Genetic and Phenotypic Analysis of *Soybean mosaic virus* Resistance in PI 88788 Soybean

Irfan Gunduz, Glenn R. Buss, Pengyin Chen, and Sue A. Tolin

First, second, and third authors: Department of Crop and Soil Environmental Sciences, Virginia Polytechnic Institute and State University, Blacksburg 24061; and fourth author: Department of Plant Pathology, Physiology, and Weed Science, Virginia Polytechnic Institute and State University, Blacksburg 24061.

Current address of P. Chen: Department of Crop, Soil, and Environmental Sciences, University of Arkansas, Fayetteville 72701.

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ABSTRACT

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Resistance to *Soybean mosaic virus* (SMV) was identified in PI 88788 soybean, a germ plasm accession from China that is used widely as a source of resistance to soybean cyst nematode. Strains SMV-G1 through -G7 infected the inoculated leaves of PI 88788 but were not detected in upper, noninoculated trifoliolate leaves. Inheritance of resistance was determined by inoculating progenies of crosses of PI 88788 with susceptible cvs. Essex and Lee 68 with SMV strains G1 and G7. Allelomorphous relationships with known genes for resistance to SMV were tested in crosses with the resistant genotypes PI 96983, L29, and V94-5152, possessing

Rsv1, *Rsv3*, and *Rsv4* genes, respectively. Data analyses showed that resistance in PI 88788 to SMV-G1 is controlled by a single, partially dominant gene; however, to SMV-G7, the same gene was completely dominant. The PI 88788 gene was independent of the *Rsv1* and *Rsv3* loci, but allelic to *Rsv4* in V94-5152. Expression of the *Rsv4* gene in PI 88788 resulted in a reduced number of infection sites and restricted short- and long-distance movement of virus, rather than hypersensitivity. A unique late susceptible phenotype was strongly associated with heterozygosity. This gene has potential value for use in gene pyramiding to achieve resistance to several SMV strains, as well as for rate-reducing resistance.

Additional keywords: broad resistance, *Glycine max*, late susceptibility, partial dominance.

Soybean mosaic virus (SMV; genus *Potyvirus*, family *Potyviridae*) is one of the most common viral diseases of soybean (*Glycine max* (L.) Merr.) and can cause severe yield losses (13, 21). Controlling crop losses depends on utilization of resistant cultivars because no other effective way of managing the disease currently is available. To date, three SMV-resistant loci, *Rsv1*, *Rsv3*, and *Rsv4*, have been named. Resistance genes at these loci confer differential reactions to SMV strains G1 through G7 as defined by Cho and Goodman (14,15). A single dominant gene for SMV resistance was identified in PI 96983 and designated as *Rsv* (later renamed as *Rsv1*) (9,18). *Rsv1* has been mapped to soybean molecular linkage group "F" in a cluster of resistance (*R*) genes (36,37). The symbol *Rsv2* previously was assigned to the gene in OX670, which presumably was derived from Raiden (6). However, we have shown that OX670 carries two genes; one is derived from Raiden and is allelic to the *Rsv1* locus while the other is derived from Harosoy and allelic to the *Rsv3* locus (17). A unique *Rsv2* locus does not appear to exist in OX670 or its ancestors; thus, the gene symbol *Rsv1-r* was assigned to the SMV *R* gene in Raiden (11). A single dominant gene derived from Columbia and conditioning systemic necrosis to SMV-G1 and -G4 was found to be independent of *Rsv1* and was designated as *Rsv3* (7). *Rsv3* has been mapped to molecular linkage group "B2" (23). L29, an isoline of Williams with SMV resistance derived from Hardee, also has been shown to contain *Rsv3* (5). A new resistance gene independent of *Rsv1* and *Rsv3* has been reported in PI 486355 (24). This new gene, labeled *Rsv4* (20), was isolated in V94-5152,

a selection from PI 486355 × 'Essex' (24), and registered as germ plasm (4). The *Rsv4* locus has been mapped to soybean molecular linkage group "D1b", a region of the genome with no other mapped resistance genes (20). Columbia also has been found to carry a resistance gene at an independent locus, R4, which conditions early seedling resistance to SMV-G1 and -G7 and delayed systemic, or late-susceptible, symptoms (25).

