

Inheritance of Resistance to the Pea Seed-Borne Mosaic Virus

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ABSTRACT

When *Pisum sativum* plant introductions P.I. 193586 and P.I. 193835, resistant to the pea seed-borne mosaic virus, were crossed with eight susceptible commercial cultivars, the F_1 progenies were all susceptible. Backcross and progeny-tested F_2 segregation ratios fit the hypothesis that resistance to the virus is conditioned by a single recessive gene. Populations

evaluated in F_2 were somewhat variable, but, in general, they support this hypothesis. Both P.I.'s appear to possess the same genetic factor for resistance. We propose that the recessive factor for resistance to pea seed-borne mosaic virus be designated *sbm*.

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The pea seed-borne mosaic virus (PSbMV), sometimes referred to as "pea fizzle-top virus" (2), is potentially an important pathogen of processing pea (*Pisum sativum* L.) because it is readily seed-borne, (3, 4) and also is transmitted by several aphid species (1, 3). The discovery of resistance in two pea plant

introduction accessions (5) provided the basic material needed to begin incorporating resistance into commercially acceptable, PSbMV-resistant, processing peas. This paper reports on the mode of inheritance of resistance. A preliminary report has been made (6).

MATERIALS AND METHODS.—Early generation

TABLE 1. Chi-square analyses of backcross and F₂ populations of peas segregating for reaction to the pea seed-borne mosaic virus

Cross	Number of plants observed ^a				X ²	df	Probability
	Susceptible	Segregating	Resistant	Total			
<u>Backcross plants</u>							
P.I. 193835 × (P.I. 193835 × 'New Season')	4	0	5	9	0.11	1	.75-.50
P.I. 193586 × (P.I. 193586 × New Season)	9	0	12	21	0.43	1	.75-.50
P.I. 193586 × (P.I. 193586 × 'Alsweet')	6	0	7	13	0.08	1	.90-.75
Combined	19	0	24	43	0.58	1	.50-.25
Heterogeneity					0.04	2	.99-.97
<u>Classification of F₂ plants on the basis of their progeny reaction</u>							
New Season × P.I. 193586	14	22	9	45	1.13	2	.75-.50
'New Era' × P.I. 193835	12	19	8	39	0.85	2	.75-.50
Combined	26	41	17	84	1.98	2	.50-.25
Heterogeneity					0.00	1	1.00

^a Expected ratio 1:0:1 in backcross and 1:2:1 in F₂.

seed production and disease evaluations were made in the greenhouse. Air conditioned greenhouses were used in the summer.

Two resistant plant introduction (P.I.) lines, P.I. 193586 and P.I. 193835 (5), were used as sources of resistance. The two lines were crossed reciprocally with each other and with several commercial cultivars and experimental pea lines (Tables 1 and 2). F₁ and F₂ plants from these matings, and in certain crosses the backcross and F₃ progenies, were evaluated for resistance, with the parental lines as controls.

The disease reactions of progeny and parental plants were determined from seedlings inoculated with infective pea plant sap when plants were 10- to 15-cm tall. At least two well-expanded leaflet pairs at each of two different node positions were dusted with 400-mesh Carborundum powder, and the virus inoculum then rubbed on the leaves with the forefinger. Initial disease notes were taken one week after inoculation when infected plants showed systemic vein clearing and characteristic curling of the tendrils. Symptomless plants were reinoculated once, and sometimes twice, to further test their resistant classification.

RESULTS AND DISCUSSION.—Susceptibility or resistance of the parent lines, where several plants were involved, was relatively easy to ascertain, although symptom expression on individual plants varied somewhat according to the age and titer of inoculum, age of plants at time of inoculation, and length of time after inoculation when evaluations were made. Thus, it was not always possible at any given time to be certain of the resistance or susceptibility of each individual plant.

All 81 F₁ plants derived from crosses between the two P.I. lines and eight commercial cultivars were classified as susceptible to the virus, indicating that resistance to the pea seed-borne mosaic virus is conditioned by a recessive factor or factors. F₁'s from crosses between the two resistant P.I.'s were all resistant. All but three of 92 F₂ plants derived from reciprocally intercrossing the two P.I.'s were resistant.

We believe these three plants were misclassified because, as mentioned previously, a symptom expression was not consistently clear. Therefore, we conclude that these two P.I. lines possess the same gene(s) for resistance.

Plants from backcrosses of F₁ plants to the resistant parent segregated in a ratio of one resistant to one susceptible; whereas, F₂ plants, scored on the basis of their progeny reactions, segregated in a ratio of one susceptible:two segregating:one resistant (Table 1). These segregations fit the hypothesis that resistance is dependent upon the homozygous condition of a single recessive gene pair.

If resistance is conditioned by a single recessive gene, then F₂ progeny from a cross of resistant by susceptible parental lines should segregate in the ratio of three susceptible to one resistant. The segregation patterns observed from several different crosses are presented in Table 2. Progeny within crosses involving P.I. 193835 did segregate in the expected ratio, with one exception. This was in the P.I. 193835 × 'Alsweet' cross. Data from the reciprocal cross, however, which was tested simultaneously, fit the expected 3:1 ratio. It is possible that resistant plants were incorrectly classified as susceptible, although a more logical explanation is that a susceptible P.I. 193835 plant was inadvertently used in making the initial cross. (Occasional susceptible plants have been observed in both the P.I. 193835 and P.I. 193586 lines.) Because of the possibility that a susceptible P.I. plant may have been used in the crosses, both the P.I. 193835 × Alsweet cross and its' reciprocal were excluded from the combined data shown in Table 2. Heterogeneity chi-square values between reciprocal crosses are shown in Table 2 only when they exceeded the 5% level of probability.

