Sweet potato viruses in South Africa and the effect of viral infection on storage root yield

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A survey was conducted during 2001–2003 of sweet potato grown by small-scale farmers in the seven main sweet potato-producing provinces of South Africa to determine the incidence of viral diseases in the field. Based on symptoms observed, disease incidence was relatively low. The highest levels of infection observed were between 9–10 % in the Western Cape and Mpumalanga. Symptoms were less prevalent or absent in the Eastern Cape, Limpopo, Gauteng, KwaZulu-Natal and North West. However, grafting onto *Ipomoea setosa* and subsequent testing by ELISA indicated that 81 % of the samples were infected by viruses. Nine viruses were identified. *Sweet potato feathery mottle virus* (SPFMV) was the most common and occurred in 73 % of the samples tested. *Sweet potato mild mottle ipomovirus* (SPMMV) and *Sweet potato latent potyvirus* (SPLV) were detected in 3 % and 5 % of the samples, respectively. Five viruses were found for the first time in South Africa, viz. *Sweet potato chlorotic fleck virus* (SPCFV), *Sweet potato mild speckling virus* (SPMSV), *Sweet potato chlorotic stunt crinivirus* (SPCSV-East and West African strains), *Sweet potato virus Y* (SPVY) and *Sweet potato virus G* (SPVG). The effect of viral infection on storage root yield of ten sweet potato varieties was also studied over two seasons. Total yield was on average reduced by between 12 and 22 %, and marketable yield by 21 to 38 %. Reduction in marketable yield was mainly due to increased cracking of the storage roots. The new Agricultural Research Council varieties Monate and Ndou, and the 1994-5-1 breeding line produced marketable yields of 34–48 tonnes ha^{-1} even when infected with viruses, whereas the established South African varieties Mafutha and Natal Red yielded only 4–16 tonnes ha^{-1}.

Key words: reduced yield, South Africa, SPCSV, SPFMV, SPMMV, SPVG, SPVY, sweet potato, virus detection.

Sweet potato (*Ipomoea batatas* (L.) Lam.), is grown primarily for its storage roots, which constitute a very important and reliable source of food for resource-poor people in central, southern and eastern African countries (Moyo et al. 1999; Laurie 2004). Viral diseases are the major biotic constraint to sweet potato production in eastern and southern Africa (Owour 2000). *Sweet potato feathery mottle virus* (SPFMV) infects the crop worldwide (Moyer & Salazar 1989). Other viruses reported include *Sweet potato mild mottle ipomovirus* (SPMMV) (Hollings et al. 1976), *Sweet potato chlorotic stunt crinivirus* (SPCSV) (Karyeija et al. 2000; Gibson & Aritua 2002) and the potyviruses *Sweet potato virus G* (SPVG) (Colinet et al. 1994) and *Sweet potato virus Y* (SPVY) (Ateka et al. 2004). *Cucumber mosaic virus* (CMV) has been reported to severely infect sweet potato in Israel (Cohen et al. 1988). SPFMV, SPMMV and *Sweet potato latent potyvirus* (SPLV) infection of sweet potato has been recorded in South Africa (Thompson & Mynhardt 1986; Jericho & Thompson 2000), and studies indicated that elimination of viruses can lead to an increase in yield of up to 80 % (Joubert et al. 1974; Laurie et al. 2000).

The objectives of this study were to establish the incidence of sweet potato viral infection in the seven main sweet potato-growing provinces of South Africa, to observe the prevalence of viral symptoms in the field in each province, and to determine the identity and distribution of viruses infecting sweet potato grown particularly by small-scale farmers in the country. Evidence is also presented regarding the effect of four of the most common viruses as co-infections on the root yield of local and imported sweet potato varieties over two seasons.

Materials and methods

Field assessment of sweet potato viruses

Field surveys were conducted between January 2001 and August 2003 in the seven main sweet potato-growing provinces of South Africa, namely Limpopo, Mpumalanga, KwaZulu-Natal, Western Cape, Gauteng, Eastern Cape and North West. All

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areas were surveyed according to a W-pattern and 5–25 sampling points were randomly selected along this pattern depending on the number and size of the fields in the area. Viral disease severity was assessed at each sampling point on a scale of 0–5, with 0 representing no symptoms, 1–3 mild to moderate symptoms, and 4–5 severe to very severe symptoms. The incidence of sweet potato viral disease symptoms per province was calculated according to Snedecor & Cochran (1980).

