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Identification of quantitative trait loci for resistance to whitebacked planthopper, *Sogatella furcifera*, from an interspecific cross *Oryza sativa* × *O. rufipogon*

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Quantitative trait loci (QTLs) for resistance to whitebacked planthopper (WBPH), *Sogatella furcifera*, were determined using 202 backcrossed inbred lines derived from a cross between the recurrent parent Xieqingzao B and an accession of *Oryza rufipogon*, and then tested using chromosome segment substitution lines (CSSLs) selected from BC_3F_3 populations of the same cross. Seedling mortality with WBPH infestation was measured to evaluate the resistance. Three QTLs were detected and the wild alleles always had the effect for decreasing seedling mortality. There were *qWph2* located in the interval RM1285-RM555 on the short arm of chromosome 2, *qWph5* in RM3870-RZ70 on the long arm of chromosomes 5, and *qWph9* in RG451-RM245 on the long arm of chromosomes 9, among which *qWph9* had the most stable effect. Duplicate lines of seven CSSLs carrying homozygous alleles of *O. rufipogon* at *qWph9* were tested with WBPH infestation. Significant effects at $P = 0.01$ were observed for all the 14 lines. On the average, the seedling mortality was decreased by 55.2%. Validation of the major effect of *qWph9* on enhancing WBPH resistance not only provides a useful QTL but also a series of breeding materials for rice improvement.

Key Words: WBPH resistance, quantitative trait locus, backcrossed inbred line, chromosome segment substitution line, *Oryza sativa* L., *O. rufipogon* Griff.

Introduction

The whitebacked planthopper (WBPH), *Sogatella furcifera* Horváth, is one of the most serious sucking insect pest of rice, *Oryza sativa* L. Exploitation of the host plant resistance has been generally considered the most economical and environment-friendly approach for the management of this pest. Six major genes conferring WBPH resistance in rice have been identified, including *Wbph1* (Sidhu and Khush 1979), *Wbph2* (Angeles *et al.* 1981, Ravinder *et al.* 1982), *Wbph3* (Hernandez and Khush 1981), *wbph4* (Hernandez and Khush 1981), *Wbph5* (Wu and Khush 1985), and *Wbph6* (Min *et al.* 1991). Three of them, *Wbph1*, *Wbph2* and *Wbph6*, were tagged to DNA markers on chromosomes 7, 6 and 11, respectively (Causee *et al.* 1994, Liu *et al.* 2001, Ma *et al.* 2001). Breeding programs on incorporating genes for WBPH resistance into improved germplasm was initiated soon after the discovery of the resistance genes (Khush 1980).

In recent years, more and more attentions have been paid to the detection of quantitative trait loci (QTLs) associated with WBPH resistance. A number of permanent populations derived from inter-subspecies rice crosses were used, including recombinant inbred lines of Asominori/IR24 (Yamasaki *et al.* 1999a), double haploid lines of IR64/Azucena (Kadirvel *et al.* 1999, Geethanjali *et al.* 2009), Zaiyeqing 8/Jingxi 17 (Sogawa *et al.* 2001) and Chunjiang 06/TN1 (Sogawa *et al.* 2005), and backcrossed inbred lines (BILs) of Nipponbare/Kasalath//Nipponbare (Yamasaki *et al.* 2003b). QTLs for WBPH resistance were detected on all the 12 rice chromosomes except chromosome 9, some of which were validated using chromosome segment substitution lines (CSSLs) (Yamasaki *et al.* 1999b, 2000a, 2003a). Two additional QTLs, *Wbph7*(t) and *Wbph8*(t), were located on chromosomes 3 and 4, respectively, using an introgression line of *O. officinalis* as the resistance donor (Tan *et al.* 2004).

