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Symptoms, aetiology and serological analysis of sweet potato virus disease in Uganda

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Sweet potato virus disease (SPVD) is the name used to describe a range of severe symptoms in different cultivars of sweet potato, comprising overall plant stunting combined with leaf narrowing and distortion, and chlorosis, mosaic or vein-clearing. Affected plants of various cultivars were collected from several regions of Uganda. All samples contained the aphid-borne sweet potato feathery mottle potyvirus (SPFMV) and almost all contained the whitefly-borne sweet potato chlorotic stunt closterovirus (SPCSV). SPCSV was detected by a mix of monoclonal antibodies (MAb) previously shown to react only to a Kenyan isolate of SPCSV, but not by a mixture of MAb that detected SPCSV isolates from Nigeria and other countries. Sweet potato chlorotic fleck virus (SPCFV) and sweet potato mild mottle ipomovirus (SPMMV) were seldom detected in SPVD-affected plants, while sweet potato latent virus (SPLV) was never detected. Isolates of SPFMV and SPCSV obtained by insect transmissions together induced typical symptoms of SPVD when graft-inoculated to virus-free sweet potato. SPCSV alone caused stunting and either purpling or yellowing of middle and lower leaves when graft-inoculated to virus-free plants of two cultivars. Similarly diseased naturally inoculated field plants were shown consistently to contain SPCSV. Both this disease and SPVD spread rapidly in a sweet potato crop.

Introduction

Sweet potato virus disease (SPVD) is the name commonly used in Africa to describe a range of severe symptoms on sweet potato generally attributed to virus infection. Symptoms vary with plant genotype but typically include stunted plants (Fig. 1a) with small leaves, the latter often also being distorted, narrow (strap-like) and crinkled, with a chlorotic mosaic and/or vein-clearing, giving affected plants an overall pale appearance (Fig. 1c). SPVD is widespread and regarded as a serious problem in Africa, affected plants commonly yielding less than half that of symptomless ones (Mukiibi, 1977; Hahn, 1979). In Nigeria, the disease has been associated with the presence of an aphid-borne potyvirus now known to be sweet potato feathery mottle virus (SPFMV) and with a closterovirus transmitted by the whitefly *Bemisia tabaci* (Schaefers & Terry, 1976; Clark & Moyer, 1988). This Nigerian closterovirus was first called sweet potato chlorotic stunt (SPCSV) (Schaefers & Terry, 1976) and later SPVD-associated closterovirus (Winter *et al*., 1992). A closterovirus isolated in Israel from sweet potato was named sweet potato sunken vein virus (SPSVV) (Cohen *et al*., 1992) but has since been shown to be serologically similar to the West African closterovirus (Hoyer *et al*., 1996; Vetten *et al*., 1996). Consequently, it has been proposed to the Closterovirus Study Group of the International Committee for the Taxonomy of Viruses that the name SPCSV should be retained for this closterovirus (H. J. Vetten, Institut für Biochemie und Pflanzenvirologie, 1996, personal communication) and this name is therefore used throughout this paper. Results of serological tests with polyclonal and monoclonal antibodies (MAb) have recently shown (Vetten *et al*., 1996; H. J. Vetten, 1997, unpublished data) that an isolate of SPCSV from Uganda and others from Kenya and Tanzania are serologically distinct from most other non-East African isolates (Nigeria, Gabon, Israel, Brazil, Argentina, USA and Taiwan).

African sweet potato production is concentrated in East Africa, especially around Lake Victoria; Uganda has the largest sweet potato production in Africa and the fourth largest in the world. SPVD is the most important disease of sweet potato in the Lake Victoria region (Geddes, 1990). Sheffield (1957) identified two viruses

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Figure 1 (a) Purpled (extreme left), SPVD (centre) and symptomless (extreme right) field plants of cultivar Tanzania, respectively, reacting in ELISA assays negatively to SPFMV and positively to SPCSV, reacting positively to both SPFMV and SPCSV, and reacting negatively to both SPFMV and SPCSV; (b) A close-up of a field plant cultivar Tanzania infected with SPCSV and showing the purpling symptom. (c) A close-up of an SPVD-affected plant cultivar Tanzania infected with both SPCSV and SPFMV, showing leaf narrowing, distortion and mosaic.

commonly infecting sweet potato in Kenya, Tanzania and Uganda and distinguished them as sweet potato viruses A and B. Virus A was aphid-borne and seems likely to have been SPFMV; B was whitefly-borne and may have been what is now known as sweet potato mild mottle ipomovirus (SPMMV) (Hollings *et al.*, 1976) or SPCSV (Schaefers & Terry, 1976). Wambugu (1991) reported several viruses to be common in Uganda and Kenya, including SPFMV and SPMMV, whereas Carey *et al.* (1996) reported only SPFMV to be common in

diseased sweet potato plants in Uganda and Kenya. The whitefly-borne SPCSV was not tested for in either of these surveys because of a lack then of diagnostic reagents for SPCSV. SPFMV causes mild or no symptoms in sweet potato (Clark & Moyer, 1988; Brunt *et al.*, 1996) and seemed unlikely to be the cause of SPVD. SPMMV also causes relatively mild symptoms in sweet potato (Hollings *et al.*, 1976). The main purpose of this present study was therefore to determine the common cause of SPVD in Uganda.

Materials and methods

Test plants

Ipomoea setosa, the near-universal indicator of sweet potato viruses (Moyer *et al.*, 1989) including SPFMV (Stubbs & McLean, 1958) and SPCSV (Schaefers & Terry, 1976), was grown from seed originally provided by the International Potato Centre (CIP, Lima, Peru). Virus-free *in vitro* cultures of the sweet potato cultivars Tanzania (CIP pathogen-tested list 440166) and Wagabolige (CIP pathogen-tested list 440167), both recently officially released in Uganda, were also provided by CIP. At Namulonge Agricultural and Animal Production Research Institute (NAARI), plants were transferred to pots of soil in an insect-proof screenhouse. Cultivar Tanzania is moderately resistant to infection with SPVD and cultivar Wagabolige is resistant.

Insect transmission

A culture of the aphid *Myzus persicae* was maintained on cabbage. Fasted apterous adults were allowed brief (*c.* 2·5 min) access to leaves of SPVD-affected field plants of sweet potato cultivar Tanzania before being given overnight access to *I. setosa* seedlings (five/seedling). Adult whiteflies (predominantly *B. tabaci*), caught in a field of sweet potato at NAARI that contained mostly SPVD-affected plants, were caged overnight in batches of *c.*50 on *I. setosa* seedlings. Plants were kept in an insect-proof screenhouse.

Serological assays for viruses

In tests for SPFMV, SPMMV, sweet potato chlorotic fleck virus (SPCFV) (Fuentes & Salazar, 1992) and sweet potato latent virus (SPLV) (Brunt *et al.*, 1996), nitrocellulose membrane-enzyme-linked immunosorbent assay (NCM-ELISA) kits provided by CIP were used. These included positive controls pre-applied to NCM. Tests for SPCSV were carried out using triple antibody sandwich (TAS)-ELISA in microplates. For coating of plates, IgG were isolated from a rabbit antiserum raised to a purified preparation of the Israeli isolate of SPCSV (=SPSVV) kindly donated by J Cohen (Cohen *et al.*, 1992) and used at a concentration of 2 μg mL^{-1}. Murine MAb were also prepared against this isolate as well as against bacterially expressed coat protein of a Kenyan isolate of SPCSV (= SPSVV; *sensu* Hoyer *et al.*, 1996). A mixture (Mix-1) was made of culture fluids containing MAb reacting solely to the isolate of SPCSV from Kenya, and another (Mix-2) was made from culture fluids containing MAb reacting solely to isolates from Israel, Nigeria and the Americas (Vetten *et al.*, 1996). These mixtures were used as the first detecting antibodies and both were used at titres previously found at the Institut für Biochemie und Pflanzenvirologie (IBP), Braunschweig, Germany to be effective for detecting, respectively, a Kenyan and a Nigerian isolate of SPCSV. Commercially obtained (Sigma Chemical Co. Ltd, Poole, UK) goat anti-mouse alkaline phosphatase-conjugated antibodies were then used at a dilution of 1:1000 and the test completed using nitrophenyl phosphatase substrate.

