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RESEARCH

Identification of Soybean Genotypes Resistant to *Fusarium graminearum* and Genetic Mapping of Resistance Quantitative Trait Loci in the Cultivar Conrad

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ABSTRACT

Fusarium graminearum, a necrotroph, has emerged as an important soybean [*Glycine max* (L.) Merr.] seedling pathogen in Ohio. An effective management strategy for seedling pathogens is host resistance; thus, the objectives of this research were to identify and characterize sources of resistance to *F. graminearum*. Twenty-four soybean genotypes were screened for resistance using a rolled-towel assay; seeds were inoculated with 2.5×10^4 macroconidia mL^{-1}. A disease severity index (DSI) was calculated on the basis of the ratio of lesion length to total plant length at 7 days after inoculation. Five genotypes had high levels of resistance to *F. graminearum*, with DSIs ranging from 24 to 41.5%. These included the cultivar Conrad, which has high levels of partial resistance to the hemibiotroph, *Phytophthora sojae*. A population of 262 $F_{6:8}$ recombinant inbred lines derived from a cross of Conrad × Sloan (susceptible) was then evaluated for resistance. The same rolled towel method was used, and resistance to *F. graminearum* segregated as a quantitative trait. The DSI ranged from 22 to 100% and the broad-sense heritability estimate was 0.72. Four putative quantitative trait loci (QTLs) were identified from Conrad on chromosomes 8, 13, 15, and 16, and one putative QTL from Sloan mapped to chromosome 19. The putative QTLs identified in this population were not the same as those that confer resistance to *P. sojae*, which suggests that different loci are required for resistance to these two different types of seedling pathogens.

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Abbreviations: BLUP, best linear unbiased predictor; CIM, composite interval mapping; cM, centimorgan; DAI, days after inoculation; DSI, disease severity index; IM, interval mapping; LOD, logarithm of odds; LSD, least significant difference; MLG, molecular linkage group; QTL, quantitative trait locus; PAMSA, PCR amplification of multiple specific alleles; PCR, polymerase chain reaction; R, resistant parent; RIL, recombinant inbred line; S, susceptible parent; SNP, single nucleotide polymorphism; SSR, simple sequence repeat.

Soil-borne seed and seedling pathogens prevent emergence and result in reductions in plant stand. In Ohio, this often results in replanting of a given field repeatedly during the spring. Over the past decade, the occurrence of replanting as a result of seedling diseases of soybean has increased in Ohio (Broders et al., 2007a, 2007b). Several factors may have contributed to this increase in seedling disease incidence and severity including: earlier planting dates that result in seeds planted under cooler and wetter environmental conditions that delayed germination; consecutive years of above-average rainfall during April and May; a high proportion of crop production land in long-term no-till soil conservation systems; shifts in seed treatment fungicide chemistries; and changes in the base soybean germplasm (Broders et al., 2007a, 2007b; Fernandez and Fernandes, 1990; Sneller, 2003; Workneh et al., 1999; Wrather et al., 2001, 2003; Wrather and Koenning, 2006, 2009). As a result of these changes

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in environment and production practices, one of the soybean seedling pathogens that emerged as a problem in the last decade is the necrotroph *Fusarium graminearum* (teleomorph: *Gibberella zeae*) (Broders et al., 2007b; Ellis et al., 2011; Xue et al., 2006, 2007). Symptoms on soybean caused by *F. graminearum* typically include damping-off with water-soaked lesions at the site of infection, associated with a pink to red-brown discoloration. Broders et al. (2007b) recovered 112 *F. graminearum* isolates from soybean and corn (*Zea mays* L.) seedlings collected from over 30 locations in Ohio where stand establishment was a problem. They reported that these isolates were moderately pathogenic to soybean in greenhouse pot assays, and 105 were highly pathogenic to seed in petri plate assays. Ellis et al. (2011) subsequently determined that high levels of inoculum of this pathogen were needed for disease development. Thus, no-till and reduced tillage practices may contribute to the increase in *F. graminearum* disease incidence because these leave large quantities of crop residue on the soil surface and provide favorable soil conditions for both pathogen growth and survival (Baird et al., 1997; Fernandez and Fernandes, 1990; Cotton and Munkvold, 1998; Leslie et al., 1990; Sutton, 1982; Windels et al., 1988; Workneh et al., 1999).

