Etiology of Sweet Potato Chlorotic Dwarf Disease in Argentina

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ABSTRACT

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Chlorotic dwarf (CD), the most important disease in the sweet potato-producing regions of Argentina, is caused by the synergistic combination of two aphid-transmitted potyviruses with a whitefly-transmitted crinivirus. Sweet potato feathery mottle virus, sweet potato mild speckling virus, and a crinivirus (serologically related to sweet potato chlorotic stunt virus) were associated with CD. The synergistic combination of these three viruses reproduced the disease.

Additional keywords: *Bemisia tabaci*, *Myzus persicae*, sweet potato sunken vein virus, synergistic virus interactions

There are approximately fifteen viruses known to affect sweet potatoes (*Ipomoea batatas* (L.) Lam.) (3,5,27). Sweet potato feathery mottle potyvirus (SPFMV) (26) is the most common virus found wherever sweet potatoes are cultivated, causing variable symptoms and damage to crops (5,23,25, 27). SPFMV in synergistic combination with a whitefly-transmitted crinivirus (identified as sweet potato sunken vein virus [SPSVV] in Israel [6,17] and sweet potato chlorotic stunt crinivirus [SPCSV] in Nigeria [36]) is known to cause sweet potato virus disease (SPVD) in Africa (14–16,33, 34,37,39). SPCSV has been recommended as the preferred name (40) for the crinivirus found in SPVD. SPSVV reported from Uganda (20) apparently is a different strain of SPCSV.

A disease known as *batata crespa* (curly sweet potato) in Argentina, caused by sweet potato vein mosaic potyvirus (SPVMV) (28), had devastated cv. Criolla Amarilla by 1970. Yield reductions of 84% in the cultivar were reported (29). Later, SPFMV again was detected in affected plants of the same cultivar (30). Because of *batata crespa*, a new cultivar (Morada-INTA, tolerant to both SPFMV and SPVMV) was adopted by Argentinean farmers in 1978. The cultivar is now grown on more than 90% of the area planted to sweet potato in Argentina. Since 1984, Morada-INTA has been affected by a severe disease, termed chlorotic dwarf (CD) based on symptoms in infected plants (9). CD-affected plants are stunted, and their leaves show severe mosaic, blisters, distortion, and reduced leaf area. Symptoms become more severe as the number of vegetative propagations after infection increases (2). CD disease causes yield reductions of up to 80% (8). Previous serological (enzyme-linked immunosorbent assay [ELISA]) work demonstrated that SPFMV was always present in affected plants. However, immunosorbent electron microscopy and decoration (ISEM-D) with SPFMV antiserum indicated that other viruses (undecorated flexuous particles) also were present. Later, two viruses serologically related to sweet potato mild speckling virus (SPMSV) and SPCSV were isolated from CD-affected plants. SPMSV has been reported as a new potyvirus based on molecular characterization of its coat protein (1) and is serologically unrelated to five other sweet potato viruses: SPFMV, sweet potato latent (SwPLV), sweet potato mild mottle (SPMMV), sweet potato chlorotic flecks (SPCFV), and C-6 viruses (3,10,12).

In this paper we report on how the viruses involved in CD disease of sweet potato were isolated and identified and demonstrate that a synergistic combination of three viruses (SPFMV, SPMSV, and SPCSV) reproduced the disease.

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MATERIALS AND METHODS

Source of inoculum. Stem cuttings of cv. Morada-INTA growing in a farmer's field in Cordoba and showing severe CD symptoms (stunting, chlorosis, severe mosaic, and leaf deformation [Fig. 1]) were collected, rooted, and maintained in a growth chamber. These plants were the source of inoculum for the study.

Transmission tests. Sap from test plants was homogenized and diluted in 0.05 M potassium phosphate buffer, pH 7.2, containing 0.2% sodium sulfite and rubbed on leaves of *Nicotiana benthamiana* Domin., *N. clevelandii* Grey, and *Ipomoea setosa* Ker.

Apterous aphids from a healthy colony of *Myzus persicae* Sulz. were reared on *Raphanus sativus* L. and used for transmission studies. A group of fasted aphids was allowed to probe briefly (one probe) on plants of sweet potato cv. Morada-INTA showing typical CD symptoms (21). One aphid was transferred to each of 50 healthy *I. setosa* seedlings and allowed to feed for 12 h before being killed with an aphicide (Pirinicab); the process was repeated once. *I. setosa* plants from both experiments were tested by nitrocellulose membrane-ELISA (NCM-ELISA) and ISEM-D against SPFMV and SPMSV antisera, respectively (discussed below). In another experiment, the efficiency of aphids in transmitting SPFMV and SPMSV from infected to healthy *I. setosa* was determined. Fasted aphids were given a 5-min virus acquisition access period on infected plants. Two aphids were transferred to each of 50 healthy plants (two repetitions), allowed to feed for 12 h, and killed.

