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# Recent Advances in the Genetic and Biochemical Mechanisms of Rice Resistance to Brown Planthoppers (*Nilaparvata lugens* Stål)

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**Abstract:** Rice (*Oryza sativa* L.) is the staple food of more than half of Earth's population. Brown planthopper (*Nilaparvata lugens* Stål, BPH) is a host-specific pest of rice responsible for inducing major losses in rice production. Utilizing host resistance to control *N. lugens* is considered to be the most cost-effective method. Therefore, the exploration of resistance genes and resistance mechanisms has become the focus of breeders' attention. During the long-term co-evolution process, rice has evolved multiple mechanisms to defend against BPH infection, and BPHs have evolved various mechanisms to overcome the defenses of rice plants. More than 49 BPH-resistance genes/QTLs have been reported to date, and the responses of rice to BPH feeding activity involve various processes, including MAPK activation, plant hormone production, $Ca^{2+}$ flux, etc. Several secretory proteins of BPHs have been identified and are involved in activating or suppressing a series of defense responses in rice. Here, we review some recent advances in our understanding of rice–BPH interactions. We also discuss research progress in controlling methods of brown planthoppers, including cultural management, trap cropping, and biological control. These studies contribute to the establishment of green integrated management systems for brown planthoppers.

**Keywords:** rice; brown planthopper; defense responses; BPH-resistance genes; integrated pest management



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## 1. Introduction

The brown planthopper (*Nilaparvata lugens* Stål, BPH) is a host-specific herbivore that is widespread in Asia, Australia, and the South Pacific islands [1]. *N. lugens* soaks up phloem sap by inserting needle-like stylets into the vascular tissue of rice (*Oryza sativa* L.) [2]. Large amounts of BPHs often gathered in groups to harm plants, and caused wilting, yellowing, and even death of rice plants, as well as "hopperburn" in BPH-susceptible rice fields [3]. BPHs are also vectors of various viruses of rice, such as the grassy stunt virus and ragged stunt virus, which were introduced into rice plants during the *N. lugens* feeding process [4–6]. Direct and indirect economic losses induced by BPH feeding in Asia alone exceed hundreds of millions of dollars on an annual basis [7]. Brown planthoppers have become one of the most serious pests that harm rice production [3].

Currently, the application of chemical insecticides remains the major approach to controlling BPH in the field [8]. However, the widespread use of these compounds is hazardous to human health and the environment and has side effects that impact the natural enemies of BPH [9]. In addition, the indiscriminate use of pesticides can promote

the emergence of insecticide resistance in BPHs [10,11]. Wu et al. found that the insecticide resistance to different insecticides (including imidacloprid, buprofezin, thiamethoxam, pymetrozine fufprole, chlorpyrifos, sulfoxafor, nitenpyram) of 69 *N. lugens* populations collected from eight Chinese provinces improved to varying degrees [12]. This led to a significant reduction in the toxicity efficiency against BPH [13–16]. Therefore, other BPH management strategies that are greener, healthier, and more sustainable must be developed. Utilizing the inherent resistance genes of rice to cultivate resistant rice varieties has been widely considered as the most cost-effective method for sustainable BPH control [17–19].

DNA sequence data show that the host of BPHs began to gradually transfer from *Leersia* to rice approximately 2.5 million years ago [1]. Rice has since evolved sophisticated defense systems to resist BPH infection, and BPHs have evolved various mechanisms to overcome these defenses [1]. Here, we review recent advances in research on the detection of BPH-resistance genes/QTLs, the mechanisms by which rice resists BPH infestations, and the roles of BPH secretory proteins in activating or suppressing rice defenses, and discuss their utilization in diminishing damage caused by brown planthoppers. Additionally, we discuss the research progress in *N. lugens* controlling methods. The insights from this review will enhance our understanding of the survival competition mechanism between rice and BPH and aid the development of strategies to establish green integrated BPH management.

## 2. BPH-Resistance Gene Mapping

The *indica* cultivar Mudgo, the first BPH-resistant rice germplasm, was identified in 1969 by the International Rice Research Institute [20]. *Bph1*, the first BPH-resistance gene identified from Mudgo, was mapped on chromosome 12 [21]. In recent decades, more than 49 BPH-resistance genes/QTLs have been detected due to the development of molecular marker technology and methods for evaluating the resistance of rice to BPHs [2,3,22–24]. Among these 49 genes/QTLs, 33 (*Bph37* from IR64; *Bph38(t)*, *Bph33(t)*, *bph19*, *Bph31*, *Bph44(t)*, *qBph4.3*, *Bph33*, *Bph30*, *Bph41*, *Bph40*, *qBph4.1*, *Bph3*, and *qBph4.2* from IR65482-17, *qBph4.4*, *Bph17*, and *qBph4.2* from Rathu Heenati; *Bph27(t)*, *Bph6*, *Bph44*, *Bph42*, *Bph25*, and *Bph37* from SE382; *Bph32*, *bph4*, *Bph43*, *Bph28(t)*, *bph2*, *bph7*, and *Bph9* from Kaharamana; and *Bph1*, *Bph26*, and *Bph9* from Pokkali) were derived from traditional cultivated rice species; the rest were derived from wild rice varieties, including *Bph13(t)*, *bph11*, *qBph3*, *Bph14*, *qBph4*, and *Bph15* from *O. officinalis*; *Bph12* from *O. latifolia*; *Bph35*, *Bph36*, *Bph27*, and *bph29* from *O. rufipogon*; *Bph21* and *Bph20(t)* from *O. minuta*; *Bph34* from *O. nivara*; and *Bph18* and *Bph10* from *O. australiensis* (Table 1). Rice varieties containing one or more BPH-resistance genes/QTLs have been developed, and the cultivation of these varieties has greatly reduced the loss of rice yield induced by BPH feeding [25].

**Table 1.** BPH-resistance genes/QTLs discovered in rice.

| Gene | Germplasm | Chromosome | Linked Markers | Reference |
|---|---|---|---|---|
| *Bph37* | IR64 | 1L | RM302, YM35 | [26] |
| *Bph38(t)* | Khazar | 1L | 693369, id1012165 | [27] |
| *Bph33(t)* | RP2068 | 1L | RM488, RM11522 | [28] |
| *Bph13(t)* | *O. officinalis* | 3S | AJ09b, AJ09c | [29] |
| *bph19* | AS20-1 | 3S | RM6308, RM3134 | [30] |
| *Bph31* | CR2711-76 | 3L | PA26, RM2334 | [31] |
| *bph11* | *O. officinalis* | 3L | G1318 | [32] |
| *qBph3* | IR02W101 (*O. officinalis*) | 3L | t6, f3, c3-14 | [33] |
| *Bph14* | B5 (*O. officinalis*) | 3L | SM1, G1318 | [34] |
| *Bph44(t)* | IRGC 15344 | 4S | 344-0-6, 344-1-2 | [24] |
| *qBph4.3* | Salkathi | 4S | RM551, RM335 | [35] |
| *Bph33* | Kolayal, Poliyal | 4S | H99, H101 | [36] |
| *Bph30* | AC-1613 | 4S | SSR28, SSR69 | [2] |

**Table 1.** *Cont.*

| Gene | Germplasm | Chromosome | Linked Markers | Reference |
|---|---|---|---|---|
| *Bph41* | SWD10 | 4S | SWRm_01617, SWRm_01522 | [37] |
| *Bph40* | SE232, SE67, C334 | 4S | - | [2] |
| *Bph12* | *O. latifolia* | 4S | RM16459, RM1305 | [3] |
| *qBph4.1* | Rathu Heenati | 4S | - | [38] |
| *Bph3* | Rathu Heenati | 4S | RHD9, RHC10 | [39] |
| *Bph35* | RBPH660 (*O. rufipogon*) | 4S | PSM16, R4M13 | [40] |
| *qBph4.2* | IR65482-17 | 4S | RM261, S1, XC4-27 | [41] |
| *Bph15* | B5 (*O. officinalis*) | 4S | RG1, RG2 | [3] |
| *qBph4.4* | Salkathi | 4S | RM335, RM5633 | [35] |
| *Bph36* | *O. rufipogon* | 4L | S13, X48 | [42] |
| *qBph4* | IR02W101 (*O.officinalis*) | 4S | p17, xc4-27 | [33] |
| *Bph20(t)* | *O. minuta* | 4S | B42, B44 | [43] |
| *Bph17* | Rathu Heenati | 4S | RM8213, RM5953 | [44] |
| *qBph4.2* | Rathu Heenati | 4L | - | [38] |
| *Bph27* | *O. rufipogon* | 4L | RM16766, RM17033 | [42] |
| *Bph27(t)* | Balamawee | 4L | Q52, Q20 | [45] |
| *Bph6* | Swarnalata | 4L | H, Y9 | [46] |
| *Bph44* | Balamawee | 4L | Q31, RM17007 | [24] |
| *Bph34* | *O. nivara* | 4L | RM16994, RM17007 | [47] |
| *Bph42* | SWD10 | 4L | SWRm_01695, SWRm_00328 | [37] |
| *Bph25* | ADR52 | 6S | S00310 | [48] |
| *bph29* | *O. rufipogon* | 6S | BYL8, BID2 | [49] |
| *Bph37* | SE382 | 6S | - | [22] |
| *Bph32* | Ptb33 | 6S | RM19291, RM8072 | [50] |
| *bph4* | Babawee | 6S | RM190, C76A | [51] |
| *Bph43* | IRGC 8678 | 11L | 16-22, 16-30 | [23] |
| *Bph28(t)* | DV85 | 11L | Indel55, Indel66 | [52] |
| *bph2* | ASD7 | 12L | RM7102, RM463 | [53] |
| *bph7* | T12 | 12L | RM3448, RM313 | [54] |
| *Bph10* | *O. australiensis* | 12L | RG457 | [55] |
| *Bph9* | Kaharamana | 12L | RM463, RM5341 | [56] |
| *Bph1* | Mudgo | 12L | em5814N, em2802N | [57] |
| *Bph26* | ADR52 | 12L | DS72B4, DS173B | [58] |
| *Bph18* | *O. australiensis* | 12L | BIM3, BN162 | [59] |
| *Bph9* | Pokkali | 12L | InD2, RsaI | [54] |
| *Bph21* | *O. minuta* | 12L | S12094A, B122 | [43] |

S, short arm of chromosome; L, long arm of chromosome.