Since changes in insect biotypes are a continuing threat to rice production, there is always a need to identify and introduce new resistance genes into rice varieties. Wild relatives of cultivated rice are highly diversified and host various genes conferring resistance to biotic and abiotic stresses, thus providing a valuable gene pool for rice genetic improvement. The common wild rice, *O. rufipogon* Griff., is considered to be the progenitor of *O. sativa*, but this close relative of cultivated rice has not been utilized for the identification of genes conferring resistance to WBPH or another important sucking insect pest of rice, brown planthopper (BPH), *Nilaparvata lugens* Stål. The objectives of this study are to identify QTLs for WBPH resistance using BILs derived from an interspecific cross *O. sativa* × *O. rufipogon*, and to test the QTL effect in the genetic background of cultivated rice.

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Materials and Methods

Plant materials

The mapping population consists of 202 BILs derived from the inter-specific cross Xieqingzao B//Xieqingzao B/Dwr (Chen *et al.* 2006), in which *O. sativa* ssp. *indica* var. Xieqingzao B (hereafter referred to as XB) is the maintainer line of the dwarf-abortive cytoplasmic male sterile line Xieqingzao A, and Dwr is an accession of *O. rufipogon* from Dongxiang, Jiangxi Province, China. Using total DNA extracted from fresh leaf of a single plant for each of the 202 lines at BC_1F_5, a linkage map consisting of 149 DNA markers and spanning 1306.4 cM was constructed in our previous studies (Chen *et al.* 2006, Huang *et al.* 2008). Because the original *O. rufipogon* accession is not longer available, three DNA samples, including DNA from the recurrent parent XB and two DNA pools each consisting of 10 randomly-selected BILs, were used for parental survey of polymorphism. In the BIL population, genotypes were scored in relation to the DNA fragment detected for XB: lines showing XB fragment only were recognized as XB homozygotes, showing non-XB fragment only as Dwr homozygotes, and showing both XB and non-XB fragments as heterozygotes (Chen *et al.* 2006). Seeds were harvested from the plants providing DNA samples and used to derive BILs for the evaluation of WBPH resistance in this study.

After *qWph9* for WBPH resistance was detected in the XB//XB/Dwr BIL population, CSSLs were developed for the validation of this QTL. One BIL carrying introgression segments for *qWph9* was selected and backcrossed to XB for two generations, followed by two generations of selfing. The BC_3F_3 population was genotyped with simple sequence repeat markers RM215, RM245, RM1026 and RM205 on chromosome 9. In the assembled genome of Nipponbare (www.gramene.org), RM215, RM1026 and RM205 are located at the positions of 21189110, 22605178, and 22720660 bp of the 23011239 bp-long chromosome 9, respectively. The physical position of RM245 remains to be clarified, but it has been mapped between RM215 and RM205 (www.gramene.org) and was located between RM215 and RM1026 with tightly linkage to RM1026 in our recent studies (our unpublished data). Seven plants showing XB genotype at RM215 and Dwr genotypes at RM245, RM1026 and RM205, were selected. The resultant BC_3F_4 lines were grown with 12 plants per line in the winter-spring season in 2007–2008 in Lingshui, Hainan Province, China. At maturity, two seed samples were collected from each line. When an identical phenotype was observed among the 12 plants of a line, seeds from a single plant were collected as one sample and those from the remaining plants were mixed as the other. When segregation was observed, two single plants showing distinguishable phenotype were harvested respectively.

