Molecular mapping and transfer of a novel brown planthopper resistance gene bph42 from Oryza rufipogon (Griff.) To cultivated rice (Oryza sativa L.)

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Abstract
Background Brown planthopper (BPH), Nilaparvata lugens (Stål), is one of the most destructive pests of rice accounting for 52% of annual yield loss. The breakdown of resistance against known BPH biotypes necessitates the identification and deployment of new genes from diverse sources. The current study aimed at mapping and transfer of a novel BPH resistance gene from the wild species of rice O. rufipogon accession CR100441 to the elite rice cultivar against BPH biotype 4.

Methods and Results The phenotypic screening against BPH biotype 4 was conducted using the standard seedbox screening technique (SSST). Inheritance study using damage score caused by BPH infestation at the seedling stage indicated the presence of a single major recessive gene with the segregation ratio of susceptible to resistant plants in 3:1 (210:66, \( \chi^2 = 0.17 \leq \chi^2_{0.05,1} = 3.84 \)). The genotyping of the mapping population was done using polymorphic microsatellite markers between PR122 and O.rufipogon acc.CR100441 spanning all the 12 chromosomes of rice. A total of 537 SSR markers were used to map a BPH resistance gene (designated as bph42) on the short arm of chromosome 4 between RM16282 and RM6659. QTL analysis identified a peak marker RM16335 contributing 29% of the phenotypic variance at 40.76 LOD.

Conclusions The identified marker co-segregates with the bph42 and hence could be efficiently used for marker-assisted selection (MAS) for the transfer of resistance into elite rice cultivars. The introgression lines with higher yield and BPH resistance were identified and are under advanced yield trails for further varietal release.

Keywords Brown planthopper · Oryza rufipogon · QTL mapping · Linkage map · Bulked segregant analysis · Marker-assisted selection

Introduction

Rice (Oryza sativa L.) is one of the staple food crops that supply a major dietary calorific intake to one-third of the human population, globally. Rapid climate change and various abiotic and biotic stresses are the major constraints of global rice productivity. Rice productivity has been constantly challenged by insects, pests, and diseases through the rapid evolution of resistant pathotypes/biotypes. Among the insect-pests, Brown planthopper (BPH) Nilaparvata lugens (Stål) is a major pest of rice that hampers grain yield by feeding and crippling the host plant. BPH extracts the food assimilates of rice plants using its stylet type mouthparts by sucking plant sap and oviposit their eggs in stems [33]. The outbreaks of BPH cause yield loss up to 60% under high infestation [17, 38]. Both nymphs and adults feed by sucking the cell saps of rice leaves which lead to dehydration of leaves, decrease in the number of tillers, retardation of plant growth, and development. BPH causes serious damage to rice plants directly by feeding on phloem sap giving a scorched appearance called ‘hopper burn’ [7]
and in most severe cases kills the entire plant during flowering. BPH indirectly damages the rice crop by transmitting ragged stunt virus (RRSV) and grassy stunt virus (RGSV) [54]. It has been reported ~10–40% yield loss due to these viruses [45]. Therefore, it is an urgent need to control BPH to ensure global food security.

Numerous pesticides have been recommended to control BPH infestation; however, the excessive use of these chemical pesticides leads to a resurgence of resistance in BPH biotypes and poses a high risk of environmental pollution [8]. Therefore, identification and utilization of host plant resistance from landraces and wild germplasm to develop resistant rice cultivars are considered as one of the most effective, economic, and environment-friendly approaches to manage BPH [4]. The program for genetic analysis and identification of the BPH resistance gene started in 1968 [30]. Afterward, numerous efforts were made to explore and identify BPH resistance genes from diverse germplasm resources. So far, 41 genes/QTLs have been identified from cultivated and wild species of rice and assigned to different chromosomes of rice. Among them, nine genes (Bph14, Bph3/Bph17, Bph26, bph29, Bph18, Bph6, Bph32, Bph9, and Bph30) have been isolated using the positional cloning approach [19]. Most of the BPH resistance genes are found in clusters on the rice chromosome. For example, Bph3/Bph17 [40], Bph6 [31], Bph12(t) [49], Bph15 [50], bph18(t) [20], Bph20(t) [32], Bph27(t) [9], Bph27 [12], Bph30 [43], Bph34 [18] and Bph36 [21] and Bph41 [44] have been mapped on chromosome 4 and eight genes, Bph1 [15], bph2 [41], Bph10 [28], Bph9 [39], Bph18(t) [14], Bph19(t) [6], Bph21(t) [32] and Bph26(t) [51] were mapped on chromosome 12 suggesting these two chromosomes as hotspots for BPH resistance genes in rice against biotype 1, 2 and 3. This phenomenon of distribution of genes on chromosomes in clusters is consistent with the pattern of R genes [23]. However, only a few of the BPH resistance genes showed broad-spectrum resistance in monogenic rice lines [11] and the majority of the genes identified are ineffective against the evolving BPH biotype(s) prevalent in North-Western India [36]. Thus, scouting of new genes conferring BPH resistance against biotype 4 is urgently required.

