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Identification of Quantitative Trait Loci Conditioning Partial Resistance to *Phytophthora sojae* in Soybean PI 407861A

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ABSTRACT

Improving resistance for Phytophthora root and stem rot is an important goal in soybean [*Glycine max* (L.) Merr.] breeding. The objective of this study was to identify quantitative trait loci (QTLs) conferring partial resistance to *Phytophthora sojae* in an OX20-8 × PI 407861A cross, in which PI 407861A was used as the source of partial resistance. One hundred fifty-seven F_7–derived recombinant inbred lines (RILs) were evaluated for partial resistance to *P. sojae* isolate OH25 using a tray test. Composite interval mapping identified three QTLs on chromosomes 8, 13, and 15 by a genomewide logarithm of odds (LOD) threshold and six QTLs on chromosomes 3, 4, 10, and 18 by chromosomewide LOD threshold. The phenotypic variance explained by each QTL ranged from 2.4 to 8.6%, with a total of 41.6%. Quantitative trait loci on chromosomes 3 and 8 are novel QTLs first reported for partial resistance to *P. sojae* in this study. All nine QTLs were localized near known resistance gene rich regions or those previously reported for resistance to other soil-borne pathogens. Genomewide distribution of the QTLs in this population was distinct from the QTLs identified from previous studies with 'Conrad' but many were in common with those identified with another South Korean soybean accession. This indicates that the underlying genes for partial resistance in these exotic accessions may be distinct from genes controlling variation for partial resistance observed in Conrad. Plant Introduction 407861A is a promising genetic source of alleles that can be used to increase the genetic diversity and enhance levels of partial resistance to *P. sojae* in North American soybean cultivars.

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Abbreviations: BLUP, best linear unbiased predictor; Chr., chromosome; CIM, composite interval mapping; LOD, logarithm of odds; OPA, Oligo Pool All; PCR, polymerase chain reaction; PV, phenotypic variance; QTL, quantitative trait locus; R, resistance; RIL, recombinant inbred line; S, susceptible; SCN, soybean cyst nematode; SNP, single nucleotide polymorphism; SSR, simple sequence repeat.

Phytophthora root and stem rot, caused by *Phytophthora sojae* (Kaufmann and Gerdemann), is one of several root diseases that impact soybean in the north central region of the United States (Schmitthenner, 1985). Phytophthora root and stem rot suppresses yield of soybean and annual economic losses up to US$300 million in the United States have been reported (Wrather and Koenning, 2009). *Phytophthora sojae* is a soil-borne oomycete that can survive as oospores in soil or soybean plant debris for many years and germinates under saturated soil conditions (Schmitthenner, 1985). Infection by *P. sojae* can occur on roots throughout the growing season. Disease symptoms include pre- and postemergence seedling damping off at early growth stages and brown lesions that will develop beginning from the root progressing up the stem when infections occur at later growth stages followed by wilting, chlorosis, and plant death.

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Genetic host resistance is one of the most effective strategies to control both biotic and abiotic stresses in many cropping systems. Two types of genetic host resistance have been reported in the soybean–*P. sojae* interaction. Single dominant genes (*Rps*) mediated resistance confers an immune type of resistance to a limited number of *P. sojae* isolates that carry the cognate avirulence (*Avr*) gene (Gijzen and Qutob, 2009). To date, 15 *Rps* alleles (*Rps1a*, *Rps1b*, *Rps1c*, *Rps1d*, *Rps1k*, *Rps2*, *Rps3a*, *Rps3b*, *Rps3c*, *Rps4*, *Rps5*, *Rps6*, *Rps7*, *Rps8*, and *Yu25*) have been mapped to nine loci on chromosomes (Chrs.) 3, 13, 16, and 18 (Anderson and Buzzell, 1992; Athow and Laviolette, 1982; Athow et al., 1980; Buzzell and Anderson, 1992; Demirbas et al., 2001; Diers et al., 1992; Gordon et al., 2006; Kilen et al., 1974; Mueller et al., 1978; Sun et al., 2011; Weng et al., 2001).

In addition to *Rps* gene-mediated resistance, partial resistance is another form of genetic resistance that is used to manage *P. sojae*, which has also been called quantitative resistance, field resistance, rate-reducing resistance, and tolerance (Schmitthenner, 1985; Tooley and Grau, 1982; Walker and Schmitthenner, 1984). During the last decade, many studies have focused on efforts to learn more about the expression of this type of resistance and to identify genomic quantitative trait loci (QTLs) associated with partial resistance to *P. sojae* using biparental mapping populations derived from crosses between susceptible and resistant genotypes. A majority of these studies have been focused on the soybean cultivar Conrad, which does not possess known *Rps* genes but has high levels of partial resistance to *P. sojae* (Burnham et al., 2003; Fehr et al., 1989; Han et al., 2008; Wang et al., 2010, 2012; Weng et al., 2007). Initially, two QTLs were mapped in Conrad to Chrs. 2 and 13 in three separate populations using tray tests (Burnham et al., 2003). These same loci were subsequently identified in two additional populations, Conrad × OX760-6-1 (susceptible [S]) with a different phenotypic assay (layer test) with four different isolates (Han et al., 2008) and Conrad × Hefeng25 (resistance [R]) with multiple field tests (Li et al., 2010). Several additional QTLs were identified on Chrs. 12, 13, 14, 16, 17, 18, and 19 in a larger Conrad × 'Sloan' population using tray tests (Wang et al., 2010, 2012) and on Chr. 16 in Conrad × OX760-6-1 population with field tests (Weng et al., 2007).

