], and Pi-ta and Avr-Pita [184]. PRR, on the other hand, recognize generic motifs of the pathogen and initiate reaction, e.g., rice \( Xa21 \)-resistance gene recognizes the tyrosine-sulphated protein, RaxX [232]. Currently, more than a dozen examples of direct physical interactions are available, and interestingly, other domains are reported to interact physically to initiate defense response [89].

3.3.2. Guard Model

The Guard Model envisages an indirect physical interaction between R and effector proteins. The model predicts that R proteins act by monitoring/guarding the indispensable effector target (called guardee) and any modification of this target by the effector results in the activation of the R protein [233]. This indirect perception could explain how multiple effectors could be perceived by a single R protein, thus enabling a handful of \( R \) genes to provide protection against a great variety of pathogens. Classical examples of the hypothesis are tomato RCR3 and Pto and \( Arabidopsis \) RIN4 and PBS1 [133,234]. In rice, three gene pairs coding for CC-NLRs are understood to act in a Guard Model. These genes include \( RGA4/RGA5, Pik-1/Pik-2, \) and \( Pi5-1/Pi5-2 \) [44,235–238]. The proteins RGA4/RGA5 is necessary for AVR-Pia- and AVR1-CO39-induced and Pia- and Pi-CO39-mediated blast resistance in rice. Similarly, Pikh-1 proteins act as guardees and perceive AvrPik-h protein, which further induces Pikh-2-mediated resistance response.

3.3.3. Decoy Model

The Decoy Model envisages that the arms race of host–pathogen interaction leads to the evolution of ‘decoys’, which mimics effector targets to trap the pathogen into a recognition event, but itself has no function either in the development of disease or resistance. Decoys might evolve from effector targets by two ways: (a) gene duplication and evolution and (b) mimicking effector targets (target mimicry) [177,234]. The current understanding suggests that these interactions drive the evolution of ‘decoys’ in pathogens as well. Interestingly, decoys undergo a similar manipulation as the component they mimic, but play an opposite role, either by preventing manipulation of the component they mimic or by triggering a molecular recognition event. Three different types of decoy are reported: (a) Receptor decoys: mimics to absorb ligands, (b) bodyguard decoys: protecting secreted virulence factors, and (c) sensing decoys: mimics effector targets acting as coreceptors with
two different modes: sponge and bait [239]. Currently, there are few cases illustrating decoy mechanisms, however, much remains to be discovered. The discovery of more decoys shall enhance our understanding of disease resistance and provide a means to improve host immunity e.g., decoy engineering of PBS1 in Arabidopsis plants confers a broader resistance spectrum [240]. Interestingly, a putative decoy protein, zinc finger–BED type (ZBED), was investigated for its role in rice resistance and susceptibility to the blast fungus [241]. Similarly, the RATX1 domain of RGA5 acts as a decoy domain for recognizing the M. oryzae effector AVR-Pia [242].

4. Resistance Response of Rice to Blast Disease

4.1. Resistance Response Based on Quantitative Trait Loci (QTL)

Resistance to rice blast in some cases is known to be inherited as quantitative trait regulated by many genes. Contemporary approaches that are employed for other polygenetic traits are also applicable for studying the quantitatively inherited blast resistance. The basic QTL mapping approaches such as single marker analysis (SMA), simple interval mapping (SIM), and interval mapping approaches (IM) have been extensively used for detecting major and minor QTLs linked with complete/partial resistance to rice blast [108,243]. Mapping genomic regions for quantitative blast resistance has gained importance due to race non-specificity of the resistance, which provides stable protection against the pathogen [244]. For the first time, blast-resistance QTL was identified in a widely grown African variety, Moroberekan [143]. There are also instances in which the blast resistance has been shown to be regulated by race-specific resistance genes such as Pi34, Pi5, Pi21, and Pb1. [146,245–248]. To date, more than 500 QTLs controlling resistance to blast disease in rice have been identified and mapped on different rice chromosomes [91]. Most of these QTL were identified from bi-parental populations derived from japonica and indica subspecies, with the assistance of various marker systems such as RFLP, SSR, and SNP [249]. The QTLs exhibiting modest individual effect with race non-specific or broad-spectrum resistance provide durable resistance [250]. Thus, it is important to identify and use novel QTLs with broad spectrum resistance for the development of resistant cultivars.