A total of 753 sweet potato samples in the form of cuttings were randomly collected at the various sampling points during the surveys. Each cutting was transplanted to sterilised potting soil in a 20-cm-diameter plastic pot and pots with developing plants were maintained for further analysis at a day/night temperature of 30/20°C at the ARC-Vegetable and Ornamental Plant Institute (VOPI), Pretoria. Four symptomatic specimens of a wild *Ipomoea* sp. growing amongst sweet potatoes were also collected for virus analysis. The species was subsequently identified by the National Botanical Institute of South Africa as *Ipomoea senensis* (Ders.) Choisy.

Infectivity assays

Six-hundred-and-fifteen surviving plants from the present survey, as well as 28 from a commercial farm near Marble Hall in Mpumalanga Province and 51 from a survey conducted in 1997–1999, were indexed by grafting onto *Ipomoea setosa* Kerr. at the three-leaf stage. Grafted plants were maintained in the above greenhouse and were monitored for viral symptoms over a period of six weeks.

Serological tests

Symptomatic leaves from the bottom, middle and top of the grafted *I. setosa* plants, and leaves from sweet potato plants maintained in the greenhouse, were analysed serologically. Samples were tested with three types of enzyme-linked immunosorbent assays (ELISA), viz. nitrocellulose membrane-based ELISA (NCM-ELISA), using Hybond C Extra membrane (Amersham Life Science, Buckinghamshire, UK), triple-antibody sandwich ELISA (TAS-ELISA) and double-antibody sandwich ELISA (DAS-ELISA). Antisera for virus analysis were provided by the International Potato Centre (CIP), Lima, Peru, and the Institut für Biochemie und Pflanzenvirologie, Braunschweig, Germany. Assays were carried out according to the instructions provided. CMV antiserum was produced at ARC-VOPI. NCM-ELISA was used to test for SPFMV, SPMMV, SPLV, *Sweet potato chlorotic fleck virus* (SPCFV), *Sweet potato mild speckling virus* (SPMSV), *Sweet potato caulimolike virus* (SPCaLV), C-6 virus and CMV. SPVY and SPVG were tested for by DAS-ELISA and SPCSV by TAS-ELISA, using polyclonal (PAb's) and monoclonal (MAb's) antibodies.

For DAS- and TAS-ELISA, visual assessment was supported by taking plate readings using a Flow Titertek® Multiskan Plus ELISA plate reader (Labsystems, Finland) at 405 nm.

Effect of viral infection on storage root yield of sweet potato varieties

The following sweet potato varieties were selected for evaluation based on their potential to be used by resource-poor farmers: Bosbok, Mafutha and Natal Red (South African varieties), Mamphenyane, Monate and Ndou (new ARC varieties), 1994–5–1 (ARC breeding line), Excel, CN 1656–97 and Xushu 18 (imports from CIP). Trials were conducted in 2001/2002 and 2003.

Infected sweet potato plants collected during the 1997–1999 survey were used as source of inoculum of SPFMV as a single infection and of SPFMV+SPMMV as a mixed infection. However, it was subsequently established that these sources were also infected with SPVY and SPVG and the inocula were therefore designated virus combination A = SPFMV+SPVY+SPVG, virus combination B = SPFMV+SPMMV+SPVY+SPVG, and C = negative control/virus-free. The virus-free control plants were provided by the ARC-VOPI sweet potato virus indexing scheme for multiplication and distribution of virus-free sweet potato material (Laurie & Stock 1997).

Cuttings of the selected varieties were inoculated in duplicate by side-grafting virus-infected cuttings onto them. Virus-free control plants were provided by ARC-VOPI. Three months after inoculation, cuttings of the grafted varieties were indexed by grafting onto *I. setosa.* The indicator plants were monitored for symptom development and tested by NCM-ELISA and DAS-ELISA to confirm the presence of the viruses in the inoculated germplasm. Cross-infection was prevented by maintaining the three treatments in separate insect-proof greenhouses. Plants that tested positive were multiplied and transplanted to the field in October 2001 in a multiplication plot for the

Table 1. Incidence of viral disease symptoms in sweet potato grown by small-scale farmers in the main sweet potato-growing provinces of South Africa.

