AN ABSTRACT OF THE THESIS OF

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Title: Inheritance of Resistance to the Lentil Strain of Pea Seedborne Mosaic Virus in Pisum sativum L.

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The inheritance mode of pea-resistance to the Lentil Strain of Pea Seedborne Mosaic Virus (PSbMV-L) was studied. In addition, commercial pea cultivars, Plant Introduction (P.I.) lines and Oregon State University (OSU) breeding lines were tested for susceptibility to this virus. These lines were also tested for their reaction to Bean Yellow Mosaic Virus (BYMV) and pathotype 1 of Pea Seedborne Mosaic Virus (PSbMV-P1) to further elucidate the relationships of Pisum resistance to the three viruses.

Susceptible cultivars, 'Sounder', 'Abador', 'Quincy' and 'Avon' were hybridized with resistant cultivars, 'Little Marvel' and OSU B445-66. Data on reaction to PSbMV-L inoculation were obtained for F1, F2, and F3 progeny and selected backcrosses. F1 tests indicated that resistance was recessive. Most F2 populations segregated as ratios of 3 susceptible: 1 resistant indicating that a single recessive gene controls resistance. Populations of most F3 families fit ratios of 3 susceptible or segregating: 1 resistant. The F3 family data fit ratios of 1 susceptible: 2 segregating: 1 resistant in some of the tests. In remaining tests, failure to fit this expected ratio was assumed to be due to random escapes (susceptible, but not infected), or to a klendusic reaction in progenies from 'Quincy'. Segregation of backcrosses from two crosses in a 1 susceptible: 1

resistant ratio supported the single recessive gene hypothesis.

Of 202 commercial cultivars screened screened for PSbMV-L resistance, 144 were resistant and 58 were susceptible. Cultivars resistant to PSbMV-L were also resistant to BYMV, and cultivars susceptible to PSbMV-L were susceptible to BYMV except for 'Quincy' and 'Avon', which were susceptible to PSbMV-L but resistant to BYMV. All of 9 OSU lines and all 32 of selected P.I. lines tested were resistant to PSbMV-L. The P.I. lines had been previously tested and were known to be resistant to PSbMV-P1. All 9 OSU breeding lines and all but one P.I. line were resistant to BYMV, indicating a possible linkage relationship between resistance to PSbMV-L and BYMV. Three of 178 cultivars, 27 of 32 P.I. lines, and all of 9 OSU breeding lines tested were resistant to both PSbMV-P1 and PSbMV-L. The results suggested that there is a tendency for resistance to PSbMV-P1 and resistance to PSbMV-L to be associated in pea cultivars.

Inheritance of Resistance to the Lentil Strain of Pea Seedborne Mosaic Virus in *Pisum sativum* L.

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INTRODUCTION

Pea seedborne mosaic virus (PSbMV), first identified in 1966 in Czechoslovakia (Musil,1966), has since been reported in several countries throughout the world (Bos, 1970b; Fry, 1980; Hampton, 1969; Inouye, 1967; Mink et al., 1969; Stevenson and Hagedorn, 1969). International transportation and exchange of pea seed, and the seedborne nature of this virus creates a potential problem for breeders and seed producers.

PSbMV-infected pea seed is the principal means by which the virus perennates, is widely distributed, and is established in field plantings. Field inoculum from seed-infected plants is disseminated locally by aphid spp. vectors. Although PSbMV can be detrimental to a commercial pea crop by decreasing yield, it is the seed producers and plant breeders who are most affected by contamination of the seed crop.

PSbMV-infected plants are at times difficult to detect visually. Depending on the host genotype, disease expression may range from symptomless to whole-plant necrosis. Visual disease detection may also fail when PSbM symptoms are confused by other disease symptoms. Because PSbMV may escape visual detection, infected seed may be inadvertantly saved. Although industry-wide control efforts have been helpful, PSbMV has been detected in peas growing in the Pacific Northwest as recently as the summer of 1987.

A distinct strain of PSbMV, designated the lentil strain (PSbMV-L), was discovered in 1982 as a contaminant in the U.S. Plant Introduction collection of lentils (*Lens culinarus*) at Pullman, Washington (Hampton, 1982). Although generally resembling the type strain (PSbMV-P1),

there were a number of dissimilarities. Most importantly for this study, the lentil strain differs from the type strain in pea genotypic resistance as indicated by differential reactions in pea cultivars. It was observed that the pea host range of PSbMV-L was more limited than that of PSbMV- P1. It has been reported that pea cultivars resistant to Bean Yellow Mosaic Virus (BYMV) are also resistant to PSbMV-L (Goodell and Hampton, 1983). In essence, PSbMV-L resistance in peas was not conferred by the sbm-1 gene which confers resistance to PSbMV-P1, and PSbMV-L resistance was associated with the mo gene conferring resistance to BYMV.

The purpose of this study was to determine the mode of inheritance of resistance to PSbMV-L in peas, to test pea cultivars, Plant Introduction (P.I.) lines, and Oregon State University (OSU) breeding lines for their reaction to PSbMV-L, and to test their reaction to BYMV and PSbMV-P1, to clarify host resistance relationships among the three viruses.

REVIEW OF LITERATURE, RELEVANT VIRUSES

Pea Seedborne Mosaic Virus-P1

The presence of Pea Seedborne Mosaic Virus was initially reported in Czechoslovakia (Musil, 1966). Subsequently, it was recognized in Japan (Inouye, 1967), in the U.S. in 1968 (Hampton, 1969; Mink et al., 1969; Stevenson and Hagedorn, 1969), and in The Netherlands (Bos, 1970). Canadian pea breeding lines were discovered to be infected in 1974 (Hampton et al., 1976), and in New Zealand, seed produced in the 1978 growing season was found to be contaminated with the virus (Fry, 1980). In order to clarify the relationships among various isolates, seven originating from Czechoslovakia, Japan, The Netherlands and the U.S. were compared, and found to be variants of the same virus (Hampton et al., 1981). Initially referred to as Pea Leafrolling Virus, Pea Fizzletop Virus, Pea Leafroll Mosaic Virus, and Pea Seedborne Mosaic Virus, the name Pea Seedborne Mosaic Virus was formally adopted in 1974 (Mink et al., 1974).

Particles of PSbMV, a member of the Potato Virus Y group, contain 5% single-stranded RNA (Knesek et al., 1974) and comprise modal lengths approximating 770 nm (12 nm diameter) (Hampton et al., 1974). Initial reports of the virus suggested that particles of some isolates were shorter than this but it was found that breakage was occurring in the electron microscope preparatory process unless 3.5% gluteraldehyde was included as a fixative. Most PSbMV isolates produce pinwheel inclusions in leaf cells but rarely in root cells (Hampton, 1973; Inouye, 1967). Infected plant juice is highly infective when initially extracted from plants but loses its infectivity within 24 hours when extracted from plant leaves and 96 hours when extracted from root tissue (Knesek et al., 1974). When leaf tissue is diluted to 10^{-1} or 10^{-2} it

remains infective for at least 96 hours suggesting the presence of inhibitors in leaf tissue. The dilution end-point is between 10^{-3} and 10^{-4} and thermal inactivation point is 55 degrees. It loses its ability to infect with freezing.

Procedures for virus purification were described by Knesek et al. (1974) and Hamilton and Nichols (1978). Physico-chemical properties of virus particles include a sedimentation coefficient of 148±1 S and a buoyant density in CsCl of 1.329 g/cm^3.

The virus is stylet borne by at least nine aphid species including Myzus persicae (Sulzer), the green peach aphid, Acyrthosiphon pisum (Harris), the pea aphid, Macrosiphon euphorbiae (Thomas), the potato aphid, Dactynotus escalantii (Knowlton), Macrosiphon rosae (L.), Ovatus crataegarius (Walker), and Rhopalosiphum padi (L.) (Aapola and Mink, 1973; González and Hagedorn, 1971). An acquisition time of less than 30 seconds is required and infectivity is lost in five minutes with the pea aphid and 30 minutes with the green peach aphid.

Symptoms of PSbMV in mechanically inoculated pea plants first appear as a transient vein clearing followed by a light leaf mottle and distortion. Plants become stunted, due to progressive shortening of internodes, and eventually culminate in a tight rosette (Mink et al., 1969). Symptoms in mechanically inoculated Plant Introduction lines range from whole plant necrosis to slight vein clearing and leaf roll. Intermediate symptoms consist of stunting, swollen veins, and distorted leaves and tendrils. These differences in symptom intensity appear to be due to modifying genes (Hampton, 1980a).

Pea plants infected through the seed exhibit symptoms with severity ranging from traces of vein swelling but an otherwise healthy appearance to marked

stunting, vein clearing and downward rolling of leaf margins (Hampton, 1972).

Field symptoms of PSbMV include plant stunting, leaf size reduction, distortion of leaves, thickened and tightly rolled leaves, and rosetting. In addition, leaves tend to be color cast and may remain green after healthy plants have senesced. These plants also were inclined to resist frost damage (Hampton and Baggett, 1970; Mink et al., 1969).

