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Evaluation of Ancestral Lines of U.S. Soybean Cultivars for Resistance to Four Soybean Viruses

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ABSTRACT

Fifty-two North American (NA) ancestral soybean [*Glycine max* (L.) Merr.] lines were screened for resistance to *Bean pod mottle virus* (BPMV), *Soybean mosaic virus* (SMV) strains G1 and G5, *Tobacco ringspot virus* (TRSV), and *Tobacco streak virus* (TSV). Seven ancestors, 'CNS', 'Haberlandt', 'Ogden', 'Peking', PI 71506, PI 88788, and 'Tokyo', were resistant to SMV-G1. Sixteen entries, 'A.K. (Harrow)', 'Capital', CNS, FC 33243, Haberlandt, 'Illini', 'Improved Pelican', 'Laredo', 'Lincoln', 'Mandarin', 'Mandarin (Ottawa)', Ogden, 'Palmetto', Peking, PI 88788, and Tokyo were resistant to SMV-G5. All ancestral lines tested were susceptible to BPMV and TRSV. Only one ancestor, 'Tanner', was resistant to TSV. On the basis of cultivar registration articles through 2002, there were 15 public soybean cultivars with reported resistance to SMV. The possible donors of resistance for each were identified. Two soybean ancestors, CNS and Ogden, were the most important possible sources of SMV resistance genes in U.S. commercial soybean cultivars, as the pedigree of 75 and 56% of the reported resistant cultivars contained CNS and Ogden, respectively. In most of the cultivar registration articles, reactions to SMV were not reported. With the relatively high frequency of SMV resistance in major ancestral lines, SMV resistance in U.S. cultivars may be more common than expected.

Soybean viral diseases have become increasingly common and economically important in the USA in recent years (Tolin, 1999). The increase in virus vectors is likely to exacerbate the problem in the future (Giesler et al., 2002; Hartman et al., 2001a; Mabry et al., 2003). In Illinois, BPMV and SMV cause the two most common viral diseases in soybean fields, with TRSV and TSV found less frequently (Hartman et al., 2001b). In addition to direct yield losses due to virus infection, it is also known that some viruses, like BPMV and SMV alone or in combination, impact seed appearance by causing seed coat mottling (Hobbs et al., 2003).

Bean pod mottle virus is a beetle-transmitted virus that causes leaf mottling, mosaic, and leaf distortion on soybean (Gergerich, 1999). This virus was first reported in soybean in Arkansas, North Carolina, and Virginia in 1958 (Gergerich, 1999). It has spread throughout the soybean-growing areas in the USA (Ghabrial et al., 1990; Mabry et al., 2003; Milbrath et al., 1975; Pitre et al., 1979), causing yield losses of 10 to 52% (Hopkins and Mueller, 1984; Horn et al., 1973; Myhre et al., 1973; Quinones et al., 1971; Ross, 1968).

Soybean mosaic virus is an aphid-transmitted virus that occurs in all soybean production areas of the world (Hill, 1999). Leaves of SMV-infected plants often appear mottled and distorted, and yield losses of 8 to 35% have been reported (Hill, 1999; Quinones et al., 1971; Ross, 1968). Cho and Goodman (1979) defined SMV strains G1 through G7 using reactions on a set of soybean differentials. *Soybean mosaic virus* strain G1 was the most common strain identified when 98 SMV isolates were tested from a worldwide seed collection (Cho and Goodman 1979), and it has been shown to have a high rate of seed transmission (Bowers and Goodman, 1991). *Soybean mosaic virus* G5 has been shown to cause severe reduction in weight of infected plants and seed yields (Tu, 1989). All strains, including G1 and G5, cause seed mottling (Bowers and Goodman, 1991).

Tobacco ringspot virus was first described in the USA in 1941 (Samson, 1942). Bud blight is the most severe symptom of TRSV infection in soybean, but vein necrosis, flower and pod abortion, and stunting also can be common. This disease was first reported in the U.S. Midwest in 1946 (Allington, 1946). Yields may be reduced by 25 to 100%, with the greatest losses occurring when young plants are infected (Demski et al., 1999a).

