Advances in the etiology of sweet potato (*Ipomoea batatas* (L.) Lam) yellow curling disease in Argentina

Avances en la etiología de la enfermedad del encrespamiento amarillo de la batata (*Ipomoea batatas* (L.) Lam) en Argentina

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Originales: *Recepción:* 17/02/2022 - *Aceptación:* 18/10/2022

Abstract

Sweet potato yellow curling (YC), the most severe disease of sweet potato detected in Argentina, causes symptoms and damage to sweet potato crops in all cultivated regions. Since 2010/11, the presence of four viruses has been detected in symptomatic cv. Arapey INIA: two potyviruses non-persistently transmitted by *Myzus persicae* (sweet potato feathery mottle virus, SPFMV and sweet potato virus G, SPVG)*;* a closterovirus, sweet potato chlorotic stunt virus (SPCSV) and a geminivirus, sweet potato leaf curl virus (SPLCV), both transmitted by *Bemisia tabaci* in a semi-persistent and persistent manner, respectively. All the plants were collected from fields in Colonia Caroya, Córdoba province, Argentina. The objectives of the present work are to isolate and identify the virus or viruses involved in YC disease of sweet potato, and to elucidate the viral combination that reproduces YC symptoms. The most severe YC symptoms for this genotype in the field were only reproduced by a combination of the four viruses. The symptoms include chlorosis, stunting, mosaic, blistering, leaf curling, chlorotic spots, chlorotic patterns, leaf area reduction and distortion, and upward curling of leaf edges. The presence of each virus was detected by serological (DAS, NCM and TAS-ELISA) and molecular (PCR) tests. It is concluded that the interaction of SPFMV, SPVG, SPCSV and SPLCV is needed for the development of YC symptoms.

Keywords

Ipomoea batatas • sweet potato feathery mottle virus • sweet potato virus G • sweet potato leaf curl virus • sweet potato chlorotic stunt virus • Arapey INIA • Koch's postulates

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Resumen

El encrespamiento amarillo (EA), la enfermedad más severa detectada en Argentina, causa síntomas y daños en cultivos de batata en toda la región productora. Desde 2010/11 se ha detectado la presencia de cuatro virus en plantas sintomáticas del cv. Arapey INIA recolectadas en lotes de Colonia Caroya, provincia de Córdoba. Los virus son sweet potato feathery mottle virus (SPFMV) y sweet potato virus G (SPVG), dos potyvirus transmitidos de forma no persistente por *Myzus persicae*; un closterovirus: sweet potato chlorotic stunt virus (SPCSV) y un geminivirus: sweet potato leaf curl virus (SPLCV), ambos transmitidos por *Bemisia tacabi* de manera semipersistente y persistente, respectivamente. Los objetivos de este trabajo fueron aislar e identificar el o los virus involucrados en la enfermedad EA de la batata y determinar la combinación de virus que reproduce la sintomatología de EA Solo la combinación de los cuatro virus permitió reproducir la sintomatología más severa del encrespamiento amarillo observada a campo en dicho genotipo. Los síntomas incluyen clorosis, achaparramiento, mosaico, ampollado, enrulado de la hoja, manchas cloróticas, diseños cloróticos, reducción y distorsión del área foliar, bordes de la hoja curvados hacia arriba. La presencia de cada uno de los virus se detectó mediante pruebas serológicas (DAS, NCM y TAS-ELISA) y moleculares (PCR). Se concluye que la interacción de SPFMV, SPVG, SPCSV y SPLCV es necesaria para el desarrollo de EA.

Palabras claves

Ipomoea batatas • sweet feathery mottle virus • sweet potato virus G • sweet potato leaf curl virus • sweet potato chlorotic stunt virus • Arapey INIA • postulados de Koch

Introduction

Sweet potato, *Ipomoea batatas* (L.) Lam, is a perennial plant of the *Convolvulaceae* family, native to northwestern South America (6, 16). It is ranked among the 10 most important food crops worldwide, with a production of 89,487,835 tons in an area of 7,400,472 hectares in 2020 (8). The major sweet potato production areas are located in China and around the Great Lakes of East Africa, but the crop is also important in many other countries (6, 20). According to the International Potato Center, developing countries concentrate 95% of the production, but with low yields. Latin America produces only 1.5 million tons (8).