Province	No. of areas surveyed	Total no. of sampling points	Total symptom score	Mean score	Incidence (%)[a]
Limpopo					
Klipspruit, Riverside, Arthur Stone, Dingleydale, Tshidani, Mianzwi, Tshiombo, Maseromola Park, Greenside	9	77	0	0	0
Mpumalanga					
Gutjwa, Mgcobaneni, Beverbreed, Hazyview, Numbi, Ntsikadzi, Tonga, Burgershall, Lundi	9	117	106.7	0.912	10.0
Eastern Cape					
Alice, Thombo, Lower Mpako, Mpako, Port Alfred, Ncambendlana, Bathurst, Mqanduli	8	96	5.1	0.053	0.7
Western Cape					
Saron, Friemersheim, Goedverwag, Ebenhaeser, Pacaltsdorp	5	121	53.3	0.440	9
North West					
Magolego, Themba	2	39	2.0	0.051	2.5
KwaZulu-Natal					
Mvundleni	1	13	0	0	NC
Gauteng					
Cullinan	1	13	10.1	0.777	NC

[a]Calculated according to Snedecor & Cochran (1980).
NC = incidence not calculated because only one area was surveyed.

production of planting material. The first field trial was established in December 2001 with material taken from the multiplication plot.

The second trial was aimed at determining the cumulative effect of viral infection over time. This consisted of the previously virus-infected (2001 trial) and the newly infected plants. The previous season's infection was sustained by maintaining virus-infected plants from the previous trial after harvesting in sterilised potting soil in 20-cm-diameter plastic pots in the above greenhouses to separate treatments from each other during the winter of 2002. For new infections, negative plants were again inoculated by side-grafting the virus-infected cuttings onto them. Viral infection was confirmed following the same procedures as in the 2001 trial. Virus-tested plants were multiplied and transplanted to a new field plot towards the end of October 2002. The second field trial was established in January 2003 with material from this source.

Both trials were laid out according to a randomised complete block design with three replicates per treatment, i.e. virus combination A, virus combination B, and the healthy control. Each replicate comprised 30 plants at inter- and intra-row spacing of 1 m and 0.3 m , respectively. Maize (*Zea mays* L.) was planted between treatments to restrict cross-infection from one treatment to the other by aphids. The trials received supplementary fertilisation totalling the equivalent of 160 kg N, 90 kg P and 178 kg K ha^{-1} (2001/2002 trial) and 136 kg N, 80 kg P and 144 kg K ha^{-1} (2003 trial). Fertiliser was applied before planting and four and eight weeks after planting. Irrigation was applied when required. The plants had a growth period of five months and were harvested at the end of May 2002 and mid-June 2003, respectively. Storage roots were graded as marketable (good-quality roots of 100–1200 g) or unmarketable (<100 g, >1200 g, cracked, rotten, damaged by insects).

Data were analysed using GenStat (2003). Analysis of variance was used to test for differences between treatments and treatment means were separated by Fisher's protected *t*-test least significant difference.

Results

Field assessment of sweet potato viral diseases

Table 1 summarises the incidence of sweet potato viral infection in the seven provinces

surveyed. Typical viral symptoms were observed in Mpumalanga and the Western Cape but rarely in the Eastern Cape, Limpopo, Gauteng, North West and KwaZulu-Natal. Mpumalanga was the only province where viral symptoms were conspicuously expressed. The most common symptoms observed were those typical of potyviruses, namely vein-clearing, leaf crinkling, chlorotic spots, and purple ringspots on older leaves. Symptoms of sweet potato viral diseases (SPVD) in general, such as severe vein-clearing, leaf distortion, chlorosis, mosaic and stunting, were also evident at Hazyview (Ntsikadzi) and Gutjwa in Mpumalanga. The mean percentage incidence of visual symptoms in all provinces ranged between 0 % (Limpopo) and 10 % (Mpumalanga) (Table 1).

Infectivity assays

Symptoms observed on *I. setosa* were typical of potyviruses and included vein-clearing, chlorotic spots, chlorosis and diffuse mottling. After six weeks of symptom monitoring, 134 (19 %) of the 694 indexed sweet potato samples expressed no symptoms on *I. setosa.* These samples were assumed to be virus-free after confirmation that the graft had taken.