Numerous genetic sources of partial resistance to *P. sojae* have been identified, which include breeding lines and plant introductions (Dorrance and Schmitthenner, 2000; Li et al., 2010; Nguyen et al., 2012; Tucker et al., 2010; Wu et al., 2011). In QTL analysis of some of these resistance sources, only a few of the QTLs were mapped to locations similar to those identified in Conrad populations: on Chrs. 13 and 16 in V71-370 (R) × PI 407162 (S) (Tucker et al., 2010) and Chrs. 13 and 17 in the S99-2281 (R) × PI 408105A population (S) (Nguyen et al., 2012). The majority of the QTLs from these studies were mapped to chromosomes other than those found in Conrad. For example, QTLs were mapped on Chr. 20 in the V71-370 × PI 407162 population (Tucker et al., 2010), Chrs. 6, 10, and 15 in the Su88-M21 (R) × Xinyiniaoheidou (S) population (Wu et al., 2011), and Chrs. 1, 2, 3, 4, 7, and 20 in the OX20-8 (S) × PI 398841 (R) population (Lee et al., 2013). Quantitative trait loci were also mapped to the same chromosomes as those previously identified in Conrad but at distinct locations separated by as much as 40 cM (Li et al., 2010; Tucker et al., 2010). Several QTLs reported in these new sources, other than Conrad, also contributed 16 to 42% of phenotypic variance (PV) (Li et al., 2010; Tucker et al., 2010; Wu et al., 2011; Lee et al., 2013). Race specificity of *Rps* gene-mediated resistance and the emergence of *P. sojae* populations virulent to the *Rps* genes deployed in commercial cultivars highlight the importance of combining both types of resistances for a more effective disease management strategy against *P. sojae* in soybean (Dorrance et al., 2003; Walker and Schmitthenner, 1984).

Numerous South Korean Plant Introductions were identified as new sources of resistance that may contain unique genes conferring *Rps*-gene resistance or partial resistance to *P. sojae* (Burnham et al., 2002; Dorrance and Schmitthenner, 2000). North American soybean cultivars reportedly have a narrow genetic base as they were bred and selected from a small numbers of ancestors (Carter et al., 2004; Gizlice et al., 1994; Hyten et al., 2006). South Korean germplasm selected for resistance to *P. sojae* was shown to be genetically distinct from the U.S. collection through cluster analysis of genetic distance using simple sequence repeat (SSR) markers (Burnham et al., 2002). Therefore, the South Korean soybean germplasm would also be useful for increasing the genetic diversity of the U.S. soybean germplasm.

In an earlier study, the South Korean PI 407861A exhibited levels of partial resistance that were higher than that observed in Conrad (Dorrance and Schmitthenner, 2000). The present study aimed to characterize QTLs associated with partial resistance to *P. sojae* in a recombinant inbred line (RIL) population derived from a cross of OX20-8 (S) × PI 407861A (R). Identification of QTLs in new genetic sources will broaden the genetic base for current and future breeding programs to improve partial resistance of soybean cultivars against *P. sojae* as well as contribute to the investigation of a genomewide network of genes and QTLs associated with partial resistance to *P. sojae* in soybean.

MATERIALS AND METHODS

Plant Materials and DNA Extraction

A $F_{7:8}$ RIL population derived from a cross of OX20-8 and PI 407861A was used for this study. OX20-8 is a breeding line developed in Ontario, Canada, and highly susceptible to *P. sojae*. The initial cross was done in 2005 and seven F_1 seeds were planted

in the greenhouse to self. Then single-seed descent was used to advance the generation from F_2 to F_7 plants. A total of 157 F_7 plants were threshed individually to yield $F_{7:8}$ seeds. The DNA was extracted from young leaf tissue collected from parental lines and single F_7 plants at the V1 or V2 stage using a modified cetyltrimethylammonium bromide method (Mian et al., 2008).