Current management strategies for seedling diseases, such as those caused by *F. graminearum*, rely heavily on seed treatment with fungicides and improvement of soil drainage through tillage and tiling (Ellis et al., 2011; Grau et al., 2004; Nelson, 1999). However, many of the broad-spectrum seed treatments, such as Rival {19.8% captan [*N*-(trichloromethylthio)cyclohex-4-ene-1,2-dicarboximide], 8.4% pentachloronitrobenzene, 8.4% terrazole [1,2,4-thiadiazole,5-ethoxy-3-(trichloromethyl)], and 1.0% thiabendazole [2-(thiazol-4-yl)benzimidazole]; Gustafson)} and Captan (37.4% captan; Bayer Crop Science) are no longer available, and a shift to chemistries that have narrower pathogen efficacy profiles has occurred. In two previous studies, the efficacy of fungicide seed treatments for *F. graminearum* was evaluated (Broders et al., 2007b; Ellis et al., 2011). Fludioxonil [4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile; Maxim 4S, Syngenta Crop Protection Inc.)] and captan provided higher levels of control than the newer class of strobilurin fungicides (Broders et al., 2007b; Ellis et al., 2011). Although fludioxonil was effective both in amended agar plate assays (Broders et al., 2007b) and as a fungicide seed treatment (Ellis et al., 2011), mutants insensitive to fludioxonil were readily generated in the amended agar plate assays (Broders et al., 2007b). While no such mutants have been recovered from the field, there are additional production costs associated with planting treated seed or intensifying tillage and tiling. Thus, more effective and less costly disease management strategies such as disease resistance are a priority for the long-term management of *F. graminearum*.

Disease resistance is a promising strategy for management of *F. graminearum*. Recent shifts in soybean germplasm have occurred as industry now dominates in the development and delivery of new cultivars compared with the public sector (Diers and Kim, 2008). As such, there is limited diversity among elite lines from some companies, due in part to the limitation of the exchange of germplasm (Sneller, 2003). The emergence of *F. graminearum* as a pathogen of soybean may also be linked to these shifts in germplasm. The first hypothesis of this study is that resistance to *F. graminearum* is present in adapted soybean cultivars that were widely grown before the recent emergence of this seedling pathogen.

There are three general types of plant resistance to pathogens: (i) innate immunity or basal resistance which is nonspecific recognition of broadly conserved pathogen features such as flagellin from bacteria or chitin from fungal cell walls (Boller and Felix, 2009; Boller and He, 2009; Chisholm et al., 2006; Jones and Dangl, 2006; Medzhitov and Janeway, 1997); (ii) qualitative (complete, vertical, major-gene, or narrow-spectrum) resistance, which is usually conditioned by a single gene and is involved in specific recognition of pathogen effectors or their targets (Chisholm et al., 2006; Flor, 1955, 1971; Jones and Dangl, 2006; van der Plank, 1968); and (iii) quantitative (partial, horizontal, minor-gene, or broad-spectrum) resistance, which is conditioned by multiple genes with partial effect (Poland et al., 2009; van der Plank, 1968; Young, 1996). Based on current knowledge, the type of resistance that is observed often depends on the biology of the pathogen (Glazebrook 2005; Hammond-Kosack and Parker, 2003; Oliver and Ipcho, 2004; Poland et al., 2009). Necrotrophic pathogens tend to have wide host ranges and rapidly kill their host, usually through the secretion of toxins. In contrast, biotrophic pathogens have a more intimate association with their host, require living host cells, are host specific, and often establish themselves by forming specialized structures such as appresoria and haustoria. A number of plant pathogens have lifestyles that are intermediate, and these are referred to as hemibiotrophs. They establish infection in a similar manner to biotrophs, but later in the infection process they begin to actively spread through the host and kill host cells (Agrios 2005; Erwin and Ribeiro, 1996; Qutob et al., 2002). In some host pathosystems, qualitative resistance is often targeted toward host-specific pathogens, which are host specific (hemibiotrophs or biotrophs). For many interactions, qualitative resistance is often less durable than quantitative resistance because pathogens can more easily adapt to single gene–mediated resistance (Poland et al., 2009; St. Clair, 2010). In contrast, quantitative resistance usually provides broad-spectrum protection toward all isolates or strains of a pathogen regardless of biology (biotrophic or necrotrophic), with only a few cases of qualitative resistance reported for necrotrophs (Oliver and Ipcho, 2004).

