Phytophthora Root Rot Resistance in Soybean E00003

Zhongnan Zhang  
*Michigan State University*

Jianjun Hao  
*Michigan State University*

Jiazheng Yuan  
*Michigan State University*

Qijian Song  
*USDA– ARS Soybean Genomics and Improvement Laboratory, Beltsville, MD, qijian.song@ars.usda.gov*

D. L. Hyten  
*USDA-ARS, Soybean Genomics and Improvement Laboratory, Beltsville, Maryland, david.hyten@unl.edu*

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Authors
Zhongnan Zhang, Jianjun Hao, Jiazheng Yuan, Qijian Song, D. L. Hyten, P. B. Cregan, Guorong Zhang, Cuihua Gu, Ming Li, and Dechun Wang

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Phytophthora Root Rot Resistance in Soybean E00003

Zhongnan Zhang, Jianjun Hao, Jiazheng Yuan, Qijian Song, David L. Hyten, Perry B. Cregan, Guorong Zhang, Cuihua Gu, Ming Li, Dechun Wang*

ABSTRACT

Phytophthora root rot (PRR) is a devastating disease in soybean [Glycine max (L.) Merr.] production. Michigan elite soybean E00003 is resistant to Phytophthora sojae and has been used as a resistance source in breeding. Genetic control of PRR resistance in this source is unknown. To facilitate marker-assisted selection (MAS), the PRR resistance loci in E00003 and their map locations need to be determined. In this study, a genetic mapping approach was used to identify major PRR-resistant loci in E00003. The mapping population consists of 240 F₄-derived lines developed by crossing E00003 with the P. sojae susceptible line PI 567543C. In 2009 and 2010, the mapping population was evaluated in the greenhouse for PRR resistance against P. sojae races 1, 4, and 7, using modified rice (Oryza sativa L.) grain inoculation method. The population was genotyped with seven simple sequence repeat (SSR) and three single nucleotide polymorphism (SNP) markers derived from bulk segregant analysis. The heritability of resistance in the population ranged from 83 to 94%. A major locus, contributing 50 to 76% of the phenotypic variation, was mapped within a 3 cM interval in the Rps1 region. The interval was further saturated with more BARCSOY SSRs and SNPs with TaqMan assays. Two SSRs and three SNPs within the Rps1k gene were highly associated with PRR resistance in the mapping population. The major resistance gene in E00003 is either allelic or tightly linked to Rps1k. The molecular markers located in the Rps1k gene can be used to improve MAS for PRR resistance.

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Phytophthora sojae infects soybeans at any growth stage primarily via the root system. High soil moisture favors disease development since P. sojae zoospores are only produced in saturated soil (Ho, 1969). The disease can only be partially managed using methods such as cultural practices and seed treatments with fungicides (Dorrance and Dennis, 2009; Schmitthenner, 1985). Integrated management strategies that combine host resistance with seed treatments were studied by Dorrance et al. (2009), who concluded that selecting resistant cultivars held the greatest utility.

On the basis of the results of six decades of research, both partial and race-specific resistances to P. sojae have been identified (Burnham et al., 2003a; Dorrance et al., 2004). Eight loci, with a total of 15 genes, have been reported as responsible for race-specific resistance (Anderson and Buzsell, 1992; Athow and Laviolette, 1982; Athow et al., 1980; Buzsell and Anderson, 1992; Demirbas et al., 2001; Diers et al., 1992; Gao and Bhattacharyya, 2008; Kilin et al., 1974; Mueller et al., 1978; Sun et al., 2011; Weng et al., 2001).

A high-yielding Michigan soybean line, E00003, was found to be resistant to P. sojae races 4 and 7 in the Uniform Soybean Tests in Northern States 2002 (Crochet, 2002). It has been intensively used as a source of resistance to P. sojae in the Michigan State University (MSU) soybean breeding program. There is insufficient evidence to trace the ancestor that contributed the PRR resistance to this line, thus it is unknown whether it carries new PRR resistance genes or new alleles at existing resistance loci. The objective of this study was to characterize PRR resistance in E00003 and to develop breeder-friendly markers to facilitate marker-assisted selection.