The host range of PSbMV includes at least 47 plant species from 12 families but only a few are categorized as being highly susceptible (Aapola et al., 1974). These included Chenopodium foetida Schrad., Lathyrus odoratus, Lens culinaris Medik., Pisum sativum, and 20 Vicia ssp. Recently chickpea, Cicer arientinum, was added to the list of species susceptible to PSbMV (Alconero, 1986). In Wisconsin, one highly susceptible species, Vicia villosa, was studied for its potential as an overwintering host, and was found to be effective under experimental conditions, however, PSbMV was not seed-transmitted in this host (Stevenson and Hagedorn, 1973b).

High temperatures under greenhouse conditions may accelerate symptom development, although overall intensity remains constant over a temperature range from 16 to 28 degrees C. (Stevenson and Rand, 1970). In the field, low temperatures seem to decrease the level of symptom expression although it was not known if this was due to less infectivity of the virus or some other factor (Kraft and Hampton, 1980).

Studies have been made on the effect of PSbMV on yield. In single row tests of two field pea cultivars, yield was decreased, with yield reduction attributed to smaller seed (Chiko and Zimmer, 1978). A number of PSbMV- infected pea cultivars were tested under plot conditions for their yield response (Kraft and Hampton, 1980). In general, results indicated that yields of

fresh peas and dry seed were reduced only when young plants were infected and virus incidence was high. When cultivars were tested using the New Zealand strain of PSbMV, it was found that, generally, yield reduction was not significant (Ovendon and Ashby, 1981). Only one out of seven susceptible cultivars had a significant yield reduction and another one out of seven produced significantly smaller seed when infected.

Seed transmission rates vary widely. The ability to transmit PSbMV through seed appears to be cultivar specific with some cultivars not transmitting through seed at all. In general, the Alaska and Alsweet pea types have the highest rate of seed transmission although exceptions exist (Stevenson and Hagedorn, 1970). Most infected seed has originated in breeding plots but may also occur in commercial seedlots (Fry, 1980; Hampton et al., 1976; Knesek and Mink, 1970; Mink and Parsons, 1978).

PSbMV was found associated with the embryo, endosperm and testa of immature seeds and with the embryo and cotyledons in mature seeds with growth cracks (Stevenson and Hagedorn, 1973a). It was associated with all parts of the pea inflorescence including pollen, although tests indicated a very low percent (0.85%) of virus transmission through pollen. PSbMV has been recovered from dry seed stored for a year (Knesek and Mink, 1970) and more than 10 years by others (Hampton, unpublished).

Techniques were developed to recognize seed infected with PSbMV. One study indicated that in Perfection-type peas, seed size did not seem to have an effect on seed transmission, however, seeds having growth cracks in the seed coat had a significantly higher level of virus transmission (Stevenson and Hagedorn, 1970). In another study, two pea lines were examined for differences in

seed appearance between infected and noninfected (Mink and Parsons, 1978). In a round seeded line most infected seed was wrinkled. In the other line, a flat-seeded variety, the highest incidence of infected seed was in small seeds and normal looking seeds.

In addition to seed transmission through peas, the virus is also seed transmitted through lentils (Hampton and Muehlbauer, 1977), and at a low rate through Vicia articulata, V. narbonensis and V. pannonica (Hampton and Mink, 1975).

When 1835 Plant Introduction lines from the Northeastern Regional Plant Introduction Station at Geneva, NY were screened for infected seed, 420 were found to be infected (Hampton and Braverman, 1979). Most of these had been introduced or had become infected by aphid transmission from introduced lines during seed-increase plantings after 1969. Elimination of the virus from introduced lines through the selection of virus-free plants has been demonstrated (Hampton, 1983). Alconero et al. (1985) reported that the elimination of infected seed from the U.S. Plant Introduction collection could affect the genetic diversity of some pea lines and recommended the use of tissue culture for virus elimination. However, selection of virus-free plants has been successful and PSbMV-P1 is expected to be eliminated from the Pisum P.I. collection by 1990 (Hampton, personal communication).

Sources of resistance to PSbMV in peas were found within the Pisum Plant Introduction collection (Baggett and Hampton, 1972; Stevenson and Hagedorn, 1971). P.I. 193585 and 193586 were utilized as resistant parents in an inheritance study conducted by Hagedorn and Gritton (1973). Resistance to PSbMV-P1 in peas was found to be conferred by a single recessive gene designated sbm Confirmation of this information was made by Hampton and Marx (1981). Provvidenti and Alconero (1988a) have

recently referred to this gene as sbm-1. Linkage studies with the wlo gene indicate that the sbm-1 gene is located in Pisum linkage group VI (chromosome 6) (Gritton and Hagedorn, 1975).

The recessive allele of the Sbm gene is quite common in the U.S. Plant Introduction collection, but with many lines being mixtures of susceptible and immune individuals (Hampton, 1980b). Both sources of PSbMV-P1 immunity and inoculum seem to have originated in P.I. lines from northern India, leading to the hypothesis that this area may be the center of origin for the virus (Hampton, 1986)

Alconero et al. (1986) compared three PSbMV isolates, P-1 and P-4 from pea, and L-1 from Lens Plant Introduction accessions. All three reacted similarly in serological tests, were morphologically indistinguishable, and were generally alike in host range (although differences in symptom expression occurred), and seed transmission capabilities. Differences in pea cultivar host range and timing of the onset of symptoms resulted in their being categorized as pathotypes of PSbMV. P-1 is the typical PSbMV pathotype most often encountered in infected pea cultivars and producing typical stunting, leafroll, and mosaic symptoms. P-4 differs from P-1 in two ways: first, symptoms were delayed in about half of the pea cultivars tested and these latent types were cultivars resistant to BYMV; and secondly, two P.I. lines resistant to pathotype P-1 pathotype were susceptible to pathotype P-4 . The L-1 strain differed from pathotypes P-1 and P-4 in having lentil as its natural inoculum reservoir and infecting only pea cultivars which did not produce delayed symptoms when inoculated with pathotype P4. These same cultivars were susceptible to BYMV. L-1 and PSbMV-L have the same inoculum reservoir and pea host range and have been

classified as belonging to pathotype 2 (P2). Provvidenti and Alconero (1988a) have reported that the gene conferring resistance to P2, designated sbm-2 is linked to mo on chromosome 2. A second gene conferring resistance to P2 but not linked to mo was designated sbm-3. In a recent personal communication, Provvidenti has indicated that sbm-3 is linked closely with wlo on Chromosome 6 and confers resistance to all known pathotypes of this virus group. Resistance to pathotype 4 was also found to be conferred by a gene designated sbm-4 (Provvidenti and Alconero, 1988b). Recent personal communication with Provvidenti indicates that sbm-4 is also found on Chromosome 6.

The New Zealand isolate of PSbMV , PSbMV-N.Z., was initially found in the pea cultivar `Pamaro' and has been referred to as PSbMV-Pam (Ashby et al., 1986). Symptoms of PSbMV-N.Z. have been reported to be very mild and sometimes latent. Resistance to this isolate was reported to be associated with the mo gene for resistance to BYMV (Goodell and Hampton, 1983). It is classified with the L-1 strain (P2) (Alconero et al., 1986) Serologically, using direct ELISA, it was possible to differentiate the New Zealand isolate from a typical PSbMV (PSbMV-P1) isolate, but not from PSbMV-L (Ashby et al., 1986).

Pea Seedborne Mosaic Virus-Lentil

After lentils were found to be capable of seed transmitting PSbMV, Lens culinarus Plant Introduction accessions were screened for infected seed (Hampton, 1982). A seedborne virus was isolated. Based on symptomology in lentil and pea cultivars, serology using SDS-gel tests, and immunosorbant electron microscopy this virus appeared to be typical PSbMV. However, distinct differences were discovered between this newly found virus, designated PSbMV-L, and PSbMV-P1. First,

lentils were the natural inoculum reservoir for PSbMV-L while peas were the type strain inoculum reservoir. Pea genetic distinctions were also evident. Resistance to PSbMV-L proved to be independent of the gene for resistance to PSbMV-P1 in both lentils and peas. The lentil strain, like PSbMV-L1 and PSbMV-NZ, was non-infective to pea cultivars containing the homozygous recessive form of the Mo gene conferring BYMV resistance, and was infective to cultivars susceptible to BYMV. Because the lentil strain resembles PSbMV but differs in pathogenicity in the same manner as the L-1 strain, it has been referred to as a variant of L-1 (Alconero et al., 1986) and as belonging to Pathotype P2. PSbMV-L differs from the L-1 strain in its symptom severity in pea cultivars, with the lentil strain being less virulent.

Other distinctions between PSbMV-L and the typical PSbMV were found (Hampton, 1982). With ELISA, they differed quantitatively when using PSbMV antiserum. Additionally, PSbMV-L virus particles were likely to break when techniques appropriate for PSbMV were used in virus purification and electron microscopy.

Transmission of PSbMV-L through seed and by aphids was studied by Goodell and Hampton (1984). Based on results from a single pea cultivar, PSbMV-L appears to be transmitted through the seed less efficiently than typical PSbMV. Using the pea aphid, Acyrthosiphon pisum, PSbMV-L was found to be aphid transmissible in a trimodal manner indicating that the virus could survive and be spread farther than if it were transmitted in a typically non- persistant manner. As temperatures increased, the amount of virus transmitted through aphids decreased, although the reason was not clear.

Bean Yellow Mosaic Virus

BYMV, first described in 1934, has a worldwide distribution and a wide host range especially within the families Leguminosae and Liliacae (Bos, 1970a). It is also known as Bean virus 2, Phaseolus virus 2, Gladiolus mosaic virus and Pea mosaic virus.

