Three Pea Seedborne Mosaic Virus Pathotypes from Pea and Lentil Germ Plasm

R. ALCONERO, Germplasm Resources, Northeast Regional Plant Introduction Station, USDA, ARS, and R. PROVVIDENTI, and D. GONSALVES, Department of Plant Pathology, Cornell University, New York State Agricultural Experiment Station, Geneva 14456

ABSTRACT

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Three isolates of pea seedborne mosaic virus (PSbMV), P-1 and P-4 from pea (*Pisum sativum*) and L-1 from lentil (*Lens culinaris*) germ plasm accessions, were distinguished by their capacity to infect pea genotypes. Resistance in peas was isolate-specific. Resistance to the L-1 isolate was associated with bean yellow mosaic virus resistance and also with a delayed reaction to isolate P-4. Several pea germ plasm accessions were resistant to all three isolates. All isolates were infective to 26 genetic lines of chickpea (*Cicer arietinum*), a new host, but not to 12 accessions of pigeon pea (*Cajanus cajan*). Antisera produced against the cytoplasmic inclusion protein induced in peas by P-1 and L-1 were useful in detecting infections by the indirect ELISA method and were generally more sensitive than antisera to the viral protein.

Germ plasm collections of pea (*Pisum sativum* L.) and lentil (*Lens culinaris* Medik.) may not only be reservoirs for pea seedborne mosaic virus (PSbMV) but also valuable sources of resistance to this pathogen (3,10,12,13,17). Resistance in pea to the common strain of PSbMV is reported to be conditioned by a single recessive gene, designated *sbm* by Hagedorn and Gritton (8). Infection in this host species may be latent or may be expressed by a wide variety of symptoms ranging from rapid and lethal to delayed and transitory (11,15). This variety of plant responses in pea has suggested a modifier-gene system affecting resistance to this virus (11). Recently, variants of PSbMV have failed to infect pea cultivars susceptible to the common strain but resistant to bean yellow mosaic virus (BYMV). Gene *mo*, responsible for resistance in peas to BYMV, has been reported to confer resistance to these variants of PSbMV (6). Resistance in lentil to the common strain of PSbMV is conferred by a single recessive gene, designated *sbv* by Haddad et al (7). Inheritance of resistance to the lentil strain is yet to be determined.

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Breeding for resistance to PSbMV in peas may be affected by the kind of plant response to infection. For example, pea progenies from certain apparently PSbMV-immune parents may show susceptibility to the virus only after a series of inoculations (11). Likewise, the variety of plant responses to PSbMV infection may affect virus eradication procedures when slow development of the virus in test plants results in delays in detection with bioassay or serological methods (2,9). The interaction between PSbMV and pea or lentil becomes more apparent as we study more representatives of the host and pathogen. The following report distinguishes three isolates of PSbMV by differential responses of pea genotypes to infection, demonstrates the value of cytoplasmic inclusion protein (CIP) antisera to detect the virus in plant tissues, and notes a new leguminous species, chickpea (*Cicer arientinum* L.), susceptible to PSbMV.

MATERIALS AND METHODS

Virus isolation and tests for infection. The isolates were obtained from seedborne infections of single plants in germ plasm accessions PI 432112 (isolate from lentil labeled L-1), PI 179458 (isolate from pea labeled P-1), and PI 471128 (isolate from pea labeled P-4). Prior to their use, these isolates were passed through three single-lesion transfers in *Chenopodium quinoa* Willd. Stock cultures were maintained in pea plants of cultivar Ranger, which also served as a source of inoculum. A range of possible host species was tested for susceptibility to the three strains. The tests were made in aphid-free greenhouses at 22–30 C with fluorescent or sodium vapor lights augmenting sunlight when needed. Twelve test plants of each host per isolate were manually inoculated when the first two true leaves expanded and again 1 wk later unless otherwise mentioned.