Serological tests

Table 2 summarises the serological test results of the symptomatic samples tested further with ELISA procedures. SPFMV was the most prevalent, occurring in all seven provinces, with 73 % of the samples infected. SPVY and SPVG were present in 32 and 34 % of the samples tested, respectively. These two viruses were commonly detected in samples already infected with SPFMV and other viruses. TAS-ELISA detected low levels of SPCSV in the sweet potato leaves tested. About 3 and 1 % of the samples tested were infected with East African and West African strains of SPCSV, respectively. SPCSV-WA was detected only in samples from the commercial farm near Marble Hall in Mpumalanga Province. SPCSV was not always transferred to *I. setosa.* Attempts to retest SPCSV-infected plants using grafted *I. setosa* leaves yielded negative results, while leaves of a SPCSV-infected sweet potato plant continued to test positive. Symptomatic wild *I. senensis* plants were infected with SPCSV-EA, SPFMV, SPVG and SPVY.

SPLV, SPCFV and SPMSV were detected in samples from Limpopo, Mpumalanga and Gauteng. SPLV and SPCFV were also present in a few samples from the Eastern Cape, and SPCFV and SPMSV in some from the Western Cape. The above three viruses were mostly detected in samples already infected with either SPFMV or other potyviruses, and seldom as a single infection. SPMMV was present in about 3 % of all samples tested. CMV was detected in only one sample from the Western Cape, whereas SPCaLV and C-6 were not detected at all. Mixed infections were more prevalent and only 11 % of the samples were infected by a single virus. Three per cent of the samples induced symptoms on *I. setosa,* but tested negative in the serological tests.

Effect of viral infection on storage root yield of sweet potato

Viral infection generally decreased yield significantly compared to the control after only one season of infection. Yield variables of the first (2001) and second (2003) trials are given in Table 3.

Sweet potato varieties varied in their reaction to viral infection. Response also varied with virus treatment. Monate and 1994–5–1 exhibited a significant decrease in both marketable and total yield, but Monate had a greater decrease with virus treatment B than with virus treatment A, whereas 1994–5–1 was more sensitive to virus treatment A. The reduction in marketable yield could mainly be ascribed to an increased incidence of cracked storage roots. Cracking was significantly aggravated by viral infection in six of the ten sweet potato varieties tested, the exceptions being Monate, Bosbok, Natal Red and Mamphenyane.

Monate, Ndou and 1994–5–1 are valuable as they still produced high marketable yields of 34–48 tonnes ha^{-1} following both virus treatments (Fig. 1). The medium-yielding varieties were Bosbok, Excel and Mamphenyane, with 16–32 tonnes ha^{-1} of marketable yield after viral infection. The poor-yielding varieties were CN1656–97, Natal Red, Xushu 18 and Mafutha, with marketable yields severely reduced by viral infection to only 4–16 tonnes ha^{-1}.

Discussion

This study represents the first detailed identification of sweet potato viruses in South Africa and assessment of their effect on root yield. Results indicated that viruses constitute a significant constraint to sweet potato production in both

Table 2. Viruses present in sweet potato plants collected in the seven main sweet potato-growing provinces of South Africa, as determined by ELISA.