Given that *F. graminearum* was only recently reported as a seedling pathogen to soybean, no studies have been reported to identify or characterize resistance to this pathogen in this host. In Ohio and other Midwest states, *F. graminearum* causes Fusarium head blight of wheat (*Triticum aestivum* L.) and barley (*Hordeum vulgare* L.) (Bai and Shaner, 1994; McMullen et al., 1997), and ear and stalk rot of corn (Sutton, 1982). In these and other hosts, resistance to *F. graminearum* is quantitative (Anderson et al., 2001; Bai et al., 1999; Buerstmayr et al., 2002; Gervais et al., 2003; Waldron et al., 1999; Zhou et al., 2002) and can also be greatly influenced by the environment (Bai and Shaner, 1994, 1996, 2004; Buerstmayr et al., 2011; Snijders and Van Eeuwijk, 1991). Quantitative resistance in soybean has been extensively studied for both hemibiotrophic and necrotrophic root pathogens, including *Phytophthora sojae* (Burnham et al., 2002, 2003; Dorrance and Schmitthenner, 2000; Han et al., 2008; Tooley and Grau, 1982; Tucker et al., 2010; Walker and Schmitthenner, 1984; Wang et al., 2010; Weng et al., 2007); *Fusarium virguliforme* (telomorph belonging to *Nectria sensu lato*) (Chang et al., 1996; Neto et al., 2007; Hnetkovsky et al., 1996; Iqbal et al., 2001; Meksem et al., 1999; Njiti et al., 1996, 2002); *Macrophomina phaseolina* (Mengistu et al., 2007; Paris et al., 2006; Smith and Carvil, 1997); and *Rhizoctonia solani* (teleomorph *Thanatephorus cucumeris*) (Bradley et al., 2005; Zhao et al., 2005).

Several soybean populations that are segregating for resistance to *P. sojae* are available (Gordon et al., 2007). If at least one of these same populations segregated for resistance to *F. graminearum*, this would allow for comparison of resistance loci to both hemibiotrophic and necrotrophic soybean root pathogens. As such, the second hypothesis for this study was that resistance to *F. graminearum* is inherited quantitatively and is conferred by different loci compared to hemibiotrophic root pathogens. Therefore, the objectives of this study were (i) to identify sources of resistance to *F. graminearum* and (ii) to map resistance to *F. graminearum* in a recombinant inbred line (RIL) population that was also segregating for resistance to *P. sojae*.

MATERIALS AND METHODS

Phenotypic Assay

Twenty-four soybean genotypes (Table 1) were chosen for an evaluation of resistance toward *F. graminearum* using two criteria: (i) possession of known resistance genes or partial resistance to other soybean pathogens and (ii) prior use as parents for the development of existing genetic mapping populations. A rolled towel assay as described previously by Ellis et al. (2011) was used. *Fusarium graminearum* isolate Fay11 was selected from an earlier survey (Broders et al., 2007b) in which there was very little variability among the 105 isolates evaluated for pathogenicity on soybean seed. For the assay, 20 seeds of each genotype were placed in a row on a moistened towel and each seed was inoculated with a 100 μL suspension of 2.5×10^4 macroconidia mL^{-1}. Another moistened towel was placed over the inoculated seed and the towels were rolled and placed in 25-L buckets, with 24 towels per bucket. There were three buckets, with one towel for each genotype in each bucket. A black plastic bag was placed over each bucket, and the buckets were placed in a growth chamber at 22°C. The experimental design was a randomized complete block, with three replicates. Because each of the three buckets provided a physical separation between the replicates of the genotype and that the microenvironment was homogeneous within buckets but heterogeneous among buckets, we considered each bucket to be a block and random effect in the analysis. The experiment was conducted twice. Seven days after inoculation (DAI), total lesion length and plant length were recorded for each seedling. A disease severity index (DSI) was calculated by dividing the lesion length by the total length of the seedling and multiplying by 100.

Because the data were converted into percentages using the DSI calculation and the range in percentages exceeded 40%, the data were arcsine-transformed as suggested by Little and Hills (1978) and were then analyzed using the general linear model procedure (PROC GLM) of SAS 9.2 (SAS Institute Inc.). Levene's test of homogeneity of variance was used to compare experiments. Because there was no significant difference ($P = 0.16$) between experiments, the data were pooled and analyzed together. Means were compared using Fisher's protected least significant difference (LSD) at $P = 0.05$.

Table 1. Mean disease severity index following inoculation of soybean seed with 100 μL of 2.5×10^4 macroconidia mL^{-1} of *Fusarium graminearum* in a rolled-towel assay.