Virus and CIP purification. The method of Yeh and Gonsalves (18) was used to purify CIP from plants infected with isolates P-1 and L-1. The two virus isolates were purified according to the method of Dougherty and Hiebert (5) with some modifications. Infected shoots of Ranger pea were harvested and frozen 24–28 days after inoculation. Tissues were homogenized in cold potassium phosphate buffer (0.5M, pH 7.5, with 0.1% Na_2SO_3 and 0.01 M ethylenediaminetetraacetic acid) at 2 ml/g of tissue for 2 min, then in cold chloroform and carbon tetrachloride at 0.5 ml of each per gram of tissue for an additional minute. The homogenate was centrifuged at 400 *g* for 5 min. The supernatant was saved, and the pellets were resuspended in phosphate buffer and centrifuged at 400 *g* for 5 min. The combined supernatants were centrifuged at 1,000 *g* for 5 min, the pellets were discarded, and the supernatant was centrifuged at 10,000 *g* for 20 min. At this stage, the pellets contained the cytoplasmic inclusions and the supernatant contained the virus. The virus was precipitated by adding polyethylene glycol (PEG, mol wt 8,000, 4% [w/v] final concentration) while stirring for 1.5 hr at 4 C. The precipitated virus was collected by centrifugation at 10,000 *g* for 10 min and resuspended in 40 ml of 0.02 M phosphate buffer, pH 8.2, with 0.1% 2-mercaptoethanol. The virus preparation was centrifuged in 30% (w/v) CsCl gradient at 80,000 *g* for 24 hr at 6 C. The virus zone was removed and diluted in an equal volume of buffer, then the virus was precipitated by adding 0.3 M NaCl and 5% PEG while stirring 1 hr at 6 C. The preparation was centrifuged at 10,000 *g* for 15 min, and the pellet was resuspended in 11 ml of 0.02 M $KHPO_4$ with 0.1% 2-mercaptoethanol, pH 8.2, before a second 24-hr CsCl centrifugation at 80,000 *g* as a final purification step.

Polyacrylamide gel electrophoresis in minislab gels (Ideal Scientific Co, Corvallis, OR) was used to monitor the quality of samples during the purification procedures. The samples were dissociated in 0.1 M tris-HCl, pH 6.8, 2.5% sodium dodecyl sulfate, 5% 2-mercaptoethanol, 5% sucrose, and 0.001% bromophenol blue. Gels and buffers were prepared according to Laemmli (16). Electrophoresis was with a constant voltage at

18V/cm for 1 hr. Protein bands were visualized by staining with Coomassie Blue R-250.

Preparation and use of antisera in ELISA. Purified viral antigen (2 mg) in 1 ml of 0.037 M potassium phosphate buffer, pH 7.6, was emulsified with 1 ml Freund's complete adjuvant. Part of the emulsion (about 0.2 ml) was injected into the toe pad of a New Zealand rabbit, and the rest of the emulsion was injected intramuscularly into the hind legs. The injection procedure was repeated three times at weekly intervals, except subsequent antigen (1 mg/L) was injected with Freund's incomplete adjuvant. Purified CIP antigen in 1 ml of 0.12 M guanadine-HCl, 0.02 M tris-HCl, pH 8.2, was emulsified and injected as for purified virus antigen. Rabbits were bled every 7–10 days after the third injection for a period of 2 mo. The direct double-antibody sandwich method (4) and an indirect method (18) of enzyme-linked immunosorbent assay (ELISA) were used to detect the presence of the virus in test plants. In the direct method, test samples were used at 1:20 (w/v) in phosphate-buffered saline, 1% polyvinyl-pyrrolidone (mol wt 40,000), 0.001 M ethylenediaminetetraacetic acid, and 1 drop per 1,000 ml of polyoxethylene sorbitan monolaurate (Tween 20). Micro-ELISA wells were coated with 5 μg/ml γ-globulin, and the globulin-akaline phosphatase conjugate was used at 1:400. In the indirect method, test samples were used at 1:20 (w/v) in coating buffer and the antiserum was used at 1:1,000 dilution. Antiserum to a known PSbMV isolate obtained from R. O. Hampton and used in earlier studies (2) was used to compare ELISA reactions with those obtained with antisera produced from P-1 and L-1 antigens. Reactions were measured with an Artek 210 micro-ELISA spectrophotometer (Artek Systems Corporation, Farmingdale, NY). Samples were diluted when necessary to obtain the appropriate readings.