Sources of SPVD-affected plants

Virus-diseased field plants of specific cultivars (Table 5) were obtained from field crops at NAARI, which is situated in Mpigi District in Central Uganda. SPVD-affected plants of local landraces were obtained from farmers' crops in Mpigi District and also in Iganga and Tororo Districts in eastern Uganda, in Rakai District in southern Uganda and from Fort Portal (Kabarole) in western Uganda.

The effect of SPCSV on yield

Whitefly transmissions to *I. setosa* caused a disease subsequently identified as SPCSV (see the Results). This disease was graft-transmitted to sweet potato cultivar Tanzania. Thirty mounds *c.* 0·3 m high, of the type normally used in Uganda for sweet potato (Bashaasha *et al.*, 1995), were made with soil, over which had been erected a large screenhouse covered with 'Nikofence 36' netting (Clovis-Lande Associates Ltd, Tonbridge, Kent, UK). The mounds were arranged in 10 blocks, each of three mounds, with 1 m between the centres of mounds. Mounds were planted on 9 November 1995 with cuttings of cultivar Tanzania. Within each block, one mound was planted with three virus-free cuttings, one mound with three SPCSV-infected cuttings, and one mound was planted with three cuttings from SPVD-affected field plants previously sprayed with Applaud to kill any insects. The virus-free and SPCSV-infected cuttings were of similar sizes (*c.* 0·3 m long) and ages; however, SPVD-affected cuttings were stunted because of the effects of the disease. The different planting treatments were applied at random in each block; a single row of mounds planted with virus-free cuttings surrounded the experiment. The experiment was harvested on 30 April 1996; fresh weights of storage roots and foliage were measured for each mound.

Results

Virus symptoms observed in field plants

Characteristic symptoms of SPVD observed in sweet

Table 1 Symptoms of SPVD in different Ugandan sweet potato cultivars

Cultivar	Symptoms of SPVD
Tanzania	Plant stunted, leaves narrow and stunted, crinkled and with a pale green mosaic
Wagabolige	Plant stunted, leaves small and crinkled, pale green with chlorosis along major veins
Tororo 3	Plant stunted, small puckered leaves with a chlorotic mottle
Sowola	Plant stunted, leaves narrow and stunted with a chlorotic mottle
New Kawogo	Plant stunted, leaves small with chlorosis along major veins
Bwanjule	Plant stunted, leaves narrow with a distorted edge, purple with a pale green mottle
Bitambi	Plant stunted, leaves small, crinkled with chlorosis along major veins
Kimotoka	Plant stunted, leaves small, crinkled with chlorosis along major veins

potato cultivars at NAARI Farm and in farmers' fields are listed in Table 1. They generally comprised severe stunting (Fig. 1a), leaves becoming crinkled and distorted (often narrow and strap-like in genotypes such as cultivar Tanzania (Fig. 1c) having divided leaves), and either a pale green mosaic (Fig. 1c) or clearing of the major veins. Some cultivars with SPVD also had purpling on lower leaves. Another virus symptom commonly observed in field plants comprised a stunting of the whole plant and either a purpling of lower and middle leaves, for example in cultivar Tanzania (Fig. 1b), or a chlorotic yellowing, as in cultivar Wagabolige. No other symptoms characteristic of virus infections have been observed at NAARI Farm or in farmers' fields in these two cultivars over a 3-year period, during which several hundred diseased plants of each cultivar have been examined. In other sweet potato cultivars examined at NAARI, or sweet potato landraces examined in farmers' fields throughout Uganda, a similar identification of two common symptom types could be made, one being SPVD and the other a purpling or yellowing of foliage combined with stunting.

Field spread of purpling and SPVD in cultivar Tanzania

The occurrence of plants with the purpling symptom or of SPVD was monitored in a field of cultivar Tanzania planted with symptomless (but not virus-indexed) cuttings obtained from a field at NAARI Farm. There was a rapid spread of the purpling disease followed by an even more rapid spread of SPVD (Table 2). When samples of plants with purpling and SPVD were tested for SPFMV by NCM-ELISA, 54 of 57 plants with SPVD were positive for SPFMV but only six of 57 plants with purpling were positive (at this stage of the investigation TAS-ELISA for SPCSV was unavailable).