Bemisia tabaci Gennadius reared on poinsettia (*Euphorbia pulcherrima* Willd.) plants was used for whitefly transmissions. Approximately 100 *B. tabaci* adults were allowed to feed on a CD-affected plant for 48 h. After the acquisition period, the aphids were transferred to healthy *I. setosa* and *N. benthamiana* and allowed to feed for 48 h (13).

The viruses isolated through aphid and whitefly transmission were serologically tested for other flexuous sweet potato viruses (SwPLV, SPMMV, SPCFV, and C-6).

Virus purification. At the beginning of the research, viruses from CD-affected plants (mixture of SPFMV, SPMSV, and SPCSV) were purified by the method reported by Cohen et al. (7), with some modifications. Leaves of cv. Morada-INTA were homogenized (1:2, wt/vol) with 0.5 M borate buffer, pH 8.0, containing 0.01 M EDTA and 0.05% thioglycolic acid. After mixing the homogenate with 1 volume of carbon tetrachloride, 1 volume of chloroform was added dropwise, and the mixture was clarified by centrifugation at 10,000 × *g* for 10 min. The virus was sedimented from the aqueous phase by centrifugation at 90,000 × *g* for 2 h through a 25% sucrose cushion in 0.05 M borate buffer, pH 8.0, containing 0.001 M EDTA. Pellets were resuspended in the same buffer and sedimented through sucrose-CsCl step gradients (0 to 41% CsCl in borate buffer containing 20% sucrose) at 100,000 × *g* for 5 h at 8°C. The virus band was collected

and dialyzed in 0.05 M borate buffer and further purified through a second CsCl gradient. After SPFMV and SPMSV were isolated, purification of the viruses was performed with leaves of *I. setosa* infected with each virus, following essentially the same procedure.

Antisera production and serological tests. Purified virus preparations of CD (a mixture of three viruses) as well as SPFMV and SPMSV (in Freunds' complete adjuvant) were injected into New Zealand rabbits by multiple intradermal injections and intramuscularly 10 and 30 days later in Freunds' incomplete adjuvant. Antisera were used in NCM-ELISA (22,31) and double-antibody sandwich-ELISA (DAS-ELISA), essentially as described by Clark and Adams (4).

A SPFMV monoclonal antiserum (MAb) prepared against isolate SPV-I from the SPVD complex from Nigeria was provided by G. Thotappilly (IITA, Lagos, Nigeria). Polyclonal antiserum for SPCSV was a gift from J. Cohen (Volcani Center, Bet-Dagan, Israel). MAb mixes 1 and 2, which recognize SPCSV from Eastern Africa and Nigeria (as well as from other areas), respectively, was provided by J. Vetten (Federal Biological Research Center, Institute of Biochemistry, Agriculture, and Forestry, Braunschweig, Germany) (38). MAb PTY1, which recognizes cryptotopes in aphid-transmitted potyviruses (19), was obtained from a commercial supplier (Agdia Elkhart, IN).

For ISEM-D, the protocol of Milne and Lesemann (24) was used. For sensitizing grids, antisera was diluted 1:50 in TBS (20 mM Tris base, 500 mM NaCl, pH 7.5) and incubated on the grids for 30 min. Samples from mesophyll tissue adjacent to veins were macerated in TBS containing 0.2% sodium sulfite (1:10, wt/vol) and centrifuged for 5 min at 2,240 × *g*. For decoration of virus particles, antisera was diluted 1:10 and incubated for 15 min on the grids.

Inoculations with three viruses in different combinations. This experiment was designed to determine the virus combination(s) that would reproduce CD in sweet potato. Scions of *I. setosa* plants infected individually with each of the three isolated viruses (SPFMV, SPMSV, and SPCSV) were double or triple side-grafted onto healthy plants of cv. Morada-INTA and *I. setosa*. Healthy plants of cv. Morada-INTA grafted with scions from CD-affected plants were used as controls.

All grafted plants were kept in a growth chamber with 16 h of illumination (12,000 lux) at 26°C and 8 h of darkness at 20°C and 80% relative humidity.

DAS-ELISA was used to confirm infection of grafted *I. setosa* and cv. Morada-INTA with SPFMV and SPMSV. Infection with SPCSV was confirmed by ISEM-D with a polyclonal SPCSV antiserum.

