International Journal of
Molecular Sciences

MDPI

Review

Prospects of Understanding the Molecular Biology of Disease Resistance in Rice

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Received: 17 January 2018; Accepted: 5 March 2018; Published: 10 April 2018

Abstract: Rice is one of the important crops grown worldwide and is considered as an important crop for global food security. Rice is being affected by various fungal, bacterial and viral diseases resulting in huge yield losses every year. Deployment of resistance genes in various crops is one of the important methods of disease management. However, identification, cloning and characterization of disease resistance genes is a very tedious effort. To increase the life span of resistant cultivars, it is important to understand the molecular basis of plant host–pathogen interaction. With the advancement in rice genetics and genomics, several rice varieties resistant to fungal, bacterial and viral pathogens have been developed. However, resistance response of these varieties break down very frequently because of the emergence of more virulent races of the pathogen in nature. To increase the durability of resistance genes under field conditions, understanding the mechanismof resistance response and its molecular basis should be well understood. Some emerging concepts like interspecies transfer of pattern recognition receptors (PRRs) and transgenerational plant immunitycan be employed to develop sustainable broad spectrum resistant varieties of rice.

Keywords: rice; disease resistance; breeding; biotic stress; marker assisted selection; signaling pathways; transcription factor

1. Introduction

Rice is one of the major cereal crops that fulfils more than 23% calorie needs of people worldwide and is a staple food crop for half of the world population living in Asia, where its cultivation covers approximately 92% of total acreage [1]. It is expected that the world population will exceed eightbillion by the year 2025 and to meet the increased global food and calorie demands, the total grain production has to be increased by up to 50% [2]. To fulfill this goal, it is imperative to decrease crop losses due to biotic and abiotic stresses [3]. Rice cropsare affected by around 70 pathogens, especially viruses, bacteria, fungi and nematodes that damage the crop severely and ultimately reduce yield [4]. Minimizing the losses caused by these diseases will thus increase total rice production. Deployment of resistance genes in rice is the best practice to manage diseases and reduce environmental damageby reducing the application of agro-chemicals. Development of disease resistance rice varieties has beenachieved by the implementation of plant breeding approaches including conventional breeding like introduction of exotic lines, backcross breeding, and modern biotechnological approaches such as molecularmarker assisted backcross breeding, gene pyramiding, etc.Molecular mapping of disease resistance genes, their cloning and generation of transgenic lines using genetic engineering methods are very promising approaches. These techniques explore the natural diversity present within the rice gene pools. However, natural diversity is not enough to continuously generate new resistance

cultivars. To generate more variation, artificial mutations can be randomly created in the rice genome or the genes involved in the resistance mechanism can be directly targeted. Development of new resistant varieties is needed to protect crops from pathogens. Understanding themolecular basis of resistance against multiple pathogens is also important for the sustainable use of resistance sources under the pressure of global climate change.

We now understand that there are two tiers of defense in pathosystems: PAMP (Pathogen associated molecular pattern)-triggered immunity (PTI) and effector-triggered immunity (ETI) which act in a typical host plant against the pathogen attack. The PTI is the first level of defense barrier in the plant and this process begins by getting the perception or through sensing of microbe- or pathogen-associated molecular patterns (MAMPs or PAMPs). The second level of defense in the plant is the ETI which recognizes effector molecules delivered by pathogens inside host cell that have surpassed the PTI [5]. A primary characteristic of the plant innate immunity is activation of the PTI. The perception of PAMPs and MAMPs by pattern recognition receptors (PRRs) directs to the foundation of several downstream defense related signaling consequences. Thus, the virulence capability of a pathogen depends upon the deactivation of PTIs by its effector molecules [6]. The stimulation of PTI generates signaling networks of mitogen-activated protein (MAP) kinase, transcriptional recoding facilitated by transcription factors such as WRKY and formation of different reactive oxygen species (ROS) inside the host plant [7]. The ETI is activated by the binding of major *R* gene receptors of host plant and effectors of pathogen that activates host defense mechanism and generally leads to localized cell death surrounding the pathogen invasion [8]. In this review, molecular aspects of rice disease resistance, including the general approaches used for developing resistant plants, the genetic basis of host resistance, signal transduction pathways, defense mechanism, regulation of defense mechanism by regulatory elements and their perspectives in rice resistance will be discussed in response to fungal, bacterial and viral pathogens.

2. Mechanism of Plant Defense against Biotic Stresses

The defense system of plants comprises of multiple barriers ranging from outer barrier, like waxy cuticles to internal barriers like resistance and defense response genes. Plant resistance is entirely dependent on a network of signaling pathways involving innate immunity and a class of resistance genes [9–11]. Primarily, defense systems of plants can be categorized into two classes, basal defense and specific defense systems. The basal defense system checks the entry of pathogens in the plants andprovides immunity at the beginning of infection. This defense system is much effective against necrotrophic pathogens. The latter specific defense mechanism operates effectively against biotrophs and hemibiotrophs through hypersensitive response (HR) developed by programmed cell death at the surroundings of infection sites and thereby limits pathogen growth and disease development. This mechanism comes forward to control the pathogen when first level defense is breached [9,12]. The basal defense system or innate immunity is a generalized barrier which does not discriminate between pathogens, unlike the specific defense system which is mediated by a highly specific set of genes called resistance (*R*) genes. In the beginning of infection, waxy cuticle, and the cell wall restrict pathogens entry, however, most pathogens infecting plants like fungi harbor secretory proteins which degrade these barriers [13–15]. After the entry of pathogens inside the host cell, molecules get released (called Microbial/Pathogen Associated Molecular Pattern; MAMP or PAMP) which are composed of peptidoglycan, ergosterol lipopolysaccharide, and bacterial flagelin proteins. The innate immune system recognizes these proteins with the help of receptors present on the plasma membrane of host cell called pattern recognition receptors (PRRs) to further restrict the progress of infection providing MAMP-triggered immunity (MTI). PRRs also detect molecules that are native components of the host which, however, get released when damage is done by pathogens and are known as damage-associated molecular patterns (DAMP). The binding of these components also activates PTI and downstream defense responses [16,17]. Overall, the recognition of MAMP/PAMP or DAMP leads to the activation of PTI causing different reactive oxygen species (ROS) production, initiation of mitogen-activated

protein (MAP) kinase activity and various transcription factoractivation restricting the spread of pathogens completely [7].

Several studies have proven that expression of defense response (DR) genes like *Chitinase*s and *Phenylalanineammonia-lyase* (*PAL*) can directly correlate with host resistance [18,19]. Rice germin-like proteins (OsGLP) are a class of DR genes, present in a QTL along with *R* genes and are potentially associated with resistance of rice, as silencing of these genes increased the susceptibility against two major fungal pathogens, sheath blight and rice blast [20]. *OsPAL4* is associated with broad spectrum disease resistance in rice [19]. A LysM receptor like kinase (RLK), *OsCERK1,* regulates cytoplasmic *OsRLCK176* and *OsRLCK185*, recognizes chitin and peptidoglycans activating immune signaling pathways in rice against *Magnaporthe oryzae* and *Xanthomonas oryzae* pv. *oryzae* (*Xoo*). *OsCERK1*, *OsRLCK176* and *OsRLCK185* follow the same signaling pathways; however, *OsRLCK176* and *OsRLCK185* function downstream in the signaling pathway governed by *OsCERK1*. The phosphorylation of *OsRLCK185* is mediated by *OsCERK1* to activate MAPK pathways. The *Xoo* effector Xoo1488 inhibits the phosphorylation of *OsRLCK185* causing pathogen resistance [21,22]. Three *RLKs* and *OsWAK* genes in rice are transcriptionally co-regulated as these *RLKs* are known to be required for enhanced resistance against *M. oryzae* [23]. Transcriptome and micro RNA analysis of two varieties of rice (resistance versus susceptible) revealed different signaling pathways as well as variable micro RNA expression causing change in innate immunity against *M. oryzae* [24]. A lectin *RLK* from rice is also found to show association with innate immunity and interacts with depolymerizing factor of actins [25].

Most of the above-mentioned examples are mediated by pattern recognition receptors (PRRs). Basically, PRRs belong to receptor like kinase (RLK) and receptor like protein (RLP) classes. The RLK has a single membrane-embedded domain, a kinase domain present intracellularly and a domain present extracellularly for sensing ligand molecules, whereas RLP has a membrane-embedded extracellular region but lacks kinases region [26]. RLPs are structurally and functionally similar to Toll like receptors (TLR) of animals [27]. The extracellular ligand-sensing domain of PRR is generally rich in leucine repeats [28,29]. In contrast to animals, plants are sessile and are affected by biotic, abiotic and multiple other factors, therefore, plants may have higher numbers of RLK and RLP than animals. Approximately, 640 RLKs and 90 RLPs are reported in rice [30].

PAMP marker detection which lead to plant immunity is a widespread and effective mechanism to restrict most of the pathogens. However, in spite of this, multiple pathogens including fungi and bacteria possess a varietyof proteins that enter the plant and manipulate innate immunity of hosts whileremaining undetected. In this scenario, a second line of defense comes into action that includes resistance (*R*) gene, composed of mainly nucleotide binding site (NBS), Leucine Rich Repeat (LRR) domains and other domains [5]. The *R* genes impart resistance against pathogens by recognizing secreted effector proteins called avirulence (*Avr*) genes and provide immunity, i.e., effectortriggered immunity (ETI). This resistance provided by *R* genes could be the result of either direct interaction with the *Avr* gene or indirect interaction that involves other mediator proteins. The indirect interaction of *R-Avr* involves finding modifications of other host proteins (effector targets) that interact with the effectors. By this interaction, a cascade of defense response networks are activated leading to HR, restricting pathogen growth at the infection site. The immunity provided by the innate immune response and *R* genes is altogether mediated by a complex network of signaling pathways leading to the activation of defense-responsive genes such as pathogenesis-related genes (*PR*), reactive oxygen species (ROS), *glucanases*, *chitinases*, secondary metabolites, physiology of stomata closure, and deposition of callose and lignin. The products of these genes largely act at the protein levels against fungal and bacterial pathogens and are involved in regulation at the mRNA level. In contrast to the defense mechanism against viral diseases, they generally actat the RNA level [31–34].

