Pea Cultivar Susceptibility and Inheritance of Resistance to the Lentil Strain (Pathotype P2) of Pea Seedborne Mosaic Virus

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ABSTRACT. Commercial pea (*Pisum sativum* L.) cultivars, plant introduction (PI) lines, and Oregon State Univ. (OSU) breeding lines were tested for resistance to pathotype P2 (lentil strain) and pathotype P1 (type strain) of pea seedborne mosaic virus (PSbMV) and to bean yellow mosaic virus (BYMV) to assess the relative proportion of resistant and susceptible pea genotypes. Of the 161 commercial cultivars tested, 117 (73%) were resistant and 44 were susceptible to PSbMV-P2. Of these PSbMV-P2-resistant cultivars, 115 were tested for resistance to BYMV and all were resistant. Of the 44 PSbMV-P2-susceptible cultivars, 43 were tested for BYMV susceptibility and all were infected except two, 'Quincy' and 'Avon', both of which were susceptible to a BYMV isolate in another laboratory. Of 138 commercial cultivars inoculated with PSbMV-P1, all were susceptible. All PI lines and OSU breeding lines that were resistant to PSbMV-P1 were resistant also to PSbMV-P2. The high percentage of commercial cultivars resistant to PSbMV-P2 was probably attributable to the close linkage of genes *sbm-2* and *mo* and the widespread use by breeders of BYMV-resistant 'Perfection' and 'Dark Skin Perfection' in developing new pea cultivars. Segregation ratios in progenies of three separate crosses between PSbMV-P2-resistant and PSbMV-P2-susceptible cultivars closely fit the expected 3 susceptible : 1 resistant ratio expected for resistance conferred by a single recessive gene.

Resistance to pea seedborne mosaic virus (PSbMV) has been an important objective of many pea (*Pisum sativum*) breeders since sources of resistance were initially reported (Baggett and Hampton, 1972; Stevenson and Hagedorn, 1971). The early resistance screening of pea germplasm involved the type strain (PSbMV-P1). Several pea breeding lines resistant to PSbMV-P1 have been released from the pea breeding program at Oregon State Univ. (OSU) (Baggett and Hampton, 1977, 1994; Baggett and Kean, 1988).

An additional strain of PSbMV, virulent to peas, was isolated from plant introduction (PI) accessions of lentil (*Lens culinaris*) (Hampton, 1982). Designated PSbMV-L and later renamed pathotype P2 (Alconero et al., 1986), the lentil strain is similar to PSbMV-P1 but is less efficiently seed transmitted, has a primary natural reservoir in *L. culinaris*, and is unable to infect most U.S. pea cultivars (Goodell and Hampton, 1984). Resistance to PSbMV-P2 is distinct from resistance to PSbMV-P1 (gene *sbm-1*; Gritton and Hagedorn, 1975; Hagedorn and Gritton, 1973), being conferred either by gene *sbm-2* or *sbm-3* (Provvidenti and Alconero, 1988a). Gene *sbm-2* is linked with *mo*, which confers resistance to bean yellow mosaic virus (BYMV; chromosome 2). Accordingly, cultivars resistant to BYMV were found by Alconero et al. (1986) to be resistant to pathotype 2 (PSbMV-P2). Gene *sbm-1*, conferring resistance to PSbMV-P1, is linked to genes *sbm-3* and *sbm-4* on chromosome 6 (Provvidenti and Alconero, 1988a, 1988b). Thus, most pea genotypes resistant to PSbMV-P1 would be expected to resist infection by PSbMV-P2.

The objectives of this study were to 1) assess the proportion of

representative pea genotypes susceptible to PSbMV-P2, relative to PSbMV-P1, with emphasis on cultivars from U.S. seed companies; 2) examine the linkage of PSbMV-P2 and BYMV resistance in a wide range of commercial cultivars and *P. sativum* PIs; and 3) reexamine, using distinct germplasm sources, the inheritance of resistance to the original strain of PSbMV-P2 isolated from lentil by Hampton (1982).