Over 20 aphid species transmit BYMV in a nonpersistant manner (Bos, 1970a).

Symptoms of BYMV in peas range from a light green mottle to a bright yellow mosaic to necrosis depending on the virus strain (Bos, 1970b; Bos et al., 1974). Infected pea plants exhibit little or no height reduction (Ford and Baggett, 1965).

The environment, especially temperature, has an affect on BYMV symptom expression. Schroeder et al. (1966) found that plants heterozygous for the Mo gene express disease symptoms only under a high temperature regime. Swenson (1968) found that after aphid inoculation with BYMV, more pea plants showed symptoms when post-inoculation temperatures were maintained at 30 C. than when maintained at lower temperatures. Light and nutrient levels, however, did not consistantly affect the rate of infection. Thermal isolates, selected for when high temperature regimes suppressed or destroyed the type strain, were reported to produce mild BYMV symptoms in BYMV-resistant peas (Provvidenti and Schroeder, 1963).

The original Perfection-type peas were considered immune to BYMV but some new cultivars of this type were later discovered to be susceptible. The `loss of immunity' was attributed to incorporation of susceptible germplasm into breeding programs rather than to circumvention of immunity by the virus (Ford, 1963).

Resistance to BYMV in peas was found to be conferred by a single recessive gene designated mo (Johnson and Hagedorn, 1958; Yen and Fry, 1956). Studies have linked

mo with *Wb*, *K*, and the isozyme marker, *Pgm-p*, thus placing the gene on chromosome 2 (Weeden et al., 1984; Yen and Fry, 1956).

BYMV was divided into three general groups based on biological and serological relationships by Bos et al. (1974) and Jones and Diachun (1977). In accordance with these workers I have considered Group I to be the severe forms, including the isolates referred to as the pea necrosis strain (Bos,1970b), clover yellow vein virus (Hollings and Nariani, 1965), and isolates inducing necrosis in beans (Tatchell and Baggett, 1985); Group II to be the typical BYMV strains, including the Scott isolate used in this study which induces a yellow mosaic in both beans and peas; and Group III to include the Pea Mosaic strains which are non-infectious to most bean cultivars. Based on RNA comparisons and newly found genetic differences, some scientists regard these three groups as being three distinct viruses (Provvidenti, 1987; Reddick and Barnett, 1983; Weeden et al., 1984).

MATERIALS AND METHODS

Description of Viruses

All three viruses used in this study were originally provided by Dr. R.O. Hampton, U.S.D.A., Corvallis, Oregon, as fresh or dried inoculum, and subsequently maintained on susceptible hosts in the greenhouse.

The PSbMV-L isolate originated from infected Lens P.I. 297772 at Pullman, Washington. For this study, it was maintained in 'Sounder' peas. Symptoms of PSbMV-L infection include pronounced plant stunting, leaf distortion and mottle, and pods containing no seeds or a few small, cracked seeds (Figure 1). Infected plants have a tendency to proliferate basal branches and to senesce later than healthy plants.

The Scott isolate (BYMV-Scott) of BYMV used in the study was originally typed by W.J. Zaumeyer and later provided by Dr. R.O. Hampton. It belongs to the Subgroup II (or 'type' category) as classified by Jones and Diachun (1977). Symptoms in peas induced by BYMV-Scott include a bright yellow mosaic with little plant stunting or overall distortion (Figure 2). Symptoms in beans include a slight stunting and a typical yellow mosaic on leaves. BYMV-Scott was maintained in 'Red Kidney' beans or 'Sounder' peas.

The SL25 isolate of PSbMV utilized in this study was originally isolated from infected peas from Canada. It belongs to the P1 pathotype classification of Alconero et al. (1986). Symptoms of PSbMV-P1 infection include an initial vein clearing 7 days after mechanical inoculation, followed by stunting, downward leaf roll and a light mosaic (Figure 3). Under conditions used in this study, PSbMV-P1 symptoms were less severe than symptoms of PSbMV-L. PSbMV- P1 was maintained in the greenhouse in either 'Sounder' or 'Little Marvel' peas.

Figure 1. Severe stunting and leaf distortion in 'Sounder' peas inoculated with PSbMV-L

Figure 2. Bright yellow mosaic in pea plants inoculated with a Group II isolate of BYMV

Figure 3. Plant stunting and leafroll in 'Sounder' peas inoculated with PSbMV-P1

Parental Lines

Choice of parents for the inheritance study was based on their reactions to PSbMV-L, PSbMV-P1, and BYMV in preliminary tests (Table 1).

'Sounder', a determinate, freezer-type pea, was developed by Rogers Bros. Seed Company. It served both as a susceptible parent and a susceptible check variety because it produced moderate to severe symptoms when inoculated with any of the three viruses used in the study. It was also used to maintain a source of inoculum for the tests because of its high resistance to powdery mildew which was a problem under greenhouse conditions.

'Abador', a cultivar developed by Asgrow Seed Company has small, pale green leaves and seeds. It is susceptible to many diseases in the greenhouse, including root rots, powdery mildew, and all three of the viruses used in this study. 'Abador' was used as a susceptible parent in the inheritance study.

'Little Marvel', an early, determinate old variety is a popular garden pea of the freezing type. In the inheritance study it was used as a parent resistant to PSbMV-L. Since 'Little Marvel' is resistant to PSbMV-L and BYMV but susceptible to PSbMV it was included in all PSbMV- L tests as a check for contamination of PSbMV-L by PSbMV- P1. 'Little Marvel', along with 'Sounder', was used as an inoculum reservoir for PSbMV-P1 in which it produces clear, moderate symptoms.

B445-66, an OSU breeding line developed for resistance to PSbMV-P1 (Baggett and Hampton, 1977) is an F3 selection from crosses between a PSbMV-P1 resistant P.I. line and OSU lines of commercial freezing type. B445-66 is the only parental line resistant to all three viruses used in this study.

'Quincy', a large leaved cultivar, developed by W. Brotherton Seed Company, has reduced stipules presumably

Table 1. Reactions of parental lines to virus strains in preliminary tests

Lines	PSbMV-L	BYMV	PSbMV-P1
Sounder	S[1]	S	S
Abador	S	S	S
Little Marvel	R	R	S
B445-66	R	R	R
Quincy	S	R[2]	S
Avon	S	R[2]	S

1. S indicates susceptibility as shown by development of symptoms; R indicates resistance or lack of symptom development.

2. Results obtained differ from results obtained by Dr. R. Provvidenti in Geneva, NY.

conferred by the St gene. 'Quincy' was included in the study because of its apparent resistance to BYMV and susceptibility to PSbMV-L, an uncommon combination. This cultivar is difficult to infect with PSbMV-L, but when infected, readily exhibits typical symptoms. This phenomenum, presumably caused by modifying genes, is referred to as klendusity.

'Avon', also developed by W. Brotherton Seed Company, is an early freezing type pea. It is similar to 'Quincy' in its susceptibility to PSbMV-L and apparent resistance to BYMV, but is easier to infect with PSbMV-L. 'Avon' was used as a susceptible parent in the inheritance study.

Hybridization

PSbMV-L susceptible parental lines, 'Sounder', 'Abador', 'Quincy', and 'Avon' were hybridized with resistant 'Little Marvel' and B445-66 (Table 2). All pea hybridizations were performed in the greenhouse. In order to produce vigorous plants and to better ensure successful hybrid seed production, each pot was limited to a maximum of 4 plants.

Flower buds on the female parent, with receptive stigmas but undehisced anthers, were selected and emasculated with forceps. Pollen from an open flower of the male parent was then brushed onto the stigma of the female flower. Forceps were dipped in alcohol between pollinations to kill contaminating pollen.

Reciprocal combinations were produced for all crosses, and complete backcrosses were made for two crosses and their reciprocals (Tables 2 and 3).

Seed Production

F2 and F3 generation seeds were produced both in the greenhouse and in the field. Production in the greenhouse allowed generation advancement in the

Table 2. Crosses and reciprocals produced for inheritance study from hybridizations between PSbMV-L susceptible and resistant cultivars

Cross Designation	Parentage Female		Male
K1	Sounder	x	B445-66
K1R	B445-66	x	Sounder
K2	Abador	x	B445-66
K2R	B445-66	x	Abador
K3	Quincy	x	B445-66
K3R	B445-66	x	Quincy
K4	Sounder	x	Little Marvel
K4R	Little Marvel	x	Sounder
K5	Quincy	x	Little Marvel
K5R	Little Marvel	x	Quincy
K6	Avon	x	B445-66
K6R	B445-66	x	Avon
K7	Avon	x	Little Marvel
K7R	Little Marvel	x	Avon

Table 3. Backcrosses produced for inheritance study

Cross Designation	Backcross Combination[1]	Parentage[2]
K1	BC R	(Sounder x B445-66) x B445-66
		(B445-66 x Sounder) x B445-66
		B445-66 x (Sounder x B445-66)
		B445-66 x (B445-66 x Sounder)
	BC S	(Sounder x B445-66) x Sounder
		(B445-66 x Sounder) x Sounder
		Sounder x (Sounder x B445-66)
		Sounder x (B445-66 x Sounder)
K4	BC R	(Sounder x L. Marvel) x Little Marvel
		(L. Marvel x Sounder) x Little Marvel
		Little Marvel x (Sounder x L. Marvel)
		Little Marvel x (L. Marvel x Sounder)
	BC S	(Sounder x Little Marvel) x Sounder
		(Little Marvel x Sounder) x Sounder
		Sounder x (Sounder x Little Marvel)
		Sounder x (Little Marvel x Sounder)

1. BC R= Backcross to resistant parent; BC S= Backcross to susceptible parent.

2. Parents in parenthesis are the F1 generations of the cross involved.

off-season but limited the amount of seed produced. Field seed production yielded a large amount of seed but was limited to a single generation per year.