Viral diseases are considered a major constraint to sweet potato production worldwide (22, 38, 40), due to the vegetative propagation of this species and the consequent cumulative effect of virus infections (17, 29). The first report of a sweet potato viral disease in Argentina was published in the 1970s. This disease, named "batata crespa", affected cv. Criolla Amarilla and was caused by the potyvirus sweet potato vein mosaic virus (SPVMV) (30). Later, sweet potato feathery mottle virus (SPFMV) was detected in the same cultivar (31). In 1978, Argentine farmers adopted a new cultivar, Morada INTA, tolerant to both potyviruses (SPVMV and SPFMV). However, during the 1980s, this new cultivar was affected by a severe disease called sweet potato chlorotic dwarf, caused by a synergistic combination of two aphid-transmitted potyviruses (SPFMV and sweet potato mild speckling virus, SPMSV) with a whitefly-transmitted closterovirus serologically related to sweet potato chlorotic stunt virus (SPCSV) (7). Sweet potato chlorotic dwarf was successfully controlled using propagation material obtained from disease-free areas. Nevertheless, in all sweet potato-producing areas of Argentina, cv. Morada INTA has been progressively replaced by cv. Arapey INIA, an Uruguayan genotype (7).

In 2010/11, a new viral disease with severe and variable symptoms was detected in this cultivar; it was named sweet potato yellow curling disease (YC). YC causes significant yield losses, with records of up to 90% loss in experimental trials and commercial plots. In addition, YC affects the quality of the storage roots (23).

Serological tests confirmed the presence of SPFMV (4) and SPCSV (Unpublished data) in symptomatic plants. Moreover, in these plants, two pathogens that had not been described in Argentina at that time, sweet potato virus G (SPVG), a potyvirus (33) and the geminivirus, sweet potato leaf curl virus (SPLCV) (34), were detected through serological tests and PCR, respectively.

Since YC is presumed to be caused by a viral complex, this work aimed to isolate and identify the virus or viruses involved in YC disease of sweet potato and to elucidate the viral combination that reproduces YC symptomatology.

Material and methods

Source of inoculum

More than 100 plants of sweet potato cv. Arapey INIA showing chlorosis, stunting, mosaic, chlorotic rings, chlorotic patterns, blistering, distortion, reduction of foliar area, and upward leaf curling were randomly collected from 12 fields in Colonia Caroya (31°01'16.8'' S 64°03'42.2'' W), Colón department, Córdoba province, Argentina. Plants were transplanted to pots and maintained in the greenhouse at 25°C under controlled conditions of humidity (70-75% RH) and photoperiod (16 h light) for further analysis.

Transmission assays

Apterous aphids from a healthy colony of *Myzus persicae* Sulz. were reared on *Raphanus sativus* L. and used for transmission studies. Fasted aphids were allowed to probe briefly (one probe) on plants of sweet potato cv. Arapey INIA showing typical YC symptoms. Aphids were transferred to healthy individuals of the indicator plant *Ipomoea setosa* (one aphid per each one of 44 plants) and allowed to feed for 12 h; then they were killed with an aphicide (Pirimicarb) (7, 13). A repetition was performed using sweet potato cv. Okinawa 100 plants infected with SPVG, SPCSV and SPLCV. The procedure was similar to the one described previously but involved 46 *I. setosa* plants.