3. Resistance Gene Architecture and Resistance Hypothesis

Plant resistance *R* genes are generally a multi domain gene family having NB-ARC (nucleotide-binding adaptor shared by APAF-1, R proteins, and CED-4) as a primary domain and Leucine Rich Repeats (LRR)

as an associated domain. The nucleotide binding site (NBS) domains play a key role in host defense mechanisms [35–37]. The LRR domain has a role in the recognition of pathogen gene products [9,38]. The NBS-LRR domains are functionally dependent on each other and are under simultaneous pressure [39]. In NBS domains, multiple motif sequences like MHDV, RNBS-A, RNBS-B and GLPL have also been reported, that are crucial in maintaining the function of the NBS domain [40]. In eukaryotes, these are collectively called NLRs (Nucleotide binding domain, Leucine-rich Repeat). In rice, approximately 438 putative *NLR* genes have been reported [41,42].

Various models have been given to describe the interaction of *R*- and *Avr*-genes. The very first model put forward was the gene-for-gene hypothesis. According tothis hypothesis, the *R* gene product of the host plant interacts directly with its corresponding *Avr* gene product of the pathogen [43]. This hypothesis was followed by multiple cases of *R-Avr* interactions, i.e., a single *NLR* gene recognizes its counterpart *Avr* effector and imparts resistance to the pathogen [5,44]. This type of direct interaction has been reported in the case of the rice blast resistance protein Pi54 and its counterpart Avirulence protein, AvrPi54 [45]. Indirect interaction between *R*- and *Avr*-gene products is called the Guard–Guardee model, in which *R*-genes monitor the target and interaction of effector molecules and modification of this target acts as an indicator of pathogen attack. Two proteins of *Solanum lycopersicum* (tomato), Pto and Prf follow this mechanism to interact with the AvrPto proteins of the bacterial pathogen *Pseudomonas syringae* [46], however, recognition of effectors in multiple pathogens by indirect means lead to a modified hypothesis called the decoy model which states that an effector protein may have generated multiple targets in the host (decoy proteins) by duplication events [47]. The decoy model has been reported in multiple studies for effector *Pto*, *Bs3*, *RCR3*, and *RIN4* [47]. Recently, in some studies, the multiple decoy domain has also been found to show a link with NLR proteins [48–50]. The NLR proteins with decoy domain were firstly reported in poplar tree plants having BED domain as a decoy domain [51]. This type of *R* gene function was reported in a study in which sugar transferase, BED-type zinc finger andcarbohydrate esterase 4 superfamily acted as decoy domains in nine putative *R* genes and in a resistance gene (*Xa1)* of rice, and further speculated that these domains may integrate in *R* genes to functions as sensors [52].

This type of *R* gene action has also been validated in rice-*M. oryzae* pathosystem [48,53,54], where RGA5 and Pik-1, two proteins of rice contain RATX1/HMA domain whichare similar to gene *Pi21*, interact physically with two effectors, *Avr-Pia* as well as with *Avr-Pik* of *M. oryzae*. In another study, Kroj et al. [55] reported that the NLR and decoy domain association is extensive and common in plants. Actually, they mined 31 plant genomes for finding NLR with putative decoy domains and further evaluated the role of NLR-BED decoy domain protein in rice for resistance against *M. oryzae* using overexpression and knockout mutant analysis. Although few R proteins arealready known to contain decoy domains, e.g., the NLR CHS3 has zinc-finger domain [56], the rice resistance protein Pi-ta contains a thioredoxin domain [57], Xa1 comprises a BED domain [58] and Pi54 contains a unique zinc finger (NFX) domain [42,59]. Sarris et al. [60] also found that the fused domain with multiple NLR proteins may act as bait for pathogen effectors as these domains areknown to interact with pathogens.

4. Signal Molecules and Their Networks Involved in Rice Defense Response

The plant cell initiates a network of defense signaling cascades on the perception of pathogen elicitors through PRRs and disease resistance (R) proteins.This results in a defense response to confine the pathogen within the infection site. The defense response includes changes in membrane permeability and ion fluxes (Ca^{2+}, K^{+}, H^{+}), generation of ROS and NO, production of pathogenesis responsive (PR) proteins (glucanases, chitinases, defensins), cell wall strengthening (callose and lignin deposition), phytoalexin synthesis and activation of kinase cascades accompanied by hypersensitive response (HR) [5]. This defense response is mediated by cross-communicating sets of endogenous signal molecules, including phytohormones, ROS and NO which in turn are regulated by numerous transcription factors [61]. These Jasmonic Acid (JA), Salicylic Acid (SA), and Ethylene (ET) are the archetypical players in the regulation of defense signal transduction cascades.

4.1. Mitogen-Associated Protein (MAP) Kinase and Ca^{2+} Signaling

Following pathogen perception, plant receptors activate ion channels, GTP binding proteins, and kinases, which in turn activate secondary messengers to amplify signals to the diverse set of downstream cascades. The MAP kinase cascade is the prevalent component of plant defense signaling, mainly in the PTI signaling pathway. It comprises of three interlinking proteins where MAPKKK stimulates MAPKK which in turn stimulate MAPK. Thus, activated MAPK stimulates TF and other downstream signaling molecules through trans-phosphorylation reactions [62]. A total of 17 MAPKs have been identified from *O. sativa*; however, only five MAPKs (*OsWJUMK1*, *OsMAPK4*, *OsMAPK5*, *OsMAPK6*, and *MAPK12 OsBWMK1*, for blast and wound induced MAP kinase) have been characterized for their function [63]. Ca^{2+} influx is another essential and conserved event in the plant defense responses which stimulates intracellular signaling directly or through Ca^{2+} sensors. The Ca^{2+} sensors can be either Ca^{2+}-dependent protein kinases (CDPKs), chimeric Ca^{2+}/calmodulin-dependent protein kinases (CCaMKs) or CDPK-related kinases (CRKs) [64]. Activation of typical CDPKs stimulates the CCaMKs, enzymes or TFs in the downstream processes of defense response. The CDPKs and MAPKs act either synergistically or independently in the innate immune responses.

4.2. Role of ROS and NO in Rice Defense Signaling

The generation of reactive oxygen species (superoxide, O^{2-} and H_2O_2 accompanied by oxidative burst) is a conserved early response in plant defense signaling cascades, generally accompanied by programmed cell death/hypersensitive response during plant–pathogen interaction. The NADPH oxidase, peroxidase and oxidases are the sources of ROS generation in cell organelles, including mitochondria, chloroplasts and peroxisomes [65]. This oxidative burst drives the cell wall reinforcements or cell wall strengthening for cellular protection. The pathogen avirulence factors induce the production of nitric oxide (NO) which potentiate the induction of defense-related gene expression and secondary metabolite synthesis [66], thereby, playing a key role in disease resistance in plants.

4.3. Phytohormone-Mediated Signaling

Plant immunity follows a binary model for phytohormone signaling, including SA- and JA/ET-dependent pathways, which interact in a mutually antagonistic way to trade-off pathogens, which is similarin rice. Moreover, SA often induces resistance against hemibiotrophs and biotrophs whereas necrotrophs are usually deterred by JA/ET-dependent cascades [67]. However, rice crop reveals new insights and unique features ofplant immunity as compared to *Arabidopsis* [68]. The endogenous level of SA often increases substantially for induction of the pathogenesis-related (PR) proteins and defense response. However, involvement of the SA in rice defense mobilization was more reliantupon SA signaling rather than the variation in its endogenous level or its *de novo* synthesis [68,69]. The SA pathway in rice shares *OsNPR1/OsNH1*, *OsTGAs*, *OsWRKY13* [70] and *OsWRKY45* (autoregulated) [71] as downstream signaling components which in turn induce PR protein accumulation (Figure 1) to confer resistance against rice blast and bacterial blight. JA is often considered to be predominantly effective against necrotrophs but enhances susceptibility to biotrophs. However, monocots including rice did not completely follow this dichotomy [72,73]. Accumulating evidence showsthat JA is a powerful signal to trade-off hemibiotrophs and biotrophs including *X. oryzae* and *M. oryzae* in rice [74,75] as well as to defend-off necrotroph including *R. solani* [75]. Moreover, ET functions synergistically with JA signaling and acts as a two-faced regulator in rice defense signaling since its application enhances disease resistance against pathogens such as rice blast, whereas it leads to disease susceptibility in case ofbacterial blight depending on pathogen's infection biology [76].