4.2. Resistance Gene Mediated Resistance

4.2.1. The Blast Resistance Genes Identified, Mapped, and Cloned in Rice

Deploying host plant resistance for disease management is an eco-friendly and most viable approach to manage the disease as a wide range of resistance alleles are available in diverse rice germplasms [123]. In rice, nearly 100 rice blast resistance genes have been identified and mapped in rice till date (Table 3, Figure 6) [3]. Among the identified resistance genes, 38 have been cloned and are known to encode proteins with nucleotide-binding sites (NBS) and leucine-rich repeat (LRR) domains (Table 4) [101,251]. Exceptions include Pi-d2, which encodes a B-lectin kinase domain protein, pi21 that encodes a proline-rich protein with a heavy metal domain, and Pdr that encodes an atypical protein with an armadillo repeat [4,101]. Pik, Pikm, Pik-p, Pi1, Pike, Pi5, Pia, and Pi-CO39 contain two NBS-LRR protein structural genes for blast resistance. Pi5-1, Pb1, pi21, and Pi63 genes are induced by pathogen infection, while the remaining genes express constitutively. The majority of the cloned R genes induce resistance against leaf blast at the seedling stage, while only a few R genes, such as Pb1, Pi25, Pi64, and Pi68 confer resistance to panicle blast [252–255]. Most of the identified blast resistances are clustered on chromosomes 6, 11, and 12 (Figure 6), while cloned blast resistance genes are clustered on chromosome 6 and 11 (Figure 7). Notably, a large number of R genes (28 R genes) are mapped on to chromosome 11, and chromosomes 3, 7, and 10 carry solitary R genes, respectively. The genes Pi2, Pi54, Pi19, Pigm, and Pi7t mapped on to chromosome 6 are known to provide broad spectrum resistance, and so is Pi54, which is mapped on to chromosome 11. A great majority of blast resistance genes are distributed into clusters of tightly linked genes. At least three major clusters of blast resistance genes have been detected in rice on chromosomes 6, 11, and 12 (Figure 6). Several studies have demonstrated that the genetic control of blast resistance is complex and
involves both major and minor genes with additive/complementary interactions [256]. To be ahead in the evolutionary race between pathogen and host, there is a need for continuous identification of new $R$ genes from the previously untapped genetic resources [177].

![Figure 6. Rice blast resistance genes identified and mapped on to different rice chromosomes.](image)

**Figure 6.** Rice blast resistance genes identified and mapped on to different rice chromosomes.

![Figure 7. Cloned and characterized blast resistance genes on different chromosomes.](image)

**Figure 7.** Cloned and characterized blast resistance genes on different chromosomes.
Table 4. The cloned and functionally characterized rice blast resistance genes.