Marker Genotyping and Linkage Map Construction

Ninety-six parental genotypes, including OX20-8 and PI 407861A, were initially genotyped with the Universal Soybean Linkage Panel 1.0 containing 1536 single nucleotide polymorphisms (SNPs) at Dr. Perry Cregan's laboratory at the United States Department of Agriculture, Agricultural Research Service, Beltsville, MD. An Oligo Pool All (OPA) assay was organized with a subset of 384 SNPs to genotype multiple populations using Illumina GoldenGate BeadXpress SNP genotyping (Illumina Inc.). Based on the initial SNP genotyping, 250 SNPs in this OPA assay were polymorphic between OX20-8 and PI 407861A. All 157 RILs were genotyped according to the protocol from Illumina at the Molecular and Cellular Imaging Center at the Ohio Agricultural Research and Development Center and scored for the polymorphic SNPs. Selected SSR markers (Song et al., 2004, 2010) were added to increase genome coverage. The polymerase chain reactions (PCRs) had 20 μL final volume containing 50 ng of template DNA, 1x PCR buffer, 1.0 mM of $MgCl_2$, 50 μM of each of the deoxyribonucleotide triphosphates, 0.1 μM of each of forward and reverse primers (IDT Inc.), and 1.0 U of *Taq* polymerase (GeneScript Corp.). The PCR program was 95°C for 5 min followed by 32 cycles of denaturing at 95°C for 30 s, annealing at 48 to 61°C for 30 sec, and extension at 72°C for 45 sec. An additional 10 min of extension at 72°C followed at the end of the last cycle. The PCR product was resolved on a 4% high-resolution agarose gel (Research Products International Corp.) by gel electrophoresis. The genetic map was constructed with JoinMap (Van Ooijen, 2006) using the Kosambi's mapping function. Linkage was determined at the logarithm of odds (LOD) threshold of 3.0 with a maximum map distance of 50 cM. The order of markers in linkage groups was compared with the Consensus Map 4.0 (Hyten et al., 2010).

Phenotypic Assay for Partial Resistance

The 157 $F_{7:8}$ RILs of the OX20-8 × PI 407861A population were evaluated for partial resistance using tray tests that measure the lesion length that developed on roots 7 d after inoculation with *P. sojae* as previously published (Burnham et al., 2003; Tucker et al., 2010; Wang et al., 2010). Briefly, ten 7-d-old seedlings per RIL were placed on a tray with a polyester cloth and a wicking pad, and a 1-cm wound was made on the tap root 20 mm below the crown with a scalpel. An agar-mycelial slurry from a 7-d-old culture of *P. sojae* isolate OH25 (virulence to 1a, 1b, 1c, 1k, and 7) was placed over the wound. The seedlings were covered with the polyester cloth, 10 trays were bundled, and two bundles (20 trays) were vertically placed in a 25-L plastic bucket with 2 L of deionized water. The lesion length (mm) on each seedling was measured from the inoculation site to the edge of lesion margin 7 d after inoculation. This experiment was arranged in a randomized complete block design with three replications completed over time, during 26 Sept. through 10 Oct., 30 Sept. through 14 Oct., and 13 Oct. through 27 Oct. 2011, respectively. Each bucket held 20 trays, 16 RILs, which were randomly selected, two parents, and two checks (Conrad and Sloan). There were 10 buckets in a single replication of the experiment. The check cultivars, Conrad and Sloan, have high level of partial resistance and moderate susceptibility to *P. sojae*, respectively (Burnham et al., 2003).

Statistical Analysis of Phenotypic Assay Data

The mean lesion length of 10 seedlings from each RIL was analyzed to obtain the best linear unbiased predictor (BLUP) by PROC MIXED procedure in SAS (SAS 9.1; SAS Institute, 2007) (Stroup, 1989). These BLUP values represented the estimated and relative phenotypic values, adjusted by excluding random effects, among RILs within the mapping population. The model was $Y_{ijkl} = \mu + R_i + B(R)_{ij} + C_k + G(C)_{kl} + \varepsilon_{ijkl}$, in which μ is the overall mean, R_i is the effect of the ith replication, $B(R)_{ij}$ is the effect of the jth bucket in the ith replication, C_k is the effect of the kth class of entry (k = 1, 2, 3, 4, and 5 for OX20-8, PI 407861A, Conrad, Sloan, and RIL, respectively), $G(C)_{kl}$ is the effect of the lth genotype within class for recombinant inbred lines only (genotypic variance [σ_G^2]), and ε_{ijkl} is the experimental error (σ^2). Class of entry was assumed to be a fixed effect and all other terms random effect. Variance components were estimated using the restricted maximum likelihood method. The broad-sense heritability on a line-mean basis was calculated as $\sigma_G^2/(\sigma_G^2 + \sigma^2/r)$, in which r is the number of replications per RIL.

Quantitative Trait Locus Identification

MapQTL 5 (Van Ooijen, 2004) interval mapping was performed to identify QTLs, and subsequently composite interval mapping (CIM) was done using "multiple-QTL method" function along with the selected cofactors by "automatic cofactor selection" function. Walking speed was set to 1 cM. Genome-wide and chromosomewide LOD thresholds were calculated by a 1000-permuation test at $\alpha = 0.05$ (Churchill and Doerge, 1994) and were used to determine the significance of each QTL. Although a genomewide LOD threshold is used in general, Lander and Kruglyak (1995) proposed that a certain level of association between marker and trait was meaningful to report even though not significant at experimentalwise 5% error rate. Subsequently, chromosomewide LOD thresholds were suggested as the lower levels of significance by Van Ooijen (1999). Logarithm of odds thresholds are dependent on chromosome map length and marker density (Van Ooijen, 1999); therefore, chromosomewide LOD thresholds were applied in this study, as the map lengths of chromosomal linkage groups varied because some chromosomes were not represented as a single linkage group. The total PV (%) explained by all QTLs was calculated using PROC REG in SAS (SAS Institute, 2007). The genetic map and locations of the QTLs identified in this study were graphically presented using MapChart 2.2 (Voorrips, 2002).