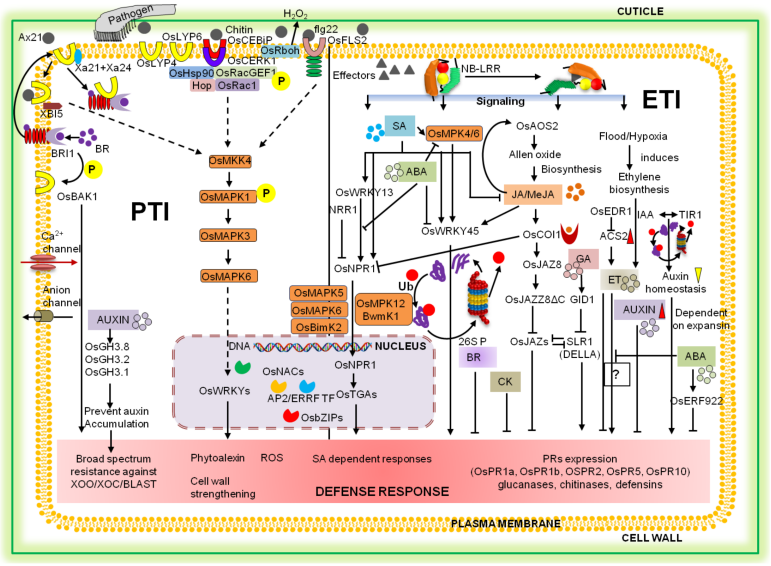

Figure 1. Schematic representation of rice defense signaling cascades. Following the pathogen perception bypattern recognition receptors(PRRs) and R proteins, the rice plant initiates the diverse set of signaling cascades at different levels (PAMP-triggered immunity (PTI), left side and ETI, right side) involving numerous signal molecules, viz. ROS, NO, MAPKs, CDPKs, phytohormones to trade-off the pathogen invasion. In case of PTI, the host cell recognizes the common molecular pattern associated with most of the pathogens using PRRs (OsLYP6, OsFLS2, OsCEBiP, OsCERK1, Xa21, Xa24) and initiates MAPK kinase cascades (OsMKK4–OsMAPK6) that actually activate host defense responses via various transcriptional regulatory factors (OsWRKYs, OsNACs, OsNPR1, OsTGAs, OsbZIPs). However, PTI is suppressed by pathogen effectors, where they are encountered by the resistance genes (*NBS-LRRs*) that lead signaling to activate defense responses through phytohormonal activities. The archetypical defense pathways, SA and JA/ET pathways, mainly antagonistic to each other, are responsible for resistance against biotrophs and necrotrophs, respectively. The defense response includes production of PR proteins (glucanases, chitinases, defensins), production of ROS and NO, change in ion fluxes (Ca^{2+}), cell wall strengthening (callose and lignin deposition) to confine the pathogen dissemination and disease development. GA, CK and Auxin act as negative regulators of plant innate immunity. BR prompts or suppresses disease susceptibility based on pathogen lifestyle or colonization. Furthermore, abscisic acid (ABA), well-known in abiotic stress tolerance, plays an ambiguous role, i.e., is both a positive and negative regulator of rice disease resistance based on the type and stage of infection; however, it predominantly actsas a negative regulator. The abbreviations used in the figure above represent viz. SA-salicylic acid; JA-Jasmonic acid, MeJA-Methyl Jasmonate; GB-Gibberellins, BR-Brassinosteroid; ET-Ethylene, CK-Cytokinin; ABA-Abscisic Acid, OsNPR1-Non-expressor of PR1 (NH1, NPR1 homolog1); OsCOI1-Coronatine Insensitive1 (JA receptor); OsJAZ8-Jasmonate ZIM domain protein, HPL3-Hydroperoxide lyase; ACS2-Enzyme for ET biosynthesis (ACC Synthase); OsEDR1-Enhanced Disease resistance 1 (TR1-like kinase); SLR1-slender rice1 (DELLA protein); GID1-encodes GA receptor; BRI1-BR Insensitive 1 (RLK) BR receptor.

There is extensive crosstalk between various phytohormones in rice against biotic stresses. For example, SLR1 (a rice DELLA protein) is an important regulator of GA signaling and suppresses GA biosynthesis. In addition, SLR1 mediates resistance against hemibiotrophs and biotrophs, but not necrotrophs in rice through integration and amplification of SA- as well as JA/ET-dependent signaling [77]. Moreover, cytokinin (CK) acts synergistically with SA signaling and enhances resistance against hemibiotrophic and biotrophic pathogens [78]. Moreover, auxin accumulation enhances disease susceptibility in rice. For instance, over-expression of *OsGH3.8*, *OsGH3.1*, and *OsGH3.2* prevents auxin accumulation and is responsible for enhanced broad spectrum resistance against *Xoo*/*M. oryzae*/*Xoc* pathogens in innate immunity [79]. Auxin signaling stimulates *OsWRKY31*-depending expression of defense-related and auxin responsive genes which in turn suppress the auxin sensitivity and

enhances the resistance against rice blast [80]. Furthermore, brassinosteroids (BR) are reported to be involved in modulation of innate immunity based on BAK1-dependent and BAK1-independent defense response [81]. Surprisingly, abscisic acid (ABA), often known to be involved in abiotic stress tolerance is a negative regulator of biotic stress response. For example, exogenous applications of ABA and ABA biosynthesis inhibitor suppress resistance and reduce susceptibility against cold stress and rice blast, respectively [82,83]. In summary, the defense signaling network is a complex and cross-communicating network of interaction between signal molecules, including ROS, NO, MAPK, CDPKs and phytohormones, and any impairment in this signaling network enhances the chance of disease susceptibility in rice plants.

5. Role of Regulatory Elements in Rice Disease Resistance

Plant responses towards biotic stresses are specially facilitated by the action of different phytohormones in a network of signaling pathways, in which salicylic acid (SA) reaction to stress are an antagonistic way of the responses generated by jasmonic acid (JA)/ethylene transport (ET) pathways. In these signaling networks, the SA pathway is mainly associated with the responses developed against biotrophic pathogens. However, the responses induced by necrotrophic pathogens are synergistically regulated by JA and ET pathways. This synergistic action is also involved in plant defenses against various insect pests [84]. Some chemicals are also known to induce SA-derived responses in the plant (like probenazole, benzothiadiazole (BTH) which increase the level of SA in rice plants to mimic as biotrophic pathogens) [85]. The SA pathway plays a key role in the systemic acquired resistance (SAR) defense mechanism by activation of *NPR1* gene. Afterward, this gene provides broad spectrum resistance response against the pathogens. However, it does not provide immunity to the plant alone; several signaling transductions simultaneously work altogether to produce the resistance response [86]. *NPR1* protein is active only in its monomeric form, otherwise, it is present in a dimeric inactive form and the monomeric change is induced by the SA pathway, in which SAs breakdown the disulphide bonds of dimeric protein to generate monomers [87]. This protein directly interacts with transcription factors (TFs) of the *TGA* gene family to activate defense response [88]. Nevertheless, it also negatively regulates SAR in the plants with the help of *WRKY*-TFs [89]. For instance, *OsNPR1/NH1* functions as a repressor or negative regulator to provide resistance in rice against bacterial blight by manipulating expression levels of other defense- and photosynthesis-related genes [69,90,91]. The key *WRKY*-TFs involved in the the SA-derived resistance response is *WRKY45*, which is induced by SA activity and treatment of plants with BTH. However, it has been reported that *WRKY45* works independently and is not directly related to the *NH1* (homolog of *NPR1*) in rice [92]. This TF is reported to activate resistance response in transgenic lines against *M. oryzae* after treatment with BTH. Its physical interaction with a panicle blast resistance gene *Pb1* has been reported to provide broad spectrum resistance to *M. oryzae* [93]. In many studies, it has been concluded that *WRKY45* plays a major role in defense response of rice against rice blast and bacterial blight pathogens [92,94]. However, rice *WRKY45* has two different alleles (*OsWRKY45-1*, *OsWRKY45-2*) in indica and japonica subspecies and both the alleles were found to show resistance to *M. oryzae*, but, not to bacterial diseases [94]. The allele *OsWRKY45-1* does not provide resistance to the bacterial pathogens *Xoo* and *Xoc*, whereas, *OsWRKY45-2* shows direct involvement in bacterial resistance [94].

Very few TFs act as master regulators in responses to various biotic stresses in plants besides *WRKY45* [94]. *WRKY13* is another major regulatory factor to transmit signals from *WRKY45* to downstream functioning *WRKY*-TFs such as *WRKY42*. *WRKY13* follows the SA-pathway-dependent disease resistance mechanism and shows association with resistance responses against *M. oryzae* and *Xoo* [95,96]. Furthermore, this regulatory factor has two cis-elements (*PRE2* and *PRE4*), which are identified as pathogen-responsive regulatory elements [70,97]. Overall, *WRKY13* regulation is mediated by the SA pathway and pathway-related genes, while JA pathway-related signals are suppressed during this regulation [95]. Several *WRKY*-TFs other than master regulators have been identified and characterized; these play a role in rice resistance response for *M. oryzae* [79,98–102], *X. oryzae* [103] and

Rhizoctonia solani [104,105]. Hence, the *WRKY*-TFs family is one of the major transcription regulatory elements involved in rice resistance response through SA, JA, ET and ABA signaling networks, because all these signals are interlinked and have crosstalk between them.

Several other major and minor non-*WRKY*-TFs have also been identified in rice. *NAC* is a major regulatory element that has been recognized to induce and impart resistance response in rice plant through the activation of *PR* genes. Many *NAC*-TFs are reported to provide innate immunity in rice, mainly against *M. oryzae.* Several findings have been documented with reference to *NAC*-TF induced resistance in rice and these are, *ONAC122*, *ONAC131*, *OsNAC6*, *OsNAC19*and *OsNAC111* [106–109]. Another transcription factor, basic leucine Zipper (*bZIP*), was found to regulate the signal transduction- and the defense-related genes in rice, thus restricting *M. oryzae* infection [110]. In a study based on *bZIP*, *OsBBI1*, a *bZIP* gene was identified that regulates the resistance spectrum in rice for diverse groups of *M. oryzae* by altering the first level of defense mechanism in host plant [111]. In addition, *OsBRR1* was also found to have resistance regulating action in rice against a certain set of *M. oryzae* isolates [112]. Therefore, various types of transcription factors play an important role in inducing immune response in rice via positive or negative gene regulation. C_2H_2-type TF was discovered to regulate rice resistance in a manner of non-race-specific to *M. oryzae*, while another *AP2/ERF* (*OsEREBP1*) controlled the rice resistance mechanism in a specific manner against *X. oryzae* [113,114]. Expression of ethylene responsive transcription factors *OsBIERF1*, *OsBIERF3* and *OsBIERF4* was induced by infection with *M. oryzae*, suggesting their role in biotic stresses [115]. Other rice TFs associated with resistance response towards biotic stresses are *OsDR10*, *OsGAP1*, *OsRac1*, for *X. oryzae* [116–118], *OsAOS2*, *Rir1b* for *M. oryzae* [119,120], *OsPLDβ1*, *OsDR8*, *OsSBP* for *X. oryzae* and *M. oryzae* [88,121,122]. Interestingly, some regulatory elements in rice like *Rir1b* are expressed at a much higher level when rice is infected with its non-host pathogen *Pseudomonas syringae* pv. *syringae* and that accumulated transcript of the *Rir1b* gene produced an enhanced resistance response in rice against *M.oryzae* [120]. Furthermore, *OsRac1* is reported to be essential for innate immunity in rice to *M. oryzae*, however, it also directly interacts with *Pit*, an NBS-LRR resistance gene. This interaction enhances effectiveness of the resistance in rice to *M. oryzae* [123]. Another *OsGH3.1* regulatory element is reported to help rice by providing resistance against a fungal infection [124].