| Genes & Alleles | Encoded Protein | Chr. No | Cognate AVR Gene | Chromosomal Location | Donor | Reference |
|-----------------|-----------------|---------|-------------------|----------------------|-------|-----------|
| Pish            | NLR             | 1       | -                 | 33,136,846–33,145,541| Nipponbare | [257]   |
| Pi35            | NLR             | 1       | -                 | 33,838,140–35,206,760| Hokkai 188 | [111] |
| Pi37            | NLR             | 1       | -                 | 33,116,117–33,124,371| St. No. 1 | [112] |
| Pi64            | NLR             | 1       | -                 | 33,098,072–33,104,550| Yangmaogu | [254] |
| Pi-t            | NLR             | 2       | AVR-Pib           | 35,979,234           | K59    | [258,259]|
| P21             | Proline-rich metal binding protein | 4 | - | 19,836,301–19,835,131 | Owarihatamochi | [96] |
| Pi63            | NLR             | 4       | -                 | 31,553,065–31,558,406| Kahei   | [260] |
| PiPR1           | NLR             | 4       | -                 | 316,00,121–31,604,201|         | [261] |
| Pi9             | NLR             | 6       | AVR-Pi9           | 2,410,176–2,418,568  | 75-1-127 | [37,133]|
| P2              | NLR             | 6       | -                 | 1,043,5816–10,441,907| Jefferson | [262] |
| Piz-t           | NLR             | 6       | Avr-Pizt          | 10,387,509–10,390,465| Zenith  | [36,262]|
| Pi50            | NLR             | 6       | -                 | 10,375,846–10,380,263| Er-Ba-zhan (EBZ) | [97] |
| Pizk            | NLR             | 6       | -                 | 10,087,244–10,478,622|         | [263] |
| Pigm            | NLR             | 6       | -                 | Near to 10,435,816–10,441,907 | Gumei4 | [100,264] |
| Pi-d2           | B-lectin receptor kinase | 6 | - | 17,164,851–17,160,330 | Digu | [137] |
| Pi-d3           | NLR             | 6       | -                 | 13,058,027–13,055,162| Digu    | [265,266]|
| Pi25            | NLR             | 6       | -                 | 13,058,027–13,055,162| Gumei2  | [253] |
| Pi3-3-A4        | NLR             | 6       | -                 | 13,058,027–13,055,162| A4 (Oryza rufipogon) | [267] |
| Pi36            | NLR             | 8       | -                 | 2,878,953–2,890,634  | Kasalath | [268] |
| Pi5             | NLR             | 9       | -                 | 9,674,695–9,674,000  | RIL260  | [236] |
| Pii             | NLR             | 9       | AVR-Pii           | 9,674,695–9,674,000  | Hitomebore | [16,269] |
| Pi56            | NLR             | 9       | -                 | 9,777,527–9,780,698  | Sanhuangkan No. 2 | [270] |
| Pi-b            | NLR             | 11      | -                 | 14,705,215–14,714,572| Modan   | [252,271]|
| Pik             | NLR             | 11      | AVR-Pik           | 27,984,697–27,989,134| Kusabue | [16,272]|
| Pik-p           | NLR             | 11      | AVR-Pikp          | 27,978,568–27,980,621| K60     | [237] |
| Pikm            | NLR             | 11      | AVR-Pikm          | 27,984,697–27,989,134| Tsuyuake | [235] |
| Pike            | NLR             | 11      | -                 | 27,984,697–27,989,134| (Pik allele) | Xiangzao143 | [273] |
| Pik-h           | NLR             | 11      | -                 | 27,984,697–27,989,134| (Pik allele) | K3 | [238] |
| Pii             | NLR             | 11      | -                 | 27,984,697–27,989,134| (Pik allele) | C101LAC | [274] |
| Pi5             | NLR             | 11      | AVR-Pi5           | 25,262,834–25,264,520| Tetep   | [39,275]|
| Pi54rh          | NLR             | 11      | Avr-Pi54          | 25,262,834–25,264,520| (Pi54 allele) | Oryza rhizomatis (ncrp 002) | [171] |
| Pi54sfof        | NLR             | 11      | AVR-Pi54          | 25,262,834–25,264,520| (Pi54 allele) | Oryza officinalis (ncrp0004) | [89] |
| Pia             | NLR             | 11      | AVR-Pia           | 6,546,026–6,541,924  | Sasanishiki | [16,244,276] |
| Pi-CO39         | NLR             | 11      | AVR-CO39          | 6,888,057-6,291,466  | CO39   | [142,277]|
| Pi-ta           | NLR             | 12      | AVR-Pita          | 10,612,068–10,606,359| Yashiro-mochi | [32,162] |
| Pi65            | LRR-RLK         | 12      | -                 | 28,376,327–28,379,731| GangYu129 | [278] |
| Ptr             | ARM repeat domain protein | 12 | - | 10822534–10833768 | M2354 | [101] |

NLR, nucleotide-binding leucine-rich repeat; AVR, avirulence; Chr. No, Chromosome number.
4.2.2. Resistance Response Mediated by Alleles of Known R Genes

The deployment of cultivars introgressed with resistance (R) genes is most effective and eco-friendly approach to control blast disease [4]. Among the 38 cloned and characterized blast R genes (Table 4), except for pi21, which is a recessive R gene, the remaining 37 are dominant genes. Most of the cloned blast resistance genes are alleles of the previously cloned rice blast R genes and few of them represent a new rice blast R locus [260,279]. Considering that more than 400 NBS–LRR gene sequences are identified in a rice genome and alleles of rice blast R genes may confer distinct resistance spectra to M. oryzae isolates, allele mining of cloned rice blast R genes in rice germplasms would reveal more favorable R alleles for rice blast resistance breeding [89,280]. TILLING (Targeting Induced Local Lesions in Genomes) and PCR based mining are the two main approaches for the identification of better, new and superior alleles of disease resistance genes [281,282]. Natural mutations such as transition, transversion, point mutation, and insertion, and deletions (InDels) are the main driving forces for the generation and evolution of new alleles. With the availability of enormous database information, desired and superior alleles can be easily identified and retrieved [282]. The allele mining approach identifies new haplotypes and evolution patterns of R genes [282]. The superior allelic variants with novel resistance specificities can be deployed in breeding programs to achieve broad spectrum resistance to blast.