Tobacco streak virus causes Brazilian bud blight of soybean, and was first reported in Brazil in 1950 (Demski et al., 1999b) and was first reported in soybean in the USA in Iowa (Fagbenle and Ford, 1970) and subsequently has been found in several other states. *Tobacco streak virus* was found in 2002 in a soybean field at the Crop Science Research and Education Center (CSREC), University of Illinois, Urbana, IL (G.L. Hartman, 2002, unpublished data) and causes similar symptoms on soybean to TRSV, including bud necrosis.

Three known loci, Rsv_1, Rsv_3 and Rsv_4, confer resistance to SMV (Palmer et al., 2004). Even though BPMV and TSV can cause severe damage to soybean, no resistance to either virus has been reported in *G. max*, although a mild symptom reaction to BPMV has been reported in the cultivar Semmes (Ross, 1986), and resistance to BPMV has been reported in other *Glycine* species (Scott et al., 1974; Zheng et al., 2003). Resistance to TRSV has been reported in *G. max* PI 92713 and PI 154194 (Demski et al., 1999a) and in *G. soja* PI 407287 (Orellana, 1981).

Soybean cultivars grown in NA have a narrow genetic base. Gizlice et al. (1994) identified a group of 35 soybean introductions and first progeny cultivars that con-

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Abbreviations: BPMV, *Bean pod mottle virus*; CSREC, Crop Science Research and Education Center; ELISA, enzyme-linked immunosorbent assay; NA, North American or North America; SMV, *Soybean mosaic virus*; TRSV, *Tobacco ringspot virus*; TSV, *Tobacco streak virus*.

tributed 95% of the genes, and 91 accessions that contributed 99% of the genes in modern (1947–1988) NA public soybean cultivars. The objectives of this study were: (i) to screen the major ancestral lines of NA soybean cultivars for resistance to BPMV, SMV strains G1 and G5, TRSV, and TSV; and (ii) to check registration articles for all public cultivars released through 2002, record pedigree information for all cultivars cited as being resistant, and evaluate the responses to infection by SMV strains G1 and G5 of selected public cultivars that were reported to be SMV-resistant.

MATERIALS AND METHODS

Soybean Lines

Fifty-two soybean accessions (Table 1) were selected to represent the major ancestral lines of NA cultivars as identified by Gizlice et al. (1994). 'Williams 82' was used as the susceptible check.

Registration articles were examined for all public cultivars released through 2002. Pedigree information was recorded for all cultivars cited as being BPMV, SMV, TRSV, or TSV-resistant. Fifteen public cultivars identified as SMV-resistant in the literature and the privately developed cultivar 'Marshall', which has been used as a differential type to distinguish among SMV strains were planted and inoculated with SMV strains G1 and G5 in the greenhouse.

All soybean seeds were obtained from the USDA Soybean Germplasm Collection in Urbana, IL.

Virus Isolates

Bean pod mottle virus isolate 98 was collected from infected soybean at the CSREC, Urbana, IL, and was maintained by continuous transfer and stored long term in desiccated refrigerated leaves. It belongs to subgroup I (Gu et al., 2002). *Soybean mosaic virus* strains G1 and G5 were obtained from J. Hill, Iowa State University, and maintained by continuous greenhouse transfer and stored long term in lyophilized leaves. Classifications of isolates as G1 and G5 were confirmed on a set of soybean differentials (Cho and Goodman, 1979). The TRSV-grape isolate was obtained from the American Type Culture Collection (Manassas, VA), maintained by continuous greenhouse transfer, and stored long term in fresh leaf material frozen at −80°C. The TSV-UIUC isolate, which was identified as TSV by positive enzyme-linked immunosorbent assay (ELISA) reaction using TSV-specific antibodies, was collected from infected soybean at the CSREC and maintained by continuous greenhouse transfer.