Many of the SMV resistance genes discovered to date are at the *Rsv1* locus. However, several of them are alleles at this locus and exhibit resistance to lower-numbered strains of SMV, but either susceptibility or necrosis to higher-numbered strains (9,11,14). On the other hand, alleles at the *Rsv3* locus show a susceptible reaction to lower-numbered strains but a resistant reaction to higher-numbered strains (Table 1). PI 88788, an early soybean introduction from China, has been used widely as a source of soybean cyst nematode resistance. Two dominant genes and one recessive gene condition resistance to soybean cyst nematode in PI 88788 (29). We have recognized that it is also resistant to SMV, but the inheritance of resistance to SMV in PI 88788 has not been studied. The objective of this study was to determine (i) the reaction of PI 88788 to SMV strains G1 through G7, (ii) the inheritance of resistance to SMV in PI 88788, and (iii) the allelomorphous relationship of the resistance gene or genes in PI 88788 with previously identified resistance genes. We also demonstrate that the mechanism of action of this dominant gene is to reduce viral infection and accumulation and delay both local and vascular movement. A unique late-susceptible phenotype is described and is strongly associated with certain heterozygotes.

Corresponding author: S. A. Tolin; E-mail address: stolin@vt.edu

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MATERIALS AND METHODS

The response of PI 88788 to SMV strains G1 through G7 was tested, but only two strains, SMV-G1 and SMV-G7, were used to

screen F₁ plants, F₂ populations, and F_{2,3} families. These two strains induce differential responses in parental genotypes PI 96983 and L29, carrying *Rsv1* and *Rsv3*, respectively (Table 1). The sources of the virus strains, maintenance of the virus cultures, inoculation procedures, greenhouse conditions, and field plot techniques were as described previously (9).

PI 88788 was crossed with SMV-susceptible cvs. Lee 68 and Essex to study the inheritance of resistance. It also was crossed with resistant lines PI 96983 and L29, which contain *Rsv1* and *Rsv3*, respectively, as well as with V94-5152, which contains *Rsv4* (4,5,20,23). 'Peking', a plant introduction from China resistant to SMV (G. R. Buss, unpublished data) and carrying a recessive gene for resistance to *Peanut mottle virus* (32), also was crossed with PI 88788. Crosses were made either in the greenhouse or in the field at Blacksburg, VA. Crosses were distinguished from selfs in the F₁ and F₂ generation using leaf shape, seed color, flower color, pubescence color, and maturity as markers.

F₁ plants were grown in the greenhouse or in the field without SMV inoculation at Blacksburg, VA and harvested individually. F₂ plants were grown in the field without virus inoculation at either Blacksburg or Warsaw, VA and were harvested individually to obtain F_{2,3} families. Remnant F₁ seed, F₂ populations, and F_{2,3} families were tested with SMV-G1 in the greenhouse and field and with SMV-G7 in the greenhouse at Blacksburg, VA. An average of 5 F₁ plants, 200 F₂ plants per population, and 20 F_{2,3} plants from each of 50 F_{2,3} families per cross were inoculated. Individual plant reactions were examined at 10, 20, 30, and 40 days post-inoculation (dpi) and classified into four distinct phenotypes: resistant (R) (symptomless or with only local necrotic lesions on inoculated leaves), necrotic (N) (local and systemic necrosis that developed into stem tip necrosis and often plant death 10 to 15 dpi), susceptible (S) (mosaic, first trifoliolate showing vein clearing by 7 dpi, that developed into mosaic on all leaves), and late-susceptible (LS) (no symptoms until 14 to 21 dpi, then transitory chlorotic islands or rings appeared on trifoliolate leaflets). Plants showing systemic necrosis or late susceptibility in F₂ populations and F_{2,3} progenies were combined with resistant plants for testing fits to standard Mendelian ratios (10). The rationale for combining these phenotypes into the resistant genotype is based on our previous genetic analyses of the segregation of resistance at the *Rsv1* locus (9, 10) and the R3 and R4 (later called *Rsv3* and *Rsv4*) loci (17,24,25).

To confirm that the LS phenotype was a heritable trait, 200 F₂ plants from PI 88788 × Essex and PI 88788 × Lee 68 were planted in the virus nursery at Blacksburg, VA, and inoculated with SMV-G1. Susceptible, late-susceptible, and resistant plants were tagged and harvested to obtain F_{2,3} families, which then were planted in the greenhouse and inoculated with SMV-G1. Then, χ^2 tests were performed to determine the goodness-of-fit of the observed segregation to expected genetic ratios.

