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Review

# Understanding Brown Planthopper Resistance in Rice: Genetics, Biochemical and Molecular Breeding Approaches

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**Abstract:** Brown planthopper (BPH, *Nilaparvata lugens* Stål) is the most devastating pest of rice in Asia and causes significant yield loss annually. Around 37 BPH resistance genes have been identified so far in *indica*, African rice varieties along with wild germplasms such as *Oryza officinalis*, *O. minuta*, *O. nivara*, *O. punctata*, *O. rufipogon* and *O. latifolia*. Genes/QTLs involved in BPH resistance, including *Bph1*, *bph2/BPH26*, *Bph3*, *Bph6*, *bph7*, *BPH9*, *Bph12*, *Bph14*, *Bph15*, *Bph17*, *BPH18*, *bph19*, *Bph20*, *Bph21*(t), *Bph27*, *Bph27*(t), *Bph28*(t), *BPH29*, *QBph3*, *QBph4*, *QBph4.2*, *Bph30*, *Bph32*, *Bph33*, *Bph35* and *Bph36*, have been fine-mapped by different researchers across the globe. The majority of genes/QTLs are located on rice chromosomes 1, 3, 4, 6, 11 and 12. Rice plants respond to BPH attack by releasing various endogenous metabolites like proteinase inhibitors, callose, secondary metabolites (terpenes, alkaloids, flavonoid, etc.) and volatile compounds. Besides that, hormonal signal pathways mediating (antagonistic/synergistic) resistance responses in rice have been well studied. Marker-assisted breeding and genome editing techniques can also be adopted for improving resistance to novel BPH biotypes.

**Key words:** rice; brown planthopper; resistance; wild germplasm; marker-assisted breeding; genome editing; secondary metabolite

Insect pests have always emerged as a major constraint to agriculture, resulting in significant loss of yield as well as deterioration in grain quality. Rice, one of the most important cereal crops in Asia-pacific region is a host to wide range of insects that feed on it. Among these insect pests, brown planthopper (BPH, *Nilaparvata lugens* Stål) is the most devastating pest of rice, accounting for about 20% to 80% of yield loss and an overall economic loss to around $300 million in Asia annually (Min et al, 2014).

BPH causes serious damage to rice crops by sucking the sap from the xylem and phloem tissues, which ultimately leads to ‘hopper burn’. BPH also causes indirect damage by transmitting viral diseases such as grassy stunt virus and ragged stunt virus (Cabautan et al, 2009). Currently, application of chemical pesticides such as imidacloprid is the main method of controlling BPH population, which is expensive as well as hazardous to health and environment. It kills natural predators and ultimately develops insecticide resistant BPH biotypes (Tanaka et al, 2000). Hence, host-plant resistant is the most economic, effective and eco-friendly approach to manage insects and increase yields (Jena et al, 2006).

Over the period of times, different BPH biotypes varing in virulence pattern to different rice genotypes

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have evolved (Sogawa, 1978). Four recognized biotypes have been categorized in BPH population (Khush et al, 1985; Brar et al, 2009). Biotypes 1 and 2 are mostly prevalent in East and South-east Asia whereas biotype 3 is originated in laboratory by rearing insect on a resistant variety (Panda and Heinrichs, 1983). The most devastating biotype in South Asia, especially in Indian sub-continent, is biotype 4. In the course of time, new virulent biotypes may evolve, which can overcome the existing resistant gene (Jing et al, 2014). In recent decades, it has been documented that BPH is showing variation in its ability to adopt resistance in host plants. The first ever virulence variation in BPH was reported in 1970s, when *indica* rice cultivar Mudgo having *Bph1* resistance gene was introduced commercially to manage the pest population. The selection pressure caused by this gene leads to emergence of a new virulent planthopper which tolerates the effect of *Bph1* (Myint et al, 2009). In subsequent years, new virulent BPH biotypes evolved, which are dominating the resistance genes. Till date, the evolution of BPH biotype is not well understood, but there are many reports of plant diseases or pest combinations, which indicate that virulence is mainly attributed to the loss of specific effector proteins that are recognized by host plants to induce resistance responses.

In nature, to protect from insect damage, plants establish resistance mechanisms in three different ways including antibiosis, antixenosis and tolerance (Alam and Cohen, 1998). Antibiosis is one major mechanism in conferring resistance to BPH, which mostly affects the insect behavior like survival, feeding or reproduction following infestation. The plant tissue triggers its immune response, which includes activation of inhibitory genes, secretion of toxic substances and formation of external barriers like thick cuticle or callose plugging. Antixenosis mechanism avoids insect pest damage through repelling or disturbing the insects, thereby reducing pest colonization and oviposition. In addition to these two existent mechanisms, the tolerance mechanism is a peculiar type, in which plants can produce good quality crop with little or no decrease in fitness despite being attacked (Strauss and Agarwal, 1999). The genes confer their resistance through one or combination of these three defense mechanisms. Therefore, it is always beneficial to analyze the type of resistance mechanism operating in resistant varieties, which can be introduced into susceptible rice genotypes through various breeding methods.

Over the past few decades, considerable efforts have been made in identification of BPH resistance genes due to advancement in molecular genetics and genomics by use of different types of molecular markers such as SSR, InDel and SNP. To date, 37 resistance genes have been identified in rice varieties (Du et al, 2020; Haliru et al, 2020), out of which 9 potential genes i.e. *Bph3*/*Bph17*, *Bph6*, *BPH9*, *Bph14*, *Bph15*, *BPH18*, *BPH26*, *BPH29* and *Bph32* have been cloned and characterized. Identification of these genes has intensified marker-assisted breeding as well as pyramiding of these genes into elite susceptible cultivars for achieving durable resistance against BPH.

## Genetic evaluation of BPH resistance

In rice improvement programme, gaining insights into the genetics and identification of suitable genes in the plant population is of utmost important. For this, the available rice germplasm resources have to be screened and evaluated for BPH resistance/susceptibility. Several screening techniques have been followed to assess the degree of resistance of host plant with respect to infestation such as Standard Seed Box test (Fujita et al, 2013), modified Standard Seed Box test (Panda and Khush, 1995) and occasionally by examining the inherent mechanism of host that inhibits the insect attack. In the Standard Seed Box test method, seedlings at the 2/3 leaf stage are infested with 2nd or 3rd instar BPH nymph followed by scoring of each seedling as per the standard evaluation system. The modified Seed Box test performs the screening by using the seed box in a screen house where it utilizes nymphs of BPH with independent selection of plant substances at the young (seedling) stage or sometimes across various developmental stages of plants. Furthermore, this method also assesses the damage to the seedlings by the progeny of an initial infestation with a set of nymphs. This method is being recognized as standard method because of suitability of time and space management for evaluating germplasms and breeding materials. However, this test is influenced by various environmental and developmental factors such as temperature, humidity, nymph stage, biotype and natural enemy. Another approach which indirectly evaluates plant's innate response is by examining the physiological and biochemical reactions of BPH (feeding rate, fecundity and survival) on different rice varieties. Parameters measured include honeydew excretion, host choice, colonization and feeding behavior (Pathak et al, 1982; Klingler et al, 2005; Sangha et al, 2008). During the process of screening and evaluation, maximum caution needs to be taken care of the purity of BPH population (Hu et al, 2016).