To separate SPCSV from SPLCV (semi-persistent and persistent transmission, respectively), non-viruliferous *Bemisia tabaci* Gennadius whiteflies reared on poinsettia (*Euphorbia pulcherrima* Willd.), a sweet potato plant cv. Arapey infected with YC, and 42 healthy seedlings (second unfolded leaf) of *I. setosa* were placed in protected cages with anti-aphid mesh, at 25°C, in a greenhouse, under controlled conditions of humidity (70-75% RH) and photoperiod (16h light). The whiteflies were removed from the cages after 1h and up to 48h of exposure to insect feeding. At 21 days after inoculation, the plants were checked for the presence of SPCSV and SPLCV by TAS-ELISA and PCR, respectively (25).

Verification of SPFMV, SPVG, SPCSV and SPLCV presence

Infection by SPFMV, SPVG and SPCSV was checked by serological tests on all inoculated plants. A local antiserum was used for SPFMV detection. The SPVGas and SPCSVas were provided by S. Fuentes (International Potato Center, Lima, Peru) and H. J. Vetten (Federal Biological Research Center, Institute of Biochemistry, Agriculture, and Forestry, Braunschweig, Germany), respectively (7, 9). SPLCV was tested by PCR (34). The production of local SPVGas is discussed below.

SPVG virus purification and antiserum production

After SPVG isolation, virus particles were purified from *I. setosa*-infected leaves following the method described by Di Feo *et al.* (2000). The virus band was collected from a sucrose-CsCl step gradient (0 to 41% CsCl in borate buffer containing 20% sucrose) after ultracentrifugation (100000 x g for 5 h at 8°C), and dialyzed against 0.05 M borate buffer. Purified virus preparation of SPVG (0.02 mg/ml) was injected into a female New Zealand rabbit. Three inoculations were performed at 20-day intervals. The first inoculation (1ml of the purified virus + 2 ml of Freund's complete adjuvant) was administered by multiple intradermal injections, whereas the other two inoculations (1 ml of the purified virus + 1 ml of incomplete Freund's adjuvant) were administered intramuscularly. Blood sample collection started 20 days after the last injection. The titers were evaluated by Nitrocellulose membrane enzyme-linked immunosorbent assay (NCM-ELISA) (21, 32), Double-antibody sandwich enzyme-linked immunosorbent assay (DAS-ELISA) (5) and Immunosorbent electron microscopy plus decoration (ISEM+D) (26).

To evaluate the optimal dilution to be used in NCM-ELISA, SPVG antiserum was serially diluted from 1:500 to 1:1024000 in Tris saline buffer + 2% skimmed milk. Samples were

macerated in extraction buffer (TBS + 0.2% Na_2SO_3) in a 1/50 (w/v) proportion and blotted onto a nitrocellulose membrane. Positive controls were *I. setosa* plants infected with SPVG, sweet potato cv. Arapey INIA infected with YC and sweet potato cv. Okinawa infected with the complex (SPVG + SPCSV + SPLCV). Negative controls were healthy *I. setosa* and sweet potato plants. In addition, a sample of *I. setosa* infected with SPFMV was included to rule out the possible cross-reaction between the antiserum and this potyvirus.

For the DAS-ELISA test, IgG was purified from the SPVG antiserum. Enzyme conjugate was prepared with the purified IgG and Phosphatase Alkaline VII S (SIGMA -Aldrich) (10 mg protein/ml suspension of $SO_4(NH)_4$; 30000 enzyme units/ml). DAS-ELISA test was performed using a NUNC polystyrene plate with 96 flat-bottomed wells as support. Three dilutions of IgG (1:500, 1:1000 and 1:2000) were tested against three dilutions of enzyme conjugate (1:500. 1:1000 and 1:1500). Samples were macerated in extraction buffer (PBS + 0.05% Tween 20 + 2% PVP+ 2% skimmed milk + 0.2% SO_2Na_2) in a 1/10 and a 1/1000 proportion, respectively. Positive and negative controls were sweet potato plants infected with SPVG and healthy plants, respectively. Absorbance was measured using a spectrophotometer (Dynatech Laboratories, Model MRX) at 405 nm.

