Characterization of Isolates of Four Aphid-Transmitted Sweet Potato Viruses

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ABSTRACT

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Isolates of four aphid-transmitted viruses (feathery mottle [FMV], russet crack [RCV], internal cork [ICV], and chlorotic leaf spot [CLSV]) from sweet potatoes were purified from mechanically inoculated plants of *Ipomoea nil* with yields of 8–18 mg of virus per kilogram of tissue. An antiserum was prepared for each isolate and the reciprocal titers were from 512 to 1,024 in homologous microprecipitin tests. Based on three serological procedures and particle morphology, the four isolates are closely related strains of sweet potato feathery mottle virus. In microprecipitin tests, the titers of each heterologous reaction were the same as the homologous reaction or one dilution step less; cross-absorption with each heterologous antigen removed all antibodies to the homologous virus; and intensities of homologous and heterologous reactions closely resembled those obtained with the ELISA technique. The normal length of the flexuous rod-shaped particles of FMV, RCV, ICV, and CLSV, were 829 ± 38, 834 ± 39, 838 ± 38, and 845 ± 32 nm, respectively. The four isolates caused similar symptoms in the foliage of *Ipomoea* spp., but differences were observed in the symptoms on roots of infected sweet potato plants. Plants of Jersey inoculated with RCV, ICV, and CLSV had russet crack symptoms that were not present in the FMV-inoculated plants or in the controls.

Aphid-transmitted viruses occur in all major sweet potato-producing areas and may cause reduction in yield (12,20) and loss of market quality (5) in some cultivars. This group of viruses is not well understood (4). Martyn (16) listed four viruses: sweet potato internal cork virus, sweet potato leaf spot virus, sweet potato mosaic virus A, and sweet potato ringspot virus. Steinbauer and Kushman (26) listed three: internal cork virus, chlorotic leaf spot virus, and russet crack virus. Most of these viruses have been characterized by host range and symptomatology, a few by their styletborne relationship to aphids (17,23,27,29) or by flexuous rod-shaped particles ~840 nm long (22), and fewer have been purified (4,19). Thus, Campbell et al (4) proposed that aphid-transmitted, styletborne viruses of sweet potato should be considered as strains of one virus until differences other than symptom expression were demonstrated. They chose "sweet potato feathery mottle virus" (FMV) (9) as the preferred name for this virus and hypothesized from results with cross-protection tests that russet crack is caused by a strain of FMV; ie, FMV-RC (4). A virus recovered from sweet potato cultivar Georgia Red in North Carolina had the characteristics of, and was identified as, FMV (18). During a later survey of viruses of sweet potato in North Carolina, differentiation of two strains of FMV was based on the host reactions of *Ipomoea nil, I. purpurea*, and *Chenopodium amaranticolor* (19). One strain was designated as FMV-RC because one isolate was tested and caused russet crack on Jersey sweet potato. Sweet potato vein mosaic virus (SPVMV) from Argentina has shorter particles than FMV isolates and, thus, was recognized as a virus distinct from FMV (21,22).

The objectives of the present paper were to isolate, purify, and compare particle morphology, serology, and symptomatology in *Ipomoea* spp. of four aphid-transmitted viruses of sweet potatoes.

MATERIALS AND METHODS

Virus isolates. Feathery mottle virus (FMV) and russet crack virus (RCV) were the Californian isolates used previously (4,22). They differ in symptomatology: FMV does not cause russet crack symptoms on cultivar Jersey sweet potato (*I. batatas* (L.) Lam.), whereas the RCV isolate does. Isolates of internal cork (ICV) and chlorotic leaf spot (CLSV) viruses were obtained from infected Porto Rico plants kindly supplied by W. J. Martin, Louisiana State University, Baton Rouge 70803. The plants with internal cork were a selection of Porto Rico maintained as a susceptible check in the LSU breeding program whereas the CLS source plants produced foliar symptoms but no internal cork in the storage roots (W. J. Martin, *personal communication*). Thus, they fitted the symptomatology upon which they were described as distinct aphidborne viruses (16,26). Before experimental work was started, each isolate was graft inoculated to *I. setosa* Ker. or *I. hederacea* (L.) Jacq. and consecutively transmitted by aphids (*Myzus persicae* (Sulz.)) to *I. setosa* following an acquisition feeding period of <1 min and by sap inoculation to *I. setosa*. From this host the isolates were graft transmitted to Jersey sweet potatoes that were kept as virus stock plants. For experimental work, the isolates were grafted from virus stock plants to *I. setosa*, then sap-transmitted to *I. incarnata* Choisy, and later sap-transmitted to host-range and virus increase plants.