Genotype	Mean % seedlings affected†
PI 399073	94.3a
Sloan	89.6ab
Archer	87.8abc
Resnik	86.2bc
Ripley	85.6bc
Strong	82.8bcd
Williams	81.8bcd
Kottman	81.7bcd
Williams 82	81.8cd
Ohio-FG5	80.9cd
OX-208	78.2cd
L85-2325	77.8cde
Dennison	74.4de
L83-570	74.3de
Jack	73.4de
Stressland	72.8def
Ohio-FG1	65.9ef
Flint	65.2ef
OHS 303	60.5f
Prohio	41.5g
Conrad	35.0hg
PI 408211B	33.5hg
HC99-2846	31.7hg
PI 424354	24.0h
Mean	69.2

†Mean disease severity represents the lesion length divided by the total length of the seedling multiplied by 100. Disease severity data were arcsine transformed; the actual means are reported in the table. The experimental design was randomized complete block, with three replications. The experiment was repeated for a total of two times. Values followed by the same letter were not significantly different according to Fisher's protected least significant difference ($P < 0.05$) based on the arcsine-transformed data.

Plant Material for Quantitative Trait Locus (QTL) Mapping

A population of 262 $F_{6:8}$ RILs derived from a cross between the partially resistant (R) cultivar Conrad (Fehr et al., 1989) and the susceptible (S) cultivar Sloan (Bahrenfus and Fehr, 1980) was evaluated for partial resistance to *F. graminearum*. The population was developed from eight F_1 plants, and each generation was advanced through single seed descent. This population was previously used to map QTLs associated with resistance to *P. sojae* (Wang, 2011).

Phenotypic Assay for QTL Mapping

Twenty seeds from each of the 262 RILs were evaluated for resistance using the rolled towel method, as already described. The experimental design was an augmented randomized incomplete block, with R and S parents included as checks in each bucket that contained 21 to 22 RILs. The 262 RILs were separated into three groups of approximately 87 RILs and were evaluated over three separate time points, with a period of 24 h between each inoculation. At 7 DAI the DSI data were taken as previously described. This was repeated for a total of two experiments.

The DSI data were analyzed using a mixed model to obtain the best linear unbiased predictor (BLUP) (Stroup, 1989). The BLUP equation was

$$Y_{ijklm} = \mu + R_i + I(R)_{ij} + K(IR)_{ijk} + C_l + G(C)_{lm} + \varepsilon_{ijklm} \quad [1]$$

where Y_{ijklm} = observation of *i*th experiment, *j*th incomplete block (time), *k*th bucket, *l*th class, and *m*th genotype; μ = overall mean; R_i = effect of the *i*th experiment; $I(R)_{ij}$ = effect of the *j*th incomplete block (time) in the *i*th experiment; $K(IR)_{ijk}$ = effect of the *k*th bucket in the *j*th incomplete block (time) of the *i*th experiment; C_l = effect of the *l*th class (Conrad, Sloan, and RILs); $G(C)_{lm}$ = effect of *m*th genotype within *l*th class for recombinant lines only (genotypic variance); and ε_{ijklm} refers to sampling variation from plant to plant within an experimental unit. One class was assigned for each parental line and another class for all of the RILs. Class of entry was assumed to be a fixed effect, and all other terms were random. This model permitted an analysis in which checks (parents) were fixed effects and RILs were random. Variance components were estimated using restricted maximum likelihood (Burnham et al., 2003; Tucker et al., 2010; Wang et al., 2010). Heritability was calculated on a family mean basis as $\sigma^2_{G(C)}/[\sigma^2_{G(C)} + \sigma^2_{\varepsilon}/2]$.

Molecular Mapping

To develop the initial genetic map for this study, a subset of 147 RILs from the full population of 262 RILs were genotyped with 128 polymorphic single nucleotide polymorphisms (SNPs) and 65 simple sequence repeat (SSR) markers as described in Wang (2011) for identifying resistance to *P. sojae*. SNP markers were assayed by the VeraCode GoldenGate Genotyping Kit (Illumina Inc.) and analyzed using the Illumina BeadXpress Reader (Illumina Inc.); SSR markers were assayed as described in following text. An initial map consisting of these 193 markers was developed using JoinMap 4.0 with the Kosambi function (van Ooijen, 2006).