Province	Area	No. of samples	Incidence[a]												
			NCM-ELISA								DAS-ELISA		TAS-ELISA		
			SPFMV	SPMMV	SPLV	SPCFV	SPMSV	C-6	CMV	SPCaLV	SPVY	SPVG	SPCSV-EA	SPCSV-WA	Negative with ELISA	Single infections
Present survey (2001–2003)																
Limpopo	Klipspruit	30	21	0	1	0	0	0	0	0	–	–	–	–		
	Arthur Stone	15	13	0	3	1	0	0	0	0	–	–	–	–		
	Dingleydale	12	7	0	0	0	0	0	0	0	–	–	–	–		
	Riverside	15	11	0	0	1	0	0	0	0	–	–	–	–		
	Tshiombo	67	60	3	3	1	2	0	0	0	–	–	–	–		
	Total	139	112 (81)	3 (2)	7 (5)	3 (2)	2 (1)	0 (0)	0 (0)	0 (0)	–	–	–	–	6 (4)	36 (26)
Mpumalanga	Gutjwa	130	103	0	4	6	0	0	0	0	59	68	17	0		
	Hazyview	40	25	0	5	6	1	0	0	0	28	29	0	0		
	Beverbreed	31	29	4	6	1	0	0	0	0	19	12	0	0		
	Tonga	18	18	1	1	0	0	0	0	0	10	11	0	0		
	Mgcobaneni	37	18	0	0	0	0	0	0	0	32	34	0	0		
	Marble Hall	28	19	0	1	0	1	0	0	0	9	10	3	7		
	Total	284	212 (75)	5 (2)	17 (6)	13 (5)	2 (1)	0 (0)	0 (0)	0 (0)	157 (55)	164 (58)	20 (7)	7 (2)	13 (5)	12 (4)
Eastern Cape	Alice	4	3	0	0	0	0	0	0	0	3	4	0	0		
	Thombo	3	2	0	0	0	0	0	0	0	1	0	0	0		
	Mpako	8	9	1	0	1	0	0	0	0	4	7	0	0		
	Port Alfred	16	5	1	1	0	0	0	0	0	3	4	1	0		
	Ncambendlana	5	1	0	0	0	0	0	0	0	1	1	0	0		
	Bathurst	12	2	0	1	0	0	0	0	0	2	3	0	0		
	Total	48	22 (46)	2 (4)	2 (4)	1 (2)	0 (0)	0 (0)	0 (0)	0 (0)	14 (29)	19 (40)	1 (2)	0 (0)	1 (2)	2 (4)
Western Cape	Saron	17	14	0	0	0	0	0	0	0	12	11	0	0		
	Friemersheim	13	8	0	0	0	0	0	0	0	2	7	1	0		
	Pacaltsdorp	10	10	0	0	0	0	0	0	0	6	1	0	0		
	Ebenhaeser	19	14	0	0	1	2	0	0	0	10	10	0	0		
	Goedverwag	30	17	0	0	1	0	0	1	0	9	13	0	0		
	Total	89	63 (71)	0 (0)	0 (0)	2 (2)	2 (2)	0 (0)	1 (1)	0 (0)	39 (44)	42 (47)	1 (1)	0 (0)	2 (2)	16 (18)
Gauteng	Cullinan	27	27	0	2	2	1	0	0	0	–	–	–	–		
	Total	27	27 (100)	0 (0)	2 (7)	2 (7)	1 (4)	0 (0)	0 (0)	0 (0)	–	–	–	–	0 (0)	0 (0)
KwaZulu-Natal	Mvundleni	40	5	0	0	0	0	0	0	0	3	1	0	0		
	Total	40	5 (13)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	3 (8)	1 (3)	0 (0)	0 (0)	1 (3)	5 (13)
North West	Themba	6	4	0	0	0	0	0	0	0	3	3	0	0		
	Magolego	10	10	0	0	0	0	0	0	0	3	10	0	0		
	Total	16	14 (88)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	6 (38)	13 (81)	0 (0)	0 (0)	0 (0)	2 (13)

Continued on p. 20

Table 2 (*continued*)

Province	Area	No. of samples	Incidence[a]												
			NCM-ELISA							DAS-ELISA		TAS-ELISA		Negative with ELISA	Single infections
			SPFMV	SPMMV	SPLV	SPCFV	SPMSV	C-6	CMV	SPCaLV	SPVY	SPVG	SPCSV-EA	SPCSV-WA		
Previous survey (1997–1999)																
Mpumalanga	Tonga	9	9	3	1	1	0	0	0	0	–	–	–	–		
	Thulamhashe	3	3	0	1	0	0	0	0	0	–	–	–	–		
	Mamfene	24	24	3	5	2	0	0	0	0	–	–	–	–		
	Hoxane	3	3	0	1	0	0	0	0	0	–	–	–	–		
	Total	39	39 (100)	6 (15)	8 (21)	3 (8)	0 (0)	0 (0)	0 (0)	0 (0)	–	–	–	–	0 (0)	0 (0)
Limpopo	Tshiombo	12	12	4	1	1	1	0	0	0	–	–	–	–		
	Total	12	12 (100)	4 (33)	1 (8)	1 (8)	1 (8)	0 (0)	0 (0)	0 (0)	–	–	–	–	0 (0)	0 (0)
Total no. samples		694	506	20	37	25	8	0	1	0	219	239	22	7	23	73
Overall percentage			73	3	5	4	1	0	0.1	0	32	34	3	1	3	11

[a]Number (percentage) of samples testing positive.
SPFMV = *Sweet potato feathery mottle virus*, SPMMV = *Sweet potato mild mottle ipomovirus*, SPLV = *Sweet potato latent potyvirus*, SPCFV = *Sweet potato chlorotic fleck virus*, SPCaLV = *Sweet potato caulimolike virus*, SPMSV = *Sweet potato mild speckling virus*, CMV = *Cucumber mosaic virus*, SPVY = *Sweet potato virus Y*, SPVG = *Sweet potato virus G*, SPCSV (EA and WA) = *Sweet potato chlorotic stunt crinivirus* (East African and West African strains).
– = samples not tested for the particular virus.

small-scale and commercial farming. SPFMV was the most prevalent virus and poses a serious threat to sweet potato production, as previously indicated by Jericho & Thompson (2000). The low infection rate of SPMMV compared to SPFMV can probably be ascribed to its dependence on transmission by whiteflies (*Bemisia tabaci* (Genn.) (Homoptera: Aleurodidae)), which were limited in distribution in the areas sampled during the surveys.

