VIRGINIA POLYTECHNIC INSTITUTE AND STATE UNIVERSITY Department of Plant Pathology, Physiology and Weed Science, and Department of Agronomy Blacksburg, VA 24061

## Genetics of reaction to soybean mosaic virus (SMV) in the cultivars 'Kwanggyo', 'Marshall', and PI 96983

Several genes conditioning resistance to SMV have been found and some have been assigned gene symbols. In addition, a series of SMV strain groups has been differentiated by their interactions with a selected group of cultivars (Cho and Goodman, 1979; Lim, 1985). We have undertaken a study of the genes conditioning the reactions of certain differential cultivars to SMV in an attempt to establish their relationships with symbolized genes. Recently, we have established the number of genes conditioning SMV resistance in each of the cultivars Kwanggyo, Marshall and PI 96983.

The isolate SMV-VA, which was classified into Cho and Goodman's strain group Gl (Hunst and Tolin, 1982), was used to inoculate F<sub>3</sub> lines from crosses shown in Table 1. All work was done under field conditions using methods described by Roane et al. (1983). We found that PI 96983 has two genes conditioning reaction to SMV-VA and that Kwanggyo and Marshall each have one gene; these are either allelic or are very closely linked.

	Clas					
Cross	Homozygous resistant	Segregating	Homozygous susceptible	x <sup>2</sup>	Ratio tested	Р
Essex (RXS)	69	71	9	0.465	7:8:1	>0.7
Essex x Marshall (SXR	) 47	80	51	1.421	1:2:1	>0.3
Kwanggyo x						
Lee 68 (RXS)	30	48	15	4.935	1:2:1	>0.05
Kwanggyo x Marshall (RXR	) 82		0		e nguran	- SACT 

Table 1. Segregation of F3 lines for reaction to SMV-VA (= G1)

<sup>+</sup>Two lines segregated 16:1 and 23:2.

134

Kiihl and Hartwig (1979) identified one gene, <sub>RSV</sub>, in PI 96983. They used isolates of SMV which were later assigned by Cho and Goodman (1982) to strain groups G2 and G3. These strains must have one or more virulence genes capable of defeating one of the resistance genes in PI 96983. Similarly, Buzzell and Tu (1984) detected only one gene in a PI 96983 descendant, L78-379. Either the strains they used, G7 and G7A, can also defeat one of the genes in PI 96983, or else only one of the two genes from PI 96983 occurs in L78-379.

The single genes found to condition the resistance of Kwanggyo and Marshall to SMV have not yet been tested against any other symbolized gene, but we plan to conduct such experiments. At present, we are concerned about the poor fit of the Kwanggyo x Lee 68 cross to a monogenic ratio. When we produced the  $F_3$  seed in the field, the  $F_2$  plants were inoculated with SMV and some failed to produce seed. This may have brought about a deficiency of homozygous susceptible lines. Kwanggyo will be given further attention.

## References

- Buzzell, R. I. and J. C. Tu. 1984. Inheritance of soybean resistance to soybean mosaic virus. J. Heredity 75:82.
- Cho, E.-K. and R. M. Goodman. 1979. Strains of soybean mosaic virus: Classification based on virulence in resistant cultivars. Phytopathology 69: 467-470.
- Cho, E.-K. and R. M. Goodman. 1982. Evaluation of resistance in soybeans to soybean mosaic virus strains. Crop Sci. 22:1133-1136.
- Hunst, P. L. and S. A. Tolin. 1982. Isolation and comparison of two strains of soybean mosaic virus. Phytopathology 72:710-713.
- Kiihl, R. A. S. and E. E. Hartwig. 1979. Inheritance of reaction to soybean mosaic virus in soybeans. Crop Sci. 19:372-375.
- Lim, S. M. 1985. Resistance to soybean mosaic virus in soybeans. Phytopathology 75:199-201.
- Roane, C. W., S. A. Tolin and G. R. Buss. 1983. Inheritance of reaction to two viruses in the soybean cross 'York' x 'Lee 68'. J. Heredity 74:289-291.