Many studies have reported allele mining of blast resistance genes from wild and cultivated species of rice [170,283–286]. Studies of Pi-ta gene in wild (AA and CC genome) and cultivated species of rice indicated a consensus conserved sequence before divergence [287]. In another study, Pi-ta orthologs identified in 26 rice accessions from 10 different countries were shown to display a dimorphic pattern of nucleotide polymorphism and low nucleotide diversity at the LRD region of the orthologs [16]. To study allelic variants and flanking sequence of Pi-ta, 159 geographically diverse accessions of Oryza species (AA genome) have been used [288]. The Pi-ta and Pi9 alleles have been studied extensively in Indian landraces [289,290]. Five unique and novel Pi-ta variants were identified from local landraces of rice. Notably, strong selective sweeps as indicated by the high value of Pi (non/syn) on the LRD were inferred to shape the evolution of the new alleles at Pi-ta locus [290]. Other blast resistance loci such as Pid3 and Pi9 have been explored to study the nucleotide polymorphism and evolutionary pressure [265,291]. A nucleotide polymorphism study of the Piz-t locus of Indian landraces indicated positive selection pressure on the locus and the role of diversification of the LRR domain in the evolution of a gene [292]. PCR-based allele mining for blast resistance gene Pi54 from six cultivated rice lines and eight wild rice species was undertaken to understand its structural variation and its impact on the phenotypes. A high nucleotide variation was recorded between cultivated and wild species (35–90%) compared to variation in cultivated species (1–20%) [177]. The Pi54 allele mining in 92 rice lines indicated an extensive variability in the allelic sequences and unique haplotypes linked to resistance alleles. InDel polymorphisms in the allelic variants have been targeted for developing markers for the identification of better allele(s) and their introgression in commercial rice cultivars, employing marker-assisted selection [293]. Similarly, the allelic diversity of Pi54 gene has also been studied in 885 Indian rice laces that have shown resistance to naturally existing pathogens as well as against 5 unique strains of the blast pathogen. Nine new alleles of Pi54 were identified based on the sequence comparison to the Pi54 reference sequence as well as to already known Pi54 alleles [279]. Allelic mining of the blast resistance Pid3 locus in 3000 rice genome project (3 K RGP) genomes revealed that most japonica rice accessions harbored pseudogenes due to premature stop mutations, while Pid3 alleles in most of the indica rice accessions were identical to the functional haplotype, which had a similar resistance spectrum as the previously reported Pid3 gene [294]. In another study, 13 novel alleles of Pi9 were identified based on tandem-repeat regions from 361 resistant rice varieties [295].
5. Molecular Mechanisms of Leaf and Panicle Blast

Blast infects the rice crop at all stages of its growth, starting from the nursery to the grain filling stage, under favorable environmental conditions. Blast pathogen likes leaf wetness, high humidity, longer free moisture periods, a night temperature between 18–24 °C, and an absence of wind at night time. Fungal spores are produced and spread under high relative humidity conditions, however, no spore production is observed below 89% relative humidity. The sporulation process increases with relative humidity above 93%. The most appropriate temperature for spore germination, lesion formation, and sporulation is 25–28 °C [296].

Of the two commonly recognized phases of the disease, the leaf blast occurs during the plant’s vegetative stage, while the neck blast (a near synonym of panicle blast) appears during the reproductive stage. The flow of photosynthates to growing grains is blocked at the base of the panicle during neck blast infection, resulting in chaffy grains or empty panicles. Under epidemic conditions, the damage inflicted by neck blast infection could be twice as severe as leaf blast with losses approaching up to 70% of the anticipated yield [297]. Although more than 100 R-genes for leaf blast resistance are known, very few genes for resistance to neck blast have been identified and located on the rice genome. The disease response of leaf and panicle to blast infection is different, and the varieties that are susceptible to leaf blast are resistant to neck blast and vice versa [297,298]. Some of the cultivars resistant at the seedling stage become susceptible to neck blast [299]. The reported susceptibility of leaf blast resistance genotypes to neck blast and vice versa has suggested that the different genes are involved in resistance to leaf and neck blast [297,300]. In other studies, the gene/QTLs for panicle blast resistance were mapped to the genomic locations harboring major leaf blast resistance genes, thereby suggesting the existence of common genes for resistance to both phases of the disease [301]. Pb1 gene mapped on the long arm of chromosome 11 in an indica cultivar ‘Modan’ is the first panicle blast resistance gene to be identified from rice [147]. The gene was introgressed into several varieties in Japan and has shown durable resistance to blast for almost 30 years [252]. The gene exhibits lower expression levels at the seedling stage but its expression reaches peak during full-heading stage, thus accounting for its strong resistance to panicle blast. These findings have been taken to reflect that the temporal and spatial expression pattern of a blast resistance gene is a major factor in deciding whether the gene will offer protection to leaf or neck blast or both phases of the disease [302]. Zhuang et al. [156] identified a blast resistance gene Pib25(t) that provides resistance to both leaf and neck blast. Ma et al. [254] identified a resistance gene Pib64, which is constitutively expressed in all the tissues and provides protection against both leaf and neck blast. Noenplab et al. [301] reported co-localization of QTLs for leaf and neck blast resistance on the same genomic regions on chromosomes 1, 11, and 12.