ISEM+D was performed according to the protocol described by Milne & Lesemann (1978). Copper grids were first covered with the SPVG-specific antiserum, diluted 1:1000 in TBS (20 mM Tris base, 500 mM NaCl, pH 7.5) and incubated for 30 min. Samples co-infected with SPVG + SPCSV were macerated in TBS containing 0.2% sodium sulfite (1/10 w/v). For decoration of virions, antiserum was diluted at 1:50 and incubated on grids for 15 min. Moreover, the modal length of virions obtained from purified preparations was determined. Observations were made under an electron microscope (JEOL JEM EX II 1220®).

Inoculation of healthy sweet potato plants with four viruses in different combinations

This experiment was designed to determine the viral combination(s) that reproduces YC in sweet potato. Scions of *I. setosa* plants infected individually with each of the four isolated viruses (SPFMV, SPVG, SPCSV and SPLCV) were double, triple and quadruple side-grafted onto 10 healthy plants of cv. Arapey INIA (obtained by "*in vitro*" meristem culture) and *I. setosa*, respectively. Six healthy plants of cv. Arapey INIA grafted with scions from YC-affected plants were used as controls.

Grafted plants were kept in protected cages with anti-aphid mesh in a greenhouse at 25°C under controlled conditions of humidity and photoperiod.

The presence of SPFMV was determined by NCM-ELISA using local antiserum (7). To detect SPCSV, a triple-antibody sandwich enzyme-linked immunosorbent assay (TAS-ELISA) was performed, as described by Gibson *et al.* (1998), using antisera provided by H. J. Vetten (Federal Biological Research Center, Institute of Biochemistry, Agriculture, and Forestry, Braunschweig, Germany). Infection with SPVG was confirmed by NCM-ELISA using the local antisera produced in this study. The presence of SPLCV was verified by PCR using the primers SPG1 and SPG2 designed to bind to conserved regions in open reading frames C2 and C1, and to amplify a 912-bp fragment (18).

Results

Transmission assay

Only SPFMV was transmitted to healthy *I. setosa* plants from sweet potato cv. Arapey INIA infected with the YC complex. However, the frequency of isolation using single aphids (*M. persicae*) was 77% for SPFMV and 13% for SPVG when the inoculum source was sweet potato cv. Okinawa 100 infected with SPVG, SPCSV and SPLCV. Virus sources were maintained by grafting onto *I. setosa* and sweet potato cv. Arapey INIA in insect-proof cages in the greenhouse.

Transmission of SPCSV and SPLCV from sweet potato plants infected with YC showed that one hour after the *I. setosa* seedlings were removed from the cage, 100% of the plants were infected with SPCSV and 0% with SPLCV. On the other hand, at 48 h, 100% of the plants were infected with SPLCV and 33% with SPCSV. Those plants negative for closterovirus were selected for further testing.

Virus purification and serology

SPVG was purified from single-infected *I. setosa* plants. The A_{260}/A_{280} ratio and viral concentration of the band extracted from the CsCl gradient were 1.16 and 0.161 mg/ml, respectively. According to the NCM-ELISA test, the optimal dilution of the antiserum was 1:512000, without cross-reaction with SPFMV.

In DAS-ELISA, optimal values of A_{405} were obtained after 90 minutes of reaction. The most suitable dilutions of IgG and enzyme conjugate for virus diagnosis were 1:1000 and 1:500, respectively (table 1), whereas 1/10 w/v was the most appropriate dilution of sweet potato plant tissue.

Table 1. Absorbance values (A405) in DAS-ELISA using SPVGas.

Tabla 1. Valores de absorbancia (A405) en DAS-ELISA utilizando SPVGas.

	IgG dilution (v/v)								
	1/500			1/1000			1/2000		
	IgG-AP conjugate dilution (v/v)								
	1/500	1/1000	1/1500	1/500	1/1000	1/1500	1/500	1/1000	1/1500
I. setosa infected with SPVG	1.038	0.549	0.363	0.967	0.504	0.285	0.667	0.374	0.205
Healthy plants	0.071	0.021	0.019	0.087	0.008	0.023	0.046	0.028	-0.012

When the SPVG antiserum was used for ISEM + Decoration tests, differentially decorated virions were observed in samples of tissue infected with SPVG +SPCSV (figure 1).