Materials and Methods

CULTIVAR SUSCEPTIBILITY STUDY. Each pea accession was inoculated separately with the three virus isolates, BYMV-Scott and the P1 and P2 pathotypes of PSbMV. All tests for resistance were conducted in the greenhouse using mechanical inoculation. The PSbMV-P2 isolate, originating from infected *Lens culinaris* Medic. PI 297772, Pullman, Wash., was maintained in 'Sounder' pea. Our isolate of PSbMV-P1 was obtained from infected pea seeds and maintained on 'Little Marvel' pea. The Scott isolate of BYMV, which belongs to subgroup II of Jones and Diachun (1977), was maintained on 'Red Kidney' bean (*Phaseolus vulgaris* L.). Inoculum was prepared from the youngest symptomatic leaves by macerating them with mortar and pestle in 0.02 M potassium phosphate buffer, pH 7. The macerate was rubbed on pea leaflets that had been dusted with 400-mesh silicon carbide. Each pot contained, in addition to the inoculated plants, a single noninoculated control plant. Plants were first inoculated when leaflets at the first two to three nodes were fully expanded, about 10 to 14 d after planting. Inoculation was repeated usually 5 to 7 d later on newly expanded leaflets. Plants were rinsed with water after inoculation. Greenhouses were maintained at 21 °C day and 15 °C night temperatures. For the inheritance study, a 16-h photoperiod was maintained using supplemental low-spectrum (cool-white) fluorescent lighting (about 50 $\mu\text{mol}\cdot\text{m}^{-2}\cdot\text{s}^{-1}$).

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Table 1. Reactions of PSbMV-P2-resistant (R) and PSbMV-P2-susceptible (S) commercial cultivars to PSbMV-1 and BYMV.

PSbMV-P2	Total	No. of cultivars					
		BYMV			PSbMV-1		
		R	S	Not tested	R	S	Not tested
R	117	115	0	2	0	100	17
S	44	2	41	1	0	38	6
Total	161	117	41	3	0	138	23

Included in the tests were 161 pea cultivars from U.S. seed companies (23 of these were not tested with PSbMV-P1), 32 PI lines, many of which were known to be PSbMV-P1-resistant, and 10 OSU breeding lines known to be resistant to PSbMV-P2. Six to ten seeds of each cultivar were planted per 20-cm pot, leaving one plant per pot as a noninoculated control. All other plants were mechanically inoculated twice. Commercial cultivars that remained symptomless in the initial test were replanted and retested in the same manner. During the second test, cultivars that were not obviously infected, but included abnormal plants, were assayed for the presence of virus by back-inoculation to susceptible plants.

The PI lines were planted and tested twice. In the first test, symptomless lines were observed for 60 d before being discarded. In the second test, every line was assayed by back-inoculation to susceptible plants. Plants inoculated with PSbMV-P1 and PSbMV-P2 were assayed by bulking and macerating approximately equal amounts of tissue from each inoculated plant per line and mechanically back-inoculating about six 'Sounder' plants. The OSU breeding lines were tested twice. Any suspect plants were individually assayed on 'Sounder'. When required, plants were assayed for presence of BYMV by back-inoculation to 'Red Kidney' bean.

INHERITANCE STUDY. PSbMV-susceptible cultivars 'Sounder' and 'Abador' were crossed reciprocally to B445-66, an OSU line previously released as PSbMV-P1-resistant germplasm (Baggett and Hampton, 1977). Similarly, 'Sounder' was crossed to PSbMV-P2-resistant 'Little Marvel'. F_2 and F_3 seeds were produced in the greenhouse and field. F_3 families were derived by saving seeds from random individual F_2 plants. These progenies were tested for susceptibility in the greenhouse using the cultural and test methods described above in the cultivar-susceptibility study. Nine plants were usually tested for each F_3 family.

Based on the results of homogeneity tests, reciprocal crosses were combined for analysis.

Results and Discussion

Space limitations preclude describing the reactions of each commercial cultivar tested. Individuals interested in reactions of specific genotypes should contact the second author. Of the 161 commercial cultivars tested, 117 (73%) were classified as resistant to PSbMV-P2 (Table 1). Of these PSbMV-P2-resistant cultivars, 115 were tested further and found to be resistant to BYMV. Of the 44 PSbMV-P2-susceptible cultivars, 43 were tested for BYMV reaction and all but two, 'Quincy' and 'Avon', were susceptible. The classification of these two is questionable because R. Provvidenti (personal communication) was able to infect them with a separate isolate of BYMV.

