For corresponding crosses,the total, pooled, and homogeneity chi-square values were all found acceptable for the two F_2 genetic ratios used respectively to test goodness-of-fit.

These data indicated that the aberrant white-stem phenotype is a recessive characteristic in peanuts. The acceptance of both the monogenic and digenic models suggest that one of the two genes for the related germplasm P-0062 is present as homozygously recessive alleles. Whereas in the unrelated pigmented parents, the two genes are homozygous for dominant alleles. Since the cultivated peanut has been classified as a diploidized allotetraploid[5], these findings would seem to suggest a theory of residual heterozygosity, where duplicate loci between genomes are perpetuated in a homozygous but not homogenous condition.

Segregation in F_3 progenies resulting from selfed individual F_2 plants having pigmented stems was as expected, except for two crosses, P-0062 × white stem (Table I) and Gujarat narrowleaf × white stem (Table II). Significant deviation occurred only in F_3 hybrid populations involving the white-stem male parent. Since no reciprocal differences were found in the F_2 generation, the reasons for this phenomenon in these cross combinations are uncertain. It should be noted that in both segregating generations, significantly ($P < 0.01$) reduced survival was observed for white-stem seedlings, probably due to direct sunlight causing lethality. However, all classifiable progenies from white-stem F_2 plants bred true for the character. Thus, the F_3 data appear to confirm the F_2 inheritance results.

Gene symbols ws_1 and ws_2 are proposed for the two independent recessive loci controlling the white-stem phenotype in peanuts. Upon crossing, pigmented $Ws_1Ws_1Ws_2Ws_2$ and $Ws_1Ws_1ws_2ws_2$ or $ws_1ws_1Ws_2Ws_2$ parental lines × white stem genotype, $ws_1ws_1ws_2ws_2$, resulted in digenic and monogenic models, respectively.

References

1. BADAMI, V. R. K. *Arachis hypogaea* (the Groundnut). Ph.D. thesis. Cambridge (Eng.) Univ. Library. 1928.
2. BALAIAH, C., P. S. REDDY, and M. V. REDDI. Genic analysis in groundnut: I. Inheritance studies on 18 morphological characters in crosses with Gujarat narrow leaf mutant. *Proc. Indian Acad. Sci.* 85B:340-350. 1977.
3. BRANCH, W. D. Screening for genetic tolerance to cold temperature during germination in peanuts (*Arachis hypogaea* L.). Ph.D. thesis, Okla. State Univ. (Mic. no 7801211). Univ Microfilms, Ann Arbor, Mich. (Diss. Abstr. Internal. 38(9):3987 B, 1978). 1976.
4. GOPANI, D. D. and N. L. VAISHNANI. Two mutant forms of groundnut (*Arachis hypogaea* L.). *Indian J. Agric. Sci.* 40:431-437 1970.
5. HAMMONS, R O. Genetics of *Arachis hypogaea*. *In* Peanuts—Culture and Uses. Am. Peanut Res. Educ. Assn., Stillwater, Okla. p. 135-173. 1973.
6. HANNA, W., B. MULLINIX, and L. GRIMES. Computer programs for analyses of inheritance and linkage data. *Crop Sci.* 18:517. 1978.
7. HAYES, T. R. The classification of groundnut varieties. With a preliminary note on the inheritance of some characters. *Trop. Agric.* (West Indies) 10: 318-327. 1933.
8. JADHAV, G. D. and N. N. SHINDE. Genetic studies in groundnut (*Arachis hypogaea*). *Indian J. Agric Res.* 13:93-96. 1979.
9. KATAYAMA, Y. and T. NAGATOMO. Crossing experiments in various strains of peanut. *Bull. Fac. Agric. Miyazaki Univ.* 9:99-126. 1963.
10. PATEL, J. S., C. M. JOHN, and C. R. SESHADRI. The inheritance of characters in the groundnut *Arachis hypogaea Proc. Indian Acad. Sci.* 3(B): 214-233. 1936.
11. SRIVASTAVA, D. P. Inheritance of purple pigmentation on stem and branches in groundnut. *Sci. Cult.* 43.42-43 1977.

The Journal of Heredity 73:302-303. 1982.

Inheritance of resistance to soybean mosaic virus in *Phaseolus vulgaris*

R. Provvidenti, D. Gonsalves, and P. Ranalli

ABSTRACT: In cross and backcross populations of the bean cultivar Great Northern 1140 (resistant to soybeam mosaic virus (SMV)) with the susceptible line Black Turtle 2 (a selection of Black Turtle Soup), resistance was conferred by a single but incompletely dominant gene (*Smv*). In seed lots of individual plants of SMV-infected Black Turtle 2 and the cultivar Pioneer, which had been kept in storage for more than a year, virus transmission ranged from 0 to 4 percent.

NATURAL occurrence of soybean mosaic virus (SMV) is commonly associated with the soybean crop. Infected seed of *Glycine max* (L.) Merr. represents the main source of this virus that is subsequently spread by several aphid species in a stylet-borne manner[1]. Experimentally, it has been demonstrated that SMV can infect a number of leguminous species, including the common bean (*Phaseolus vulgaris*)[4,6,8,9,12-14].

During annual surveys of viral diseases affecting the bean crop in New York State, we have recovered SMV from bean plants exhibiting symptoms closely resembling those caused by bean common mosaic virus (BCMV). The identity of the causal agent was determined by diagnostic species, serology, and electron microscopy. In greenhouse tests using SMV isolates from soybean many domestic cultivars and plant introductions of *P. vulgaris* responded only with localized infection (resistant). A smaller number of lines reacted with severe systemic symptoms resembling those of BCMV (susceptible), or with local necrotic lesions followed by systemic necrosis and death of plants (local and systemic hypersensitivity). The objective of this study was to elucidate the resistance to SMV in the cultivar Great Northern 1140 (GN1140), which had been previously found to be resistant to the severe strain of bean yellow mosaic virus[11].

Materials and Methods

Genetic studies were based on populations that had been derived from crosses and backcrosses between the SMV-resistant GN1140 with the susceptible line Black Turtle 2 (BT2), a selection of the cultivar Black Turtle Soup[10]. All seed were obtained from plants grown exclusively in the greenhouse and maintained free of BCMV infection. Plants of F_1, F_2, and reciprocal backcross generations were tested with an isolate of SMV (NY76-6), which had been recovered from the Altona soybean. Inoculum was prepared by homogenizing young leaves of infected Altona plants with 0.01 M phosphate buffer (K^+) at pH 7.5. Plants were mechanically inoculated when the primary leaves were fully expanded and thereafter reinoculated on the first trifoliolate. Recovery of SMV was attempted on plants of Altona and Black Turtle 1 (BT1) (another selection of Black Turtle Soup[10]). Altona is not infected by BCMV, and BT1 with SMV responds with numerous necrotic local lesions and systemic necrosis, but is highly resistant to most of the strains of BCMV[10].

Enzyme-linked immunosorbent assay (ELISA)[3] also was employed, using an antiserum to SMV provided by R. M. Lister, Purdue University. Twenty-two additional isolates of SMV, including the strains characterized by Cho and Goodman[2], were used to test plants of GN1140, BT1, BT2, and Pioneer. Seed lots derived from single plants of BT2 and Pioneer systemically infected with SMV were planted in the greenhouse to determine seed transmission of SMV. Prior to their use seed had been kept for more than one year in a seed storage. The work was conducted in an insect-free greenhouse, which was maintained at 25°-30°C.

The first and second authors are affiliated with the Department of Plant Pathology, New York State Agricultural Experiment Station, Cornell University, Geneva, NY 14456; the third author with the Istituto Sperimentale per le Culture Industriali, Bologna, Italy.

Results

Parental reaction. Plants of the resistant parent, GN1140 did not exhibit local or systemic symptoms when inoculated with SMV. Assays, however, revealed that there was virus infection, but it was confined to the inoculated leaves. Plants of the susceptible parent, BT2, reacted with local chlorotic spots that eventually turned necrotic. Systemic symptoms were severe and plants remained stunted with leaves considerably reduced in size. Trifoliolates showed a variety of symptoms that included green mottle veinbanding, blistering, downward cupping, and distortion. Seed production was reduced and some plants died prematurely.

Reaction of F_1 and F_2 generations. F_1 plants reacted to SMV inoculation with local chlorotic spots followed by systemic symptoms, that initially consisted of scattered chlorotic spots and then by a mild to moderate mottle. Plants, however, remained vigorous and only slightly stunted. Seed production on these plants compared favorably with those of noninoculated controls. In F_2 populations segregation was in the ratio 1:2:1. Thus, three recognizable classes of plants were evident: 1) resistant, which were free of systemic infection; 2) plants systemically infected but vigorous and productive as those of F_1 generation (intermediate resistance); and 3) susceptible with symptoms comparable to those of BT2. Progenies of plants exhibiting intermediate resistance also segregated in the ratio 1:2:1 confirming that the heterozygotes could be distinguished phenotypically from both parents.

Reaction of reciprocal backcross generations. Plants that derived from progenies of F_1 plants crossed with the resistant parent, segregated in a ratio of 1 resistant (no systemic infection) to 1 with intermediate resistant. The progenies of F_1 plants that had been crossed with the susceptible parent also segregated in approximately equal number of intermediate resistant and susceptible individuals.

From the data presented in Table I, it is concluded that resistance of SMV in *P. vulgaris* cv. GN1140 is conditioned by a single, but incompletely dominant factor. For this gene the symbol *Smv* (soybean mosaic virus) is proposed.

Reaction of GN1140 to other isolates of SMV. When plants of GN1140 were mechanically inoculated with 22 additional isolates of SMV, including the seven recognized strains[2], virus infection was localized to the inoculated leaves, which failed to develop symptoms. Plants of BT1 reacted to all the isolates with numerous but rather distinct necrotic local lesions followed by systemic necrosis and death. Similarly, plants of BT2 and those of Pioneer develop systemic symptoms closely resembling those of BCMV.

Seed transmission of SMV in bean. Three of seven seed lots of BT2 yielded plants that were infected with SMV (1/30, 0/24, 0/31, 1/48, 0/16, 0/15, and 1/32). Five of nine lots of Pioneer contained plants infected with SMV (2/50, 0/30, 2/47, 0/31, 1/46, 2/53, 0/27, 1/32, and 0/19). Thus, the percentage of SMV infection ranged from 0 to 4 percent. The identity of the virus infecting every single plant was confirmed using the indicator host Altona soybean and by serology. Work is in progress to localize the virus in infected seed.

Discussion

The results show that in the bean cultivar GN1140 resistance to SMV is conferred by an incompletely dominant factor. However, the expression of this gene (*Smv*) appears to be influenced by external factors. During late spring and summer, the heterozygotes were clearly distinguishable from homozygous resistant or susceptible plants, but during autumn and winter they failed to show symptoms or were inconspicuously affected. Consequently, at times only two classes of plants resistant and susceptible could be recognized. Since temperature was easily controlled and natural light was supplemented with fluorescent tubes for a 14-hour photoperiod, this shift toward full dominance may have been caused by reduced light intensity or a change in light quality.

The cultivar GN1140 also was found to be resistant to 23 isolates of SMV, including the strains of this virus characterized by Cho and Goodman[2] using differential soybean cultivars. Thus, whereas in *G. max* resistance to SMV appears to be essentially strain specific, in *P. vulgaris* cv. GN1140 resistance was not associated with any of the available SMV isolates.

There are numerous reports in the literature regarding the seed-borne nature of SMV in soybeans[1], but the few previous attempts to demonstrate it in *P. vulgaris* were unsuccessful[5,8,9]. Our results indicate that this virus can be transmitted through seed of BT2 and Pioneer. As for soybeans, seed transmission may depend upon several factors[7,13].