Most of the BPH-resistance genes/QTLs identified to date were located on six of twelve chromosomes (chromosomes 1, 3, 4, 6, 11, and 12), and their distribution on chromosomes was clustered (Figure 1). Three genes (*bph11*, *qBph3,* and *Bph14*) were clustered between 35.60 and 35.80 Mb of chromosome 3L [29,32,34]. A total of 21 genes were located on chromosome 4: five genes (*Bph44(t)*, *qBph4.3*, *Bph33*, *Bph30,* and *Bph41*) were clustered between 0.17 and 1.10 Mb on chromosome 4S [2,24,35–37]; 11 genes (*Bph40*, *Bph12*, *qBph4.1*, *Bph3*, *Bph35*, *qBph4.2*, *Bph15*, *Bph36*, *qBph4*, *Bph20(t),* and *Bph17*) were clustered between 4.44 and 9.38 Mb on chromosome 4S [2,3,33,38–44]; and *Bph27(t)*, *Bph6*, *Bph44*, *Bph34,* and *Bph42* were clustered on chromosome 4L between 20.60 and 21.80 Mb [24,37,45–47]. The *Bph37* that from SE382, *Bph25*, *bph29, Bph32* and *bph4* were present on chromosome 6S between 0.21 and 1.47 Mb [22,48–51]. The *Bph43* and *Bph28(t)* genes were clustered between 16.79 and 16.96 Mb of chromosome 11L [23,52]. Some regions of these genes in the same cluster might overlap, indicating that these genes were not the same but were tightly linked, or that they were the same gene. These clustered genes might also constitute different alleles

of the same gene that mediate responses to different BPH populations. In the same region on chromosome 12L, a total of eight BPH-resistance genes have been isolated. Sequence alignment revealed that these genes were alleles, and four allelotypes were identified. An assessment of the BPH resistance of the four allelotypes revealed that the resistance to BPH populations conferred by allelotypes of the same resistance gene varies [54].

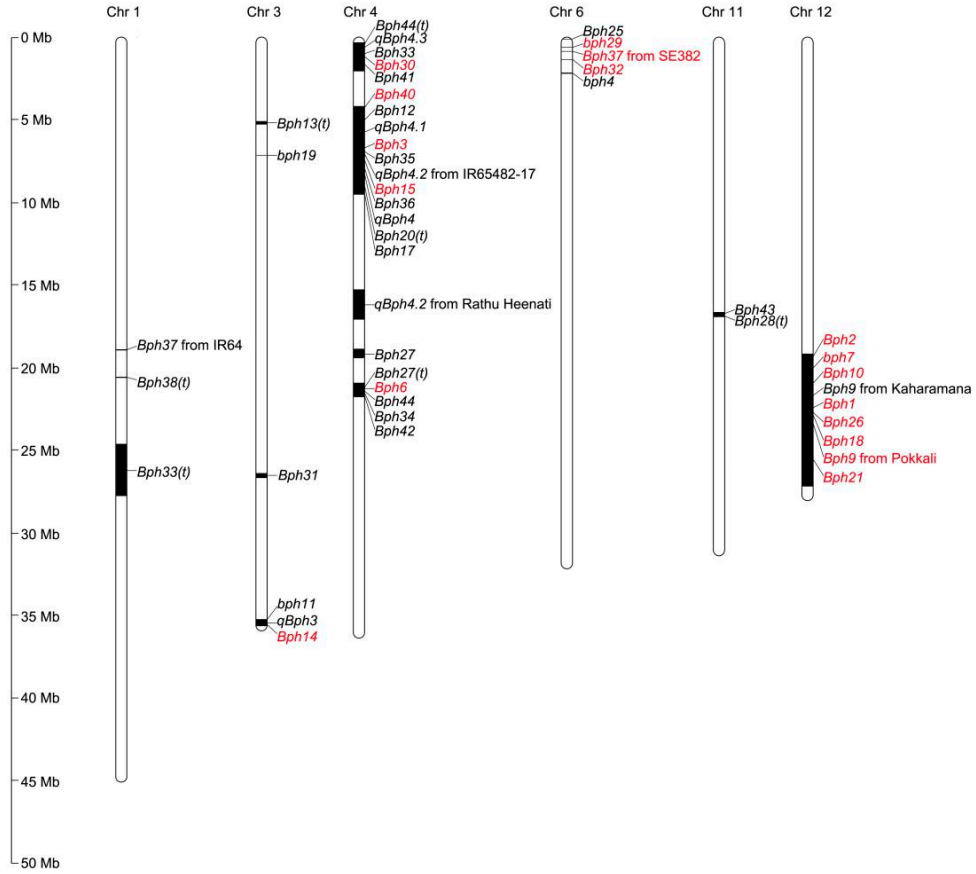


**Figure 1.** Distribution of BPH-resistance genes/QTLs on rice chromosomes. Numbers on the left indicate the physical distance. Black bars represented the position of BPH-resistance genes/QTLs on rice chromosomes. Red represents genes that have been cloned, and black represents genes that have not been cloned.

## 3. Cloning and Mechanisms of BPH-Resistance Genes

A total of 17 BPH-resistance genes have been isolated to date (Table 2). These genes can be classified into seven types based on the types of encoded proteins. Coiled-coil, nucleotide-binding, and leucine-rich repeat (CC–NB–LRR, CNL) protein is encoded by *Bph14* [34]. *Bph1*, *Bph2*, *Bph7*, *Bph9*, *Bph10*, *Bph18*, *Bph21*, *Bph26*, and *Bph37* encode atypical CC–NB–LRR proteins [22,54,59]. *Bph15* and *Bph3* encode lectin receptor-like kinases (LecRKs) [39,60]. Leucine-rich repeat domain (LRD)-containing proteins are encoded by *Bph30* and *Bph40* [2]. *Bph6* encodes an atypical LRR protein [46]. A B3 DNA-binding domain protein is encoded by *bph29* [49]. *Bph32* encodes a short consensus repeat (SCR) domain-containing protein [50]. The high variation in the types of proteins encoded by BPH-resistance genes reflected the high diversity in BPH-resistance mechanisms. These genes have been used to develop resistant rice varieties for the sustainable prevention and control of BPHs [25].

**Table 2.** BPH-resistance genes isolated from rice.

| Gene | Germplasm | Chromosome | Encoded Protein | Defense Mechanism | Reference |
|---|---|---|---|---|---|
| *Bph14* | B5 | 3L | CC-NB-LRR | SA↑, callose deposition | [34] |
| *Bph9* | Pokkali | 12L | CC-NB-NB-LRR | SA↑ | [54] |
| *Bph1* | Mudgo | 12L | CC-NB-NB-LRR | - | [54] |
| *Bph2* | ASD7 | 12L | CC-NB-NB-LRR | - | [54] |
| *bph7* | T12 | 12L | CC-NB-NB-LRR | - | [54] |
| *Bph10* | IR65482-4-136-2-2 | 12L | CC-NB-NB-LRR | - | [54] |
| *Bph18* | IR65482-7-216-1-2 | 12L | CC-NB-NB-LRR | - | [59] |
| *Bph21* | IR71033-121-15 | 12L | CC-NB-NB-LRR | - | [54] |
| *Bph26* | ADR52 | 12L | CC-NB-NB-LRR | - | [58] |
| *Bph37* | SE382 | 6S | CC-NB | - | [22] |
| *Bph6* | Swarnalata | 4L | Atypical LRR | SA↑, JA↑, CK↑, enhanced cell walls | [46] |
| *Bph30* | AC-1613 | 4S | LRD | enhanced cell walls, IAA↓ | [2] |
| *Bph40* | SE232, SE67, C334 | 4S | LRD | enhanced cell walls | [2] |
| *Bph15* | B5 | 4S | Lectin receptor kinase | *OsPR1a*↑, *OsLOX*↑, *OsCHS*↑ | [60] |
| *Bph3* | Rathu Heenati | 4S | Lectin receptor kinase | - | [39] |
| *bph29* | RBPH54 | 6S | B3 DNA-binding | SA↑, JA/ET↓ | [49] |
| *Bph32* | PTB33 | 6S | SCR | - | [50] |

S, short arm of chromosome; L, long arm of chromosome; CC, coiled coil domain; NB, nucleotide-binding domain; LRR, leucine-rich repeat domain; LRD, leucine-rich domain; SCR, short consensus repeat; SA, salicylic acid; JA, jasmonic acid; CK, cytokinin; IAA, indole-3-acetic acid; ET, ethylene; ↑, up-regulation; ↓, down-regulation.

### 3.1. CC–NB–LRR Gene

*Bph14* was the first BPH-resistance gene to be cloned. This gene was isolated from the highly resistant line B5, which is a chromosome fragment infiltration line derived from the wild rice species *Oryza officinalis*. *Bph14* was first mapped on the long arm of chromosome 3 between markers R1925 and R2443 using the segregating population from the cross between B5 and Minghui 63 [3]. Du et al. fine-mapped *Bph14* and ultimately located it within the 34 kb interval between molecular markers SM1 and G1318 [34]. Two candidate genes, *Ra* and *Rb*, are present in this region. Transgenic functional verification revealed that *Ra* was *Bph14*. *Bph14* encodes a typical CNL protein, which is mainly accumulated in the vascular tissue. The salicylic acid (SA) signaling pathway was activated and callose deposition was induced in phloem during BPH feeding on plants expressing *Bph14* [34]. Additional studies have shown that BPH14 interacts with the transcription factors (TFs) OsWRKY46 and OsWRKY72 and activates the expression of the receptor-like cytoplasmic kinase gene *RLCK281* and the callose synthase gene *LOC_Os01g67364.1* in rice [23].

### 3.2. Atypical CC–NB–LRR Genes

A rare CC–NB–LRR protein with two NB domains (CC–NB–NB–LRR, CNNL) was encoded by *Bph9* [54]. Following BPH infestation, the SA signaling pathway was rapidly activated in plants expressing *Bph9* [54]. The CC domain of BPH9 has been shown to confer resistance to attack from BPHs. The NB1 and NB2 domains in BPH9 protein have been shown to be essential for the resistance of BPH9 to BPHs. NB2 domain with the intact NB function motifs repressed the activation of the CC domain. However, the NB1 domain

did not have this function, as its sequence differed greatly from the NB function motifs. The LRR domain is responsible for the activation of BPH9 during BPH infestation [23]. *Bph9* was isolated from the *indica* rice variety Pokkali, and it was located in the interval on chromosome 12L between the markers InD2 and RsaI. The location of *Bph9* overlapped with the position intervals of seven other BPH-resistance genes (*Bph1*, *Bph2*, *Bph7*, *Bph10*, *Bph18*, *Bph21*, and *Bph26*). Genomic sequence alignment and analyses of the chromosomal locations of these genes have shown that the aforementioned eight genes were allelic to each other. Four allelotypes could be classified according to their sequences, and they conferred varying levels of resistance to three brown planthopper populations [54].

Another unusual CC–NB–LRR protein that lacked the LRR domain and only contained CC and NB domains was encoded by *Bph37* [22]. *Bph37* was mapped between 1.20 and 1.57 Mbp on chromosome 6S. In this region, a typical CC–NB–LRR protein was encoded by *LOC_Os06g03500* in the BPH-susceptible varieties Nipponbare and Kasalath. Whereas the premature termination of translation of *LOC_Os06g03500* in BPH-resistance variety SE382 was due to one base inserted in the second exon, which explained the absence of the LRR domain. Functional verification indicated that *LOC_Os06g03500* cloned from SE382 was *Bph37* [22]. The isolation of *Bph37* and studies of the domains of BPH14 and BPH9 suggest that the functions of the CC, NB, and LRR domains in BPH-resistance proteins may vary.