**MATERIALS AND METHODS**

Genetic mapping was used to identify PRR resistance loci in E00003. Phenotypic data were obtained from greenhouse trials, and genotypic data were obtained with simple sequence repeats (SSRs) and single nucleotide polymorphisms (SNPs). Resistance loci reported in the literature were first tested with SSRs in those regions.

**Mapping Population**

The genetic mapping population consists of 240 F₂-derived lines developed with the single seed descent method (Brim, 1966) from the cross E00003 × PI 567543C. E00003 is resistant to P. sojae, whereas PI 567543C is susceptible to P. sojae. The seeds of the mapping population were harvested in the fall of 2009 in the field of the Agronomy Farm at MSU located at East Lansing, MI.

**Evaluation for Phytophthora sojae Resistance**

Three P. sojae races, 1, 4, and 7, were used to evaluate the mapping population and its two parents in the MSU Plant Science Greenhouses in a total of six trials in 2009 and 2010. In each pathogen-inoculated trial, 12 seeds from each inbred line as one replicate, plus 36 seeds from each parent as three replicates, were planted in Baccato soil mix (Michigan Peat Company). The pathogen isolates were kindly provided by Dr. Anne Dorrance from the Department of Plant Pathology at Ohio State University. The greenhouse was maintained at 26°C day, 15°C night temperature, and sodium vapor lights were used to supplement light during the day (14 h).

The rice (Oryza sativa L.) grain inoculation method originally developed for evaluation of tree species for Phytophthora resistance (Homes and Benson, 1994) was used in this study. The rice grain inoculum was prepared in 250-ml flasks. In each 250-ml flask, 25 g of white long-grain rice and 20 mL distilled water were mixed thoroughly. The flask was covered with double-layered aluminum foil and autoclaved for 40 min twice in 24 h. After cooling, the rice grains in the flask were inoculated with three disks (5-mm diam.) of P. sojae mycelia obtained from the margin of an actively growing culture for each race. The flask was shaken daily to prevent the grains from compacting and ensure that all grains were uniformly colonized. After about 12 to 14 d of incubation, the rice grains were sampled and assayed for fungal colonization on V8 medium (Miller, 1955) 2 d before use as inoculum. As used by Holmes and Benson (1994), the rice inoculum was mixed well with sand, incorporated into a peat-vermiculite medium and sprinkled on the surface of a tray containing tree seeds. The method was modified by burying three P. sojae colonized rice grains together with one soybean seed in the Baccato soil mix, which provided the most appropriate disease pressure to maximize the difference of the PRR resistance levels of the resistant and susceptible parents of the mapping population in our preliminary tests.

Each soybean seed was buried in the soil with three P. sojae colonized rice grains at a depth of 2.5 cm in 12.5-cm-deep square pots (700051C, T.O. Plastics). The 12 seeds of each line were planted in two pots with six seeds in each pot. The pots were watered every other day for 14 d, and the numbers of live seedlings were counted. For the six trials, survival index (SI) of line j for trial i was calculated as follows: SI = [the number of plants of line j surviving in the trial i]/[12 × 100]. Survival index values of the parents, E00003 and PI 567543C, were calculated as SI = [(the number of plants surviving in the trial i)/36] × 100. The SI ranges from 0% for the most susceptible lines to 100% for the most resistant lines.

A separate germination test with non-pathogen-inoculated seeds was conducted in the greenhouse in 2010. Germination index (GI) of line j was calculated as follows: GI = (the number of plants of line j germinated/12) × 100. Adjusted survival index (ASI) of line j for trial i was defined as follows: ASI = [(the number of plants of line j surviving in the trial i)/12 × GI] × 100.