Test for seed transmission in chickpeas. Twenty-five plants of each of five chickpea (*Cicer arietinum*) accessions, ILC 35, ILC 260, Sonora 80, Surutato 77, and PI 273879, were inoculated twice with P-1, P-4, or L-1. One hundred seeds that developed from infected plants were tested for PSbMV by observing the seedlings in the greenhouse and by serology using the indirect ELISA method.

Bioassays. *Chenopodium amaranticolor* Coste & Reyn. grown in the greenhouse for 3–4 wk before inoculation was used as a bioasay host in host range studies concurrently with ELISA serology.

Electron microscopy. Expressed sap from infected leaves of Ranger pea was stained with 2% phosphotungstate, pH 6.5, examined by transmission electron microscopy, and 210–226 virus particles were measured per isolate with a grid standard.

RESULTS

Electron microscopy and serology. Flexuous, rod-shaped particles with a modal length of 750–780 nm were observed in leaf sap of Ranger pea infected with P-1, P-4, or L-1. The three isolates were detected by both direct and indirect ELISA when antisera to P-1 and L-1 virions were used as well as with the antiserum to a known PSbMV isolate obtained from R. O. Hampton. Antisera to CIP from Ranger pea infected with P-1 or L-1 reacted much more strongly with the three isolates in the indirect ELISA than in the direct method. In the indirect test, reactions were stronger between all three antigens and both CIP antisera than they were with antisera prepared from either virus (Table 1). Extracts from samples infected with L-1 reacted more strongly with all five antisera in parallel tests than did those from P-1- and P-4-infected tissues, and the strongest reactions were observed in lentil samples. Samples infected with the P-4 isolates usually gave the lowest A_{405nm} absorbance in direct and indirect ELISA. Surutato 77 chickpea samples infected with P-1 isolate reacted the strongest when the Hampton H(v) antiserum was used as conjugate in plates coated with either P-1, L-1, or Hampton PSbMV γ-globulin in direct ELISA (Table 1).

Host range. Species tested and found not susceptible to infection by the three isolates after back-inoculation to *C. amaranticolor* were *Cajanus cajan* (L.) Huth. (12 accessions tested), *Datura stramonium* L., *Glycine max* (L.) Merr. 'Bragg,' *Gomphrena globosa* L., *Lycopersicon esculentum* Mill. 'Marglobe,' *Nicotiana benthamiana* L., *N. glutinosa* L., *N. tabacum* L. 'Samsun NN,' *Solanum melongena* L. 'Black Magic,' and *Vigna unguiculata* (L.) Walp. 'California Cowpea #5.' Species susceptible to all three isolates were *Chenopodium amaranticolor* (local lesions only), *C. quinoa* (local lesions with P-4 but also systemic with P-1 and L-1), *Phaseolus vulgaris* L. (local lesions in Black Turtle Soup), *Vicia faba* L. var. *minor* (systemic mosaic, stunting, and leaf malformation), *Pisum sativum, Lens culinaris,* and *Cicer arietinum.* In the last three species, more representative lines were tested and different responses were observed.