Considerably more variability was observed in the progeny reaction of the crosses involving P.I. 193586: F₂ progenies of seven of the 11 crosses segregated as expected (three susceptible:one resistant). However, in the remaining four crosses the observed segregation failed to fit the 3:1 ratio. Deviations from

TABLE 2. Chi-square analyses of F₂ populations of peas segregating for reaction to the pea seed-borne mosaic virus

Cross	Number of plants			X ²	df	Probability
	Susceptible	Resistant	Total			
'New Season' × P.I. 193835	70	15	85	2.45	1	.25 - .10
P.I. 193835 × New Season	48	17	65	0.05	1	.90 - .75
L No. 1493 × P.I. 193835	32	7	39	1.03	1	.50 - .25
P.I. 193835 × L No. 1493	38	12	50	0.03	1	.90 - .75
L No. 993 × P.I. 193835	104	25	129	2.17	1	.25 - .10
P.I. 193835 × L No. 993	79	19	98	1.65	1	.25 - .10
'Dark Skin Perfection' × P.I. 193835	65	16	81	1.19	1	.50 - .25
P.I. 193835 × Dark Skin Perfection	85	41	126	3.82	1	.10 - .05
Heterogeneity				4.30	1	.05 - .02
'Alsweet' × P.I. 193835	74	15	89	3.15	1	.10 - .05
P.I. 193835 × Alsweet	80	1	81	24.40	1	<.005
Heterogeneity				5.52	1	.02 - .01
'Sprite' × P.I. 193835	72	33	105	2.31	1	.25 - .10
P.I. 193835 × L No. 1183	311	87	398	2.09	1	.25 - .10
P.I. 193835 as male combined ^a	343	96	439	2.30	1	.25 - .10
Heterogeneity				6.86	4	.25 - .10
P.I. 193835 as female combined ^a	561	176	737	0.49	1	.50 - .25
Heterogeneity				7.14	4	.25 - .10
All P.I. 193835 crosses combined ^a	904	272	1,176	2.20	1	.25 - .10
Heterogeneity				14.60	9	.25 - .10
Dark Skin Perfection × P.I. 193586	183	88	271	8.07	1	<.005
P.I. 193586 × Dark Skin Perfection	134	65	199	6.23	1	.02 - .01
L No. 1493 × P.I. 193586	16	8	24	0.89	1	.50 - .25
P.I. 193586 × L No. 1493	55	31	86	5.60	1	.02 - .01
L No. 993 × P.I. 193586	18	7	25	0.12	1	.75 - .50
P.I. 193586 × L No. 993	50	21	71	0.79	1	.50 - .25
Alsweet × P.I. 193586	94	16	110	6.41	1	.02 - .01
P.I. 193586 × Alsweet	118	27	145	3.15	1	.10 - .05
Sprite × P.I. 193586	123	32	155	1.57	1	.25 - .10
P.I. 193586 × Sprite	62	29	91	2.29	1	.25 - .10
Heterogeneity				3.85	1	.05 - .02
P.I. 193586 × Sprite	61	12	73	2.85	1	.10 - .05
P.I. 193586 as male combined	434	151	585	0.21	1	.75 - .50
Heterogeneity				16.85	4	<.005
P.I. 193586 as female combined	480	185	665	2.82	1	.10 - .05
Heterogeneity				18.09	5	<.005
All P.I. 193586 crosses combined	914	336	1,250	2.36	1	.25 - .10
Heterogeneity				35.62	9	<.005
All crosses combined ^a	1,818	608	2,426	0.00	1	>.90
Heterogeneity				54.76	20	<.005

^a Does not include Alsweet × P.I. 193835 and reciprocal crosses.

expectation were not consistent, however, as in some crosses an excess of susceptible plants was observed and in other crosses a deficiency. This is apparent from the heterogeneity values for the combined data involving P.I. 193586. The deviations may have been due to difficulties in classification as reactions were not always clear. The combined data, whether the P.I. was used as the male or the female, closely fit a 3:1 ratio; but the tests for heterogeneity indicate that the progeny of the individual crosses were not segregating in a homogeneous manner.

Segregations obtained by combining data from all of the crosses involving both P.I.'s (except P.I. 193835 × Alsweet and reciprocal), closely fit a 3:1 ratio. However, data from the individual crosses were not homogeneous as shown by the highly significant chi-square value given in Table 2.

Considering the overall F₂ data, segregation of plants for reaction to the pea seed-borne mosaic virus seems to follow the hypothesis of a single recessive factor for resistance, although there were differences among individual crosses. The segregation ratios of

backcross and progeny-tested F_2 's strongly support the single-factor hypothesis.

We propose that the recessive factor for resistance to pea seed-borne mosaic virus be designated *sbm*. The alternative dominant allele conditioning susceptibility would be *Sbm*. Therefore, the genotype of the two resistant lines used in this study, P.I. 193586 and P.I. 193835, would be *sbmsbm* and that of the susceptible parent lines, *SbmSbm*.

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