Evaluation of WBPH resistance

The WBPH resistance of the 202 BILs was evaluated in the summer of 2004 at the China National Rice Research Institute (CNRRI), Hangzhou, Zhejiang Province, China. The BILs and the recurrent parent XB were tested in two replications in plastic trays of $60 \times 45 \times 10$ cm^3. In each replication, 20 germinated seeds from each BIL were sown in a row of 20 cm with 3 cm spacing between rows. Three rows of the susceptible control Taichung Native 1 (TN1) were grown at random in each tray. Unhealthy seedlings were removed before infestation. The WBPH colony used for infestation was originated from WBPH macropterous adults collected from the paddy field at CNRRI in July 2002. The population was reared and maintained on the susceptible TN1 rice plants in the greenhouse at CNRRI. At the 2nd leaf stage, 2nd- to 3rd-instar nymphs of WBPH were released to the trays at a density of 10 insects per seedling. The test materials were measured for seedling mortality when the TN1 plants reached 95–100% of seedling mortality. The same evaluation was used for the duplicate samples of the seven CSSLs at CNRRI in 2008, except that three rows of XB were randomly grown in each replication and the original WBPH macropterous adults were collected from the paddy field at CNRRI in August and September 2007.

Data analysis

In the BIL population, seedling mortality measured from each replication and the mean values over two replications were used for QTL mapping, respectively. QTL analysis was performed using Composite Interval Mapping (CIM) and Multiple Interval Mapping (MIM) options of Windows QTL Cartographer 2.5 (Wang *et al.* 2005). Firstly, QTLs were mapped with CIM at a threshold of LOD = 3.0. Then, the significance of the multiple putative QTLs, as well as epistasis between the QTLs, was simultaneously tested with MIM. QTLs were designated following the rules recommended by McCouch and CGSNL (2008).

Seedling mortality of the CSSLs was analyzed using the following approach. Insect damage is similar to disease incidence in which a reasonable assumption can be made that the observed number of dead seedling m_{ij} on the i-th replication for the j-th line follows the binomial distribution with parameter π_{ij} and total number seedling n_{ij}, and the binomial probability at the dead seedling could be modeled by a generalized linear model (GLM) with a logit link (Piepho *et al.* 1999). The model for the probability of seedling mortality on the j-th line on the i-th replication is

$$\text{logit}(\pi_{ij}) = \log\left(\frac{\pi_{ij}}{1-\pi_{ij}}\right) = \eta_{ij} = \mu + rep_i + line_j + \varepsilon_{ij} \qquad (1)$$

where the term η_{ij} is known as the linear predictor in GLM, and the logit function is the so-called link function of the model, linking the binomial probability to the linear predictor η_{ij}, μ is the general effect, rep_i the effect for i-th replication, $line_j$ the effect for the j-th line, and ε_{ij} random error for the j-th line on the i-th replication.

The SAS GLIMMIX procedure was used to estimate the effect and make comparison of test lines with the recurrent parent XB by applying Dunnett-Hsu adjustment (SAS Institute 2006).

Table 1. Two-way ANOVA of seedling mortality in the BIL population

Source	SS	MS	*F*	*P*
Among the BILs	270305.6	1372.1	3.51	6.85×10^{-18}
Between the replications	6617.2	6617.2	16.93	5.68×10^{-5}
Error	76977.3	390.7		
Total	353900.0			

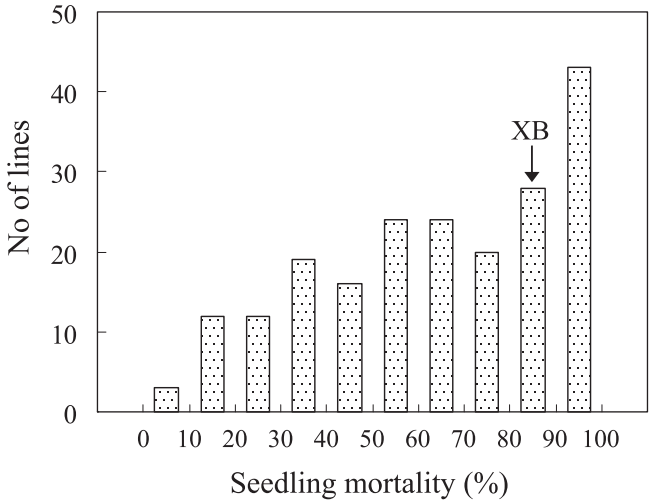

Fig. 1. Distribution of seedling mortality for resistance to whitebacked planthopper in the BIL population.