Reactions of pea genotypes. All pea cultivars tested were infected by the P-1 isolate and expressed symptoms within 7 days of the first inoculation. Symptoms in the greenhouse throughout the year consisted of early leafroll and veinclearing, developing into a mosaic, usually with some stunting. Isolate P-4 also infected all pea cultivars tested with severity equal to that of P-1, but in many cultivars, symptom expression was delayed 7–10 days in winter and infection was erratic. Detection by ELISA was also delayed in these cultivars infected by P-4. In early summer (May and June), this delay was often only 3–4 days, and more test plants became infected in the first inoculation. The long delay of infection by P-4 in cultivar Bonneville during winter could

Table 1. Serological reactions (infected:healthy ratios) between P-1, P-4, and L-1 pathotypes of pea seedborne mosaic virus (PSbMV) in pea, lentil, and chickpea and antisera to standard PSbMV, P-1 and L-1 virions, and cytoplasmic inclusion protein (CIP)

	Indirect ELISA												
	Ranger pea (antisera)				Tekoa lentil (antisera)				Surutato 77 chickpea (antisera)				
Antigens	P-1(v)	P-1(CIP)	L-1(v)	L-1(CIP)[a]	P-1(v)	P-1(CIP)	L-1(v)	L-1(CIP)	P-1(v)	P-1(CIP)	L-1(v)	L-1(CIP)	H(v)[b]
P-1	2.96[c]	5.84	2.38	8.56	2.94	7.07	2.36	3.66	2.49	5.27	2.88	6.13	4.81
P-4	2.90	5.23	2.46	7.65	2.82	4.57	2.31	2.49	2.30	4.40	2.52	4.12	3.65
L-1	4.00	7.87	3.44	11.57	5.48	12.25	4.77	11.25	2.88	8.06	3.54	9.13	5.99
	Direct double-sandwich ELISA/Surutato 77 chickpea												
Antisera													
Coating		P-1(v)	P-1(v)	P-1(v)		L-1(v)	L-1(v)	L-1(v)			H(v)	H(v)	H(v)
Conjugates		P-1(v)	L-1(v)	H(v)		P-1(v)	L-1(v)	H(v)			P-1(v)	L-1(v)	H(v)
Antigens													
P-1		3.25	2.63	6.30		4.64	2.44	7.65			4.35	2.95	7.91
P-4		1.97	2.65	2.20		2.25	2.06	2.66			2.12	2.12	2.61
L-1		4.71	2.65	2.80		5.27	2.04	3.92			4.66	2.36	3.82

[a](v) and (CIP) = antisera prepared with virus and CIP, respectively, from peas, lentils, and chickpeas.
[b]H(v) = antiserum from standard PSbMV from R. O. Hampton.
[c]Numbers refer to the average A_{405nm} reading of four sample wells divided by the average A_{405nm} reading of four control wells in the same micro-ELISA plate. A_{405nm} for controls ranged from 0.05 to 0.48; A_{405nm} for test samples ranged from 0.89 to 4.62.

be shortened and erratic infections improved if Bonneville showing symptoms was used as the inoculum source instead of Ranger. The host range of P-4 remained the same, whichever source of inoculum was used. Isolate L-1 infected fewer pea cultivars, and those not infected with this isolate were the same as those for which delays in symptom expression were observed in inoculations with P-4 (Table 2). No cases of latent infection were detected in L-1 infections. L-1 infection invariably resulted in marked leaf cupping, leaf malformations, mosaic, and shortened internodes leading to severe stunting.

Plant introductions (PIs) of peas reported homogeneously immune to PSbMV by Hampton and Braverman (11) also did not become infected by P-1, P-4 or L-1, except for PIs 347470 and 347494, where a few plants were infected by P-4 and L-1. PIs 193586 and 193835, reported resistant to PSbMV by Stevenson and Hagedorn (17), remained uninfected by the three strains in our trials. In PIs reported as sources of PSbMV immunity (12); 343305 was infected by the three strains; 347329, 347422, and 347464 were infected by P-1 only; and 347466, 347467, and 347492 remained uninfected by the three strains (Table 3). Representatives of pea differentials used to distinguish PSbMV isolates (14) were also used to distinguish between P-1, P-4, and L-1 isolates. PIs 269818 and 269774 were consistently infected by P-4 only. PI 174319 responded to P-1 infection with a rapid, whole-plant necrosis but was only moderately affected by P-4 and severely stunted by L-1. PI 269804 was severely affected by L-1 but only moderately by P-1 and P-4.