F2 seeds from F1 plants of the same cross were bulked at harvest. In contrast, seeds from single F2 plants were harvested as single F3 families. Each family consisted of all the F3 seeds from an F2 plant and was used as a progeny test of the F2 parent plant.

Environmental Conditions and Plant Culture

While seed production was carried out either in a greenhouse or in the field, virus testing was limited to the greenhouse in order to avoid contaminating OSU breeding materials with PSbMV-L and PSbMV-P1. It was also necessary to avoid interference of tests by natural infection by pea enation mosaic and other viruses which occur in the Corvallis area.

For resistance tests, 6 to 9 seeds were planted in No. 10 cans or plastic pots holding approximately 2.5 liters of soil. For hybridization and seed production, each pot contained no more than 4 plants for maximum seed yield. A pasteurized soil medium containing 1 part sandy- loam soil: 1 part sand: 1 part peat: 2 parts pumice with added plant nutients was used for all greenhouse plantings.

General testing and seed production took place in greenhouse rooms maintained at 21 degrees C. days and 15 degrees C. night temperatures. In the room used for the inheritance tests, higher temperatures of 25 degrees C. days and 19 degrees C. nights were provided to optimize symptom development.

During the winter, due to low natural light intensity and short days, supplemental low spectrum (cool-white) fluorescent lighting (approximately 50 microeinstein $m^{-2} \times s^{-1}$), 16 hour photoperiod, was provided for the inheritance tests.

Insect and disease problems in the greenhouse were controlled with appropriate pesticides.

Inoculation Method

PSbMV-L inoculum was maintained in 'Sounder' because symptom development and expression was rapid and intense. 'Sounder' also has the advantage of having a high level of resistance to powdery mildew which at times reaches high levels in peas growing in the greenhouse. BYMV was usually maintained in 'Red Kidney' beans, which are not host to PSbMV-P1 or PSbMV-L, to ensure that the BYMV inoculum would not become contaminated. 'Red Kidney' beans rapidly produce symptoms of BYMV (7 to 10 days after mechanical inoculation) and are a dependable indicator of the presence of viable virus particles. When a large amount of inoculum was needed, infected 'Sounder' plants were utilized as an additional source, PSbMV was maintained in either 'Sounder' or 'Little Marvel' plants, both of which show typical symptoms. In addition to these fresh inoculum sources, freeze-dried inoculum was available for renewing the virus source when necessary.

For the inheritance study, an individual test was planted with approximately 100 F2 individuals or 100 F3 families, and included parental lines and control cultivars.

Inoculum was prepared from the youngest leaves showing virus symptoms by macerating them in a mortar and pestle in 0.02 Molar potassium phosphate buffer, pH 7. This macerate was rubbed on pea leaflets which had been dusted with 400 mesh carborundum (silicon carbide). Each pot contained a single uninoculated control plant. The first inoculation was performed when leaflets at the first two to three nodes were fully expanded, approximately 10 to 14 days after planting. The second inoculation was usually performed 5 to 7 days later on

newly expanded leaflets. After rubbing the leaves with inoculum, plants were rinsed with water to avoid injury caused by the buffer.

Screening of Pea Cultivars, P.I. Lines, and Breeding Lines For Virus Resistance

Seeds for approximately 200 pea cultivars were obtained from seed companies. In addition, 32 Plant Introduction lines known to include a high proportion of PSbMV-P1 resistant lines and 9 PSbMV-resistant OSU breeding lines were obtained. All of these cultivars and lines were tested for their reacton to PSbMV-L, BYMV and PSbMV-P1.

Between 6 and 10 seeds of each cultivar were planted in a single pot. Leaving one plant per pot as an uninoculated check, all other plants were mechanically inoculated twice. All cultivars that remained symptomless in the initial test were replanted and retested in the same manner. During the second test, cultivars which were not obviously infected but included abnormal or suspicious looking plants, were assayed for the presence of virus by inoculation onto a susceptible host.

The Plant Introduction lines were planted and tested twice. In the first test, symptomless lines were observed for 60 days before being discarded. In the second test, every line was assayed by susceptible host inoculation. Plants inoculated with PSbMV-P1 and PSbMV-L were assayed by bulking an approximately equal amount of tissue from each inoculated plant within a line and mechanically inoculating one pot containing an average of 6 'Sounder' plants. The BYMV tested plants were assayed in the same way on 'Red Kidney' beans although each pot contained just three plants.

The OSU breeding lines were tested twice. Any suspicious or abnormal looking inoculated plants were individually assayed on 'Sounder'.

Chronology of the Study

Spring	1984	Initial crosses made.
Summer	1984	F2 seed production in field.
Fall	1984	Crosses and backcrosses made. F3 families produced from field-grown F2 seed. Additional F2 seed produced in greenhouse.
Winter	1985	Cultivar testing started.
Spring	1985	Inheritance tests.
Summer	1985	Additional F3 families produced in field.
Fall	1985	Inheritance tests.
Spring	1986	Inheritance tests.
Fall	1986	Inheritance tests. Cultivar and P.I. line testing.
Winter	1987	Inheritance tests. Cultivar and P.I. line testing.

RESULTS AND DISCUSSION

Response of Parent Cultivars to Virus Inoculations

PSbMV-L: Systemic symptoms of PSbMV-L in 'Sounder' included an initial pronounced height reduction, leaf distortion, vein clearing, and a brighter over-all yellow- green color. Pods were small and contained none to few tiny seeds, many with split seed coats. Symptoms were induced by PSbMV-L infection in 89% of 'Sounder' plants two weeks after inoculation (Table 4).

'Abador' reacted severely when inoculated with PSbMV- L. Plants infected with PSbMV-L displayed an extreme height reduction so that they were approximately one-third of the height of the noninoculated check plants. Leaf size was greatly reduced and leaves were tightly curled. Approximately ten days after mechanical inoculation, 'Abador' began showing necrosis in the newest tissue, along with the appearance of wilting. This was followed by the proliferation of basal branches. Eventually many of the 'Abador' plants died, apparently from PSbMV-L infection. Eighty-seven per cent (Table 4), of 'Abador' plants became infected when inoculated with PSbMV-L.

'Quincy' was difficult to infect with PSbMV-L and had a large number of escapes. When inoculation was successful, this cultivar showed strong, clear symptoms consisting of leaf size reduction and distortion. Fifty-nine percent of 'Quincy' plants were infected when inoculated with PSbMV-L in the inheritance study (Table 4).

Symptoms of PSbMV-L in 'Avon' were typical and of similar intensity to symptoms in 'Sounder'. 'Avon' was easily infected with PSbMV-L showing a 93% infection rate for all plants tested in the inheritance study (Table 4).

Both 'Little Marvel' and OSU B445-66 were resistant to PSbMV-L.

Table 4. Parental lines inoculated with PSbMV-L in inheritance tests

Cultivar	No. of Plants Infected	Not Infected	Percent Infected
Sounder	142	18	89
Abador	79	12	87
Quincy	26	18	59
Avon	95	7	93
Little Marvel	0	62	0
B445-66	0	251	0

BYMV: When infected with BYMV, 'Sounder' displayed a slight height reduction, but leaf and pod size did not appear to be greatly affected. A bright yellow mosaic developed on the leaves approximately 2 to 3 weeks after inoculation.

With BYMV, symptoms were apparent in 'Abador' earlier than in any of the other susceptible parental lines. Along with leaf-size reduction, symptoms included a bright yellow mosaic covering more leaf surface than that produced by BYMV in 'Sounder'. Many BYMV-infected 'Abador' plants died prematurely, approximately 4 to 6 weeks after inoculation.

'Quincy', under greenhouse conditions at OSU, was resistant to BYMV although Dr. R. Provvidenti, under his greenhouse conditions at Geneva, NY, was able to infect 'Quincy' with this virus. Attempts were made to infect 'Quincy' under high greenhouse temperatures and supplemental lighting but no infections resulted. Likewise, attempts to cause infection by inoculating with several isolates of BYMV failed to induce infection (Table 5).

'Avon' was similar to 'Quincy' in its apparent, but disputed, resistance to BYMV.

'Little Marvel' and OSU B445-66 were both resistant to the BYMV-Scott isolate used in this study. B445-66 did, however, produce symptoms consisting of a white to pale- green mosaic when inoculated with three BYMV isolates from bellbeans (Table 5).

PSbMV-P1: In 'Sounder', symptoms of PSbMV-P1 infection included an overall plant stunting, a faint leaf mottle, and leafrolling.

In 'Abador', PSbMV-P1 symptoms consisted of general plant stunting and leafroll. Symptoms in 'Abador' were not as severe with PSbMV-P1 as with the other two viruses, and most plants survived the infection.

