Influence of Soybean Monoculture on *Phialophora gregata* f. sp. *sojae* IGS-Genotype B Isolate Aggressiveness

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ABSTRACT

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Many soybean accessions described as resistant to brown stem rot (BSR) are preferentially colonized by isolates of *Phialophora gregata* f. sp. *sojae* genotype B. These isolates are generally considered less aggressive than isolates of *P. gregata* f. sp. *sojae* genotype A because they cause minor or no foliar symptoms characteristic of BSR. However, variation in aggressiveness has been observed among isolates of *P. gregata* f. sp. *sojae* genotype B. To determine if BSR-resistant soybean accessions would preferentially select for more aggressive isolates of *P. gregata* f. sp. *sojae* genotype B, monocultures of both BSR-resistant or BSR-susceptible accessions were established at the Arlington Agriculture Research Station, Arlington, WI. BSR-susceptible cv. Corsoy 79 and BSR-resistant plant introduction (PI) 567.157A were inoculated under greenhouse conditions with a total of 39 isolates of *P. gregata* f. sp. *sojae* genotype B obtained from the different monocultures. BSR severity was determined as the percentage of symptomatic foliar and internal stem tissue. Overall, BSR severity was low and did not exceed 20% for either foliar or stem symptoms. Isolates of *P. gregata* f. sp. *sojae* genotype B caused more severe foliar ($P < 0.0001$) and stem ($P = 0.0008$) symptoms of BSR on PI 567.157A than on Corsoy 79. Analysis of BSR stem symptom severity indicated an interaction ($P = 0.0124$) between soybean accession and the origin of isolates of *P. gregata* f. sp. *sojae* genotype B. Isolates of *P. gregata* f. sp. *sojae* genotype B obtained from the monoculture of a BSR-susceptible or -resistant accession were more aggressive than isolates from a mixed resistant and susceptible soybean monoculture. The relationship between the origin of isolate of *P. gregata* f. sp. *sojae* genotype B and isolate aggressiveness was more apparent for PI 567.157A than for Corsoy 79. Results of this study indicate that the monoculture of resistant or susceptible soybean favors an increase in the aggressiveness of isolates of *P. gregata* f. sp. *sojae* genotype B. Furthermore, results suggest that resistance to genotype A may be genetically different from resistance to genotype B.

Brown stem rot (BSR) is a disease that continues to impact soybean (*Glycine max*) production in many areas of the north-central United States (37). The symptoms associated with BSR include browning of the vascular and pith tissues (stem symptoms) and interveinal necrosis and chlorosis, wilting, and defoliation of the leaves (foliar symptoms). Although BSR can be successfully managed with crop rotation (3,22) and soil tillage (3,36), host resistance remains the preferred method of control (9). However, resistance to BSR in commercially available cultivars is limited to two main sources; cultivars containing one or more *Rbs* genes (9) or those derived from plant introduction (PI) 88788 (18,27). In addition to a narrow genetic base, the resistance in these cultivars has been defined as a reduction in symptom severity (3,4,18) and not by the prevention or restriction of pathogen colonization (7,20,24).

The causal agent of BSR, *Phialophora gregata* f. sp. *sojae* (syn. *Cadophora gregata*; *Cephalosporium gregatum*), is a residue-borne fungal pathogen that survives between soybean crops as a saprophyte in host tissue colonized during its parasitic phase (2,20). Isolates of *P. gregata* f. sp. *sojae* can be classified as either genotype A or genotype B based on an insertion or deletion (INDEL) in the intergenic spacer region of ribosomal DNA (7). The two genotypes are common throughout the north-central United States and are often found in the same field. Genotypes A and B can infect both BSR-resistant and -susceptible soybean cultivars as well as concurrently colonizing the same plant. However, data from several studies suggest that genotypes A and B exhibit cultivar preference (7,17,24). Soybean cultivars classified as resistant are colonized at a greater frequency by isolates of genotype B than genotype A while cultivars classified as susceptible are colonized at a greater frequency by isolates of genotype A than genotype B. Among resistant cultivars, preferential colonization is further influenced by the source of resistance. Cultivars with resistance derived from PI 88788 are colonized significantly more by *P. gregata* f. sp. *sojae* genotype B than

resistant cultivars with one or more *Rbs* genes (7,17,24).

Isolates of *P. gregata* f. sp. *sojae* genotypes A and B also differ in the type and severity of symptoms they produce (12,16). Overall, isolates of *P. gregata* f. sp. *sojae* genotype A cause both BSR foliar and stem symptoms while isolates of genotype B cause stem discoloration and minor or no foliar symptoms. On BSR-susceptible cultivars, isolates of genotype A cause greater symptom severity than isolates of genotype B and are considered more aggressive (12,16). However, variation in aggressiveness exists within populations of both genotypes. Isolates of genotype A range from benign to highly aggressive while isolates of genotype B range from benign to mildly aggressive (12,15).

Prior to the identification of *P. gregata* f. sp. *sojae* genotypes, the monoculture of a BSR-resistant cultivar was believed to be as effective as crop rotation with a nonhost in decreasing disease incidence and severity (33). Although this conclusion was thought to be due to a decrease in inoculum quantity of *P. gregata* f. sp. *sojae*, current data suggest the decreases in disease incidence and severity may have been the result of a change in the genotypic structure of the *P. gregata* f. sp. *sojae* population in favor of genotype B (16,17). Today, the interaction between soybean genotype and genotypes A and B of *P. gregata* f. sp. *sojae* remains ambiguous. Because host resistance is the preferred method for controlling BSR, understanding the influence of soybean genotypes on *P. gregata* f. sp. *sojae* genotype B is necessary for the development and deployment of effective disease management strategies. To study the influence of host genotype on *P. gregata* f. sp. *sojae* genotype B, soybean accessions differing in resistance and susceptibility to BSR were monocultured from 2000 to 2005 with the goal of elucidating the interaction between *P. gregata* f. sp. *sojae* and its soybean host. As part of this goal, the present study was developed to determine whether the continuous cropping of genetically different soybean accessions influenced aggressiveness, defined here as the amount of disease (symptom development), among isolates of *P. gregata* f. sp. *sojae* genotype B.

MATERIALS AND METHODS

In 2000, a field site was established at the Arlington Agricultural Research Sta-

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tion, Arlington WI, in an area with no history of soybean or *P. gregata* f. sp. *sojae* (17). Briefly, 10 soybean accessions were selected based on their genetic resistance or susceptibility to BSR (Table 1). Monocultures of each accession were established by planting each accession in the same 38-by-9 m plot from 2000 through 2004 (Fig. 1). In 2005, plots were reduced in size to 3 by 9 m, which allowed each accession to be planted in an area previously planted to itself as well as that of the nine other accessions (Fig. 1).

When soybean plants reached beginning maturity (R7), 15 plants were arbitrarily selected from within each plot, bulked into five groups of 3 plants each, and air dried at room temperature (2 to 3 days). For each bulk, approximately 13 to 15 cm of the lower stem was ground and dilution plated onto a semi-selective medium (1,20). Plates were then placed in the dark at 12°C for 4 weeks. Following incubation, approximately 10 colonies of *P. gregata* f. sp. *sojae* were arbitrarily selected from each plate and transferred onto quarter-strength potato dextrose agar (PDA) (25 g of peeled, sliced potato tubers, 1.25 g of

dextrose, 7.5 g agar, and 500 ml of deionized water). After 5 to 7 days of incubation at room temperature, one to five pure-culture isolates were identified and transferred onto full-strength PDA. Following a subsequent 2-week incubation at room temperature, subcultures of each isolate were transferred to glass vials containing quarter-strength PDA and stored at 12°C. The remaining mycelium was then used for DNA extraction and the genotype of each isolated determined by polymerase chain reaction (7). Of the 1,143 isolates of *P. gregata* f. sp. *sojae* obtained, 71% were identified as genotype B (17).