Results

Phenotypic performance of the BIL population

Great variation on the seedling mortality was observed in the BIL population. Analysis of variance (Table 1) indicated highly significant variation among the lines ($P = 6.85 \times 10^{-18}$), suggesting that genetic factors for WBPH resistance were segregated in the population. Highly significant variation was also observed for replications ($P = 5.68 \times 10^{-5}$), and the coefficient of determination between the two replications was estimated as 0.31. On average over two replications, the seedling mortality of the recurrent parent XB was 85.0% while the values among the BILs ranged from 6.7 to 100.0% with continuous variation (Fig. 1).

Since significant variation was observed between the two replications, data from each replication and the average data were independently used for QTL analysis. In addition, QTL analysis was performed using a subset of 150 BILs in which seedling mortality difference between the two replications was smaller than 30.0%.

QTL analysis using data from the original 202 BILs

Using the linkage map consisting of 149 DNA markers and spanning 1306.4 cM, QTLs for WBPH resistance measured in seedling mortality were determined with Windows QTL Cartographer 2.5. Totally three QTLs were detected using data from the 202 BILs, of which none was detected in both replications (Table 2 and Fig. 2).

Table 2. QTLs for WBPH resistance detected in the BIL population

Replication	QTL	Interval	LOD	Additive effect[a]	Variance explained (%)
I	*qWph5*	RM3870-RZ70	3.91	−11.1	7.6
	qWph9	RG451-RM245	3.58	−10.9	8.0
II	*qWph2*	RM1285-RM555	3.25	−9.1	6.5
Average	*qWph5*	RM3870-RZ70	3.61	−10.3	8.6
	qWph9	RM1896-RM201	3.28	−9.5	7.8

[a] The effect on seedling mortality (%) when a Xieqingzao B allele was replaced by an *O. rufipogon* allele.

Two QTLs were detected in Replication I. They were *qWph5* and *qWph9* located in the intervals RM3870-RZ70 and RG451-RM245 on the long arms of chromosomes 5 and 9, respectively. The wild alleles at *qWph5* and *qWph9* decreased the seedling mortality by 11.1% and 10.9%, explaining 7.6% and 8.0% of the phenotypic variance, respectively. The two QTLs were also detected using the average data, showing slight lower LOD scores and additive effects as compared with the values estimated from Replication I (Table 2). When *qWph5* and *qWph9* were simultaneously tested with MIM, they exhibited no significant digenic interaction (LOD = 0.025) while the individual effects remained to be significant.

QTL *qWph2* located in the interval RM1285-RM555 on the short arm of chromosome 2 was the only QTL detected in Replication II. It explained 6.5% of the phenotypic variance, and the wild allele decreased the seedling mortality by 9.1%. This QTL was not detected in Replication I and showed no significant effect based on the average data.

One more interval, RM1896-RM201 on chromosome 9, appeared to harbor a QTL responsible for seedling mortality variation in Replication I (Fig. 2). However, this interval was closely linked to *qWph9* and had a wide distance of 24.8 cM. To avoid the detection of artificial QTL effects due to real QTL in surrounding intervals, chromosome 9 was divided into two segments, one extending from RM316 to RM1896 and the other from RM201 to RM245, and QTL analysis was re-run using the genetic map consisting of 13 linkage groups. The effect at either RM1896 or RM201 was not longer significant, whereas *qWph9* remained significant with LOD score of 3.72. In addition, no significant effect was detected in the interval RM1896-RM201 when the 150 BILs having smaller phenotypic variation between replications was analyzed (Fig. 2). Thus no claim was made for the existence of QTL in this interval.

