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Evaluation of Soybean Cultivars, 'Williams' Isogenic Lines, and Other Selected Soybean Lines for Resistance to Two *Soybean Mosaic Virus* Strains

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ABSTRACT

***Soybean mosaic virus* (SMV) is one of the most common soybean viruses worldwide. The resistance or susceptibility of most commercial soybean cultivars to SMV is not known. The objectives of this study were to evaluate resistance to SMV strains G1 and G5 of current soybean cultivars, isogenic lines with different *Rsv* genes and alleles in 'Williams' or 'Williams 82' background, and selected soybean lines with reported or observed SMV resistance. Commercial and precommercial soybean cultivars were screened for resistance to SMV strains G1 and G5. Based on multiple tests, 1.5% and 6.7% of the 850 cultivars were resistant to SMV-G1 and SMV-G5, respectively. No cultivars were resistant to both strains. Expression of different SMV resistance genes in Williams isogenic lines inoculated with both SMV strains indicated that lines with *Rsv1-y* from 'Dorman', or unnamed resistance genes from 'Kosamame', and 'Sodendaizu', were resistant to G1 and susceptible to G5. Lines with *Rsv1* alleles from PI 96983, 'Marshall', or 'Ogden' were resistant to both strains, and lines with *Rsv1* alleles from 'Raiden', 'SS 74185' (PI486355), or 'Suweon 97' were resistant to G1 and produced a systemic necrosis reaction with G5. Lines with *Rsv3-h* from 'Hardee' were susceptible to G1 and resistant to G5. Isogenic lines with SMV resistance genes from 'Buffalo' showed either a resistant–resistant or resistant–susceptible reaction to the two SMV strains, suggesting the presence of more than one SMV resistance gene. Ten selected lines with reported or observed resistance to SMV were inoculated with the two SMV strains. Some lines were resistant to either G1 or G5, and some were resistant to both strains.**

Soybean mosaic virus is one of the most common soybean viruses worldwide and is aphid and seed transmitted (Hill, 1999). The introduction of the Asian soybean aphid (*Aphis glycines* Matsumura) is likely to exacerbate SMV problems in the United States in the future (Hartman et al., 2001). Symptoms of SMV can include mosaic, leaf mottling, leaf distortion, dwarfing of plants, or systemic necrosis. Causing significant yield loss, SMV is regarded as economically important in many areas. A wide range of soybean yield losses has occurred due to SMV infection (Hill, 1999; Ross, 1968; Zhang, 1979). *Soybean mosaic virus* can cause seed coat mottling and reduce the quality of soybean seeds, particularly in edible soybean cultivars (Hobbs et al., 2003).

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Strains of SMV differ in their symptom expression on soybeans. *Soybean mosaic virus* isolates have been grouped into strains G1 through G7 based on their ability to infect a set of soybean differentials (Cho and Goodman, 1979). Efforts to control SMV mainly involve development and utilization of soybeans with SMV resistance. Various sources of SMV resistance and resistance genes have been identified in soybeans (Chen et al., 1991; Cho and Goodman, 1982; Gunduz et al., 2001; Liao et al., 2002; Lim, 1985; Wang et al., 1998, 2005; Zheng et al., 2005).

To date, three loci, *Rsv1*, *Rsv3*, and *Rsv4* have been reported to control SMV and have been used in soybean breeding programs (Palmer et al., 2004). The *Rsv1* locus (Kiihl and Hartwig, 1979) is multi-allelic with nine known alleles (Palmer et al., 2004). Buzzell and Tu (1984) initially identified *Rsv2* in Raiden; however, subsequent research (Chen et al., 2001; Wang et al., 1998) showed that Raiden actually contains an *Rsv1* allele. The *Rsv3* gene, identified in Hardee, controls resistance to SMV strains G5, G6, and G7, but not other SMV strains (Buss et al., 1999). The *Rsv4* gene found in SS 74185 (PI 486355) and PI 88788 controls resistance to all known SMV strains (Gunduz et al., 2004; Ma et al., 1995).