6. Breeding Approaches to Control Diseases in Rice

The genus *Oryza* contains 23 species, of which only twoare cultivated and the rest 21 are wild types [125]. *Oryza sativa* (Asian rice) and *O. glaberrima* (African rice) are the cultivated species of rice and *O. sativa* is the only species of rice grown worldwide, whereas *O. glaberrima* is limited to some parts of West Africa. The *O. sativa* also has two subspecies indica and japonica. Japonica subspecies are further grouped into temperate and tropical rice with their growing suitability, and tropical japonica is known as javanica, which is considered as a separate subspecies of *O. sativa*. Now, it is a well-established fact that genetic diversity iskey to the improvement of any crop plant by transferring the genes for useful traits from the land races and wild-relatives into elite cultivars. Thus, natural genetic diversity is a major resource for various plant breeding programs, including resistance breeding for biotic stresses. Only 6 of 21 wild species, namely *O. glumaepatula*, *O. breviligulata*, *O. meridionalis*, *O. longistaminata*, *O.* rufipogon and *O. nivara*, and two cultivated *Oryza* species *O. sativa* and *O. glaberrima* have been utilized in rice crop improvement programs via breeding techniques, because they make the primary gene pool (AA genome) and share genetic constituents without any hindrance [126–128]. Besides, two additional gene pools, secondary and tertiary are reported for rice, but they are not suitable for traditional breeding programs, however, several useful traits taken from them have been exploited with the help of biotechnological tools and other approaches in conjunction with the breeding programs. Conventional breeding methods are the same for all kinds of agronomically important traits used in rice improvement programs. There are two types of resistance reported in the plant against biotic stresses, i.e., partial and complete resistance [129]. Partial resistance is governed by more than one gene and also called quantitative resistance or polygenic resistance. It provides non-race-specific resistance

against the pathogens through quantitative resistance loci [130], while complete resistance is controlled by a single gene. It possesses a qualitative character and provides race-specific resistance against the pathogens. Nevertheless, sometimes, a single gene acts both ways and provides complete as well as partial resistance towards pathogens. Both cases of resistance have been reported in rice to control various plant pathogens. Major techniques employed in the rice resistance breeding are given below.

6.1. Introduction of Resistant Exotic Lines

Introduction of new germplasm lines and local landraces is an alternative way to augment the genetic diversity of crop plants in a confined area where local germplasm lacks resistance to biotic stresses [131]. It is a process to bring foreign genetic material for evaluation and utilize selected agronomically superior lines as a resistant variety in such areas where a certain pathogenic strain causes disease to all the local cultivars. It is a quick method to resolve the problem, but bringing genetic material across the globe is very tough due to country-wise laws that make this method very limiting in disease resistance breeding programs. However, the introduction of exotic lines was reported to be an efficient method for development of disease resistant cultivars [132]. For instance, Thippeswamy et al. [133] reported that the most of exotic rice germplasm taken from the International Rice Research Institute (IRRI) have shown resistance to local South Indian races of blast fungus, indicating high potential of this method in resistance breeding.

6.2. Introgression of Resistance Genes

Management of diseases in rice through the use ofresistant cultivars is one of thebest ways to tackle the problem because it reduces the pesticide application in rice fields, hence, minimizing the cost of crop production and subsequently lowering agrochemical pollution in the fields. The deployment of resistant cultivars to various rice diseases has already been published [134–136]. Land races and wild relatives of rice are usually used as sources for introgression of a new resistance gene into elite cultivar. Nevertheless, introgression of a resistance gene is challenging with conventional breeding methods because of the linkage-drag of undesirable traits that is very hard to break in spite of many generations of back-crosses [137]. Backcross breeding is normally employed for the introgression of resistance genes by inserting single disease resistant genes into a susceptible high yielding elite cultivar. Many near isogenic rice lines (NILs) have been generated through backcross breeding and employed in the development of resistant rice variety. Plant breeders also develop disease-resistant hybrids and cultivars using a conventional hybridization method. In this method, combining the genes of agronomically important traits, including disease resistance from different sources are practiced to improve the crop plants. Several cultivars of crop plants have been developed to enhance the disease resistance using this method [138].

6.3. Pyramiding of Resistance Genes

Gene pyramiding is one of the most effective plants breeding strategies for achieving multiple and durable resistance against many plant diseases. In this breeding method, genes pyramided from different genotypes in single cultivars offer long-term resistance towards pathogens and has turned into a plant breeders' tool to generate broad-spectrum disease-resistant cultivars which restrict emergence of various pathogen races. Many different approaches such as composite breeding [139,140], synthetics crosses, and multiline crop breeding [141–143] have been utilized for developing gene pyramided lines of crop plants against various biotic stresses. Multiline breeding is also an important method of resistance improvement in crop plants [141–143]. It is also known as the "dirty crop" breeding method as in this approach, an elite cultivar is improved by developing many isogenic or near isogenic lines (NILs). The NILs are created by transferring a single resistance gene from various sources in different single plants by several rounds of backcrossing and, thus, each line has a separate resistance gene. Afterward, the NILs are bulked and the bulkedlines are called multiline as they have many lines containing separate resistance genes. This tool assists in gene pyramiding to pool several resistance

genes together into a single genotype offering durable resistance. These lines are morphologically similarplants that may be genetically less different [141]. Rice breeders have generated many blast disease-resistant cultivars using this breeding method [143–145].

7. Molecular Breeding Approaches for Disease Resistance

There are various problems associated with the classical breeding methods for development of resistant cultivars; they require longer time periods, are more effort and labor intensive, they transfer undesirable genes along with the resistance genes by hybridization, they exhibit frequent resistance breakdown due to high mutagenic ability of pathogen and develop new pathogenic races. There are limited natural sources for resistance, and poor understanding ofthe resistance mechanism in the traditional breeding methods. Hence, there isa necessity to advance innovative and more efficient methods to overcome these problems. With the improvement of molecular genetics and biotechnological knowledge, several modern tools have been derived for this rationale. Considering these facts, some modern techniques of plant breeding related to disease resistance in rice against various fungi, bacteria and viruses have been employed.

7.1. Marker-Assisted Selection and Mapping of Resistance Genes

Traditional plant breeding for resistance is mostly dependent upon the phenotypic symptoms which are strongly related to environmental conditions in the field, consequently, newly emergingvirulent race of pathogens cannot be easily identified, and the introduction of improved resistant varieties cannot be reliable [146]. Unlike conventional breeding, marker-assisted selection (MAS) is very effective in disease resistance breeding because major resistance in the plant is operated by single or a few genes [130]. It is very useful in such host–pathogen interaction where both resistance (*R*) gene and avirulence (*Avr*) gene show interaction in the manner of gene-for-gene fashion [147]. Molecular markers increase the efficiency of conventional breeding by choosing the markers that are tightly linked to the desired traits. Similarly, molecular markers are also beneficial for identifying loci that operate quantitative traits [148]. Molecular markers, SSR, SNP, EST, RAPD, AFLP and RFLP are normally used to map several major resistance genes and QTLs in rice (Tables 1 and 2).

Table 1. Details of the resistance genes identified from rice.

Chromosome	Genes	References
1	*(Xa29(t)), (Pit, Pitp(t), Pi37, Pi35(t), Pi24(t), Pi27, Pish)*	[136,149–155]
2	*(xa24(t)), (Pi-b, Pi25(t)) (Pid1(t), Pi-Da(t)), (Pi-y1(t), Piy2(t)), (Pig(t), Pi-tq5, Pi14(t), Pi16(t))*	[149,153,156–163]
3	*(Xa11), (xa42), (Pi66(t))*	[164–167]
4	*(Xa1), (Xa2), (Xa12), (Xa14), (xa31(t)), (Pi39(t), pi21, Pikur1, Pi(t))*	[58,168–178]
5	*(xa5), (Pi26(t), Pi23, Pi10)*	[153,179–182]
6	*(Xa7), (Xa27), (xa33(t)), (Pi27(t), Pi-tq1, Pi8, Pi13(t), Pi22(t), Pigm(t)), (Piz-5, Piz-t), (Pi40(t), Pi59(t), Pi9, Pi2-1, Pid2), (Pi25(t), Pi26),(Piz), (Pi13), (Pi2-2, Pi50(t))*	[153,161–163,181,183–201]
7	*(xa8), (Pi17(t))*	[202,203]
8	*(xa13), (Pi29(t), Pi33, Pizh, Pi36, pi55(t), PiGD-1(t))*	[153,204–213]
9	*(Pi15, Pi2(t), Pi3(t), Pi5(t), Pi56(t))*	[214–218]
10	*(Pi28(t), PiGD-2(t))*	[153,213]

Table 1. *Cont.*

Chromosome	Genes	References
11	*(Xa3/Xa26), (Xa4), (Xa6, xa9), (Xa10), (Xa21), (Xa22), (Xa23, Xa30(t), Xa32(t), Xa35(t), Xa39, Xa40(t)), (Pi7(t)), (Pik, Pik-p), (Pi30(t), Pi60(t), Pilm2, Pikg),(Pik-h, Pik-s), (Pi-hk1), (Pi54), (Pi-1(t), (Pb1, Pise1, Pikur2, Pi38, Pif, Pi34, Pia, PiCO39(t), Pi44(t), Pi49, Pik-m, Pi18(t), Pi47), (Pi1)*	[129,149,153,159,161,162,183,197,219–254]
12	*(xa25/Xa25(t)), (Pita-2), (Pi31(t), Pi32(t)), Pi61(t), Pi-tq6, (Ipi(t), IPi3(t)), (Pi21(t), Pi58(t), Pi51(t), Pi-GD-3(t), Pi48,Pi24(t), Pi-42(t), Pi62(t)), (Pi6(t), Pi4(t)), (Pi12(t), Pi19(t), Pita, Pi39(t), Pi20(t))*	[149,153,159,161,178,181,192,194,213,252,255–264]

Xa indicates resistance gene to bacterial blight disease (*Xoo*) and *Pi* represents resistance gene against blast disease (*M. oryzae*).

Table 2. Chromosome wise list of identified QTLs against various pathogen induced diseases in rice.