### 3.3. LRD Genes

Recently, the novel BPH-resistance gene *Bph30*, which encodes an LRD protein, was cloned from the cultivated rice variety AC-1613, and it was mainly expressed in the sclerenchyma cells of the rice leaf sheath [2]. BPH30 promotes the deposition of cellulose and hemicellulose in the sclerenchyma cell wall, which increases the cell wall stiffness and sclerenchyma thickness [2]. These structural changes impeded the ability of planthoppers to pierce the sclerenchyma with their stylets and feed on the phloem, thus conferring broad-spectrum resistance to planthoppers in rice [2]. Through the analysis of homologous genes and genome-wide association studies, the *Bph30*-like gene, *Bph40*, was isolated from the cultivated rice varieties SE232, SE67, and C334. *Bph40* encodes an LRD protein that was identified as BPH30. BPH40 has been shown to promote the deposition of cellulose and hemicellulose in the sclerenchyma cell wall, which might be similar to the resistance mechanism of BPH30 [2]. A total of 27 *Bph30*-like genes that encode proteins containing LRDs were identified in the Nipponbare genome. Whether other *Bph30*-like genes confer resistance to BPHs remains unclear. Future studies of *Bph30*-like genes may provide additional genetic resources and aid the development of more efficient strategies for isolating new BPH-resistance genes [2].

### 3.4. LecRK Genes

*Bph15* encodes an LecRK protein. This gene was derived from the resistant line B5, and it was initially mapped to a 0.4 cM interval on the short arm of chromosome 4 [3]. Next, a more refined genetic map was developed, and *Bph15* was located in a 47 kb interval between markers RG1 and RG2 [3]. *Bph15* from this region was isolated, and silencing this gene in rice weakened the anti-xenosis effect of BPHs [60]. *Bph3* was mapped to a 79 kb interval on the short arm of chromosome 4 [39]. The isolation and characterization of *Bph3* have indicated that *Bph3* is actually a cluster of genes encoding three lectin receptor-like kinases (OsLecRK1–OsLecRK3) [39]. Individual genes or a combination of two genes only confer partial resistance to BPHs, whereas the presence of all three genes confers durable and broad-spectrum resistance to BPHs and WBPHs in rice [39]. BPH15 and BPH3 are all localized to the plasma membrane, indicating that these four proteins might be pattern recognition receptors that receive herbivory-associated molecular patterns [23].

### 3.5. Other Types of BPH-Resistance Genes

*Bph6* encodes an atypical LRR protein [46]. This gene was initially mapped on the long arm of chromosome 4 between the simple sequence repeat (SSR) markers Y9 and Y19.

It was derived from the Bangladesh landrace Swarnalata [3]. Guo et al. fine-mapped this gene and isolated it from the interval between the molecular markers H and Y9 [46]. BPH6 interacts with OsEXO70E1 and facilitates exocytosis [46]. *Bph6* confers broad-spectrum resistance to planthoppers by reinforcing the cell wall and activating SA, jasmonic acid (JA), and cytokinin (CK) signaling [46]. Recent studies have shown that BPH6 interacts with OsEXO70H3 and *S*-adenosylmethionine synthetase-like protein (SAMSL), which facilitates SAMSL secretion to the apoplast, where it promotes lignin deposition in the cell wall [61].

*bph29* is a recessive gene that was isolated from RBPH54. *bph29* was previously positioned on chromosome 6S between markers RM435 and RM540 [62]. Subsequent studies reduced the mapping range of *bph29* to 24 kb between markers BYL8 and BID2. A B3 DNA-binding domain protein is encoded by *bph29*. BPH infestation activates the SA signaling pathway, whereas it suppresses the JA/ethylene (ET) signaling pathway in RBPH54 [49]. *Bph32* was initially identified between markers RM19291 and RM8072 on the short arm of chromosome 6. This region was approximately 170 kb and 190 kb in 9311 and Nipponbare, respectively. Bioinformatics and DNA sequence comparison mediated the isolation of *Bph32* from Ptb33 [50]. *Bph32* encodes a protein with an SCR domain, and this protein confers resistance to BPHs by antibiosis [50].

## 4. Responses of Rice to BPH Infection

The host of BPHs started shifting from *Leersia* to rice approximately 0.25 million years ago [1]. BPHs then began to feed specifically on rice plants [3]. Rice plants have evolved multiple mechanisms to resist attack from BPHs (Figure 2) [2,3,46]. Several studies have focused on clarifying the molecular mechanisms of BPH resistance, and these studies have enhanced our understanding of the responses of rice to BPH feeding [3,46].

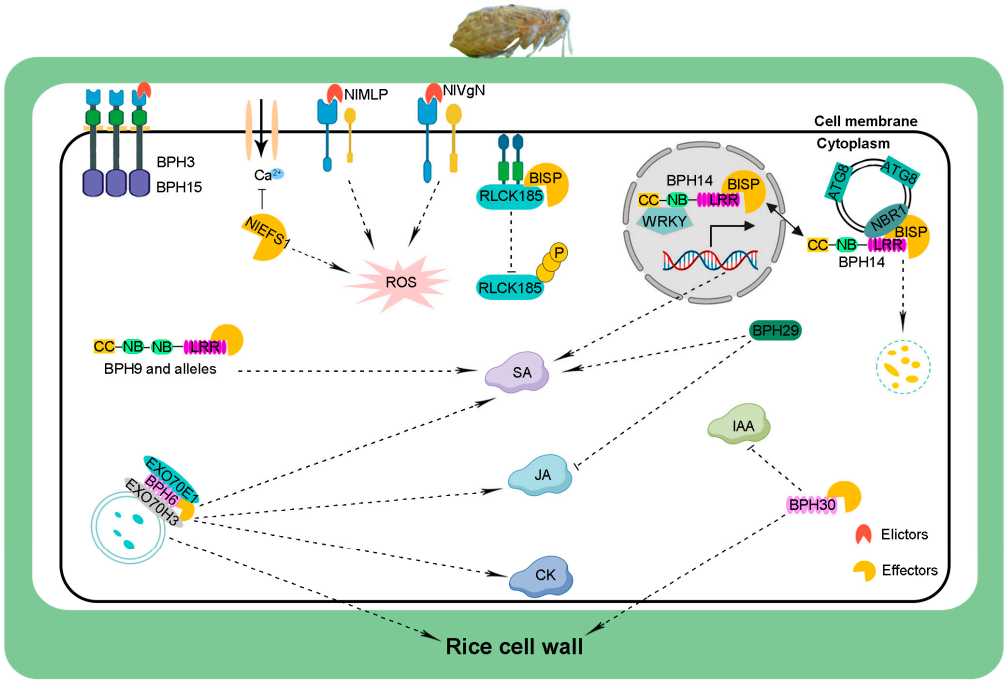


**Figure 2.** Model of rice-brown planthopper interactions. During BPH feeding on rice, elicitors and effectors were secreted into rice cells. Elicitors are perceived by PRRs, activating basic immune responses, such as elevated levels of ROS. However, effectors suppress the first-layer immune responses, such as BISP, which interacts with OsRLCK185 and suppresses its phosphorylation. The BPH-resistance proteins recognize these effectors, triggering second-layer immune reactions, including the activation or suppression of phytohormone signaling pathways and enhancement of the cell wall of leaf sheaths, thus inhibiting BPH from sucking phloem sap. ROS, reactive oxygen species; SA, salicylic acid; JA, jasmonic acid; CK, cytokinin; IAA, indole-3-acetic acid.

### 4.1. MAPK Signal Transduction

Mitogen-activated protein kinases (MAPKs) are a group of very conservative protein kinases in eukaryotes [63,64]. The activation of MAPKs is an early reaction of plant exposure to biotic and abiotic stress [63]. Biochemical and genetic studies have revealed that MAPK cascades connect the different stimuli and downstream responses in plants [23]. Several *OsMAPK* genes have been shown to alter the defense gene expression or phytohormone levels to regulate the resistance of rice to BPHs. *OsMAPK20-5* is a group D MAPK gene, and its expression was rapidly increased following female BPH infestation. BPH feeding increased the contents of ET and nitric oxide (NO) in *OsMAPK20-5* silencing plants, which increased the BPH resistance of rice [64]. *OsMKK3* was significantly induced after BPH infestation. The contents of JA, JA-Ile, and ABA were significantly increased, whereas the SA level was decreased in plants overexpressing *OsMKK3* during BPH feeding, thus compromising the preference for BPH feeding, survival rate, and reproduction [65]. Nanda et al. showed that the expression of *OsMPKs* was remarkably influenced by BPH population type, rice variety, and infestation period [66]. *OsSPL10* negatively regulated the resistance of rice against BPH. In *spl10* mutant plants, genes related to the MAPK signaling pathway were remarkably upregulated during BPH feeding [67]. NlDNAJB9 is a BPH salivary protein that is highly expressed in salivary glands. In the *NlDNAJB9* overexpression plants, MAPK cascades and other defense pathways were induced [68].

### 4.2. Phytohormones

Plant hormones play important roles in rice counteracting BPH. JA and SA are two of the most well-studied hormones involved in BPH resistance [69,70]. In *Bph14*-containing plants, BPH infestation increased SA content and the expression of SA-related genes, such as *EDS1*, *NPR1*, *ICS1*, *PAL*, and *PAD4* [34]. Similar changes have been observed in *Bph9*- or *bph29*-containing plants following BPH feeding [49,54]. Exogenous spraying of SA increased the resistance level of rice to BPHs, suggesting that SA positively regulated BPH resistance [46]. It is generally believed that JA and SA are two antagonistic plant hormones that play opposite roles in the resistance of rice to phloem-sucking insects [61]. However, this might not always be the case. In plants expressing *Bph6*, SA, and JA seemed to participate in the resistance in a synergistic manner [46]. Recent studies have shown that JA-deficient mutants are susceptible to BPHs, and SA deficiency has no effect on BPH resistance [70]. These findings indicate that the functions of JA and SA in the response of rice to attack from BPHs might vary with genotypes and genetic backgrounds.

CK, ET, gibberellins (GA), brassinosteroids (BR), abscisic acid (ABA), and indoleacetic-3-acid (IAA) were also related to the rice defense against BPHs. In *Bph6*-containing plants, the CK content and the expression of synthetic genes increased substantially between 12 h and 24 h following BPH feeding, and the BPH resistance of plants was significantly increased after treatment with CKs [46]. ET is a defense phytohormone that has multiple impacts on insect infestations. *OsACS2*, the 1-aminocyclopropane-1-carboxylic acid (ACC) synthase gene, plays a role in herbivore-induced ET biosynthesis in rice. Knockdown of *OsACS2* decreased the emission of ET and enhanced BPH resistance in rice [71]. The expression of *OsGID1*, a GA receptor gene in rice, was induced during BPH feeding. Overexpression of *OsGID1* improved the BPH resistance level of rice, which was attributed to the increase in the level of lignin and the upregulation expression of three SA pathway-related WRKY genes (*OsWRKY33*, *OsWRKY30*, and *OsWRKY13*) [72]. Exogenous spraying of BR activated JA pathways and suppressed SA pathways, which increased the susceptibility of rice to BPHs [73]. ABA is a key phytohormone that is not only involved in the regulation of plant development but also in the responses to stress. Exogenous treatment with ABA enhanced callose synthase activity but suppressed β-1,3-glucanase activity, which inhibited BPH feeding [74]. Recent studies have shown that IAA negatively regulates the BPH resistance of rice plants [75].