Table 2 The spread of purpling and SPVD in a crop of sweet potato cultivar Tanzania at NAARI Farm near Kampala

Date	Total numbers of plants with		
	Purpling	SPVD	No disease
17 April 1995	Planted as symptomless cuttings		
1 June 1995	28 (1%)	49 (2%)	2211
19 September 1995	535 (27%)	112 (6%)	1321
24 November 1995	536 (30%)	1098 (62%)	149

Plants with purpling or yellowing symptoms similar to those in the graft-inoculated sweet potato were also observed in a field that had been planted with cuttings of virus-free sweet potato cultivars Tanzania and Wagabolige. Again, SPFMV, SPMMV, SPCFV and SPLV were not detected.

Transmission and serology tests

Cuttings of cultivar Tanzania established in a screenhouse from a typical SPVD-affected field-grown plant at NAARI were found by ELISA to contain SPFMV and SPCSV, but not SPMMV, SPCFV or SPLV. Grafting scions to *I. setosa* caused, within 1–2 weeks, a severe and permanent disease on the latter, comprising stunting of the main stem and leaves, general chlorosis of the leaves and necrosis sometimes leading to plant death. Aphid transmissions from the cuttings to *I. setosa* initially induced in the latter a mosaic, then vein-clearing followed eventually by the production of symptomless leaves. These symptoms were quite different from those obtained by graft-inoculation but were typical of those reported for SPFMV in this indicator plant. The presence of SPFMV was confirmed by NCM-ELISA. When the *I. setosa* infected with SPFMV were grafted to virus-free sweet potato cultivars Tanzania and Wagabolige, no symptoms were induced over a period of observation lasting more than 1 year, although again back-grafting to *I. setosa* seedlings and testing with ELISA showed that these plants contained SPFMV. Plants of *I. setosa* inoculated using whiteflies, collected from severely diseased field plants at NAARI, became stunted and leaves were also brittle, cupped downwards and chlorotic (but not necrotic); ELISA gave reactions with Mix-1 of the MAbs to SPCSV but not with other antibodies, showing that these plants contained SPCSV but not the whitefly-borne SPMMV or SPFMV, SPCFV or SPLV.

Virus-free cuttings of cultivars Tanzania and Wagabolige were grown in pots of soil in a screenhouse, four plants of one cultivar to a pot and four pots of each cultivar. In each pot, one cutting was grafted with *I. setosa* infected with SPFMV, one with *I. setosa*

Table 3 The absorbances (A_{405nm})* generated in TAS-ELISA using MAb Mix-1 for SPCSV of duplicate sap samples of *Ipomoea setosa* and sweet potato plants cultivar Tanzania graft-inoculated with SPCSV and/or SPFMV

	Inoculated with SPCSV	Not inoculated with SPCSV
I. setosa	1·508; 1·586	0·008; 0·004
I. setosa + SPFMV	0·654; 0·665	0·004; 0·012
cv. Tanzania	0·688; 0·624	0·013; 0·004
cv. Tanzania + SPFMV	0·238; 0·246	0·008; 0·005

*Blanked on wells containing substrate only.

infected with SPCSV, one with *I. setosa* infected with SPFMV and SPCSV, and one cutting was mock-grafted. After *c.* 4 weeks the plants of cultivar Tanzania inoculated with SPFMV + SPCSV developed very stunted growth, old lower leaves became purple, and new leaves were narrow and strap-like with a pale green mosaic; similarly inoculated plants of cultivar Wagabolige also became very stunted, with pale green chlorotic leaves showing pronounced vein-clearing. These symptoms resembled those observed in the field and described as SPVD in each cultivar. After about 7 weeks, the plants of cultivar Tanzania inoculated with SPCSV became stunted and developed purple middle and lower leaves, and the plants of cultivar Wagabolige inoculated with SPCSV also became stunted and developed golden yellow leaves, symptoms resembling in each cultivar the other common virus-like symptoms observed in crops (see above). TAS-ELISA absorbance values for SPCSV in *I. setosa* and cultivar Tanzania plants are given in Table 3. All plants mock-grafted or grafted with SPFMV remained symptomless. The results are summarized diagrammatically in Fig. 2.