**Genetic Analysis**

Genotyping of the mapping population with SSR markers was performed as described by Zhang et al. (2010). All genomic regions that had been suggested in the literature were considered as potential PRR resistance loci (Table 1). Bulked segregant analysis (BSA), as proposed by Michelmore et al. (1991), was used to obtain possible genomic regions of major PRR resistance. For each inoculation trial, 15 resistant lines with the largest SI and 15 susceptible lines with the smallest SI were selected to form one resistant bulk pool and one susceptible bulk pool. A total of 132 SSR markers covering the genomic regions with reported PRR resistance loci were first selected to test the bulks. Genomic regions potentially associated with PRR resistance were saturated further with more SSR markers and additional BARCSOY SSR markers (Song et al., 2010). Then, a
subpopulation of 94 individual lines, as well as the two parents, was genotyped with polymorphic and trait-associated markers identified by BSA. The remaining lines of the entire mapping population were then genotyped with the markers that showed association with PRR resistance in the initial set of 94 lines. Ten DNA samples including the susceptible and resistant parents, two segregating bulks, and six resistant inbred lines from the mapping population were genotyped with the Illumina Infinium BeadChip (Illumina, Inc.) containing 52,041 soybean SNP markers (Song et al., 2013). Three polymorphic SNPs associated with PRR resistance among the 10 samples were selected as candidates for TaqMan SNP allele-specific genotyping assays for the population. The sequences were subjected to the Customer TaqMan Assay Design Tools of Applied Biosystems (ABI) to obtain allele-specific primers and probes, which were synthesized by ABI. The PCR reaction mixture for the TaqMan assay consisted of 20 ng of genomic DNA, 0.15 uL of 10X TaqMan Assay, and 1.5 uL of 2X ABI Genotyping Master mix containing a modified Taq DNA polymerase, reaction buffer, MgCl2, and dNTPs (ABI). After 10 min preincubation at 95°C, 45 PCR cycles were conducted with 10 sec denaturation at 95°C, 30 sec annealing at 60°C, and 10 sec extension at 72°C. A final melting cycle for nonspecific amplicon screening was performed by raising the temperature to 95°C for 10 sec, lowering the temperature to 40°C for 30 sec, then increasing the temperature to 85°C with continuous fluorescent acquisition followed by cooling to 40°C on the LightCycler 480. Data were analyzed by the Roche Applied Science software version 1.5.0.

### RESULTS AND DISCUSSION

#### Phenotype Distribution

*Phytophthora sojae* races 1, 4, and 7 were used to screen the mapping population for resistance to PRR. Survival index of the susceptible parent PI 567543C varied from 0.0 to 5.6%, while that of the resistant parent E00003 ranged from 41.7 to 91.7% (Table 2). E00003 showed significantly lower SI in trials inoculated with race 4 (44.4%) than with race 1 (91.7%) in 2009 at significant level $\alpha = 0.05$, which indicates that E00003 is less resistant to race 4 than to race 1. The SIs of the three replicates were very closely correlated (pairwise correlation coefficients = 0.79, 0.88, and 0.93; all $p$ values < 0.01). Therefore, one replication was sufficient for each trial. Two-sample $t$ test showed there was no significant difference between the 2 yr for races 1 and 7, indicating that year is not a confounding factor in the heritability calculation ($p$ value = 0.47 and 0.25, respectively, for races 1 and 7). A significant difference ($p$ value = 0.00065) was detected in race 4 between the 2 yr; therefore, the heritability was not estimated for race 4. In races 1 and 7, the broad sense heritability ranged from 83 to 94%. The correlation coefficients of race-specific SI between the 2 yr were all significant for the three *P. sojae* races, 0.89, 0.77, and 0.78, respectively. Continuous phenotypic distribution of SI with major peaks was observed for each trial, with SI ranging from 0 to 100% (Fig. 1). Among the entire mapping population, 90% of the lines had a GI above 87.5%. Germination index was applied as a phenotypic trait in QTL analysis, however no QTL with LOD heritability ($H^2$) (Fehr, 1987) of PRR resistance to different races was calculated in a fixed model as follows:

\[
H^2 = \frac{\sigma^2_g}{\sigma^2_g + \sigma^2_e / r},
\]