Wild species of rice are the reservoir for many biotic and abiotic stress resistance genes [5]. Wild species including O. latifolia, O. minuta, O. eichingeri O. nivara, O. australiensis, O. rufipogon, O. officinalis, and O. punctata have been reported to carry resistance genes against different evolving BPH biotypes [46]. Most of the distant genomes are still untapped because of the several fertilization barriers. The transfer of genes from related “AA” genome species is relatively easier than distantly related species due to the high level of homology at the chromosome level. At Punjab Agricultural University, India, the wild species of rice O. rufipogon (2n = 2X = 24) carrying AA genome was identified as a promising resistance source against Biotype 4 of BPH over the past 4 years of screening. A mapping population was developed by hybridizing O. rufipogon accession CR100441 (BPH resistant) and Punjab Rice 122 (PR122) (BPH susceptible) with the objectives of understanding the genetics of resistance and identification of chromosomal location and markers linked to resistance gene for the efficient transfer of BPH resistance to the elite rice cultivars.

Materials and methods

Plant materials and development of mapping population

A collection of 1003 wild accessions of rice were screened against BPH biotype 4 at Punjab Agricultural University (PAU), Ludhiana, Punjab, India during the years 2012 and 2013, and 159 resistant accessions were identified [36]. Out of identified resistant accessions, one highly resistant O. rufipogon accession CR100441 (Score 1) was selected as a donor for genetic analysis, mapping, and transfer of BPH resistance. The O. rufipogon accession CR100441 was crossed with high-yielding but highly susceptible cultivar PR122 to generate F1. The F1 hybrid was backcrossed with PR122 to generate BC1F1. The BC2F2 population and BC2F3 progenies were used for the genetic analysis and mapping of the BPH resistant locus (Supplementary Fig. 1).

Raising and maintaining BPH

The seeds of the susceptible rice cultivar Taichung Native 1 (TN1) were sown in the earthen pots filled with puddled clay soil and plants were allowed to grow for 30 days under the insect-proof glasshouse conditions before feeding to insects. BPH biotype 4 insect culture was maintained on the 30 days old TN1 susceptible plants in a separate glasshouse maintained at 28 ± 2°C and 75 ± 5% relative humidity. Rectangular cages made of steel (0.68 × 0.50 × 0.50 m) covered with nylon size stitched along all sides except the top were used for rearing the test insect. For screening purposes, the 2nd and 3rd instar nymphs obtained thereafter were then released on the screening tray [36].

Phenotypic screening

The seedling of BC2F2, BC2F3, BC2F4, and BC2F5 populations were screened using SSST proposed by [10]. For screening of BC2F2 population, 276 pre-germinated seeds
of each individual along with parents were sown in wooden/plastic trays (45 cm × 35 cm × 10 cm) containing well-puddled soil (Supplementary Fig. 2a). An insect-proof greenhouse was used to raise the progenies which were maintained at 30±2°C, 80±5% relative humidity. After 14 days, the 2nd to 3rd instar nymphs of the hopper were released at 10–12 nymphs per seedling (Supplementary Fig. 2b). The wooden trays were covered with a fine-gauge nylon net on the iron cage (Supplementary Fig. 2c). The damage score was recorded following a 0–9 scale using the Standard Evaluation System (SES) when all seedlings of TN1 were dead. Each plant was scored as 0 = no visible damage, 1 = partial yellowing of the first leaf, 3 = first and second leaves of most of the plants partially yellow, 5 = pronounced yellowing and stunting or about half of the plants wilted or dead, 7 = more than half plants wilted or dead and remaining plants severely stunted, 9 = all plants wilted and dead [13]. For the evaluation of BC2F2 population, the plants with damage scores 1–3 were scored as resistant and 7–9 were scored as susceptible, (Supplementary Fig. 2d).