Reactions of chickpea genotypes. Twenty-six chickpea lines were tested for resistance to the three isolates and were found susceptible in varying degrees (Table 4). 1CCC4 was the least affected, showing only light systemic mosaic to all three isolates after a relatively long (3–4 wk) incubation period. Isolate P-1 caused light mosaic in all lines tested, and L-1 was severe in all but 1CCC4, causing leaf and shoot necrosis with stunting as well as mosaic. P-4 usually caused a well-developed mosaic, necrotic lesions in leaves, shoot-tip death, and stunting. No seed transmission was detected in the five accessions tested.

Reactions of lentil and pigeon pea genotypes. Four lentil cultivars, Red Chief, Brewer, Eston, and Tekoa, were tested and found susceptible to the three isolates. P-1 and P-4 were relatively mild and L-1 usually caused severe reactions, especially in Tekoa. Pigeon peas (*Cajanus cajan*) did not become infected in two trials (12 plants per isolate tested per trial) with PIs from Iran (394033, 394988, 396017, 397058, 397272, 397394, 399637, and 399640), from India (183295 and 249631), from Argentina (304646), and from Mexico (312195).

DISCUSSION

Virus isolates P-1, P-4, and L-1 have similar morphology, host range, symptomatology, seed transmission capacity, and serological reactions to other isolates of PSbMV. They differ, however, in their infection of pea genotypes and therefore may be considered pathotypes of PSbMV. Isolate P-1, causes the well-known leafroll and mosaic in pea cultivars that are commonly associated with the common strain of PSbMV (9). Isolate L-1 is much more severe in susceptible pea cultivars. This isolate in our tests only infected pea cultivars that were also susceptible to BYMV, as does the PSbMV-L isolate from lentils (12). Pea cultivars resistant to BYMV were also resistant to the L-1 isolate. Unlike the PSbMV-L isolate, L-1 may be easily purified with the same procedures used for the purification of the P-1 isolate. Pea, lentil, and chickpea reactions to L-1 were generally more severe than to isolates P-1 and P-4.

Isolate P-4 differs from P-1 and L-1 in two main characteristics. It infects peas in PIs 269818 and 269774, which are resistant to P-1 and L-1, is also infective to pea cultivars susceptible to P-1 but not to L-1, and is infective in a different manner than P-1. Its development in these cultivars is delayed and infectivity is erratic, especially during the winter months in the greenhouse. It may be significant that delay in symptom expression, such as in pea cultivars Sparkle and Wando, for example, is associated with resistance to infection by isolate L-1, and furthermore, resistance to BYMV. We are investigating these apparent associations. Delays in symptom expression and PSbMV detection in infected tissues have been noted previously (2,9). The genetic basis in the host and relations to pathotypes of PSbMV need further clarification.

Table 2. Responses of pea (*Pisum sativum*) cultivars to manual inoculation with pathotypes P-1, P-4, and L-1 of pea seedborne mosaic virus[a]

Pea cultivar	Virus pathotypes		
	P-1	P-4	L-1
Alaska	+	+	++
Alderman	+	+	++
Dwarf Green Sugar	+	+	++
Freezonian	+	+	++
Giant Stride	+	+	++
Green Arrow	+	+	++
Lincoln	+	+	++
Mammoth Melting Sugar	+	+	++
Ranger	+	+	++
Thomas Laxton	+	+	++
World's Record	+	+	++
Bonneville	+	+D	−
Early Frosty	+	+D	−
Hundredfold	+	+D	−
Knight	+	+D	−
Laxtonian	+	+D	−
Laxton Progress	+	+D	−
Laxton Superb	+	+D	−
Little Marvel	+	+D	−
Perfected Freezer	+	+D	−
Progress #9	+	+D	−
Sparkle	+	+D	−
Wando	+	+D	−

[a] + = Relatively mild systemic mosaic, leafroll, and stunting; +D = delay of 7–10 days in development of symptoms, infection erratic in winter; ++ = severe stunting, mosaic, and leaf malformation; and − = no infection.