### 4.3. Transcription Factors

The defense responses of rice against BPHs are usually accompanied by the regulation of defense-related gene expression and defense-associated signaling transduction, and TFs play important roles in regulating these processes [76]. The expression of *OsWRKY45* was induced by BPH infestation and played a negative role in the BPH resistance of rice. In *OsWRKY45*-silenced plants, the content of $H_2O_2$ and ET was increased following BPH feeding, thus reducing the feeding, oviposition, and survival rate of BPH and delaying nymph development [77]. *OsWRKY53* positively regulated BPH resistance by increasing $H_2O_2$ production during BPH infestation [78]. *OsMYB30*, an R2R3 MYB TF, directly upregulated the expression of *OsPAL6* and *OsPAL8*, which encoded two key enzymes in the phenylalanine ammonia–lyase pathway and conferred BPH resistance in rice [69]. *OsERF3* encodes an ethylene-responsive factor that reduces the BPH resistance of rice, which might stem from the decrease in the BPH-elicited $H_2O_2$ content [79]. The microarray and RNA sequencing results revealed significant differences in both the number and expression of differentially expressed TFs in resistant and susceptible materials following BPH feeding [80].

### 4.4. Metabolites

Changes in large amounts of metabolites, including primary metabolites, secondary metabolites, and defense compounds, have been observed in rice following BPH infestation [34,75,81]. The contents of amino acids, which are the main metabolites in phloem sap and essential nutrients for BPHs, were significantly reduced in BPH-resistance rice varieties during BPH feeding [75]. This might motivate BPHs to seek BPH-sensitive materials to acquire more nutritious sap [75]. Lipid profiles of rice leaf sheaths showed that the sterol biosynthetic pathway in the susceptible variety Nipponbare and wax biosynthesis and phytol metabolism in resistant *Bph6*-transgenic plants were activated during BPH feeding [61]. A recent study showed that *Bph30* coordinated the flow of primary and secondary metabolites through the shikimate pathway, which conferred BPH resistance [75]. Serotonin is widespread in living organisms, and its synthesis was induced following BPH infestation. The suppression of serotonin biosynthesis increases levels of SA and enhances BPH resistance [81]. Schaftoside is a flavonoid that binds to the BPH CDK1 kinase NlCDK1 and affects its protein kinase activity, which reduces the survival of BPHs [82]. Callose is a well-studied compound involved in BPH resistance [23]. In BPH-resistant varieties, callose deposition blocked the phloem, which inhibited BPH feeding. In susceptible varieties, BPH infestation activated callose-hydrolyzing enzymes, which induced the degradation of callose and facilitated BPH feeding [34,46].

### 4.5. Calcium Signaling

$Ca^{2+}$ is an important second messenger that is widespread in eukaryotes and plays a role in diverse biological processes [83]. $Ca^{2+}$ influx was the earliest response of rice to BPH infestation [3]. *NlSEF1*, which is strongly expressed in the salivary glands of BPHs, encodes a $Ca^{2+}$-binding protein that functions as an effector [84]. During BPH feeding, NlSEF1 is secreted into rice cells and decreases the cytosolic $Ca^{2+}$ content, which is beneficial for the survival and feeding of BPHs [84]. This change in $Ca^{2+}$ concentration is thought to function as a signal that elicits callose synthesis [61].

### 4.6. MicroRNAs

MicroRNAs (miRNAs) are single-stranded non-coding RNAs with a length of approximately 23 nt [85]. miRNAs bind target mRNAs through base complementary pairing to degrade them or inhibit translation, which mediates post-transcriptional gene silencing in both animals and plants [86]. Some studies have shown that miRNAs are involved in the responses of plants to external stimuli [87,88]. Wu et al. identified 23 miRNAs that were differentially expressed in *Bph15* introgression plants (P15) and the susceptible recipient line 9311 (PC) prior to BPH feeding [89]. A total of 104 and 80 differentially expressed miRNAs

were identified in P15 and PC, respectively, following BPH infestation [89]. Significant differences in the abundance and expression levels of differentially expressed miRNAs in BPH-resistance and susceptible varieties before and after BPH feeding have also been identified in several other studies [90,91]. *OsmiR156*, the main regulatory factor of individual plant development, negatively regulates the BPH resistance of rice by increasing levels of JA [92]. Dai et al. found that *OsmiR396* silenced the expression of the *OsGRF8*, reducing the accumulation of transcripts of *OsF3H* and inhibiting flavonoid biosynthesis, thus negatively regulating BPH resistance in rice [93]. The results of these studies suggest that miRNAs play key roles in mediating the resistance of rice to BPHs. Additionally, these studies provided new target genes that could aid the breeding of BPH-resistance varieties.

## 5. BPH-Secreted Proteins That Involved in Rice–BPH Interactions

BPHs are typical piercing sucking insects that penetrate rice tissue with their stylets and suck phloem sap [94]. During the puncturing process, BPHs secrete a large number of proteins into rice tissues [95]. These secreted proteins are essential for the feeding success of BPHs and serve as key signaling molecules for initiating or suppressing rice immune responses (Figure 2) [96,97]. Several advances have been made in our understanding of BPH secretory proteins in recent years, and these studies have provided new insights into the interactions between rice and brown planthoppers [96–98].

### 5.1. BPH Elicitors

Elicitors are BPH secretory proteins that can be recognized by plants and trigger primary immune responses [99]. Mucin-like proteins are widespread in microorganisms [100]. *NlMLP* encodes an *N. lugens*-secreted mucin-like protein (NlMLP) identified from the BPH salivary glands [96]. During BPH feeding, NlMLP is secreted into rice tissues and induces rice defense responses, including the activation of the JA signaling pathway and MAP kinase, $Ca^{2+}$ mobilization, and callose deposition [96]. NlMLP is indispensable for the assembly of stylet sheaths, and its silencing inhibits BPH feeding and performance [96]. Yolk proteins are crucial for egg development. The major precursors of yolk proteins, vitellogenins (Vgs), are usually cut into two segments [97]. NlVgN is the N-terminal subunit of the Vgs of BPHs and is present in saliva and eggs. The secretion of NlVgN into rice tissue induced direct defense responses, such as the production of JA-Ile, JA, cytosolic $Ca^{2+}$, and $H_2O_2$, as well as indirect defense reactions, including the release of volatiles to attract female *A. nilaparvatae* wasps, which are natural enemies of BPHs. NlVg is also essential for the survival of BPHs, and disruptions in *NlVg* expression have a major effect on the feeding, development, and reproduction of BPHs [101]. *N. lugens* salivary protein 1 (NlSP1) was identified from the BPH salivary proteome. The secretion of NlSP1 into plants increases defense-related gene expression, $H_2O_2$ levels, and the deposition of callose [96].

### 5.2. BPH Effectors

Secretory proteins, known as effectors, can weaken defense responses [102,103]. *NlEG1* was identified in salivary glands and encoded an endo-β-1,4-glucanase with endoglucanase activity. The silencing of *NlEG1* reduced the ability of BPH stylets to puncture rice tissue. However, *NlEG1* silencing had no effect on the ability of BPHs to consume an artificial diet. NlEG1 did not induce defense-related responses following its secretion into rice tissues via BPH feeding. These findings suggest that NlEG1 functions as an effector that reduces the resistance conferred by the cell wall [104]. The flavone tricin is widespread in rice plants and can enhance the resistance of rice to BPHs. Gong et al. identified an effector, BPH salivary protein 7 (NlSP7), and found that it decreased the tricin level in rice, which promoted BPH feeding [98]. In addition to suppressing immune responses, effectors can be recognized by specific resistance proteins, activating more intense immune responses [102,105]. The effector BPH14-interacting salivary protein (BISP) was identified in a recent study. BISP interacts with OsRLCK185, which attenuates its autophosphorylation to suppress basal

defense responses. The BPH-resistance protein BPH14 can bind to BISP to activate other resistance pathways to stop BPH feeding [106].

In addition to being able to activate or inhibit the defense responses of rice, the aforementioned secreted elicitors and effectors are also indispensable for the feeding, development, and reproduction of BPHs. Therefore, these proteins may become new targets for controlling BPH.

## 6. Conclusions and Future Perspectives

To date, more than 49 BPH-resistance genes/QTLs have been identified, and the utilization efficiency of these genes in the breeding of BPH-resistance varieties has been low; this has resulted in the homogeneity of BPH-resistance genes in rice varieties [25]. The application of these BPH-resistant varieties will impose enormous selection pressure on BPH to favor the evolution of mechanisms to overcome these resistance genes [107]. BPH populations capable of overcoming the defenses conferred by common BPH-resistance genes have been observed in the laboratory [108,109]. There are several explanations for the lack of utilization efficiency of these BPH-resistance genes. First, the resistance levels of BPH-resistance genes derived from different donors were greatly affected by genetic backgrounds and environments, and this induced significant variation in resistance in rice varieties containing the same resistance gene. Second, traditional approaches for introducing genes are time-consuming and laborious. Few germplasm resources containing different types of BPH-resistance genes and excellent agronomic traits have been developed. Consequently, donor parents with high BPH resistance and good agronomic traits are lacking. Third, the aforementioned resistance genes are mostly incompletely dominant, and the BPH resistance of heterozygous individuals was weaker than that of homozygous individuals [2]; thus, more labor and time will be required for BPH-resistance breeding, especially for hybrid rice breeding. Fourth, the BPH resistance mechanisms remain unclear, which impedes the cultivation of varieties with broad-spectrum resistance and the efficacy of gene pyramiding. However, the discovery of novel types of BPH-resistance genes and defense mechanisms has facilitated major advances in the breeding of BPH-resistance rice varieties [2,46]. A series of BPH-resistance varieties have been cultivated using traditional and molecular-assisted breeding approaches, and this has aided efforts to control BPH populations and enhance rice yield [25].

BPH-susceptible genes and miRNAs in rice merit increased attention, especially in light of the rapid development of genetic engineering technology [110,111]. To date, few BPH susceptibility genes have been identified. *OsERF3* plays a negative role in BPH resistance in rice by suppressing the biosynthesis of $H_2O_2$. The silencing of *OsERF3* enhanced the BPH resistance of rice [79]. *CYP71A1* is a cytochrome P450 gene. Knockout of *CYP71A1* in rice plants blocked serotonin synthesis but increased SA levels, which enhanced BPH resistance [81]. *OsACS2*, which encodes an ACC synthase, negatively regulates BPH resistance. The silencing of *OsACS2* increased the release of 2-heptanol and 2-heptanone, which are two repulsive volatiles, and suppressed BPHs infestation by attracting their natural enemy, *Anagrus nilaparvatae* [71]. Two miRNAs have also been identified to negatively regulate BPH resistance in rice [92,93]. Using genetic engineering methods to reshape the expression of these sensitive genetic materials, making BPH-susceptible varieties with excellent agronomic traits resistant is a fast and cost-effective breeding approach [112]. Although reprogramming these sensitive genetic materials may have deleterious effects on crop growth and yield, an improved understanding of BPH-resistance regulatory networks will greatly aid efforts to develop varieties that are resistant to BPHs without compromising yield [112].