Chromosome	QTLs	References
1	*(1.1, 1.2, 1.3, 1.4, 1.5, 1.6, 1.7, 1.8),(AQEN002, AQEN011, AQEN076, AQEN078), AQGJ001,(ch1q1, ch1q2, ch1q3a, ch1q3b, h1q4, ch1q5),(GWr, H30r, HDr, HMr, T30r, VC1,VC2), Pi-24(t), qABB-1, qBBR1,(qbk1.1, qbk1.2, qbk1.3), qBK1_628091, qBLASTa-1, qBlsr1,(QBr1a, QBr1b), qBSfR1,(qDLA1, qLN1-1, qLN1-2, qLS1), qDLA-1-1, qFSR1, qrbr-1.2,(qRBR-1-1(t), rbr1a, qRBR-1-2(t), rbr1b, qRBR-1-4(t), rbr1d), qSB-1,(QSbr1a, QSbr1b), qShB1, qStv1,*	[129,153,196,199,265–283]
2	*(2.1, 2.2, 2.3, 2.4, 2.5, 2.6, 2.7),(AQEN032, AQEN069),(BSq2.1v&i, BSq2.2v&i),(ch2q1, ch2q2, ch2q3, ch2q4, ch2q5, ch2q6, ch2q7), Pi-25(t), qBLASTads, qBlsr2, qBR2.1,(QBr2a, QBr2b, QBr2c), qBS2,(qDLA2, qLN2), qFSR-2-4,,(qRBR-2(t), rbr2),(qrbr-2.2, qrbr-2.3), qSB-2,(QSbr2a, QSbr2b), qShB2,(qShB2-1, qShB2-2),(qStv-12,qStv-9), Rh-2,(S, VC2)*	[129,153,196,199,266,267,272–274,278,280–282,284–292]
3	*(3.1, 3.2, 3.3, 3.4, 3.5, 3.6, 3.7, 3.8, qFSR-3-5, qFSR-3-9), AQEN012,(ch3q10, ch3q2, ch3q3, ch3q4, ch3q5, ch3q6, ch3q7, ch3q8, ch3q9), qBBR-3, qBBR3-1, qBFR3, qbk3.1, qBLASTads-3,(qBlsr3a, qBlsr3b, qBlsr3c, qBlsr3d), QBr3,(qDLA3-1, DLA3-2, qLN3, qLS3), qDLA-3-3 ,(qrbr-3.1, qrbr-3.2), qRBSDV-3, qSB-3,(qSB-3-1, qSB-3-2), QSbr3, Qsbr3a, qShB3,(qShB3-1, qShB3-2, qShB3-3), qSTV-3, Qxa-3, Rh-3*	[129,196,199,266,269,270,272–274,276,278,280–282,288,290,292–298]
4	*(4.1, 4.2, 4.3, 4.4, 4.5, 4.6), qFSR-4-1,(AQEN022, AQEN063, AQEN065), AQGJ003, BSq4.1v&i,(ch4q1, ch4q2),(CQAC1, pi21, CQAC2), GWr, qBB-4, qBBR-4,(qBFR4-1(t), qBFR4-2(t)), qBK4_31750955,(qBlsr4a, qBlsr4b), QBr4, qBSfR4, qDLA'96-4, qFSR4, qLS4,(qrbr-4.1, qrbr-4.2),(qSB-4-1, qSB-4-2),(QSbr4a, QSbr4b, QSbr4c), qStv-4*	[129,196,199,265–268,271,273–278,280,281,284,286,291,293,299,300]
5	*(5.1, 5.2, 5.3, 5.4, 5.5), AQEN004, ch5q1, Pi-26(t), qBB-5, qBBR-5,(qBlsr5a, qBlsr5b), QBr5,(qDLA'95-5, qNBL-5),(qrbr-1.1, qrbr-11.1, qrbr-11.3, qrbr-2.1), qSB-5,(QSbr5a, QSbr5b), qShB5, qSTV5, xa5*	[129,153,199,266,268,273,274,276,278,281,282,290,293,298,301,302]
6	*(6.1, 6.2, 6.3, 6.4, 6.5), qFSR-6-7,(AQEN005, AQEN014),(AQGJ005, AQGJ007, AQGJ024),(BSq6.1v, BSq6.2i),(ch6q1, ch6q2, ch6q3a, ch6q3b, ch6q4, ch6q5, ch6q6a, ch6q6b, ch6q7), Pi-27(t), qBBR-6,(qBLASTads-6, Pi-tq1), qBR6.1,(qDLA-6, qLSNN-6),(qrbr-6.1, qrbr-6.2),(qSB-6-1, qSB-6-2), qShB6, xa7*	[129,153,199,265,266,272,276,278,280,282,284,286,293,298,303]
7	*(7.1, 7.2, 7.3, 7.4, 7.5, 7.6), AQEN007,(AQGJ009, AQGJ010),(ch7q1, ch7q2, ch7q3, ch7q4, ch7q5), qABB-7, qBBR7, qBLASTa-7, QBr7, qDLA7,(qRBR-7-2(t), rbr7b), qSB-7, qStv7, Rh-7*	[129,196,199,265,266,268,269,272,274,279,288,292,302]
8	*(8.1, 8.2, 8.3, 8.4, 8.5), qFSR-8-3, AQEN008,(BSq8.1i, BSq8.2v),(ch8q1, ch8q2, ch8q3, ch8q4, ch8q5, ch8q6, ch8q7), Pi-29(t),qBB-8-2, QBr8,(qDLA8, qLN8, qLS8),(qRBR-8(t), rbr8),(qrbr-8.1, qrbr-8.2, qrbr-8.3),(qSB-8-1, qSB-8-2), Qsbr8a, VC2*	[129,153,196,199,266–268,274,278–280,284,286,296]

Table 2. *Cont.*

Chromosome	QTLs	References
9	*(9.1, 9.2, 9.3, 9.4), AQGJ027, BSq9.1v,(ch9q1, ch9q2, ch9q3, ch9q4, ch9q5, ch9q6), CQAC3, qABB-9, qBLASTads, QBr9, qBR9.1, qBS9,(qDLA'95-9, qLSNN'96-9, qNBL-9),(qrbr-9.1, qrbr-9.2),(qRBR-9-1(t), rbr9a, qRBR-9-2(t), rbr9b), qSB-9,(qSB-9-1, qSB-9-2),(QSbr9a,QSbr9b), qShB9-1, qStv-9, s*	[199,213,265–268,272,274,276,278–282,284,285,289,291,299,301]
10	*(10.1, 10.2, 10.3, 10.4),(qFSR-10-2, qFSR-10-5),(ch10q1a, ch10q1b, ch10q2, ch10q3, ch10q4, ch10q5a, ch10q5b, ch10q6), Pi-28(t), qABB-10, QBr10, qFSR10, qLS10, qRBSDV-10, qSB-10*	[153,196,199,266,268,277,280,281,286,295]
11	*(11.1, 11.2, 11.3, 11.4, 11.5), qFSR-11-2,(AQEN009, AQEN028, AQEN066), AQGJ013,(BSq11.1v&i, BSq11.2v),(ch11q1, ch11q2, ch11q3, ch11q4, ch11q5), Pi-30(t), Pi-lm2, qABB11, qBFR11, qBlsr11, QBr11a, QBr11b, qBS11, qBSfR11,(qDLA11, qLN11, qLS11-1, qLS11-2), qRBSDV-11, qSB-11, qStv11,(qSTV-11a, qSTV-11b, qSTV-11c)*	[129,153,196,199,265,266,268,274,277,281,284,286,294,297,304,305]
12	*(12.1, 12.2, 12.3, 12.4, 12.5), qFSR-12-5,(AQEN025, AQEN043, AQEN072),(AQGJ014, AQGJ015, AQGJ016), BSq12.1v,(ch12q1,, ch12q2, ch12q3, ch12q4, ch12q5, ch12q6, ch12q7, ch12q8, ch12q9, ch12q10, ch12q11), CQAC4,(Pi-31(t), Pi-32(t)), qBB-12,(qBLASTa12-2, qBLASTads-12-1, Pi-tq6), (qDLA-12-2, qDLA-12-5), qFSR12,(qrbr-12.1, qrbr-12.2), qSB-12,(QSbr12a, QSbr12b, QSbr12c), qStv-12,(s, VC2)*	[129,153,199,265–268,272,276–278,280,281,284,286,291,299]

In this regard, resistance to blast disease has been recorded in rice as qualitative and quantitative [4]. Over 100 genes giving complete/partial blast resistance in rice have been documented [1,306]. Moreover, most of the blast *R* genes were characterized by a map-based cloning approach. For instance, *Pi54* is a major blast *R* gene and provides broad-spectrum resistance against many races of *M. oryzae*; it was cloned by map-based cloning approach from chromosome 11 of rice [238]. Similarly, Kumar et al. [257] also identified a broad-spectrum blast resistance gene *Pi42(t)* that was mapped on the chromosome 12 using the molecular marker techniques, but it is yet to be cloned and characterized. Both chromosomes 11 and 12 are the major blast resistance-gene-containing chromosomes. Besides, chromosomes 2 and 6 are also reported to have a large number of blast resistance genes (Figure 2). Another molecular marker-based approach allele mining was reported to clone and characterize many orthologues and alleles of blast *R* genes from wild rice [307–311]. Sheath blight considered the second most devastating fungal disease of rice which is caused by the necrotrophic fungus *Rhizoctonia solani* worldwide. Developing resistant cultivars against *R. solani* is very difficult due to the absence of a single major resistance gene, even though some resistance QTLs have been identified and used in rice improvement programs [312]. Brown spot caused by the necrotrophic fungus *Cochliobolus miyabeanus* (formerly known as *Helminthosporium oryzae*) is third in a row to damage rice crops by fungi. This fungus causes yield losses up to 52% [313]. For this fungus, only resistance QTLs have been detected [314]. Bakane disease does not have aneconomic importance as other major fungal diseases of rice, such as blast and sheath blight. It is caused by the *Gibberella fuzikori* species complex (GFSC). This complex comprises many species of *Fusarium fuzikori*, *F. proliferatum*, *F. concenticum* and *F. verticillioides*. The fungus causes infection through roots or crowns resulting in partially filled or chaffy grains. Only eight QTLs have been identified for the disease [270,315,316]. In addition, false smut caused by *Ustilaginoidea virens* is another minor fungal disease of rice. So far, except for tenQTLs, no major resistance-conferring gene has been identified from rice against this disease [286,317,318].