INOCULATION RESULTS. All 138 commercial cultivars tested against PSbMV-P1 were found to be susceptible. With few exceptions, pea cultivars derived from PSbMV-P1-resistant PI accessions are still under development.

The PI lines were selected for testing because of reported resistance to PSbMV-P2. These were generally resistant to PSbMV-P2, PSbMV-P1, and BYMV. Matching resistance to BYMV and PSbMV-P2 is predictable by the description of Linkage Group I (Provvidenti and Alconero, 1988a), in which genes *mo* and *sbm-2* are clearly linked on chromosome 2. Resistance to PSbMV-P1, conferred by *sbm-1*, is independent of *sbm-2*, occurring in Linkage Group II on chromosome 6. Resistance to PSbMV-P1 is strongly linked to PSbMV-P2 and PSbMV-P4 resistance, since genes *sbm-3* and *sbm-4* are also contained in Linkage Group II (Provvidenti and Alconero, 1988b).

Nine OSU breeding lines that had been released as sources of PSbMV-P1 resistance (Baggett and Hampton, 1977; Baggett and Kean, 1988) were included. All nine were resistant to all three viruses. All originated from F_2 populations screened only for resistance to PSbMV-P1 and were tested against PSbMV-P2 and

Table 2. Discrepancies among tests for specific virus resistance in plant introduction (PI) lines of *Pisum sativum*.

PI line	Response to inoculation ²			Literature source
	PSbMV-P2	BYMV	PSbMV-P1	
347455	R	R	S (H)	3 ^y
347456	R	R	R (H)	3
347464	R	R (S)	S (H)	1,2,3,4
347465	R	R (S)	S	4
347466	R	R (S)	R (H)	1,2,3,4
347467	R	R (S)	S (R,H)	1,2,3,4
347470	R (H)	S	R	1,3
347484	R	R	R (H)	3
347492	R	R (S)	R (H)	1,2,3,4
357023	R	R	R (H)	3

²Prior results differed from those in this table in having been reported as (H) = heterogeneous seedlot; mixed reactions or (R) = resistance or (S) = susceptible.

^yPrior reports with differing results were 1 = Alconero et al., 1986; 2 = Hampton, 1980; 3 = Hampton, 1986; 4 = Provvidenti, personal communication.

BYMV only after they were in advanced generations. The proportion of PSbMV-P2- or BYMV-susceptible plants in early generations is not known.

The high percentage of commercial cultivars resistant to PSbMV-P2 in our tests cannot be the result of intentional selection by pea breeders because PSbMV-P2 has not been of importance in peas and has generally been unknown to commercial breeders until recently. The prevalence of PSbMV-P2 resistance is instead attributable to the linkage of gene *sbm-2* with *mo*, conferring BYMV resistance. Resistance to BYMV has long been recognized as a positive factor in pea breeding and its prevalence indicates widespread use by breeders of Perfection-type canning peas and the freezing pea 'Dark Skin Perfection' in developing new commercial processing cultivars. Most of the Perfection-type cultivars developed in the United States are resistant to BYMV (Hagedorn, 1951).

The susceptibility of all 138 commercial cultivars to PSbMV-P1 indicates that all derive PSbMV-P2 resistance from gene *sbm-2*, not *sbm-3*. The susceptibility of all to PSbMV-P1 indicates the presence of gene *Sbm-3* (PSbMV-P2-susceptible), in phase with gene *Sbm-1*, in Linkage Group II.

Our results sometimes disagreed with other reports of screening tests, perhaps due to within-line heterogeneity, seed mixtures of PI accessions (Hampton, 1980), or incorrect number assignments during repository seed increases; differing inoculation methods; or distinct virus isolates tested. Of 32 PI lines tested, none became infected, whereas 2 had been reported susceptible by others (Table 2). We found one susceptible to BYMV, five resistant but disputed, and the remaining twenty-six resistant and undisputed. Twenty-eight of the thirty-two lines were resistant to PSbMV-P1, with five disputed, and four susceptible with three disputed. It should be noted that PI lines of peas are almost always very tall types and difficult to test, often producing mild, easily masked symptoms.