## Sources of BPH resistance genes

Pioneer work on the BPH resistance was initiated with the search for various potential donors and transfer of the resistance genes from these sources to elite susceptible varieties. The first BPH resistance gene was discovered in 1967 (Pathak et al, 1969). Following this preliminary identification, Athwal et al (1971) identified two genes, namely *Bph1* and *bph2*, conferring BPH resistance from Mudgo and ASD7, respectively (Table 1).

The resistant *indica* cultivar, IR26, harbouring *Bph1* gene became susceptible due to the development of a new race (biotype) (Khush, 1971). Eventually, a new recessive gene *bph2* was identified and subsequently introgressed into IR26 (Khush, 1992). The *bph2* resistance gene being durable is highly used in the breeding line, and the cultivar IR36 possessing this gene is widely grown, which exerts tremendous pressure on the biotype 2 (Jena and Kim, 2010). As a consequence, a new biotype BPH biotype (biotype 3) evolved. This new biotype evolution is in accordance with the 'Boom and Bust cycle' theory. Subsequently, two resistance loci, *Bph3* and *bph4*, were identified in two Srilankan varieties, i.e. Rathu Heenati and Babawee, respectively, and were introgressed into many elite rice cultivars (Lakashminarayana and Khush, 1977). This significant finding leads to the development of a series of new BPH resistant rice varieties including IR56, IR60, IR68, IR70 and IR72 (Jena and Kim, 2010).

A study was conducted to establish the possible allelic relationship among the four major genes *Bph1*, *bph2*, *Bph3* and *bph4* discovered at that time (Ikeda and Kaneda, 1981). *Bph1* with *bph2* genes segregate independently of both *Bph3* and *bph4*, while *Bph1* and

**Table 1. List of brown planthopper (BPH) resistance genes in rice.**

| Chr | Gene | Flanking marker | Location (Mb) | Germplasm source | Resistant to biotype | Reference |
|---|---|---|---|---|---|---|
| 1 | *Bph33*(t) | RM488, RM11522 | 24.80–28.00 | RP2068 | ND | Naik et al, 2018 |
| 1L | *BPH38*(t) | SNPs 693, 369, 10, 112, 165 | 20.71–21.23 | Khazar | Biotype 3 | Balachiranjeevi et al, 2019 |
| 1 | *BPH37* | RM302, YM35 | ND | IR64 | ND | Yang et al, 2019 |
| 3 | *bph11* | ND | 35.60–35.80 | *O. officinalis* | ND | Hirabayashi et al, 1998 |
| 3S | *Bph13*(t) | AJ09b230, AJ09c | 5.18–5.70 | IR54745-2-21 (*O. officinalis*) | Biotypes 1, 2, 3 and 4 | Renganayaki et al, 2002 |
| 3S | *bph19* | RM6308, RM3134 | 7.18–7.24 | AS20-1 | Biotype 2 | Chen et al, 2006 |
| 3 | *qBph3* | RM3180, RM2453 | 18.27–20.25 | Rathu Heenati | ND | Kumari et al, 2010 |
| 3L | *BPH31* | PA26, RM2334 | 26.26–26.74 | CR2711-76 (*indica*) | Biotype 4 | Prahalada et al, 2017 |
| 4S | *Bph12* | RM16459, RM1305 | 5.21–5.66 | B14 (*O. latifolia*) | ND | Qiu et al, 2012 |
| 4S | *Bph15* | RM261, S16 | 6.68–6.90 | B5 (*O. officinalis*) | ND | L üet al, 2014 |
| 4S | *QBph4.1* | P17, xc4-27 | 6.70–6.90 | IR02W101 (*O. officinalis*) | ND | Hu et al, 2015b |
| 4S | *QBph4.2* | RM261, S1 | 6.58–6.89 | IR65482-17-511 (*O. australiensis*) | ND | Hu et al, 2015a |
| 4S | *Bph17* | RM8213, RM5953 | 6.93–6.97 | Rathu Heenati | ND | Sun et al, 2005 |
| 4S | *Bph20*(t) | MS10, RM5953 | 8.20–9.60 | IR71033-121-15 (*O. minuta*) | ND | Rahman et al, 2009 |
| 4S | *Bph30* | RM16294, RM16299 | 0.90–0.94 | AC-1613 (*O. indica*) | Biotypes 1, 2 and 3 | Wang et al, 2018 |
| 4S | *Bph33* | H99, H101 | 0.91–0.97 | Kolayal and Poliyal | ND | Hu et al, 2018 |
| 4S | *Bph36* | S13, X48 | ND | *O. rufipogon* Griff | Biotypes 1 and 2 | Li et al, 2019 |
| 4S | *Bph12* | RM16459, RM1305 | 5.21–5.66 | B14 (*O. latifolia*) | Biotype 2 | Qiu et al, 2012 |
| 4L | *Bph6* | Y19, RM119 | 21.36–21.39 | Swarnalata | Biotype 4 | Qiu et al, 2010 |
| 4L | *Bph34* | RM16994, RM17007 | 21.15–21.30 | IRGC104646 (*O. nivara*) | Biotype 4 | Kumar et al, 2018 |
| 4L | *Bph27* | RM273, RM471 | 19.12–19.20 | GX2183 (*O. rufipogon*) | Biotype 2 | Huang et al, 2013 |
| 4L | *Bph27*(t) | RM471, RM5742 | 20.79–21.33 | Balamawee | ND | He et al, 2013 |
| 4L | *Bph35* | PSM16, RM413 | 6.28–6.93 | RBPH660 | ND | Zhang et al, 2020 |
| 6S | *Bph3* | RM469, RM588 | 1.21–1.40 | Rathu Heenati | Biotypes 1, 2, 3 and 4 | Jairin et al, 2007 |
| 6S | *bph4* | RM190, C76A | 1.20–1.76 | Babawee | Biotypes 1, 2, 3 and 4 | Kawaguchi et al, 2001 |
| 6S | *BPH25* | S00310 | 0.20–1.71 | ADR52 | ND | Myint et al, 2012 |
| 6S | *BPH29* | BYL7, BID2 | 0.48–0.49 | RBPH54 (*O. rufipogon*) | Biotypes 1 and 2 | Wang et al, 2015 |
| 6S | *Bph32* | RM19291, RM8072 | 1.21–1.40 | Ptb33 | ND | Ren et al, 2016 |
| 11L | *Bph28*(t) | Indel55, Indel66 | ND | DV85 | ND | Wu et al, 2014 |
| 12 | *BPH10* | RM260, RM313 | 19.00–23.00 | *O. australiensis* | ND | Ishii et al, 1994 |
| 12 | *Bph1* | BpE18-3 | 13.10–13.28 | Mudgo, TKM6 | Biotypes 1 and 3 | Kim and Sohn, 2005 |
| 12L | *bph2/Bph26* | KAM4 | 22.13–23.18 | IR1154-243 | Biotypes 1 and 2 | Murai et al, 2001 |
| 12L | *bph2* | RM463, RM7102 | 13.21–22.13 | ASD7 | ND | Sun et al, 2007 |
| 12L | *bph7* | RM3448, RM313 | 19.95–20.87 | T12 | Biotype 4 | Qiu et al, 2014 |
| 12L | *BPH9* | RM463, RM5341 | 19.11–22.13 | Kaharamana | Biotypes 1, 2 and 3 | Su et al, 2006 |
| 12L | *BPH9* | OPR04, S2545 | 19.00–22.50 | Pokkali | ND | Murata et al, 2001 |
| 12L | *BPH18*(t) | RM463, S15552, 7312.T4A | 22.25–23.48 | IR65482-7-216 (*O. australiensis*) | Biotypes 1, 2, 3 and 4 | Jena et al, 2006 |
| 12L | *Bph21*(t) | RM3726, RM5479 | 23.28–24.41 | IR71033-121-15 (*O. minuta*) | Biotype 1 | Rahman et al, 2009 |