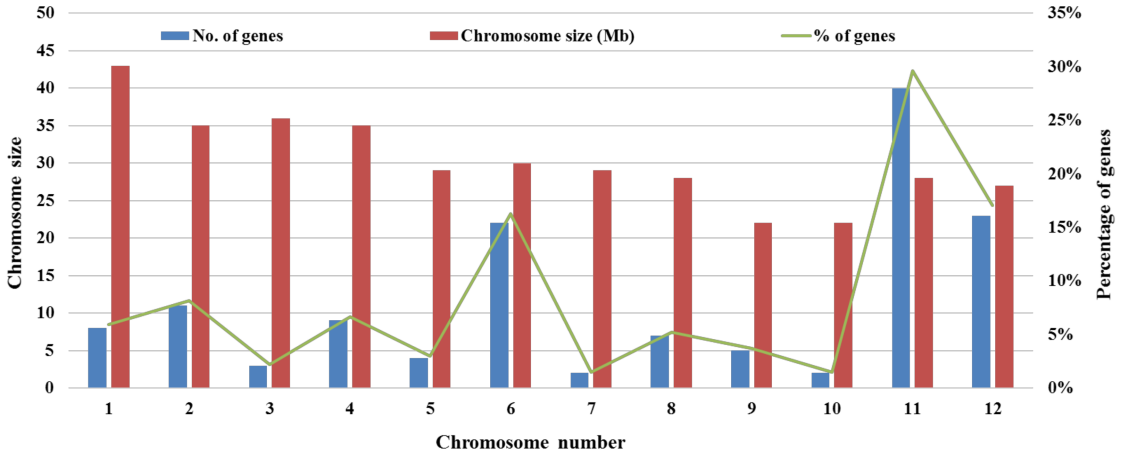

Figure 2. Chromosome-wise distribution of major resistance genes identified from rice. Numbers 1–12 represent the chromosome of rice. Percentage of resistance gene sharing on each chromosome is shown in green solid line, while blue and red bars represent number of resistance genes on each chromosome and chromosome size (Mb), respectively.

Several bacterial diseases also damage rice crops and major bacterial disease that cause significant yield losses in rice arediscussed here. A bacterial blight disease caused by the biotrophic bacterium *Xanthomonas oryzae* pv. *oryzae* (*Xoo*) is the utmost significant bacterial disease of rice, especially in tropical and subtropical areas. So far, more than 40 major resistance genes and several QTLs have been identified in rice using the marker-assisted selection method [319].Another bacterial disease, bacterial streak is caused by *X. oryzae* pv. *oryzicola* (*Xoc*) and it is a serious problem in Asian rice cultivation [320]. In this case, one *Xo1* locus in rice has been found to confer complete resistance to *Xoc* [321]. Numerous QTLs showing resistance to *Xoc* have been registered [319]. Two bacterial diseases, bacterial grain rot and bacterial seedling rot, produced by a necrotrophic bacterium called *Burkholderia glumae*, are important diseases of rice at the global level [322]. No single major resistance gene against *B. glumae* has been identified yet, but resistance QTLs have been reported through marker-assisted selection [314].

Many viral diseases havealso been reported in rice and some of them are mentioned here, like rice stripe disease [323], rice yellow mottle disease [324,325] and rice tungro disease [326]. Rice stripe disease iscausedby the infection of *Rice stripe virus* which is an RNA virus transmitted by small vector brown plant hoppers. Five major resistance QTLs have been reported with the help of molecular marker techniques, and one of them wasmolecularly characterized at the nucleotide sequence level [327]. Rice tungro disease (RTD) consists of a *Rice tungro spherical virus* (RTSV) and a *Rice tungro* bacilliform virus (RTBV) and the disease is one of the most destructive diseases that causes yield constraint in rice-growing areas of tropical Asia. Both RTSV and RTBV are transferred in the host plant by an insect vector green leaf hopper (GLH) [328]. RTSV is freely transmitted by GLH, while RTBV can be transferred by GLH only in the presenceof helper virus RTSV [329]. Marker-assisted selection for resistance against RTSV has been effectively applied to develop RTSV-resistant rice lines [330]. Most of the major resistance genes and QTLs are plotted by chromosome-wise and remaining genes and QTLs could not be positioned on the respective chromosomes due to lack of their physical location (base pair) information (Figures 3 and 4). Chromosomal distribution analysis showed that chromosomes 1, 6 and 11 had greater numbers of major genes and QTLs than other chromosomes, whereas considering only QTLs 2/3 rice chromosomes shared more or less equal numbers of loci (Figure 3). Chromosomes 4, 5, 7 and 10 contained less QTLs for disease resistance in rice (Figure 4).

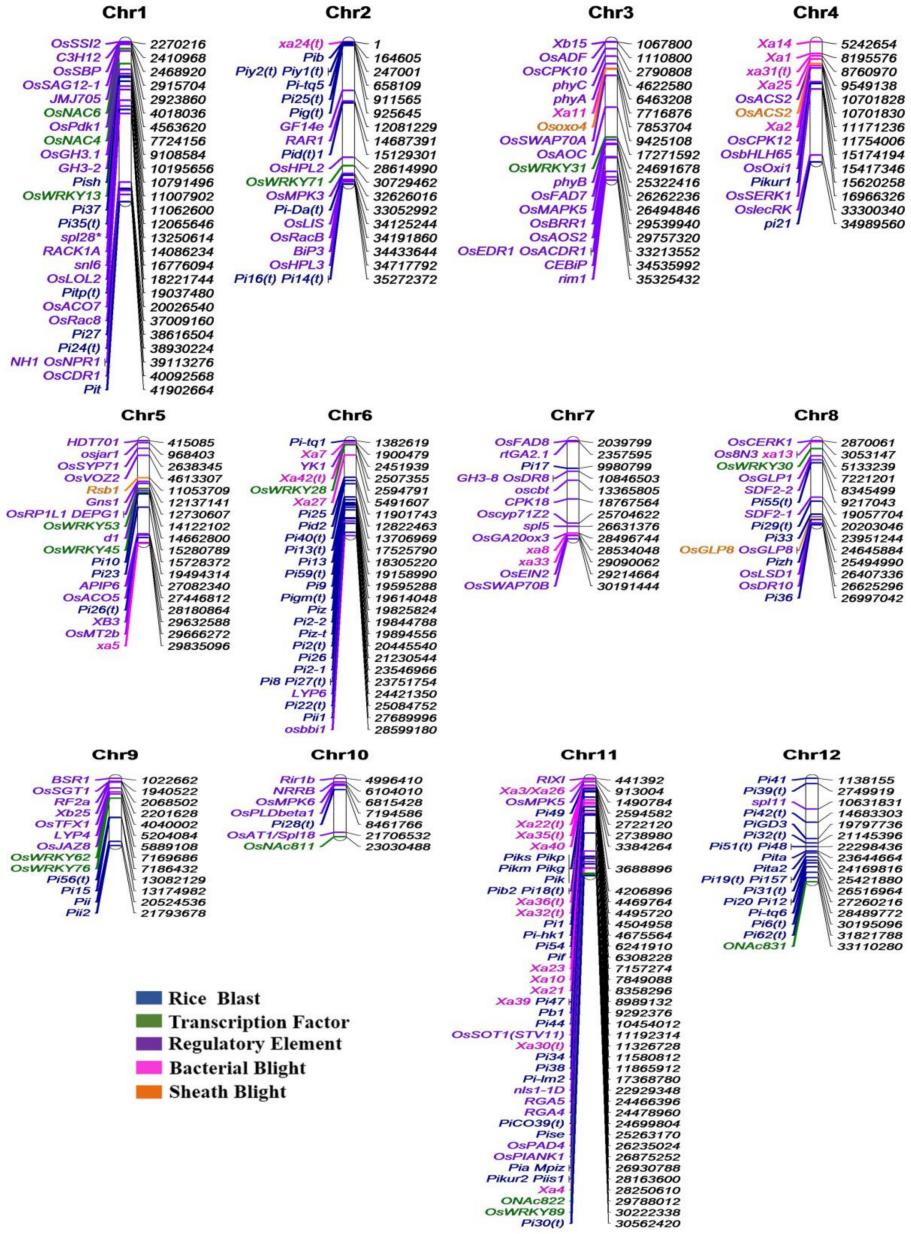

Figure 3. Distribution of major resistance genes according to theirphysical location on the respective chromosomes. Different disease resistance gene categories plotted on the chromosomes are indicated by five color codes. The plot was generated on the basis of the nearest linked molecular makers.