To further define the putative QTLs identified for resistance to *F. graminearum* with the initial map, a subset of the aforementioned 193 markers, consisting of 57 SSR and 26 SNP markers, were assayed on the full population of 262 RILs. This subset of markers was localized to regions of putative QTL or associated with resistance to *Fusarium* spp. An additional 15 SSR markers were assayed on the full population in regions of putative QTL for resistance to *F. graminearum* and also to ensure thorough coverage of regions in the genome that were associated with resistance to *F. virguliforme* and *S. sclerotiorum*. The 26 SNP markers assayed on the full population were converted to "polymerase chain reaction (PCR) amplification of multiple specific alleles" (PAMSA) markers designed using the protocol from Gaudet et al. (2007) (Supplemental Table 1). SSR and PAMSA markers were amplified by polymerase chain reaction (PCR) using a modified protocol from Gordon et al. (2007). A 12.5-μL PCR mix containing 50 ng of genomic DNA, 1× PCR buffer (Promega), 130 μM of each dNTP, 2 mM $MgCl_2$ (Promega), 0.4 μM forward and reverse primers, as well as 0.5 to 1.0 U of *Taq* DNA polymerase (Promega), for the SSR and PAMSA markers, respectively. A touchdown program was used in which the annealing temperature decreased from either 60°C to °50 or 58°C to 48°C by 1°C each cycle for the first 10 cycles, and the annealing temperature was then kept at 50°C or 48°C for the remaining 28 cycles. PCR products were analyzed by electrophoresis on 4% 3:1 HRB agarose (AMERSCO Inc.) containing 0.7% GelRed (Phenix Research Products) for 120 to 180 min at 150 V in the RapidRun Agarose Buffer (Affymetrix/USB Inc.). The products were sized with TriDye 100 bp DNA ladder (UVP Inc.). The gels were photographed using the Kodak Electrophoresis Documentation and Analysis System 290 (EDAS, 290: Kodak Company) and the product bands were scored. The targeted region map generated with the 98 molecular markers assayed on the full population of 262 RILs was created using JoinMap 4.0 and the Kosambi mapping function (van Ooijen, 2006).

QTL Analysis

A preliminary analysis using interval mapping (IM) was performed using MAPQTL to identify putative QTLs from the initial map of 193 molecular markers assayed on the subset of 147 RILs. The walking speed was 1.0 centimorgan (cM) for QTL analysis (van Ooijen, 2004). Permutation tests with 1000 iterations were performed on each linkage group and on the whole genome to estimate significant logarithm of odds (LOD) scores (Churchill and Doerge, 1994).

A second round of IM and composite interval mapping (CIM) were performed using MAPQTL to further delimit QTL using the targeted region map developed from 98 markers assayed on the full population. The walk-speed and permutations were as described above. One-way ANOVA with the PROC GLM procedure (SAS Institute Inc.) was used to confirm significant associations between marker genotypes and phenotypic variation. QTL Network 2.0 (Yang et al., 2008), based on a linear mixed model (Wang et al., 1999), was used to confirm the main effect QTL identified using MAPQTL and to detect if interactions between QTL were significant. A window size and filtration size of 10, with a 1-cM walking speed

was used in the analysis. Permutation tests with 1000 iterations were performed on each linkage group with a Gibbs sample size of 20,000. The significance level threshold was 0.05. Gibbs sampling as defined by Wang et al. (1994) is a Monte Carlo algorithm that generates a sequence of random samples from the joint distributions through sampling from and updating conditional distribution.

RESULTS AND DISCUSSION

Sources of Resistance

There was a wide range of responses among the 24 soybean genotypes following inoculation with *F. graminearum* in the initial germplasm screening experiment (Table 1). The differences in the arcsine-transformed DSI data for the 24 soybean genotypes were highly significant ($P < 0.0001$). The genotypes with the highest levels of resistance and lowest disease severities, 24.0 to 41.5%, included plant introductions (PIs) 424354 and 408211B, the breeding line HC99-2846 (Mian, 2006), and the cultivars Conrad and Prohio (Mian et al., 2008) (Table 1, Fig. 1). Soybean genotypes with the highest disease severities, 81.7 to 94.3%, included PI 399073 and the cultivars Sloan, Archer (Cianzio et al., 1991), Resnik (McBlain et al., 1990), and Ripley (Cooper et al., 1990) (Table 1, Fig. 1). None of the genotypes that expressed high levels of resistance to *F. graminearum* in this study exhibited an immune response because all developed minor lesions following inoculation.

The results for this select group of 24 genotypes suggest that resistance to *F. graminearum* may be common in soybean. Similarly, moderate levels of resistance to *M. phaseolina* were also found among 24 commercial cultivars in one study (Smith and Carvil 1997) and among 18 breeding lines and six cultivars in another (Mengistu et al., 2007). If resistance to pathogens such as *F. graminearum* and *M. phaseolina* is common in soybean genotypes, it may not be necessary for breeders to focus initial efforts on unadapted germplasm when developing new cultivars. Instead, efforts could be focused on screening advanced breeding lines for resistance. Since resistance to *F. graminearum* was readily identified within a very limited sampling, this may indicate that shifts and changes in the base soybean germplasm may have contributed to the emergence of this organism as a seed and seedling pathogen of soybean in Ohio. An evaluation of representative cultivars that were grown before 2000, compared to those that are available today, would be necessary to confirm this hypothesis.