Ishihara et al. [303] identified a major QTL, qPbm11, for panicle blast resistance in the genomic region on chromosome 11, from where panicle blast resistance locus, Pb1, has previously been identified in cultivar Modan. However, the absence of Pb1-encoded transcripts in the panicles of qPbm11 genotype Miyazakimochi has suggested that the qPbm11 is different from Pb1. Fang et al. [304] identified a panicle blast resistance QTL, qPhb-11-1, located on the long arm of chromosome 11. The gene occupies a different genomic position compared to two panicle blast resistance loci Pb1 and qPbm11 previously identified from the same chromosome. The gene expression studies have suggested that the genes such as Pb1 that are expressed during the heading stage shall display neck and panicle blast resistance, while those showing constitutive expression such as Pib64 are expected to provide protection against both the phases of the disease. These studies have provided a plausible explanation for observed inconsistencies in the reaction of leaf blast resistance varieties to neck blast and vice-versa, which were previously ascribed solely to either shifts in race composition of the pathogen or changes in environmental conditions during the crop season.
6. Management of Blast Disease Using Host Resistance

6.1. Introgression of QTLs for Blast Resistance

The introgression of R and QTLs genes in rice is considered to be one of the best ways to control blast disease [305]. Therefore, deploying single or multiple QTLs that impart the partial resistance and non-race specificity is a priority in rice breeding nowadays [306,307]. Though there are more than 500 reported QTLs for blast resistance, only few of them have been deployed under field conditions. There are various reports in which QTLs have successfully been transferred in rice to achieve broad spectrum resistance against rice blast disease. Pyramiding of resistance QTLs in cultivated varieties has been practiced to increase disease resistance levels, e.g., two QTLs (qBl1 and qBl11) identified from the rice cultivar Jao Hom Nin (JHN) were introgressed into the Thai glutinous jasmine rice cultivar RD6 MAB [308]. Using the MAS approach, elite indica rice lines were developed by bringing together multiple QTLs from IR64 and JNJ into a single background, and resulting lines have shown broad spectrum resistance against Thai blast isolates [309]. A new glutinous rice variety was developed by pyramiding Sub1, badh2, qBl1, and qBl11 loci from the rice lines IR85264 (Sub1), TDK303 (badh2), and RGD07529 (qBl1+qBl11) into a single background [310]. Further, Fukuoka et al. [311] combined the partial resistance genes and QTLs (pi21, Pi34, qBR4-2, and qBR12-1) to enhance the blast resistance in rice. Suwannual et al. [312] used four blast resistance QTLs from two rice lines for the development of pyramided broad spectrum blast resistance rice lines of a popular variety RD6. The RD6 introgression lines carrying a high number of QTLs displayed broad-spectrum resistance to prevalent blast pathogen races. Recently, the introgression of a major QTL qBL3 for leaf and neck blast resistance into a susceptible rice variety BPT5204 resulted in progeny lines showing field resistance to leaf and neck blast [123].

6.2. Introgression of R-Genes for Blast Resistance

Of the various means available to curb the blast disease, breeding resistance varieties is the most suitable, ecologically safe, and cost-effective strategy. Since the resistance to blast in rice involves gene-for-gene interactions, the varieties carrying single R genes often succumb to disease due to the appearance of virulent races of the pathogen, which is due to the mutation of avirulence gene to evade detection by the corresponding host R-gene [313]. Pyramiding of multiple R-genes has been advocated to foster enduring resistance to blast [314]. Genetic mapping and molecular cloning of different blast resistance genes has provided a gamut of linked or gene-based markers for the efficient selection of resistance genes in breeding programs. Several blast resistance genes have been deployed in rice using different genomics-assisted approaches for achieving long-lasting resistance to blast (Table 5). Of the various R genes, the Pi54 gene cloned from rice line Tetep in our laboratory [95] has been deployed more extensively, both globally and in India, in combination with other blast resistance genes for achieving durable resistance to blast (Table 6).