Table 1. Descriptive statistics of mean lesion length (mm) of parents, checks, and 157 $F_{7:8}$ recombinant inbred lines (RILs) of the OX20-8 × PI 407861A population.

Trait	Parent and check†				RIL population			
	OX20-8	PI 407861A	Conrad	Sloan	n§	Mean	Range	SD
Lesion length (mm)	40.7 a‡	17.5 c	20.0 c	33.3 b	157	27.5	9.1– 53.0	7.04

†Conrad has a high level of partial resistance. Sloan is moderately susceptible.

‡Numbers followed by same letter are not significantly different based on Fisher's least significant difference test at $P < 0.0001$ (PROC GLM; SAS Institute, 2007).

§Number of RILs assayed.

RESULTS

Evaluation of Partial Resistance in the Mapping Population

The level of partial resistance for each RIL was evaluated based on lesion length 7 d after *P. sojae* inoculation in phenotypic assay (tray test). In this study, the mean lesion lengths of 157 RILs were continuously distributed between 9.1 to 53.0 mm and there was significant difference among the RILs ($P < 0.0001$). The mean lesion lengths were 40.7, 33.3, 20.0, and 17.5 mm for OX20-8, Sloan, Conrad, and PI 407861A, respectively. The mean lesion lengths were significantly smaller in the resistant parent PI 407861A than the susceptible parent OX20-8 ($P < 0.0001$) over the experiments as expected, and the mean (27.5 mm) of all RILs was intermediate between the two parents. Conrad had significantly shorter mean lesion length than the moderately susceptible check Sloan (Table 1). The mean lesion lengths of the RILs were normalized into BLUP values using the mixed model. The frequency of the BLUP values among the RILs had a normal distribution between –9.3 and 12.9 and least square means of the parents and checks were 13.3, 5.9, –7.4, and –9.9 for OX20-8, Sloan, Conrad, and PI 407861A, respectively (Fig. 1). A lower BLUP value indicates a higher level of partial resistance in the present study. The broad-sense heritability for the partial resistance was 0.75.

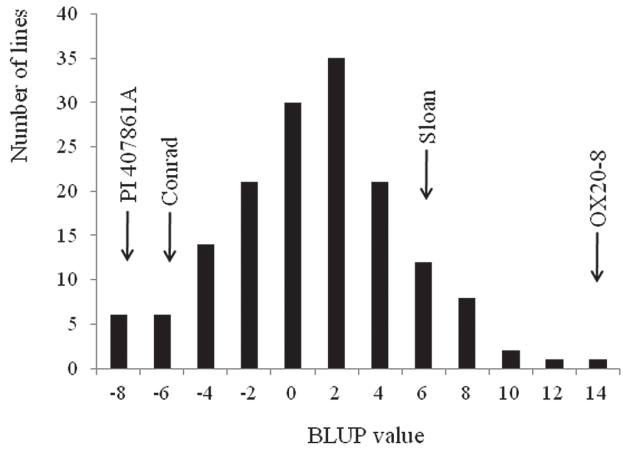

Figure 1. Frequency distribution of best linear unbiased predictor (BLUP) values estimated from mean lesion length (mm) of 157 recombinant inbred lines in the OX20-8 × PI 407861A population. The estimated values of two parents, Conrad, and Sloan, are indicated by arrows. A lower BLUP value means a higher level of partial resistance to *Phytophthora sojae* isolate OH25. OX20-8 is the susceptible parent, PI 407861A is the partially resistant parent, Conrad is the check for high levels of partial resistance, and Sloan is the moderately susceptible check.

Genotypic Map Construction

Of 384 SNPs in the OPA, 250 SNPs were polymorphic between OX20-8 and PI 407861A and 212 SNPs were used to construct an initial genetic map of the OX20-8 × PI 407861A population. Sixty-eight SSR markers were additionally genotyped to fill the large gaps on the initial map of with those 212 SNP markers. A total of 280 markers were integrated to the genetic map of the mapping population (Supplemental Fig. S1). This genetic map consisted of 36 linkage groups and spanned 1895.6 cM with an average distance of 6.7 cM between markers. The genetic map corresponded to 1648.3 cM based on the comparison to the latest soybean reference map (Hyten et al., 2010), which covered 71% of the soybean genome. The order of the markers was in accordance with the Consensus Map 4.0 (Hyten et al., 2010), with the exception of a few tightly linked markers.