Field Cage and Greenhouse Experiments

Screening for SMV-G1 resistance in ancestral germplasm was conducted from 1992 to 1994. Screening ancestral lines for resistance to the other viruses in this study was done in 2002 and 2003. Screening for resistance to SMV-G1 and G5 in public cultivars reported to have SMV resistance was also done in 2002 and 2003. Screening from 1992 to 1994 was completed at CSREC, Urbana, IL, in the field during the growing season and in a greenhouse during the winter. All screening completed in 2002 and 2003 was done in a greenhouse.

Field experiments were completed inside a 13- by 19-m cage covered with a nylon fabric (mesh size of 12 openings cm^{-1}) to exclude insect vectors. Each tested line was planted in hills with 12 seeds per hill, and were placed 40 cm apart in all directions. Hills were thinned to eight plants after emergence.

In the greenhouse, eight seeds of each line tested were planted in a pasteurized soil mixture (1:1:1 soil–sand–peat) in plastic flats (52- by 37-cm) or in 10-cm plastic pots with a soilless mix (Sunshine Mix LC1, Sun Gro Horticulture, Inc., Bellevue, WA) and covered with coarse vermiculite. All entries without symptoms were retested using five to six plants.

Inoculum consisted of extracts from infected leaves of Williams 82 plants maintained in the greenhouse that were prepared by homogenizing infected leaves in chilled 0.025 *M* KPO_4 buffer, pH 7.1, plus 0.01 *M* sodium sulfite with sterilized pestles and mortars. Pestles were used to apply inoculum to carborundum-dusted leaf surfaces. Plants were inoculated with

Table 1. Reactions of 52 U.S. soybean ancestral lines to inoculation with two strains of *Soybean mosaic virus* (SMV), *Bean pod mottle virus* (BPMV), *Tobacco ringspot virus* (TRSV), and *Tobacco streak virus* (TSV).

	SMV				
Entry	G1	G5	BPMV	TRSV	TSV
'A.K. (Harrow)'	S†	R	S	S	S
'Arksoy'	S	S	S	S	S
'Bansei'	S	S	S	S	S
'Biloxi'	S	S	S	S	S
'Capital'	S	R	S	S	S
'CNS'	R	R	S	S	S
'Dunfield'	S	S	S	S	S
FC 31745	S	S	S	S	S
FC 33243	S	R	S	S	S
'Flambeau'	S	S	S	S	S
'Haberlandt'	R	R	S	S	S
'Illini'	S	R	S	S	S
'Improved Pelican'	S	R	S	S	S
'Jackson'	S	S	S	S	S
'Jogun'	S	S	S	S	S
'Kanro'	S	S	S	S	S
'Korean'	S	S	S	S	S
'Laredo'	S	R	S	S	S
'Lincoln'	S	R	S	S	S
'Macoupin'	S	S	S	S	S
'Mammoth Yellow'	S	S	S	S	S
'Manchu'	S	S	S	S	S
'Mandarin'	S	R	S	S	S
'Mandarin (Ottawa)'	S	R	S	S	S
'Manitoba Brown'	S	S	S	S	S
'Mansoy'	S	S	S	S	S
'Mukden'	S	S	S	S	S
'Ogden'	R	R	S	S	S
'Palmetto'	S	R	S	S	S
'Patoka'	S	S	S	S	S
'Peking'	R	R	S	S	S
'Perry'	S	S	S	S	S
PI 54610	S	S	S	S	S
PI 54610-1	S	S	S	S	S
PI 54610-4	S	S	S	S	S
PI 71506	R	S	S	S	S
PI 80837	S	S	S	S	S
PI 81041	S	S	S	S	S
PI 88788	R	R	S	S	S
PI 180501	S	S	S	S	S
PI 200492	S	S	S	S	S
PI 240664	S	S	S	S	S
PI 360955B	S	S	S	S	S
PI 438471	S	S	S	S	S
PI 438477	S	S	S	S	S
'Ralsoy'	S	S	S	S	S
'Richland'	S	S	S	S	S
'Roanoke'	S	S	S	S	S
'S-100'	S	S	S	S	S
'T109'	S	S	S	S	S
'Tanner'	S	S	S	S	R
'Tokyo'	R	R	S	S	S

† S = susceptible, R = resistant. Classification based on virus symptoms and ELISA reaction: susceptible = with symptoms and positive ELISA reactions; resistant = symptomless and with negative ELISA reactions.