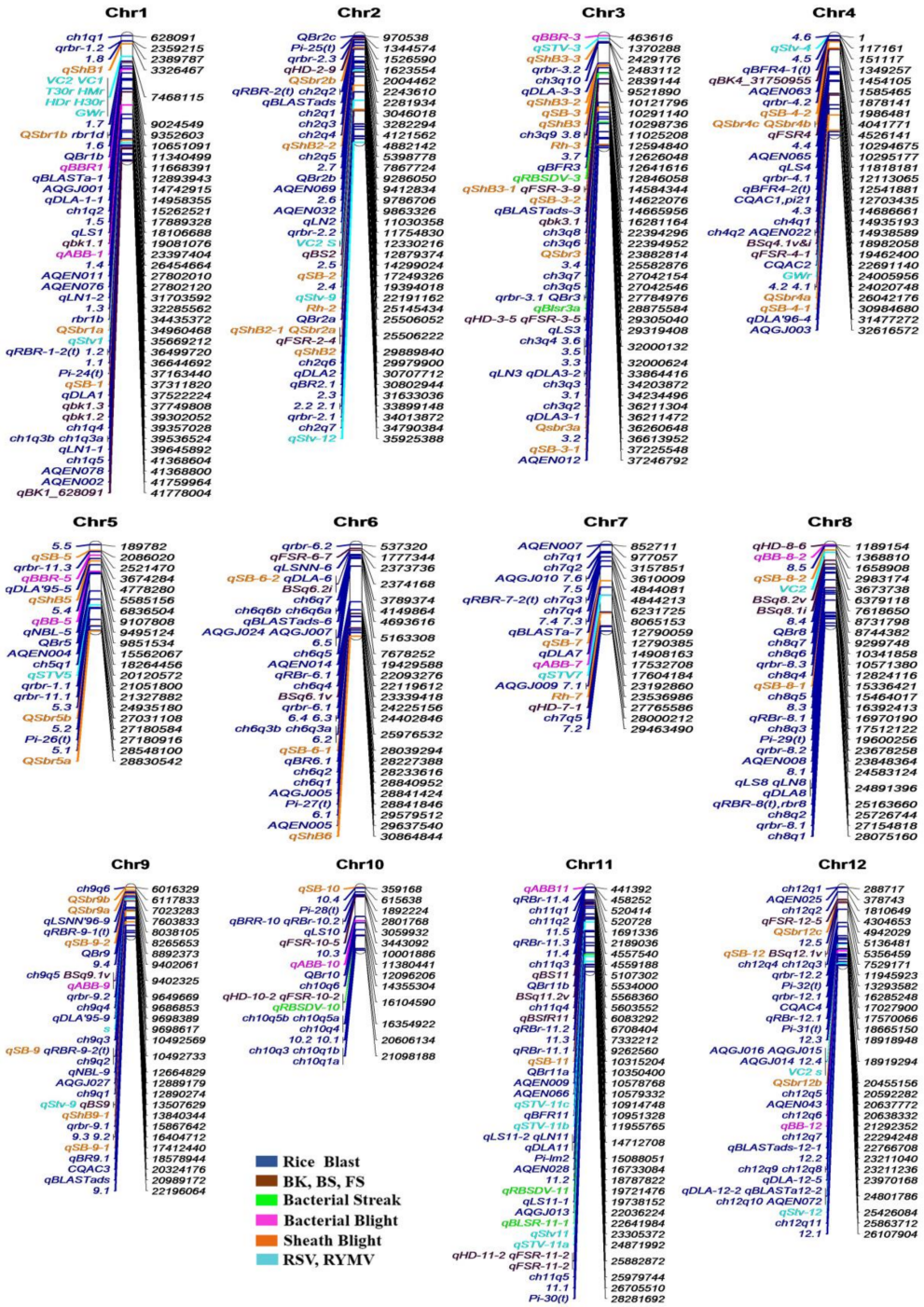

Figure 4. QTLs distribution on rice chromosomes. Separate color codes are given for each group of QTLs. The physical distribution of QTLs is derived by the nearest linked molecular markers on each chromosome. BK, BS, FS, RSV and RYMV represent Bacterial Streak, Brown Spot, False Smut, *Ricestripe virus* and *Rice yellow mottle virus* resistance QTLs, respectively.

7.2. Resistance Gene Pyramiding by MAS

Pyramiding is combining of genes into a single cultivar or line using back cross breeding. On the other hand, it is a strategy to combine two or more than two genes present in multiple parents into a single genotype with all of the target genes. Gene pyramiding is widely used for combining multiple pest or disease resistance genes for specific races of an insect or pathogen to generate durable

resistance. By applying marker-assisted selection (MAS) and molecular marker back cross breeding (MMBC), it reduces the breeding duration in gene pyramiding and helps in crop improvement program. Different *R* genes often provides resistance to a number of different biotypes, races or isolates, thus making it durable. Several studies related to gene pyramiding have been reported in rice for disease resistance [331–336]. Recently, a group of four QTLs was pyramided in a rice line, giving resistance against a population of diverse *M. oryzae* isolates in different field conditions [337]. Similarly, many bacterial blight resistance genes have been pyramided and have developed improved rice cultivars using marker-assisted selection [338].

7.3. Genome-Wide Association Study of Resistance Genes

Association mapping is an alternative QTL mapping approach, conducting association analysis of genotypes versus phenotypes of a large population; it is based on linkage disequilibrium (LD) or the non-independence of alleles. In this method, SSR and SNP markers have been widely used to effectively identify marker and disease resistance associations in rice [339–341].Genome-wide association studies (GWASs) reflect association relationships of genome-wide distributed marker traits; such studies have become gradually popular in rice genetics with the advancement of high-throughput next generation sequencing (NGS) approaches and SNP chip techniques [342,343].GWAS is a very potent strategy which can be used for the understanding of the genetic basis of complex traits that has been exclusively useful for rice [332]. Nowadays, GWAS has been used in combination with NGS like genotyping by sequencing (GBS) to identify the SNP markers associated with resistance phenotypes in rice. Recently, an association study was performed based on 184 000 SNPs generated by GBS which helped in associating25 genomic regions with blast resistance in the rice genome [344].

7.4. Mutation Breeding for Rice Resistance

Mutation breeding is also known as the reverse genetics approach, in which mutants are generatedusing physical or chemical mutagens and evaluated for their resistance to various diseases. Most of the mutants generated by this method are deleterious in nature, but very few show promising effects for agronomically important traits including disease resistance. This approach is very useful in those crops which do not have much genetic diversity, but it has been routinely used in rice for disease resistance [345–347]. Various mutagenic agents like γ, UV, X-rays irradiation (Physical mutagens), ethyl methane sulfonate (EMS), methyl methane sulfonate (MMS) and colchicine, (Chemical mutagens) are generally used for the induction of mutations. However, biological mutagens like T-DNA insertion and transposable elements have been widely exploited by plant breeders to generate mutant lines [348]. The main drawbacks of mutation breeding arethe restricted influence in producing dominant alleles with less efficiency and also its random nature.

8. Transgenic Approaches for Disease Resistance in Rice

With the recent advances in sequencing technologies, various genes involved in pathogenesis pathways and plant innate immunity have been dissected and used for developing durable disease-resistant crops through transgenic approaches like over-expression/gene complementation tests, Small RNA (microRNA), RNA interference (RNAi), CRISPR/Cas systems [349]. However, genetically modified (GM) plants derived usingthese approaches have been adapted in the main stream of agriculture at a very small scale. Due to various potential biosafety issues, introduction of new allergens into food, horizontal gene transfer from GM plants to non GM plants or microbes, genetic diversity loss, affecting non target organisms, and socioeconomic and ethical concerns [350–353], involved with the transgenic approaches. Although there is no doubt that the GM technology has played an important role to fulfil our increased demands in the field of medicine and some non-food crop plants, plant molecular biology and biotechnology techniques have taken a rapid progress in the identification and cloning of genes involved in plant defense responses. In the following sections, various transgenic approaches being used for the development of disease resistance in rice are explained.

8.1. Over-Expression/Functional Complementation Test in Rice Resistance

The term "overexpression" means increased expression of targeted genes beyond the normal expression level. The functional complementation assay (FCA) is an in vivo assay commonly used to validate gene function and its essentiality. For biotic stresses, the number of genes has already been characterized using the complementation test. For developing resistance, several blast resistance (*R*) genes from rice have already been identified and characterized [1,311,354].Now, many *R* genes have been fully characterized in rice and their effectiveness has beentested using various approaches (Table 3). The resistance spectrum of *R* gene orthologues by mining superior alleles from wild species with a broader defense spectrum has also been reported [307,311].

Table 3. List of selected rice resistance and defense response genes characterized by genetic engineering.

Gene	Type	Method	Pathogen	Reference
Pib	NBS-LRR	OE	*M. oryzae*	[355]
Pi-ta	NBS-LRR	OE	*M. oryzae*	[262]
Pi9	NBS-LRR	OE	*M. oryzae*	[193]
Pi-2	NBS-LRR	OE	*M. oryzae*	[190]
Pi5	CC-NB-LRR	OE	*M. oryzae*	[356]
Pi21	Proline-rich protein	RNAi	*M. oryzae*	[357]
Pi36	CC-NB-LRR	OE	*M. oryzae*	[263]
Pi37	NBS-LRR	OE	*M. oryzae*	[151]
Pi54	NBS-LRR	OE	*M. oryzae*	[358]
pi-d2	B-lectin domain	OE	*M. oryzae*	[195]
Pik	CC-NBS-LRR	OE & RNAi	*M. oryzae*	[359]
Pikm	NBS-LRR	OE	*M. oryzae*	[360]
Pik-p	CC-NBS-LRR	OE & RNAi	*M. oryzae*	[361]
Pi54rh	NBS-LRR	OE	*M. oryzae*	[307]
Pi54of	CC-LRR	OE	*M. oryzae*	[311]
Pi54 and *Pi54rh*	NBS-LRR	OE	*M. oryzae*	[362]
mpi and *pci*	Proteinase inhibitors	OE	*M. oryzae*& Insect pests	[363]
chi11 and *ap24*	Rice chitinase & Tobacco osmotin	OE	*R. solani*	[364]
OsBRR1	LRR-RLK	OE	*M. oryzae*	[112]
OsWAK1	Protein kinase	OE	*M. oryzae*	[365]
Dm-AMP1	Antifungal plant defensin	OE	*M. oryzae*&*R. solani*	[366]
OsACS2	Ethylene biosynthetic gene	OE	*M. oryzae*&*R. solani*	[367]
At. NPR1	Defense gene	OE	*R. solani*	[368]
(Loc_Os11g47510)	Chitinase	OE	*R. solani*	[369]
PR-5	Thaumatin-like protein	OE	*R. solani*	[370]
RCH10 and *AGLU1*	Chitinase & Alfalfa β-1,3-glucanase gene	OE	*M. oryzae*&*R. solani*	[371]
OsCPK4	Protein kinase	OE	*M. oryzae*&*R. solani*	[372]
OsCPK10	Protein kinase	OE	*M. oryzae*&*R. solani*	[373]
miR160a and *miR398b*	miRNA	OE	*M. oryzae*	[374]
miR169a	miRNA	OE	*M. oryzae*	[375]
osa-miR7695	miRNA	OE	*M. oryzae*	[376]
OsPGIP1	Polygalacturonase inhibiting proteins	OE	*R. solani*	[377]
BSR1	Receptor like kinase	OE	*Xoo*, *M. oryzae*, *Burkholderiaglumae*, *Cochliobolusmiyabeans*, RSV	[378]
OsOSM1	Osmotin protein (PR)	OE	*R. solani*	[379]
OsPGIP1	Polygalacturonase inhibiting proteins	OE	*R. solani*	[380]
OsPR1b	PR	OE	*Xoo*	[381]
OsSAMS1	*S*-adenosyl-L-methionine synthetase	RNAi	RDV	[382]
OsDCL1	Dicer-like	RNAi	*M. oryzae*	[383]
miR528	miRNA	OE	RDV	[384]
miR168	miRNA	RNAi	RDV, RSV	[385]
ALS	Acetolactate Synthase	CRISPR/Cas9	Herbicide	[386]
OsSWEET13	Sucrose transport	TALENs	*Xoo*	[387]
MoHrip1 and MoHrip2	Effector protein	OE	*M. oryzae*	[388]
MoSM1	Secreted protein	OE	*M. oryzae*&*Xoo*	[389]
RPMK1-1 and RPMK1-2	Kinases	RNAi	*R. solani*	[390]
chi11	Chitinase	OE	*R. solani*	[391]
OsSWEET11, OsSWEET14	Sucrose transporter	CRISPR/Cas9	*Xoo*	[392]
OsSWEET14	Sucrose transporter	TALEN	*Xoo*	[393]
OsERF922	ERF	CRISPR/Cas9	*M. oryzae*	[394]

Over-expression: OE, Rice sheath blight pathogen: *R. solani*, *Rice stripe virus*: RSV, *Rice dwarf virus*: RDV, Ethylene Responsive Factor: ERF.