Isolates of *P. gregata* f. sp. *sojae* genotype B were separated into four categories based on the cropping history of the plot from which the isolate was obtained (Fig. 2). Ten isolates were arbitrarily selected from within each of the four cropping histories and evaluated for aggressiveness based on BSR foliar and internal stem symptom severity (Fig. 2; Table 2). Spore suspensions of each isolate were prepared

in 500-ml flasks containing 250 ml of either green bean broth (40 g of Gerber 2nd Foods green bean baby food [Gerber, Freemont, MI] and 500 ml of deionized water) or soybean seed broth (80 g of soybean seed, cv. Bell, and 1 liter of deionized water). Flasks were inoculated with three plugs of mycelium from an actively growing culture on PDA and incubated at room temperature on a rotary shaker for approximately 2 weeks. Following incubation, green bean and soybean seed broth cultures were combined and blended in 1-liter beakers with a Braun (Boston) vertical hand mixer and the concentration of conidia and mycelial fragments determined with a hemacytometer. Inoculum was adjusted to 1×10^6 conidia and mycelium per milliliter with de-ionized water and stored at -20°C until inoculation.

Seed of BSR-susceptible soybean cv. Corsoy 79 (24) and BSR-resistant PI 567-157A (21) were germinated in medium-grade vermiculite in 25.4-by-50.8-cm, 96-well flats containing commercial-grade

Fig. 1. Field design and layout for studying the influence of monocropping brown stem rot (BSR)-resistant (0–4) and BSR-susceptible (5–9) soybean accessions on stem and soil populations of *Phialophora gregata* f. sp. *sojae*. **A**, Field design from 2000 through 2004 in which accessions were planted continuously in the same location in each year. **B**, Field design for 2005 in which the direction of planting was rotated 90° and the plot size reduced so that each accession was planted onto the monoculture of itself as well as that of the other nine accessions. Figure depicts one of four replications at Arlington, WI.

Table 1. Soybean accessions grown from 2000 through 2005 at Arlington, WI

Soybean accession	BSR reaction ^a	BSR-R source or gene ^b	BSR-S soybean accession in parentage
BSR 101	R	<i>Rbs</i> 1,3	...
IA 2008R	R	<i>Rbs</i> 1,3	...
LN92-120334	R	<i>Rbs</i> 2	...
Dwight	R	PI 88788	...
Jack	R	PI 88788	...
Sturdy	S	...	Century
Corsoy 79	S	...	Corsoy
Hardin	S	...	Corsoy
Pioneer 9234	S	...	Peking
Williams 82	S	...	Peking

^a Reaction of soybean accession to brown stem rot (BSR); R = resistant and S = susceptible.

^b Gene or genes conferring resistance or source (plant introduction [PI]) used to obtain resistance to BSR.

Fig. 2. Diagrammatic representation of the four soybean cropping histories from which isolates of *Phialophora gregata* f. sp. *sojae* genotype B were obtained. R_m = brown stem rot (BSR)-resistant accession monocultured from 2000 through 2004. S_m = BSR-susceptible accession monocultured from 2000 through 2004. R_a = BSR-resistant accession planted in 2005 from which the isolate of *P. gregata* f. sp. *sojae* genotype B was obtained. S_a = BSR-susceptible accession planted in 2005 from which the isolate of *P. gregata* f. sp. *sojae* genotype B was obtained.

Bradyrhizobium japonicum (Nitragin Inoculants, Liphatech Inc., Milwaukee, WI). Seedlings were inoculated approximately 14 days after seeding, at the VC-V1 growth stage (unifoliate leaves fully expanded), using a modified root-dip method as previously described (16). Briefly, nine seedlings of each cultivar were removed from vermiculite and the roots were rinsed in water. Roots were trimmed so that approximately 3 to 4 cm of root remained, and the seedlings were placed into a 250-ml beaker containing inoculum of a single isolate for 20 min. The method was repeated for each isolate, including two isolates of *P. gregata* f. sp. *sojae* genotype A (Table 2) (16), which were included as a positive control. Negative control plants were mock inoculated in the same method by using a sterilized, quarter-strength mixture of green bean and soybean seed broths.

Immediately following inoculation, seedlings were transplanted to 15.2-cm fiber pots (Formart Containers, Inc., Cedarburg, WI) containing a 1:1 mixture of Sun Gro (Bellevue, WA) Metro Mix-300 and Pro-Moss-TBK (Quakertown, PA) sphagnum peat moss. Each cultivar-isolate combination was replicated three times, with three plants per pot, and arranged in a completely randomized blocked design (RCBD). Plants were fertilized once a week with Peter's 20-10-20 peat-lite special and treated as needed with Safer brand (Safer, Inc., Woodstream Corp., Lititz, PA) sulfur spray or 1.6% ultrapure oil (Whitmire Micro-Gen Research Laboratories, Inc., St. Louis) plus 0.1% Latron B-1956 spreader-sticker (Dow Agrosciences, LLC., Indianapolis, IN) for control of powdery mildew. Plants were grown for approximately 6 to 7 weeks until the R2 to R3 growth stage (full flowering to beginning pod). Each plant per pot was then rated for BSR foliar and internal stem symptom severity by visually estimating the percentage of tissue symptomatic of BSR and assigning a Horsfall-Barratt (HB) rating (19). This study was conducted three times (three experimental runs), consecutively, from 16 October 2008 to 11 June 2008 at the West Madison Agricultural Research Station Greenhouses, Madison, WI. Greenhouses were programmed with a 14-h photoperiod supplemented by metal halide and high-pressure sodium lamps, with average daytime and nighttime temperatures at 26 and 18°C, respectively.

All ratings based on HB values were converted to percentages prior to analysis by using the Elanco conversion tables (30). Disease severity values for each of the three plants per pot were averaged and the means analyzed as an RCBD by using PROC MIXED (SAS version 9.1; SAS Institute, Cary, NC). Isolate of *P. gregata* f. sp. *sojae* genotype B, experimental run, and the interaction of experimental run-block were treated as random effects. Least

significant difference comparisons of the means were made by using the LSMEANS and PDIF options. Comparisons of the mean were conducted at $P < 0.05$. Contrast statements were used for individual mean comparisons. Model assumptions were examined with PROC UNIVARIATE and studentized residuals prior to analysis.

RESULTS

Symptom development in BSR-susceptible cv. Corsoy 79 inoculated with *P. gregata* f. sp. *sojae* genotype A suggest that greenhouse environmental conditions were conducive for disease development during all three experimental runs (Table 3). However, foliar symptom severity was noticeably lower for Corsoy 79 in the third experimental run, which was likely the

result of higher than average greenhouse temperatures during May and June (9). In spite of this, BSR foliar and internal stem symptom severity was lower for BSR-resistant PI 567.157A than for susceptible Corsoy 79 in each experimental run when inoculated with *P. gregata* f. sp. *sojae* genotype A (Table 3).

As expected (12,16), both foliar and internal stem symptom severity was low (<20%) on Corsoy 79 and PI 567.157A inoculated with *P. gregata* f. sp. *sojae* genotype B for all three experimental runs. Similar to the results observed for inoculation with genotype A, foliar symptom severity was visibly reduced in the third experimental run for both Corsoy 79 and PI 567.157A inoculated with isolates of genotype B (data not shown). Regardless

Table 2. Designation and origin of isolates of *Phialophora gregata* f. sp. *sojae*

Isolate	Genotype ^a	Soybean accession		Cropping history ^d
		Monoculture (2000–2004) ^b	Sampled accession (2005) ^c	
1014-5A	B	Dwight	LN92-12033	R-R
1019-3	B	Dwight	Jack	R-R
1026-1	B	Jack	Jack	R-R
2010-4	B	LN92-12033	IA 2008R	R-R
2082-5	B	BSR 101	BSR 101	R-R
3037-3	B	LN92-12033	BSR 101	R-R
3047-2	B	Jack	Jack	R-R
3078-1B	B	Dwight	Jack	R-R
4023-1	B	IA 2008R	Jack	R-R
4079-4	B	LN92-12033	LN92-12033	R-R
1017-2A	B	Dwight	Pioneer 9234	R-S
1021-5B	B	Jack	Hardin	R-S
1023-5	B	Jack	Pioneer 9234	R-S
2060-2	B	Jack	Pioneer 9234	R-S
3095-3	B	BSR 101	Williams 82	R-S
4006-2	B	Dwight	Hardin	R-S
4009-1	B	Dwight	Pioneer 9234	R-S
4075-2	B	LN92-12033	Hardin	R-S
4081-5	B	Jack	Sturdy	R-S
1031-1A	B	Williams 82	IA 2008R	S-R
1035-1	B	Williams 82	Jack	S-R
1040-4A	B	Williams 82	BSR 101	S-R
2078-5	B	Sturdy	Jack	S-R
2091-5B	B	Williams 82	BSR 101	S-R
3027-4	B	Pioneer 9234	IA 2008R	S-R
3081-3	B	Corsoy 79	LN92-12033	S-R
4019-2	B	Hardin	Jack	S-R
4019-2	B	Hardin	Jack	S-R
4033-5	B	Corsoy 79	Jack	S-R
1033-1C	B	Williams 82	Sturdy	S-S
1037-3	B	Williams 82	Corsoy 79	S-S
2043-1	B	Corsoy 79	Corsoy 79	S-S
2075-5B	B	Sturdy	Pioneer 9234	S-S
2076-5	B	Sturdy	Williams 82	S-S
2098-1(1)	B	Williams 82	Corsoy 79	S-S
3025-3A	B	Pioneer 9234	Hardin	S-S
3063-5	B	Hardin	Sturdy	S-S
4049-2	B	Williams 82	Williams 82	S-S
4065-2	B	Sturdy	LN92-12033	S-R
4098-5	B	Pioneer 9234	Pioneer 9234	S-S
3051-2	A	IA 2008R	Corsoy 79	R-S
Fulton-OH ^e	A	Unknown	Unknown	Unknown

^a Genotype of *P. gregata* f. sp. *sojae* isolate determined by polymerase chain reaction (7).

^b Soybean accession grown in the same location for 5 years.

^c Soybean accession planted in 2005 from which the isolate of *P. gregata* f. sp. *sojae* was obtained.

^d Reaction of soybean accession to brown stem rot (BSR) grown in the same location for 5 years (first letter) followed by the reactions of the soybean accession to BSR planted in 2005 from which the isolate of *P. gregata* f. sp. *sojae* was obtained (second letter). R = resistant and S = susceptible.

^e Standard, aggressive isolate of *P. gregata* f. sp. *sojae* genotype A (7,15,16).

of the differences in symptom severity obtained across the three experimental runs, preliminary analysis did not indicate an effect of experimental run on either foliar or stem symptom severity. Therefore, data from all three experimental runs was combined for analysis.

BSR-resistant PI 567.157A expressed greater ($P < 0.0001$) foliar and stem ($P < 0.0008$) symptom severity than susceptible Corsoy 79 when inoculated with isolates of *P. gregata* f. sp. *sojae* genotype B (Fig. 3). Analysis of BSR stem symptom severity revealed an interaction ($P = 0.0124$) between soybean accession and the origin of isolates of genotype B. Isolates of genotype B obtained from the monoculture of a BSR-resistant or -susceptible accession caused greater ($P = 0.0020$) stem symptom severity on BSR-resistant PI 567.157A than isolates obtained from a mixed resistant and susceptible soybean monoculture (Fig. 4). On BSR-susceptible cv. Corsoy 79, isolates of genotype B obtained from the monoculture of a BSR-susceptible accession caused greater ($P = 0.0573$) stem symptom severity than isolates obtained from a mixed soybean monoculture or the monoculture of a BSR-resistant accession (Fig. 4). As with the results observed on PI 567.157A, isolates of genotype B obtained from the monoculture of a BSR-resistant accession tended to cause greater stem symptom severity on Corsoy 79 than isolates from a mixed soybean monoculture; however, this difference was not significant. Analysis of BSR foliar symptoms showed no relationship between the origin of isolates of genotype B and BSR foliar symptom severity (Fig. 4).