Although studies of the mechanisms by which rice plants resist attack from BPHs can provide important insights that could aid the control of BPHs, studies of BPHs are also needed to achieve effective control of their populations. Many genomic, transcriptomic, and proteomic data on BPHs have been obtained in recent years, and several important elicitors and effectors have been discovered [96]. In addition to their roles in activating

or inhibiting plant immunity, these proteins also play key roles in BPH feeding, and this merits increased research attention [96,97]. RNA interference can be used to silence target gene expression via double-stranded RNA (dsRNA) [113]. Although there are several challenges associated with the use of dsRNA, including its low stability in the environment, low absorption efficiency, and low intracellular delivery efficiency, dsRNA insecticides provide a green approach that could be effective for controlling infestations of insect pests [112,114–117]. This novel type of biopesticide has been used to control various diseases of plants in a sustainable and eco-friendly manner [118–122]. A system that co-delivers insecticide and dsRNA was recently developed that could be used to silence insecticide resistance genes in pests, which would increase the susceptibility of insect herbivores to insecticides [114,123–125]. This provides an optional approach for BPH control.

In addition, other environmentally friendly strategies for BPH management should also be considered. Cultural management is a simple and important eco-friendly method for controlling BPH, although it might not provide immediate results [107]. Crop rotation plays a significant role in restricting population of BPH. For instance, rotating rice with non-host plants or BPH-resistant varieties can remarkably decrease BPH populations [107]. Trap cropping is a useful strategy for BPH control and is commonly used in Asian countries [126]. Trap plants grow together with the major crop, attracting pests away from the major crop or attracting natural enemies, thereby protecting the main plants from pests [126–128]. One study showed that highly susceptible rice plants that were planted 20 d earlier around the main rice field attracted a large number of BPHs to feed, thus reducing the population on the major crop [126]. The pulling power of trap plants, planting time, and required space should be comprehensively considered before choosing trap crops [126]. Biological control, in which natural enemies such as predators and parasitoids play a major role, is another eco-friendly method for rectifying the harm of BPHs [107]. Predators of eggs, such as *Cyrtorhinus lividipennis*, larvae predators, including *Cyrtorhinus lividipennis*, *Lycosa. Pseudoannulata*, and *Migrovelia douglasi*, and predators of both larvae and adults, such as *Synharmoni octomaculata (F.)* and *Paederus fuscipes Curtis*, were found to significantly decrease BPH populations [129,130]. The parasitoids, such as *Oligosita yasumatsui*, *Anagrus* spp., and *Pseudogonatopus* spp. were also identified and considered indispensable factors in the biological control of BPHs [129,131].

In summary, the introduction of major BPH-resistance genes into rice varieties remains the most important method for BPH control. However, studies of the BPH resistance mechanisms of rice and improvements in genetic engineering technologies, as well as changes in agricultural practices will facilitate the development of a green tridimensional defense system to control BPH populations.

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## References


1. Zheng, X.; Zhu, L.; He, G. Genetic and molecular understanding of host rice resistance and *Nilaparvata lugens* adaptation. *Curr. Opin. Insect Sci.* **2021**, *45*, 14–20. [CrossRef]
2. Shi, S.; Wang, H.; Nie, L.; Tan, D.; Zhou, C.; Zhang, Q.; Li, Y.; Du, B.; Guo, J.; Huang, J.; et al. *Bph30* confers resistance to brown planthopper by fortifying sclerenchyma in rice leaf sheaths. *Mol. Plant* **2021**, *14*, 1714–1732. [CrossRef] [PubMed]
3. Muduli, L.; Pradhan, S.K.; Mishra, A.; Bastia, D.N.; Samal, K.C.; Agrawal, P.K.; Dash, M. Understanding brown planthopper resistance in rice: Genetics, biochemical and molecular breeding approaches. *Rice Sci.* **2021**, *28*, 532–546. [CrossRef]
4. Chang, X.; Wang, F.; Fang, Q.; Chen, F.; Yao, H.; Gatehouse, A.; Ye, G. Virus-induced plant volatiles mediate the olfactory behaviour of its insect vectors. *Plant Cell Environ.* **2021**, *44*, 2700–2715. [CrossRef]
5. Sarao, P.S.; Sahi, G.K.; Neelam, K.; Mangat, G.S.; Patra, B.C.; Singh, K. Donors for resistance to brown planthopper *Nilaparvata lugens* Stål from wild rice species. *Rice Sci.* **2016**, *23*, 219–224. [CrossRef]
6. Feng, C.; Wan, Z.; Lan, L.; Le, K. Rice responses and resistance to planthopper-borne viruses at transcriptomic and proteomic levels. *Curr. Issues Mol. Biol.* **2015**, *19*, 43–52.
7. Min, S.; Lee, S.W.; Choi, B.R.; Lee, S.H.; Kwon, D.H. Insecticide resistance monitoring and correlation analysis to select appropriate insecticides against *Nilaparvata lugens* (Stål), a migratory pest in Korea. *J. Asia-Pac. Entomol.* **2014**, *17*, 711–716. [CrossRef]
8. Lu, K.; Chen, X.; Liu, W.; Zhang, Z.; Wang, Y.; You, K.; Li, Y.; Zhang, R.; Zhou, Q. Characterization of heat shock protein 70 transcript from *Nilaparvata lugens* Stål: Its response to temperature and insecticide stresses. *Pestic. Biochem. Phys.* **2017**, *42*, 102–110. [CrossRef]
9. Alam, M.J.; Das, G. Toxicity of insecticides to predators of rice brown planthopper: Wolf spider and carabid beetle. *J. Sci. Food Agric.* **2020**, *3*, 9–13. [CrossRef]
10. Wu, J.; Ge, L.; Liu, F.; Song, Q.; Stanley, D. Pesticide-induced planthopper population resurgence in rice cropping systems. *Annu. Rev. Entomol.* **2020**, *65*, 409–429. [CrossRef]
11. Ge, L.Q.; Huang, L.J.; Yang, G.Q.; Song, Q.S.; Stanley, D.; Gurr, G.M.; Wu, J.C. Molecular basis for insecticide-enhanced thermotolerance in the brown planthopper *Nilaparvata lugens* Stål (Hemiptera: Delphacidae). *Mol. Ecol.* **2013**, *22*, 5624–5634. [CrossRef]
12. Wu, S.; Zeng, B.; Zheng, C.; Mu, X.; Zhang, Y.; Hu, J.; Zhang, S.; Gao, C.; Shen, J. The evolution of insecticide resistance in the brown planthopper (*Nilaparvata lugens* Stål) of China in the period 2012–2016. *Sci. Rep.* **2018**, *8*, 4586. [CrossRef]
13. Zeng, Q.; Yu, C.; Chang, X.; Wan, Y.; Ba, Y.; Li, C.; Lv, H.; Guo, Z.; Cai, T.; Ren, Z.; et al. $CeO_2$ nanohybrid as a synergist for insecticide resistance management. *Chem. Eng. J.* **2022**, *446*, 137074. [CrossRef]
14. Jin, R.; Wang, Y.; He, B.; Zhang, Y.; Cai, T.; Wan, H.; Jin, B.R.; Li, J. Activator protein-1 mediated *CYP6ER1* overexpression in the clothianidin resistance of *Nilaparvata lugens* (Stål). *Pest Manag. Sci.* **2021**, *77*, 4476–4482. [CrossRef] [PubMed]
15. Liao, X.; Jin, R.; Zhang, X.; Ali, E.; Mao, K.; Xu, P.; Li, J.; Wan, H. Characterization of sulfoxaflor resistance in the brown planthopper, *Nilaparvata lugens* (Stål). *Pest Manag. Sci.* **2019**, *75*, 1646–1654. [CrossRef] [PubMed]
16. Jin, R.; Mao, K.; Liao, X.; Xu, P.; Li, Z.; Ali, E.; Wan, H.; Li, J. Overexpression of *CYP6ER1* associated with clothianidin resistance in *Nilaparvata lugens* (Stål). *Pestic. Biochem. Physiol.* **2019**, *154*, 39–45. [CrossRef]
17. Sharma, H.C. Host plant resistance to insects: Modern approaches and limitations. *Plant Prot. Assoc. India* **2007**, *35*, 179–184.
18. Gurr, G.M.; Liu, J.; Read, D.; Catindig, J.; Cheng, J.A.; Lan, L.P.; Heong, K.L. Parasitoids of Asian rice planthopper (Hemiptera: Delphacidae) pests and prospects for enhancing biological control by ecological engineering. *Ann. Appl. Biol.* **2015**, *158*, 149–176. [CrossRef]
19. Khush, G.S. Green revolution: The way forward. *Nat. Rev. Genet.* **2001**, *2*, 815–822. [CrossRef]
20. Pathak, M.; Cheng, C.; Fortuno, M. Resistance to *Nephotettix impicticeps* and *Nilaparvata lugens* in varieties of rice. *Nature* **1969**, *223*, 502–504. [CrossRef]
21. Hirabayashi, H.; Ogawas, T. RFLP mapping of Bph-1 (brown planthopper resistance gene) in rice. *Jpn. J. Breed.* **1995**, *45*, 369–371. [CrossRef]
22. Zhou, C.; Zhang, Q.; Chen, Y.; Huang, J.; Guo, Q.; Li, Y.; Wang, W.; Qiu, Y.; Guan, W.; Zhang, J.; et al. Balancing selection and wild gene pool contribute to resistance in global rice germplasm against planthopper. *J. Integr. Plant Biol.* **2021**, *63*, 1695–1711. [CrossRef] [PubMed]
23. Simon, E.V.; Hechanova, S.L.; Hernandez, J.E.; Li, C.P.; Tülek, A.; Ahn, E.K.; Jairin, J.; Choi, I.R.; Sundaram, R.M.; Jena, K.K.; et al. Available cloned genes and markers for genetic improvement of biotic stress resistance in rice. *Front. Plant Sci.* **2023**, *14*, 1247014. [CrossRef] [PubMed]
24. Kiswanto, I.; Soetopo, L.; Adiredjo, A.L. Identification of novel candidate of brown planthopper resistance gene *Bph44* in rice (*Oryza sativa* L.). *Genome* **2022**, *65*, 505–511. [CrossRef]
25. Liu, M.; Fan, F.; He, S.; Guo, Y.; Chen, G.; Li, N.; Li, N.; Yuan, H.; Si, F.; Yang, F.; et al. Creation of elite rice with high-yield, superior-quality and high resistance to brown planthopper based on molecular design. *Rice* **2022**, *15*, 17. [CrossRef] [PubMed]
26. Yang, M.; Cheng, L.; Yan, L.; Shu, W.; Wang, X.; Qiu, Y. Mapping and characterization of a quantitative trait locus resistance to the brown planthopper in the rice variety IR64. *Hereditas* **2019**, *156*, 22. [CrossRef] [PubMed]
27. Balachiranjeevi, C.H.; Prahalada, G.D.; Mahender, A.; Jamaloddin, M.; Sevilla, M.A.L.; Marfori-Nazarea, C.M.; Vinarao, R.; Sushanto, U.; Baehaki, S.E.; Li, Z.; et al. Identification of a novel locus, *BPH38(t)*, conferring resistance to brown planthopper (*Nilaparvata lugens* Stål.) using early backcross population in rice (*Oryza sativa* L.). *Euphytica* **2019**, *215*, 185. [CrossRef]