Serological detection of viruses in diseased field plants

Following the above results, tests were made to determine which part of diseased field plants gave the most reliable detection of SPCSV. Samples from middle leaves gave both the highest absorbance values ($0{\cdot}780 \pm 0{\cdot}127$ compared with $0{\cdot}322 \pm 0{\cdot}107$ from bottom leaves, and only $0{\cdot}096 \pm 0{\cdot}041$ in top leaves) and detected SPCSV in all diseased plants, so all subsequent samples were taken from there. Cuttings were obtained from 106 plants of a range of local cultivars, all with symptoms involving some combination of stunting, chlorosis, mosaic, vein-clearing or purpling, from farmers' fields in several locations in Uganda (Table 4). All samples but one contained both SPFMV and SPCSV, a few were additionally infected with either SPCFV or SPMMV (but none with all four viruses), and one sample was infected with SPFMV and SPMMV only. SPCSV was detected in all cases only by MAb Mix-1. Plants infected with SPCFV as well as SPFMV and SPCSV seemed to have typical symptoms of SPVD, although perhaps slightly more severe. Although plants with SPVD are stunted, they typically remain upright. In contrast, the five plants containing SPMMV, SPFMV and SPCSV were all weak as well as stunted, and had a straggly appearance; leaves had a wavy edge and a chlorotic mottle; the plant containing only SPMMV and SPFMV was also straggly but had only occasional chlorotic spots on leaves instead of a general mottle.

Tests were also carried out on field plants of cultivars Tanzania and Wagabolige at NAARI that were symptomless, purpled (cultivar Tanzania) or yellowed (Wagabolige), or had the symptoms of SPVD typical for each variety (Table 5).; SPCSV was detected by MAb Mix-1, but never by Mix-2 in all plants with SPVD, yellowing or purpling. SPFMV was detected in all plants with SPVD but only in about a third of yellowed or purpled plants and in only one symptomless plant. Similar results were obtained with symptomless and SPVD-affected plants of cultivars Tororo 3 and New Kawogo. TAS-ELISA tests for SPCSV using Mix-1 and NCM-ELISA for SPFMV on a further 10 plants each of cultivars Tanzania, Wagabolige, Sowola, Bwanjule and Tororo 3, all with SPVD, were also all positive.

The effect of SPCSV on yield

Plants grown from SPCSV-infected cuttings produced about a quarter the foliage of uninfected cuttings; SPVD-affected cuttings from the field scarcely grew and produced *c.* 3% of the foliage of uninfected cuttings (Table 6). Storage root yield was poor in the screenhouse

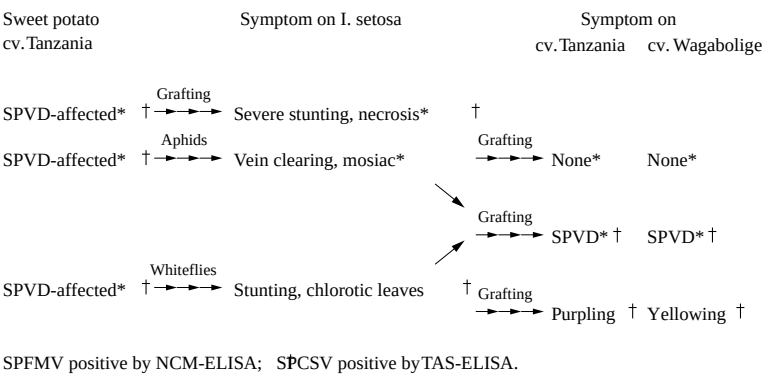


Figure 2 A diagrammatic summary of the results of the transmission of aphid- and/or whitefly-borne components of SPVD to *I. setosa* and sweet potato.

SPFMV positive by NCM-ELISA; SPCSV positive by TAS-ELISA.