6.3. Transgenic Approach for Blast Management

The transgenic approach is one the important components in rice blast disease management. The initial cloning and characterization of the R genes was performed using the transgenic approach by expressing these genes in susceptible rice lines (Table 3). As discussed in details in the earlier section, 38 blast R were transformed into different rice lines. Although large-scale field release of these transgenic lines is not reported, the developed lines are a valuable resource for deployment whenever regulatory approval is given. Among the 38 cloned blast R genes, Pi54 and its orthologs are widely studied using different methods through transgenics (Table 6).
### Table 5. Resistance genes introgressed in rice for blast improvement program.

| Target Gene | Recipient Parent | Chromosome | Marker Used | Reference |
|-------------|------------------|------------|-------------|-----------|
| Pi2         | C815S            | 6          | RM527       | [315]     |
| Pigm        | Kongyu 131, Longjing 26, Kenjiandao 6 | 6 | M80362 | [316] |
| Pigm        | KT27S            | 6          | G8900       | [316]     |
| P9          | E32              | 6          | In2-3       | [317]     |
| P9          | R288             | 6          | Clon2-1     | [318]     |
| P9          | QZ11S            | 6          | Nbs21       | [319]     |
| P40         | Osancik-97, Halilbey | 6 | 9871.T7E2b | [320] |
| Pi1         | BPT5204          | 11         | RM224       | [321]     |
| Pi54        | BPT5204          | 11         | Pi54MAS     | [322]     |
| P1          | R1, R2           | 11         | RM224       | [323]     |
| Pi54        | MTU1010          | 11         | Pi54MAS/RM206 | [324] |
| P46/Pi-ta   | Hanghui 179      | 11, 12     | RM224, YL155/YL87 // YL155/87 | [325] |
| P1/Pi54/Pi-ta | Mushk Budji     | 11, 12, 12 | Pi54MAS, RM224, YL155/YL87 // YL155/87 | [323] |
| Pi/b/Pik    | K6415            | 2, 11      | N5b, K6415  | [326]     |
| Pi/b/Pi54   | MR219            | 2, 11      | RM208, RM206 | [107] |
| P1/Pi2     | GD-75            | 6, 11      | RM144, AP22 | [327]     |
| P1/Pi2     | Pusa RH-10       | 6, 11      | RM5926, AP5659-5 | [326] |
| P1/Pi2, P1/Pigm | GZ63S, 97S, R084, R609 | 6, 11 | RM224, ZJ58.7, AP22 | [328] |
| P2/Pi54    | PB1121           | 6, 11      | AP5659-5, RM206 | [329] |
| P2/Pi54    | PRR78            | 6, 11      | AP5930, RM206 | [330] |
| Piz-t/Pi54, P9/Pi54 | 07GY31      | 6, 11      | Z4794, Pkh-1 | [331] |
| P2/P1/Pi54  | Swarna-Sub1      | 6, 11      | Pi54MAS, RM224, AP5659-5 | [332] |
| P1/P2/D12  | Jin 23B          | 6, 11, 12  | RM144/RM224, P12-4/HC28, RM277/RM309 | [333] |
| P9/Pi-ta   | Pusa Basmati 1   | 6, 12      | AP5659-5, YL155/YL87 // YL155/87 | [334] |
| P1/P2/P33  | Kuboyar          | 6, 8, 11   | RM224, RM527, RM310 | [335] |
| P1/P2/P33  | ADT43            | 6, 8, 11   | RM224, RM527, RM25 | [336] |
| P2/Pi54    | Sambha Mahsuri   | 6, 11      | Pi54MAS, AP5659-5 | [337] |

### Table 6. Applications of Pi54 gene in developing blast resistance rice.

| Country       | Approach          | Applications                               | Cultivar Developed       | Reference |
|---------------|-------------------|--------------------------------------------|--------------------------|-----------|
| China; Beijing| MAS-Gene pyramiding | Pi9, Pi1, and Pi54 for blast resistance | NILs                     | [331]     |
| China; Yangzhou| MAS-Gene pyramiding | Combination of major R genes including Pi54 for blast resistance | NILs | [338] |
| China; Wuhan | MAS-Gene pyramiding | Pi54, Pi37, Pi4, Pi54, Pigm, P45, Ppkm, and Ph1 for blast resistance | Improved Y58S, GuangZhan60S (GZ63), C815S and HD9802S | [339] |
| India (ICAR-IIRR) | MAS-Gene pyramiding | Pi54 blast and Xa21, xa13 blast resistance | MTU1010 | [324] |
| India (ICAR-IARI) | MAS-Gene pyramiding | Pi2, Pi54 blast and xa13, Xa21 blast resistance | PB1121-NILs and PB6-NILs | [329] |
| India (ICAR-IIRR) | MAS-Gene pyramiding | Pi54 blast and Xa2 blast resistance | DRR17B | [340] |
| India (ICAR-IIRR) | MAS-Gene pyramiding | Pi54 and Pi2 blast resistance | Improved Samba Mahsuri | [337] |
Table 6. Cont.