28. Naik, S.B.; Divya, D.; Sahu, N.; Sundaram, R.M.; Sarao, P.S.; Singh, K.; Lakshmi, V.J.; Bentur, J.S. A new gene *Bph33(t)* conferring resistance to brown planthopper (BPH), *Nilaparvata lugens* (Stål) in rice line RP2068-18-3-5. *Euphytica* **2018**, *214*, 53. [CrossRef]
29. Renganayaki, K.; Fritz, A.K.; Sadasivam, S.; Pammi, S.; Harrington, S.E.; McCouch, S.R.; Kumar, S.M.; Reddy, A.S. Mapping and progress toward map-based cloning of brown planthopper biotype-4 resistance gene introgressed from *Oryza officinalis* into cultivated rice, *O. sativa*. *Crop Sci.* **2002**, *42*, 2112–2117. [CrossRef]
30. Chen, J.; Wang, L.; Pang, X.; Pan, Q. Genetic analysis and fine mapping of a rice brown planthopper (*Nilaparvata lugens* Stål) resistance gene *bph19(t)*. *Mol. Genet. Genom.* **2006**, *275*, 321–329. [CrossRef]
31. Prahalada, G.D.; Shivakumar, N.; Lohithaswa, H.C.; Gowda, D.K.S.; Ramkumar, G.; Kim, S.R.; Ramachandra, C.; Hittalmani, S.; Mohapatra, T.; Jena, K.K. Identification and fine mapping of a new gene, *BPH31* conferring resistance to brown planthopper biotype 4 of India to improve rice, *Oryza sativa* L. *Rice* **2017**, *10*, 41. [CrossRef]
32. Hirabayashi, H.; Angeles, E.R.; Kaji, R.; Ogawa, T.; Brar, D.S.; Khush, G.S. Identification of a brown planthopper resistance gene derived from *O. officinalis* using molecular markers in rice. *Breed. Sci.* **1998**, *48*, 82.
33. Hu, J.; Xiao, C.; Cheng, M.; Gao, G.; Zhang, Q.; He, Y. Fine mapping and pyramiding of brown planthopper resistance genes *QBph3* and *QBph4* in an introgression line from wild rice *O. officinalis*. *Mol. Breed.* **2015**, *35*, 3. [CrossRef]
34. Du, B.; Zhang, W.; Liu, B.; Hu, J.; Wei, Z.; Shi, Z.; He, R.; Zhu, L.; Chen, R.; Han, B.; et al. Identification and characterization of *Bph14*, a gene conferring resistance to brown planthopper in rice. *Proc. Natl. Acad. Sci. USA* **2009**, *106*, 22163–22168. [CrossRef]
35. Mohanty, S.K.; Panda, R.S.; Mohapatra, S.L.; Nanda, A.; Behera, L.; Jena, M.; Sahu, R.K.; Sahu, S.C.; Mohapatra, T. Identification of novel quantitative trait loci associated with brown planthopper resistance in the rice landrace Salkathi. *Euphytica* **2017**, *213*, 38. [CrossRef]
36. Hu, J.; Chang, X.; Zou, L.; Tang, W.; Wu, W. Identification and fine mapping of *Bph33*, a new brown planthopper resistance gene in rice (*Oryza sativa* L.). *Rice* **2018**, *11*, 55. [CrossRef]
37. Tan, H.; Palyam, S.; Gouda, J.; Kumar, P.P.; Chellian, S.K. Identification of two QTLs, BPH41 and BPH42, and their respective gene candidates for brown planthopper resistance in rice. *Sci. Rep.* **2022**, *12*, 18538. [CrossRef] [PubMed]
38. Kamolsukyeunyong, W.; Ruengphayak, S.; Chumwong, P.; Kusumawati, L.; Chaichoompu, E.; Jamboonsri, W.; Saensuk, C.; Phoonsiri, K.; Toojinda, T.; Vanavichit, A. Identification of spontaneous mutation for broad-spectrum brown planthopper resistance in a large, long-term fast neutron mutagenized rice population. *Rice* **2019**, *12*, 16. [CrossRef]
39. Liu, Y.; Wu, H.; Chen, H.; Liu, Y.; He, J.; Kang, H.; Sun, Z.; Pan, G.; Wang, Q.; Hu, J.; et al. A gene cluster encoding lectin receptor kinases confers broad-spectrum and durable insect resistance in rice. *Nat. Biotechnol.* **2015**, *33*, 301–305. [CrossRef] [PubMed]
40. Zhang, Y.; Qin, G.; Ma, Q.; Wei, M.; Yang, X.; Ma, Z.; Liang, H.; Liu, C.; Li, Z.; Liu, F.; et al. Identification of a major resistance locus *Bph35* to brown planthopper in rice (*Oryza sativa* L.). *Rice Sci.* **2020**, *27*, 237–245.
41. Hu, J.; Xiao, C.; Cheng, M.; Gao, G.; Zhang, Q.; He, Y. A new finely mapped *Oryza australiensis*-derived QTL in rice confers resistance to brown planthopper. *Gene* **2015**, *561*, 132–137. [CrossRef]
42. Li, Z.; Xue, Y.; Zhou, H.; Li, Y.; Usman, B.; Jiao, X.; Wang, X.; Liu, F.; Qin, B.; Li, R.; et al. High-resolution mapping and breeding application of a novel brown planthopper resistance gene derived from wild rice (*Oryza rufipogon* Griff). *Rice* **2019**, *12*, 41. [CrossRef]
43. Rahman, M.L.; Jiang, W.Z.; Chu, S.H.; Qiao, Y.L.; Ham, T.H.; Woo, M.O.; Lee, J.; Khanam, M.S.; Chin, J.H.; Jeung, J.U.; et al. High-resolution mapping of two rice brown planthopper resistance genes, *Bph20(t)* and *Bph21(t)*, originating from *Oryza minuta*. *Theor. Appl. Genet.* **2009**, *119*, 1237–1246. [CrossRef]
44. Sun, L.; Su, C.; Wang, C.; Zhai, H.; Wan, J. Mapping of a major resistance gene to the brown planthopper in the rice cultivar Rathu Heenati. *Breed. Sci.* **2005**, *55*, 391–396. [CrossRef]
45. He, J.; Liu, Y.; Liu, Y.; Jiang, L.; Wu, H.; Kang, H.; Liu, S.; Chen, L.; Liu, X.; Cheng, X.; et al. High-resolution mapping of brown planthopper (BPH) resistance gene *Bph27(t)* in rice (*Oryza sativa* L.). *Mol. Breed.* **2013**, *31*, 549–557. [CrossRef]
46. Guo, J.; Xu, C.; Wu, D.; Zhao, Y.; Qiu, Y.; Wang, X.; Ouyang, Y.; Cai, B.; Liu, X.; Jing, S.; et al. *Bph6* encodes an exocyst-localized protein and confers broad resistance to planthoppers in rice. *Nat. Genet.* **2018**, *50*, 297–306. [CrossRef] [PubMed]
47. Kumar, K.; Sarao, P.S.; Bhatia, D.; Neelam, K.; Kaur, A.; Mangat, G.S.; Brar, D.S.; Singh, K. High-resolution genetic mapping of a novel brown planthopper resistance locus, *Bph34* in *Oryza sativa* L. × *Oryza nivara* (Sharma & Shastry) derived interspecific $F_2$ population. *Theor. Appl. Genet.* **2018**, *131*, 1163–1171. [PubMed]
48. Myint, K.K.M.; Fujita, D.; Matsumura, M.; Sonoda, T.; Yoshimura, A.; Yasui, H. Mapping and pyramiding of two major genes for resistance to the brown planthopper (*Nilaparva talugens* [Stål]) in the rice cultivar ADR52. *Theor. Appl. Genet.* **2012**, *124*, 495–504. [CrossRef]
49. Wang, Y.; Cao, L.; Zhang, Y.; Cao, C.; Liu, F.; Huang, F.; Qiu, Y.; Li, R.; Luo, X. Map-based cloning and characterization of *BPH29*, a B3 domain-containing recessive gene conferring brown planthopper resistance in rice. *J. Exp. Bot.* **2015**, *66*, 6035–6045. [CrossRef]
50. Ren, J.; Gao, F.; Wu, X.; Lu, X.; Zeng, L.; Lv, J.; Su, X.; Luo, H.; Ren, G. *Bph32*, a novel gene encoding an unknown SCR domain-containing protein, confers resistance against the brown planthopper in rice. *Sci. Rep.* **2016**, *6*, 37645. [CrossRef]
51. Kawaguchi, M.; Murata, K.; Ishii, T.; Takumi, S.; Mori, N.; Nakamura, C. Assignment of a brown planthopper (*Nilaparvata lugens* Stål) resistance gene *bph4* to the rice chromosome 6. *Breed. Sci.* **2001**, *51*, 13–18. [CrossRef]
52. Wu, H.; Liu, Y.; He, J.; Liu, Y.; Jiang, L.; Liu, L.; Wang, C.; Cheng, X.; Wan, J. Fine mapping of brown planthopper (*Nilaparvata lugens* Stål) resistance gene *Bph28(t)* in rice (*Oryza sativa* L.). *Mol. Breed.* **2014**, *33*, 909–918. [CrossRef]

