Identification of QTL for Resistance to Sclerotinia Stem Rot in Soybean Plant Introduction 194639

T. D. Vuong, B. W. Diers, and G. L. Hartman*

ABSTRACT

Sclerotinia stem rot of soybean [*Glycine max* (L.) Merr.], caused by *Sclerotinia sclerotiorum* (Lib.) de Bary, is a difficult disease to manage, although some gains have been made through breeding for quantitative resistance. The objective of the present study was to map quantitative trait loci (QTL) controlling partial resistance to Sclerotinia stem rot from the soybean plant introduction (PI) 194639. The resistance QTL were mapped in a population of 155 $F_{4:5}$ recombinant inbred lines (RILs) developed from the hybridization of the partially resistant parent, PI 194639, to the susceptible cultivar Merit. The population was evaluated for Sclerotinia stem rot resistance using a cut stem inoculation technique and was genotyped with 134 simple sequence repeat (SSR) markers. Broad-sense heritability of lesion length (LL) after inoculation with the cut stem technique in the population was 0.57. Two putative QTL-controlling LL were identified by composite interval mapping (CIM) and mapped to linkage groups (LGs) A2 and B2, with likelihood of odds scores of 5.6 and 3.5, respectively. The LG A2 QTL was linked to the marker Sat_138 and explained 12.1% of the phenotypic variation for LL. The LG B2 QTL was proximal to the marker Satt126 and explained 11.2% of the phenotypic variance. Two minor QTL also were mapped onto LGs K and L, explaining 5.5% of the total phenotypic variation. A multivariate model that included all significant QTL explained 27% of the observed phenotypic variation of LL. These results suggest that SSR markers associated with resistance QTL mapped in this study for Sclerotinia stem rot resistance may be useful for marker-assisted breeding programs in soybean.

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Abbreviations: CIM, composite interval mapping; DAI, days after inoculation; LG, linkage group; LL, lesion length; LOD, likelihood of odds; PCR, polymerase chain reaction; PI, plant introduction; QTL, quantitative trait loci; RIL, recombinant inbred line; SSR, simple sequence repeat.

Sclerotinia stem rot [caused by *Sclerotinia sclerotiorum* (Lib.) de Bary] of soybean [*Glycine max* (L.) Merr.] is an important disease in the north-central United States. The fungus that causes Sclerotinia stem rot attacks many hosts, including canola (*Brassica napus* L.), dry bean (*Phaseolus vulgaris* L.), sunflower (*Helianthus annuus* L.), and tomato (*Solanum esculentum* L.) (Grau and Hartman, 1999). The initial infection of soybean occurs primarily by ascospores infecting senescing flower petals (Sutton and Deverall, 1983). Lesions are commonly observed on the main stems or on lateral branches (Boland and Hall, 1988). The fungus then proceeds to girdle stems and branches, resulting in plant death and yield losses. The occurrence and importance of the disease have been reported in most north-central states in the United States (Grau and Hartman, 1999). Specifically in Illinois, yield losses from commercial fields were shown to range from 0 to nearly 100% (Hartman et al., 1998; Hoffman et al., 1998).

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Under field conditions, both physiological resistance and escape mechanisms were reported to influence Sclerotinia stem rot disease severity (Boland and Hall, 1987; Coyne et al., 1974). In a study of dry bean, Coyne et al. (1974) concluded that modified plant architecture of cultivar Great Northern had a direct effect on disease escape mechanisms to *S. sclerotiorum*. Boland and Hall (1987) reported that maturity, lodging, plant height, and other canopy architecture traits were associated with disease escape in soybean to *S. sclerotiorum*. Nelson et al. (1991) also concluded that increased lodging, which resulted in a dense canopy favorable to disease development, was correlated with greater disease severity.

Inheritance of resistance to *S. sclerotiorum* has been reported in many crop plants including soybean (Abawi et al., 1978; Arahana et al., 2001; Fuller et al., 1984; Hoffman et al., 1999; Kim and Diers, 2000). In dry bean, resistance was initially reported to be under the control of a single gene (Abawi et al., 1978); however, others concluded that the resistance was multigenic, with each gene having a minor effect and influenced by the environment (Fuller et al., 1984; Miklas and Grafton, 1992; Miklas et al., 2004).