QTL analysis using data subset having smaller variation between replications

When QTL analysis was performed using 150 BILs in which seedling mortality difference between the two replications was smaller than 30.0%, no QTL was detected at the threshold of LOD = 3.0 (Table 3 and Fig. 2). The highest

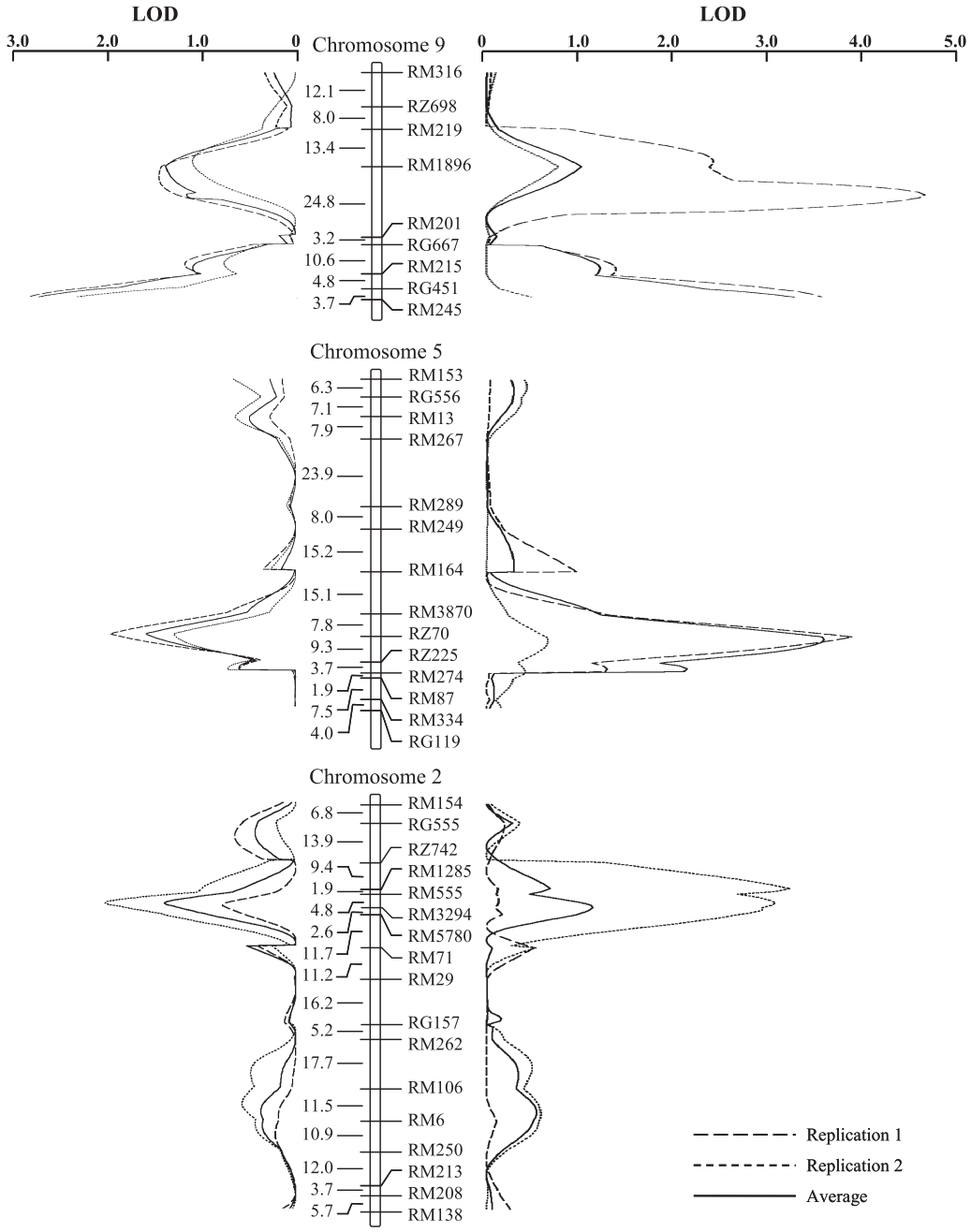

Fig. 2. LOD profiles of QTLs for WBPH resistance detected using data from the original 202 BILs (right hand side) and a subset of 150 BILs having smaller phenotypic variation between the two replications (left hand side). Marker names are listed on the right hand side of the chromosome with the genetic distances in Kosambi cM (Kosambi 1944) indicated on the left.