The symptoms incited by SMV in susceptible bean plants can be easily confused with those incited by BCMV. Similarly, the symptoms caused by watermelon mosaic virus 2 (WMV-2) mimic those of bean yellow mosaic virus (BYMV)[10]. Both SMV and WMV-2 presently can be considered of minor importance, since most of the cultivars in use are resistant. But the potential importance of these viruses must not be underestimated, particularly SMV, which can be seed-transmitted. We have found that a number of plant introductions currently used as sources of resistance to root-rot (PI 109859, PI 165426, PI 165435, PI 203598, and others) are susceptible to these two viruses.

The bean line BT1 proved to be a more sensitive and reliable local lesion host than Kentucky Wonder, which was widely used as assay host before the advent of ELISA.

Table I. Segregation ratios in cross and backcross populations of *Phaseolus vulgaris* cv. Great Northern 1140 (GN1140) with line Black Turtle 2 (BT2) for resistance to soybean mosaic virus

Populations	No. plants: resistant	No. plants: intermediate resistant	No. plants: susceptible	Expected ratio	Goodness-of-fit (P)
GN1140	150	0	0		
BT2	0	0	125		
(GN1140 × BT2)F_1	0	55	0		
(GN1140 × BT2)F_2	68	139	61	1:2:1	.69
(GN1140 × BT2)F_1 × GN1140	25	29	0	1:1	.60
(GN1140 × BT2)F_1 × BT2	0	46	50	1:1	.64

References

1. BOS, L. Soybean mosaic virus. C.M.I./A.A.B. Description of plant viruses. No. 93. 1972.
2. CHO, K. K. and R. M. GOODMAN. Strains of soybean mosaic virus: classification based on virulence in resistant soybean cultivars. *Phytopathology* 69:467–470. 1979.
3. CLARK, M. F. and A. N. ADAMS. Characteristics of microplate method of enzyme-linked immunosorbent assay for the detection of plant viruses. *J. Gen. Virology* 34:475–483. 1977.
4. COSTA, A. S., L. N. COSTA, L. D. ALMEIDA, and E. BULISANI. Susceptibilitade de certos grupos de feijoeiro a infeccao sistematica pelo virus do mosaic da soja. *Fitopat. Brasileira* 3:27–37. 1978.
5. ——, J. VEGA, and O. GASPAR. Non-transmission of the soybean virus through bean seeds from infected plants. *Summa Phytopath.* 4:24. 1978
6. GALVEZ, G. E. Host range, purification, and electron microscopy of soybean mosaic virus. *Phytopathology* 53:388–393. 1963.
7. GOODMAN, R. M., G. R. BOWERS, JR., and E. H. PASCHAL II. Identification of soybean germplasm line and cultivars with low incidence of soybean mosaic virus transmission through seed. *Crop. Sci.* 19:264–267. 1979.
8. IIZUKA, N. Seed transmission of viruses in soybean. *Tohoku Natl. Agr Exp. Sta. Bull.* 45: 131–141. 1973.
9. MURAYAMA, D. and Y. H. HAN. Occurrence of soybean mosaic virus in Taiwan. *Pl. Protect. Bull.* 13:75–85. 1971.
10. PROVVIDENTI, R. Inheritance of resistance to watermelon mosaic 2 in *Phaseolus vulgaris*. *Phytopathology* 64:1448–1450. 1974.
11. —— and W. T. SCHROEDER. Resistance in *Phaseolus vulgaris* to the severe strain of bean yellow mosaic virus. *Phytopathology* 63:196–197. 1973.
12. QUANTZ, L. Untersuchungen uber das Gewohnliche Behnenmosaikvirus and das Sojamosaikvirus. *Phytopath. Z.* 43:79–101. 1961.
13. SINGH, B. R., D. R. SINGH, and H. K. SAKSENA. A mosaic disease of soybean at Kanpur. *India Sci. Cult.* 42:53–54. 1976.
14. WALTERS, H. J. Leguminous hosts of soybean mosaic virus. *Pl. Dis. Reptr* 47:726–728. 1963.