| Country                  | Approach               | Applications                          | Cultivar Developed          | Reference |
|--------------------------|------------------------|---------------------------------------|----------------------------|-----------|
| India (ICAR-IARI)        | MAS-Gene pyramiding    | *Pi54*, *Pi1*, *Pita*, *Pi2*, and *Pi9* | PBI NILs                  | [314]     |
| India (PJTSAU, Hyderabad)| MAS-Gene pyramiding    | *Pi54* and *Pi1* for blast resistance  | Tellahamsa                | [341]     |
| ICAR-IARI                | MAS-Gene pyramiding    | *Pi5* and *Pi54* blast resistance     | Basmati restorer PRR78     | [342]     |
| ICAR-IIRR                | MAS-Gene pyramiding    | *Pi54* blast and Xa2 blight resistance | IR58025B                  | [343]     |
| ICAR-IARI                | MAS-Gene pyramiding    | *Pi54* blast, *xa13*, *Xa21* blight and QTL qSBR11-1 ShB resistance | Improved Pusa Basmati 1 | [344]     |
| ICAR-IIRR                | MAS                    | *Pi1*, *Pi2*, *Pi33*, and *Pi54* for blast resistance | ADT 43 NIL               | [345]     |
| ICAR-IIRR                | MABB                   | *Pi1*, *Pi2*, and *Pi54*              | 16 introgressed lines     | [346]     |
| Universiti Putra, Malaysia| MABB                  | *Pi54* (Pi-kh) and *Pi-b*            | MR219                     | [327]     |
| ICAR-IIRR                | MABB                   | *Pi54* blast resistance               | Swarna                    | [347]     |
| ICAR-IIRR                | MABB                   | *Pi54* introgression for blast resistance | Samba Mahsuri            | [322]     |
| ICAR-IIRR                | MABB                   | *Pi2*, *Pi54*, *Xa21*, *xa13*, and *xa5* | Improved Samba Mahsuri   | [348]     |
| UAS & Tech, Kashmir      | MABB                   | *Pi54*, *Pi1*, and *Pita*            | Mushk Budji               | [323]     |
| ICAR-IIRR                | MABB                   | *Pi5* and *Pi54*, *Pi5*              | Swarna-Sub1               | [332]     |
| China: Yangzhou          | MABB                   | *Pi1*, *Pi33*, and *Pi54*, *Pi5*     | 15-pyramided lines        | [349]     |
| TNAU                     | MABB                   | *Pi54* introgression for blast resistance | Restorer lines            | [350]     |
| ICAR-IIRR                | MABB                   | *Pi54*, *Pi1*, *Xa21*, and *xa13*    | Tellahamsa                | [341]     |
| ICAR-IARI                | Allele mining          | *Pi54* allele mining land races and wild rice | -                         | [351]     |
| Switzerland (ETH Zurich)| Allele mining          | *Pi54* mining from 885 Indian rice genotype | -                         | [279]     |
| ICAR-NRCPB               | Allele mining          | *Pi54* mining from 92 rice lines      | -                         | [293]     |
| ICAR-IARI                | Allele mining          | *Pi54* mining from 100 rice germplasm | -                         | [352]     |
| ICAR-NRCPB               | Allele mining          | *Pi54* mining from land races and wild rice | -                         | [170]     |
| Tohoku University, Japan | Allele mining          | *Pi54* evolution in the *Oryza* genus | -                         | [353]     |
| China; Yancheng          | Allele mining          | Field resistance for blast *Pi-ta*, *Pigm*, and *Pi54* for blast disease | Rice accessions           | [354]     |
| Malaysia                 | Over-expression        | Constitutive expression of *Pi54* homologue from rice line PH9 | Transgenic- MR219         | [355]     |
| ICAR-NRCPB               | Over-expression        | *Pi54* orthologue from *O. officinalis* | Transgenic TP309          | [89]      |
| ICAR-NRCPB               | Over-expression        | *Pi54* orthologue from *O. rhizomatis* | Transgenic TP309          | [171]     |
| ICAR-NIPB                | Over-expression        | *Pi54*                               | Transgenic TP309          | [169]     |
| China; Chengdu           | Over-expression        | *Pi8*, *Pi25*, and *Pi54*             | Kasalath, Zhengan 10      | [356]     |

6.4. Genome Editing of Immunity Regulators

Sequence-specific nucleases (SSNs), such as 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 recently proven to be extremely effective tools for plant genome editing [357]. Since it has become feasible to use the bacterial CRISPR/Cas mechanism in eukaryotes, which in itself is simple to design, fairly affordable, and multiplexing compliant [358], it has consequently superseded other approaches. CRISPR/Cas9 has proved to be the most effective SSN to date and has been used to alter the genomes of key crops including rice [359].
Susceptibility factor-encoding genes are often potential targets for genome editing as knockout of a single gene can significantly improve tolerance [360]. Müller and Munné-Bosch found that plant ethylene responsive factors (ERF) have a role in stress tolerance regulation [361]. Furthermore, RNAi silencing of rice ERF922 in cultivar Zhonghua 17 improves resistance to M. oryzae, suggesting that this gene may function as a resistance negative regulator [362]. Consistent with these findings, the CRISPR/Cas9-targeted knockouts of ERF transcription factor have shown an improved resistance to rice blast [363]. The gene-edited mutants exhibited a reduced number of blast lesions following pathogen infection compared to wild-type plants at both the seedling and tillering stages. Furthermore, no significant differences were observed between mutant lines and the wild-type plants for different agronomic traits tested.