Figure 1. Symptoms of *Fusarium graminearum* infection 7 days after inoculation (DAI) on plant introduction (PI) 424354 and Conrad (high levels of resistance) compared with Williams and Sloan (lower levels of resistance). Seed was inoculated with 2.4 × 10^4 macroconidia mL^{-1} in a rolled towel assay.

QTL for Resistance

The cultivar Conrad was resistant to *F. graminearum* in the germplasm screening and Sloan was susceptible, so putative QTL for *F. graminearum* resistance were mapped in a Conrad × Sloan $F_{6:8}$ RIL population. BLUP values for the 262 RILs of the Conrad × Sloan $F_{6:8}$ population had a normal distribution, indicating that the resistance was quantitatively inherited (Fig. 2). The DSI BLUP values ranged from −30.71 to 21.20 (DSI 22.2% to 100.0%). The broad-sense heritability estimated from the DSI ratings for this population was 0.72. In this study, the R and S checks were consistent among blocks (buckets) and across experiments.

The final linkage map constructed from the 208 molecular markers covered 961.1 cM across 34 fractional molecular linkage group (MLGs; unlinked markers were not included). Marker order in the constructed map was mostly in accord with the Soybean Consensus Linkage Map 4.0 (Hyten et al., 2008), and the constructed map covered approximately 43% of the consensus map. Sixteen of the 20 chromosomes had good coverage with approximately 1 to 20 cM distances between markers, and there were only a few regions with more than 20 cM between markers. The four chromosomes with poor

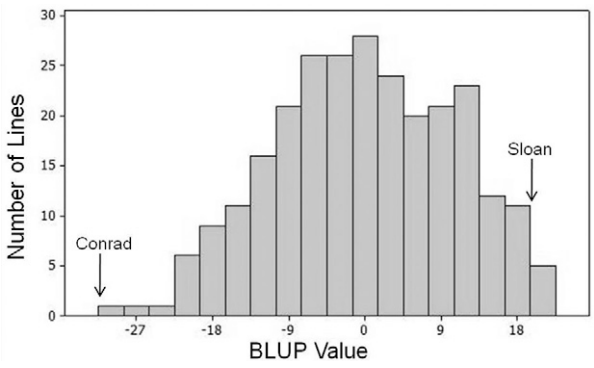

Figure 2. Distribution of best linear unbiased predictor (BLUP) values for the disease severity index (DSI) among $F_{6:8}$ recombinant inbred lines derived from a cross of Conrad (resistant) × Sloan (susceptible) at 7 days after inoculation with a 2.5 × 10^4 macroconidia mL^{-1} of *Fusarium graminearum*.

coverage included chromosomes 4 (MLG C1), 6 (MLG C2), 11 (MLG B1), and particularly chromosome 20 (MLG I), for which only two polymorphic SSR markers were identified. One of these two markers was Sat_268, which is in a region previously reported to be associated with resistance to *F. virguliforme* (Iqbal et al., 2001).

Four putative QTL from the cultivar Conrad were identified through both IM and CIM using MAPQTL 5.0 (van Ooijen 2004) on chromosomes 8 (MLG A2), 13 (MLG F), 15 (MLG E), and 16 (MLG J), and one putative QTL from the cultivar Sloan was identified through IM and CIM on chromosome 19 (MLG L) (Table 2, Fig. 3). The LOD scores ranged from 2.2 to 3.4 for CIM, which was significant based on the empirically determined threshold for $P = 0.05$ (Churchill and Doerge, 1994) (Table 2). The total phenotypic variation explained by the putative QTL detected with IM and CIM was 30 and 24%, respectively, with each individual QTL contributing approximately 3 to 11% (Table 2). QTLNetwork 2.0 identified the same four QTLs as CIM, with the QTL on chromosome 19 falling just below the 0.05 false discovery rate (data not shown). No interactions among the QTLs were detected (data not shown).

Table 2. Quantitative trait loci for partial resistance to *Fusarium graminearum* that were identified via interval mapping (IM) and composite interval mapping (CIM) using 262 $F_{6:8}$ recombinant inbred lines of Conrad (resistant) × Sloan (susceptible).

				IM		CIM			
Chromosome	Interval	Nearest marker	Consensus map†	LOD	Exp. var.	LOD	Exp. var.	LOD thr.‡	R^2§
	cM		cM		%		%		%
8	0.0–10.8	BARC_051847_11270	101.9	3.1	6.0	3.1	5.0	1.4	9.2
13	21.0–23.8	FLOWER_COLOR W1/w1 locus	17.4	1.9	3.6	2.8	4.4	1.5	5.1
15	0.0–19.0	BARC_025663_049888	47.9	3.6	11.2	3.4	6.7	1.1	7.2
16	12.2–21.9	Satt693	38.0	3.0	5.9	2.9	4.3	1.4	5.2
19	31.7–40.3	BARCSOYSSR_19_1452		1.8	3.5	2.2	3.3	1.8	3.6

†Consensus map position (Hyten et al. 2008) of the nearest marker in each interval.

‡Threshold of significance for LOD for each chromosome based on permutation tests of 1000 iterations ($P < 0.05$; Churchill and Doerge, 1994).

§R2 values from one-way ANOVA.

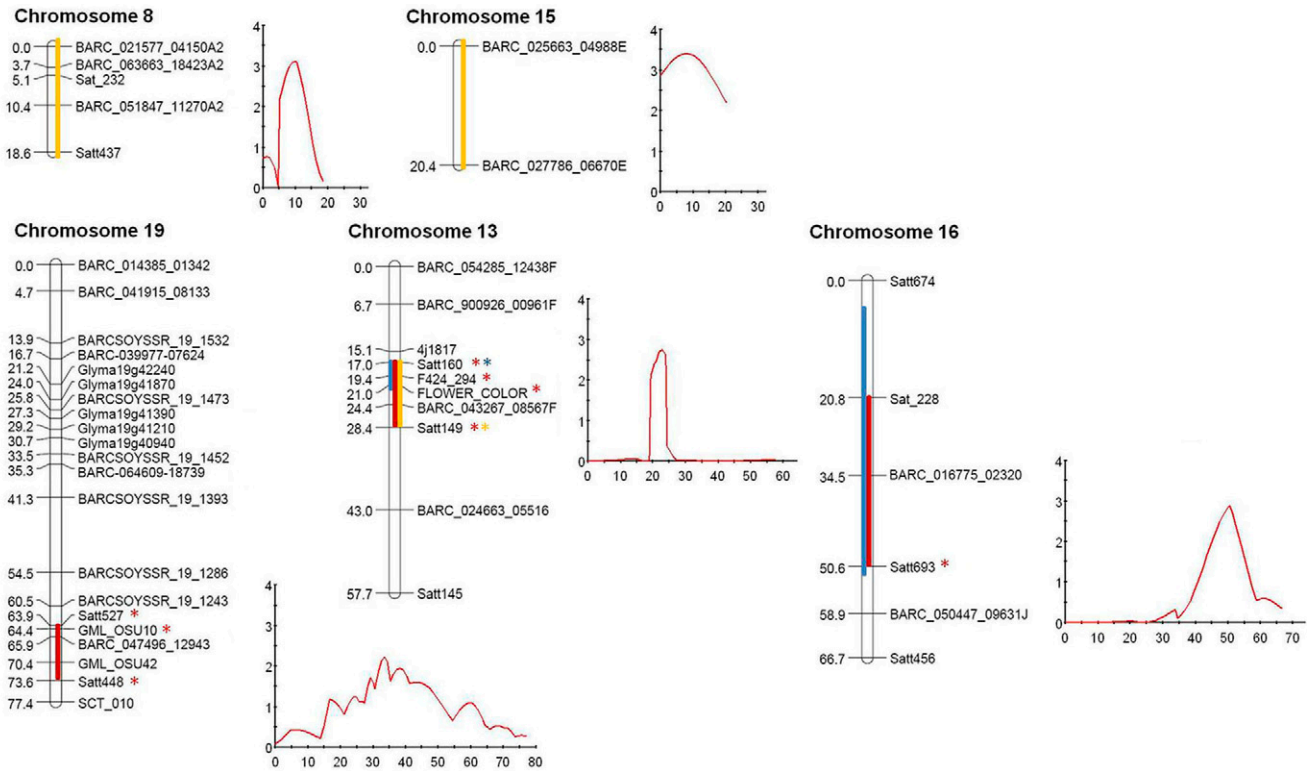

Figure 3. Genetic maps generated from the genotype data from the Conrad (resistant) × Sloan (susceptible) $F_{6:8}$ recombinant inbred lines using JoinMap4.0 (van Ooijen, 2006) and logarithm of odds (LOD) charts of the putative quantitative trait loci (QTLs) from composite interval mapping analysis by MapQTL 5.0 (van Ooijen, 2004). The chromosome number and assigned molecular linkage group is listed above each linkage group. Colored lines indicate regions where putative QTLs overlap with QTLs identified for other soybean pathogens, and an asterisk indicates significance of a molecular marker from both pathogens. The different pathogens are indicated by color: blue (*Fusarium virguliforme*), red (*Phytophthora sojae*), and yellow (*Sclerotinia sclerotiorum*).