The Varietal Information Program for Soybeans (VIPS, www.vipsoybeans.org) provides experimental results of over 800 cultivars from about 70 companies each year (ISA 2005). This information includes yield, protein, oil content, and resistance to various diseases and pests. Since 2004, SMV has been one of the pathogens used in disease resistance screening of VIPS cultivars.

The occurrence of resistance to SMV-G1 and -G5 in current soybean cultivars has not been studied, although results from earlier research indicated that resistance to SMV-G5 was more common than resistance to SMV-G1 in soybean ancestral lines (Wang et al., 2005) from which most modern day cultivars are derived (Gizlice et al., 1994). The primary objectives of this study were: (i) to evaluate current VIPS soybean cultivars for resistance to SMV-G1 and -G5; (ii) to compare the expression of resistance to the two strains in isogenic lines with different *Rsv* genes and alleles in Williams or Williams 82 background; and (iii) to evaluate reactions to both SMV strains in selected soybean lines with reported or observed SMV resistance.

MATERIALS AND METHODS

Soybean Germplasm

Seed of 850 U.S. soybean cultivars, entered into the 2004 VIPS (ISA, 2005; www.vipsoybeans.org) were obtained for

Abbreviations: ELISA, enzyme-linked immunosorbent assay; SMV, *Soybean mosaic virus*; VIPS, Varietal Information Program for Soybeans.

SMV resistance evaluation. Additionally, 19 isogenic lines developed by backcrossing different *Rsv* genes into Williams or Williams 82 soybeans were obtained from the USDA Soybean Germplasm Collection. Additionally, there were 10 selected lines included that either were reported to be resistant to SMV or observed to be resistant to SMV in our prior research. These were the University of Illinois line L97–946; the Virginia Polytechnic Institute and State University line V97–9001; Iowa State University 'IA 3010'; Asgrow 'AG 4201'; DeKalb 'DKB 4651'; University of Tennessee '5601T'; and the USDA, ARS, North Carolina State University 'N6201', 'N7001', 'N7101', and 'N7102'. In all screenings (VIPS cultivars, isogenic lines, and other selected lines) multiple repetition (three times or more) of testing of putative resistant cultivars and lines to verify resistance ensured that those cultivars or lines were definitely resistant. Williams 82 was used as the susceptible check in all tests.

Virus Strains

SMV strains G1 and G5 were originally obtained from J. Hill, Iowa State University, and maintained by continuous greenhouse transfer and stored long term in lyophilized leaves at −20°C. Classifications of isolates as G1 and G5 were confirmed on a set of soybean differentials (Cho and Goodman, 1979).

ELISA and Tissue Blot Evaluation

Trifoliolate leaf samples from individual plants were tested for the presence of SMV, by ELISA (double antibody sandwich [DAS]) (Clark and Adams, 1977) using Agdia antibodies and protocol (Agdia, Inc., Elkhart, IN) or by tissue blot (Lin et al., 1990; Srinivasan and Tolin, 1992), 2 to 3 wk after inoculation. A conjugated SMV antibody–alkaline phosphatase label (Agdia, Inc.) was used in tissue blots. Sample wells that gave absorbance values (at 405-nm wavelength) more than twice those of the healthy soybean control wells were considered positive in ELISA, and sample blots that gave a blue color were considered positive in tissue blot. Evaluations of resistance or susceptibility were based on ELISA or tissue blot reactions.