Chr, Chromosome; S, Short arm; L, Long arm; ND, No data.

*bph2* as well as *Bph3* and *bph4* are closely linked. Based on trisomic analysis, the genetic loci of *Bph3* and *bph4* are positioned on chromosome 6S (Ikeda and Kaneda, 1981). Khush et al (1985) identified a single recessive gene *bph5* conveying resistance to BPH biotype IV in rice cultivar ARC10550, which segregated independently of *Bph1*, *bph2*, *Bph3* and *bph4*. Later, Deen et al (2017) demonstrated that the resistance conferred in this cultivar is governed by five major loci located on chromosomes 1, 3, 6, 8 and 12 and not by *bph5*.

Through genetic analysis of 17 *Oryza* cultivars, Kabis and Khush (1988) reported a single dominant resistance gene and a recessive gene that segregate independently of *bph5*. They designated these new identified genes as *Bph6* and *bph7*. Similarly, genetic studies along with allelism test using some selected resistant varieties identified a new recessive resistant gene *bph8* (Nemoto et al, 1989). Another dominant gene identified from Kaharamana (Su et al, 2006) and Pokkali (Murata et al, 2001) was designated as *BPH9*. In addition to this extensive search for resistance gene in cultivated varieties, new potential genes were searched in wild germplasms. *BPH10* is the first gene to be reported harbouring in a wild relative (*O. australiensis*) of cultivated rice (Ishii et al, 1994). Subsequently, *bph11* and *Bph12* were reported in the genetic background of wild rice (*O. officinalis*) (Table 1).

## Mapping of BPH resistance genes

From the beginning of 21st century, much progress has been made on mapping of BPH resistance loci in resistant varieties. Till date, 38 BPH resistance genes/ QTLs have been identified in many rice varieties including African rice varieties and wild germplasm (Du et al, 2020; Haliru et al, 2020). The majority of these resistance genes localize on 5 out of the 12 rice chromosomes (chromosomes 1, 3, 4, 6 and 12). Four clusters (namely, A, B, C and D) have been reported on three chromosomes (Balachiranjeevi et al, 2019). Cluster A is located on long arm of chromosome 12 and contains 8 loci. Clusters B and D are present on the short and long arms of chromosome 4, containing 10 and 5 loci, respectively. Cluster C is identified on the short arm of chromosome 6, which includes 5 gene loci. Apart from these clusters, the rest of the reported genes are distributed on chromosomes 1, 3 and 11. Till date, 26 genes/QTLs have been fine-mapped (Table 1).

Recently, a dominant gene *Bph30* having strong antibiotic response has been fine mapped in a 0.90–0.94 Mb region flanked by SSR-28 and SSR-69 on chromosome 4 (Wang et al, 2018). In addition, two markers RM16294 and RM16299 tightly linked to *Bph30* have been successfully applied for introgression of the gene into elite lines. Another dominant gene *BPH31* having a stable and broad spectrum resistantce has been identified in an *indica* cultivar (CR2711-76). This gene is fine mapped on the long arm of chromosome 3 (Prahalada et al, 2017). It exhibits very effective and stable resistance against the most prevalent biotype 4 in Indian subcontinent by utilizing all the three kinds of defence mechanism explained earlier. This thus facilitates the improvement of popular local cultivars against pest attack. By transferring *BPH31*, an improved Jaya line has been developed that shows strong resistance to BPH biotypes of India and the Philippines.

Genetic analysis of two Srilankan BPH resistant rice cultivars (Kolaya and Poliyal) leads to detection of a resistant gene *Bph33* (Hu et al, 2018), which is fine mapped to a 60 kb region between two InDel markers (H99 and H101) on the short arm of chromosome 4. The gene exhibits durable resistance during the plant growth period from the seedling to tillering stages, similar to *Bph6* and *BPH9* (Zhao et al, 2016; Guo et al, 2018). Naik et al (2018) reported similar genomic segment as that of *Bph33* on chromosome 1 and designated it as *Bph33*(t). The new locus is identified using advanced generation recombinant inbred lines derived from RP2068 and is the first gene to be located on chromosome 1, which is defined by two flanking markers RM488 and RM11522.

In plant breeding activities, ancestral character species or related species always remain a potential source for various desirable genes. Kumar et al (2018) identified a BPH resistance locus named *Bph34* on the long arm of chromosome 4 by high resolution mapping using $F_2$ and $F_{2:3}$ populations derived from *O. sativa/O. nivara* cross.

Another resistance locus, *Bph35*, has been identified from RBPH660, an introgression line derived from *O. rufipogon* (Zhang et al, 2020). This locus, accounting for 51.17% of phenotypic variation, is mapped to the candidate region of chromosome 4 between InDel markers PSM16 and RM413, where *QBph4* and *QBph4.2* are located. However, these two QTLs have not been cloned so far, and their allelic nature with *Bph35* is not confirmed. *Os04g0193950*, encoding a putative NB-ARC (nucleotide-binding adaptor shared by APAF-1, R proteins and CED-4) and LRR (leucine-rich repeat) domain protein with nine non-synonymous SNP substitutions in its coding sequence regions, might be

the candidate gene for *Bph35*. Thus, a total of five genes form a cluster on chromosome 4L [*Bph6*, *Bph27*, *Bph27*(t), *Bph34* and *Bph35*].