The inheritance of resistance to Sclerotinia stem rot in soybean also has been shown to be quantitative (Arahana et al., 2001; Han et al., 2007; Kim and Diers, 2000). Kim and Diers (2000) analyzed the field resistance of a population derived from a cross between cultivar Williams 82 and NK 'S19-90' and identified three resistance quantitative trait loci (QTL) on molecular linkage groups (LGs) C2, K, and M. Arahana et al. (2001) used the detached leaf inoculation technique to evaluate five populations derived from crosses between the susceptible cultivar Williams 82 and the partially resistant cultivars Corsoy 79, Dassel, Vinton 81, DSR 173, and NK 'S19-90'. They detected a total of 28 resistance QTL, which were mapped onto 15 LGs. Of these, many QTL detected from different genetic populations were mapped to the same intervals. In a greenhouse evaluation, Han et al. (2003) used a stem inoculation technique and identified seven QTL responsible for Sclerotinia stem rot resistance. Among these QTL, both Han et al. (2003) and Arahana et al. (2001) mapped QTL to the same intervals on LGs A2, B2, D2, and L.

Because the environment plays a large role in the success or failure of the pathogen to develop Sclerotinia stem rot, especially in the field, it is critical to control the environment when attempting to map QTL for physiological resistance. This need for environmental control has resulted in the development of several laboratory and greenhouse inoculation methods to evaluate Sclerotinia stem rot resistance in soybean. Among these methods, the cut stem inoculation method (Vuong et al., 2004) was shown to be a reliable and nondestructive method to quantitatively evaluate Sclerotinia stem rot resistance in controlled environmental conditions. This inoculation method also was shown to be highly correlated with disease severity under field conditions (Vuong et al., 2004).

Vuong and Hartman (2003) reported that PI 194639 showed greater resistance levels based on restricted stem lesion development of the disease compared to other PIs and current commercial varieties. The objective of our study was to map QTL controlling Sclerotinia stem rot resistance from PI 194639.

MATERIALS AND METHODS

Genetic Population

A genetic population was developed by crossing the Sclerotinia stem rot–susceptible cultivar Merit to the partially resistant PI 194639 (Vuong and Hartman, 2003). Five F_1 seeds were obtained from crossing in a greenhouse during the summer of 1999 and winter of 2000. After hybridity was verified, F_1 plants were grown to maturity to produce F_2 seed, and single-seed descent was used to inbreed to the F_4 generation. From these F_4 plants, 155 $F_{4:5}$ recombinant inbred lines (RILs) were developed for study.

Genetic Marker Analysis

Genomic DNA was isolated from young leaf tissue of four to five plants for each RIL and the parents following the CTAB protocol described by Sagai-Maroof et al. (1984), with minor modifications. Briefly, ground leaf tissue was mixed with CTAB extraction buffer, followed by an incubation period at 65°C for 1 h. A 24:1 mixture of chloroform and isoamyl alcohol was added to the suspension followed by shaking and centrifugation. The aqueous layer was colleted and treated with RNase. The DNA was precipitated by mixing with precooled isopropanol and centrifuged. The DNA pellet was washed with ethanol and dissolved in TE (Tris-HCl-EDTA, pH 8.0) buffer. The DNA concentration was quantified with a spectrophotometer (NanoDrop Technologies Inc., Centreville, DE) and diluted to 10 ng μL^{-1} for polymerase chain reaction (PCR) amplification.