Table 4 Viruses detected by TAS-ELISA (SPCSV) and NCM-ELISA in SPVD-affected sweet potato obtained from different locations in Uganda

Location	SPCSV		SPFMV	SPCFV	SPMMV	SPLV	Number of plants tested
	Mix-1	Mix-2					
Rakai	20	0	20	2	0	0	20
Fort Portal	14	0	14	1	0	0	14
Mpigi	15	0	15	1	0	0	15
Tororo	9	0	9	0	0	0	9
Tororo	10	Not tested	11	0	5	0	11
Iganga	37	Not tested	37	1	1	0	37
Total	105	0	106	5	6	0	106

for virus-free and diseased plants; however, SPCSV-infected cuttings produced only *c.* 13% and SPVD-affected cuttings produced only *c.* 2% of the yield of uninfected cuttings.

Discussion

These results demonstrate that SPFMV was present in all and SPCSV was present in all but one of *c.* 250 sweet potato plants affected by a range of severe symptoms described collectively as SPVD and obtained from several areas of Uganda. The symptoms, although differing somewhat between cultivars, included stunting and chlorosis and deformation of the leaves; these were often narrow (strap-like) in varieties with deeply lobed leaves, crinkled and with a mosaic or vein-clearing (Fig. 1a,c). SPMMV and SPCFV were detected in only a few such plants and SPLV was detected in none. Dual infection with SPCSV and SPFMV induced typical symptoms of SPVD in two cultivars. These surveys and experimental transmissions show, therefore, that dual infection with SPCSV and SPFMV is the main cause of SPVD in Uganda. Since the same cause has been indicated in West Africa (Schaefers & Terry, 1976), it seems likely that dual infection by SPCSV and SPFMV is the main cause of SPVD throughout Africa. This occurrence of SPCSV and SPFMV as the apparent causal agent in over 99% of plants recognizable as being affected with SPVD indicates that the term is useful (whilst not being completely specific) as a diagnostic as well as a descriptive term. SPCSV has also been detected in diseased sweet potato plants in the Americas (Pio- Ribeiro *et al.*, 1994, 1996) and Israel (Cohen *et al.*, 1992).

The experimental inoculations to cultivars Tanzania and Wagabolige also showed that SPCSV alone causes a disease. That it is the same disease as is commonly observed in crops in Uganda was confirmed by the consistent detection of only SPCSV in naturally diseased plants. Typical symptoms involve stunting of the plant and either a yellowing or purpling (depending on the cultivar) of the foliage (Fig. 1b); the disease spread rapidly in a field of cultivar Tanzania. SPFMV was also detected in a few plants with these symptoms. It seems likely that such plants had only recently been infected by SPFMV and symptoms of SPVD had not yet developed; indeed, most (but not all) cuttings of plants of purpled Tanzania developed SPVD when grown on in a

Table 5 Detection of SPFMV and SPCSV by TAS-ELISA in field plants of sweet potato cultivars Tanzania (Tz), Wagabolige (Wb) and Tororo 3 (Tor) affected with SPVD or a purpling or yellowing symptom

Plant sample	SPFMV	SPCSV: Mix-1		SPCSV: Mix-2	
	Positives/total sample	Positives/total sample	Absorbance (A_{405nm})	Positives/total sample	Absorbance (A_{405nm})
Tz–SPVD	35/35	35/35	0·967 ± 0·105	0/35	0·096 ± 0·024
Tz–purpled	14/35	35/35	1·440 ± 0·100	0/35	0·096 ± 0·026
Tz–symptomless	1/35	0/35	0·095 ± 0·003	0/35	0·095 ± 0·002
Virus-free control			0·102 ± 0·006		0·099 ± 0·005
Wb–SPVD	5/5	5/5	0·782 ± 0·167	0/5	0·082 ± 0·010
Wb–yellowed	1/5	5/5	1·059 ± 0·124	0/5	0·093 ± 0·006
Wb -symptomless	0/5	0/5	0·094 ± 0·006	0/5	0·100 ± 0·006
Tor–SPVD	8/8	8/8	0·533 ± 0·063	0/8	0·089 ± 0·004
Tor–symptomless	0/8	0/8	0·094 ± 0·003	0/8	0·093 ± 0·003
Virus-free control			0·085		0·090
Total					
SPVD	48/48	48/48		0/48	
Purpled/yellowed	15/40	40/40		0/40	
Symptomless	1/48	0/48		0/48	

Table 6 The mean yield (fresh weight in kg) in a screenhouse of mounds planted with virus-free, SPCSV-inoculated or SPVD-affected cuttings of sweet potato cultivar Tanzania

	Virus-free	SPCSV-infected	SPVD-affected	LSD ($P=5\%$)
Foliage	3·6	0·96	0·11	0·87
Storage roots	0·64	0·085	0·012	0·15

screenhouse. This disease has not been reported before (apparently because it has been confused with nutrient deficiency) but can by itself apparently cause considerable loss of storage roots as well as stunting foliage growth. Infection with SPFMV alone induces no obvious symptoms (Gibson *et al.*, 1997).