Screening Experiments

Screening for SMV-G1 and -G5 resistance in VIPS cultivars and isogenic and selected lines was conducted in the greenhouse from the winter of 2004 through the summer of 2005. For initial G1 screening of VIPS cultivars, six seeds of each line were planted in a 10-cm-diam pot in soil-less mix (Sunshine Mix LC1, Sun Gro Horticulture Inc., Bellevue, WA) and thinned to four plants after emergence. All entries that were ELISA negative were retested at least twice. For the retest for resistance to G1, seeds were planted in 4 by 12 cell plastic inserts (each cell was 6 by 4 by 5.5 cm) inside plastic trays, one entry per cell, and each entry thinned to two plants per cell. Seeds were planted in soil-less mix (Sunshine Mix LC1) and covered with coarse vermiculite. Symptom notes were taken 2 to 3 wk after inoculation. Williams 82 was planted as a susceptible check. To conserve space, all G5 initial VIPS screening was done in 8 by 12 inserts (each cell was 3 by 4 by 5.5 cm) in plastic trays, one plant per line. All tissue blot negative entries were planted again and retested at least twice more in flats with 4 by 12 inserts, two plants per line.

Isogenic lines and selected lines were planted in 4 by 12 inserts, four plants per line, and evaluated for virus symptoms visually and for virus infection by tissue blot 2 to 3 wk after SMV inoculation. These two sets of lines were tested separately and were retested at least twice to verify consistency of reaction.

Inoculum was prepared from extracts of infected leaves of Williams 82 plants maintained in the greenhouse, by grinding infected leaves with sterilized pestles and mortars in chilled 0.025 *M* KPO_4 buffer, pH 7.1, plus 0.01 *M* sodium sulfite. Pestles were used to apply inoculum to carborundum-dusted leaf surfaces. Plants were inoculated 7 to 10 d after planting at the unifoliolate growth stage. Two to three weeks after inoculation, trifoliolate leaves were examined for virus symptoms and tested by ELISA or tissue blot.

RESULTS

Susceptible Williams 82 plants had typical mosaic symptoms 2 wk after inoculation with either SMV-G1 or -G5, although G1 mosaic symptoms were milder than mosaic symptoms produced by G5. Evaluations of resistance or susceptibility in the 850 VIPS cultivars were based on ELISA or tissue blot reactions. Along with visual symptoms, all inoculated Williams 82 plants had positive reactions in ELISA and tissue blot tests. For the 850 VIPS cultivars inoculated with SMV-G1, 13 (1.5%) were ELISA negative and two cultivars had a mix of individual plants that were ELISA negative and positive (Table 1). Repeated inoculations and ELISA tests confirmed that 13 cultivars were resistant and that the two cultivars were segregating. Of these 15 cultivars resistant or segregating to SMV-G1, three were in maturity group IV and 12 were in maturity group V (Table 1). Fifty-seven (6.7%) VIPS entries were tissue blot negative for SMV-G5 (Table 1). Resistance to SMV-G5 in all of the 57 cultivars was confirmed by tissue blot tests in retesting. Of these 57 cultivars, 47 were in maturity group II, two cultivars were in maturity group III, and eight cultivars were in maturity group IV (Table 1). None of the cultivars were resistant to both SMV-G1 and -G5 strains (Table 1).

Williams isogenic lines with *Rsv1*, *Rsv1-m*, *Rsv1-t*, or an unnamed resistance gene from Buffalo, were resistant to both G1 and G5 (Table 2). Isogenic lines with *Rsv1-y* (Zheng et al., 2005) or unnamed resistance genes from Buffalo, Kosamame, or Sodendaizu were resistant to G1 but not G5, while an isogenic line with *Rsv3-h* was resistant to G5 but not G1 (Table 2). Isogenic lines with *Rsv1-r* or the genes from SS 74185 (PI 486355) or Suweon 97 (PI 483084; Chen et al., 2002) were resistant to G1 and responded with a systemic necrosis reaction to G5 (Table 2).

Among the 10 other cultivars and lines tested with previously observed or reported resistance, seven were resistant to SMV-G1, six were resistant to SMV-G5, and three were resistant to both SMV-G1 and -G5 (Table 3).