Fig. 1. Chlorotic dwarf-affected sweet potato cv. Morada-INTA showing mild (right) and severe (center) symptoms. Healthy control at left.

Table 1. Detection[a] of three viruses in different plants by nitrocellulose membrane enzyme-linked immunosorbent assay with monoclonal antibodies (MAb) for sweet potato chlorotic stunt virus (SPCSV) and polyclonal antibodies (PAb) for sweet potato feathery mottle virus (SPFMV) and sweet potato mild speckling virus (SPMSV)

	Antiserum		
Plant[b]	SPCSV MAb mix 2	SPFMV PAb	SPMSV PAb
CD-affected sweet potato cv. Morada-INTA	+	+	+
Ipomoea setosa with SPFMV	–	+	–
Nicotiana benthamiana grafted with CD	+	–	+
SPCSV from Israel	+	–	–
SPVD from Nigeria	+	+	–
I. setosa with SPFMV from CD	–	+	–
I. setosa with SPMSV from CD	–	–	+
I. setosa with SPCSV from CD	+	–	–
Healthy cv. Morada-INTA	–	–	–
Healthy *I. setosa*	–	–	–

[a] Positive (+) and negative (–) detection determined by visual assessment.
[b] CD = chlorotic dwarf; SPVD = sweet potato virus disease.

Table 2. Detection of viruses in infected sweet potato plants by double-antibody sandwich enzyme-linked immunosorbent assay with antisera to partially purified viruses from plants with chlorotic dwarf (CD) disease or infected with sweet potato mild speckling virus (SPMSV) or sweet potato feathery mottle virus (SPFMV)

	Antiserum		
Plant disease	SPMSV	SPFMV	CD
SPFMV	– (0.004)[a]	+ (0.516)	+ (1.714)
SPMSV	+ (0.303)	– (0.005)	+ (0.361)
CD	+ (1.731)	+ (1.597)	+ (2.062)
SPCSV + SPMSV	+ (1.473)	– (0.004)	+ (0.318)
SPCSV + SPFMV	– (0.006)	+ (0.919)	+ (1.938)
SPCSV + SPMSV+ SPFMV	+ (2.040)	+ (0.837)	+ (1.999)
Critical point[b]	0.050	0.020	0.030

[a] A_{405nm} values 90 min after initiation of reaction. – = negative reaction; + = positive reaction.
[b] A_{405nm} values of negative control plus three times the standard deviation.

RESULTS

Isolation of viruses. Two potyviruses were transmitted to healthy *I. setosa* plants by aphids from CD-affected plants. The viruses were SPFMV and SPMSV, and the frequency of isolation from single aphids (*M. persicae*) was 32 and 26%, respectively. Twenty-eight percent of *I. setosa* plants were infected with both viruses; fourteen percent were not infected with either virus (no symptoms and no serological reaction with any antisera). SPMSV also was isolated, independent of SPFMV, in some sap-inoculated *N. benthamiana* and *N. clevelandii* plants.

Both viruses were maintained in aphid- or sap-inoculated *I. setosa* in insect-proof

cages in the greenhouse. The efficiency of two aphids per plant in transmitting SPFMV and SPMSV from infected to healthy *I. setosa* was 70%.

Mild mosaic developed in both *I. setosa* and *N. benthamiana* plants 15 days after transmission of the crinivirus by whiteflies. The presence of this virus and its relationship to SPCSV was shown by ISEM-D.

Virus purification and serology. Viruses were purified in a mixture (SPFMV, SPMSV, and SPCSV) from CD-affected sweet potato plants or separately (SPFMV and SPMSV) from *I. setosa* infected with each virus alone. Antisera produced in rabbits reached a titer of 1:300,000 for SPMSV, 1:1,000,000 for SPFMV, and 1:500,000 for CD (the three viruses together) in NCM-ELISA. Viruses detected from different diseased plants are shown in Table 1. CD antiserum reacted with the three viruses isolated from CD-affected plants: strongly with SPFMV, intermediately with SPMSV (Table 2), and weakly with SPCSV (data not shown). When CD antiserum was used to detect SPCSV, it was detected by ISEM-D and NCM-ELISA (after cross-absorption of antiserum with healthy plant proteins).

Isolated SPFMV did not react to the MAb SPFMV SPV-I. SPFMV, but not SPMSV, reacted with MAb PTY1. The isolated crinivirus reacted in NCM-ELISA with SPCSV polyclonal antiserum and MAb mix 2.