Curtis W. Roane Sue A. Tolin Dept. of Plant Pathology, Physiology & Weed Science

Glenn R. Buss Dept. of Agronomy

## Application of the gene-for-gene hypothesis to soybean-soybean mosaic virus interactions

The gene-for-gene hypothesis has been found applicable to several hostparasite interactions in which the parasite is a fungus. It has most satisfactorily explained interactions involving cereal rust and powdery mildew diseases in which cases the pathogens are obligate parasites and the genes of both the parasites and hosts may be manipulated in genetic tests. Generally, the gene-for-gene hypothesis has not been applied to host-virus systems because virulence genes in viruses cannot be manipulated by hybridization. However, by assuming that each having soybean mosaic virus (SMV) strain possesses one or no virulence gene (having no virulence gene means that the strain can cause symptoms only in a host having no resistance gene), we may soon be able to "read out" host genotypes for resistance to SMV simply by inoculating cultivars with carefully selected SMV strains. Such a system has been described for *Phaseolus vulgaris* and bean common mosaic virus (Drijfhout, 1978).

In Table 1, the cumulative genetic research on SMV resistance in soybean is summarized. Information is taken from published works of Buzzell and Tu (1984); Cho and Goodman (1979, 1982); Kiihl and Hartwig (1979); Lim (1985); and Roane, Tolin and Buss (1983, 1986). From Table 1, it may be seen that:

- PI 96983, carrying Rsv, segregates monogenically when progenies of its crosses are inoculated with SMV strains G2, G3, G6, G7, G7A, and C14 (Buzzell and Tu, 1984; Kiihl and Hartwig, 1979; Lim, 1985), but digenically when inoculated with G1 (Roane et al., 1986). Therefore, PI 96983 must have a second resistance gene labelled here Rsv?.
- 2. Strains G2, G3, G6, G7, G7A, and C14 each must possess a virulence gene capable of "defeating" one of the genes in PI 96983 but G1 must lack such a virulence gene. This is also apparent from Table 1 where it is shown that G1 does not produce symptoms on cultivars carrying resistance genes; therefore, G1 should be used in all genetic studies because it should detect any resistance gene.
- B. If strains G2, G3, G6, G7, G7A, and C14 each possess a single virulence gene, progenies of some cultivars other than PI 96983, which showed monogenic inheritance when inoculated with one of these strains, could show digenic inheritance when progenies are inoculated with G1. Note that our assumptions do not preclude the possibility that some virus strains may have more than one virulence gene.

136

- 4. 'Ogden' has been assumed to have a gene,  $rsv^t$ , at the Rsv locus (Kiihl and Hartwig, 1979). However, this gene may be allelic with Rsv?, the second gene in PI 96983, rather than with Rsv. Proof of this hypothesis requires the inoculation of partitioned  $F_3$  lines of PI 96983 x Ogden and a susceptible cv. x Ogden with strains Gl and G2 or G3.
- 5. 'York' has been assumed to carry rsv<sup>t</sup> because of its descent from 'Tokyo', Ogden, and 'Hood' (Kiihl and Hartwig, 1979; Roane et al., 1983). York is a selection from a cross of 'Dorman' x Hood. Cho and Goodman (1982) have placed York in the cultivar group with 'Davis', Dorman and 'Ware'. Thus, York could have derived a gene for SMV resistance from either Hood or Dorman. A genetic study of York x PI 96983, York x Ogden and York x Davis using Gl and G2 to inoculate partitioned F<sub>3</sub> lines from the crosses will be necessary to determine if York carries rsv<sup>t</sup> or Rsv?, the second gene in PI 96983.
- 6. By virtue of their identical responses to virus strains, 'Raiden' and 'Suweon 97' belong to the same cultivar group; therefore, Suweon 97 must carry Rsv<sub>2</sub> (Buzzell and Tu, 1984; Cho and Goodman, 1982). Since this is assumed but not proven, the Suweon 97 gene is labelled Rsv<sub>2</sub>?.
- 7. 'Kwanggyo' and 'Marshall' have alleles or closely linked genes as shown in the previous report (Roane et al., 1986). Their responses to seven SMV strains differ; therefore, they must not have the same gene or allele.
- 8. Lim (1985) reported a gene in PI 486355 which segregated independently of Rsv and Rsv<sub>2</sub> (Rsv<sub>3</sub>? in Table 1) but which was not labelled. Crosses of PI 486355 with Ogden, Marshall, Kwanggyo and York are needed to find out if the PI 486355 gene is unique.

Obviously, the several assumptions for the eight points above require proof. To be able to designate properly the genes involved in SMV reactions requires that the strains of SMV and the differential cultivars be maintained by someone or at some laboratory. At present, we have samples of Cho and Goodman's strain groups Gl-G7 in storage and have seed of their differential cultivars. However, we do not have Raiden, Suweon 97 or PI 486355. In addition, we urge that researchers obtaining results relative to this problem should communicate their results promptly to other interested persons so that effort will not be duplicated.

Cultivars <sup>a</sup>	Genes in cultivars	Virus strain group and systemic symptoms for strain/host interaction								
		G1 (SMV-VA) <sup>b</sup> 0	G2 (SMV-1) 1	G3 (SMV-1B)		G5	G6	G7	G7A 1	C14
				1		es in s ?	trains - 1			1
PI 96983 (Buffalo)	Rsv + Rsv?	0 <sup>c</sup> (2) <sup>d</sup> R6 <sup>e</sup>	0(1) K,L	0(1) K	0	0	0(1) B	N(1) B,L	0(1) B	0(1) L
Ogden (Tokyo, Hood)	rsvt	0	0(1) K	N(1) K	0	0	0	N	-	
Davis (Dorman, York, Ware)	Rsv?f	0(1) R3	0	0	N	S	S	S	-	- Entry
Raiden = PI 360844 (Suweon 97)	Rsv <sub>2</sub>	0	0	0	0	0	0(1) B	0	0(1) B	N(1) L
Suweon 97 = PI 483084 (Raiden)	Rsv <sub>2</sub> ?	0	0(1) L	0	0	0	0	0(1) L	-	N(1) L
PI 483355	Rsv <sub>3</sub>	0	0	0	0	0	0	0(1) L	S	0(1) L
Marshall	Rsv?	0(1) R6	N	N	0	0	N	N		
Kwanggyo	Rsv?	0(1) R6	0	0	0	N	N	N	-	2

Table 1. Summary of soybean mosaic virus strains, cultivars for differentiating them, and genetics of their interactions

<sup>a</sup>Cultivars in parentheses give same reactions to strains as those accompanying them but not in parentheses. <sup>b</sup>Viruses in parentheses are synonyms for the G strains.

<sup>c</sup>Symptoms reported: 0 = no symptoms, N = systemic necrosis, S = systemic mottling, - = no report. <sup>d</sup>Number of genes in a host conditioning the reaction to a strain group.

<sup>e</sup>Letters on second line in each cluster refer to published reports -- B = Buzzell & Tu, 1984; K = Kiihl & Hartwig, 1979; L = Lim, 1985; R3 = Roane, Tolin & Buss, 1983; R6 = Roane, Tolin & Buss, 1986.

f Rsv? connotes dominant resistance, the four Rsv?'s shown may or may not be alleles.

## References

- Buzzell, R. I. and J. C. Tu. 1984. Inheritance of soybean resistance to soybean mosaic virus. J. Heredity 75:82.
- Cho, E.-K. and R. M. Goodman. 1979. Strains of soybean mosaic virus: Classification based on virulence in resistant cultivars. Phytopathology 69: 467-470.
- Cho, E.-K. and R. M. Goodman. 1982. Evaluation of resistance in soybeans to soybean mosaic virus strains. Crop Sci. 22:1133-1136.
- Drijfhout, E. 1978. Genetic interaction between *Phaseolus vulgaris* and bean common mosaic virus with implications for strain identification and breeding for resistance. Centre for Agri. Publishing and Documentation. Wageningen, Netherlands. 98 pp.
- Hunst, P. L. and S. A. Tolin. 1982. Isolation and comparison of two strains of soybean mosaic virus. Phytopathology 72:710-713.
- Kiihl, R. A. S. and E. E. Hartwig. 1979. Inheritance of reaction to soybean mosaic virus in soybeans. Crop Sci. 19:372-375.
- Lim, S. M. 1985. Resistance to soybean mosaic virus in soybeans. Phytopathology 75:289-291.
- Roane, C. W., S. A. Tolin and G. R. Buss. 1983. Inheritance of reaction to two viruses in the soybean cross 'York' x 'Lee 68'. J. Heredity 74:289-291.
- Roane, C. W., S. A. Tolin and G. R. Buss. 1986. Genetics of reaction to soybean mosaic virus (SMV) in the cultivars Kwanggyo, Marshall, and PI 96983. Soybean Genet. Newsl. 13:134-135.

Curtis W. Roane Sue A. Tolin Dept. of Plant Pathology, Physiology & Weed Science

Glen R. Buss Dept. of Agronomy