DISCUSSION

Over 91% of the VIPS cultivars were susceptible to both SMV strains. The low frequency of SMV resistance in these cultivars could portend that SMV has the potential to become a greater problem in the future now that a major vector, the soybean aphid, has become established in the region where those cultivars are grown.

Resistance to SMV-G5 was more common than resistance to G1 in the 850 cultivars tested, even though G5 is

Table 1. Resistance of soybean cultivars entered into the 2004 Illinois Varietal Information Program for Soybeans† to *Soybean mosaic virus* (SMV) strains G1 and G5.

Cultivar	Maturity Group	Company	SMV‡ G1	SMV‡ G5
9283	2.8	Agsource	S	R
9285	2.8	Agsource	S	R
5B288 RR	2.8	Atlas	S	R
274 NRR	2.7	Beck	S	R
DSR-277 RR	2.7	Dairyland	S	R
DKB 28–52	2.8	Dekalb	S	R
DKB 28–53	2.8	Dekalb	S	R
DKB 46–51	4.6	Dekalb	S	R
4960 RR	4.9	Delta Grow	R	S
DK 4763 RR	4.7	Delta King	S	R
DK 5161 RR	5.1	Delta King	R	S
DK 5366 RR	5.3	Delta King	R	S
DK 5465 RR	5.4	Delta King	R	S
DK 55T6	5.5	Delta King	R	S
DK XTJ 54 J9	4.9	Delta King	R	S
DP 4546 RR	4.5	Delta+Pine Land	R	S
D 2615 RR	2.7	Diener	S	R
8354 RR	3.5	Excel	S	R
8530 NNRR	5.3	Excel	Seg	S
FA 7264	2.6	Farm Advantage	S	R
8184 RR	2.8	Fontanelle	S	R
HS 5248	5.2	FS Hisoy	R	S
HS 2725	2.7	FS Hisoy	S	R
HS 2815	2.8	FS Hisoy	S	R
HS 2861	2.8	FS Hisoy	S	R
X 2846	2.8	FS Hisoy	S	R
H-2712 RR	2.7	Golden Harvest	S	R
H-2739 RR	2.7	Golden Harvest	S	R
H-2929 RR	2.9	Golden Harvest	S	R
H-4772 RR	4.7	Golden Harvest	S	R
SS 9405 RR	2.9	Henkel	S	R
H 283 NRR	2.8	Horizon	S	R
H421N	4.2	Horizon	S	R
612 RR	2.6	Hughes	S	R
IP 2991N	2.9	IPAP	S	R
287 RR	2.8	Kruger	S	R
287 RR/SCN	2.8	Kruger	S	R
287A RR/SCN	2.8	Kruger	S	R
289+ RR	2.8	Kruger	S	R
474 RR/SCN	4.7	Kruger	S	R
E 2884 R	2.8	Latham	S	R
L 2900 R	2.9	Latham	S	R
2896	2.8	Lewis	S	R
C 2777 NRR	2.7	LG Seeds	S	R
9530 RR	5.3	M+D Seed	R	S
9550 RR	5.5	M+D Seed	R	S
Everest RR	5.3	Merschman	Seg	S
SiouxIIRR	2.7	Merschman	S	R
AE RR 53 116	5.3	Midwest Premium Gen	R	S
M 2808 RR	2.8	Monier	S	R
2A73 RR	4.7	NC +	S	R
S 38-T8	3.8	NK Brand	S	R
93M30	2.3	Pioneer	S	R
95B32	5.3	Pioneer	R	S
PB-2732 RR	2.7	Prairie Brand	S	R
PB-2794 NRR	2.7	Prairie Brand	S	R
PB-2643 RR	2.6	Prairie Brand	S	R
5247 RR	2.7	Roeschley	S	R
RT 5130N	5.1	Southern States	R	S
S2783–4	2.7	Stine	S	R
HC-2262 RR	2.6	Trelay	S	R
HC-2282 RR	2.8	Trelay	S	R
HC-2284 RR/SCN	2.8	Trelay	S	R
4380CN	4.3	Trisler Trisoy	S	R
V 284 RR	2.8	Vigoro	S	R
V 28N5 RR	2.8	Vigoro	S	R
V 47N3 RR	4.6	Vigoro	S	R
2574 RR	2.7	Wilken	S	R
2678 RR	2.7	Wilken	S	R
2685 RR	2.8	Wilken	S	R
RR 2284	2.8	Willcross	S	R
RR 2295 N	2.9	Willcross	S	R