Quantitative Trait Locus Identification

Composite interval mapping for each chromosome was used to estimate the location of each QTL associated with partial resistance to *P. sojae* isolate OH25. A total of nine QTLs were identified on Chrs 3, 4, 8, 10, 13, 15, and 18 from both OX20-8 and PI 407861A with a genomewide and chromosomewide LOD thresholds, which explained 41.6% of PV (Tables 2 and 3; Fig. 2). Three QTLs were significant at the genomewide LOD threshold (Table 2). Two QTLs (designated QTL-13 and QTL-8) were mapped between Sat_234 and BARCSOYSSR_13_1131 on Chr. 13 with LOD score of 5.0 and between BARC-051883-11286 and BARC-042715-08379 on Chr. 8 with LOD of 3.6 (Table 2; Fig. 2). QTL-13 and QTL-8 accounted for 8.8 and 7.2% of PV and their additive effects were –1.27 and –1.28, respectively (Table 2). The resistance alleles of these loci were contributed by PI 407861A as indicated by negative additive effects (Table 2). Another QTL (QTL-15) was identified between BARC-055329-13210 and BARCSOYSSR_15_0160 on Chr. 15 with LOD score of 4.4 (Table 2; Fig. 2). This QTL contributed 7.2% of PV, with an additive effect of 1.12, which was contributed by OX20-8 (Table 2).

Table 2. Quantitative trait loci for partial resistance to *Phytophthora sojae* isolate OH25 identified based on the genomewide logarithm of odds (LOD) threshold in the OX20-8 × PI 407861A population.

Chr. †	Loci	Position (cM)‡	Flanking markers	Composite interval mapping			
				LOD	Threshold§	R^2 (%)¶	*a*#
8	QTL-8	102 to 114	BARC-051883-11286 to BARC-042715-08379	3.6	3.1	7.2	–1.28
13	QTL-13	49 to 57	Sat_234 to BARCSOYSSR_13_1131	5.0		8.6	–1.27
15	QTL-15	16 to 19	BARC-055329-13210 to BARCSOYSSR_15_0160	4.4		7.2	1.12

†Chr., chromosome. The number followed by letters indicates separate linkages of the chromosome.

‡Genetic positions (cM) of the flanking markers of quantitative trait loci (QTLs) listed based on the Consensus Map 4.0 (Hyten et al., 2010). Approximate positions (cM) were given to BARCSOYSSR markers based on the nearest single nucleotide polymorphism marker (Song et al., 2010).

§A genomewide LOD threshold at $\alpha = 0.05$ calculated by a 1000-permutation test was 3.1.

¶Phenotypic variance explained by the single QTL.

#*a*, additive effect. The negative additive effects indicate that PI 407861A contributes resistance alleles for the QTLs.

Table 3. Quantitative trait loci for partial resistance to *Phytophthora sojae* isolate OH25 significant at chromosomewide logarithm of odds (LOD) threshold identified in the OX20-8 × PI 407861A population.

Chr. †	Loci	Position (cM)‡	Flanking markers	Composite interval mapping			
				LOD	Threshold§	R^2 (%)¶	*a*#
3	QTL-3a	21 to 27	BARC-028645-05979 to BARCSOYSSR_03_0317	2.4	1.7	3.6	–0.82
3	QTL-3b	65 to 85	Sat_091 to Sat_125	2.8	1.6	4.6	–0.97
4	QTL-4a	5 to 16	BARC-038359-10052 to BARC-054289-12451	1.6	1.4	2.4	0.71
4	QTL-4b	44 to 54	BARC-024445-04886 to BARC-061079-17031	1.6	1.5	2.5	0.69
10	QTL-10	70 to 100	BARC-060257-16508 to BARC-015925-02017	2.8	1.9	4.6	–0.93
18	QTL-18	70 to 81	BARC-040163-07672 to BARC-041331-07965	2.0	1.9	3.5	–0.87

†Chr., chromosome. The number followed by letters indicates separate linkages of the chromosome.

‡Genetic positions (cM) of the flanking markers of quantitative trait loci (QTLs) listed based on the Consensus Map 4.0 (Hyten et al., 2010). Approximate positions (cM) were given to BARCSOYSSR markers based on the nearest single nucleotide polymorphism marker (Song et al., 2010).

§Chromosomewide LOD threshold at $\alpha = 0.05$ was calculated by a 1000-permutation test using MapQTL5 (Van Ooijen, 2004).

¶Phenotypic variance explained by the single QTL.

#*a*, additive effect. The negative additive effects indicate that PI 407861A contributes resistance alleles for the QTLs.

Additional six QTLs were identified by CIM with a chromosomewide LOD threshold for each chromosome (Table 3). Two QTLs were identified on Chrs. 3 and 4 each; they individually contributed less than 5% of PV (Table 3; Fig. 2). Two loci (QTL-3a and QTL-3b) were mapped between BARC-028645-05979 and BARCSOYSSR_3_0317 and between Sat_091 and Sat_125, respectively, on two partial linkages of Chr. 3 (Fig. 2). The genetic distance between the two intervals was 40 cM according to the Consensus Map 4.0 (Hyten et al., 2010). Similarly, two loci (QTL-4a and QTL-4b) were located on the separate linkages of Chr. 4, between BARC-038359-10052 and BARC-054289-12451 and between BARC-024445-04886 and BARC-061079-17031, respectively. QTL-4a was located approximately 30 to 50 cM distant from QTL-4b according to the Consensus Map 4.0 (Fig. 2). Interestingly, the resistance alleles for the two QTLs on Chr. 4 were contributed by OX20-8 while the resistance alleles for both QTLs on Chr. 3 were from PI 407861A (Table 3). In addition, two more loci (QTL-10 and QTL-18) were detected between BARC-060257-16508 and BARC-015925-02017 on Chr. 10 and between BARC-040163-07672 and BARC-041331-07965 on Chr. 18 (Table 3; Fig. 2). The resistance alleles at these QTLs were also contributed by PI 407861A (Table 3).