Observations were made under a transmission electron microscope (JEOL JEM EX II 1220®) (X150000).

Observaciones realizadas al Microscopio electrónico de transmisión (JEOL JEM EX II 1220®) (X150000).

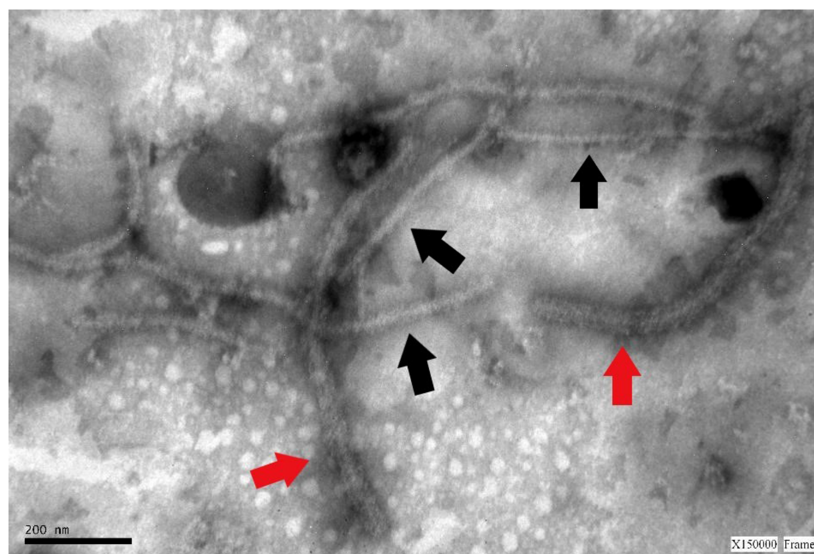


Figure 1. Virus particles from tissue samples co-infected with SPVG + SPCSV. ISEM+D coated 1/2000 with SPVG antiserum, decoration 1/50 (v/v), and contrasted with 2% uranyl acetate (red arrow).

Figura 1. Viriones de SPVG de muestras de tejido coinfectado con SPCSV, ISEM + D sensibilizado con antisuero SPVG diluido 1/2000, decorado con antisuero diluido 1/50 (v/v), y contrastado negativamente con acetato de uranilo al 2% (flecha roja).

A modal length of 850-900 nm (150 virions) from purified suspensions was determined (figure 2, page XXX), which corresponds to the range of length established for potyviruses (37).

The modal length (850-900 nm) corresponds to the range of length established for potyviruses.

La longitud modal (850-900 nm) corresponde al rango de longitud establecido para los potyvirus.

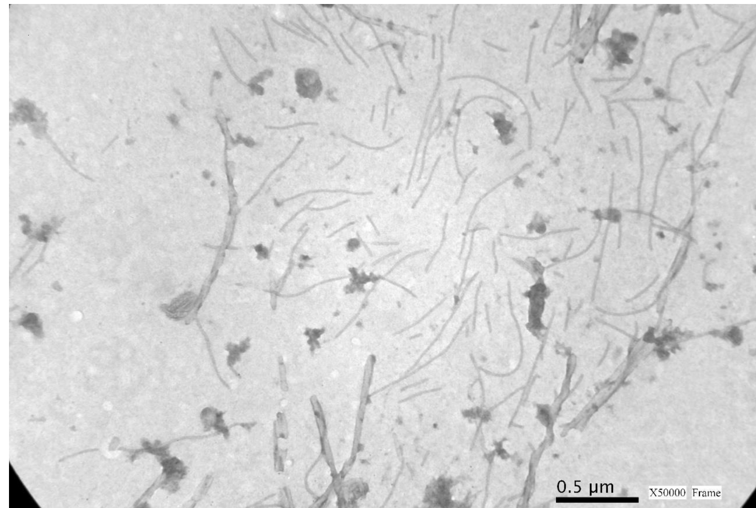


Figure 2. SPVG viral particles from purified suspensions, observed under a transmission electron microscope JEOL JEM EX II 1220® (X50000).