In Nigeria, SPCSV alone causes no symptoms in sweet potato (Schaefers & Terry, 1976). Our contrasting results suggest that SPCSV in Uganda has more severe effects than that in Nigeria. Furthermore, in *I. setosa*, our Ugandan isolate of SPCSV causes severe stunting, chlorosis and a downward cupping of leaves, whereas Nigerian SPCSV has been reported to cause only stunting and mild chlorosis but no leaf malformation (Schaefers & Terry, 1976); mild, not readily recognizable symptoms (Rossel & Thottappilly, 1985); or stunting and inward-rolling leaves but leaf chlorosis only 'under the most favourable conditions' (Winter *et al.*, 1992).

Additionally, in Nigeria, Hahn (1979) found that SPVD-affected plants had a foliage and storage root yield >20% of symptomless controls, whereas SPVD-affected sweet potato in Uganda had a foliage and storage root yield <5% of virus-free controls. Clones of sweet potato selected for virus resistance in Nigeria (Hahn *et al.*, 1981) have also proved susceptible in Uganda (Mwanga *et al.*, 1991).

In addition to Ugandan SPCSV apparently being more severe than Nigerian isolates, it has been shown previously that an isolate of SPCSV from neighbouring Kenya can be distinguished serologically from isolates obtained from Nigeria, Israel and the Americas (Hoyer *et al.*, 1996; Vetten *et al.*, 1996). The results of the present study show that this difference is general; all samples of SPCSV obtained from several regions in Uganda and from a range of cultivars were detected by MAb Mix-1 (which do not react to a Nigerian isolate) whereas none was detected by MAb Mix-2 (which do react to a Nigerian isolate). One important extrapolation from our work is therefore that phytosanitary controls must be enforced to prevent this more severe strain of SPCSV being moved about or from Africa. Furthermore, although sweet potato originates from the Americas and the East African (but not the West African) serotype of SPCSV are serologically related to lettuce infectious yellows virus (Hoyer *et al.*, 1996), which has been reported only from North America, this serological diversity of SPCSV in Africa suggests a possible African origin of this virus, consistent with a possible African origin of its insect vector, *B. tabaci* (Campbell *et al.*, 1995).

In Nigeria, SPVD has been reported to result from the synergistic effects of the whitefly-borne virus SPSCV and SPFMV (Clark & Moyer, 1988), and SPCSV is acquired readily only from sweet potato infected with both viruses (Schaefers & Terry, 1976). In the present study, TAS-ELISA absorbance values were high for SPCSV plants infected by this virus alone and there was no evidence of an increased SPCSV titre in plants dually infected with it and SPFMV, in fact rather the reverse (Tables 3 and 6). This lack of synergistic effect of SPFMV on Ugandan SPCSV may be a further difference between SPCSV from Uganda and Nigeria but it needs to be confirmed by parallel tests conducted at a single location.

Sweet potato is a daily household food for rural and urban populations in Uganda (World Bank, 1993; Bashaasha *et al.*, 1995) and elsewhere in much of East Africa. SPVD is the most damaging disease of sweet potato in East Africa (Geddes, 1990). Understanding the epidemiology of SPVD and developing control strategies, particularly selecting resistant genotypes, requires an ability to identify correctly the presence of the causal viruses. The development and validation of TAS-ELISA for SPCSV in East Africa is therefore of considerable strategic importance. NCM-ELISA kits have been developed by CIP for detecting SPFMV, the aphid-borne component of SPVD. TAS-ELISA has now shown that SPCSV is the common whitefly-borne component of SPVD and is an effective test for this virus. This development of robust methods for detecting both component viruses causing SPVD should enable work on its control to progress more rapidly.

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