All plants with questionable symptoms, as well as 10 to 20 randomly selected susceptible, symptomless, and necrotic plants, were tested to confirm the presence or absence of SMV by tissue-blot immunoassays on nitrocellulose membranes (33). Invasion of inoculated leaves by SMV-G1 and -G7 was tracked by leaf print immunoassays of randomly selected leaves. Briefly, this was done

by imprinting leaves onto hardened filter paper (S&S 410 Filter Paper; Schleicher and Schuell, Keene, NH) (16), decolorizing with Triton X-100, and treating with polyclonal antibody to purified SMV, alkaline phosphatase-conjugated goat anti-rabbit immunoglobulin G (Sigma-Aldrich, St. Louis), and the substrate 5-bromo-4-chloro-3-indolyl phosphate and nitro tetrazolium salt (NBT/BCIP) (Zymed Laboratories, Inc., San Francisco).

RESULTS

Host phenotypic reactions. Susceptible cvs. Essex and Lee 68 exhibited typical veinal clearing in first trifoliolate leaves 6 to 8 dpi and misshapen and puckered trifoliolate leaves 15 to 35 dpi, typical of SMV symptoms (Fig. 1A and B). Leaf print immunoassay of SMV-G1-inoculated Essex unifoliolate leaves revealed 20 to 30 infection foci by 9 dpi and complete invasion of the inoculated leaf at a 23-dpi assay time (Fig. 2A and D). In contrast, PI 88788 exhibited no visible symptoms on unifoliolate leaves inoculated with SMV strains G1 through G7 or on any other leaves (Table 1). Only a few foci of infection could be detected by leaf print immunoassay of unifoliolate leaves inoculated with SMV strains G1 (Fig. 2B and E) and G7 (Fig. 2C and F). The area invaded by either G1 or G7 was barely detectable (<1 mm²) at 9 dpi (Fig. 2B and C) and enlarged to 2 to 3 mm² by 23 dpi (Fig. 2E and F). Leaves of PI 88788 sampled up to 42 dpi showed little increase in area invaded by the virus (data not shown). Neither macroscopically visible symptoms nor virus were detected on the upper leaves in the later growth stages.

The F₁ plants from the PI 88788 R × S crosses inoculated with G1 exhibited the LS phenotype (Table 2). The F₁ plants of PI 88788 × Essex exhibited mosaic symptoms, but not until 19 dpi. Later, predominant symptoms were chlorotic vein banding and chlorotic rings, forming green islands (Fig. 1C). Symptoms did not appear on PI 88788 × Lee 68 F₁ plants until 35 dpi, suggesting that the susceptible parent influences the susceptibility of the heterozygote. Several F₁ plants from the PI 88788 × Lee 68 cross also exhibited the LS phenotype upon inoculation with SMV strains G2, G3, G5, or G6 (data not shown). Symptoms developed by 14 dpi with G6, by 26 dpi with G5, and not until 35 dpi with G2 and G3. No symptoms developed following inoculation of either heterozygote with G7.

Inheritance of resistance to SMV-G1 and -G7 in PI 88788. The F₂ populations of PI 88788 × Essex and PI 88788 × Lee 68 segregated to fit a 3:1 R:S ratio based on the initial reaction to SMV-G1 at 7 to 10 dpi (data not shown). However, at 20 dpi, LS plants were observed in the F₂ populations from PI 88788 × Essex and PI 88788 × Lee 68. The number of LS plants increased in the 30- and 40-dpi readings. The segregation pattern, based on the 40-dpi reading of the PI 88788 (R) × Essex (S) F₂ population, was consistent with a 1:2:1 R:LS:S ratio (Table 2). The same population planted and inoculated in the field, as well as the PI 88788 × Lee 68, did not segregate in this ratio but did fit a 3:1 R+LS:S ratio.