Host range and symptomatology. Mechanical transmissions were made by triturating infected tissue with a mortar and pestle in 0.05 M phosphate buffer, pH 7.5, containing 0.02 M ascorbic acid and 0.1% charcoal (Nórit SG extra, American Norit Co., Jacksonville, FL 32208). This mixture was rubbed with cotton-tipped swabs onto corundum-dusted leaves or cotyledons of seedlings of *I. incarnata, I. nil* (L.) Roth, and *I. setosa*. In an early trial, more *I. incarnata* were infected when swabs were used (21 of 63 plants) than when the leaves were finger rubbed (12 of 60 plants).

Virus-free plants of sweet potato cultivars Jersey, Porto Rico, and Garnet were produced by meristem culture, maintained, and assayed for freedom from virus as previously described (4). They were inoculated by cleft-grafting infected scions of *I. incarnata*. The scions consisted of two or three node sections. These were bound with latex bandage and covered with a plastic bag for 5–7 days. The grafted scions always remained alive for 2 wk and generally continued to grow indefinitely. Eight to 10 wk after inoculation, the sweet potato roots were washed free of soil and examined for russet crack symptoms.

Seed transmission. Seeds were collected from infected plants of *I. incarnata* grown in the greenhouse, immersed in concentrated sulfuric acid for 30 min, washed, dried, and planted in sterilized potting mix in the greenhouse. The seedlings were observed for 2 mo. Healthy plants of *I. incarnata* were mechanically inoculated and used as positive checks to be sure the environment was favorable for symptom expression. Uninoculated seedlings from virus-free plants were kept as negative checks to detect virus contamination.

Electron microscopy. During purification, preparations of each

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isolate were applied to carbon-coated collodion membranes on grids (48 μm, 300-mesh), negatively stained with 2% potassium phosphotungstate (KPT) (pH 6.5), and examined with an RCA EMU-3G electron microscope.

For particle measurement, serologically specific electron microscopy (SSEM) was used following the procedure of Derrick and Brlansky (2,8). Grids (74 μm, 200-mesh) with carbon-coated collodion membranes were floated on drops of FMV antiserum diluted 1:250 with 0.05 M Tris buffer, pH 7.5, for 30 min and washed five times on drops of Tris buffer. Immediately after being washed, the grids were placed for 30 min on drops of sap from infected *I. incarnata* extracted with Tris buffer 1:5 (w/v). The grids were washed three times in drops of Tris buffer containing 0.4 M sucrose and twice in distilled water. Preparations were stained by placing the grids on 5% uranyl acetate in water for 10 min. Excess water was removed with a filter paper and the grids were dried. Electron micrographs were taken by using a Zeiss EM9 electron microscope calibrated with a carbon replica grating with 1,136 lines per millimeter. Particle length was determined by enlarging the negatives with a projector and measuring the particles with a small flexible plastic ruler. One hundred particles were measured and the normal length was calculated from the measurements of 85 particles (1).