Table 3. Reactions of plant introductions of *Pisum sativum* to P-1, P-4, and L-1 pathotypes of pea seedborne mosaic virus[a]

Accession	Virus pathotypes		
	P-1	P-4	L-1
PI 193586	R	R	R
PI 193835	R	R	R
PI 269774	R	S	R
PI 269818	R	S	R
PI 343305	S	S	S
PI 347329	S	R	R
PI 347422	S	R	R
PI 347464	S	R	R
PI 347466	R	R	R
PI 347467	R	R	R
PI 347470	R	R[b]	R[b]
PI 347492	R	R	R
PI 347494	R	R[b]	R[b]

[a] R = resistant and S = susceptible.
[b] These lines include some susceptible plants.

Table 4. Responses of chickpea (*Cicer arietinum*) to manual inoculation with pathotypes P-1, P-4, and L-1 of pea seedborne mosaic virus[a]

Chickpea accession	Virus pathotypes		
	P-1	P-4	L-1
ILC 35	+	++	+++
ILC 132	+	+++	+++
ILC 171	+	++	+++
ILC 202	+	++	+++
ILC 260	+	++	+++
ILC 294	+	++	+++
ILC 482	+	+++	+++
ILC 484	+	++	+++
ILC 517	+	+++	+++
ILC 523	+	+++	+++
ILC 591	+	+++	+++
ILC 1002	+	++	+++
ILC 1929	+	++	+++
ILC 3279	+	++	+++
VEW 1004	+	++	+++
VC 5	+	+++	+++
CP 8	+	++	+++
C 235	+	++	+++
1CCC4	+	+	+
PI 273879	+	++	+++
Aztec	+	++	+++
Giza 1	+	++	+++
Lyons	+	+++	+++
Sonora 80	+	++	+++
Spanish White	+	+++	+++
Surutato 77	+	+++	+++

[a] + = Relatively mild systemic mosaic; ++ = systemic mosaic, foliar necrotic lesions, shoot tip death, some stunting; and +++ = systemic mosaic, extensive shoot necrosis, and severe stunting.

We found that antisera to the cytoplasmic inclusions induced by isolates P-1 and L-1 reacted more strongly to the antigens in sap from infected plants than antisera prepared from the purified virus in indirect ELISA. This corroborates the observations made by Yeh and Gonsalves with papaya ringspot virus and watermelon mosaic virus (18). The weaker serological reactions observed in samples infected with isolate P-4 probably reflect the lower concentration of the virus in the plants. A slower virus replication may result in a delay in symptom expression in pea genotypes that are resistant to BYMV and could be useful in field resistance to P-4.

Resistance to the three pathotypes obtained from the pea and lentil accessions is present in the two lines used by Stevenson and Hagedorn (17), as well as in several other plant introductions. Further studies of PSbMV pathotypes in germ plasm collections are needed to determine if there are virus strains that attack these genotypes. Discrepancies in reported responses of pea introductions to infections by PSbMV may be due to inadvertent changes in the germ plasm accessions. We have observed genetic changes in pea accessions as a result of possible inadvertent mixtures and the elimination of PSbMV-infected individuals (2). The accessions used to distinguish PSbMV pathotypes should be as genetically stable as possible to avoid problems in identification.

Host range studies and descriptions of PSbMV do not include chickpea as a susceptible species (1,14). We found all 26 accessions tested susceptible, with 1CCC4 being the least affected by the three pathotypes. W. J. Kaiser in Pullman, WA, has also observed PSbMV infection in chickpea under experimental conditions (*personal communication*). These observations, as well as those of PSbMV in lentil seed (12) indicate a need for more studies of PSbMV in pulse crops.

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