Seed from eight randomly selected F₂ plants of PI 88788 × Essex, growing in the field and showing LS symptoms, were planted in the greenhouse and inoculated with SMV-G1. Segregation in

TABLE 1. Reaction of parental soybean genotypes to seven *Soybean mosaic virus* (SMV) strain groups

Parents (genotype)	SMV strains ^a						
	G1	G2	G3	G4	G5	G6	G7
Essex (rsv)	S	S	S	S	S	S	S
Lee 68 (rsv)	S	S	S	S	S	S	S
PI 96983 (<i>Rsv1</i>)	R	R	R	R	R	R	N
L29 (<i>Rsv3</i>)	S	S	S	S	R	R	R
V94-5152 (<i>Rsv4</i>)	R	R	R	R	R	R	R
PI 88788	R	R	R	R	R	R	R

^a R, resistant (symptomless); N, necrotic (systemic necrosis); S, susceptible (mosaic).

Fig. 1. Symptoms on soybean in the greenhouse at various days postinoculation (dpi) with *Soybean mosaic virus* strain G1. **A**, Unifoliate and first trifoliate leaves of 'Essex' (susceptible) at 7 dpi, showing local chlorotic lesions and vein clearing. **B**, Upper trifoliate leaves of Essex at 35 dpi, showing mosaic and leaf curl symptoms. **C**, Upper trifoliate leaves of heterozygous F_1 plants of PI 88788 (resistant) $\times$ Essex at 32 dpi, showing the partial resistance response of chlorotic vein banding and green islands.

the eight LS $F_{2.3}$ families of PI 88788 $\times$ Essex was consistent and homogeneous with a 3:1 R:S ratio at 9 dpi and 1:2:1 R:LS:S ratio at 41 dpi (Table 3).

Inoculation with SMV-G7 did not induce symptoms on F_1 plants of PI 88788 $\times$ Essex and PI 88788 $\times$ Lee 68 (Table 2). F_2 populations of the same crosses segregated in a 3:1 R:S ratio (Table 2). $F_{2.3}$ families of PI 88788 $\times$ Lee 68 and PI 88788 $\times$ Essex segregated in a 1:2:1 (all R:[3(R+LS):1S]:all S) phenotypic ratio when inoculated with SMV-G1 (Table 4). The $F_{2.3}$ population of PI 88788 $\times$ Essex segregated to fit a 1:2:1 (all R:[3R:1S]:all S) phenotypic ratio when inoculated with G7 (Table 4). All $F_{2.3}$ lines from PI 88788 $\times$ Essex exhibited the same reaction to both strains, except that LS plants were observed in the segregating lines inoculated with SMV-G1 but not with SMV-G7 (Tables 2 and 4).

Allelic relationship of the resistance gene in PI 88788 with *Rsv1*, *Rsv3*, and *Rsv4*. The F_2 population from PI 88788 (R) $\times$ PI 96983 (R, *Rsv1*) segregated to fit a digenic ratio of 15:1 R+LS+N:S when inoculated with SMV-G1 (Table 2). $F_{2.3}$ lines from the same cross exhibited a segregation ratio of 7:4:4:1 (all R:[15(R+LS+N):1S]:[3(R+N+LS):1S]:all S) (Table 5). Segregation in the F_2 population from L29 (R, *Rsv3*) $\times$ PI 88788 (R) was consistent with a digenic ratio of 15:1 R:S when inoculated with SMV-G7 (Table 2). $F_{2.3}$ families from the same cross showed a 7:4:4:1 (all R:[15R:1S]:[3R:1S]:all S) segregation ratio (Table 6). There were no susceptible plants identified in the F_2 and $F_{2.3}$ populations from V94-5152 (R, *Rsv4*) $\times$ PI 88788 (R) when inoculated with SMV-G1 (Tables 2 and 4). Progeny of crosses between Peking and PI 88788 also were all resistant (data not shown).

DISCUSSION

It is very clear from our data that the resistance gene in PI 88788 is independent of *Rsv1* and *Rsv3*. The data suggest that the single, dominant resistance gene in PI 88788 is allelic to the SMV resistance gene at the *Rsv4* locus in V94-5152. No phenotypic differences were observed in the expression of the two sources of *Rsv4*; therefore, it is likely that they are the same allele. The gene carried by Peking also appears to be at the *Rsv4* locus.