This is the first report of the occurrence and wide distribution of the potyviruses SPVY and SPVG on sweet potato in South Africa. SPCFV, SPMSV and SPCSV EA and WA strains are also reported for the first time in South Africa, but appear to be restricted in distribution.

TAS-ELISA proved to be reliable and accurate when testing for the presence of SPCSV in sweet potato leaves. However, retesting for SPCSV using grafted *I. setosa* leaf material was found to be unreliable. The fact that co-infection of SPFMV and SPCSV does not always result in the expression of severe SPVD condition in South Africa can probably be ascribed to differences in climatic conditions which seem to cause variation in virus concentration and the expression of symptoms on sweet potatoes in the field. A thorough characterisation of SPCSV to explore the mechanism behind its synergism with SPFMV may provide answers as to why the synergy does not always result in the expression of SPVD symptoms in local sweet potato fields.

The fact that 3 % of the samples that induced symptoms on *I. setosa* failed to react with any of the antisera, is possibly an indication of the presence of as yet unidentified viruses. This was also shown in Tanzania where plants with virus-like symptoms did not test positively with the antisera used (Ndunguru & Kapinga 2007).

Yield losses in storage roots of sweet potato have been associated with viral infection since the 1970s in South Africa (Joubert et al. 1974). Yield losses in excess of 70 % have been ascribed to the synergistic effect of SPFMV and SPCSV in Uganda (Gibson et al. 1998; Gibson & Aritua 2002). The present study showed that, even during the first season after infection, total and marketable yield is on average reduced by 14.2 and 14.8 tonnes ha^{-1}, respectively. Although yield loss and quality of storage roots have been determined with only two combinations of mixed viral infection, it is evident that the presence of viruses lowers the

Table 3. Effect of viral infection on storage root cracking and yield of selected sweet potato varieties.

Variety	Treatment[a]	Root cracking (%)[bc]			Marketable yield (tonnes ha^{-1})[b]			Total yield (tonnes ha^{-1})[b]		
		2001	2003A	2003B	2001	2003A	2003B	2001	2003A	2003B
Monate	C	2.9	5.1	12.8	57.5	51.7	46.2	84.1	71.5	68.7
	A	7.8	8.0	6.9	57.4	42.4*	44.8	76.1	59.4*	66.7
	B	2.8	8.7	19.0	41.6*	31.1*	36.4*	50.7*	48.1*	57.4*
1994–5–1	C	13.2	11.3	13.6	65.4	44.2	35.3	81.9	57.0	47.8
	A	26.4*	24.6	8.0	26.6*	29.7*	45.3*	40.3*	45.2*	62.2*
	B	9.0	15.2	22.2	64.2	33.1*	36.8	75.6	52.4	58.7
Ndou	C	0	2.3	0	69.7	50.3	44.6	82.7	63.3	68.0
	A	7.6	6.1	23.9*	53.1*	44.6	28.4*	67.4*	62.7	51.3*
	B	0	6.1	8.6	48.0*	32.4*	43.6	60.5*	51.8*	60.0
Bosbok	C	3.8	2.8	5.0	53.6	22.3	39.6	62.8	30.1	49.8
	A	5.7	0	0	34.3*	30.1	12.0*	44.9*	37.1	18.2*
	B	0	0	0	38.1*	21.3	22.7*	46.3*	35.0	32.7*
CN 1656–97	C	19.1	26.3	12.8	26.8	18.5	19.4	50.0	36.0	33.4
	A	53.3*	70.7*	7.0	8.4*	1.1*	1.0*	41.2	29.9	31.5
	B	28.9	46.5*	19.0	22.6	7.5*	0.7*	44.2	31.0	20.8*
Excel	C	10.6	23.1	–	31.8	31.1	–	42.9	48.6	–
	A	14.5	16.4	–	36.9	27.6	–	50.5	50.6	–
	B	38.6*	36.2	–	16.4*	17.1*	–	38.0	38.3	–
Mafutha	C	6.3	35.3	26.5	38.5	18.0	24.5	52.9	35.6	35.5
	A	18.5*	66.0*	41.4*	32.5	2.8*	12.5*	47.2	21.2*	28.9
	B	26.0*	58.7*	27.1	12.6*	2.3*	17.2	23.1*	15.9*	29.9
Natal Red	C	48.8	65.4	76.9	23.9	6.7	2.3	62.3	55.3	45.6
	A	58.2	78.5	78.3	9.6*	0.9	1.0	45.4*	36.9*	34.5
	B	51.1	76.3	75.9	23.9	2.3	3.3	64.5	43.5*	36.4
Xushu 18	C	26.3	51.4	43.1	45.4	16.0	20.5	69.0	56.0	52.6
	A	46.3*	81.0*	46.1	18.7*	1.1*	10.3*	51.8*	52.5	48.5
	B	53.8*	82.2*	44.0	14.8*	1.3*	18.3	54.8*	48.7	53.0
Mamphenyane	C	–	–	11.2	–	–	26.1	–	–	43.8
	A	–	–	18.0	–	–	30.7	–	–	45.0
	B	–	–	14.1	–	–	26.8	–	–	41.8
Overall mean		21.5	31.6	28.3	35.9	22.5	25.1	56.0	44.8	45.3
Mean C		14.5	23.4	21.9	45.8	28.5	30.3	65.4	49.7	49.9
Mean A		26.5*	36.9*	32.3*	30.6*	21.1*	21.2*	51.6*	44.0*	42.5*
Mean B		23.4	34.4*	30.8*	31.4*	17.8*	23.9*	50.8*	40.7*	43.4*