53. Sun, L.; Wang, C.; Su, C.; Liu, Y.; Zhai, H.; Wan, J. Mapping and marker-assisted selection of a brown planthopper resistance gene *bph2* in rice (*Oryza sativa* L.). *Acta Genet. Sin.* **2006**, *33*, 717–723. [CrossRef] [PubMed]
54. Zhao, Y.; Huang, J.; Wang, Z.; Jing, S.; Wang, Y.; Ouyang, Y.; Cai, B.; Xin, X.; Liu, X.; Zhang, C.; et al. Allelic diversity in an NLR gene *BPH9* enables rice to combat planthopper variation. *Proc. Natl. Acad. Sci. USA* **2016**, *113*, 12850–12855. [CrossRef]
55. Ishii, T.; Brar, D.S.; Multani, D.S.; Khush, G.S. Molecular tagging of genes for brown planthopper resistance and earliness introgressed from *Oryza australiensis* into cultivated rice *O. sativa*. *Genome* **1994**, *37*, 217–221. [CrossRef]
56. Su, C.; Zhai, H.; Wang, C.; Sun, L.; Wan, J. SSR mapping of brown planthopper resistance gene *Bph9* in Kaharamana, an *indica* rice (*Oryza sativa* L.). *Acta Genet. Sin.* **2006**, *33*, 262–268. [CrossRef] [PubMed]
57. Sharma, P.N.; Ketipearachchi, Y.; Murata, K.; Torii, A.; Takumi, S.; Mori, N.; Nakamura, C. RFLP/AFLP mapping of a brown planthopper (*Nilaparvata lugens* Stål) resistance gene *Bph1* in rice. *Euphytica* **2002**, *129*, 109–117. [CrossRef]
58. Tamura, Y.; Hattori, M.; Yoshioka, H.; Yoshioka, M.; Takahashi, A.; Wu, J.; Sentoku, N.; Yasui, H. Map-based cloning and characterization of a brown planthopper resistance gene *BPH26* from *Oryza sativa* L. ssp. *indica cultivar ADR52*. *Sci. Rep.* **2014**, *4*, 5872.
59. Ji, H.; Kim, S.R.; Kim, Y.H.; Suh, J.P.; Park, H.M.; Sreenivasulu, N.; Misra, G.; Kim, S.M.; Hechanova, S.L.; Kim, H.; et al. Map-based cloning and characterization of the *BPH18* gene from wild rice conferring resistance to brown planthopper (BPH) insect pest. *Sci. Rep.* **2016**, *6*, 34376. [CrossRef]
60. Cheng, X.; Wu, Y.; Guo, J.; Du, B.; Chen, R.; Zhu, L.; He, G. A rice lectin receptor-like kinase that is involved in innate immune responses also contributes to seed germination. *Plant J.* **2013**, *76*, 687–698. [CrossRef]
61. Mishra, A.; Barik, S.R.; Pandit, E.; Yadav, S.S.; Das, S.R.; Pradhan, S.K. Genetics, mechanisms and deployment of brown planthopper resistance genes in rice. *Crit. Rev. Plant Sci.* **2022**, *41*, 91–127. [CrossRef]
62. Yang, L.; Li, R.; Li, Y.; Huang, F.; Chen, Y.; Huang, S.; Huang, L.; Liu, C.; Ma, Z.; Huang, D.; et al. Genetic mapping of *bph20(t)* and *bph21(t)* loci conferring brown planthopper resistance to *Nilaparvata lugens* Stål in rice (*Oryza sativa* L.). *Euphytica* **2012**, *183*, 161–171. [CrossRef]
63. Hettenhausen, C.; Schuman, M.C.; Wu, J. MAPK signaling: A key element in plant defense response to insects. *Insect Sci.* **2015**, *22*, 157–164. [CrossRef] [PubMed]
64. Li, J.; Liu, X.; Wang, Q.; Huangfu, J.; Schuman, M.C.; Lou, Y. A group D MAPK protects plants from autotoxicity by suppressing herbivore-induced defense signaling. *Plant Physiol.* **2019**, *179*, 1386–1401. [CrossRef] [PubMed]
65. Zhou, S.; Chen, M.; Zhang, Y.; Gao, Q.; Noman, A.; Wang, Q.; Li, H.; Chen, L.; Zhou, P.; Lu, J.; et al. OsMKK3, a stress-responsive protein kinase, positively regulates rice resistance to *Nilaparvata lugens* via phytohormone dynamics. *Int. J. Mol. Sci.* **2019**, *20*, 3023. [CrossRef] [PubMed]
66. Nanda, S.; Wan, P.; Yuan, S.; Lai, F.; Wang, W.; Fu, Q. Differential responses of *OsMPK*s in IR56 rice to two BPH populations of different virulence levels. *Int. J. Mol. Sci.* **2018**, *19*, 4030. [CrossRef] [PubMed]
67. Lu, L.; Sun, Z.; Wang, R.; Du, Y.; Zhang, Z.; Lan, T.; Song, Y.; Zeng, R. Integration of transcriptome and metabolome analyses reveals the role of *OsSPL10* in rice defense against brown planthopper. *Plant Cell Rep.* **2023**, *42*, 2023–2038. [CrossRef] [PubMed]
68. Gao, H.; Lin, X.; Yuan, X.; Zou, J.; Zhang, H.; Zhang, Y.; Liu, Z. The salivary chaperone protein NlDNAJB9 of *Nilaparvata lugens* activated plant immune responses. *J. Exp. Bot.* **2023**, *27*, erad154.
69. He, J.; Liu, Y.; Yuan, D.; Duan, M.; Liu, Y.; Shen, Z.; Yang, C.; Qiu, Z.; Liu, D.; Wen, P.; et al. An R2R3 MYB transcription factor confers brown planthopper resistance by regulating the phenylalanine ammonia-lyase pathway in rice. *Proc. Natl. Acad. Sci. USA* **2020**, *117*, 271–277. [CrossRef] [PubMed]
70. Xu, J.; Wang, X.; Zu, H.; Zeng, X.; Baldwin, I.T.; Lou, Y.; Li, R. Molecular dissection of rice phytohormone signaling involved in resistance to a piercing-sucking herbivore. *New Phytol.* **2021**, *230*, 1639–1652. [CrossRef]
71. Lu, J.; Li, J.; Ju, H.; Liu, X.; Erb, M.; Wang, X.; Lou, Y. Contrasting effects of ethylene biosynthesis on induced plant resistance against a chewing and a piercing-sucking herbivore in rice. *Mol. Plant* **2014**, *7*, 1670–1682. [CrossRef]
72. Chen, L.; Cao, T.; Zhang, J.; Lou, Y. Overexpression of *OsGID1* enhances the resistance of rice to the brown planthopper *Nilaparvata lugens*. *Int. J. Mol. Sci.* **2018**, *19*, 2744. [CrossRef]
73. Pan, G.; Liu, Y.; Ji, L.; Zhang, X.; He, J.; Huang, J.; Qiu, Z.; Liu, D.; Sun, Z.; Xu, T.; et al. Brassinosteroids mediate susceptibility to brown planthopper by integrating with the salicylic acid and jasmonic acid pathways in rice. *J. Exp. Bot.* **2018**, *69*, 4433–4442. [CrossRef]
74. Liu, J.; Du, H.; Ding, X.; Zhou, Y.; Xie, P.; Wu, J. Mechanisms of callose deposition in rice regulated by exogenous abscisic acid and its involvement in rice resistance to *Nilaparvata lugens* Stål (Hemiptera: Delphacidae). *Pest Manag. Sci.* **2017**, *73*, 2559–2568. [CrossRef]
75. Shi, S.; Zha, W.; Yu, X.; Wu, Y.; Li, S.; Xu, H.; Li, P.; Li, C.; Liu, K.; Chen, J.; et al. Integrated transcriptomics and metabolomics analysis provide insight into the resistance response of rice against brown planthopper. *Front. Plant Sci.* **2023**, *14*, 1213257. [CrossRef]
76. Alves, M.S.; Dadalto, S.P.; Gonçalves, A.B.; de Souza, G.B.; Barros, V.A.; Fietto, L.G. Transcription factor functional protein-protein interactions in plant defense responses. *Proteomes* **2014**, *2*, 85–106. [CrossRef]
77. Huangfu, J.; Li, J.; Li, R.; Ye, M.; Kuai, P.; Zhang, T.; Lou, Y. The transcription factor OsWRKY45 negatively modulates the resistance of rice to the brown planthopper *Nilaparvata lugens*. *Int. J. Mol. Sci.* **2016**, *17*, 697. [CrossRef] [PubMed]