Due to continuous selection pressure, pathogens overcome *R*-mediated host resistance in a few years [395]. To overcome such a scenario, gene stacking methods are used, i.e., combing two or more

genes for developing crops with combined protection against several pathogens. Recently, two *R* gene alleles, i.e., *Pi54* and *Pi54rh* were stacked together in a susceptible rice variety to confer enhanced combined host resistance against *M. oryzae* [362,396].Similarly, transgenic rice expressing two fusion genes *mpi* and *pci* (proteinase inhibitors) has displayed high resistance towards insect attack and rice blast infection [363]. Richa et al. [369] also developed transgenic rice plants harboring novel *chitinase* gene (LOC_Os11g47510) through genetic transformation resulting in much higher resistance against sheath blight (ShB) disease. Over-expressing thaumatin-like protein (TLP) in a rice line showed an enhanced level of ShB resistance compared to the control plants [370].Using a gene stacking method, chitinase gene (*RCH10*) and glucanase gene (*AGLU1*) together provide resistance to both ShB and rice blast pathogen in a susceptible rice variety [371]. SA, JA and ET plant hormones play key roles in defense responses and signaling [397,398]; they are also known to activate many defense-associated kinases, different transcription factors and various *PR* genes, as these genes have been reported to increase the host resistance in many transgenic rice lines [399]. Transgenic rice expressing *OsACS2* gene encoded 1-aminocyclopropane-1-carboxylic acid synthase, which is a key enzyme of ET biosynthesis, under the regulation of a pathogen-inducible promoter resulted in higher ET production and enhanced resistance against rice blast and sheath blight diseases [367]. In a similar study, *MoSM1*-overexpressing transgenic rice showed an improved resistance against *M. oryzae*, and *Xoo* by modulating SA/JA signaling pathways [389]. Interestingly, some reports mentioned increasing host resistance because of protein elicitors produced by plant pathogens. Two elicitors, namely, *MoHrip1* and *MoHrip2* cloned from *M. oryzae* fungus, when overexpressed in rice result in lower levels of disease severity compared to the controls [388]. Small RNAs including siRNAs (short-interfering RNAs) and miRNAs (microRNAs) are the major groups associated with post-transcriptional gene regulation affecting eukaryotic immunity. These are the short non-coding RNAs having a consistentmode of biogenesis and mechanism of action. With the advancement in sequencing technology, large sets of miRNAs have been identified and characterized in various crops for both biotic and abiotic stresses. Using deep sequencing, Li et al. [374] explored miRNA for rice immunity against *M. oryzae*. Transgenic rice plants with over-expressing miR160a and miR398b resulted in up-regulated defense-related genes followed by enhanced resistance against *M. oryzae*. Similarly, Campo et al. [376] identified and overexpressed novel osa-miR7695 miRNA in rice. Transgenic plants harboring osa-miR7695 have ahigh resistance spectrum against the rice blast pathogen; miR169a overexpressing rice lines were found to be highly affected by *M. oryzae* infection by altering defense related responses [375]. Thus, the miRNA acts in both ways as a positive- as well as negative-regulator towards plant immunity.

8.2. Role of RNA Interference (RNAi) in Disease Resistance

The RNA interference (RNAi) approach involves sequence-specific gene regulation mediated by small RNAs (sRNAs). In eukaryotes, RNAi emerged as one of the most precise, efficient mechanism resulting in gene regulation both at the transcriptional and post transcriptional level. RNAi plays critical roles in developmental regulation, stress response, and host defense against transposons and viruses. It involves a two-step mechanism, wherein its initial step is to degrade dsRNA and thereby generates 21–25 nucleotides long small interfering RNAs (siRNAs) through the action of RNase III-like molecules. In the final step, siRNAs associate with an RNase and make RNA-induced silencing complex (RISC), which precisely acts on the cognate partner of double-stranded mRNA and ultimately degrade the targeted mRNAs via a homology dependent manner [400–402].Various target traits have already been modified through RNAi approaches towards crop improvement (Table 3). RNA-dependent RNA polymerases (RDR) play a crucial role in gene silencing that provide resistance against the pathogens. Wagh et al. [403] reported the mutantrice line of *RDRP6* gene increased susceptibility response against *Cucumber mosaic virus* (CMV), *Rice necrosis mosaic virus* (RNMV), *X. oryzae* pv. *oryzae* and *M. oryzae*. Host-induced gene silencing (HIGS) is a mechanism that involves the silencing of pathogen genes using the RNAi tool [404]. The target genes of pathogens are expressed and dsRNA is generated using the plant machinery. This dsRNA is used as a precursor for generating smaller RNA fragments

complementary to the genes expressed distantly in the pathogen [405]. There are transgenic rice lines carrying a hybrid RNAi construct targeting two pathogen genes where pathogenicity MAP kinases *RPMK1-1* and *RPMK1-2* shows increased sheath blight resistance compared to the control lines [390]. VIGS (Virus Induced Gene Silencing) is an important tool for triggering RNAi silencing with the use of viral vectors like BMV (*Brome mosaic virus*). VIGS acts as an efficient and rapid tool for assigning gene function in plants. Using BMV-HIGS, Zhu et al. [406] reported that *MoABC1*, *MoMAC1* and *MoPMK1* *M. oryzae* genes were responsible for disease development.

8.3. CRISPR/Cas9 Immune System

With sequence-specific nucleases (SSNs), genome editing where gene insertion, deletion or replacement in the genome become a reality with enormous possibilities. SSNs are also considered as "molecular scissors" which belong to four categories, i.e., MegaN (mega nuclease), ZFNs (Zinc finger nucleases), TALENs (transcription activator-like effector nucleases) and CRISPR/Cas9 (clustered regularly interspaced short palindromic repeat/CRISPRs-associated protein 9) [407,408]. CRISPR/Cas9 system is considered as one of the most important and simple genome editing tool. Due to high simplicity and efficiency, this system becomes a powerful tool for understanding various biosynthetic pathways and resistance response mechanisms in crop plants [392]. This system is not an exception of off-target problems associated with other genome editing tools ZFNs and TALENs. In this system, the off-target percentage is usually insignificant and it can be further diminished by considering a few points like designing specific guide RNA (sgRNA) sequences, complete whole genome sequence information of the experimental crop plant and selection of highly precise computational tools for target identification [409–411]. Rice as a diploid and a monocot plant is considered one of the best choices for the CRISPR/Cas9 system. The *OsSWEET14* gene in rice results in the pathogenesis of *X.oryzae*. Modifying effector-binding sites present in the *OsSWEET14* gene promoter results in reduction ofpathogen virulence [393]. For addressing biotic stress resistance in rice using CRISPR/Cas9, Wang et al. [394] developed targeted knockout of the *OsERF922* gene and achieved improved rice blast resistance without affecting agronomic traits.

9. Conclusions and Future Perspectives

Rice wasthe firstcrop plant to be decoded at the genome level, and its sequence information has been publicly available since 2005. Thereafter, this crop has attracted lots of attention from plant molecular biologists in relation to study the disease resistance and other agronomically important traits.Although substantial advances have been attained regardinginsights into the genetic nature of disease resistance genes and signal transduction pathways along with influencing regulatory factors heading to defense response activation in rice, the complete story is still far from well-defined. Rice is a well-known model crop plant for various research activities, however, it has lesser information than other model plants like *Arabidopsis* and tobacco with reference to disease resistance. Host resistance response can be very effectively improved by using modern molecular biology and genetic engineering techniques. However, such resistance responsesaregenerally broken down by the emergence of more virulent races of the pathogen. By characterizing additional *R* genes from rice [238,262] wild species and local rice landraces, plant genotypes with varying degree of disease resistance can be obtained [412]. Understanding the signaling cascades involved in disease resistance and the host defense pathway-associated genes can be achieved by the application of the latest molecular biology approaches. These signaling genes will be very helpful for developing rice varieties with sustainable and broad spectrum resistance against various pathogens. Thus, long-term durable and broad spectrum resistance rice is our current need in view of global climatic changes which may help in the emergence of new and virulent races of the pathogens. This goal of getting broad spectrum resistance can be achieved through several emerging approaches like hostplant immunity, non-host resistance, multigene varieties, interspecific gene transfer and genome editing, etc.

Plants possess PTI and ETI innate immune systems to withstand different biotic stresses. While, pathogens are equipped with advanced effector molecules that defeat host plant immunity, many

importantconclusions have been drawn regardingthe rice–pathogen interactions using different techniques such as plant breeding, mutation breeding, marker-assisted selection, gene pyramiding, association studies, genetic engineering with complementation tests, RNAi, miRNA, CRISPR/Cas9, etc. Several important outcomes related to disease resistance have been discovered, such as hypersensitive response (HR) by major *R* gene, ROS generation, *PR* gene activation, hormone biosynthesis and their cross talk with other signaling pathways. However, the entire connections and factors engaged in the plant immune responses employing disease resistance genes are still not clear in rice. The information provided in this review will help in the understanding ofdifferent rice hostpathosystems that may lead to the development of sustainable broad spectrum disease-resistant cultivars.

Acknowledgments: Tilak Raj Sharma is thankful to the Department of Science and Technology, Government of India for J.C. Bose Fellowship and financial support for this work. Akshay Nag and Preeti Arya are thankful to Science and Engineering Research Board, Department of Science and Technology, Government of India for National Post-Doctoral Fellowship and financial support for this work.

Author Contributions: Pankaj Kumar Singh, Preeti Arya, Ritu Kapoor, Akshay Singh and Rajdeep Jaswal drafted the manuscript. Akshay Nag, Pankaj Kumar Singh and Ritu Kapoor prepared the tables. Preeti Arya, Pankaj Kumar Singh and Akshay Singh constructed the figures. Tilak Raj Sharma edited and made the final manuscript.

Conflicts of Interest: The authors declare no conflict of interest.

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