† www.vipsoybeans.org.
‡ S, susceptible; R, resistant; Seg, segregating. All other cultivars of the 850 were susceptible to both SMV strains.

Table 2. Reactions of Williams/Williams 82 isogenic lines to *Soybean mosaic virus* (SMV) strains G1 and G5.

Entry name	SMV† G1	SMV† G5	Genetic background‡
L78–379	R	R	*Rsv1* Williams(6) × PI 96983
L81–4420	R	R	*Rsv1*, *Rps1-k*, L78–379 × Williams 82
L83–542	R	R	*Rsv*? F3 from BC5 Williams(6) × Buffalo (PI 424131)
L83–551	R	R	*Rsv*? F3 from BC5 Williams(6) × Buffalo (PI 424131)
L84–2112	R	R	*Rsv1-m* Williams × (Williams(6) × Marshall)
L85–2308	R	S	*Rsv1-y*?, Williams(6) × Dorman
L86–1525	S	R	*Rsv3-h* Williams(6) × Hardee
L88–8431	R	N	*Rsv1-r* Williams(6) × Raiden (PI 360844)
L92–8151	R	N	*Rsv1-s* Williams(6) × SS 74185 (PI 486355)
L92–8580	R	N	*Rsv1-sk*? Williams(6) × Suweon97 (PI 483084)
L93–3327	R	R	*Rsv1-t* Williams(6) × Ogden
L96–1676	R	R	*Rsv*? Williams(6) × Buffalo (PI 424131)
L96–1680	R	S	*Rsv*? Williams(6) × Buffalo (PI 424131)
L96–1683	R	S	*Rsv*? Williams(6) × Buffalo (PI 424131)
L96–1687	R	R	*Rsv*? Williams(6) × Buffalo (PI 424131)
L99–7751	R	S	*Rsv*? Williams 82(6) × Kosamame (PI 171451)
L99–7761	R	S	*Rsv*? Williams 82(6) × Kosamame (PI 171451)
L00–2230	R	S	*Rsv*? Williams 82(6) × Sodendaizu (PI 229358)
L00–2232	R	S	*Rsv*? Williams 82(6) × Sodendaizu (PI 229358)

† R, resistant; S, susceptible; N, systemic necrosis reaction.
‡ Isogenic lines developed by R.L. Bernard (unpublished data, 2003). ? = unknown resistance gene or allele.

a more virulent strain based on a set of differentials (Cho and Goodman, 1979). These results were similar to those of an earlier study of SMV resistance in soybean ancestral lines where more lines were resistant to SMV-G5 than SMV-G1 (Wang et al., 2005). None of the 850 cultivars was resistant to both SMV-G1 and -G5, suggesting that the *Rsv1* allele from PI 96983 that gives resistance to both strains was not present in this group of cultivars.

Cultivars that were susceptible to G1 and resistant to G5 probably possess the *Rsv3* gene (Gunduz et al., 2001). The frequency of commercial cultivars with the *Rsv3* gene may be much higher than the frequency of commercial cultivars with the *Rsv1* allele from PI 96983.

Cultivars that were resistant to G1 and susceptible to G5 could have the *Rsv1-y* allele, (Chen et al., 1991). *Rsv1-y* and *Rpv1* (a *Peanut mottle virus* resistance gene) are closely linked, but distinct genes in York soybean (Roane et al., 1983). Dorman, a parent of York, is the likely donor of *Rsv1-y* in York (Zheng et al., 2005), as well as in Williams isogenic line L85–2308 (Table 2).