Analysis of foliar and stem symptom severity did not indicate a relationship between the different sources of BSR resistance and isolate aggressiveness. Isolates of *P. gregata* f. sp. *sojae* genotype B obtained from a PI 88788-derived BSR-resistant monoculture did not differ in aggressiveness (foliar: $P = 0.579$; stem: $P = 0.909$) from those isolates obtained from an *Rbs*-derived BSR-resistant monoculture. In addition, no differences in aggressiveness were observed for isolates of genotype B obtained from a PI 88788/*Rbs*-

Fig. 3. Brown stem rot (BSR) foliar and stem symptom severity for BSR-susceptible cv. Corsoy 79 and BSR-resistant plant introduction (PI) 567.157A inoculated with 39 isolates of *Phialophora gregata* f. sp. *sojae* genotype B. Symptom severity was assessed by visually estimating the percentage of symptomatic tissue and assigning a Horsfall-Barratt (HB) rating. HB ratings were converted to percentages prior to analysis. Values are the means of three experimental replications. Bars represent one standard error of the mean. Means marked by an asterisk (*) are significantly different at $P = 0.05$.

Fig. 4. Brown stem rot (BSR) foliar and stem symptom severity for BSR-susceptible cv. Corsoy 79 and BSR-resistant plant introduction (PI) 567.157A inoculated with 39 isolates of *Phialophora gregata* f. sp. *sojae* genotype B obtained from one of four soybean cropping histories (9–10 isolates per cropping history). RR = BSR-resistant accession monocultured from 2000 through 2004 followed by the planting of a BSR-resistant accession in 2005 from which the isolate of genotype B was obtained. RS = BSR-resistant accession monocultured from 2000 through 2004 followed by the planting of a BSR-susceptible accession in 2005 from which the isolate of genotype B was obtained. SR = BSR-susceptible accession monocultured from 2000 through 2004 followed by the planting of a BSR-resistant accession in 2005 from which the isolate of genotype B was obtained. SS = BSR-susceptible accession monocultured from 2000 through 2004 followed by the planting of a BSR-susceptible accession in 2005 from which the isolate of genotype B was obtained. Symptom severity was assessed by visually estimating the percentage of symptomatic tissue and assigning a Horsfall-Barratt (HB) rating. HB ratings were converted to percentages prior to analysis. Bars represent one standard error of the mean. Means marked by an asterisk (*) are significantly different at $P = 0.05$.

Table 3. Percent foliar and internal stem symptoms characteristic of brown stem rot (BSR) for BSR-susceptible cv. Corsoy 79 and BSR-resistant plant introduction (PI) 567.157A inoculated with isolates of *Phialophora gregata* f. sp. *sojae* genotype A

Accession	Symptom severity ^a					
	Experimental run 1		Experimental run 2		Experimental run 3	
	Foliar	Stem	Foliar	Stem	Foliar	Stem
Corsoy 79	81.1 $\pm$ 4.7	25.5 $\pm$ 7.7	93.2 $\pm$ 0.8	42.9 $\pm$ 7.3	33.7 $\pm$ 14.7	21.9 $\pm$ 7.0
PI 567.157A	48.1 $\pm$ 12.6	13.9 $\pm$ 5.3	2.4 $\pm$ 1.8	14.3 $\pm$ 3.0	0 $\pm$ 0.0	6.5 $\pm$ 1.6
LSD ^b	30.9	...	4.6	15.8	33.9	16.3
P value	0.043	0.15	<0.0001	0.003	0.051	0.061

^a Mean percent tissue symptomatic of BSR for three replications per experimental run. Symptom severity was assessed by visually estimating the percentage of symptomatic tissue and assigning a Horsfall-Barratt (HB) rating. HB ratings were converted to percentages and the values for plants inoculated with each isolate of *P. gregata* f. sp. *sojae* genotype A combined prior to analysis; $\pm$ represents one standard error of the mean. Study was conducted three times, consecutively, from 16 October 2008 to 11 June 2008.