To investigate the functional relevance of exocyst subunit proteins in plants defense mechanisms, CRISPR/Cas9 was used to alter OsSEC3A, which is reported to be associated with rice defense responses. The two exons of the OsSEC3A were targeted with two sgRNAs [364]. Edited rice plants showed enhanced immunological response and enhanced resistance to the blast disease. Blast resistance can also be realized by fine-tuning the multifunctional genes involved in rice defense signaling. CRISPR/Cas9 multiplex genome editing system was used for targeted alteration of the thermosensitive male sterile 5 gene (TMS5), rice blast susceptibility gene pi21, and bacterial leaf blight susceptibility gene xa13 [365]. Triple mutants (tms5/pi21/xa13) with homozygous frame-shift mutations in all three genes displayed thermosensitive genic male sterility with enhanced resistance to rice blast and bacterial blight.

Besides, CRISPR/Cas9 has also been used for functional validation of blast resistance genes. The R gene, Pi-d2, has been targeted for editing using the hAID*-DXTEN-Cas9n-NLS chimeric gene (dubbed rBE5) base editor to validate its role in resistance to M. oryzae [366]. Similarly, Zhao et al. [101] used CRISPR/Cas9 to confirm the function of the Ptr, a constitutively expressed resistance gene that imparts broad spectrum resistance to M. oryzae. Therefore, genome editing through CRISPR/Cas has more potential application in rice for developing varieties with enhanced blast resistance and also for the functional validation of potential defense response genes. Advanced genome-editing technologies such as base editing and prime editing could be used to install superior allelic variations precisely for developing blast resistance. In this direction, the editing of the susceptibility factors holds a great promise provided the targeted genes are chosen carefully to prevent fitness cost or yield reduction.

7. Conclusions and Future Perspective

Rice blast disease is a major threat to global rice production. Besides, M. oryzae is considered as the most potent potential biological weapon. Blast pathogen affects all parts of the rice plant from roots to panicles [4]. Since its detection dating back to 1637, constant efforts are being made to develop strategies for the effective management of rice blast disease. To date, more than 500 blast resistance QTLs have been reported, about 102 blast R genes have been mapped in rice, and 38 of these mapped genes are cloned and functionally characterized. The reported QTLs and R genes have been deployed in the genetic background of elite rice lines for resistance breeding using both conventional and genomics-assisted breeding approaches. The pathogen on the other hand displays an exceptional genomic plasticity that enables it to adapt to changes in the host, thus making it difficult for the rice breeders and researchers to rest on the present achievements to deal with the threat. There is always a chance of emergence of new virulent strains of a pathogen that can subvert the existing resistance responses [12]. Continued efforts to study the rice–Magnaporthe interaction, in order to understand the molecular mechanism of pathogenicity and resistance, are required to devise means to counteract the adaptability potential of the pathogen.

Though considerable success has been achieved in managing the disease through host resistance, the present changing climatic scenarios may alter this advantage in favor of
the pathogen in the near future. As was reported in the case of the recent outbreak of wheat blast disease in Bangladesh, climate change is going to be a big challenge to manage various major as well as minor diseases [72]. The outbreak of wheat blast, reported to be an event of host-jump of Magnaporthe to wheat, highlights the potential host jump of Magnaporthe from other crops to rice, thereby risking everything we have achieved in rice blast management. The centuries of research and recent revelations in genomics indicate that we have almost exhausted our rice genetic resources for the identification of novel, potent resistance genes for tackling emerging strains of blast pathogen. Therefore, to this end, the recent developments in the field of genome editing, mainly in CRISPR-Cas systems, appears to hold on to the future challenges. Knock-out of single and multiple genes and induction of targeted genetic variation with conventional CRISPR-Cas tools and precise editing with base editors and prime editors empower us with the ability to decipher a great deal of host–pathogen interactions and improve rice plant for blast resistance. This is largely true because till now researchers have focused on the positive regulators of blast disease resistance in rice. There lies a plethora of rice negative regulators—many of which are yet to be identified—that have the potential to provide more stable and durable resistance.

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