Figura 2. Partículas virales SPVG observadas al microscopio electrónico de transmisión JEOL JEM EX II 1220® a partir de suspensiones purificadas (X50000).

Inoculation

Symptoms caused by each virus combination are shown in table 2. The mixed infections in which SPCSV was present caused different degrees of severity.

Table 2. Symptoms induced by different combinations of sweet potato feathery mottle virus (SPFMV), sweet potato virus G (SPVG), sweet potato chlorotic stunt virus (SPCSV) and sweet potato leaf curl virus (SPLCV) in graft-inoculated *Ipomoea setosa* and sweet potato (*Ipomoea batatas*) cv. Arapey INIA.

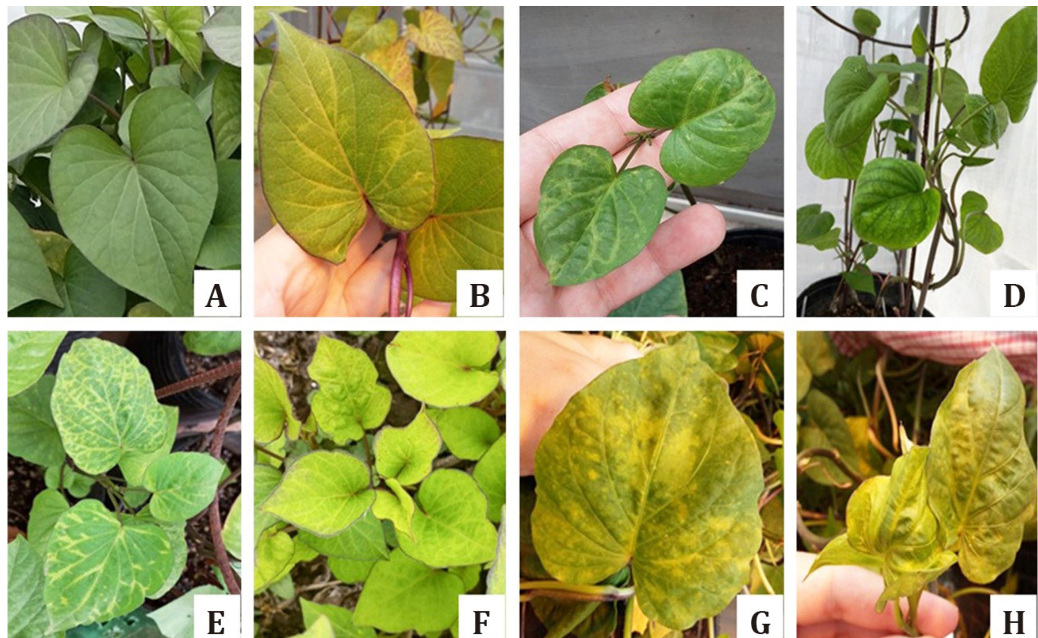
Tabla 2. Síntomas inducidos por diferentes combinaciones de sweet potato feathery mottle virus (SPFMV), sweet potato virus G (SPVG), sweet potato chlorotic stunt virus (SPCSV) y sweet potato leaf curl virus (SPLCV) en plantas de *Ipomoea setosa* y batata (*Ipomoea batatas*) cv. Arapey INIA inoculadas mediante injerto.