Further, 233 BC2F1 phenotypically superior lines with desirable characteristics were screened by sowing 30 seeds derived from single BC2F2 plants in each row along with the resistant and recurrent parent. The evaluation of BC2F3 family was done by averaging the damage score of 30 seedlings planted in each row. Based on the standard evaluation system, the mean score of each family with 0–4, 4.1–7, and 7.1–9.0 were regarded as resistant, segregating, and susceptible, respectively [10]. The same criterion was followed for the evaluation of BC2F4 and BC2F5 progenies. All resistant plants were screened twice to confirm their resistance. The descriptive statistics and distribution of BC2F3, BC2F4, and BC2F5 individuals were calculated using the ggplot2 package in R software. A Chi-square test for goodness of fit was used to study the inheritance pattern of resistance against BPH in each population.

**DNA extraction and construction of extreme bulks**

The genomic DNA was extracted from young leaves of 30-days old field-grown BC2F2 population including PR122 and O. rufipogon accession CR100441 using the standard CTAB method [34]. The genomic DNA was quantified using Nanodrop™ 8000 Spectrophotometer (Thermo Scientific, USA). Further, the quality and integrity of genomic DNA were evaluated by electrophoresis the DNA on 0.8% agarose gel was stained with Ethidium Bromide and was visualized in a gel documentation system (Biorad). The genomic DNA was normalized by adding 1X Tris EDTA buffer. Each DNA sample was further diluted to 50 ng/µl for PCR analysis.

Based on the phenotypic evaluation of the BC2F2 population, extreme bulks were prepared by pooling the equal concentration of the DNA from ten highly resistant individuals and ten highly susceptible individuals to make resistant bulk (RB) and ten highly susceptible (SB) respectively. The RB and SB were prepared in three replications, each bulk consisting of 10 different individuals in equal concentrations.

**Genotyping using rice SSR markers**

A parental polymorphism survey between PR122 and O. rufipogon acc. CR100441 was conducted using 558 SSR markers [29, 42] distributed over the 12 chromosomes of rice. The polymorphic markers were used for BSA to identify the linked markers. The Polymerase chain reaction (PCR) was applied on two bulked genotypes i.e., RS and SB including PR122 and O. rufipogon acc. CR100441. The SSR markers were amplified in a total of 10 µl PCR reaction containing 50 ng of genomic DNA (25 ng/µl), 10 pmol of each primer, 2.5 mM each of dATP, dTTP, dGTP, and dCTP, 1 unit of Taq DNA polymerase (Promega), 1X PCR buffer, and 1.5 mM MgCl2. Reactions were performed in a thermal cycler (Eppendorf) programmed at 94 °C for 5 min, followed by 35 cycles of 1 min at 94 °C, 1 min at 56 °C and 1 min at 72 °C with a final extension of 7 min at 72 °C. PCR amplified products were resolved in 3% agarose gel and individual alleles were scored in correspondence to parental alleles. Further, identified linked markers were applied to the mapping population for QTL analysis.

**Linkage map construction and QTL mapping**

The linkage map was constructed using ICI Mapping software ver. 4.0 [22]. A Chi-square test was performed to check the normal Mendelian behavior of the markers. SSR markers with distorted segregation were excluded from the linkage analysis and those having clean segregation patterns were subjected to linkage map construction. The Kosambi function was used to estimate map distance and most likely orders were preferred to plot the linkage map. A default logarithm of odds (LOD) threshold was used for grouping the anchored markers. After grouping, all markers, ordering using nTwoOpt algorithm (nearest neighbor for tour construction, and two-opt for tour improvement) was selected to construct the genetic linkage map. Each group was ripped separately with SARF (sum of adjacent recombination fractions) criteria. QTL mapping was also performed using Windows QTL Cartographer (Version 2.5_011) mapping
five randomly chosen plants from ground level to the tip of the tallest panicle and averaged, tillers were counted from five plants, randomly chosen at two places from inner rows; grains number per panicle were recorded by counting total grains (including sterile spikelets) from five panicles each in five randomly-chosen plants and grain weight was recorded by weighing fertile, filled 1,000 grains counted from a bulk of ten spikes. Data recorded for the above traits were subjected to statistical analysis as per the analysis of variance for RBD using SAS software ver. 9.2 (SAS Institute Inc., 2003) and interpretations were made accordingly.