LOD score was found for *qWph9* in the interval RG451-RM245 on chromosome 9. This QTL was detected at LOD scores of 2.83 and 2.33 in the two replications, explaining 7.4% and 6.3% of the phenotypic variance with the wild allele reducing seedling mortality by 9.8% and 9.1%, respectively. Based on the average data, *qWph9* was detected at a LOD score of 2.73, explaining 7.3% of the phenotypic variance with the wild allele reducing seedling mortality by 9.5%.

The QTL having the second highest LOD score was *qWph2* located in the interval RM555-RM3294 on chromosome 2. It was detected at a LOD score of 2.02 in Replication II, explaining 6.2% of the phenotypic variance with the wild allele reducing seedling mortality by 10.6%. However, this QTL hardly worked in Replication I, having a low LOD score of 0.75 and a low contribution of 2.2% to the phenotypic variance. No more QTLs were shown to have LOD scores higher than 2.0.

It is noted that the two QTLs showing significant effects at the threshold of LOD = 2.0 in the 150 BILs having smaller

variation between replications were among the three QTLs detected at the threshold of LOD = 3.0 using the entire 202 BILs. The remaining QTL detected using the original 202 BILs, *qWph5*, had a marginal LOD score of 1.93 in Replication I and a lower LOD score of 1.27 in Replication II. Based on the QTL effects estimated using the subset of 150 BILs (Table 3), *qWph9* was obviously more stable than other QTLs. Thus *qWph9* was taken as the first candidate for validation.

QTL effects in the CSSLs

Based on the model set up (1) with $i = 1, 2$ and $j = 1, 2, ..., 15$ (14 test lines and one check), SAS GLIMMIX procedure was used to estimate the line effect. Following the detection of highly significant variation ($P < 0.001$) among the rice lines, multiple one-sided comparisons of the CSSLs with the recurrent parent XB was made based on their LS means by applying Dunnett-Hsu adjustment (Table 4). All the 14 lines carrying *qWph9* introgression segment from *O. rufupogon* showed decrease of seedling mortality at the 1% significance level. On the average, the seedling mortality was decreased by 55.2%. These indicated that *qWph9* had a major effect on enhancing WBPH resistance in the genetic background of the *indica* rice XB and the effect was stably expressed.

Table 3. QTL Effect estimated based on a subset of 150 BILs having smaller variation between the two replications

QTL	Interval	Replication	LOD	Additive effect[a]	Variance explained (%)
qWph2	RM555-RM3294	I	0.75	−6.3	2.2
		II	2.02	−10.6	6.2
		Average	1.39	−8.4	4.2
qWph5	RM3870-RZ70	I	1.93	−9.0	4.6
		II	1.27	−7.6	3.2
		Average	1.56	−7.9	3.7
qWph9	RG451-RM245	I	2.83	−9.8	7.4
		II	2.33	−9.1	6.3
		Average	2.73	−9.5	7.3

Table 4. The combined data of the two replications and the multiple comparisons of 14 CSSLs for *qWph9* with the recurrent parent Xieqingzao B (CK) by using Dunnett-Hsu adjustment