Inoculation with SPFMV and SPMSV. Young plants of cv. Morada-INTA were grafted with *I. setosa* scions infected with SPFMV, SPMSV, or both. Plants grafted with SPFMV developed chlorotic mosaic in basal leaves after 20 days (Table 3). In plants grafted with SPMSV, only transient mild speckling was observed in basal leaves. Grafting with the two viruses did not reproduce CD symptoms, only chlorotic rings, mosaic, and light blistering in basal leaves 20 days after inoculation.

Inoculation with three viruses in different combinations. Symptoms caused by each virus or combination of viruses are shown in Table 3 and Figure 2. Combination of SPCSV with SPFMV, SPMSV, or both caused different degrees of CD severity. The combination of the three viruses induced in *I. batatas* the most severe symptomatology resembling that of CD-affected plants from the field: severe mosaic, leaf deformation and reduction, and stunting of infected plants.

Table 3. Symptoms induced by three viruses (sweet potato feathery mottle [SPFMV], sweet potato mild speckling [SPMSV], and sweet potato chlorotic stunt [SPCSV]), alone or in combination, in graft-inoculated *Ipomoea setosa* and sweet potato (*I. batatas*) cv. Morada-INTA

Host	Virus	Symptoms
I. setosa	SPFMV	Mild mosaic of first two true leaves, vein clearing; severe mosaic after fourth leaf, chlorotic spotting; very mild or no symptoms after eighth leaf
	SPMSV	Mild mosaic in first two true leaves
	SPFMV + SPMSV	Same as SPMSV alone
	SPCSV	Mild mosaic
	SPCSV + SPFMV	Mosaic, chlorosis, and leaf deformation
	SPCSV + SPMSV	Mosaic, chlorosis, severe leaf distortion ("shoe-lace"), top necrosis, and death of plants
	SPCSV + SPFMV + SPMSV	Vein clearing, mosaic, leaf deformation ("shoe-lace"), stunting, necrosis, and death of plants
I. batatas	SPFMV	Chlorotic spots only in lower leaves
	SPMSV	Occasional chlorotic speckling in some leaves
	SPFMV + SPMSV	Chlorotic rings, mosaic, and mild blistering of lower leaves
	SPCSV	Very mild mosaic in old and new leaves
	SPCSV + SPFMV	Chlorotic leaves, mild mosaic, reduction of leaf area, and leaf border curved downward
	SPCSV + SPMSV	Severe mosaic of all leaves and vein clearing
	SPCSV + SPFMV + SPMSV	Severe mosaic, leaf deformation, vein clearing, and leaf border curved downward; stunting

DISCUSSION

Because the sweet potato is a vegetatively propagated crop, accumulation and perpetuation of virus diseases is likely to become a major constraint for production (5–7,14,16, 27,32,35,39). This is especially evident in regions such as Argentina where vector activity is high and sources of virus infections, such as voluntary sweet potato plants and weeds, exist. Previous work has shown