Purification. The purification method described previously (4) was modified and used for all four isolates. Cotyledons of 2-wk-old seedlings of *I. nil* were mechanically inoculated. Infected tissue was harvested 26–30 days after inoculation, chilled at 4 C for 4 hr, and homogenized in a blender with 0.5 M borate buffer (pH 9) containing 1% anhydrous sodium sulfite and chloroform (1:2–3:1–1:5, w/v/v). The aqueous phase was collected after low-speed centrifugation at 8,000 rpm for 10 min in a GSA rotor of a Sorvall centrifuge. After the aqueous supernatant was strained through cheesecloth, Triton X-100 was added to 2% (v/v); the mixture was stirred at least 12 hr at 4 C and centrifuged at low speed. The virus was sedimented at 27,000 rpm for 2 hr in a Beckman 30 rotor, pellets were resuspended in 0.05 M borate buffer, pH 9, containing 1% Triton X-100. The virus suspension was clarified by low-speed centrifugation and given another cycle of differential centrifugation. The resuspended pellets were layered onto a 10-ml cushion of 0.05 M borate buffer (pH 9) containing 20% sucrose and 0.1% Igepon T-73 (11) and centrifuged for 2.5 hr at 22,500 rpm in a Beckman SW 25.1 rotor. Later, 1% Triton X-100 was substituted for Igepon T-73 (R. J. Shepherd, *personal communication*). Pellets were resuspended in 0.025 M borate buffer and clarified by low-speed centrifugation to give a partially purified preparation. Virus concentration was estimated by determining the extinction coefficient, $E_{260}^{0.1\%} = 2.5$ (19), which is characteristic of many rod-shaped viruses.

Antiserum production. An antiserum to each isolate was prepared by injecting rabbits at 8- to 10-day intervals. The first two injections consisted of a 1:1 emulsion of 1–3 mg of partially purified virus in Freund's incomplete adjuvant injected intramuscularly in both hind legs. A third injection with the same quantity of virus was given intravenously. The rabbits were bled by cardiac puncture 7–10 days after the last injection; subsequent bleedings were taken at 5- to 8-day intervals. Before use, each antiserum was absorbed with a partially purified extract of healthy *I. nil*. Leaves were homogenized as for virus preparations except Triton X-100 was not used. The extract was clarified by low-speed centrifugation, concentrated by high-speed centrifugation, and resuspended to 1/20th the initial volume with 0.05 M borate buffer, pH 9. An equal volume of this preparation was added to the antiserum and incubated for 2 hr at 36 C followed by a low-speed centrifugation (14,000 rpm for 20 min in a Sorvall SS-34 rotor) and a high-speed centrifugation (32,000 rpm for 2 hr in a Beckman 65 rotor). The absorption process was repeated once and the absorbed antiserum was dialyzed against 0.85% saline or the γ-globulin was precipitated with ammonium sulfate (6).

Microprecipitin test. Antiserum titers were determined in microprecipitin tests in disposable plastic petri plates. Droplets of 15 μl of 0.85% saline solution were evenly spaced in the plates with a Pipetman® (West Coast Scientific, Oakland, CA 94618) pipettor and twofold serial dilutions of antiserum were made in the drops. Preparations (15 μl) of partially purified virus or of healthy sap were added to each drop, the mixture was stirred, and the droplets were covered with mineral oil (viscosity 340/365). Plates were kept at room temperature and observed with a dissecting microscope 3 hr later. Virus used for the determination of antiserum titers was partially purified by the standard method and adjusted to a concentration of $A_{260} = 0.25$. The healthy concentrate was used at dilutions from 1/5 to 1/10. Normal serum and virus controls were included in each test. Microprecipitin tests were repeated with the preparations of antigens and antisera diluted in Tris buffer 0.01 M, pH 7.5, containing 0.85% saline (24).

Enzyme-linked immunosorbent assay (ELISA). The experimental procedures for the ELISA technique were those of Clark and Adams (6,7) and were done as outlined before (3).

RESULTS

Host reaction. In one experiment, *I. setosa, I. nil,* and *I. incarnata* were mechanically inoculated with FMV, RCV, ICV, and CLSV isolates. After 10–12 days, the infected plants began to exhibit foliar symptoms. Uninoculated checks remained symptomless throughout the test period. Foliar symptoms induced by the four isolates in the *Ipomoea* sp. were similar and showed vein clearing, chlorotic leaf spots, vein banding, crinkling, and stunting. *I. setosa* showed symptoms less frequently with any isolate than did *I. incarnata* or *I. nil* (Table 1).