Rsv4 homozygotes (Fig. 2) and heterozygotes (data not shown) show a reduced number of initial virus infection sites compared with the Essex (*rsv4*) and Lee 68 (*rsv4*) parents, with both SMV strains G1 and G7. Vascular movement of SMV-G1 in homozy-

Fig. 2. Leaf print immunoassays of inoculated leaves of **A** and **D**, Essex and **B**, **C**, **E**, and **F**, PI 88788 at **A** to **C**, 9 and **D** to **F**, 23 days postinoculation with **A**, **B**, **D**, and **E**, *Soybean mosaic virus* (SMV) strain G1 or **C** and **F**, SMV-G7. Dark blue areas indicate presence of viral antigen.

gotes is severely restricted, but in heterozygotes it was merely delayed, indicating that the gene expresses partial dominance in response to SMV-G1. The delayed vascular movement resulted in symptoms referred to here as late susceptible (LS), and was typified by F₁ plants (heterozygote) of PI 88788 (R) × Essex (S) upon inoculation with SMV-G1. LS plants also were consistently observed in segregating populations of PI 88788(R) × Essex(S) and

PI 88788 (R) × Lee 68(S). Moreover, all F_{2.3} families derived from LS F₂ plants segregated for R, LS, and S phenotypes, and LS plants always were observed in segregating F_{2.3} progenies derived from R × S crosses. These observations suggest a strong association between heterozygosity and late susceptibility.

The association between late susceptibility and heterozygosity was not as strong in populations tested in the field, as seen in the

TABLE 2. Reactions of F₁ plants and F₂ soybean populations from crosses between PI 88788 and susceptible or resistant soybean genotypes, when inoculated with *Soybean mosaic virus* (SMV) strains G1 and G7

Cross, generation	SMV strain	Number of plants ^a				Expected ratio	χ^2	P
		R	N	LS	S			
PI 88788 (R) × Essex (S) F ₁	G1	0	0	10	0	...	...	...
PI 88788 (R) × Essex (S) F ₂	G1	86	0	151	67	1R:2LS:1S	2.39	0.30
PI 88788 (R) × Essex (S) F ₂ ^b	G1	41	0	24	16	3(R+LS):1S	1.19	0.27
PI 88788 (R) × Essex (S) F ₁	G7	4	0	0	0			
PI 88788 (R) × Essex (S) F ₂	G7	210	0	0	56	3R:1S	2.21	0.14
PI 88788 (R) × Lee 68 (S) F ₁	G1	0	0	7	0			
PI 88788 (R) × Lee 68 (S) F ₂	G1	90	0	45	35	3(R+LS):1S	1.76	0.18
PI 88788 (R) × Lee 68 (S) F ₂ ^b	G1	55	0	16	22	3(R+LS):1S	0.09	0.76
PI 88788 (R) × Lee 68 (S) F ₁	G7	4	0	0	0			
PI 88788 (R) × Lee 68 (S) F ₂	G7	156	0	0	45	3R:1S	0.73	0.39
PI 88788 (R) × PI 96983 (<i>Rsv1</i>) F ₂	G1	262	4	36	26	15(R+N+LS):1S	1.57	0.21
L29 (<i>Rsv3</i>) × PI 88788 (R) F ₂	G7	272	0	0	25	15R:1S	2.38	0.12
V94-5152 (<i>Rsv4</i>) × PI 88788 (R) F ₂	G1	325	0	0	0	...	...	...

^a R, resistant (symptomless); S, susceptible (mosaic); LS, late susceptible; N, necrotic.

^b Data taken from field.

TABLE 3. Reaction of F_{2.3} families of soybean from late-susceptible F₂ plants of PI 88788 (R) × Essex (S) when inoculated with *Soybean mosaic virus* strain G1 in the greenhouse^a

F _{2,3} family	No. of plants	9 dpi					41 dpi					
		R	S	$\chi^2_{3:1}$	df	P	R	LS	S	$\chi^2_{1:2:1}$	df	P
1	15	14	1	2.69	1	0.10	4	10	1	2.87	2	0.24
2	11	7	4	0.76	1	0.39	0	7	4	3.73	2	0.15
3	14	10	4	0.09	1	0.77	3	7	4	0.14	2	0.93
4	17	14	3	0.49	1	0.48	4	10	3	0.65	2	0.73
5	16	13	3	0.33	1	0.57	2	11	3	2.37	2	0.31
6	15	11	4	0.02	1	0.89	2	9	4	1.13	2	0.57
7	16	10	6	1.33	1	0.25	6	4	6	4.00	2	0.14
8	13	9	4	0.23	1	0.63	4	5	4	0.69	2	0.71
Total	117	...	...	5.920	8	...	...	...	...	15.65	16	...
Pooled	...	88	29	0.003	1	0.96	25	63	29	0.97	2	0.62
Heterogeneity	...	...	...	5.917	7	0.55	...	...	...	14.6	14	0.41

^a dpi, days postinoculation; R, resistant (symptomless); LS, late susceptible; S, susceptible; df, degrees of freedom.