Table 5. Susceptibility of selected pea parental lines to five isolates of BYMV

Cultivar	BYMV-Scott	Bell Bean Isolates[1]			
		#1	#4	#10	#15
Quincy	0/7 [2]	0/5	0/6	0/7	0/4
Avon	0/3	0/6	0/4	0/4	0/4
Sounder	3/3	3/3	5/5	4/4	5/5
B445-66	0/7	5/5 [3]	3/6	4/5	0/8
Red Kidney [4]	3/3	3/3	3/3	0/4	4/4

1. BYMV isolates from bell beans provided by Dr. R.O. Hampton.

2. Numerator refers to number of individuals showing symptoms. Denominator refers to total number of plants of that cultivar tested.

3. Three bell bean isolates produced atypical symptoms in BYMV-Scott resistant line, B445-66.

4. Red Kidney bean included in test to differentiate the type strain from the pea strains which do not usually infect beans.

'Quincy' was readily infected when inoculated with PSbMV-P1. Symptoms of PSbMV-P1 in 'Quincy' included plant stunting and a more pronounced mottle than that produced in the other parental lines.

Symptoms of PSbMV-P1 in 'Avon' were typical and similar to the intensity produced by PSbMV-P1 infection in 'Sounder'.

'Little Marvel' was susceptible to PSbMV-P1. Symptoms of PSbMV-P1 in 'Little Marvel' were moderate and included an overall plant size reduction and leaves displaying a light mottle and leafroll.

OSU B445-66 was resistant to PSbMV-P1.

Response of Progeny Populations to PSbMV-L Inoculation

The F1 generation, except for K5 and K7 with 'Quincy' and 'Avon' as susceptible parents and 'Little Marvel' as resistant parent, generally reacted with systemic symptoms to PSbMV-L (Table 6). Occasional escapes were observed which were attributed to chance. Symptom intensity in the F1 was comparable to the intensity in the susceptible parent indicating that susceptibility was completely dominant. In the segregating generations, susceptibility and resistance were clearcut. When the F2 generation data were tested against a 3 susceptible: 1 resistant (3 S: 1 R) ratio by a Chi Square goodness of fit test, most gave Chi Square values indicating they did not deviate from expected monogenic ratios. The F3 family tests were essentially progeny tests of the F2 generation. Because there was a problem of known susceptible plants (such as F1's or susceptible parents) escaping infection when inoculated, the F3 tests were analyzed against both a ratio of 1 susceptible family: 2 segregating families: 1 resistant family (1 S: 2 SG: 1 R) and a ratio of 3 S + SG: 1 R. The tendency of susceptible plants to escape infection could potentially generate incorrect ratios by decreasing the number of susceptible families and increasing the number in

Table 6. Susceptibility of F1 pea progenies to PSbMV-L

Cross	No. of Plants		Percent
	Infected	Not Infected	Infected
K1 Sounder x B445-66	87	7	93
K2 Abador x B445-66	54	1	98
K3 Quincy x B445-66	64	16	80
K4 Sounder x L. Marvel	21	4	84
K5 Quincy x L. Marvel	0	27	0
K6 Avon x B445-66	64	3	96
K7 Avon x L. Marvel	29	46	39

the segregating category. Unless there was a problem of klendusity, the resistant category was less likely to contain incorrectly identified segregating families since all susceptible plants within the family would have to escape infection. The susceptible and segregating categories were therefore combined and the F2 phenotypic ratio tested. Table 7 shows the F3 populations analyzed against both ratios. Homogeneity tests were performed on the reciprocal data from each cross, and reciprocal F2 or F3 data were combined when appropriate (Tables 8, 9 and 10).

Segregation for resistance to PSbMV-L infection among progenies from seven crosses are discussed in the following paragraphs.

K1 (Sounder x B445-66): Data were collected from the F1, F2, F3 families, and from subsequent backcross progeny (Table 11). 'Sounder', the susceptible parent, produced symptoms in all but a few individuals that escaped infection. The F1 generation also contained a small number of plants which failed to become infected. The F2 generation did not significantly deviate from the expected 3 S: 1 R ratio, and F3 family data also fit a 1 S: 2 SG: 1 R ratio. Likewise, when all-susceptible and segregating (including escaped plants) families were combined, the data also fit a 3 S + SG: 1 R ratio. Data from backcrosses similarly fit the ratio expected if resistance is conditioned by a single recessive gene. The backcross to the resistant parent fit a 1 susceptible: 1 resistant ratio (1 S: 1 R), while the backcross to the susceptible parent were all susceptible.

K2 (Abador X B445-66): Twelve out of 91 (13%) plants of susceptible parent, 'Abador', and one F1 plant out of 55 escaped infection. The F2 and F3 generations both fit a 3 S: 1 R ratio (and 3 S + SG: 1 R), but when the F3 family data were tested against a 1 S: 2 SG: 1 R ratio, one of the reciprocals significantly deviated (Table 11). The data

Table 7. F3 families analyzed with two and three classes for reaction to PSbMV-L, reciprocal crosses not combined

	No. of Families [1]											
	Two Classes				Three Classes						Chi Square [2]	
	Observed		Expected		Observed			Expected				
Cross	S+SG	R	S+SG	R	S	SG	R	S	SG	R	3:1	1:2:1
K1	131	36	125.25	41.75	44	87	36	41.75	83.5	41.75	0.880 ns	1.060 ns
K1R	129	52	135.75	45.25	40	89	52	45.25	90.5	45.25	1.151 ns	1.641 ns
K2	143	51	145.5	48.5	25	118	51	48.5	97	48.5	0.110 ns	16.062 **
K2R	140	50	142.5	47.5	40	100	50	47.5	95	47.5	0.112 ns	1.579 ns
K4	72	27	74.25	24.75	11	61	27	24.75	49.5	24.75	0.165 ns	10.515 **
K4R	71	25	72	24	20	51	25	24	48	24	0.014 ns	0.896 ns
K5	36	17	39.75	13.25	4	32	17	13.25	26.5	13.25	1.063 ns	8.660 **
K5R	36	16	39	13	7	29	16	13	26	13	0.641 ns	3.808 ns
K6	66	32	73.5	24.5	13	53	32	24.5	49	24.5	2.667 ns	8.020 **
K6R	54	43	72.75	24.25	6	48	43	24.25	48.5	24.25	18.313 **	28.237 **
K7	59	24	62.25	20.75	7	52	24	20.75	41.5	20.75	0.486 ns	12.277 **
K7R	68	20	66	22	29	39	20	22	44	22	0.136 ns	2.977 ns

1. Family reaction to PSbMV-L infection: S= All individuals within family are susceptible; SG= Segregating S and R within family; R= Resistant family.

2. Chi square values calculated using Yates Correction Factor.

Table 8. Homogeneity of cross and reciprocal populations of the F2 generation

Cross	Chi Sq. 3 S: 1 R	Pooled Chi Sq.	Homogeneity Chi Sq.[1]	Reciprocals Combined
K1	1.156	0.575	0.625 ns	yes
K1R	0.044			
K2	0.788	0.207	0.680 ns	yes
K2R	0.099			
K3	165.271	369.127	0.300 ns	yes
K3R	204.156			
K4	3.030	3.373	0.844 ns	yes
K4R	1.187			
K5	116.564	210.721	8.426 **	no
K5R	102.583			
K6	4.734	4.738	0.787 ns	yes?[2]
K6R	0.791			
K7	3.160	5.660	0.013 ns	yes?[3]
K7R	2.513			

1. Homogeneity Chi Sq. = Total Chi Sq. - Pooled Chi Sq.

2. Combined data do not fit the expected 3 S: 1 R ratio. When tested individually, K6R fit the ratio while K6 did not.

3. Combined data do not fit the expected 3 S: 1 R ratio while the two populations, when tested individually, each fit.

Table 9. Homogeneity of cross and reciprocal populations of the F3 generation

Cross	Chi Square				Repicrocals Combined
	3S:1R[1]	Total	Pooled	Homogeneity[2]	
K1	1.145	2.594	0.015	2.579 ns	yes
K1R	1.449				
K2	0.110	0.209	0.347	0.126 ns	yes
K2R	0.099				
K4	0.214	0.270	0.246	0.024 ns	yes
K4R	0.056				
K5	1.631	2.554	2.505	0.049 ns	yes
K5R	0.923				
K6	3.532	23.519	18.426	5.093 *	no
K6R	19.987				
K7	0.574	0.817	0.031	0.786 ns	yes
K7R	0.243				

1. F3 populations analyzed against a ratio of 3 susceptible and segregating families : 1 resistant family.

2. Homogeneity Chi Sq. = Total Chi Sq. - Pooled Chi Sq.

Table 10. F3 families analyzed with two and three classes for reaction to PSbMV-L, homogeneous reciprocals combined

	No. of Families[1]										Chi Square[2]	
	Two Classes				Three Classes							
	Observed		Expected		Observed			Expected				
Cross	S+SG	R	S+SG	R	S	SG	R	S	SG	R	3:1	1:2:1
K1	260	88	261	87	84	176	88	87	174	87	0.004 ns	0.138 ns
K2	283	101	288	96	65	218	101	96	192	96	0.281 ns	13.791 **
K4	143	52	146.25	48.75	31	112	52	48.75	97.5	48.75	0.207 ns	8.836 *
K5	72	33	78.75	26.25	11	61	33	26.25	52.5	26.25	1.984 ns	11.971 **
K7	127	44	128.25	42.75	36	91	44	42.75	85.5	42.75	0.018 ns	1.456 ns

1. Family reaction to PSbMV-L: S= All individuals within family are susceptible; SG= Segregating S and R within family; R= Resistant family.