[a]C = uninfected control, A = composite of SPFMV, SPVY and SPVG, B = composite of SPFMV, SPMMV, SPVY and SPVG.
[b]2001 = first trial (plants inoculated prior to planting in 2001), 2003A & B = second trial (A = plants retained from 2001 trial and planted in 2003, and B = plants inoculated prior to planting in 2003).
[c]Data were angularly transformed to stabilise variances.
*Differs significantly from the corresponding uninfected control according to Fisher's protected *t*-test least significant difference ($P \leq 0.05$).
– = not determined.

quality of sweet potatoes by causing an increase in cracking, thus reducing marketability.

The practice by small-scale farmers to use the same planting material for several seasons is probably the main reason for the low yields reported by them. To maintain good yields, farmers must be made aware of the importance of planting virus-free material and replacing this material every two or three years to prevent virus build-up (Laurie et al. 2000). Both commercial and small-scale farmers should be encouraged to establish nursery blocks of disease-free material a sufficient distance away from their production areas to prevent the introduction of viruses. The fact that 19 % of the

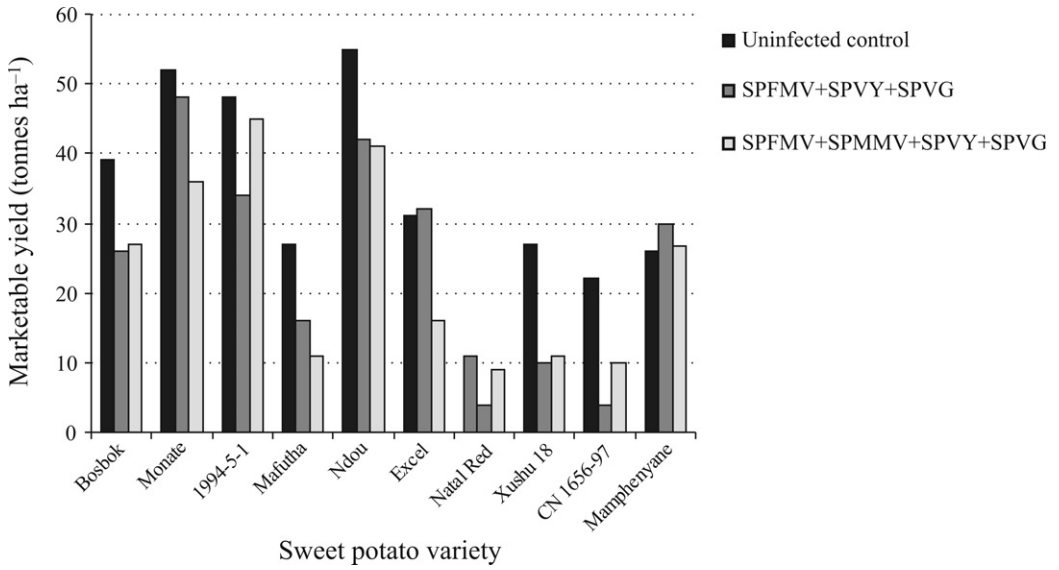

Fig. 1. Effect of viral infection on marketable storage root yield of selected sweet potato varieties. Mean of 2001, 2003A and B data.