**Phenotypic evaluation of BC₂F₅ population for agronomic traits**

The set of 224 BC₂F₅ progenies, recipient parent PR122 and donor parent *O. rufipogon* accession CR100441 were planted in a randomized complete block design (RBD) with a plot size of 7.68 m² in three replications. The crop was raised following recommended agronomic practices. Observations on yield and yield components along with other traits were recorded. Briefly, plant height was measured on

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**Fig. 1 (a):** Screening of BPH resistant *O. rufipogon* acc. CR100441 and highly susceptible cultivar PR122. (b) Evaluation of BC₂F₃ progenies against BPH biotype 4 (c) Evaluation of BC₂F₄ population against BPH biotype 4. Red arrows indicate homozygous susceptible progeny rows.
Results

Phenotyping and inheritance study

The mean BPH damage score between the two parents differed markedly in both years 2012 and 2013 (Fig. 1a). The genetic analysis of resistance present in O. rufipogon was studied in BC$_2$F$_3$, BC$_2$F$_4$, and BC$_2$F$_5$ populations (Supplementary Table 1). The inheritance pattern of genetic loci conferring BPH resistance was confirmed using screening of 276 individual BC$_2$F$_2$ plants, out of which 66 plants were found resistant and 210 plants were susceptible fitting to 3 (Susceptible): 1 (Resistant) segregation ratio ($\chi^2_c = 0.17 \leq 3.8$, $\chi^2_{0.05,1}$) (Fig. 1b). The segregation ratio indicates the recessive nature of the BPH resistance gene of O. rufipogon. The BC$_2$F$_3$ population was used to confirm the inheritance of BPH resistance in O. rufipogon acc CR100441. Out of 233 BC$_2$F$_3$ progenies, 52 progenies were resistant, 109 were segregating and 72 were susceptible. The segregation ratio was as per the 1:2:1 ratio ($\chi^2_c = 4.42 \leq \chi^2_{0.05,2} = 5.99$), thus confirming a single major genetic locus controlled BPH resistance (Table 1). Further, genetic analysis was extended to 224 BC$_2$F$_4$ and BC$_2$F$_5$ progenies where the BPH-resistance scores of BC$_2$F$_4$ population showed a continuous distribution with 106 resistant plants (1–5.5) and 118 susceptible plants (5.6–9.0) presented an acceptable fit to 1:1 ratio ($\chi^2_c = 0.64 < \chi^2_{0.05,1} = 3.84$) (Fig. 1c). The similar trend was observed in BC$_2$F$_5$ lines with 97 resistant (1–5.5 score) and 127 susceptible lines (5.6–9.0 score) owing to $\chi^2_c = 3.1 < \chi^2_{0.05,1} = 3.84$. These results exhibit the presence of BPH resistance in O. rufipogon acc CR100441.

| BPH damage score | < 3.5 | 3.6-7.0 | 7.1-9 | $\chi^2$ | $\chi^2_{0.05,1}$ |
|------------------|-------|---------|-------|---------|------------------|
| BC$_2$F$_2$ (#276) | 66    | -       | 210   | 0.17(1.3) | 3.84             |
| BC$_2$F$_3$ (#233) | 52    | 109     | 72    | 4.42 (1:2:1) | 5.99 (1:2:1) |

1. The number written in parenthesis is individual used in the experiment
2. (-) represents no individuals having BPH damage score 3.6-7.0

Fig. 2 Violin plots showing distribution of mean damage score of BC$_2$F$_3$, BC$_2$F$_4$ and BC$_2$F$_5$ individuals. Horizontal red bars indicate the average value.
of a single QTL determining BPH resistance in the selected mapping population. In addition, the distribution of mean damage score of BC$_2$F$_3$ was primarily bimodal and skewed toward the susceptible parent PR122. However, the bimodal distribution of mean damage score in BC$_3$F$_4$ and BC$_2$F$_3$ was more prominent and stabilized (Fig. 2).

**Molecular mapping of the BPH resistance gene**

A total of 194 polymorphic SSR markers throughout the genome were applied to RB, SB, and two parental lines for BSA (Supplementary Table 2). The only SSR marker RM16335 located on the short arm of chromosome 4 showed linkage to the RB as per BSA (Fig. 3a). This provided the primary indication of the presence of the genetic loci on chromosome 4 S that confers resistance to BPH. The RM16335 was also applied on the open bulks showed concurrence with the BSA (Fig. 3b). The polymorphic SSR markers were applied to the BC$_2$F$_2$ population for precise mapping of the identified gene. Out of 196 polymorphic markers, 56 markers segregating in the population were used further for mapping while the remaining markers showed distorted segregation patterns (Supplementary Table 3).

**QTL analysis**

To further confirm the effect of QTL, the linkage map was constructed using the BC$_2$F$_2$ mapping population covering 12 chromosomes with a total map length of 648.04 cM. The BPH resistant locus was mapped on the short of chromosome 4 flanked by SSR markers RM16282 and RM6659 at 40.76 LOD score exhibiting 29% phenotypic variance. This locus was tentatively designated as bph42. The RM16335 was a peak marker that was closely associated with the bph42 (Fig. 3c). The map distance between the flanking markers RM16282 and RM16335 was 6 cM (Supplementary Fig. 3).

**Validation and transfer of bph42 locus**

To validate this tightly linked marker, 224 BC$_2$F$_3$ progenies were genotyped with the peak marker RM16335 exhibited an equal ratio of amplicons corresponding to donor and recurrent parent. The marker RM16335 was perfectly co-segregating with the bph42 locus which could be utilized in the MAS program for transferring the bph42 locus into cultivated rice (Supplementary Fig. 4).

**Identification of superior resistant lines for varietal release**

The significant variation observed for different agronomic traits in the BC$_2$F$_3$ population derived from PR122/O. rufipogon acc. CR100441 (Supplementary Table 4). The resistant BC$_2$F$_3$ individual showing desirable agronomic traits such as high tiller number and thousand-grain weight and optimum plant height were selected (Supplementary Fig. 5). Considerable variation was observed for plant height (71.6-111.6 cm), the number of panicles/plant (22.6–25.6), grains/panicle (157.6-141.6), and thousand-grain weight.
A maximum increment of 26.11% was observed for the number of tillers per plant in Line144, 15.88% for grains/panicle in Line76, and thousand-grain weight 25.35% increment was observed in L-244. These outperforming lines directly or indirectly can be used in crop improvement programs. Transgressive segregation reported for plant height, panicle per plant, grains/panicle, and 1000-grain weight indicates the contribution of beneficial alleles from the wild parent. The above observation showed that the bph42 controls the BPH resistance in rice without any yield penalty which could be used for breeding high yielding cultivars with enhanced BPH resistance.

**Discussion**

**O. rufipogon as potential donors for biotic stress and productivity traits**

In this study, we reported the genetic analysis and mapping of a major recessive BPH resistance locus bph42 on the short arm of chromosome 4 from *O. rufipogon* accession CR100441. Being a progenitor of cultivated rice, *O. rufipogon* is one of the most studied wild relatives of rice serving as a rich reservoir for many useful traits and stress resistance genes. *O. rufipogon* also acts as a rich pool of favorable alleles for numerous agriculturally important traits including biotic and abiotic stress tolerance genes [27]. At PAU, an active collection of ~400 accessions of *O. rufipogon* were screened for various useful agronomic traits and disease resistance. Of which, useful donors were identified for resistance against BPH biotype 4 [36], resistance to the most recently evolved pathotypes of *Xanthomonas oryzae* pv. *oryzae* (Xoo) PbXo-10 and PbXo-8 [25]. *O. rufipogon* was also a donor for the transfer of bacterial leaf blight resistance gene named *Xa23* against Chinese (C1-C7), Philippine (P1-P10), and Japanese (T1-T3) races [53]. Approximately, 182 accessions of *O. rufipogon* were investigated for phosphorus uptake efficiency. Among them, IRGC104639, IRGC104712, and IRGC105569 have been identified to possess novel alleles of *Phosphorus uptake 1 (Pup1)* locus which could be used as potential resources for improving phosphorus uptake efficiency in rice [26]. For improving yield potential, 1780 backcross inbred lines (BILs) were developed by hybridization of 70 accessions of six wild species including *O. rufipogon* and six of them are recognized as promising donors for improving yield in cultivated rice by introgression of yield-enhancing QTLs [1, 2]. QTL for panicle length (*plt1.1, plt1.2, plt2.1, plt4.1, plt8.1, plt9.1 & plt9.1*), spikelets per panicle (*sppl.1, sppl.2 and sppl9.1*), grains per panicle (*gpp1.1, gpp8.2, and gpp12.1*), spikelet fertility percentage (*pss2.1 and pss4.1*), and 1000-grain weight (*gw4.1, gw8.1, gw9.1, gw11.1, and gw12.1*) from *O. rufipogon* in interspecific BC2 testcross population were also identified [47].

**Segregation distortion**

In our analysis, we observed that the SSR markers were distorted towards the alleles from *O. sativa* on chromosomes 2, 5, and 6. The phenomena of segregation distortion are frequently seen in inter-specific and inter-subspecific crosses. The reason behind this could be attributed to the presence of the gametophytic gene/gamete eliminator gene which is responsible for the distorted transmission of the gametes [37, 48]. Segregation distortion also affects the detection power of QTL when these loci are linked to the QTL leading to spurious detection of QTLs [52]. A study where the *S1* locus responsible for gamete elimination was mapped from *O. glaberrima* on the rice chromosome 6. Another gene, *S10* was mapped on the same location as *S1* in an inter-subspecific cross derived from *Indica* and *japonica* cross. The segregation distortion locus *S6* has already been reported in wild rice *O. rufipogon* [16, 35]. Segregation distortion on chromosomes 2, 3, and 6 was reported in the *F2* population derived from a subspecific cross between Nipponbare and Guanglui-4 [3]. It is evident from the above-cited reports that the segregation distortion loci are an integral part of the wild *Oryza* genome which diverges from the primary gene pool. Observation of strong marker segregation distortion provides an opportunity to understand the biological evolution of the gametophyte/staminate gene inherited from the wild relatives of rice.

**A novel recessive gene bph42 confers resistance to BPH**

So far, a total of 13 BPH resistance QTLs have been mapped into two distinct clusters on chromosome 4. Among them, 8 genes (*Bph3/Bph17* [40], *Bph12(t)* [49], *Bph15* [50], *Bph20(t)* [32], *Bph30* [43], *Bph36* [21], *Bph41* [44], and *QBph4.3* [24]) are clustered on the short arm of chromosome 4 whereas, 5 genes (*Bph6* [31], *Bph27* [12], *bph18(t)* [20], *Bph27(t)* [9], and *Bph34* [18]) are grouped on the long arm of chromosome 4. Based on the marker information, *Bph3/ Bph17, Bph12(t), Bph15, Bph20(t), Bph36,* and *Bph41* were located in the sub-telomeric region of chromosome 4, hence doesn’t coincide with the position of bph42. Only two of the reported genes *Bph30* and *QBph4.3* located in the same region (Fig. 4). In the present report, the bph42 completely covers *Bph30* and partially *QBph4.3*. The *Bph30* and *QBph4.3* are dominant [24, 43] in nature whereas *bph42* identified in this study is recessive in action indicating the
Novelty. Fine-mapping and map-based cloning of the \( bph42 \) gene may provide valuable information of candidate genes and its underlying mechanism that confers resistance to BPH in \( O. rufipogon \). The SSR marker RM16335 tightly linked with \( bph42 \) can be effectively used for MAS providing BPH resistance into elite rice cultivars.

Conclusions

The present study demonstrates the identification of a novel recessive BPH resistance locus from \( O. rufipogon \). The applicability of tightly linked markers with newly identified BPH resistance genes using MAS was shown for efficient introgression of this locus into the elite rice cultivar. These lines are also under multi-location yield trials and are in the process of varietal development. The advanced backcross populations developed in this study can serve as an important genetic resource for pre-breeding programs to broaden the genetic base of elite rice cultivars, particularly for yield-enhancing traits with enhanced resistance to BPH.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s11033-022-07692-8.

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Authors’ contribution Kumari Neelam, Kuldeep Singh, Yogesh Vikal: Conceptualization of the research, proofreading of the manuscript; Pavneet Kaur carried out all the genotyping and phenotyping of the mapping population; Kumari Neelam, Kishor Kumar, Ankita Babbar: Investigation, data analysis, writing of the original draft; Preetinder Singh Sarao provided BPH biotype 4 and screening facility; Renu Khanna, Rupinder Kaur, Gurjeet Singh Mangat: Handling of segregating generation, breeding and generation advancement, management of multi-location trials. All co-authors approved this manuscript before submission.

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Data Availability All the data generated and analyzed in this study are available in this article as supplementary material.

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