*O. rufipogon* is also known to be a valuable germplasm for BPH resistance. *Bph36*, a major locus, was reported from two introgression line (RBPH16 and RBPH17) developed from wild rice GX2183 (*O. rufipogon*) (Li et al, 2019). This locus was mapped on the short arm of chromosome 4 within an interval of 38 kb flanked by two InDel markers S13 and X48.

Earlier research on IR64 has already confirmed the presence of one major BPH resistance gene *Bph1*, and several minor QTLs conferring resistance (Alam and Cohen, 1998; Soundarajan et al, 2004). However, this resistance is lost due to the development of new biotypes. So, further research carried on this variety leads to the identification of gene *Bph37* on chromosome 1 flanked by RM302 and YM35 (Yang et al, 2019). This gene is quiet efficient compared to *Bph1*. *Bph37* exhibits a unique status, as it confers the tolerance mechanism rather than antibiosis or antixenosis, which is generally reported as a defence strategy. Earlier *bph7* is considered to confer tolerance to pest, which is mapped by a seedling bulk test (Qiu et al, 2014).

A molecular marker based genetic analysis of $BC_1F_5$ population derived from a cross between a BPH resistant *indica* variety Khazar and a popular susceptible line Huanghuazhan results in identification of *BPH38*(t) between 20.71 to 21.23 Mb on the long arm of chromosome 1 (Balachiranjeevi et al, 2019).

## Cloning and characterization of BPH resistance loci

Advances in next generation sequencing platform and bioinformatics methods have emerged as a major breakthrough for cloning and understanding the molecular mechanism associated with BPH resistance genes. Map-based cloning and gene isolation will enable the scientific community to apply appropriate strategies in varietal development programmes.

*Bph14* is the first gene to be cloned using map-based cloning from *O. officinalis* (Du et al, 2009). This gene encodes a coiled-coil nucleotide binding and leucin-rich repeat (CC-NB-LRR) protein that is a typical member of nucleotide-binding domain, leucine rich containing (NLR) protein family. It provides resistance at the seedling and maturity stages. Sequence comparisons indicate that the gene carries a unique LRR domain, which activates the salicylic acid (SA) signalling pathway and induces callose deposition on phloem tissue as well as trypsin inhibitor production, which in turn reduces BPH feeding on host plant (Table 2). Myint et al (2012) found two resistance genes *BPH25* and *BPH26* from an *indica* rice variety ADR52 on chromosomes 6S and 12L, respectively. The map-based cloning of *BPH26* indicated a CC-NB-LRR protein similar to that of *Bph14*, which inhibits sucking in phloem sieve element (Tamura et al, 2014). Sequence analysis confirms that *BPH26* is the same as *bph2*, which is rendered ineffective due to arising of virulent biotype in Asia. However, the resistance effect of *BPH26* is substantiated when it was used in combination with *BPH25*. *BPH18* is localized to the same locus of *BPH26* on the long arm of chromosome 12 from a resistant introgression line derived from wild rice *O. australiensis* (Ji et al, 2016). Map-based cloning and complementation test revealed that *BPH18* encodes a CC-NBS-NBS-LRR protein with two nucleotide-binding site domains. These proteins are present on membrane of endoplasmic reticulum, Golgi apparatus and pre-vacuolar compartments, suggesting that it may help recognize BPH invasion in the endo-membrane system of phloem cells. Whole genome sequencing of *BPH18* and *BPH26* reveals

**Table 2. Characterization of brown planthopper (BPH) resistant genes in rice.**

| Gene | Chr | Candidate locus | Encoded protein | Germplasm source | Defense signalling pathway | Reference |
|---|---|---|---|---|---|---|
| *Bph3* | 4 | *OsLecRK1-OsLecRK3* | Lectin receptor kinases | Rathu Heenati | – | Liu et al, 2015 |
| *Bph6* | 4 | *Gene1* (NCBI accession KX818197) | Atypical LRR | Swarnalata | JA↑, SA↑, CK↑ (Synergistic) | Guo et al, 2018 |
| *BPH9* | 12 | $R_2$ | CC-NBS-NBS-LRR | Pokkali | JA↑, SA↓ (Antagonistic) | Zhao et al, 2016 |
| *Bph14* | 3 | *Ra* (*Os03g0848700*) | CC-NB-LRR | B5 | SA↑ | Du et al, 2009 |
| *Bph15* | 4 | *LOC_Os04g12390*, *LOC_Os04g12460* | LRR and JRL | B5 | DEGs↑ | Lü et al, 2014 |
| *BPH18* | 12 | *LOC_Os12g37280*, *LOC_Os12g37290* | CC-NBS-NBS-LRR | IR65482-7-216-1-2 | – | Ji et al, 2016 |
| *BPH26* | 12 | *Os12g0559300*, *Os12g0559400*, *Os12g0559600* | CC-NB-LRR | ADR52 | JA↑, SA↑, ET↑, PR1b↑ | Tamura et al, 2014 |
| *BPH29* | 6 | *G5* (*Os06g0107800*) | B3 DNA-binding domain | RBPH54 | JA↓, SA↑ (Antagonistic) | Wang et al, 2015 |
| *Bph32* | 6 | *Os06g123200* | Unknown SCR domain | PTB33 | – | Ren et al, 2016 |

CC-NB-LRR, Coiled-coil nucleotide-binding leucine-rich repeat; CC-NBS-NBS-LRR, Coiled-coil nucleotide-binding site-leucine-rich repeat; CK, Cytokinin; DEGs, Differentially expressed genes; ET, Ethylene; JA, Jasmonic acid; JRL, Jacalin-related lectin; LRR, Leucine-rich repeat; SA, Salicylic acid; SCR, Short consensus repeat; ↑, Up-regulated; ↓, Down-regulated.

remarkable sequence difference and also differential expression to defence mechanism against the pest, although they occupy the same locus.

Zhao et al (2016) cloned *BPH9* on the long arm of chromosome 12. This gene encodes a rare type of nucleotide binding and LRR containing protein that also localizes to the endomembrane system. *BPH9* activates the SA and jasmonic acid (JA) pathways, thereby conferring both antixenosis and antibiosis modes of resistance. Further study in this aspect was extended to six genes (*Bph1*, *bph7*, *Bph10*, *BPH18*, *Bph21* and *BPH26/bph2*) reported earlier in the same locus of *BPH9*. Out of these six genes, *BPH26*/*bph2* and *BPH18* are already cloned. After cloning the rest four genes followed by comparing the sequence information with *BPH9*, it was concluded that all the seven genes in this cluster are the multiple alleles of the same locus. These alleles can be classified into four allelotypes conferring different degrees of resistance to BPH.