TABLE 4. Segregation of F_{2.3} families of soybean derived from the crosses of PI 88788 with susceptible and resistant parents when inoculated with *Soybean mosaic virus* (SMV) strains G1 and G7 in the greenhouse

Cross, parents	SMV strain	Number of families ^a			Expected ratio	χ^2	P
		All R	H	All S			
PI 88788 (R) × Essex (S)	G1	11	24	7	1(all R):2[3(R+LS):1S]:1(all S)	1.62	0.45
PI 88788 (R) × Essex (S)	G7	11	24	7	1(all R):2(3R:1S):1(all S)	1.62	0.45
PI 88788 (R) × Lee 68 (S)	G1	8	17	10	1(all R):2[3(R+LS):1S]:1(all S)	0.26	0.88
PI 88788 (R) × V94-5152 (<i>Rsv4</i>)	G1	46	0	0	...	...	...

^a R, resistant; H, segregating; S, susceptible.

TABLE 5. Segregation of F_{2.3} families of soybean derived from PI 88788 (R) × PI 96983 (R) when inoculated with *Soybean mosaic virus* strain G1 in the greenhouse

Reaction ^a	Frequency	Number of families		$\chi^2_{7:4:1}$	P
		Observed	Expected		
All R	7	28	22.75	...	...
3(R+N+LS):1S	4	12	13	...	...
15(R+N+LS):1S	4	9	13	...	...
All S	1	3	3.25	...	...
Total	16	52	52	2.54	0.47

^a R, resistant (symptomless); N, stem tip necrosis; S, susceptible; LS, late susceptible.

deficiency of LS plants and excess of R plants. Affecting factors in this case could be the age of the plants at inoculation, the inoculation procedure, or different environmental conditions. In the greenhouse, plants were inoculated at 10 days after planting (V1 stage), whereas field-grown plants were inoculated 3 to 4 weeks after planting (V2 to V3 stage). In the greenhouse, both unifoliolate leaves were inoculated by rubbing evenly with a pestle dipped in inoculum. In the field, an artist's airbrush, which is more damaging to plant tissue, was used to inoculate only one leaflet of the first or second trifoliolate leaf on each plant, using sufficient pressure and proximity to cause water-soaking of cells. The larger area of the unifoliolate leaves inoculated with SMV by pestle inoculation in the greenhouse, with minimal wounding of the inoculated cells, presumably increased the probability of establishing an infection site and leading eventually to LS symptoms. Reduced infection could have been an inoculum dosage effect, but more likely it is the result of the damage caused by air brush inoculation and cell death prior to virus movement from initially inoculated cells.

Following inoculation with SMV-G7, LS was not observed in F₁ plants, F₂ populations, and F_{2,3} families of PI 88788 × Essex and PI 88788 × Lee 68, indicating that the *Rsv4* gene is completely dominant to this pathotype. Although our data show some variation in response to the two pathotypes, it is clear that the same gene controls the resistance to SMV strains G1 and G7 in PI 88788 because all F_{2,3} families of PI 88788 × Essex populations exhibited the same reaction to both SMV strains G1 and G7.

Partial dominance implies that resistance is expressed more effectively in the homozygous (RR) than heterozygous (Rr) state. This is observed in *Rsv1* as complete resistance with homozygotes and systemic necrosis with heterozygotes (10,18). However, local and systemic necrosis also may be observed when a homozygous resistance gene at the *Rsv1* locus interacts with specific SMV strains (10). For example, *Rsv1* from PI 96983 develops systemic necrosis when inoculated with SMV-G7 (9) and *Rsv3* derived from Columbia responds to SMV-G1 with a necrotic reaction (7). These observations suggest that *Rsv1* and *Rsv3* possess receptor domains that bind viral elicitors to activate a hypersensitive defense response, consistent with the observation that these genes map to resistance gene clusters (20,23).