In another experiment, eight virus-free plants of each of three sweet potato cultivars (Jersey, Porto Rico, and Garnet) were graft-inoculated. Irrespective of virus isolate, each cultivar developed similar foliar symptoms; ie, clorotic leaf spots appeared 12–20 days after inoculation and later there was vein banding. A purple ring was observed around the chlorotic spots in Garnet and was occasionally observed in Porto Rico but never in Jersey. The isolates differed in the symptomatology of the roots of infected Jersey plants. Russet crack symptoms developed in roots of plants inoculated with RCV, ICV or CLSV (five, four, and eight plants, respectively, of eight inoculated plants) but not in roots of plants inoculated with FMV or in the nongrafted controls. A few lesions resembling those of russet crack were observed in one of eight plants of cultivar Garnet inoculated with CLSV, but not in plants inoculated with other isolates. Russet crack lesions were not observed in cultivar Porto Rico infected with any isolate.

Six cuttings were rooted from three of the Porto Rico plants inoculated with each isolate and grown for a summer in field plots at Davis. The two largest roots from each plant were stored at 20–22 C for 3 mo, sliced (5–10 mm thick), and examined for internal cork. Only four small, necrotic lesions (1 mm in cross section × 3 mm long) were found. Three were in roots from plants inoculated with ICV and one was in a root from a plant inoculated with CLSV. Fourteen of 36 roots produced from the internal cork source plants received from W. J. Martin had severe internal cork. None of 36 roots produced from virus-free Porto Rico plants had internal cork.

Seed transmission. During a 2-mo incubation period, no symptoms were observed in approximately 1,800 seedlings from

TABLE 1. Infection of *Ipomoea* spp. following mechanical inoculation with isolates of feathery mottle (FMV), russet crack (RCV), internal cork (ICV), and chlorotic leaf spot (CLSV) viruses

Virus isolate	Host		
	I. setosa	*I. incarnata*	*I. nil*
FMV	2/40[a]	31/70	16/42
RCV	3/40	32/68	22/48
ICV	6/42	35/72	15/39
CLSV	4/37	50/79	18/39
Average infection (%)	9	51	42
Uninoculated control	0/36	0/66	0/49

[a]Ratio = (number of plants with foliar symptoms)/(number of plants mechanically inoculated).

seed produced on infected *I. incarnata*. Twenty-one of 66 controls mechanically inoculated with infective sap showed symptoms after 10 days, indicating that environmental conditions were favorable for the development of foliar symptoms. None of the 200 uninoculated control seedlings developed symptoms.

Properties of the virus isolates. The ultraviolet absorption spectra of partially purified isolates were corrected for light scattering and were similar to those of other filamentous viruses with maxima at 255–262 nm, minima at 246–247 nm, and a 260/280 ratio of 1.31 ± .07 for nine preparations. Yields ranged from 8–18 mg/kg of infected tissue and the best yields were obtained during the winter. The final preparations were colorless and showed birefringence when observed between crossed-polarizing lenses, indicating the presence of anisometric particles. Negatively stained samples from these preparations contained long, aggregated, flexuous rods. The preparations were infective to *I. incarnata* and the plants showed typical symptoms; however, the specific infectivity of all isolates was low. Undiluted preparations at 0.92–1.8 mg/ml infected 30–60% of 8–12 inoculated plants. At 10-fold dilutions, they infected only 0–14% of the plants and at 100-fold dilutions, none were infective. Low-specific infectivity could be due to particle aggregation or deterioration during purification. Length measurements were made on particles from *I. incarnata* sap to avoid aggregation or fragmentation of particles in purified preparations. Long, flexuous rods were present in extracts of infected plants, but not of healthy plants, and the normal lengths were 829 ± 38 nm, 834 ± 39 nm, 838 ± 38 nm, and 845 ± 32 nm for FMV, RCV, ICV, and CLSV, respectively. The distribution of the particles in the 757–925 nm range is given in Fig. 1

Serological relationships. The partially purified preparations used as inject antigens gave antisera with titers from 512 to 2,048 for virus isolates and from 4 to 16 for healthy plant materials. The antibodies to healthy sap were absorbed leaving specific antisera with titers of 512 to 1,024 with their homologous viruses (Table 2). The precipitates in each case were of the flocculent type, which is characteristic of rod-shaped viruses. In heterologous reactions with the other three isolates, the titers were the same as or only one step lower than that of the homologous reaction (Table 2). Similar results were obtained when these experiments were repeated using 0.01 M Tris buffer containing 0.85% saline for diluting sera and antigens. Cross-absorption of any of the four antisera with any heterologous isolate eliminated any visible reaction with the respective homologous antigen.