^b LSD = least significant difference at $P = 0.05$.

resistant monoculture. Similar results were observed with isolates of genotype B obtained from BSR-susceptible soybean monocultures or a culture of different susceptible soybean accessions.

DISCUSSION

The long-term monoculture of a single agronomic crop coincides with an increase in the incidence and severity of many plant diseases. This increase has been associated with either a build-up of inoculum or an increase in pathogen aggressiveness (13,28). In previous studies with various fungal pathogens (6,8,13,14), results indicate an increase in isolate aggressiveness when resistant or susceptible cultivars are monocultured over time. Similarly, the results of this study indicate that the monoculture of soybean accessions resistant or susceptible to BSR influences the aggressiveness of isolates of *P. gregata* f. sp. *sojae* genotype B.

The more aggressive isolates of *P. gregata* f. sp. *sojae* genotype B were obtained from the monoculture of BSR-susceptible soybean accessions followed by those isolates obtained from the monoculture of BSR-resistant accessions. Although soybean was in continuous cultivation, monocultures in which both resistant and susceptible soybean accessions were grown yielded the least aggressive isolates of *P. gregata* f. sp. *sojae* genotype B. The increased aggressiveness of isolates of genotype B obtained from areas continuously cropped to purely resistant or susceptible soybean accessions compared with those obtained from a mixed soybean monoculture may be indicative of a negative feedback. In ecological studies (25,29,35), negative feedbacks are hypothesized to be a way that native systems maintain diversity in plant communities. When a single plant species occupies an area, changes are thought to take place in the soil community that decreases the growth of that species relative to another. As such, plants of one species may cause the build-up of pathogenic soilborne microorganisms that reduce the succession success of the same species. In the present study, although a single plant species was used, differences in the genetics between BSR-resistant and -susceptible accessions may be sufficient to initiate a negative feedback.

Today, economic considerations may triumph over sound agronomic practices as more soybean growers consider continuous soybean production. In production systems where crop rotations are limited or non-existent, disease management strategies may include rotating cultivars with different sources of resistance to mitigate increases in disease incidence and severity (31,34,38). Resistance to BSR in commercially available cultivars is limited to those containing one or more *Rbs* genes (9) or cultivars derived from PI 88788 (18,27). Although, genetically, these sources map

to the same region of the soybean genome (27), studies have shown that both disease development and pathogen interactions differ between cultivars derived from PI 88788 and those containing an *Rbs* gene (17,18,24). However, from the results of this study, both sources of BSR resistance favored isolates of *P. gregata* f. sp. *sojae* genotype B with similar aggressiveness. Therefore, in continuous soybean production systems, rotating cultivars with different sources of BSR resistance may not be a practical option for managing BSR.

As expected (12,16), symptom severity was generally low (<20%) for both BSR-susceptible Corsoy 79 and BSR-resistant PI 567.157A inoculated with isolates of *P. gregata* f. sp. *sojae* genotype B. However, PI 567.157A expressed more severe foliar and stem symptoms than Corsoy 79. Screening for resistance to BSR typically uses isolates of *P. gregata* f. sp. *sojae* that are described as “aggressive”, “defoliating”, or “Type 1” (10), suggesting that isolates are of the A genotype (11,18,26). In addition, numerous studies use isolate Oh2, an isolate of *P. gregata* f. sp. *sojae* genotype A that has been in continuous culture since the 1970s (4,5,23,27,32). Consequently, little is known about resistance to *P. gregata* f. sp. *sojae* genotype B. The increase in foliar and stem symptoms severity observed for PI 567.157A compared with Corsoy 79 when inoculated with genotype B suggests that (i) resistance has been identified to genotype A but not to genotype B and (ii) resistance to genotype A may be genetically different from resistance to genotype B. Results of this study indicate the need to include isolates of both genotypes in resistance breeding and to consider soybean genetics as well as reaction to BSR when making cultivar selection.

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