Table 2. Cultivars reported to be *Soybean mosaic virus* (SMV) resistant, reactions of those cultivars to SMV G1 and G5 strains of soybean mosaic virus, and SMV-resistant ancestors.†

Cultivar (MG)	SMV-resistant ancestors	G1	G5
Bay (V)	'CNS', 'Haberlandt', and 'Ogden'	S	S
Brim (VI)	'CNS', 'Haberlandt', and 'Ogden'	R	S
CF492 (IV)	'CNS', 'Lincoln', 'Ogden', and PI 71506	R	S
Clifford (V)	'CNS', 'Ogden', and PI 88788	R	R
Colfax (II)	Pedigree unknown	R	S
Dunbar (III)	'Ogden'	S	S
Epps (V)	'CNS', 'Peking', and PI 88788	R	R
Fremont (III)	'Illini' and 'Mandarin (Ottawa)'	S	S
Holladay (V)	'CNS', 'Haberlandt', and 'Ogden'	R	R
Hutcheson (V)	'CNS', 'Ogden', and PI 71506	R	S
Marshall (II)	All reported ancestors susceptible to SMV	R	R
Prichard (VIII)	'CNS', 'Ogden', 'Haberlandt', 'Peking', 'Tokyo', PI 88788, and 'Improved Pelican'	R	R
Sherman (III)	'CNS', 'Illini', and 'Lincoln'	S	S
Toano (V)	'CNS' and 'Haberlandt'	R	S
Ware (IV)	'CNS' and 'Haberlandt'	R	S
Young (VI)	'CNS' and 'Ogden'	R	S

† MG = Maturity Group; S = susceptible; R = resistant. Classification based on virus symptoms and ELISA reaction: susceptible = with symptoms and positive ELISA reactions; resistant = symptomless and with negative ELISA reactions.

BPMV, SMV, TRSV, and TSV 7 to 10 d after planting at growth stage V1 (Fehr and Caviness, 1977). Two to three weeks after inoculation, trifoliolate leaves were examined for systemic virus symptoms. Depending on the virus inoculated, susceptible reactions included leaf mosaic and distortion, stunting and bud necrosis. Resistance reactions were recorded when plants were symptomless. In cases where it was difficult to distinguish between mild and no symptoms, ELISA results were used to determine the reaction.

Enzyme-Linked Immunosorbent Assay Evaluation

Three weeks after inoculation, trifoliolate leaf samples were tested for the presence of BPMV, SMV, TRSV, and TSV by double antibody sandwich ELISA (Clark and Adams, 1977) using Agdia antibodies and protocol (Agdia, Inc., Elkhart, IN). At least three plants of each virus–soybean type combination were tested by ELISA to verify phenotypic observations. Sample wells with absorbances at 405 nm wavelength more than two times those of the control wells were considered positive reactions and indicated presence of virus in the tissue extract. Lines with a negative ELISA reaction and no systemic symptoms were considered virus resistant.

RESULTS

Evaluation of Ancestral Lines for Resistance to BPMV, SMV, TRSV, and TSV

All ancestral lines tested were susceptible to the strain of BPMV used in this research (Table 1). Symptoms caused by BPMV included mottling or mosaic on leaves, and slight to moderate leaf distortion. Infection of inoculated plants was confirmed by positive ELISA reactions.