2. Chi square values for 3 S+SG: 1 R calculated using Yates Correction Factor.

Table 11. Genetic segregation for resistance to PSbMV-L in crosses K1 through K4 reciprocal crosses combined

Cross	Generation[1]	No. of Plants or Families[2] Observed S	Observed R	Expected S	Expected R	Ratio Tested	Chi Square
K1 Sounder x B445-66	PS	97	7				
	PR	0	104				
	F1	87	7				
	F2	169	63	174	58	3S:1R	0.575 ns
	F3	260	88	261	87	3S:1R	0.015 ns
	BC R	69	60	64.5	64.5	1S:1R	0.628 ns
	BC S	41	0				
K2 Abador x B445-66	PS	79	12				
	PR	0	92				
	F1	54	1				
	F2	176	55	173.25	57.75	3S:1R	0.175 ns
	F3	284	101	288.75	96.25	3S:1R	0.281 ns
K3 Quincy x B445-66	PS	10	14				
	PR	0	27				
	F1	64	16				
	F2	142	272	310.5	104.5	3S:1R	366.946 **
K4 Sounder x L. Marvel	PS	24	0				
	PR	0	23				
	F1	21	4				
	F2	158	69	170.25	56.75	3S:1R	3.098 ns
	F3	143	52	146.25	49.75	3S:1R	0.171 ns
	BC R	40	45	42.5	42.5	1S:1R	0.188 ns
	BC S	49	4				

1. PS= Susceptible parent; PR= Resistant parent; BC R= Backcross to resistant parent; BC S= Backcross to susceptible parent.

2. Reaction to PSbMV-L: S= Number of individuals classified as susceptible; R= Number of individuals classified as resistant. F3 populations analyzed against ratio of 3 susceptible and segregating families : 1 resistant family.

indicates that there is a smaller number of all-susceptible families and a larger number of segregating families than expected. This suggests escapes in the susceptible group resulted in some of these families being classified as segregating. Backcrosses were not produced for this cross.

K3 (Quincy x B445-66): 'Quincy' was the parental cultivar that was difficult to infect with PSbMV-L. As the data indicates, both the parent and progenies displayed this tendency to escape infection (Table 11). While the F2 does not fit the expected monogenic ratio, the cross and reciprocal were homogeneous as indicated by the homogeneity test (Table 8). Although a selfed individual was detected in one F1 test, as indicated by a single plant with reduced stipules, the general difficulty in causing infection was present in all tests with 'Quincy'and was probably not due to hybridizations failures. F3 family data were not available for this cross.

K4 (Sounder x Little Marvel): Tests were performed on the F1, F2, F3, and complete backcrosses (Table 11). All data fit the ratios expected if a single recessive gene determined resistance except when the F3 family data were tested against a 1 S: 2 SG: 1 R ratio (Table 7). The high number of segregating families and low number of susceptible families suggest that a number of individuals escaped infection.

K5 (Quincy x Little Marvel): As in K3, an apparent tendency toward klendusity greatly affected the ratios obtained for the susceptible parent and progenies. All F1 individuals escaped infection, and neither of the reciprocal F2 populations fit a 3 S: 1 R ratio (Table 12). Modifying genes from both 'Quincy' and 'Little Marvel' may have contributed to the apparent tendency for escapes which resulted in the aberrant ratios observed. The F2 populations of K5 and K5R were not combined due to

Table 11. Genetic segregation for resistance to PSbMV-L in crosses K1 through K4 reciprocal crosses combined

Cross	Generation[1]	No. of Plants or Families[2] Observed S	Observed R	Expected S	Expected R	Ratio Tested	Chi Square
K1 Sounder x B445-66	PS	97	7				
	PR	0	104				
	F1	87	7				
	F2	169	63	174	58	3S:1R	0.575 ns
	F3	260	88	261	87	3S:1R	0.015 ns
	BC R	69	60	64.5	64.5	1S:1R	0.628 ns
	BC S	41	0				
K2 Abador x B445-66	PS	79	12				
	PR	0	92				
	F1	54	1				
	F2	176	55	173.25	57.75	3S:1R	0.175 ns
	F3	284	101	288.75	96.25	3S:1R	0.281 ns
K3 Quincy x B445-66	PS	10	14				
	PR	0	27				
	F1	64	16				
	F2	142	272	310.5	104.5	3S:1R	366.946 **
K4 Sounder x L. Marvel	PS	24	0				
	PR	0	23				
	F1	21	4				
	F2	158	69	170.25	56.75	3S:1R	3.098 ns
	F3	143	52	146.25	49.75	3S:1R	0.171 ns
	BC R	40	45	42.5	42.5	1S:1R	0.188 ns
	BC S	49	4				

1. PS= Susceptible parent; PR= Resistant parent; BC R= Backcross to resistant parent; BC S= Backcross to susceptible parent.

2. Reaction to PSbMV-L: S= Number of individuals classified as susceptible; R= Number of individuals classified as resistant. F3 populations analyzed against ratio of 3 susceptible and segregating families : 1 resistant family.

a lack of homogeneity although the F3 populations were combined (Tables 8 and 9). The combined F3 fit the expected 3 S + SG: 1 R ratio. In addition, K5R F3 fit the 1 S: 2 SG: 1 R ratio (Table 7). The K5 F3 family test did not fit this 1 S: 2 SG: 1 R ratio but the general health of these plants were affected by a mold infestation that eliminated many families and may have affected the ratio.

K6 (Avon x B445-66): The F1, F2 and F3 generations were tested for this cross (Table 12). All plants of the susceptible parent, 'Avon', were infected, although escapes apparently occurred in progeny populations. When K6 and K6R were combined, the F2 generation deviated from the expected 3 S: 1 R ratio. When the two populations were individually tested, K6R fit but K6 did not fit the 3 S: 1 R ratio. (Table 8) K6 and K6R of the F3 generation were not combined due to large differences between the separate ratios obtained. Although the F3 generation of K6 fit the tested 3 S + SG: 1 R ratio, the reciprocal, K6R, did not (Tables 8 and 12).

K7 (Avon x Little Marvel): The susceptible parent, 'Avon', had 7 out of 43 escapes in this test, and the majority of F1 individuals escaped infection (Table 12). As with K5, modifying factors from 'Little Marvel' and 'Avon' may have interacted to produce this tendency to escape infection. The reciprocal F2 populations were not combined because the combined data did not fit a 3 S:1 R expected ratio although, individually, the populations fit (Table 8 and 12). The combined K7 F3 populations fit a 3 S + SG: 1 R ratio. The K7R population fit a 1 S: 2 SG: 1 R ratio, while K7 deviated due to excess families in the segregating and resistant category (Tables 9, 10, and 12).

When the results of all the crosses are considered, it is apparent that the data obtained for the F3 populations should be tested against a 3 S + SG: 1 R ratio rather than a 1 S: 2 SG: 1 R ratio, due to klendusic reactions and

escapes. Thus, when these data are tested against a 3 S + SG: 1 R ratio, the F3 data combines with the F2 data in support of the conclusion that resistance is controlled by a single recessive gene. Modifying genes are implicated as the most likely and important causal factor involved in the aberrant ratios obtained from crosses with 'Quincy'.

Reaction of Pea Cultivars, P.I. Lines and Breeding Lines to Inoculations with PSbMV-L, BYMV, and PSbMV-P1

A total of 202 commercial cultivars and parental lines were tested for their reactions to the three viruses used in the study (Table 13). Of this total, 144 were resistant and 58 were susceptible to PSbMV-L (Table 14). When tested with BYMV, 144 were resistant, 55 susceptible and 3 cultivars were not available for testing. Of the 202 cultivars tested only two, 'Quincy' and 'Avon', had differential reactions to PSbMV-L and BYMV. Both of these cultivars were susceptible to PSbMV-L and resistant to BYMV. Of the total 202 cultivars, 3 were resistant to PSbMV-P1, 175 were susceptible, and 24 were not available for testing. The three PSbMV-P1 resistant cultivars included B445-66, the OSU line used as a resistant parent in the genetic study and two commercial cultivars bred specifically for resistance to this virus. Each of these three cultivars was also resistant to PSbMV-L.

Of the 32 Plant Introduction lines inoculated, none was infected by PSbMV-L, one, an apparent crossover type, was infected by BYMV, and 5 were infected by PSbMV-P1, three of which had previously been reported to contain both resistant and susceptible genotypes (Table 15). Although the proportion of PSbMV-P1 resistant lines in the Pisum P.I. collection is low, the test included a high number of resistant lines, since they were previously known to contain resistance to PSbMV-P1. Some of the results differed from published and unpublished results obtained by a number of plant scientists. Table 14 indicates agreement

or disagreement and identifies the author cited.

Since each line was bulked when assayed on a susceptible host it was not possible to distinguish between a heterogeneous and homogeneous line for the virus reaction. Therefore where these results indicate susceptibility of the line, someone else may have found it to be heterogeneous. Likewise, due to small sample size, a heterogeneous line could remain uninfected due to a chance exclusion of seed of susceptible individuals.