samples collected in this study tested negatively for viral infection indicated that there is a reasonable quantity of virus-free planting material available for this purpose. However, poor expression of the symptoms on sweet potato plants in the field will make it difficult for farmers to identify possible virus-free plants. This emphasises the importance of breeding for virus-tolerant cultivars as a long-term solution to maintain virus-free planting material. In South Africa, the virus-free sweet potato scheme at ARC-VOPI is effective in maintaining the performance of sweet potato cultivars in terms of yield (Laurie et al. 2000).

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References

Ateka E M, Barg E, Njeru R W, Lesemann D-E & Vetten H J 2004. Further characterization of sweet potato virus. 2. A distinct species of the genus potyvirus. *Archives of Virology* **149**: 225–239.

Cohen J, Loebestein G & Spiegel S 1988. Infection of sweet potato by cucumber mosaic virus depends on the presence of a whitefly-transmitted agent. *Plant Disease* **72**: 583–585.

Colinet D, Kummert J & Lepoivre P 1994. The complete nucleotide sequences of the coat protein cistron and the 3′non-coding region of a newly-identified potyvirus infecting sweet potato, as compared to those of sweet potato feathery mottle virus. *Archives of Virology* **139**: 327–336.

GenStat for Windows 2003. Introduction (7th edition). VSN International, Oxford.

Gibson R W, Mpembe I, Alicai T, Carey E E, Mwanga R O M, Seal S E & Vetten H J 1998. Symptoms, aetiology and serological analysis of sweet potato virus disease in Uganda. *Plant Pathology* **47**: 95–102.

Gibson R W & Aritua V 2002. The perspective of sweet potato chlorotic stunt virus in sweet potato production in Africa: A review. *African Crop Science Journal* **10**: 281–310.

Hollings M, Stone O M & Bock K R 1976. Sweet potato mild mottle ipomovirus. In: *Viruses of plants, descriptions and lists from the VIDE database,* 1223 (Eds A A Brunt, K Crabtree, M J Dallwitz, A J Gibbs & L Watson). CAB International, Wallingford.

Jericho C (Jr) & Thompson G J 2000. Detection of sweet potato viruses in South Africa. *African Potato Association Proceedings* **73**: 379–391.

Joubert T G, Klesser P J & Nel D D 1974. The cultiva-

tion of sweet potatoes: the value of virus-indexed material. *Farming in South Africa. Sweet Potato Series* IV, 1–3. Vegetable and Ornamental Plant Institute, Roodeplaat, Pretoria.

Karyeija R F, Kreuze J F, Gibson R W & Valkonen J P T 2000. Synergistic interaction of a potyvirus and a phloem-limited crinivirus in sweet potato plants. *Virology* **269**: 26–36.

Laurie S M 2004. Sweet potato in perspective. In: *Guide to sweet potato production in South Africa,* 1–6 (Ed. J G Niederwieser). ARC-Vegetable and Ornamental Plant Institute, Roodeplaat, Pretoria.

Laurie S M & Stock P O 1997. A scheme for the distribution of disease-free propagation material in South Africa. In: *Proceedings of the 4th triennial congress of the African Potato Association,* 287. Pretoria, South Africa, 23–28 February 1997.

Laurie S M, Thompson G J & Van den Berg A A 2000. Present state of quality and yield of Southern African sweet potato cultivars. In: *Proceedings of international workshop on sweet potato cultivar decline study,* 55–61 (Eds Y Nakazawa & K Ishiguro). Department of Upland Farming, Kyushu National Agricultural Experiment Station, Miyakonojo, Miyazaki, Japan.

Moyer J W & Salazar L F 1989. Virus and virus-like diseases of sweet potato. *Plant Disease* **73**: 451–445.

Moyo C C, Benesi I R M, Sandifolo V S & Teri J M 1999. Current status of cassava and sweet potato production and utilisation in Malawi. In: *Food security and crop diversification in SADC countries: the role of cassava and sweet potato,* 51–68 (Eds M O Akoroda & J M Teri). Proceedings of the scientific workshop of the Southern African Root Crops Research Network, