infected with non-sporulating lesions (332). The leaves on the upper third of the plant of other sublines including control lines were heavily covered with sporulating lesions (343).

It is still early to say that the four sublines producing lesions with no sporulation (hypersensitive reaction) which were found in this experiment are rust-resistant mutants. There is a need for further intensive investigation. Anyhow, this observation of the hypersensitive reaction type in M_3 soybean lines, especially the one out of three in Line No. 138 (Taichung) may suggest that an attempt to create variability for rust resistance by radiation should not be overlooked.

References

Bromfield, K. R. and C. Y. Yang. 1976. Soybean rust: Summary of available knowledge. In: Expanding the use of soybeans. R. M. Goodman, Ed. Proceedings of a conference for Asia and Oceania. College of Agriculture, University of Illinois, Urbana. (INTSOY Series 10), pp. 161-164.

Nundhapun, M. and P. Surin. 1977. Soybean reactions to rust caused by Phakopsora pachyrhizi at Mae-Joe Experiment Station. Soybean Rust Newsl. 1: 11-12.

Pupipat, U. 1977. Soybean rust research in Thailand. Soybean Rust Newsl. 1: 7-10.

Yang, C. Y. 1977. The IWGSR rust rating system. Soybean Rust Newsl. 1: 4-6.

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1) A second gene for resistance to peanut mottle virus in soybean.

Soybean (Glycine max [L.] Merr.) was first reported as a natural host of peanut mottle virus (PMV) in 1972 by research workers in Georgia (Kuhn et al., 1972). PMV on soybean has since been reported in Virginia, South Carolina, Australia, and East Africa (Demski and Kuhn, 1977). Boerma and Kuhn (1976) reported resistance to PMV in soybeans to be conditioned by a completely dominant allele at a single locus. The objective of this study was to determine if there are other genes and/or alleles that might condition resistance to PMV.

During the winter of 1975-76, several soybean cultivars and Plant Introductions were tested in the greenhouse for reaction to PMV. Based on these tests, 20 resistant soybean cultivars and Plant Introductions and two susceptible lines were selected for intercrossing in 1976. Results reported here involve only five of the selected lines (Table 1). Each of the four resistant lines, 'Arksoy', 'Peking', PI 89.784 and PI 219.789, was crossed to a susceptible line, PI 229.315. Crosses were also made between Peking and each of the other three resistant lines (Table 2).

Table 1

Parents used in crosses in 1976

Identity	Maturity Group	PMV reaction
Arksoy	VI	Resistant
Peking	V	Resistant
PI 89.784[†]	III+	Resistant
PI 219.789	V	Resistant
PI 229.315	V	Susceptible

[†]Identity not certain since flower color does not agree with that reported in RSLM 238, April 1969.

The F_1 plants for each of the seven crosses were grown in the greenhouse during the winter of 1976-77. Flower, pubescence, hilum and seed coat colors were used as genetic markers to verify crosses in the F_1 and F_2 generations. The F_2 seedlings from individual F_1 plants were grown in metal flats in the greenhouse during summer and fall of 1977. Both F_1 and F_2 plants were inoculated with PMV and scored as either resistant or susceptible.

F_1 plants for each of the following crosses were resistant to PMV: Arksoy (R) x PI 229.315 (S); PI 89.784 (R) x PI 229.315 (S); and PI 229.315 (S) x PI 219.789 (R) (Table 2). Segregating F_2 progenies from the same three crosses gave an acceptable fit to a 3 R : 1 S genetic ratio. Thus it appears that resistance to PMV in Arksoy, PI 89.784, and PI 219.789 is controlled by a single dominant gene ($\underline{Rpv}_1$) as reported previously for soybean cultivars 'CNS' and 'Dorman' (Boerma and Kuhn, 1976). It is most probable that Arksoy and Dorman carry the same gene for resistance to PMV since Arksoy 2913 is a parent

Table 2

Reactions of F_1 and F_2 soybean plants to inoculation with peanut mottle virus (PMV) and probabilities of proposed genetic ratios

Cross	PMV reaction of F_1 plants	PMV reaction of F_2 plants			Proposed genetic ratio	Chi-square probability
		Res.	Susc.	Total		
Arksoy (R) x Peking (R)	R	86	17	103	13:3	.58
		49	14	63	13:3	.49
(Pooled data from two F_1 plants)		135	31	166	13:3	.98
Peking (R) x PI 89.784 (R)	R	36	8	44	13:3	.92
Peking (R) x PI 219.789 (R)	R	37	6	43	13:3	.44
Arksoy (R) x PI 229.315 (S)	R	45	10	55	3:1	.24
Peking (R) x PI 229.315 (S)	S	--*	--	--	---	---
PI 89.784 (R) x PI 229.315 (S)	R	37	13	50	3:1	.87
PI 229.315 (S) x PI 219.789 (R)	R	40	13	53	3:1	.94

*No F_2 data available at present.

of Dorman.

If the gene conditioning PMV resistance in Peking were the same as in the other three resistant lines, then one would not expect segregation in any F_2 families from crosses among these lines. However, segregation was observed in all F_2 families from crosses of Peking with the other resistant parents. Apparently, at least two different genes for resistance were present. The only two-class F_2 dihybrid ratio that would provide a reasonable fit to the data is 13 resistant : 3 susceptible. This ratio is possible if one assumes that Peking has a recessive gene for resistance. As shown in Table 2, the data provide a very acceptable fit to that model. The susceptible reaction of the F_1 plant from the cross Peking (R) x PI 229.315 (S) seems to further substantiate the hypothesis of a recessive gene for PMV resistance in Peking. The expected F_2 genetic ratio from that cross would be 1 R : 3 S. Data are not yet available.

Based on the available data, it appears that PMV resistance in Peking is conditioned by a gene in the recessive state which is independent of the single dominant gene reported by Boerma and Kuhn. While both PI 89.784 and PI 219.789 contain genes which interact with the Peking gene in a similar manner as that from Arksoy, it remains to be shown that they contain the same dominant allele. Investigations on the allelic relationships of sources of PMV resistance are being continued.

References

Boerma, H. R. and C. W. Kuhn. 1976. Inheritance of resistance to peanut mottle virus in soybeans. Crop Sci. 16: 533-534.

Demski, J. W. and C. W. Kuhn. 1977. A soybean disease caused by peanut mottle virus. Ga. Agric. Exp. Sta. Bull. No. 196. P. 5.

Kuhn, C. W., J. W. Demski and H. B. Harris. 1972. Peanut mottle virus in soybeans. Plant Dis. Rep. 56: 146-147.

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1) Cytology of soybean haploid progeny.

Haploids are being isolated annually among individuals obtained from polyembryonic seeds associated with the North Carolina male sterile (ms_1).