BPH resistance genes that encode the membrane localized lectin receptors like kinases (LecRKs) have been cloned. *Bph15* is initially mapped and physically delineated between the flanking markers C820 and S11182 on chromosome 4S (Yang et al, 2004), and it was subsequently fine mapped to a 47 kb region between markers RG1 and RG2 (Lü et al, 2014), where the lectin receptor kinase gene, *OsLecRK*, is cloned (Cheng et al, 2013). *Bph3*, which is initially identified from a Srilankan variety Rathu Henati, is fine mapped on chromosome 6S between markers RM469 and RM588 (Jairin et al, 2007). However, Liu et al (2015) cloned *BPH3* on chromosome 4. Map-based cloning and functional characterization showed that *Bph3* is a cluster of three genes encoding the plasma membrane-localized lectin receptor kinases (*OsLecRK1*, *OsLecRK2* and *OsLecRK3*). Transgenic test revealed that genes independently confer resistance to BPH and their effect is enhanced when these three genes were pyramided, they collectively provide stable and broad-spectrum resistance. Although most of genes conferring resistance to BPH are dominant in nature, some recessive genes have also been identified with sustainable effect. *BPH29*, a recessive gene, is identified from an introgression line RBPH54 (derived from *O. rufipogon*) and fine mapped onto the short arm of chromosome 6 (Wang et al, 2015). Expression pattern analysis revealed that the tissue specific expression of *BPH29* is confined to vascular tissue on BPH attack. In response to BPH attack, this gene activates SA-signalling pathway and suppress the JA/ethylene (ET) dependent pathway. This triggers callose deposition in phloem cells following antibiosis response to BPH. Ren et al (2016) identified a BPH resistance gene *Bph32* between the markers RM19291 and RM8072 on the short arm of chromosome 6 from a variety Ptb33. This gene shares 100% sequence match with its allele in *O. latifolia*. Expression analysis revealed that *Bph32* is highly expressed in leaf sheath, which is the primary feeding sites of BPH. Overexpression of *Bph32* inhibits the feeding habit of pest after infestation.

Sometimes, BPH along with whitebacked planthopper (WBPH) simultaneously pose serious threat to rice plant, and most of genes, till now discussed, are primarily related to only BPH infestation. Guo et al (2018) map-based cloned and functionally analyzed a gene *Bph6*, which shows broad spectrum resistance to both BPH and WBPH biotypes without yield loss. *Bph6* encodes an uncharacterized LRR protein that interacts with exocyst subunit OsEXO70E1, and activates a coordinated mechanism of cytokinin (CK), SA and JA pathway to display a high-level of field resistance that is heavily infested with BPH (Table 2).

## Positional ambiguity among BPH resistance genes and gene clusters

Most of BPH resistance genes are clustered on the same or closely linked regions but they are differentiated from one another by flanking markers or by relative genetic distance. Some variation is also observed between the genes originating from the same source. For example, *Bph11* and *Bph14* are identified from the same source, but they are present on nearby location on the long arm of chromosome 3. In contrast, there are genes having two different origin sources detected on the same region. *Bph17* and *Bph20* are located on the short arm of chromosome 4 and also overlap with each other, despite being originated from Rathu Hennati and *O. minuta*, respectively (Sun et al, 2005; Rahman et al, 2009).

The regions of chromosomes 4S and 12L are considered as hotspots of BPH resistance genes, as these regions account for greater than 50% of the genes identified till date (Table 1). *Bph35* is located in the region from 6.28 to 6.93 Mb on chromosome 4. Interestingly, within this region, *QBph4* is detected in 6.70 to 6.90 Mb region between markers P17 and xc4-27 (Hu et al, 2015a), while *QBph4.2* at a 6.58–6.89 Mb region flanked by markers RM261 and S1 (Hu et al, 2015b), and *Bph15* at a region from 6.69 to 6.90 Mb in B5 (Lü et al, 2014). These above four loci are clustered in a region where one gene occupies

a position within another one because there might be overlapping within them as well as the conserved regions. This positional ambiguity can be resolved only when two QTLs *QBph4* and *QBph4.2* are cloned. Then, it would be clear whether *Bph35* (from *O. rufipogon*) is the same as that with *QBph4* (from *O. officinalis*) or *QBph4.2* (from *O. australiensis*).

Another locational and functional ambiguity surrounds three genes *Bph3*, *Bph15* and *Bph17*. *Bph3* is initially mapped at chromosome 6S (Jairin et al, 2007), which is latter cloned on chromosome 4 (Liu et al, 2015). Sun et al (2005) also reported that *Bph3* is on chromosome 4 as *Bph17*. Now, it has been acknowledged by some of the rice scientific community that the cloned gene on chromosome 4 has been reported as *Bph17*. Xiao et al (2016) observed that the amino acid sequence of the cloned *Bph17* is the same as that from Bph15-NIL (near isogenic line). Thus, *Bph15* might be the same as *Bph17*.

Identification of *Bph34* on chromosome 4L shows resemblance with *Bph6* and *Bph27*(t) genes identified earlier from *indica* rice Swarnalata and Balamawee, respectively (Kumar et al, 2018). However, these two rice varieties are susceptible and different alleles with markers linked to *Bph34* gene were amplified. Also, high-resolution mapping and insect physiological behaviour studies prove that *Bph27*(t) is non-allelic to *Bph6* (He et al, 2013). *Bph36* detected on the long arm of chromosome 4 in an interval flanked by RM16766 and RM17033, which is the same as that of *Bph27* (Li et al, 2019). Therefore, *Bph34* (from *O. nivara* acc. IRGC104646), *Bph6* (from Swarnalata), *Bph27*(t) (from Balamawee) and *Bph36* (from *O. rufipogon*) could be different alleles of the same gene or could be different genes altogether.

In BPH resistant studies, there are some instances of poor clarity regarding duplicate nomenclature of genes for the same locus without sufficient evidences. For example, *Bph27* and *Bph27*(t), *Bph33* and *Bph33*(t), are duplicate genes. *Bph33* was reported by Hu et al (2018) on the short arm of chromosome 4 while Naik et al (2018) reported it to be present on chromosome 1. This may be due to different sources of germplasm used for mapping the genes which results in different loci. Hence, to avoid this confusion, there should be clear and distinct nomenclature for this type of duplicated genes in accordance with the new Committee on Gene Symbolization, Nomenclature and Linkage (CGSNL) nomenclature system for rice (McCouch, 2008).

## Biochemical basis of BPH resistance in rice

Understanding of biochemical mechanism along with genetic factors contributing resistance in rice is of paramount importance to manage the BPH population as well as facilitate resistant breeding programme. Plant immunity against insect involves both constitutive defences like physical and chemical barriers that exist before invaders attack, whereas inducible defences include defensive mechanisms that become activated upon pest attack (Yang and Zhang, 2016). Plant epidermis acts as an important physical barrier in defence against insect attacks by preventing insect oviposition, setting or feeding. Also, chemical substances like high long to short carbon-chain compound ratio and the presence of shorter chain hydrocarbons on the plant surface serve as barriers against infestation (Woodhead and Padgham, 1988). Comparative transcriptional profiling of small brown planthopper (SBPH)-resistant and susceptible rice plants during early infestation indicates the upregulation of genes involved in the very long-chain fatty acid biosynthesis in small BPH-resistant rice plants (Zhang et al, 2015). This may be because the long-chain fatty acids (20 to 36C) are required for cuticle biosynthesis in epidermal cells that act as the first natural barrier when encountered by pathogens (Shepherd and Wynne, 2006; Samuels et al, 2008).