Host	Virus	Symptoms
I. setosa	SPVG +SPFMV	Mosaic, feathery mottling, vein clearing, chlorotic spots and leaf distortion
	SPFMV + SPCSV	Mosaic, feathery mottling, chlorotic spots, blistering and severe leaf distortion (“shoelace”)
	SPFMV + SPLCV	Feathery mottling in lower leaves and upward curling of margins of upper leaves
	SPVG + SPLCV	Mosaic and vein clearing in upper and lower leaves and upward curling of margins of upper leaves
	SPVG + SPCSV	Severe mosaic, vein clearing, and severe leaf distortion (“shoelace”) in upper leaves.
	SPCSV + SPLCV	Mosaic, blistering, leaf distortion with upward curling of margins in upper and lower leaves
	SPVG + SPFMV + SPCSV	Mosaic, feathery mottling, vein clearing and severe leaf distortion (“shoelace”) in upper and lower leaves
	SPVG + SPFMV + SPLCV	Feathery mottling and mosaic in lower and upper leaves and curling of margins in upper leaves
	SPVG + SPCSV + SPLCV	Mosaic, vein clearing and vein banding and shoelace in upper leaves
	SPFMV + SPCSV + SPLCV	Mosaic, vein clearing and feathery mottling and shoelace in upper leaves
	SPFMV + SPVG + SPCSV + SPLCV	Mosaic, vein clearing, feathery mottling, blistering and severe leaf distortion (“shoelace”)

Host	Virus	Symptoms
I. batatas	SPVG +SPFMV	Mild mosaic, chlorotic spots, vein clearing in upper leaves
	SPFMV + SPCSV	Severe leaf distortion ("shoelace"), mosaic and vein clearing
	SPFMV + SPLCV	Mosaic and feathery mottling, and upward curling of margins of upper leaves
	SPVG + SPLCV	Mild mosaic and vein clearing, upward curling of margins of upper leaves
	SPVG + SPCSV	Vein clearing, chlorosis, blistering and vein banding. Severe leaf distortion ("shoelace") in upper leaves
	SPCSV + SPLCV	Mild mosaic and upward leaf margins
	SPVG + SPFMV + SPCSV	Chlorotic spots in upper and lower leaves. Vein clearing and feathery mottling, mosaic
	SPVG + SPFMV + SPLCV	Mosaic, vein clearing and thickening, blistering, upward leaf margins
	SPVG + SPCSV + SPLCV	Vein clearing, chlorosis, mosaic, blistering and vein banding. Severe leaf distortion ("shoelace") in upper leaves
	SPFMV + SPCSV + SPLCV	Severe leaf distortion ("shoelace"), mosaic and vein clearing, upward curling of margins of upper leaves
	SPFMV + SPVG + SPCSV + SPLCV	Chlorosis, stunting, mosaic, blistering, leaf curling, chlorotic spotting, chlorotic patterns, reduction and distortion of leaf area ("shoelace"), upward curling of leaf margins

The combination of the four viruses induced the most severe symptomatology, resembling that of YC-affected plants in the field (figure 3): chlorosis, stunting, mosaic, blistering, leaf curling, chlorotic spotting, chlorotic patterns, reduction and distortion of leaf area, and upward curling of leaf margins. Young plants of cv. Arapey INIA developed this symptomatology 15 days after grafting with the combination of the four viruses.

On the other hand, *I. setosa* became symptomatic 10 days after inoculation. Symptoms varied from mosaic, vein clearing, feathery mottling and blistering to severe leaf distortion ("shoelace").



(A) Asymptomatic leaf (healthy plant); (B) SPFMV + SPVG, feathery mottle and chlorotic spots; (C) SPFMV + SPVG + SPCSV, interveinal chlorosis, chlorotic spots and upward curling of leaf margins; (E-H) SPFMV + SPVG + SPCSV + SPLCV, feathery mottle, mosaic, vein clearing, chlorotic spots, blistering, upward curling of leaf margins, leaf area reduction and distortion.

(A) Hoja asintomática (planta sana); (B) SPFMV + SPVG, moteado plumoso y punteado clorótico; (C) SPFMV + SPVG + SPCSV, clorosis internerval, punteado clorótico y curvado de hojas; (E-H) SPFMV + SPVG + SPCSV + SPLCV, moteado plumoso, mosaico, aclaramiento de nervaduras, punteado clorótico, ampollado, curvado de hojas, reducción y distorsión del área foliar.