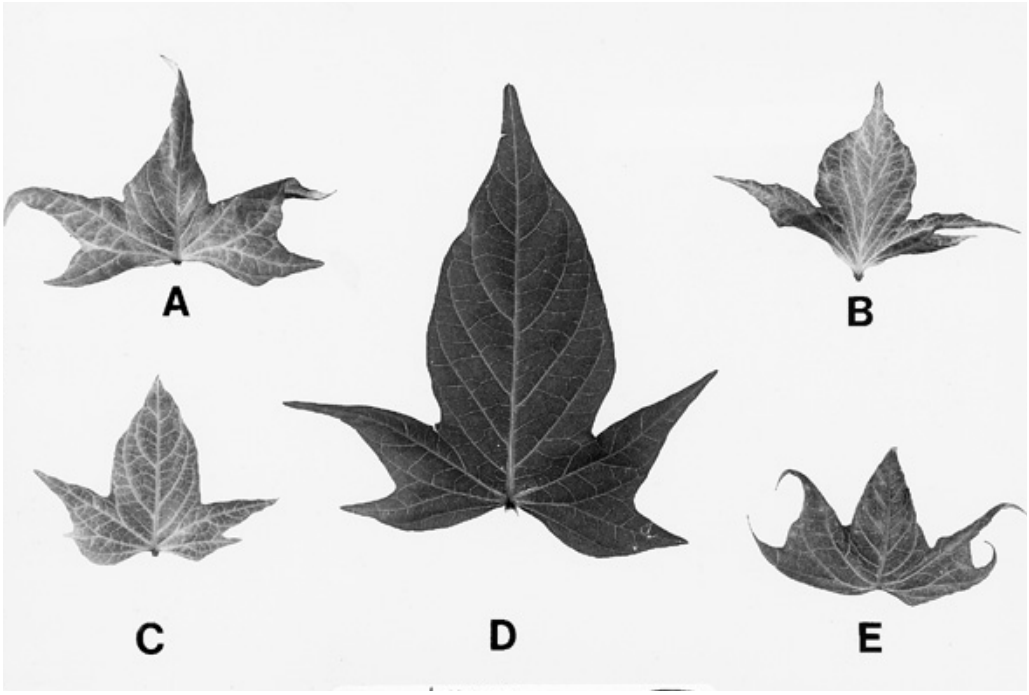

Fig. 2. Leaves of sweet potato cv. Morada-INTA infected with the viruses involved in chlorotic dwarf (CD) disease in different combinations. **(A)** Sweet potato chlorotic stunt virus (SPCSV) + sweet potato mild speckling virus (SPMSV) + sweet potato feathery mottle virus (SPFMV); **(B)** field-infected sample showing CD disease; **(C)** SPCSV + SPMSV; **(D)** healthy control; and **(E)** SPCSV + SPFMV.

that CD is the most important disease (8) in the sweet potato-producing regions of Argentina. Even though the disease resembles SPVD from Africa, our results indicate that CD disease is caused by the synergistic effect of three viruses, not two as was reported in Africa (27,33,34,36,39). Although the combinations of SPCSV with both SPFMV and SPMSV in dual combinations showed some level of synergistic interaction, symptoms were clearly more severe with a combination of all three viruses than with dual combinations. Because SPFMV hindered isolation of other viruses, we had thought that CD was caused by an unusual strain of SPFMV. However, we demonstrated that the combination of the three viruses and the different combinations of two of the three viruses account for the variability of CD symptomatology observed in the field. The most severe expression of CD was obtained when SPFMV, SPMSV, and SPCSV infected sweet potato plants simultaneously.

This situation may occur in other regions of the world as well, because all three viruses have been recorded outside Argentina. For instance, SPFMV is found wherever sweet potatoes are grown; SPCSV and its strains are present in several countries (17, 38); and SPMSV is present in Argentina, Peru. and Indonesia (S. Fuentes, *unpublished data*).

We found that the SPFMV strain from CD-affected plants is different from that reported in Nigeria (SPV-I) (33,34), because it does not infect *N. benthamiana* and is serologically unrelated to SPV-I when a MAb is used. However, a SPFMV strain serologically related to SPV-I has been found in CD-affected plants from other areas of Argentina (data not shown), indicating it is not necessarily a strain-specific combination, at least with SPFMV, that causes CD disease.

The SPCSV strain from CD seems to be different than the strain reported in Nigeria, because the CD-SPCSV caused mild mosaic in *I. setosa* (and also in sweet potato) and not chlorotic stunt, as was reported for the Nigerian isolate.

The CD disease syndrome (Fig. 1) resembles *batata crespa* (general chlorosis, vein clearing, mosaic, twisting of leaves, stunting, and reduction in size and number of roots) reported only in Argentina by 1970 (5,28). However, *batata crespa* is caused by the infection by a single aphid-transmitted potyvirus (SPVMV). The virus particle size of SPVMV differs from the other two potyviruses present in CD disease: SPVMV (761 nm) is significantly shorter than SPFMV (868 nm) but close in length to SPMSV (790 nm) (data not shown). Because SPVMV and its antiserum are not available for comparison, we do not know its relationship with SPMSV. However, the host range of SPMSV includes species in the Convolvulaceae, Chenopodiaceae, and Solanaceae families (11), while the range for SPVMV is limited to the Convolvulaceae family (28). As a result, SPVMV induced more severe symptoms in *I. setosa* (distortion, chlorosis, and size reduction of leaves) than SPMSV (Table 3).

An unusual result found in this study is that although SPFMV and SPMSV are transmitted with equal efficiency (70%) by *M. persicae*, only SPFMV reacted with MAb PTY1 (19), which recognizes cryptotopes present in potyviruses transmitted by aphids. However, this is not surprising, because some isolates of other potyviruses (papaya ringspot type W and peanut mottle) known to be transmitted by aphids also were not recognized by MAb PTY1 (18).

This work emphasizes the need to control CD by developing large-scale virus-free plant material schemes. Also, it provides the groundwork for development and use of virus-resistant cultivars. In light of our results, resistance to SPCSV would be more appropriate, although extreme resistance to SPFMV also might reduce CD incidence and its deleterious effects on sweet potato.

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