CSSL code	Subline	Total No. of seedling	Seedling mortality (%)	Decreased over CK (%)	Adj. *P* value
Wph9-1	1	38	57.9	33.4	0.0017
	2	38	42.1	49.2	<0.0001
Wph9-2	1	38	39.5	51.8	<0.0001
	2	32	28.1	63.2	<0.0001
Wph9-3	1	40	60.0	31.3	0.0023
	2	39	53.8	37.5	0.0005
Wph9-4	1	38	31.6	59.7	<0.0001
	2	39	23.1	68.2	<0.0001
Wph9-5	1	36	44.4	46.9	<0.0001
	2	35	22.9	68.4	<0.0001
Wph9-6	1	38	21.1	70.2	<0.0001
	2	38	2.6	88.7	<0.0001
Wph9-7	1	39	23.1	68.2	<0.0001
	2	36	55.6	35.7	0.0011
CK		115	91.3		

Discussion

The wild species of *Oryza* is an important resource of the genes for resistance to biotic and abiotic stresses. For the two most serious sucking insect pests of rice, introgression of genes into cultivated rice has been made from various species of the non-AA genome *O. officinalis* complex for BPH resistance and from *O. officinalis* for WBPH resistance (Brar and Khush 1997). Two genes introgressed from *O. officinalis*, *Wbph7*(t)/*Bph14* on chromosome 3 and *Wbph8*(t)/*Bph15* on chromosome 4, were found to be resistant to both WBPH and BPH (Huang *et al.* 2001, Tan *et al.* 2004). Other genes identified have shown resistance to BPH, including *O. australiensis*-introgressed *Bph10* and *Bph18*(t) on chromosome 12 (Ishii *et al.* 1994, Jena *et al.* 2006), *O. latifolia*-introgressed *Bph12* on chromosome 4 (Yang *et al.* 2002), and *O. eichingeri*-introgressed *Bph13*(t) on chromosome 2 (Liu *et al.* 2001). In the present study, three QTLs for WBPH resistance with favorable allele from *O. rufipogon* of the AA genome *O. sativa* complex were detected in a BIL population of *O. sativa* × *O. rufipogon*, of which *qWph9* having the most stable effect was validated using CSSLs.

The most reliable QTL for WBPH resistance identified in this study, *qWph9*, was located in the distal region of the long arm of chromosome 9 where no genes/QTLs for WBPH resistance have been mapped. Nevertheless, a major gene for BPH resistance (Mei *et al.* 1996) and a QTL controlling ovicidal response to BPH (Ren *et al.* 2004) were mapped in the distal region of chromosome 9. It has been shown that genes introgressed from wild rice could confer resistance to both planthopper species (Tan *et al.* 2004), so could QTLs for ovicidal response identified in cultivated rice (Yamasaki *et al.* 1999a, 2000b, 2003b). More work is needed to test whether *qWph9* confers multiple resistance against different species of planthoppers, and to clarify whether different genes mapped in this region share the same locus.

The recurrent parent XB used in this study is a major maintainer line of three-line hybrid rice. Verification of the genetic effect of QTLs for WBPH resistance in the XB background will greatly facilitate its utilization for rice improvement. The effect of the *O. rufipogon* allele at *qWph9* on enhancing WBPH resistance was validated in duplicate samples of seven CSSLs in the XB background, providing not only a useful QTL but also a series of breeding materials.

On the other hand, QTLs detected in this study only contribute a small proportion to the phenotypic variance of seedling mortality in the BIL population. This could be partly ascribed to phenotypic error due to sampling error and

variation on the vigor of the insect and the host plant, as well as to minor QTLs segregating in the population. In addition to *qWph2* and *qWph5*, minor QTLs were found in three more intervals when the "Searching for new QTLs" option of the MIM model was used after *qWph5* and *qWph9* were fixed in QTL analysis using average data of the 202 BILs. They were located in intervals RG532-RM5359 on chromosome 1, RM273-RM303 on chromosome 4 and RM219-RM1896 on chromosome 9, explaining 4.2, 4.0 and 4.5% of the phenotypic variance and showing LOD scores of 1.78, 2.03 and 2.05 in the MIM model, respectively. Verification of the effects of these minor QTLs in the genetic background of cultivated rice would be of great importance for QTL pyramiding for enhancing the WBPH resistance of rice varieties.

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