DISCUSSION

In this study, 157 $F_{7:8}$ RILs derived from a cross of OX20-8 × PI 407861A were evaluated for levels of partial resistance with the *P. sojae* isolate OH25 using the tray test assay as described in previous studies (Burnham et al., 2003; Wang et al., 2010). The levels of partial resistance were estimated according to lesion length (mm) that developed on the root and expanded into the hypocotyl 7 d after *P. sojae* inoculation. The broad-sense heritability estimated by variance components was 0.75, which was in agreement with moderate to high heritability described in the previous studies (Burnham et al., 2003; Li et al., 2010; Tucker et al., 2010; Wang et al., 2010; Wu et al., 2011). High heritability indicates PI 407861A will be a valuable source in breeding cultivars with partial resistance to *P. sojae*.

The BLUP values and molecular marker genotypes of RILs were analyzed to detect significant statistical association using CIM. As a result, a total of nine QTLs were identified in this study, but individual contributions to PV were all less than 10%. Two QTLs were first reported in this study. First, QTL-8 had the largest effect in this population according to its additive effect (–1.28). Two previous studies identified QTLs for partial resistance to *P. sojae* at different regions on Chr. 8; two loci were located either near Satt632 (46 cM in soybean Consensus Map

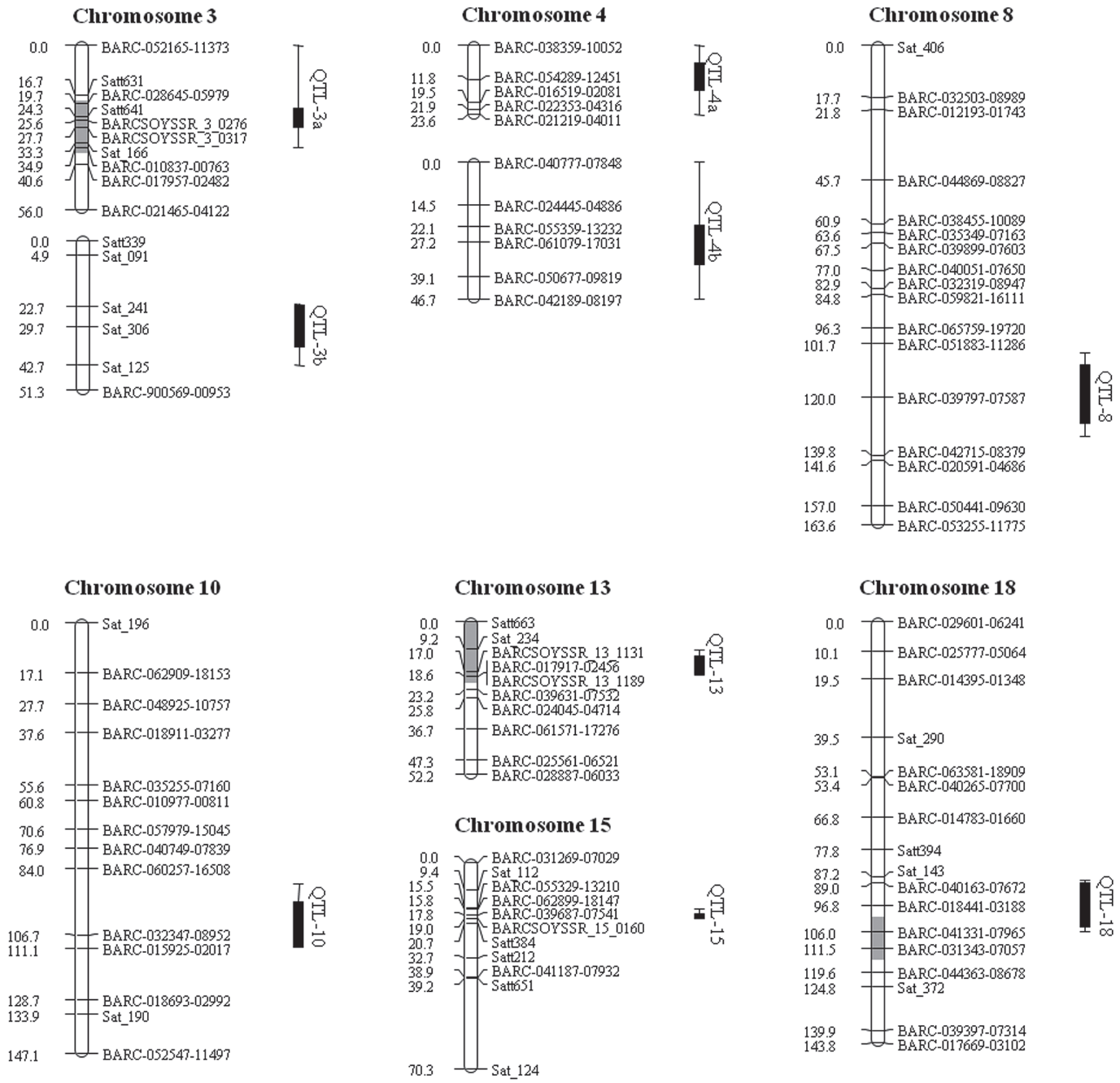

Figure 2. Partial genetic map of the OX20-8 × PI 407861A population and chromosomal locations of quantitative trait loci (QTLs) for partial resistance to *Phytophthora sojae* isolate OH25 identified by composite interval mapping with both genomewide and chromosomewide logarithm of odds (LOD) thresholds. The known *Rps*-gene regions on chromosomes 3, 13, and 18 were gray shaded on the genetic map based on the published studies (Demirbas et al., 2001; Gordon et al., 2006; Weng et al., 2001). Genetic distance (cM) and marker names are shown to the left and right of chromosomes, respectively. The bars and lines indicate the chromosomal locations of the QTLs. The one- and two-LOD intervals are displayed as black bars and solid lines to the right of chromosomes, respectively.

4.0) (Wang et al., 2012) or between Satt234 (86 cM) and Satt437 (93 cM) (Li et al., 2010). In this study, the QTL-8 was mapped between BARC-051883-11286 (102 cM) and BARC-042715-08379 (114 cM), which is over 50 and 10 cM away from the locations reported in the two previous studies. A few QTLs for seed oil and protein content were also reported in this region (Brummer et al., 1997; Chen et al., 2007). The second novel locus, QTL-3b, corresponds to the genetic location of an unnamed *Rps* gene as Satt549 (57.3 cM) and Satt660 (58.6 cM) were significantly associated with R-gene mediated resistance to the same *P. sojae* isolate used in this study (Zhang, 2009). *Rpg4* was also mapped near the location between Satt387 (44cM) and Sat_236 (47cM) (Mansur et al., 1996) and a few QTLs for oil also overlapped with QTL-3b (Chen et al., 2007; Qi et al., 2011). Co-localization of QTLs for partial resistance to *P. sojae* and oil has been previously reported (Wu et al., 2011).