Two to three weeks after inoculation, with either SMV-G1 or G5, susceptible Williams 82 plants grown in the field cage or greenhouse had mosaic symptoms and were ELISA-positive. Seven ancestors, CNS, Haberlandt, Ogden, Peking, PI 71506, PI 88788, and Tokyo, displayed no systemic symptoms and were ELISA-negative when challenged with SMV-G1 (Table 1), while the remainder of the ancestors had systemic symptoms and were ELISA-positive. Retesting these seven entries confirmed resistance to SMV G1.

Sixteen ancestral types, A.K. (Harrow), Capital, CNS, FC 33243, Haberlandt, Illini, Improved Pelican, Laredo, Lincoln, Mandarin, Mandarin (Ottawa), Ogden, Palmetto, Peking, PI 88788, and Tokyo had no systemic symptoms and were ELISA-negative when inoculated with SMV-G5 (Table 1). All other entries had systemic symptoms and were ELISA-positive. Putative resistant lines were inoculated in a second SMV-G5 test, and lack of systemic symptoms and negative ELISA tests confirmed resistance to SMV-G5. Of these, Ogden and Tokyo were previously reported resistant to SMV (Kiihl and Hartwig, 1979). Tokyo is the source of the Rsv_1^t allele in Ogden which conditions resistance to both SMV-G1 and G5. Systemic necrosis, which occurs in some SMV isolate–entry combinations, was not observed during the course of this study.

None of the ancestral lines were resistant to the TRSV-grape isolate (Table 1). All inoculated plants were ELISA-positive for TRSV. Symptoms caused by TRSV included stunted plants, distorted leaves, and bud blight, which consisted of a curving of the terminal bud that later became necrotic.

Only Tanner was resistant to the TSV-UIUC isolate (Table 1). Repeated testing of Tanner confirmed this resistance. No systemic virus symptoms occurred in Tanner, and leaves were ELISA-negative. Symptoms caused by TSV in the other lines included chlorotic spots on leaves, leaf distortion, and bud blight. All susceptible lines tested positive for TSV by ELISA.

Analysis of Pedigrees of SMV-Resistant Public Cultivars

Soybean mosaic virus resistant ancestors contained within the pedigrees for 16 public soybean cultivars reported to be resistant to SMV are shown in Table 2. It was not possible to deduce the sources of SMV resistance in the resistant cultivar Colfax (Graef et al., 1994) because it was derived from an intermated population. None of the reported ancestors of Marshall (W. Ellingson, 1993, personal communication) were found to be resistant to SMV (Wang, 1996). The resistance locus Rsv_1 was first identified in PI 96983 (Kiihl and Hartwig, 1979) and the allele in PI 96983 was shown to be resistant to SMV-G1 through G6 (Cho and Goodman, 1982). PI 96983 was not included in the ancestral lines that we tested although it is in the pedigree of 'Epps' and is the reported source of resistance to SMV (Buss et al., 1988b).

Evaluation of the Responses of Public Cultivars to SMV G1 and G5

There was information in the literature on SMV reaction for only 49 of the more than 500 public cultivars, with 34 rated susceptible and 15 rated resistant (Table 2). Of these 15 cultivars reported to have SMV resistance, 'Bay', 'Dunbar', 'Fremont', and 'Sherman' were susceptible to G1. All were susceptible to G5 except 'Clifford', Epps, 'Holladay', and 'Prichard' (Table 2). Marshall, a privately developed cultivar, was also tested and found to be resistant to both G1 and G5. None of the cultivars were reported to be resistant to

BPMV, TRSV or TSV, except the cultivar 'Semmes', which was reported to have a mild symptom reaction to BPMV (Ross, 1986). Semmes had been tested previously by us for BPMV resistance, and had visible symptoms as had been reported by Ross (1986). In this study, we did not distinguish between reactions of moderate resistance, moderate susceptibility, or susceptibility.

DISCUSSION

Among the tested ancestral lines, resistance to SMV-G5 (Cho and Goodman, 1979) was more common than resistance to SMV-G1. Conversely, among the cultivars tested that were reported to have SMV resistance, resistance to SMV G1 was more common than resistance to SMV-G5. In our test using two SMV strains, the cultivars Bay (Buss et al., 1979a), Dunbar (Graef et al., 1992), Fremont (Williams et al., 1986), and Sherman (McBlain et al., 1987) were not resistant to either strain. In their registration articles, Bay was reported to be resistant to some unidentified strains of SMV, and Dunbar was reported to be resistant to SMV (no specific strains mentioned). Differences in results between those references and our research may be due to different SMV strains used. In registration articles, Fremont was reported to be moderately resistant to SMV, and Sherman was reported to have moderate to high resistance to SMV. Moderate resistance or moderate susceptibility and susceptibility were not distinguished in our study which also may result in some differences.

Knowledge of sources of resistance to SMV is helpful in selecting parents to use in resistance breeding programs. In most of the cultivar registration articles, reactions to SMV are not reported. With the relatively high frequency of SMV resistance in major ancestral lines, SMV resistance in U.S. cultivars may be more common than expected. On the basis of results in this study, the following deductions about the possible donors of SMV resistance in public cultivars can be made.

The pedigrees of 'Brim' (Burton et al., 1994) and 'Holladay' (Burton et al., 1996) share the same SMV-resistant ancestors (CNS, Haberlandt, and Ogden). The parents of Brim were 'Young' × N73-1102. Our data indicated that Young was resistant to SMV-G1 (Table 2). The parents of N73-1102 were 'Tracy' (SMV-susceptible, data not shown) and 'Ransom' (SMV-resistant, data not shown) indicating that Haberlandt, an ancestor of Ransom, was also a potential source of SMV resistance in Brim. Brim was resistant to SMV G1, and Holladay was resistant to both SMV G1 and G5. Ogden carries the Rsv_1^t allele that conditions resistance to both SMV-G1 and G5, so it is possible that this allele also exists in Holladay. None of the three resistant ancestors of Brim were resistant to only SMV-G1. The parents of 'Toano' (Buss et al., 1987) were 'Ware' and 'Essex'. Since Essex is susceptible to SMV (Chen et al., 1991), the resistance probably came from Ware (Buss et al., 1979b). We found both Toano and Ware to be resistant to only SMV-G1, and CNS and Haberlandt were the only resistant ancestors of these two cultivars. The genetics of SMV resistance in CNS and Haberlandt have not been studied, so it is possible that at least one of those lines carries resistance alleles at two loci and that only one allele was transferred to Brim, Toano, and Ware. The resistance in Young (Burton et al., 1987) may have been derived from either CNS or Ogden. Young was resistant to SMV-G1 only, so it does not have Rsv_1^t. This would indicate that CNS had two alleles and that only the allele conditioning resistance to SMV-G1 was transferred to Young.

'CF492' (Pfeiffer et al., 1996) may have received its resistance from Ogden, CNS, PI 71506, or Lincoln. The resistant cultivar 'Hutcheson' (Buss et al., 1988a) had the same resistant ancestors except for Lincoln. Since Lincoln was resistant to SMV-G5 and CF492 was susceptible to SMV-G5, the resistance in CF492 was not likely from Lincoln. Neither CF492 nor Hutcheson had the Rsv_1^t allele, since both were susceptible to SMV G5. If CNS had multiple alleles for SMV resistance, both CF492 and Hutcheson could have a single allele from CNS, but it may be more likely that both CF492 and Hutcheson received resistance from PI 71506, since all three were only resistant to SMV-G1. Clifford (Burton et al., 1997) and 'Epps' (Hartwig, 1984) were both resistant to both SMV-G1 and G5. The pedigree of Clifford contained the resistant ancestors CNS, PI 88788, and Ogden, all of which were resistant to both SMV-G1 and G5 and could be possible donors of resistance. Any of the SMV-resistant ancestors of Epps provide resistance to both SMV-G1 and G5, but PI96983 was specifically selected as a parent to provide SMV resistance (Hartwig, 1984) so it is the most likely donor of resistance to Epps (Buss et al., 1988b).