where $\sigma^2_g$ is the mean square for genotypes, $\sigma^2_e$ is the error mean square, and $r$ is the number of years. The 2 yr were treated as two replications, with $r = 2$. Correlation coefficients for SI with the same pathogen race in 2009 and 2010 were estimated. Two-sample $t$ test was conducted to compare the means of the 2 yr for each race. Tukey’s procedure was used for multiple line comparisons. A linkage map was constructed using JoinMap 3.0 with the Kosambi function and a logarithm of odds (LOD) score of 3 (Van Ooijen and Voorrips, 2001). Composite interval mapping (CIM) was performed using QTL Cartographer V2.5 (Wang et al., 2008) to locate PRR resistance loci with LOD threshold determined by 1000 permutations (significant level $\alpha = 0.05$). The plots of LOD scores and the locus positions were generated using MapChart (Voorrips, 2002). Germination index and ∆SI for the six trials were calculated and analyzed as separate phenotypic traits in quantitative trait loci (QTL) analysis using CIM method in QTL Cartographer V2.5. Chi-square test was used to determine if the observed ratio of the genotypic classes fitted the expected ratio.
The resistant and susceptible bulks were first genotyped using SSR markers in close proximity to previously identified PRR resistance loci in E00003. Among these markers, BARCSOYSSR_03_0249 and BARCSOYSSR_03_0250 mapping population for these 200 SNPs, 76 SNPs were shown. The marker order followed the same order as the consensus map and the latest map integrated with BARCSONY SSRs (Song et al., 2004; Song et al., 2010). The interval containing the resistant locus was less than 2 cM between marker BARCSOYSSR_03_0250 and Satt675. The LOD score was estimated at 28.9 to 53.3. The $R^2$ ranged from 0.46 to 0.67, with additive effect from 18.8 to 33.4%. Since the $R^2$ only partially explained the total genetic variance as estimated by heritability, we investigated whether other PRR resistance loci existed. A total of 1328 SSRs evenly spread through the entire genome were screened with the DNA bulks, and only 15 SSRs were found to distinguish the bulks. However, none showed association with PRR resistance in the entire mapping population. Therefore, no other major PRR resistance loci were detected in E00003.

**Phytophthora Root Rot Resistance Locus Mapping**

The resistant and susceptible bulks were first genotyped with markers in close proximity to previously identified Rps resistance loci (Table 1) to investigate whether the Rps loci in E00003 were among those reported in the literature. From the BSA, a cluster of SSR markers on Chromosome 3 (linkage group N) were polymorphic between the PRR resistant and susceptible bulks. The polymorphic SSR markers were further tested with 94 lines from the mapping population. Simple sequence repeat markers Satt631, Satt675, Satt485, Satt584, and Satt624 were associated with the phenotypic data and were confirmed by genotyping the remainder of the population. The five SSR markers formed one linkage group in the linkage analysis with JoinMap (Van Ooijen and Voorrips, 2001), and a major QTL was located in a 20-cM interval between Satt631 and Satt675 when the data were analyzed using the CIM method in WinQTLCart 2.5 (Wang et al., 2008) (data not shown). The interval was in the Rps1 region (Gardner et al., 2001). Though Rps1 is close to Rps7, Rps7 was eliminated because it is susceptible to races 1, 4, 7, and 25 (Dorrance et al., 2004). Within the region flanked by Satt631 and Satt675, there is another recently identified Rps locus, RpsYu25 (Sun et al., 2011). The authors claimed that RpsYu25 is a novel locus, on the basis of different responses to a set of pathogen isolates, and developed a flow chart as a dichotomous key to differentiate Rps loci. The set of pathogens used in that study was not comparable with pathogen isolates we used; thus, it was not possible to eliminate RpsYu25 as a candidate.
The difference between the observed and the expected ratio in the F4 generation (Table 3), indicating there is no segregation distortion in the mapping population.

A new linkage map was constructed by adding the three SNPs (Fig. 2), and QTL analysis was performed using this new map. The QTL positions on the new map (BARC_1.01_Gm03_4563499_G_A), and MSUSNP03-3 (BARC_1.01_Gm03_4610670_C_T) (Supplemental Table).