Six of the nine QTLs identified in this study mapped to known *Rps*-gene regions or QTLs for partial resistance to *P. sojae*. QTL-13 in this population was identified in other populations is located in a well-documented R-gene rich region (Tucker et al., 2010; Wang et al., 2010, 2012) (Fig. 2). There are many resistance genes to bacterial blight (*Rpg1*), Phytophthora root and stem rot (*Rps3* and *Rps8*), soybean aphid (*Rag2*), and viral diseases (*Rsv1* and *Rpv1*) (Ashfield et al., 1998; Gore et al., 2002; Jeong et al., 2001; Jun et al., 2012) as well as a QTL for Sclerotinia stem rot caused by *Sclerotinia sclerotiorum* (Lib.) de Bary (Arahana et al., 2001). The QTL on Chr. 3 (QTL-3a) was mapped in the interval of 21.1 to 27 cM on the consensus map, which is in the region of *Rps1* and *Rps7*. *Rps1a* was flanked by markers Satt159 (21.3cM) and Satt009 (22.6 cM) while *Rps7* was flanked by markers Satt009 and Satt125 (34.6 cM) (Weng et al., 2007) (Fig. 2). A QTL for *P. sojae* was recently identified in this region (Nguyen et al., 2012) as well as resistance QTLs for Sclerotinia stem rot and soybean cyst nematode (SCN) (*Heterodera glycines*) and oil QTLs were mapped in this region (Arahana et al., 2001; Concibido et al., 1997; Qi et al., 2011). In the soybean Williams 82 reference genome (assembly version 1.01, http://www.phytozome.net/cgi-bin/gbrowse/soybean/, accessed 10 Sept. 2012), there are 20 archetypal resistance gene candidates (Glyma03 g04030, -04080, -04100, -04140, -04180, -04200, -04260, -04300, -04530, -04560, -04590, -04610, -04780, -04810, -05260, -05290, -05350, -05370, -05400, and -05420) annotated within the QTL-3a interval (Gm03: 2,993,383 to 5,800,563 bp). QTL-18 is located approximately 5 to 10 cM above the cluster of *Rps4*, *Rps5*, and *Rps6* genes (Fig. 2) and overlapped QTLs associated with isoflavone in seeds and resistance to SCN (Kabelka et al., 2005; Zeng et al., 2009). This genomic region also co-localized with QTLs associated with stearic acid, oleic acid, and palmitic acid in seeds (Reinprecht et al., 2006). Another locus QTL-10 was mapped in the interval of the QTL for partial resistance to *P. sojae* reported in the previous study (Wu et al., 2011) and co-localized with a glycitein (O-methylated isoflavone) QTL (Zeng et al., 2009). QTL-15 was also mapped 8 cM below a QTL for partial resistance to *P. sojae* previously reported in Wu et al. (2011), which overlapped with QTL for resistance to SCN (Qiu et al., 1999) and was located within 5 cM to QTL for resistance to Sclerotinia stem rot (Arahana et al., 2001).