78. Hu, L.; Ye, M.; Li, R.; Lou, Y. OsWRKY53, a versatile switch in regulating herbivore-induced defense responses in rice. *Plant Signal. Behav.* **2016**, *11*, e1169357. [CrossRef] [PubMed]
79. Lu, J.; Ju, H.; Zhou, G.; Zhu, C.; Erb, M.; Wang, X.; Wang, P.; Lou, Y. An EAR-motif-containing ERF transcription factor affects herbivore-induced signaling, defense and resistance in rice. *Plant J.* **2011**, *68*, 583–596. [CrossRef] [PubMed]
80. Wang, Y.; Guo, H.; Li, H.; Zhang, H.; Miao, X. Identification of transcription factors potential related to brown planthopper resistance in rice via microarray expression profiling. *BMC Genom.* **2012**, *13*, 687. [CrossRef]
81. Lu, H.; Luo, T.; Fu, H.; Wang, L.; Tan, Y.; Huang, J.; Wang, Q.; Ye, G.; Gatehouse, A.M.R.; Lou, Y.; et al. Resistance of rice to insect pests mediated by suppression of serotonin biosynthesis. *Nat. Plants* **2018**, *4*, 338–344. [CrossRef]
82. Hao, P.; Feng, Y.; Zhou, Y.; Song, X.; Li, H.; Ma, Y.; Ye, C.; Yu, X. Schaftoside interacts with NlCDK1 protein: A mechanism of rice resistance to brown planthopper, *Nilaparvata lugens*. *Front. Plant Sci.* **2018**, *9*, 710. [CrossRef]
83. Lecourieux, D.; Ranjeva, R.; Pugin, A. Calcium in plant defence-signalling pathways. *New Phytol.* **2006**, *171*, 249–269. [CrossRef]
84. Ye, W.; Yu, H.; Jian, Y.; Zeng, J.; Ji, R.; Chen, H.; Lou, Y. A salivary EF-hand calcium-binding protein of the brown planthopper *Nilaparvata lugens* functions as an effector for defense responses in rice. *Sci. Rep.* **2017**, *7*, 40498. [CrossRef] [PubMed]
85. Bartel, D.P. MicroRNAs: Target recognition and regulatory functions. *Cell* **2009**, *136*, 215–233. [CrossRef] [PubMed]
86. Axtell, M.J.; Meyers, B.C. Revisiting criteria for plant microRNA annotation in the era of big data. *Plant Cell* **2018**, *30*, 272–284. [CrossRef] [PubMed]
87. Chen, X.; Jiang, L.; Zheng, J.; Chen, F.; Wang, T.; Wang, M.; Tao, Y.; Wang, H.; Hong, Z.; Huang, Y.; et al. A missense mutation in large grain size 1 increases grain size and enhances cold tolerance in rice. *J. Exp. Bot.* **2019**, *70*, 3851–3866. [CrossRef] [PubMed]
88. Kryovrysanaki, N.; James, A.; Tselika, M.; Bardani, E.; Kalantidis, K. RNA silencing pathways in plant development and defense. *Int. J. Dev. Biol.* **2022**, *66*, 163–175. [CrossRef] [PubMed]
89. Wu, Y.; Lv, W.; Hu, L.; Rao, W.; Zeng, Y.; Zhu, L.; He, Y.; He, G. Identification and analysis of brown planthopper-responsive microRNAs in resistant and susceptible rice plants. *Sci. Rep.* **2017**, *7*, 8712. [CrossRef]
90. Nanda, S.; Yuan, S.; Lai, F.; Wang, W.; Fu, Q.; Wan, P. Identification and analysis of miRNAs in IR56 rice in response to BPH infestations of different virulence levels. *Sci. Rep.* **2020**, *10*, 19093. [CrossRef]
91. Lü, J.; Liu, J.; Chen, L.; Sun, J.; Su, Q.; Li, S.; Yang, J.; Zhang, W. Screening of brown planthopper resistant miRNAs in rice and their roles in regulation of brown planthopper fecundity. *Rice Sci.* **2022**, *29*, 559–568.
92. Ge, Y.; Han, J.; Zhou, G.; Xu, Y.; Ding, Y.; Shi, M.; Guo, C.; Wu, G. Silencing of miR156 confers enhanced resistance to brown planthopper in rice. *Planta* **2018**, *248*, 813–826. [CrossRef]
93. Dai, Z.; Tan, J.; Zhou, C.; Yang, X.; Yang, F.; Zhang, S.; Sun, S.; Miao, X.; Shi, Z. The OsmiR396-OsGRF8-OsF3H-flavonoid pathway mediates resistance to the brown planthopper in rice (*Oryza sativa*). *Plant Biotechnol. J.* **2019**, *17*, 1657–1669. [CrossRef] [PubMed]
94. Will, T.; Furch, A.C.; Zimmermann, M.R. How phloem-feeding insects face the challenge of phloem-located defenses. *Front. Plant Sci.* **2013**, *4*, 336. [CrossRef] [PubMed]
95. Huang, H.; Lu, J.; Li, Q.; Bao, Y.; Zhang, C. Combined transcriptomic/proteomic analysis of salivary gland and secreted saliva in three planthopper species. *J. Proteom.* **2018**, *172*, 25–35. [CrossRef]
96. Hu, C.; Li, Y.; Liu, Y.; Hao, G.; Yang, X. Molecular interaction network of plant-herbivorous insects. *Adv. Agrochem* 2023, *in press*. [CrossRef]
97. Tufail, M.; Takeda, M. Molecular characteristics of insect vitellogenins. *J. Insect Physiol.* **2008**, *54*, 1447–1458. [CrossRef]
98. Gong, G.; Yuan, L.; Li, Y.; Xiao, H.; Li, Y.; Zhang, Y.; Wu, W.; Zhang, Z. Salivary protein 7 of the brown planthopper functions as an effector for mediating tricin metabolism in rice plants. *Sci. Rep.* **2022**, *12*, 3205. [CrossRef]
99. Snoeck, S.; Guayazán-Palacios, N.; Steinbrenner, A.D. Molecular tug-of-war: Plant immune recognition of herbivory. *Plant Cell* **2022**, *34*, 1497–1513. [CrossRef]
100. Wang, P.; Granados, R.R. An intestinal mucin is the target substrate for a baculovirus enhancin. *Proc. Natl. Acad. Sci. USA* **1997**, *94*, 6977–6982. [CrossRef]
101. Zeng, J.; Ye, W.; Hu, W.; Jin, X.; Kuai, P.; Xiao, W.; Jian, Y.; Turlings, T.C.J.; Lou, Y. The N-terminal subunit of vitellogenin in planthopper eggs and saliva acts as a reliable elicitor that induces defenses in rice. *New Phytol.* **2023**, *238*, 1230–1244. [CrossRef]
102. Jones, J.D.; Dangl, J.L. The plant immune system. *Nature* **2006**, *444*, 323–329. [CrossRef]
103. Dangl, J.L.; Horvath, D.M.; Staskawicz, B.J. Pivoting the plant immune system from dissection to deployment. *Science* **2013**, *341*, 746–751. [CrossRef]
104. Ji, R.; Ye, W.; Chen, H.; Zeng, J.; Li, H.; Yu, H.; Li, J.; Lou, Y. A salivary endo-β-1,4-glucanase acts as an effector that enables the brown planthopper to feed on rice. *Plant Physiol.* **2017**, *173*, 1920–1932. [CrossRef]
105. Zhou, J.M.; Zhang, Y. Plant Immunity: Danger perception and signaling. *Cell* **2020**, *181*, 978–989. [CrossRef]
106. Guo, J.; Wang, H.; Guan, W.; Guo, Q.; Wang, J.; Yang, J.; Peng, Y.; Shan, J.; Gao, M.; Shi, S.; et al. A tripartite rheostat controls self-regulated host plant resistance to insects. *Nature* **2023**, *618*, 799–807. [CrossRef]
107. Jeevanandham, N.; Raman, R.; Ramaiah, D.; Senthilvel, V.; Mookaiah, S.; Jegadeesan, R. Rice: *Nilaparvata lugens* Stal interaction—Current status and future prospects of brown planthopper management. *J. Plant Dis. Prot.* **2023**, *130*, 125–141. [CrossRef]
108. Claridge, M.F.; Hollander, D. The "biotypes" of the rice brown planthopper, *Nilaparvata lugens*. *Entomol. Exp. Appl.* **1980**, *27*, 23–30. [CrossRef]
109. Saxena, R.C.; Barrion, A.A. Biotypes of the brown planthopper *Nilaparvata lugens* (Stål) and strategies in deployment of host plant resistance. *Int. J. Trop. Insect Sci.* **1985**, *6*, 271–289. [CrossRef]

110. Chen, X. Small RNAs and their roles in plant development. *Annu. Rev. Cell Dev. Biol.* **2009**, *25*, 21–44. [CrossRef] [PubMed]
111. Chen, X.; Rechavi, O. Plant and animal small RNA communications between cells and organisms. *Nat. Rev. Mol. Cell Biol.* **2022**, *23*, 185–203. [CrossRef] [PubMed]
112. Niu, D.; Hamby, R.; Sanchez, J.N.; Cai, Q.; Yan, Q.; Jin, H. RNAs—A new frontier in crop protection. *Curr. Opin. Biotechnol.* **2021**, *70*, 204–212. [CrossRef] [PubMed]
113. Wang, Y.; Li, M.; Ying, J.; Shen, J.; Dou, D.; Yin, M.; Whisson, S.C.; Birch, P.R.J.; Yan, S.; Wang, X. High-efficiency green management of potato late blight by a self-assembled multicomponent nano-bioprotectant. *Nat. Commun.* **2023**, *14*, 5622. [CrossRef] [PubMed]
114. Lv, H.; Li, X.; Li, J.; Yu, C.; Zeng, Q.; Ning, G.; Wan, H.; Li, J.; Ma, K.; He, S. Overcoming resistance in insect pest with a nanoparticle-mediated dsRNA and insecticide co-delivery system. *Chem. Eng. J.* **2023**, *475*, 146239. [CrossRef]
115. Qiao, L.; Lan, C.; Capriotti, L.; Ah-Fong, A.; Nino Sanchez, J.; Hamby, R.; Heller, J.; Zhao, H.; Glass, N.L.; Judelson, H.S.; et al. Spray-induced gene silencing for disease control is dependent on the efficiency of pathogen RNA uptake. *Plant Biotechnol. J.* **2021**, *19*, 1756–1768. [CrossRef] [PubMed]
116. Zhao, J.; Guo, H. Trans-kingdom RNA interactions drive the evolutionary arms race between hosts and pathogens. *Curr. Opin. Genet. Dev.* **2019**, *58–59*, 62–69. [CrossRef]
117. Yan, S.; Yin, M.; Shen, J. Nanoparticle-based nontransformative RNA insecticides for sustainable pest control: Mechanisms, current status and challenges. *Entomolol. Gen.* **2023**, *43*, 21–30. [CrossRef]
118. Wang, M.; Weiberg, A.; Lin, F.; Thomma, B.P.; Huang, H.; Jin, H. Bidirectional cross-kingdom RNAi and fungal uptake of external RNAs confer plant protection. *Nat. Plants* **2016**, *2*, 16151. [CrossRef]
119. Zhao, J.; Guo, H. RNA silencing: From discovery and elucidation to application and perspectives. *J. Integr. Plant Biol.* **2022**, *64*, 476–498. [CrossRef]
120. Cai, Q.; He, B.; Kogel, K.H.; Jin, H. Cross-kingdom RNA trafficking and environmental RNAi-nature's blueprint for modern crop protection strategies. *Curr. Opin. Microbiol.* **2018**, *46*, 58–64. [CrossRef]
121. Gao, J.; Huang, G.; Chen, X.; Zhu, Y. Protein S-acyl transferase 13/16 modulate disease resistance by S-acylation of the nucleotide binding, leucine-rich repeat protein R5L1 in *Arabidopsis*. *J. Integr. Plant Biol.* **2022**, *64*, 1789–1802. [CrossRef]
122. Wang, Z.; Li, Y.; Zhang, B.; Gao, X.; Shi, M.; Zhang, S.; Zhong, S.; Zheng, Y.; Liu, X. Functionalized carbon dot-delivered RNA nano fungicides as superior tools to control *phytophthora* pathogens through plant RdRP1 mediated spray-induced gene silencing. *Adv. Funct. Mater.* **2023**, *33*, 2213143. [CrossRef]
123. Yu, C.; Li, J.; Zhang, Z.; Zong, M.; Qin, C.; Mo, Z.; Sun, D.; Yang, D.; Zeng, Q.; Wang, J.; et al. Metal-organic framework-based insecticide and dsRNA codelivery system for insecticide resistance management. *ACS. Appl. Mater. Interfaces* **2023**, *15*, 48495–48505. [CrossRef] [PubMed]
124. He, B.; Chu, Y.; Yin, M.; Müllen, K.; An, C.; Shen, J. Fluorescent nanoparticle delivered dsRNA toward genetic control of insect pests. *Adv. Mater.* **2013**, *25*, 4580–4584. [CrossRef] [PubMed]
125. Ma, Z.; Zhou, H.; Wei, Y.; Yan, S.; Shen, J. A novel plasmid–Escherichia coli system produces large batch dsRNAs for insect gene silencing. *Pest Manag. Sci.* **2020**, *76*, 2505–2512. [CrossRef]
126. Sharma, A.; Shrestha, G.; Reddy, G.V.P. Trap crops: How far we are from using them in cereal crops? *Ann. Entomol. Soc. Am.* **2019**, *112*, 330–339. [CrossRef]
127. Vlahova, V. Trap cropping: A useful approach in farming systems. *Sci. Pap-Ser. A-Agron.* **2021**, *64*, 342–349.
128. Sarkar, S.C.; Wang, E.; Wu, S.; Lei, Z. Application of trap cropping as companion plants for the management of agricultural pests: A review. *Insects* **2018**, *9*, 128. [CrossRef]
129. Fahad, S.; Nie, L.; Hussain, S.; Khan, F.; Khan, F.A.; Saud, S.; Muhammad, H.; Li, L.; Liu, X.; Tabaassum, A.; et al. Rice pest management and biological control. In *Sustainable Agriculture Reviews: Cereals*; Lichtfouse, E.A., Goyal, A.B., Eds.; Springer International Publishing: Cham, Switzerland, 2015; pp. 85–106.
130. Krishnaiah, N. A global perspective of rice brown planthopper management II-after green revolution era. *Mol. Entomol.* **2014**, *5*, 46–55.
131. Lou, Y.; Zhang, G.; Zhang, W.; Hu, Y.; Zhang, J. Biological control of rice insect pests in China. *Biol. Control* **2013**, *67*, 8–20. [CrossRef]