Resistance to SMV in PI 88788 is, thus, a unique type of resistance within the SMV–soybean pathosystem because it does not appear to utilize well-characterized defense pathways. No hypersensitive response was observed in plants having the *Rsv4* gene in either a homozygous or heterozygous state following inoculation with any SMV strain. Resistance results from a restriction in cell-to-cell and vascular movement of the virus in the inoculated leaves, based on SMV-G1 and -G7 data. The LS phenotype is described that is detected only in heterozygous plants observed over a longer period of time. In this response, early restriction to virus movement by the gene was overcome, perhaps by virus-mediated suppression of gene silencing, enabling the virus to move through the vascular system and invade newly developing leaves. Movement within these leaves, however, also is restricted resulting in veinal chlorosis and green islands exhibited in the LS phenotype.

The *Rsv4* gene in PI 88788 appears to be the first report of a single dominant gene that confers resistance by interfering with viral movement. Resistance to potyviruses occurs in a number of major crop species, with a range of resistance mechanisms. Single dominant genes, such as *Rsv1* and *Rsv3* in soybean, have been shown in most cases to condition extreme resistance or hypersensitive resistance responses. Other examples include *Ry_{sto}*, conferring resistance in potato to potyviruses *Tobacco etch virus* (TEV) and *Potato virus Y* (PVY) (22), and the *Nv* gene in potato (2) conferring broad-spectrum resistance to several potyviruses. Resistance of *Arabidopsis thaliana* to TEV, however, is conditioned by two dominant, phloem functioning, genes *RTM1* and *RTM2*, which cooperate to restrict long-distance movement of the virus (12). A similar mechanism has been postulated for one resistance phenotype of *A. thaliana* to another potyvirus, *Lettuce mosaic virus* (30). However, neither of these reports recognizes an LS phenotype. The resistance conditioned by recessive genes often has been associated with a mechanism involving restriction to virus accumulation and viral movement. This type of resistance has been described for several potyviruses, including the recessive genes for resistance to *Zucchini yellow mosaic virus* and other potyviruses in cucurbits (35), PVY in pepper (1), *Potato virus A* in potato (19), and PVY and TEV in tobacco (26,31). Additionally, dominantly inherited quality trait loci mapping to seven genomic regions have been associated with resistance of pepper to *Cucumber mosaic virus* (genus *Cucumovirus*, family *Bromoviridae*) by a mechanism involving partial restriction to long-distance movement (8).

Deployment of cultivars possessing *Rsv4* may contribute to broad, effective, and long-term resistance. The gene allows limited accumulation of virus, prevents rapid long-distance movement of virus, and delays symptom expression. Of the SMV strains tested, none were found to overcome resistance at *Rsv4*, in contrast to the *Rsv1* and *Rsv3* loci which are overcome by several strains (Table 1). Strategies for managing SMV disease can include reducing the initial inoculum to a low level, reducing the temporal virus progress (such as utilizing *Rsv4* and managing virus vectors), and reducing the time span that host-pathogen populations interact (27). Thus, for SMV and related viruses, host resistance that reduces the initial success of establishing infection and rate of within-plant spread may provide benefits in terms of efficacy and durability (3,27,28). Benefits of reduction in incidence and delay in symptom appearance also have been demonstrated with transgenic resistance to SMV in soybean (34). Because *Rsv4* exhibits this mode of action as well as broad resistance to SMV strains, it should be a valuable gene to deploy in new cultivars or to pyramid with other *R* genes.

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TABLE 6. Segregation of F_{2,3} families of soybean derived from L29(R) × PI 88788 (R) when inoculated with *Soybean mosaic virus* strain G7 in the greenhouse

Reaction ^a	Frequency	Number of families		$\chi^2_{7;4;1}$	P
		Observed	Expected		
All R	7	23	20.56	...	...
3(R):1S	4	12	11.75	...	...
15(R):1S	4	8	11.75	...	...
All S	1	4	2.94	...	...
Total	16	47	47	1.88	0.60

^a R, resistant (symptomless); LS, late susceptible; S, susceptible.

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