It is not uncommon for the susceptible parent to be the source of QTLs that confer resistance. Tucker et al. (2010) previously reported that the resistance allele for a major QTL that explained approximately 42% of PV was contributed by the susceptible parent. In this study, the susceptible parent OX20-8 also provided resistance alleles at three loci (Tables 2 and 3; Fig. 2). Of the three, QTL-15 had a higher statistical significance with a LOD score of 4.4, which explained 7.2% of PV. The estimated additive effect (1.12) of QTL-15 was as high as QTL-8 or QTL-13 according to the absolute values of their additive effects (Table 2). The other two loci, QTL-4a and QTL-4b, had a relatively lower LOD score (1.6) and R^2 values (2.4 and 2.5) than QTL-15. Two QTLs for SCN resistance were previously identified in the region of the QTL-4a (Yue et al., 2001) and a QTL for resistance to sudden death syndrome caused by *Fusarium virguliforme* (Akoi, O'Donnell, Homma & Lattanzi) was reported 10 cM below QTL-4b (Njiti and Lightfoot, 2006). In addition, many QTLs related to seed weight, seed number, and seed amino acid composition were mapped in the QTL-4b region (Liang et al., 2010; Orf et al., 1999; Panthee et al., 2006).

All nine QTLs identified in this population contributed <10% of PV and co-localized with or neighbored R-genes, including *Rps* genes, or resistance QTLs for Phytophthora root rot and other root diseases. Clustering of R-genes and/or resistance QTLs is evident in many plants (Gebhardt and Valkonen, 2001; McMullen and Simcox, 1995; Wisser et al., 2005, 2006). In soybean–*P. sojae* interaction, two or more *Rps* genes are tightly linked on three genomic regions on Chrs. 3, 13, and 18 while *Rps2* was clustered with *Rmd*, *Rpp2*, and *Rj2* as well as QTLs for brown stem rot caused by the fungal pathogen *Phialophora gregata* and SCN on Chr. 16 (Graham et al., 2002; Guo et al., 2006; Patzoldt et al., 2005). A recent study in soybean suggested that over 60% of the disease resistance QTLs co-localize to 2-Mb flanking interval of predicted nucleotide-binding site-leucine-rich repeat but the disease resistance QTLs were distributed asymmetrically in both the number and function between pairs of the homeologous regions (Kang et al., 2012). Co-localization of multiple QTLs for soil-borne diseases implies these hot spots have been evolutionarily important for quantitative resistance to diverse biotic stresses in plants. These loci will be the highest priority to explore mechanisms of resistance to soil-borne pathogens but also primary targets for breeding resistance to multiple pathogens mentioned above.

Interestingly, the estimated positions of six QTLs mapped in this study overlapped or neighbored those of the QTLs previously mapped in OX20-8 × PI 398841 on Chrs. 3, 4, 13, 15, and 18 (Lee et al., 2013). The six QTLs in common included three *Rps*-gene regions mapped on Chrs. 3, 13, and 18, for which the resistance alleles were contributed by either PI 398841 or PI 407861A while OX20-8 is the donor of the positive alleles for other three loci (Tables 2 and 3; Fig. 2). Both PI 398841 and PI 407861A originated in South Korea and consequently may share common alleles contributing partial resistance to *P. sojae*. In addition to the genetic similarity of these resistance sources, the common maternal parental background of OX20-8 was probably important in identification of many common QTLs between the two populations. Recently, nested association mapping has been used

to study complex quantitative traits in maize (Poland et al., 2011; Yu et al., 2008). We plan to apply this approach to analyze QTLs for partial resistance to *P. sojae* in multiple populations sharing the common parent OX20-8.

Exotic germplasm has played an important role in providing new alleles in breeding for the resistance to biotic stresses (Carter et al., 2004). This study identified nine QTLs for partial resistance to *P. sojae* in the OX20-8 × PI 407861A population, of which two QTLs have not been previously reported. In addition to the new QTL, many QTLs were mapped to different regions and therefore are controlled by different genes from those identified in populations using Conrad, another cultivar with high levels of partial resistance. Genetic characterization of many resistant genotypes to a pathogen will provide breeders with a broader range of resistant germplasm for improvement of partial resistance to *P. sojae* as well as for the expansion of the genetic base of soybean cultivars in North America. Further studies will integrate all QTLs identified in multiple populations derived from a cross of OX20-8 and Plant Introductions and may lead to a better understanding of partial resistance to *P. sojae*.

Supplemental Information Available

Supplemental material is included with this manuscript.

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