Based on the reactions of isogenic lines L83–542, L83–551, L96–1676, L96–1680, L96–1683, and L96–1687 to SMV-G1 and -G5, Buffalo may contain two SMV resistance genes because two different patterns of resistance were found among these lines. This result does not appear to be in agreement with the report of a single dominant gene in Buffalo (Bowers et al., 1992). However, an alternative explanation for the results could be recombination within the *Rsv1* locus to produce an alternate phenotype on SMV G5 inoculation, similar to the phenomenon described by Hayes et al. (2004).

The Williams isogenic line L92–8151 (Table 2) could contain either the *Rsv1-s* allele or *Rsv4* allele or both of them from SS 74185 (PI 486355; Ma et al., 1995). But the *Rsv4* allele in SS 74185 (PI 486355) was not transferred to this isogenic line, since *Rsv4* has resistance to all SMV strains, while L92–8151 when inoculated with SMV-G5

Table 3. Reactions to inoculation with *Soybean mosaic virus* (SMV) strains G1 and G5 of selected soybean cultivars and lines with reported or observed SMV resistance.

Entry name	SMV strain†		Genetic background‡	Reference
	G1	G5		
L97–946	R	R	*Rsv*?, L87–672 × Jack	R.L. Bernard (unpubl. data, 2003)
V97–9001	R	R	*Rsv4*, Essex (5) × PI 486355	G.R. Buss (pers. comm., 2005)
IA 3010	S	R	Jacques J285 × Northrup King S29–39	R.L. Nelson (pers. comm., 2005)
AG 4201	S	R	From the same cross as DKB 4651	C.K. Moots (pers. comm., 2005)
DKB 4651	S	R	From the same cross as AG 4201	C.K. Moots (pers. comm., 2005)
5601T	R	S	F6-derived line from Hutcheson × TN89–39	Pantalone et al. (2003)
N6201	R	S	F6-derived selection from Nakasennari × Young	Carter et al. (2003b)
N7001	R	R	F4-derived selection from N77–114 × PI 416937	Carter et al. (2003a)
N7101	R	S	F6-derived selection from Vance × Jizuka	Carter et al. (2003c)
N7102	R	S	F6-derived selection from Vance × Jizuka	Carter et al. (2003d)

† R, resistant; S, susceptible.
‡ ? = unknown resistance gene or allele.

reacted with systemic necrosis. This systemic necrosis is the expected *Rsv1-s* reaction to G5. Therefore *Rsv1-s* may have been transferred to L92–8151 from SS 74185 (PI 486355).

L92–8580, the isogenic line derived from Suweon 97, reacted to SMV G5 with systemic necrosis. Suweon 97 has been reported to be resistant to SMV G5 (Chen et al., 2002). Likewise, Suweon 97 plants inoculated in our laboratory were also resistant (Hobbs et al., unpublished data, 2005). A possible explanation for these differences between L92–8580 and Suweon 97 could be the recombination phenomenon described by Hayes et al. (2004).

Ten selected lines with reported or observed SMV resistance included lines resistant to both G1 and G5, resistant to G1 but not G5, and resistant to G5 but not G1. Of the three lines that were resistant to both strains, one had *Rsv4* resistance (V97–9001) and two (L97–946 and N7001) had resistance of uncertain origin.

SMV-G1 is widely used in SMV resistance screening programs (Roane et al., 1986). One disadvantage of using SMV-G1 alone in breeding and screening programs is that it cannot detect resistance controlled by *Rsv3*. Using both SMV-G1 and -G5 when screening provides a broader spectrum of resistance to SMV and should be considered when developing SMV resistance. Based on the low frequency of SMV resistance in commercial cultivars at the present time, there is an opportunity to increase this frequency in the future through backcrossing to resistant sources and molecular marker assisted breeding.

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