Based on CIM, only 24% of the phenotypic variation could be associated with five QTL. This may be partly attributed to the lack of polymorphic markers in some regions, which was presumed to be due to the genetic similarity of Conrad and Sloan (Supplemental Table 2). The three *F. graminearum* resistance QTLs identified by CIM in this study are in regions of the genome previously reported to have QTL conferring resistance to another necrotroph, *Sclerotinia sclerotiorum*, on chromosomes 8 (Arahana et al., 2001; Vuong et al., 2008; Guo et al., 2008), 13 (Arahana et al., 2001; Guo et al., 2008), and 15 (Arahana et al., 2001; Guo et al., 2008) (Fig. 3). In addition to *S. sclerotiorum*, a number of disease resistance QTLs have been reported from the same region on chromosome 13 (Gordon et al., 2006). Other regions for resistance to *S. sclerotiorum* that were also significant for *F. graminearum* through one-way ANOVA were chromosome 9 (Arahana et al., 2001; Kim and Diers, 2000; Vuong et al., 2008), 11 (Arahana et al., 2001), and 18 (Arahana et al., 2001; Guo et al., 2008). QTLs conferring resistance to *S. sclerotiorum* were minor, with R^2 values reported to range from 4.0 to 15.7% (Arahana et al., 2001; Guo et al., 2008; Kim and Diers, 2000; Vuong et al., 2008). Conrad is moderately susceptible to *S. sclerotiorum* based on field evaluations from data collected in 1994 (Kim et al., 1999). Further studies are necessary to assess if these same loci confer resistance to both of these pathogens.

Jack (Nickell et al., 1990) and Ripley were previously identified as sources of high levels of partial resistance to *F. virguliforme* (Hartman et al., 1997; Jin et al., 1996). In this study, Jack had moderate levels of resistance to *F. graminearum*, while Ripley was susceptible (Table 1). To the best of our knowledge, neither Conrad nor Sloan has been identified as a source of resistance for *F. virguliforme*. It is also important to note that resistance to *F. virguliforme* has two different components: resistance to root rot and resistance to the translocated toxin that causes foliar symptoms (Chang et al., 1997; Hnetkovsky et al., 1996; Huang and Hartman, 1998; Kazi et al., 2008; Killebrew et al., 1988; Meksem et al., 1999; Melgar and Roy 1994; Njiti et al., 1998; Rupe 1989; Rupe et al., 1991; Stephens et al., 1993). Therefore, it seems unlikely that the QTL associated with the toxin phase of this disease would contribute toward resistance to *F. graminearum*. QTLs on chromosomes 6, 17, and 18 associated with root rot resistance to *F. virguliforme* (Kazi et al., 2008) were not associated with resistance to *F. graminearum* in this study. However, two QTLs on chromosomes 13 and 16 associated with toxin resistance to *F. virguliforme* overlapped with two putative QTLs identified by CIM in this study (Fig. 3). Kassem et al. (2006) reported that the Satt160 marker on chromosome 13 appeared to be a major determinant of seed yield. The QTL identified on chromosome 16 was significantly associated with the reduction in leaf scorch score, which is a resistance response to the toxin from *F. virguliforme* (Sanitchon et al., 2004).

Conrad is a source of partial resistance for both *P. sojae* and *F. graminearum*. There were regions on three chromosomes associated with resistance to both pathogens. Regions of the Conrad genome near Satt693 marker on chromosome 16 and between Satt160 and Satt149 markers on chromosome 13 were associated with resistance to *F. graminearum* and these same regions from the moderately susceptible parent Sloan were associated with resistance to *P. sojae*. On chromosome 19, Satt527 and GML_OSU10 were associated with resistance to *F. graminearum* in Sloan (Supplemental Table 1), while the resistance to *P. sojae* was from Conrad (Wang et al., 2010) (Fig. 3). Based on these initial results, the putative QTL from Conrad and Sloan that were identified in this population do not confer resistance to both pathogens, suggesting that different loci and possibly different mechanisms contribute to resistance to *P. sojae* and *F. graminearum*. Future studies will focus on expression analysis of resistance genes in the regions associated with resistance to these two pathogens. This may provide insights on how plants defend themselves from biotrophic and necrotrophic pathogens.

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