Polymerase chain reaction amplifications were performed for simple sequence repeat (SSR) markers in 12.5 μL final volumes containing 20 to 30 ng of template DNA, 0.3 μM of 3' and 5' end primers, 1X reaction buffer (20 mM Tris-HCl, pH 8.0, 50 mM KCl; Invitrogen, Carlsbad, CA), 3 mM $MgCl_2$, 0.4 mM of each dNTP, and 1 unit of *Taq* DNA polymerase (Invitrogen, Carlsbad, CA) and 0.5 mg mL^{-1} BSA. The thermal cycler program consisted of 32 cycles of denaturation at 94°C for 25 sec, annealing at 47°C for 25 sec, and extension at 68°C for 25 sec. A 3-min extension at 72°C followed the last cycle. The PCR products produced with fluorescence-labeled primers were separated using an ABI Prism 377 DNA sequencer (ABI Applied BioSystems, Foster City, CA) as described by Patzoldt et al. (2005). Gel images were processed to score markers using the GeneScan 3.0 program (ABI Applied BioSystems, Foster City, CA). The PCR products produced with nonfluorescence-labeled primers were separated in 3% high resolution agarose (FMC, Rockland, ME) gels. The parents of the population were evaluated with over 220 SSR markers distributed throughout 20 molecular linkage groups of the soybean genetic map (Choi et al., 2007) to identify those that were polymorphic. One

hundred and thirty-four were polymorphic, and these were used to genotype the RILs.

Disease Assessment

Parents and all RILs were planted in a greenhouse and challenged with the fungal pathogen *S. sclerotiorum,* isolate 105HT. Greenhouse growth conditions and disease response evaluation methods were conducted as previously described (Vuong et al., 2004). Soybean seeds were germinated in 15-cm clay pots containing a 1:1:1 mixture of soil, perlite, and torpedo sand. The experimental unit was a pot containing five seedlings of each RIL or parent, which were allowed to grow for 5 wk up to growth stage V5 or R1 (Fehr et al., 1971) in a greenhouse set at 25°C and a 16-h photoperiod. At 5 wk after planting, the main stems of plants were horizontally severed above either the fourth or fifth node. A single mycelial plug, prepared as previously described (Vuong et al., 2004), was placed on the cut stem, followed by a 48-h incubation period in a mist chamber at 100% relative humidity and 20 ± 1°C. Infected plants were then transferred to a greenhouse bench at 25 ± 1°C to allow for further development of lesions. Lesion length (LL) was measured for each plant at 14 d after inoculation (DAI). The experiment was a randomized complete block design with two replications separated by time.

Data Analysis

Lesion lengths among RILs were tested for normality using the UNIVARIATE procedure of SAS 9.1 (SAS Institute, Cary, NC). The Shapiro-Wilk (W) *t* statistic was used to test the null hypothesis that LLs at 14 DAI were normally distributed among RILs (Elliott, 1999). The broad-sense heritability for LL at 14 DAI was estimated according to Nyquist (1991) based on the expected means squares derived from an analysis of variance (ANOVA) with the GLM procedure of SAS.

Linkage analysis was performed with the computer program JoinMap 3.0 (Van Ooijen and Vooriips, 2001) to construct a genetic linkage map using the Kosambi mapping function. A likelihood of odds (LOD) threshold score of 3.0 and a maximum distance of 50 cM were used for initial linkage grouping of markers. Linkage groups were designated according to the soybean composite map (Song et al., 2004). Composite interval mapping (CIM) was performed to identify QTL controlling LL using the MQM method with the program MapQTL 5.0 and the appropriate cofactor selection (Van Ooijen et al., 2002). The permutation test (Churchill and Doerge, 1994) was performed with 1000 runs to determine the $P = 0.05$ genomewide significance level for declaring QTL for LL significant. The proportion of the phenotypic variance explained by the QTL effects was estimated by CIM at the QTL peaks. Regression analysis was performed between the segregation of nonlinked markers and LL to map QTL in regions where markers were not linked. The total phenotypic variance explained by the significant QTL was determined using a multivariate ANOVA model of SAS. The linkage groups with LOD plots were subsequently created using the MapChart 2.2 program (Vooriips, 2002) based on output from JoinMap 3.0 and MapQTL 5.0.

The effectiveness of marker-assisted selection with markers linked to LL QTL was tested by comparing the mean LL of lines homozygous for the positive and negative alleles for the QTL. The student's *t* test was then used to compare the mean LLs.

RESULTS AND DISCUSSION

Phenotypic Variation

Lesion length at 14 DAI among RILs in the Merit × PI 194639 population ranged from 5.2 to 18.0 cm, while LL of Merit was 14.6 cm and PI 194639 was 6.5 cm (Fig. 1). For the Shapiro-Wilk (W) test, the null hypothesis that LL among the RILs was normally distributed was not rejected using $P = 0.05$, indicating that LLs at 14 DAI were normally distributed. This normal distribution of LLs shows that resistance to Sclerotinia stem rot was quantitatively inherited. This quantitative inheritance is consistent with previous studies on the inheritance of resistance to Sclerotinia stem rot in crop plants, such as dry bean (Miklas and Grafton, 1992; Miklas et al., 2004), sunflower (Bert et al., 2002), and soybean (Han et al., 2003; Kim and Diers, 2000).

The broad-sense heritability among RILs for LL was 0.57. This estimate was similar to previously reported broad-sense heritabilities for Sclerotinia stem rot in field tests that ranged from 0.30 to 0.71 for individual environments and 0.59 across environments (Kim and Diers, 2000). Our heritability is also consistent with reported broad-sense heritabilities in dry bean that ranged from 0.27 to 0.66 for LLs of excised inoculated stems and from 0.58 to 0.77 for a disease incidence index in field tests (Miklas and Grafton, 1992).

Genetic Linkage Analysis

Seventy-five of the 134 markers used to test the population were linked, forming 15 fractional LGs covering a total of 1495 cM. Average genetic distance between markers was 8.8 cM, varying from 0.2 to 24.5 cM, and the number of markers in each LG ranged from 3 to 11. The marker order and distance between markers was highly consistent with the soybean composite linkage map (Song et al., 2004), except for two marker order flips. This included flips in the order between Satt547 and Satt244 on LG J (data not shown) and between Sat_264 and Satt577 on LG B2 (Fig. 2).

QTL Associated with Sclerotinia Resistance

Two QTL significantly associated with LL were identified (Fig. 2) through CIM using an LOD threshold of 2.7, based on permutation tests that correspond to a genomewide significance threshold of $P = 0.05$. The first QTL mapped close to Sat_138 on LG A2, had an LOD of 5.6 ($P < 0.001$), and explained 12.1% of the phenotypic variation for resistance. The allele from PI 194639 conferred greater resistance and had an additive effect on LL of −0.84 cm. The second QTL mapped near Satt126 on LG B2, explained 11.2% of the phenotypic variation, and had an LOD of 3.5 ($P < 0.001$) (Fig. 2). The allele from PI 194639 was associated with less resistance

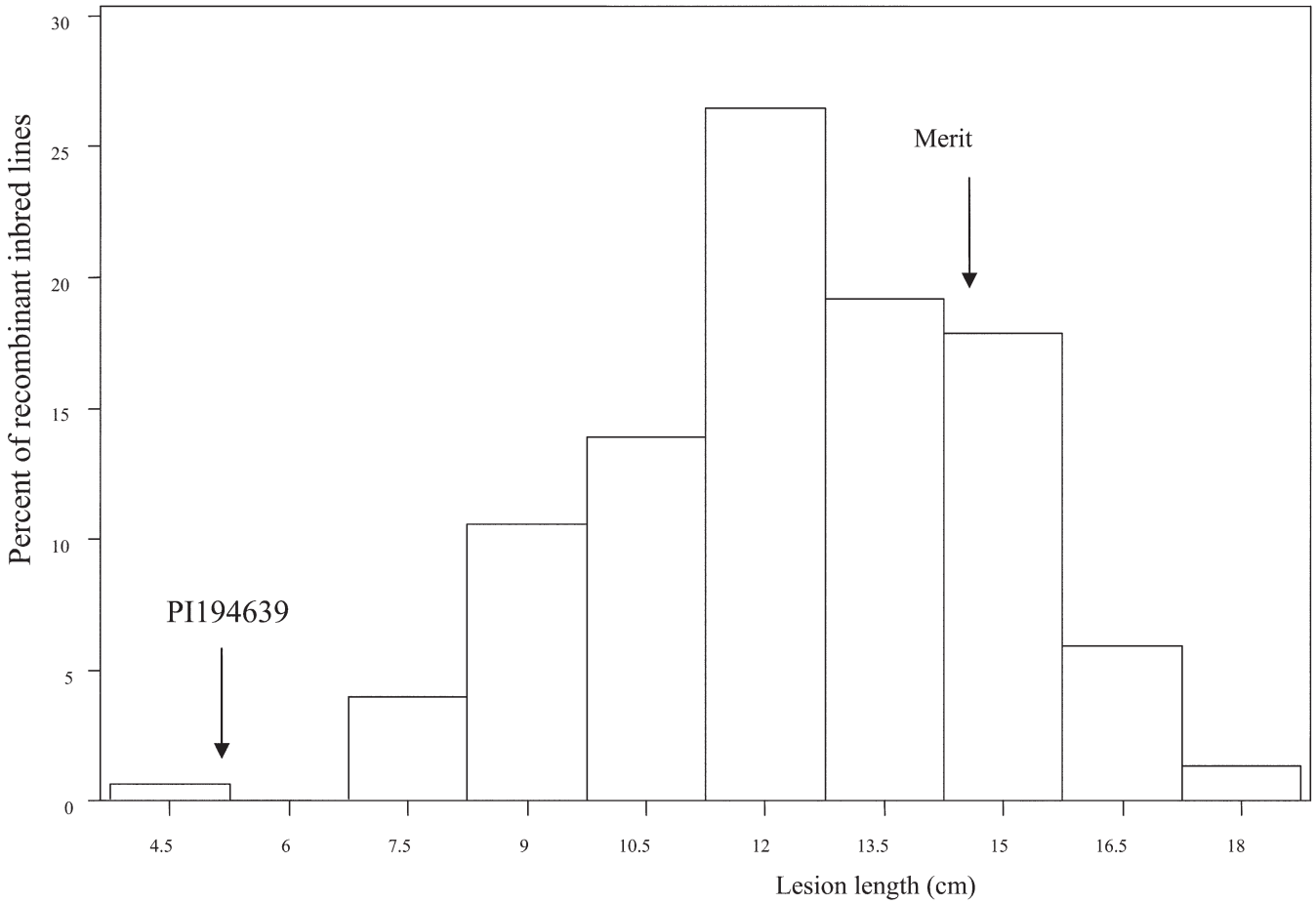

Figure 1. Distribution of lesion lengths (cm) among recombinant inbred lines (RILs) 14 d after inoculation of cut stems with mycelial plugs of *Sclerotinia sclerotiorum*. The RIL population is derived from a cross between the susceptible cultivar Merit and the partially resistant parent PI 194639.

and had an additive effect on LL of 0.86 cm. Attempts to map additional markers in the QTL interval on B2 were unsuccessful because of a lack of polymorphic markers in this region. When the markers most closely linked to the two QTL were included in a multivariate model, each marker was significant at $P < 0.001$, and together they explained 23.9% of the variation for resistance in the population. Based on regression analysis, none of the nonlinked markers were associated with LL.

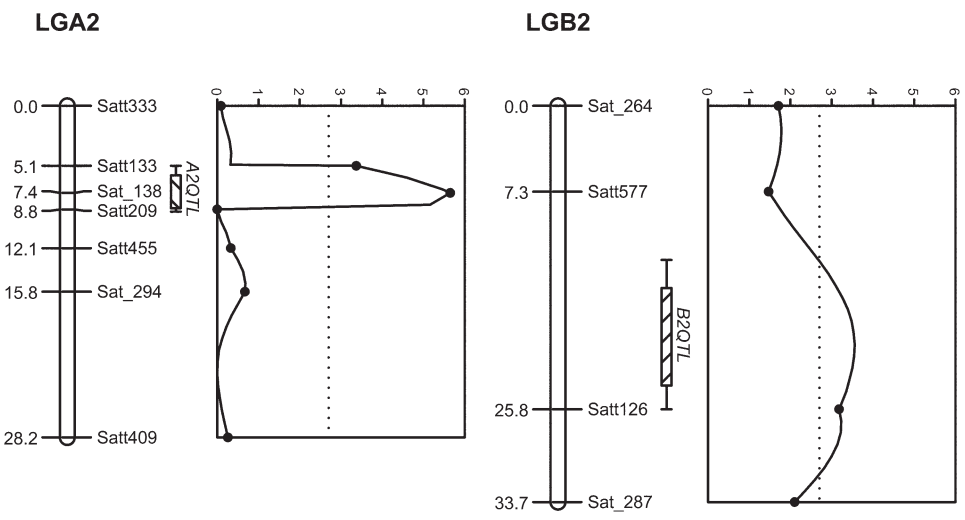

Figure 2. Likelihood of odds (LOD) plots for linkage groups A2 and B2 showing the locations of quantitative trait loci (QTL) providing resistance to Sclerotinia stem rot using an LOD threshold of 2.7. These QTL were mapped in a population of recombinant inbred lines developed from a cross between the susceptible cultivar Merit and the partially resistant parent PI 194639. The relative positions of the mapped markers are given in centiMorgan (cM).

Our mapping of QTL for Sclerotinia stem rot resistance with R^2 effects near 10% is similar to previously reported results. Arahana et al. (2001) reported R^2 values for Sclerotinia stem rot QTL that ranged from 4 to 10%. Kim and Diers (2000) mapped QTL with R^2 values that ranged from 9.6 to 7.8% across environments. Han et al. (2003) reported that two major QTL mapped on LG A1 and L explained 14 and 16% of total phenotypic variation, respectively.

The Sclerotinia stem rot resistance QTL we found on LG B2 has not been previously mapped. Both Arahana et al. (2001) and Han et al. (2003) did map Sclerotinia stem rot QTL to LG B2, but these QTL were 42 cM or greater from the QTL we mapped. In addition, Arahana et al. (2001) mapped QTL to LG A2 in two populations, but these QTL were at least 36 cM from the location where we mapped our QTL. Han et al. (2003) reported a minor QTL that was located 12 cM from the A2 QTL we mapped, and these are close enough to be potentially allelic. Additional work is needed to determine whether they are allelic or not.

In addition to the above-mentioned QTL, the markers Satt273 on LG K and Satt182 on LG L (Choi et al., 2007) also mapped putative QTL for LL. These QTL were not significant at the 2.7 threshold but were significant when the threshold was adjusted to an LOD of 2.0 ($P < 0.02$ based on the single-trait analysis) (Fig. 3). These putative QTL are interesting because they map to locations where Sclerotinia stem rot resistance QTL were previously mapped. The LG K QTL maps to the same location as a resistance QTL previously reported by Arahana et al. (2001) and within 10 cM of a QTL mapped by Kim and Diers (2000). Han et al. (2003) reported a QTL mapping 8 cM from the QTL we mapped to LG L. The LG K and L QTL each explained 5.5% of total phenotypic variation for disease resistance, and the allele from the PI parent for both QTL conferred greater resistance with additive effects of approximately −0.55 cm. When these markers were included in the multivariate model with the other two mapped QTL, the marker Satt273 was significant at $P < 0.05$ and Satt182 was not significant. The inclusion of these markers into the multivariate model increased the total phenotypic variation explained to 27%. These results suggest that the LG K and L QTL may be minor-effect resistance QTL.

It was widely known that the inheritance of resistance to Sclerotinia stem rot in soybean is complex, and no soybean genotype with complete resistance has been successfully developed. Consistent with our finding that the resistance allele for the LG B2 QTL was derived from Merit, the susceptible parent, other researchers also have mapped alleles conferring resistance to Sclerotinia stem rot from susceptible parents of their populations. Of the 28 Sclerotinia stem rot QTL mapped by Arahana et al. (2001), the resistance allele of 19 QTL was derived from the susceptible parent in at least one of the five populations they evaluated. Marker-assisted selection should be useful in helping breeders stack resistance genes from both resistant and susceptible sources into new resistant genotypes.

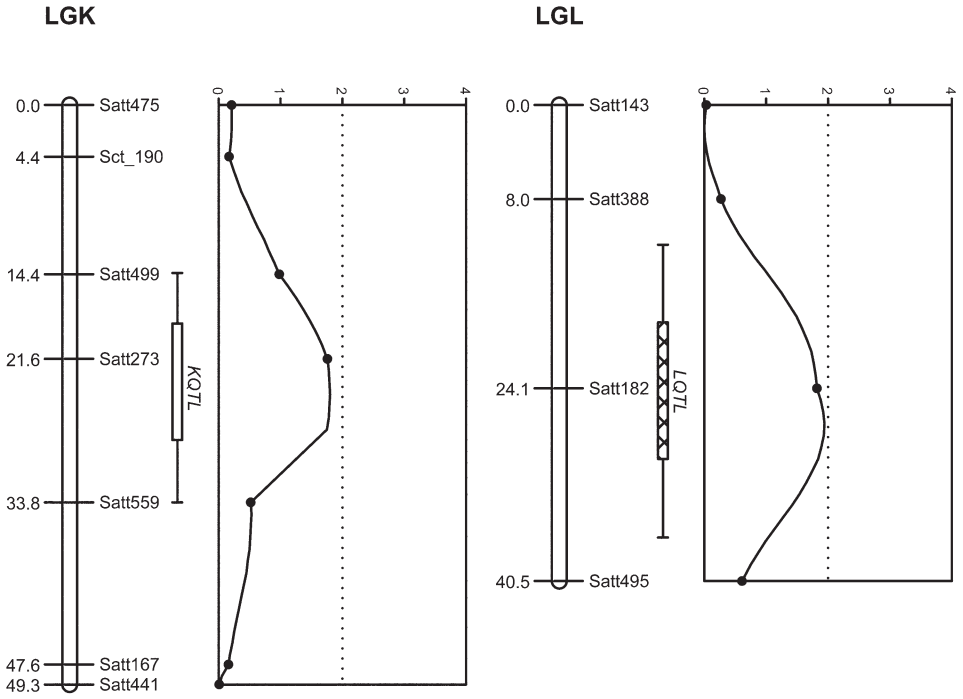

Figure 3. Likelihood of odds (LOD) plots for linkage groups K and L showing the locations of quantitative trait loci (QTL) providing resistance to Sclerotinia stem rot using an LOD threshold of 2.0. These QTL were mapped in a population of recombinant inbred lines developed from a cross between the susceptible cultivar Merit and the partially resistant parent PI 194639. The relative positions of the mapped markers are given in centiMorgan (cM).

In the present study, mean LLs of inbred lines that carried alleles from Merit, the susceptible parent, on LG A2 and carried alleles from the resistant parent, PI 194639, on LG B2 had significantly increased LLs compared to the other allelic combination (Table 1).

In summary, we mapped two new putative QTL responsible for Sclerotinia stem rot resistance to LG A2 and

Table 1. Mean lesion lengths of lines homozygous for combinations of marker alleles for quantitative trait loci associated with Sclerotinia stem rot resistance on linkage groups (LG) A2 and B2.

Pattern†	LG A2			LG B2		Lesion length
	Satt133	Sat_138	Satt209	Satt126	Satt577	
						cm
Allele a	a	a	a	a	a	12.8 A‡
Alleles a and b	a	a	a	b	b	11.8 A
Alleles b and a	b	b	b	a	a	14.7 B
Allele b	b	b	b	b	b	13.2 A

†a: homozygous for alleles from the resistant parent PI 194639; b: homozygous for alleles from the susceptible parent Merit.

‡Mean lesion lengths followed by a different letter showed statistically significant differences based on the student's *t* test at $P < 0.05$.

B2. Along with minor QTL on LG K and L, these QTL explained a considerable percentage of the heritable variation for LL in the population. These markers can be useful for marker-assisted breeding programs in soybean and can reduce dependence on phenotypic selection for this trait.

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