Figure 3. Symptoms induced in cv. Arapey INIA by different combinations of the viruses involved in sweet potato yellow curling disease.

Figura 3. Síntomas observados en diferentes combinaciones de los virus involucrados en encrespamiento amarillo cv. Arapey INIA.

Discussion

Vegetative propagation is a cultural practice that facilitates efficient virus perpetuation and dissemination way between cropping seasons or growing areas (14). While several viral diseases have been reported in sweet potato, YC is the most severe one reported in Argentina so far. It is caused by at least four viruses and produces severe symptoms and significant damage in all growing areas in the country (23).

The new disease differs from the ones previously described in Argentina. Both “batata crespa” and “sweet potato chlorotic dwarf disease” affected only sweet potato plots from Santiago del Estero and Córdoba provinces. The former was caused by SPVMV, and the latter, by three viruses: SPCSV + SPFMV + SPMSV (7, 30). Two viruses already present in Argentina (SPFMV and SPCSV) and two new viruses (SPVG and SPLCV) are involved in YC.

Mixed viral infections and synergistic complexes are frequent in sweet potato (36). SPFMV is the most common virus infecting sweet potato and occurs in all sweet potato growing regions (27). SPCSV interacts synergistically with SPFMV to cause sweet potato virus disease (SPVD), the most serious disease of sweet potato (1, 12, 19, 29). The ubiquitous presence of SPFMV has often masked the presence of other viruses in sweet potato, especially those belonging to the same family, such as SPVG; thus, detecting or isolating them is very difficult (35). It is widely known that suitable management of viral diseases that affect the sweet potato crop requires rapid and accurate detection (6). However, in previous works, it has been shown that low titers of the viral agents and the high concentration of inhibitors in the sweet potato plant hinder serological and molecular diagnosis (11, 14). The antiserum obtained allowed us to detect SPVG in single and mixed infections. In the latter case, the detection was evident, without cross-reaction with SPFMV, a ubiquitous virus in sweet potato crops (39). Detection reagents are available for local species involved in YC, such as SPVGas, which is used for routine diagnosis in IPAVE.

On the other hand, geminiviruses that infect sweet potato are widely distributed throughout the world. Twelve viruses belonging to the *Geminiviridae* family have been reported (6, 15, 28). They can cause significant losses in the production and quality of certain sweet potato genotypes, without noticeable symptom expression (41). SPLCV has been detected in several locations worldwide, including Taiwan, Japan, Israel, and the United States. The geographical range of this virus, however, is still mostly unknown (14). Leaf curl disease associated with SPLCV was first reported in Argentina in 2012 (34). Sweet potato leaf curl Georgia virus (SPLCGV) was also reported in Argentine sweet potatoes (24). The presence of both pathogens is associated with an increase in whitefly populations (3). Therefore, it is necessary to study the dispersion of the new viral species, since climate change causes the geographical expansion of vectors such as whiteflies (2, 3, 10).

The global exchange of sweet potato germplasm contributes to the wide distribution of the viruses in the sweet potato production regions (19). One effective way to prevent the spread of viruses and, therefore, control viral diseases is to use virus-tested planting material.

The new disease, called yellow curling, is a serious threat to sweet potato crops in Argentina. We are conducting studies to demonstrate its detrimental effects on the production and quality of roots.

Conclusion

In this work, the first antiserum for the fast, safe and efficient diagnosis of SPVG in the country was obtained.

The four viruses involved in YC disease were isolated and identified: SPFMV, SPVG, SPCSV and SPLCV.

The interaction of SPFMV, SPVG, SPCSV and SPLCV is needed for the development of yellow curling symptoms in sweet potato in Argentina.

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Acknowledgements

Daniela Martinelli contributed to SPVG biological characterization. Consejo Nacional de Investigaciones Científicas y Técnicas (CONICET) and Instituto Nacional de Tecnología Agropecuaria (INTA) supported this research.