Absorbance (A_{405}) values of homologous and heterologous reactions with partially purified virus preparations in ELISA (Table 3), as in the microprecipitin tests, indicated only slight differences between the two reactions.

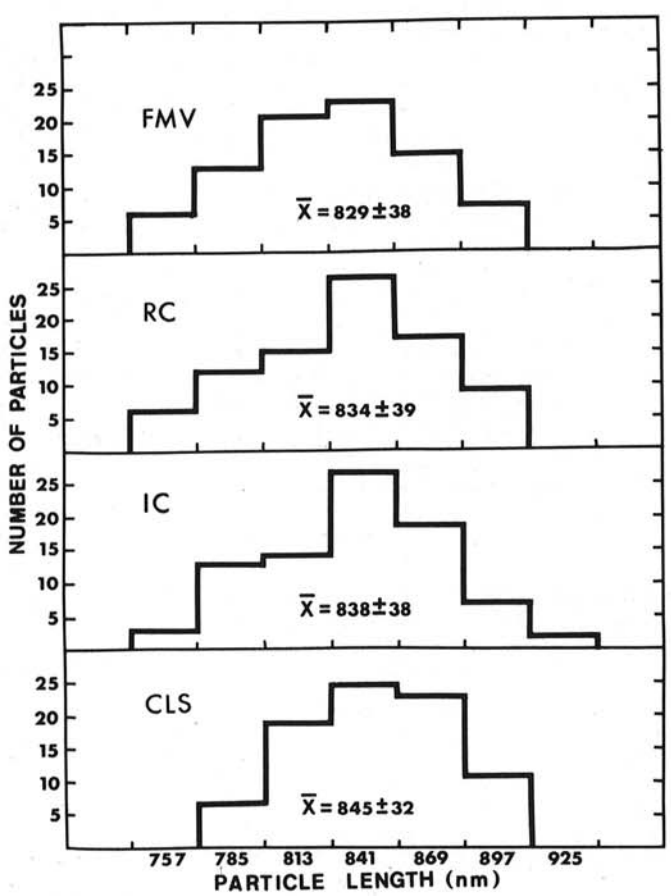

Fig. 1. Length distribution of particles of feathery mottle (FMV), russet crack (RCV), internal cork (ICV), and chlorotic leaf spot (CLSV) viruses.

DISCUSSION

We have studied four virus isolates that are aphid- and sap-transmitted, two of which were obtained from stock cultures of distinctly named entities, internal cork virus and chlorotic leaf spot virus (15). The normal particle lengths of the isolates were from 829 to 845 nm and formed a series that did not differ by one standard deviation. Other FMV isolates are in the range of 800–876 nm (4,17,19,22). The differences in particle sizes of our isolates and those reported in the literature may be caused by factors that are difficult to specify (10) such as strain, host plant (28), staining technique, experimental error in calibration of the electron microscope, and measurement errors. The serological differences between our isolates were minor whether they were compared by the microprecipitin test, by the use of cross-absorbed sera, or by ELISA. The ELISA results are significant because this method has been reported to distinguish between closely related antigens (13). Similar relationships among our isolates have been obtained with the ELISA technique applied to infected sap of *I. incarnata* (3). These isolates, therefore, on the basis of symptomatology, particle morphology, and serology are closely related and should not be classified any higher than strains of one virus. In accordance with the earlier proposal (4), we continue to refer to that virus as FMV.