Prichard (Boerma et al., 2001) was derived from 'Co82-622' × 'Howard'. Co82-622 was derived from 'Braxton' × 'Coker 368'. There are seven SMV-resistant ancestors (CNS, Ogden, Haberlandt, Peking, Tokyo, PI 88788, and Improved Pelican) in the pedigree of Prichard. Prichard and all but Improved Pelican are resistant to both G1 and G5, so the resistance in Prichard could have come from several sources.

Marshall has been frequently cited as a source of resistance to SMV and was used as one of the differential cultivars to identify SMV strains (Chen et al., 1991; Cho and Goodman, 1979). Marshall was derived from Provar × (A55-5629-4 × PI 248404) (W. Ellingson, 1993, personal communication). The progenitors of Provar and A55-5629-4 are susceptible to SMV and PI 248404 was susceptible to SMV-G1 in our tests (Wang, 1996). Marshall was reported carrying the Rsv_1^m allele (Chen et al., 1991), but the source of this allele is unknown. It is possible that Rsv_1^m arose from a mutation or perhaps there is an error in the reported pedigree of Marshall. The resistant cultivar Colfax (Graef et al., 1994) is derived from an intermated population, so the exact pedigree and potential sources of SMV resistance are unknown.

The soybean ancestors CNS and Ogden were the most common SMV-resistant ancestors of the NA public soybean cultivars. Nearly 80% of the public cultivars reported to be resistant to SMV had CNS, and nearly 60% had Ogden as an ancestor. CNS was a major ancestor,

contributing 9% of the genes to modern NA cultivars, and nearly 25% of the genes in southern adapted U.S. cultivars (Gizlice et al., 1994). Lincoln and Mandarin (Ottawa) were also major ancestral lines, contributing 18 and 12% of the genes to NA cultivars, respectively, and 24 and 17% of the genes to northern cultivars, respectively. Peking was one of the first known sources of resistance to SCN (Ross and Brim, 1957) and PI 88788 is also resistant to SCN (Epps and Hartwig, 1972); Peking and PI 88788 have been reported to contain Rsv_4 (Gunduz et al., 2004). The map location of the Rsv_4 locus occurs on molecular linkage group "D1b," a region of the genome that does not map to any SCN resistance or any other resistance genes (Hayes et al., 2000).

Our deductions were based only on the public cultivars that were described as resistant to SMV when they were released. There was no information on the SMV reactions for most cultivars, and given the relatively high number of major ancestral lines that are resistant, it is possible that there are additional cultivars that are SMV-resistant. It is also possible that resistance was lost via genetic drift due to lack of selection pressure for SMV resistance.

Several possible new sources of SMV resistance were found in this research. Further study is needed to determine the genetics of these resistance sources. Most SMV resistance alleles reported to date exist at the Rsv_1 locus, and developing cultivars with multigenic resistance will depend on identifying new resistance loci. Ancestral lines A.K. (Harrow), Capital, FC 33243, Illini, Improved Pelican, Laredo, Lincoln, Mandarin, Mandarin (Ottawa), and Palmetto may carry the Rsv_3 allele, since their reaction patterns to SMV strains G1 and G5 were identical to the patterns of soybean germplasm lines with Rsv_3 reported by Gunduz et al. (2001). The ancestor PI 71506 may carry a gene similar to Rsv_1^y in the cultivar York, since York had the same reaction pattern to SMV strains G1 and G5 (Chen et al., 1994).

Resistance was not found to BPMV and TRSV in the set of ancestral lines tested, although resistance has been reported to TRSV in two PI lines (Demski et al., 1999a). Tanner, a relatively minor ancestral type, was the only type found resistant to TSV. It would seem reasonable to conclude that given the rarity or absence of resistance to these three viruses in the soybean ancestral lines, that resistance would not occur in U.S. soybean cultivars.

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