As can be seen in Table 15, there are numerous sources of resistance to each of the three viruses in the Pisum Plant Introduction collection and most of these lines are resistant to all three viruses included in this study.

Nine OSU lines developed in a PSbMV resistance program were tested for their reaction to PSbMV-P1, PSbMV-L and BYMV (Table 16). All proved resistant to the three viruses. In addition to their resistance to infection by the three viruses, they are also all resistant to PSbMV-L1 and Pea Enation Mosaic Virus, and have differing levels of tolerance to Red Clover Vein Mosaic Virus and Bean Leafroll Virus (Baggett and Kean, 1988).

The data from the commercial cultivars, P.I. lines, and OSU lines indicate that genotypes resistant to PSbMV-P1 tend to be resistant to PSbMV-L. This relationship can best be explained by information provided by Dr. R. Provvidenti (personal communication) that a gene, sbm-3 is linked with sbm-1 on Chromosome 6 and confers resistance to all pathotypes of this virus.

Table 13. Reactions of commercial cultivars to inoculation with PSbMV-L, BYMV, and PSBMV-P1

Cultivar	Source	Virus Reaction[1] PSbMV-L	BYMV	PSbMV-P1
Summit	Crites-Moscow	R	R	S
Sun Valley		S	S	S
Image		R	R	S
Almota		R	R	S
Valley Perf.		R	R	S
Perf. 3019		R	R	NA
83 MI-3		R	R	R [2]
Dark Green Perf.		R	R	S
Scout		S	S	S
Superscout		R	R	R [2]
Hustler		R	R	S
Lance		R	R	S
Fr 259		R	R	S
Spirit		R	R	S
Early Frosty		R	R	S
Tiny		S	S	S
Rebel		R	R	S
Powder Proof		R	R	S
Venus		R	R	S
Granada		R	R	S
Trojan		R	R	S
Challis		S	S	S
Sm. Sieve Alaska		S	S	S
A-45		R	R	S
Regal 36		S	S	S
Swinger		R	R	S
Charo		S	S	S
4583		S	S	S
Kosta		R	R	S
508-4-2-4C	Canners Seed	R	R	S
2213 ES		S	S	S
7705-11F		R	R	S
8615 EP		S	S	S
7601-2-1-4F		R	R	S
X 9727-10F		S	S	S
X 9726-2F		S	S	S
X 9725-8F		R	R	S
Frontier C		R	R	S

1. S= Susceptible; R= Resistant; NA= Not available.

2. Confirmed by breeder of cultivar.

Table 13 continued.

Cultivar	Source	Virus Reaction		
		PSbMV-L	BYMV	PSbMV-P1
7705-32F	Canners Seed	R	R	S
DS Paf F		R	R	S
7708-2-3F		R	R	S
9889-2F		S	S	S
508-7C		R	R	S
9901 C		R	R	S
7025 ES		S	S	S
X 9602-2F		S	S	S
512-2F		S	S	S
7712-10C		R	R	S
7705-18F		R	R	S
8221 EP		R	R	S
8617 EP		S	S	S
9888 F		S	S	S
5147 DSP		R	R	S
9889 F		S	S	S
517-2-4		S	S	S
6060 F		R	R	S
9220 F		R	R	S
8615 EP		S	NA	S
F80139	Rogers Bros.	S	S	NA
Tempter		S	S	S
Aldot		S	S	S
Novella		R	R	S
Aurora		R	R	S
Salvo		R	R	S
F79123		S	S	S
F80152		S	S	S
Galaxie		R	R	S
Duke		R	R	S
Canjoy		R	R	S
Target		R	R	S
Novella II		R	R	S
Parlay		R	R	S
Perf 400		R	R	S
Sparkle		R	R	S
Medalist		R	R	S
F74115		R	R	S
Early Frosty		R	R	NA
Honey Pod		R	R	S

Table 13 continued.

Cultivar	Source	Virus Reaction		
		PSbMV-L	BYMV	PSbMV-P1
AVX 333-26	Sun Seed	R	R	S
AVX 339		R	R	S
AVX 382-99		S	S	NA
AVX 8-358-40		R	R	NA
Early Perfection		R	R	S
AVX 309		R	R	NA
AVX 345-23		R	R	NA
AVX 323		R	R	NA
AVX 329		R	R	NA
AVX 60-521-26		R	R	NA
AVX 8507		R	R	NA
AVX 321		R	R	NA
Anoka		R	R	S
Code 1		R	R	S
Pacemaker		S	S	S
Tonka		R	R	S
Titania		S	S	S
Duet		S	S	S
Fraser	W. Brotherton	R	R	S
Conway		R	R	S
Fr 736		R	R	S
Grant		R	R	S
L 282 Freezer		R	R	S
Small Sieve DSP		R	R	S
Popet		S	S	S
Orcas		R	R	S
82-746	Musser Seed	R	R	S
83-809		S	S	S
82-747		R	R	S
82-738		R	R	S
83-807		R	R	S
83-804		R	R	S
83-808		R	R	S
83-805		R	R	S
83-806		R	NA	NA

Table 13 continued.

Cultivar	Source	Virus Reaction		
		PSbMV-L	BYMV	PSbMV-P1
Ceras	Asgrow Seed	S	S	S
Dual		S	S	S
Frisky		S	S	S
Rally		R	R	S
Pomak		R	R	S
Mars		R	R	S
Champ		R	R	S
Dinos		R	R	S
Olympia	Agway	R	R	S
Mayfair		R	R	S
Early Snap		R	R	S
Candlelite	Gallatin Valley	R	R	S
Trident		R	R	S
Early Sw 9		R	R	S
H 680-1-3		R	R	S
Sugar Bon		R	R	S
Tripod		R	R	S
H 543-3-1-11		R	R	S
H 783-29		R	R	S
H 783-28-3		R	R	S
H 890-3-2		R	R	S
HP 6-3		R	R	S
Badger		R	R	S
Early Sw 7		R	R	S
Sugar Rae		R	R	S
Canner 2333	Pure Line Seed	R	R	S
Canner 2367		R	R	S
Fr 2484		R	R	S
Dk Skin Perf		R	R	S
Fr 2434		R	R	S
Fr 2400		R	R	S
Kodiak		R	R	S
Fr 2315		R	R	S
K-1		R	R	S
Canner 2429		R	R	S
3019		R	R	S
3040		R	R	S
Canner 81		R	R	S

Table 13 continued.

Cultivar	Source	Virus Reaction		
		PSbMV-L	BYMV	PSbMV-P1
Canner 41	Pure Line	S	S	S
Sundance		R	R	S
Arctic		R	R	S
Columbia		R	R	S
Fr 4020		R	R	S
15 Strain		R	R	S
Tahoe		R	R	S
Fr 58		R	R	NA
Fr 4024		R	R	S
Early Sw 5		S	S	S
Fr 108		S	S	S
6F		S	S	S
Fr 88		R	R	S
Banff		R	R	NA
Selway		R	R	S
11 Strain		R	R	NA
Venus		R	NA	NA
Sprite		S	S	S
7C		R	R	S
Fr 813		R	R	S
Snowflake	Johnny's	R	R	S
Knight	Harris	R	R	S
PF 60		S	S	NA
Hydra	Miscellaneous	R	R	S
Sugar Mel		S	S	S
Nofila		S	S	S
PI 261 667		S	S	S
PI 244 151		S	S	S
Green Sugar		R	R	NA
Imp. Laxton Progress		R	R	S
Frisky		S	S	NA
Sugar Ann		S	S	NA
Tall Telephone		S	S	S
Snow Pea		S	S	NA
Green Arrow		S	S	S
Wando		R	R	S
MN 108		R	R	S
Purple Podded		S	S	S

Table 13 continued.

Cultivar	Source	Virus Reaction		
		PSbMV-L	BYMV	PSbMV-P1
Aspen	Miscellaneous	R	R	S
Champ		R	R	NA
Early Sw 9		R	R	NA
Dw Grey Sugar		S	S	S
IBA Kwartella		S	S	S
Sounder	Parental lines	S	S	S
Abador		S	S	S
Little Marvel		R	R	S
B445-66		R	R	R
Quincy		S	R*	S
Avon		S	R*	S
Peru 2	Avon's Parent	S	S	S
Asgrow 3's		R	R*	S

* These results differ from those of R. Provvidenti who rated these three cultivars as susceptible to BYMV.

Table 14. Total numbers of cultivars resistant and susceptible to PSbMV-L, BYMV, and PSbMV-P1

Virus	No. of Cultivars			
	Resistant	Susceptible	Not Available	Total
PSbMV-L	144	58		202
BYMV	144	55	3	202
PSbMV-P1	3	175	24	202

Table 15. Reactions of Pisum Plant Introduction accessions to inoculation with PSbMV-L, BYMV, and PSbMV-P1

P.I. Line	Disease Reaction[1]			Literature[2] Source
	PSbMV-L	BYMV	PSbMV-P1	
269774	R*	R	R*	1,2,3,6
269818	R*	R	R*	1,2,3,6
347328	R	R	R*	5
347442	R	R	R*	5
347449	R	R	R	
347450	R	R	R	
347452	R	R	R*	5
347453	R	R	R	
347455	R	R	S**(H)	5
347456	R	R	R**(H)	5
347464	R*	R**(S)	S*	1,4,5,7
347465	R*	R**(S)	S	7
347466	R*	R**(S)	R*	1,4,5,7
347467	R*	R**(S)	S**(R,H)	1,4,5,7
347468	R	R	R*	5
347469	R	R	R*	5
347470	R**(H)	S	R*	1,5
347484	R	R	R**(H)	5
347485	R	R	R*	5
347487	R	R	R*	5
347492	R*	R**(S)	R**(H)	1,4,5,7
347494	R**	R	R*	1,5
347523	R	R	R*	5
356984	R	R	R*	5
356991	R	R	S	
357003	R	R	R*	5
357015	R	R	R*	5
357023	R	R	R**(H)	5
357024	R	R	R	
357026	R	R	R*	5
357038	R	R	R*	5
378158	R	R	R*	5

1. Tests reported in the literature agree (*), or disagree (**), with the results reported here. Reported results disagreeing with these are in parentheses: S= susceptible; H= heterogeneous; R= resistant.