There are also some volatile organic compounds [S-linalool, β-caryophyllene, green leaf volatile (GLV) etc.], whose expression level in response to BPH attack determines the plant pest interaction. S-linalool is one such volatile which is strongly induced by BPH attack (Cheng et al, 2007). Inducible S-linalool attracts predators and parasitoids but repels BPH. Similarly, GLV encoded by a gene *HPL3*, positively modulates resistance to BPH by modulating oxylipin pathway (Tong et al, 2012).

In past decade, a number of studies about plant insect interaction revealed that when insects feed or oviposit, they release some oral secretions (saliva) and ovipostion fluids on some plants which help in their survival and settlement (Du et al, 2020). These secretions act as insect elicitors or effectors, which may make insects vulnerable or lead to defence response against insects. When BPH feeds on rice, the salivary gland secrets salivary endo-β-1,4-glucanase (NlEG1), which degrades cellulose in plant cells, thereby helping the stylets to reach the phloem (Ji et al, 2017). BPH mucin like protein NlMLP, a salivary sheath component during feeding time, induces cell

death, defense-related gene expression and callose deposition in plants (Shangguan et al, 2018). When BPH is fed an transgenic rice carrying on MLP-dsRNA, it displays mortality, reduced body size and delayed maturation, suggesting that the *MLP* silencing strategy can be used to control BPH.

Plants have developed elaborated protection systems against herbivore attack (Ji et al, 2017). This protection mechanism is just similar to strategies of immunity against attack of pathogens, including pathogen-associated molecular patterns (PAMPs)-triggered immunity (PTI) and effector-triggered immunity (ETI) (Dodds and Rathjen, 2010). The recognition of effector proteins by resistance (R) proteins induces ETI. Receptor kinases and a set of NBS-LRR proteins are involved in recognizing PAMPs or effectors and turning on the host-resistance pathways. Similar mechanism is also observed in response to BPH attack. The herbivore-associated molecular patterns (HAMPs) or the herbivore associated elicitors (HAEs) are recognized by plant cells, which triggers signal transduction pathways that connect herbivore-specific elicitors to the expression of suitable defence genes (Santamaria et al, 2013).

## Hormonal signal transduction associated with BPH resistance in rice

Plant hormones play pivotal roles in regulation of defence signalling pathway to protect against herbivore attack (Pieterse et al, 2012). In response to insect attack, plant defence system activates various phytohormones, like SA, JA, ET and CK, which in turn trigger the innate immune response. Cloning of various BPH resistance genes along with the study of transcriptomics and proteomics using c-DNA array/micro-array will offer deep insights into mechanism of insect resistance.

Map-based cloning reveals that the resistance mechanism of *Bph14* is similar to the immune response of plants against pathogen attack (Du et al, 2009). Following the BPH feeding, transcripts of SA synthesis-related gene accumulate faster in the rice resistant plants possessing the *Bph14* gene compared to the susceptible type. In case of plant/pathogen interaction, SA also stimulates the defence response genes for promoting systemic acquired resistance (Jones and Dangl, 2006). A similar finding was obtained in SA synthesis gene expression in rice plants carrying *BPH29* and *BPH9* (Wang et al, 2015; Zhao et al, 2016). Two SA synthesis related genes *PAL* (phenyl-alanine ammonia-lyase) and *CHS* (chalcone synthetase) show significant high transcript level in BPH resistant varieties (RBPH54) possessing *BPH29* gene (Wang et al, 2015). However, the expression levels of two genes involved in JA synthesis pathway, *AOS2* (allene oxide synthase 2) and *LOX* (lipoxygenase), decrease rapidly after insect infestation in resistant lines, but no significant change is observed in BPH-susceptible lines. These results indicate that *BPH29* activates SA dependent pathway but is independent to JA pathway. Similarly, NILs carrying *BPH9* recode substantial increase in SA level in resistant lines with no change in susceptible lines. The overall level is lower in resistant lines than susceptible lines, suggesting the probable antagonistic relationship of SA/JA (Zhao et al, 2016) (Table 2).

There are some instances where the pathway mediating the BPH resistance is still elusive even after map-based cloning of resistance genes. The most common pathway like SA, JA/ET mediating resistance response of various genes shows no any variation in case of *BPH18*. However, *BPH26*, which occupies the same locus of *BPH18*, greatly induces the hormonal signalling pathway. In the susceptible lines and NIL-*BPH26*, JA synthesis-related genes *LOX* and *AOS2*, SA synthesis-related gene *EDS1*, ET receptor gene *EIN2* and a pathogen-related gene *PR1b* are activated after BPH infestation. In contrast, in NIL-*BPH18*, none of the defense-related genes is strongly activated by BPH insect, suggesting an unknown pathway may be involved in BPH resistance (Ji et al, 2016).

The classic binary defense model of SA and JA postulates that they have opposite roles in defences against sucking and chewing insects (Guo et al, 2018). However, contrary to existing theory, these two hormones show synergistic effect in rice plants carrying *Bph6*. The levels of both SA and JA increase rapidly upon BPH infestation in plants carrying *Bph6* compared to susceptible plants. The applications of exogenous SA and methyl jasmonate also enhance resistance to BPH and reduce insect survival on both resistant and susceptible plant varieties (Guo et al, 2018).

Besides JA and SA, there are some other hormones that control insect defense responses in plants, like CK, ET, brassinosteroids (BR), gibberellins (GA) and abscisic acid (ABA) (Du et al, 2020). The survival rates of BPH insects are significantly reduced on the CK-treated *Bph6*-NIL plants, suggesting that CK enhances resistance. In addition, CKs also positively regulate phytoalexin production. BPH feeding increases

momilactone (rice diterpenoid phytoalexins) levels in the 9311-*Bph6*-NIL plants compared to the 9311 plants (Guo et al, 2018). BRs negatively regulate BPH resistance by decreasing SA-associated gene expression while promoting JA-associated gene expression (Pan et al, 2018). Zhang et al (2017) reported that rice DELLA protein OsSLR1, which negatively regulates GA pathway, is also down-regulated by BPH infestation. Silencing *OsSLR1* enhances constitutive levels of defence-related compounds, phenolic acids, lignin and cellulose, as well as the resistance of rice to BPH.

ET, a stress hormone, acts as the modulator of the hormone-signalling backbone. ET signalling pathway receptor gene *EIN2* (ethylene insensitive 2) accumulates faster in higher levels in the wild type than in the transgenic plants having *Bph14* (Du et al, 2009) and *Bph29* (Wang et al, 2015). These evidences support that ET negatively regulates the BPH resistance in rice. Liu et al (2017) observed that exogenous application of ABA suppresses β-1,3-glucanase but induces callose synthase activity, and promotes callose deposition and thereby prevents BPH feeding.

## Roles of metabolites in defense response

Plant shows its innate immune response to BPH attack by releasing various metabolites like proteinase inhibitors, callose, secondary metabolites (terpenes, alkaloids, flavonoids and others) and volatile compounds. These substances may directly defend plants by killing or repelling BPH, and may activate various defensive pathways or attract natural predators. Insect feeding triggers proteinase inhibitor production, which affects digestive proteases followed by induced amino acid deficiencies in the insect midgut. Thereby, it restricts insect growth and development (Lison et al, 2006). Similarly, deposition of callose in resistant varieties can block the access to the phloem sap, thereby inducing insect starvation and death of BPH.

There are several secondary metabolites that induce substantial metabolic changes in both resistant and susceptible rice varieties (Du et al, 2020). BPH infestation promotes sterol biosynthesis in susceptible plants, but promotes wax biosynthesis, phytol metabolism, strengthening of gama-aminobutyric acid shunt and shikimate-mediated secondary metabolism in resistant plants (Liu et al, 2010; Zhang et al, 2018). *Bph6* enhances the level of phytoalexins in response to BPH attack. GLV, byproduct of hydroperoxide lyase (OsHPL3), also plays a key role in defence response against BPH. Loss of function of *OsHPL3* resulted in enhanced susceptibility to BPH indicating that *OsHPL3* positively modulates resistance to BPH (Tong et al, 2012).

## Breeding strategies for developing BPH resistant rice varieties

It is desirable to develop a rice variety possessing stable and broad-spectrum resistance mechanism to different BPH biotypes. For this, multiple genes need to be incorporated to a single elite variety to nullify the effect of new virulent biotypes. Transfer of multiple genes can be achieved by both conventional and new molecular tools. The conventional breeding methods are time consuming which can be reduced by using marker-assisted selection methods (Fig. 1).

### *Marker-assisted breeding for BPH resistance*

In this method, the available rice genotypes are screened to identify the genetic markers that are tightly linked with BPH resistance. The gene or QTL is identified followed by mapping the resistance genes or QTLs. The molecular markers like SSR and InDel have been widely used in backcross breeding methods to assess the presence of the introgressed resistance gene in the desired elite line (foreground selection). Also, markers can be used to accelerate the reconstitution of recipient parental genotype at other loci (background selection). Some introgressed lines possessing broad spectrum resistance have been produced by transferring *Bph3* into the genetic background of a Thai aromatic cultivated rice (Jairin et al, 2009), *Bph14* and *Bph15* into *indica* and *japonica* rice cultivars (Li et al, 2006; Hu et al, 2012; Xu, 2013). Marker-assisted introgression of *BPH31* gene into Jaya, an *indica* variety, significantly improves resistance to different BPH biotypes (Prahalada et al, 2017). Marker-assisted backcross programme is also used to transfer the resistance gene *BPH18* from IR65482-7-216-1-2 (an introgression line) into Junambyeo (an elite *japonica* cultivar). A tightly linked sequence-tagged site marker along with rigorous phenotypic selection for the quality traits leads to the development of improved lines possessing higher resistance to concerned biotype and excellent grain quality (Suh et al, 2011). Hu et al (2016) incorporated three BPH resistance genes (*Bph3*, *Bph14* and *Bph15*) into the genetic background of a semi-dwarf high-yielding *indica* cultivar, using similar strategies.

NILs developed through backcross program are important source of mapping population. Also, NILs developed through marker-assisted backcrossing are very much useful in tagging the concerned gene as

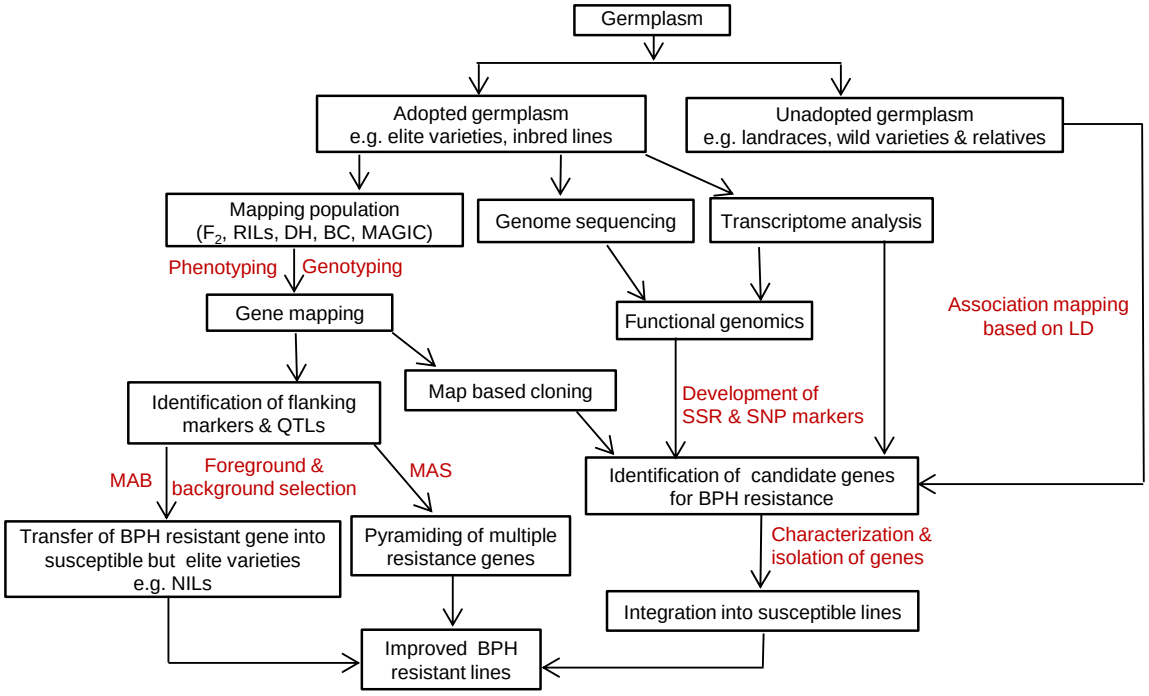


**Fig. 1. Flowchart indicating use of molecular breeding and genomics method for developing brown planthopper (BPH) resistant rice lines.**
BC, Backcross; DH, Double haploid; LD, Linkage disequilibrium; MAB, Marker-assisted breeding; MAS, Marker-assisted selection; MAGIC, Multi-parental advanced generation intercross; NILs, Near isogenic lines; QTLs, Quantitative trait loci; RILs, Recombinant inbred lines; SNP, Single nucleotide polymorphism; SSR, Simple sequence repeat.

well as high recovery of parent genome by using high density SNP chips. Using two SSR markers tightly linked to *Bph30*, a novel NIL has been developed, which shows strong antibiosis and high resistance to BPH (Wang et al, 2018). Similarly, a cross between two popular Srilankan BPH resistant lines (Kolyal and Poliyal) with a susceptible line (9311) has been successfully utilized for generating NIL and fine mapping of *Bph33* to a 60-kb region on chromosome 4S (Hu et al, 2018).

### *Gene pyramiding*

Studies have indicated that incorporating more than one resistance gene into a single rice variety increases the durability of resistance. For example, varieties harboring both *Bph14* and *Bph15* genes exhibit higher resistance compared to introgression lines containing either *Bph14* or *Bph15* (Hu et al, 2012). Jena et al (2017) reported that the NILs carrying two to three pyramided genes exhibit a stronger level of antibiosis (49.3%–99.0%) compared to the NILs possessing a single resistance gene. Qiu et al (2012) also reported that incorporating two resistance genes (*Bph6* and *BPH12*) into a common genetic background results in a significant additive effect against BPH compared to single isogenic lines. Later, Hu et al (2016) reported higher resistance of the pyramided lines containing three BPH resistance genes (*Bph3*, *Bph14* and *Bph15*) compared to single gene introgressed lines. The susceptibility of pre-NILs possessing either *Bph25* or *BPH26* gene against a particular biotype (Japan-KG-06) can be overcome by developing pyramided lines harboring both the resistance genes, indicating the possible broadening of resistance due to gene pyramiding (Myint et al, 2012). Three types of effects of gene pyramiding for BPH resistance have been reported i.e., additive, partial additive and non-additive effects, suggesting the partial or complete elimination of minor or narrow spectrum effect of one gene with the other gene having major or broad spectrum resistance (Hu et al, 2013). These studies suggest that deployment of multiple genes combine different mechanisms, which suppress the dominance of virulent biotypes in insect population and extend the stability and durability. However, there are some contradictory reports of the presence of the same level of resistance in isogenic and pyramided lines. One line carrying two genes (*Bph1* and *bph2*) exhibits the same level of resistance as the line carrying only *Bph1* or *bph2* gene, violating the additive nature of gene deployment strategies (Sharma et al, 2004; Hu et al, 2016). Hence, more studies are needed to predict

about the genetic effects of pyramiding involving the resistance genes in question.

*Multi-parent advanced generation intercross (MAGIC)*

In MAGIC design (also called funnel breeding design), multiple inbred parents are inter-crossed several times in a definite pattern to harness the genetic materials of all the parents into a common genetic background. This leads to highly diverse lines each with a unique mosaic of parental alleles. MAGIC population possesses greater advantage compared to a classical biparental population due to the use of large number of parents and genetic recombination events involved. To date, MAGIC populations have been established in a various crops including rice, maize, barley, tomato, faba bean and sorghum (Stadlmeier et al, 2018). In case of BPH resistance breeding, Satturu et al (2020) employed MAGIC panel consisting 391 lines generated from eight *indica* parents and a total of 27 041 polymorphic SNPs to identify marker-trait association. Finally, 190 significant marker associations and 92 annotated genes were identified across the chromosomes, of which 13 genes are typically associated with BPH resistance.

*Association mapping*

Primary goal of association mapping is to detect correlations between genotypes and phenotypes in a sample of individuals based on linkage disequilibrium (Varshney et al, 2005). In this method, unrelated rice genotypes or natural populations, such as wild species, ancestral cultivars and landraces, can be used, which would provide greater resolution for identifying BPH resistance genes.

*Post-transcriptional gene silencing*

RNAi basically connotes the action of small interfering RNAs and microRNAs in silencing the expression of a particular gene through the cleavage of the concerned mRNA and subsequently blocking protein synthesis. This technique is now frequently used in resistance breeding programmes of rice in general and BPH in particular. For example, silencing of Tyrosine hydrolase (*Th*) gene, a crucial survival gene involved in cuticle tanning and immunity, through microinjection of dsRNA molecules (*dsNiTh*), leads to the rapid death of the BPH population (Liu et al, 2020). In another experiment, female BPH fed with transgenic rice with silenced *AKTIP* (AKP-interaction protein) shows reduced growth with lower body weights. Detail analysis indicated the efficient blocking of *NlAKTIP* leads to significant reduction in the expression of concerned mRNA levels and the treated BPH population. This indicates that this protein is essential for growth and development of female BPH. Hence, *NlAKTIP* can be a potential target in BPH resistance breeding (Hao et al, 2015).

*Genome editing technology*

It has the potential in suitably designing varieties for resistance to various pests and diseases, by creating desirable mutations. CRISPR/Cas9 is one such genome editing technology that can be used in resistance breeding. Editing *CYP71A1* (encoding tryptamine 5-hydroxylage) by employing CRISPR/Cas9 results in an increased SA level and decreased serotonin levels in rice, which ultimately enhances BPH resistance (Lu et al, 2018). This indicates the possibility of utilizing genome editing technology for BPH resistance breeding.

## Conclusion and future perspectives

Green revolution has played a critical role in achieving food security by developing hybrids and high-yielding varieties. BPH, a potential threat to rice cultivation, is being continuously managed through the huge application of synthetic pesticides even at the cost of degrading the environment, destroying the natural predators as well as declining the plant innate immunity. Therefore, exploitation of host-plant resistance is an effective eco-friendly approach to control the BPH population and maintain the yield potential of cultivars.

The foremost step to progress for host-plant resistance is to identify and characterize the resistance gene present in the natural germplasm, particularly diverse wild species. At present, around 37 major genes/QTLs have been reported from different gene pools. Knowledge about the molecular mechanism operating for host-pest interaction can be achieved through gene mapping. Research should not only be focused on map-based cloning of BPH resistance gene but also on the genomics of the pest. It is important to identify the genes in the pest that help to overcome the resistance mechanisms operating in rice plants. Similarly, novel genes in rice imparting stable resistance mechanisms through various biochemical pathways have to be identified. After successful identification and validation, these genes can be introgressed into elite lines to develop NILs or can be pyramided into single variety by molecular breeding. Pyramiding of genes from diverse sources is the most efficient way to develop variety having broad spectrum resistance. Genome editing tools can be exploited to create specific mutations for improved resistance to novel BPH biotypes. Also, more studies are needed on the

level and pattern of expression of resistance genes when combined together in different genetic backgrounds of rice. The metabolomics and physionomics of resistance need to be explored for better understanding of host-insect interaction. Nowadays, next generation sequencing platform along with improved bioinformatics pipeline can easily pave the way for solving these problems. Using these facilities and gathering knowledge on suitable molecular method, approaches to identify new resistance genes and mechanisms can be explored.

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