Figure 1. Phenotypic distribution of survival index (Si) of the mapping population among 240 F4–derived lines from PI 567543C × E00003. Parental SIs are indicated by arrows. Survival index was calculated as: $Si_{ij} = \frac{\text{the number of plants surviving of line } j \text{ in the inoculated trial } i}{12} \times 100$.

Table 3. Segregation ratio of different genotypes of the three single nucleotide polymorphism (SNP) markers in the F4–derived mapping population E00003 × PI 567543C. The three SNP markers are MSUSNP03-1 (BARC_1.01_Gm03_4487138_A_C), MSUSNP03-2 (BARC_1.01_Gm03_4563499_G_A), and MSUSNP03-3 (BARC_1.01_Gm03_4610670_C_T).

<table>
<thead>
<tr>
<th>Molecular markers</th>
<th>Homozygous of resistance allele of E00003</th>
<th>Heterozygous</th>
<th>Homozygous of susceptible allele of PI 567543C</th>
<th>Expected ratio in F4 generation</th>
<th>Chi-square statistics</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MSUSNP03-1‡</td>
<td>108</td>
<td>26</td>
<td>100</td>
<td>7:2:7</td>
<td>0.73</td>
<td>0.39</td>
</tr>
<tr>
<td>MSUSNP03-2</td>
<td>107</td>
<td>26</td>
<td>103</td>
<td>7:2:7</td>
<td>0.55</td>
<td>0.46</td>
</tr>
<tr>
<td>MSUSNP03-3</td>
<td>105</td>
<td>24</td>
<td>101</td>
<td>7:2:7</td>
<td>0.98</td>
<td>0.32</td>
</tr>
</tbody>
</table>

A total of 240 F4–derived lines of the mapping population E00003 × PI 567543C were genotyped; individual with missing value in genotyping was not shown in this table.

Allele-specific SNP markers designed from the SoySNP50K BeadChip (Song et al., 2013).
are shown in Fig. 2, and LOD scores and $R^2$ are given in Table 4. The LOD scores were estimated to be between 32.3 and 62.6. The $R^2$ ranged from 0.50 to 0.76, with additive effects from 19.2 to 33.0%. These results indicated that about 50 to 76% of the phenotypic variation can be explained by the resistance locus in E00003. The remaining phenotypic variation was due to some other minor effect QTL or experimental errors. Though Phytophthora resistance investigated here is encoded by a major gene, the resistance gene was successfully identified by the QTL mapping method in this study. Quantitative trait loci mapping methods have also proven successful in identifying major genes in other studies (Zhang et al., 2010, 2013).

The positions of LOD peaks in Table 4 were estimated at 24.4 to 26.3 cM on the basis of the position of markers Satt631 and Satt675 from soybean consensus map (Song et al., 2004). Considering physical positions, the QTL region is located within interval 4,475,877 to 4,563,799 bp (Fig. 2), within the $Rps1k$ interval, 4,457,810 to 4,641,921 bp (Gao and Bhattacharyya, 2008). $RpsYu25$ is located between 3,338,620 and 3,465,436 bp by converting the flanking markers Satt152 and Sat_186 from interval 30.1 to 32.8 cM (Song et al., 2010; Sun et al., 2011).

To validate the gene action of the resistance allele at $Rps1k$ in E00003, SI for each genotype of the three polymorphic allele-specific SNP markers was estimated using the combined SI of 2 yr of data for the same race (Table 5). Strong evidence of additive gene action was detected, since the average SI for the heterozygous type was significantly smaller than that for the homozygous resistant type, significantly larger than that for the homozygous susceptible type, and not significantly different from the average of the two homozygous types at the significant level of $\alpha = 0.05$.