2. Numbers refer to sources listed in Literature Cited: 1= Alconero et al., 1986; 2= Ashby et al., 1986; 3= Baggett and Hampton, 1972; 4= Hampton, 1980b; 5= Hampton, 1986; 6= Hampton et al., 1981; 7= Provvidenti, pers. communication

Table 16. Reactions of OSU breeding lines to inoculation with PSbMV-L, BYMV, and PSbMV-P1

Line Designation	Virus Reaction		
	PSbMV-L	BYMV	PSbMV-P1
547-6	R	R	R
547-29	R	R	R
548-17	R	R	R
559-6	R	R	R
564-3	R	R	R
584-16	R	R	R
589-12	R	R	R
615-15	R	R	R
620-1	R	R	R

GENERAL DISCUSSION

The results of this study clearly indicate that resistance to PSbMV-L is conferred by a single recessive gene. The relationship between resistance to PSbMV-L resistance and to BYMV and PSbMV-P1 is less straightforward.

An early report from Goodell and Hampton (1983) stated that the mo gene conferred resistance to both BYMV and PSbMV-L. When 'Quincy' and 'Avon' showed apparent differential reactions to the two viruses, a linkage study was initiated as a major objective of this research. The intent was to elucidate the relationship between the mo gene and resistance to PSbMV-L through testing of F3 families of crosses using either 'Sounder' or 'Abador' as the susceptible parent. Half of each family was to be tested with BYMV and the other half with PSbMV-L. A crossover, indicated by a differential reaction to the two viruses within a family, would provide evidence that resistance to the two viruses was conferred by two separate but linked genes. With significantly large numbers of families tested, a map distance for the genes could be estimated. The linkage study was abandoned due to difficulties encountered in infecting the F3 families with BYMV, and in obtaining clear symptoms in possibly infected individuals.

Three years after commencing this study, I was informed that Dr. R. Provvidenti in Geneva, NY was studying the inheritance of PSbMV-L1 and its linkage relationship with the mo gene. PSbMV-L and PSbMV-L1 are both isolates from infected Lens Plant Introduction lines and belong to the P2 pathotype classification. It is assumed that resistance to the two isolates is conferred by the same gene. 'Quincy' and 'Avon', the apparent crossover cultivars, were tested at the Geneva, NY facility and, contrary to results of this study, were

found to be susceptible to BYMV. Reasons for my failure to duplicate the results of Provvidenti are obscure. It is probable that since he used the same virus strain, as well as others, differences in environmental conditions, such as the use of high intensity lighting probably producing high temperatures, may be implicated. Further attempts to induce infection in these two cultivars while utilizing different environmental conditions and various BYMV isolates, failed. 'Quincy' also showed a probable klendusic reaction when inoculated with PSbMV-L. Therefore, it is plausible that modifying genes which affect 'Quincy's reaction to PSbMV-L, as well as the specific environmental conditions under which the tests were performed, also acted to suppress infection with BYMV in these cultivars. If the infection with BYMV is thus totally suppressed, the cultivars will incorrectly appear to possess the recessive allele of Mo. A successful test of F2 or F3 progenies of 'Quincy' and 'Avon' with BYMV would have determined whether these parents carried the allele for BYMV susceptibility. It is also possible that the environmental conditions at Geneva, NY, may have favored the activity of modifying factors which negate the effect of the mo gene for BYMV resistance. Although these results with 'Avon' and 'Quincy' differ from those of Provvidenti, he has identified Pisum Plant Introduction accessions which exhibit a differential response to PSbMV-L and BYMV, and has confirmed by genetic studies (1988a) that resistance to PSbMV-L is conferred by a separate gene, designated sbm-2, which is closely linked with mo.

While the relationship between resistance to PSbMV-L and the mo gene has been examined by Provvidenti (1988a), the relationship between resistance to PSbMV-L and PSbMV-P1 has not been studied as closely. Among 202 commercial cultivars, 32 P.I. lines, and 9 OSU lines included in this study, a total of 38 genotypes were

resistant to both PSbMV-P1 and PSbMV-L, and 126 were susceptible to PSbMV-P1 but resistant to PSbMV-L. No line was found to be resistant to PSbMV-P1 and susceptible to PSbMV-L. Thus, it is only possible to conclude from this study that pea cultivars which carry resistance to PSbMV-P1 tend to be also resistant to PSbMV-L, but there is no apparent tendency for cultivars resistant to PSbMV-L to be resistant to PSbMV-P1.

The 100% coincidence of resistance to PSbMV-P1 and PSbMV-L among pea genotypes tested was of particular interest. There seemed to be no logical explanation of this absolute coincidence since sbm-1, controlling resistance to PSbMV-P1, and sbm-2, controlling resistance to PSbMV-L, are on different chromosomes and are, thus, genetically independent. However, the recent discovery that sbm-3 (conferring resistance to PSbMV-L, as does sbm-2, on Chromosome 6) is closely linked to waxless gene, wlo, on Chromosome 2 (Dr. R. Provvidenti, personal communication), offers a probable explanation. Genes sbm-3 and sbm-1 exist in a cluster with at least two other virus-resistance-conferring genes, all locked together in homozygous-recessive phase, near gene wlo. Accordingly, any genotype resistant to PSbMV-P1 would also contain genotype sbm-3 sbm-3, assuring resistance to PSbMV-L. Interestingly, smb-4, conferring resistance to PSbMV-4, occurs in this same gene cluster, suggesting that genotypes resistant to PSbMV-P1 and PSbMV-L (PsbMV-P2) should also be resistant to PSbMV-P4.

SUMMARY AND CONCLUSIONS

The F1 generation of all crosses except those with 'Quincy' and 'Avon' as susceptible parents was infected with PSbMV-L indicating that resistance is recessive. Some crosses involving either 'Quincy' or 'Avon' as their susceptible parent did not react typically. A klendusic reaction, presumably caused by modifying genes in combination with the environment, seemed to be suppressing infection in 'Quincy' and its progeny.

The F2 populations, except in crosses involving 'Quincy', did not significantly deviate from a 3 S: 1 R expected ratio, suggesting that a single recessive gene confers resistance to PSbMV-L. The F3 family data were analyzed for fit to a 1 S: 2 SG: 1 R genotypic ratio and to a 3 S + SG: 1 R ratio in which the all-susceptible (S) and the segregating (SG) families were combined. Half of the F3 family tests with crosses and reciprocals tested separately, failed to fit the 1 S: 2 SG: 1 R ratio but in only one cross, Avon x B445-66, did both the cross and reciprocal significantly deviate from the expected. Failure to fit a 1 S: 2 SG: 1 R ratio was always due to an excess of segregating or resistant families probably due either to chance escapes or the klendusic reaction conditioned by modifying genes. All F3 family populations, except those from K6, Avon x B445-66, fit an expected 3 S + SG: 1 R ratio. Data from backcrosses to the resistant parent fit a 1 S: 1 R ratio as expected for inheritance of a single recessive gene.

Of the 202 cultivars tested for resistance to PSbMV-L, BYMV and PSbMV-P1, 142 were resistant to both PSbMV-L and BYMV, two cultivars had apparent differential reactions, and 58 were susceptible to both viruses. The only three cultivars resistant to PSbMV-P1 were also resistant to PSbMV-L and BYMV. The two cultivars demonstrating a differential response to PSbMV-L and BYMV infection,

'Quincy' and 'Avon', were subsequently utilized as parental lines in the inheritance study. Although this differential response has been questioned by Dr. R. Provvidenti, the conclusion that resistance to PSbMV-L is controlled be a gene different from the mo gene for BYMV resistance was confirmed by his identification of a number of Pisum Plant Introduction lines exhibiting differential reactions to the two viruses.

All of 32 Plant Introduction lines tested were resistant to PSbMV-L, all but one were resistant to BYMV, and 27 were resistant to PSbMV-P1.

All of nine PSbMV-P1 resistant breeding lines from OSU were resistant to PSbMV-L and BYMV.

This study indicates that resistance to PSbMV-L in peas is conferred by a single recessive gene linked to the mo gene on chromosome 2. This single recessive gene is common in commercial cultivars, making it readily available for incorporation into a breeding program. As resistance is recessive, it is possible to fix it in a single segregating generation. It is presumed that the tight linkage of the two recessive alleles for resistance to PSbMV-L and BYMV will facilitate the simultaneous incorporation of resistance to both viruses into a single cultivar.

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