INFLUENCE OF INOCULATION SUCCESS ON IDENTIFYING RESISTANT BREEDING PROGENIES. Although PSbMV symptoms induced in

susceptible parents were conspicuous, 100% infection rates were never achieved, even with two successive PSbMV-P2 inoculations. The proportions of plants successfully infected with PSbMV-P2 in studies were 'Sounder', 142/160 (89%); 'Abador', 79/91 (87%); and 'Avon', 95/102 (93%). Consequently, it must be assumed that plants escaped infection in all populations tested. This assumption was applied to classifications of F_3 families, specifically in discerning homozygous susceptible (S) or segregating (SE) families. Families classified as homozygous resistant (R), however, were unlikely to comprise S or SE populations, a misclassification requiring that all plants of S or SE populations escape infection, even after two inoculations.

Because of lower than 100% infection rates in inoculated plant populations, we did not attempt differentiation between S and SE families. Instead, S and SE F_3 families were combined (S + SE), compared numerically to resistant (R) families, and tested for fit against the hypothetical 3:1 ratio.

INHERITANCE OF RESISTANCE TO PSbMV-P2. PSbMV-P2 susceptibility of F_1 plants and segregation ratios of populations from all three crosses (Table 3) indicate that resistance to PSbMV-P2 is due to a single recessive allele, confirming the results of Provvidenti and Alconero (1988a). Chi-square values for segregating populations were nonsignificant at $P = 0.05$, indicating that none of the observed segregation ratios deviated from that expected for a single recessive gene. F_3 data, when the 3 (S + SE) : 1 (R) ratio was tested against the 3:1 hypothetical ratio, confirmed the F_2 segregation ratio.

A few Ps (susceptible parents) and F_1 plants typically escaped infection. Accordingly, some S : R ratios in segregating populations deviated from the expected ratio, with excesses of noninfected plants, but some populations contained an excess of infected plants.

All PSbMV-P2-inoculated plants of resistant parents remained free of infection.

Table 3. Segregation for susceptibility (S) and resistance (R) to PSbMV-P2.

Cross	Generation ^y	No. of plants or families ^z				Ratio tested	Chi square
		Observed		Expected			
		S	R	S	R		
Sounder x B445-66	PS	97	7				
	PR	0	104				
	F ₁	87	7				
	F ₂	169	63	174.00	58.00	3:1	0.57 ^{NS}
	F ₃	260	88	261.00	87.00	3:1	0.02 ^{NS}
	BCR	69	60	64.50	64.50	1:1	0.63 ^{NS}
	BCS	41	0				
Abador x B445-66	PS	79	12				
	PR	0	92				
	F ₁	54	1				
	F ₂	176	55	173.25	57.75	3:1	0.17 ^{NS}
	F ₃	284	101	288.75	96.25	3:1	0.31 ^{NS}
Sounder x Little Marvel	PS	24	0				
	PR	0	23				
	F ₁	21	4				
	F ₂	158	69	170.25	56.75	3:1	3.53 ^{NS}
	F ₃	143	52	146.25	49.75	3:1	0.17 ^{NS}
	BCR	40	45	42.50	42.50	1:1	0.29 ^{NS}
	BCS	49	4				

^zSegregating and homozygous F_3 families were not distinguished; the ratio tested for F_3 families was thus 3 susceptible + segregating:1 resistant.

^yPS = susceptible parent, PR = resistant parent, BCR = backcross to resistant parent, BCS = backcross to susceptible parent.

^{NS}Nonsignificant deviation from the expected ratio at $P = 0.05$.

Both resistant parents, 'Little Marvel' and B445-66, were resistant to PSbMV-P2 and BYMV; thus, each would possess gene *sbm-2*, in Linkage Group I. However, only B445-66 was also resistant to PSbMV-P1, thus, also possessing gene *sbm-3*, in Linkage Group II [i.e. possesses both genes conferring resistance to PSbMV-P2, as found for 'Bonneville' and PSbMV-P2-resistant PI lines (Provvidenti and Alconero, 1988)]. We assume that resistance to PSbMV-P1 and PSbMV-P2 in B445-66 came from PI 269774, its pedigree source of PSbMV resistance (Baggett and Hampton, 1977).

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