Our isolates exhibited some symptomatological differences that have a bearing on the etiology of certain diseases. Three isolates (RCV, ICV, and CLSV caused russet crack symptoms in cultivar Jersey but FMV did not, confirming earlier reports (4,15). The present work provides additional evidence, particularly serological

TABLE 2. Serological relationships of feathery mottle (FMV), russet crack (RCV), internal cork (ICV), and chlorotic leaf spot (CLSV) isolates as determined by microprecipitin tests

Antiserum	Antigen[a]				Protein from healthy plants
	FMV	RCV	ICV	CLSV	
FMV	1,024[b]	512	512	512	0
RCV	256	512	256	256	0
ICV	512	512	512	512	0
CLSV	1,024	1,024	1,024	1,024	0
Normal serum	0	0	0	0	

[a] Partially purified virus at a concentration of about 0.1 mg/ml.
[b] Reciprocal titers.

TABLE 3. Serological relationships between isolates of feathery mottle (FMV), russet crack (RCV), internal cork (ICV), and chlorotic leaf spot (CLSV) viruses as determined by ELISA

Serum source of γ-globulins	Antigen[a]				Healthy concentrated sap[b]
	FMV	RCV	ICV	CLSV	
FMV	0.725[c]	0.710	0.694	0.684	0.024
RCV	0.615	0.697	0.623	0.651	0.021
ICV	0.650	0.675	0.689	0.691	0.024
CLSV	0.723	0.722	0.713	0.726	0.020

[a] Antigen concentration 5.0 μg of partially purified virus per milliliter.
[b] Concentrated sap from healthy plants, diluted to $A_{260nm} = 4.0$.
[c] Means of A_{405nm} for fourfold dilution of reacted substrates from four replicates, corrected for light scattering.

evidence, supporting the hypothesis that a strain of FMV causes russet crack (4). The four isolates are not only close serologically but results of the cross-absorption tests showed that a distinct virus is not responsible for russet crack. When the FMV that did not cause russet crack was used to cross-absorb antisera to the three isolates that cause russet crack, all homologous antibodies were removed. Thus, we conclude that russet crack is a manifestation of certain FMV isolates in cultivar Jersey and, as suggested earlier (4), that this cultivar is the best differential plant for diagnosing russet crack.

The proposal that the host reaction of *I. nil, I. purpurea*, and *C. amaranticolor* can distinguish between FMV and RCV-FMV (18) is not warranted for two reasons. First, only one isolate of a large number in each proposed group was tested on Jersey sweet potato for ability to cause russet crack. Second, among our four isolates, three of which cause russet crack, no difference is seen in host reaction on *I. nil* and none caused symptoms on *C. amaranticolor* (M. Cadena-Hinojosa, *unpublished*). These differences probably result from variability among isolates as well as environmental differences that affect symptom expression.

The etiology of internal cork of sweet potato is not yet established. This disease was prevalent for several years in the sweet potato-producing areas but is rare now because susceptible genotypes have been eliminated by selection and breeding (W. J. Martin, *personal communication*). The aphid- and sap-transmissible virus (ICV) that we recovered from the internal cork-susceptible selection of Porto Rico is clearly a strain of FMV. The foliar symptoms caused by ICV on *Ipomoea* spp., including *I. batatas* cultivars Porto Rico, Garnet, and Jersey, had the same appearance as did those caused by the other FMV isolates. The few small lesions that were found in roots of cultivar Porto Rico were not sufficient to prove that any of these FMV isolates is the cause of internal cork.

There are two distinct aphid-transmitted viruses of sweet potato: SPVMV (21,22) and FMV (4,19,22). The aphidborne virus commonly found in sweet potato in most growing areas of the world is probably FMV, which has particles about 840 nm long. SPVMV has been reported only in Argentina where it is associated with sweet potato decline and is distinguished from FMV by having particles 770 nm long (22). Both FMV and SPVMV can be placed in the potyvirus group for the following reasons: both are mechanically transmitted, aphid-transmitted in a nonpersistent manner (4,17,19,21), and associated with pinwheel tubular and laminate inclusions (14,22). Both are flexuous rods with a normal length in the 700–1,000 nm range reported for this group (10). Although some members of the potyvirus group are seed-transmitted (10,25), this phenomenon does not occur with FMV in *I. incarnata*.

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