In spite of advances in quantitative molecular genetics, owing to genomics, computation, and statistics, the bottleneck in genetic analysis is now phenotyping rather than genotyping (Walsh, 2009). The detection of $Rps1$ locus in the Michigan elite line E00003 has shown that the rice grain inoculation method is an effective high-throughput phenotyping approach to detect $Rps$ loci with major effects. The method provides the opportunity for the soil-borne pathogen to interact with its host, mimicking natural infection of soybean, and to avoid injury to epidermal tissues, which occurs in the hypocotyl splitting method. The rice grain method saves labor and time of splitting soybean seedlings. Within 14 d, thousands of rice grains can be prepared in a single batch as pathogen inoculum, and they are easier to handle. Compared with the vermiculite plate test (Thomson et al., 1991), growing the pathogen on one layer of agar in a petri dish for each single pot is unnecessary. However, to detect partial resistance to PRR, lesion length measurement using a slant board test (Burnham et al., 2003a) has proven effective (Tucker et al., 2010). Although there might be some confounding effect with germination rate or soil factors, the validity of our findings was not affected.

Phytophthora resistance in E00003 maps to the $Rps1k$ locus. To determine whether it is $Rps1k$, additional crosses need to be made between E00003 and the original $Rps1k$ parent—Williams 82 or Kingwa (Dorrance et al., 2004, Kasuga et al., 1997). The resistance gene in E00003 could be a new allele at the $Rps1$ locus or a new gene tightly linked to $Rps1$. Most importantly, two SSRs and three SNP allele-specific endpoint genotyping markers, MSUSNP03-1 (BARC_1.01_Gm03_4487138_A; C), MSUSNP03-2 (BARC_1.01_Gm03_4563499_G; A), and MSUSNP03-3 (BARC_1.01_Gm03_4610670_C; T), were identified and developed for the resistant locus in line E00003. The SNP markers are also polymorphic among commercial varieties entered in Michigan Soybean Performance Trials and significantly associated with the $Rps1$ reported by companies that entered the varieties to the trials (data not shown). With these breeder-friendly SSR and SNP markers, marker-assisted selection can be performed efficiently and effectively by using E00003 or other soybean lines containing the E00003 resistance locus as the source for PRR resistance.

**Acknowledgments**

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Table 4. Phytophthora root rot resistance locus detected in the mapping population E00003 x PI 567543C using the composite interval mapping (CIM) method. Chromosome 3 (Linkage Group N) is based on SoyBase (Grant et al., 2010).

<table>
<thead>
<tr>
<th>Trial</th>
<th>Year</th>
<th>Phytophthora sojae race</th>
<th>Peak position†</th>
<th>Marker near the peak</th>
<th>LOD (threshold)‡</th>
<th>R²</th>
<th>Additive effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2009</td>
<td>1</td>
<td>24.4</td>
<td>BARCROSSYSSR_03_0250</td>
<td>62.6(3.7)</td>
<td>0.76</td>
<td>31.6</td>
</tr>
<tr>
<td>2</td>
<td>4</td>
<td>26.3</td>
<td>MSUSNP03-1 (BARC_1.01_Gm03_4487138_A_C)</td>
<td>32.3(2.5)</td>
<td>0.50</td>
<td>19.2</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>7</td>
<td>24.4</td>
<td>BARCROSSYSSR_03_0250</td>
<td>32.1(2.4)</td>
<td>0.59</td>
<td>26.8</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>2010</td>
<td>1</td>
<td>25.4</td>
<td>BARCROSSYSSR_03_0250</td>
<td>56.01(2.8)</td>
<td>0.69</td>
<td>33.0</td>
</tr>
<tr>
<td>5</td>
<td>4</td>
<td>24.4</td>
<td>BARCROSSYSSR_03_0250</td>
<td>36.6(3.2)</td>
<td>0.56</td>
<td>24.8</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>7</td>
<td>26.2</td>
<td>MSUSNP03-1 (BARC_1.01_Gm03_4487138_A_C)</td>
<td>58.9(3.6)</td>
<td>0.70</td>
<td>32.9</td>
<td></td>
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</tbody>
</table>

†Peak position converted according to consensus map (Song et al., 2004).
‡Logarithm of odds (LOD) score threshold for each trial determined by 1000 permutations in cIM by WinQTLCart (Wang et al., 2008).
§Single nucleotide polymorphism markers from Song et al. (2013).

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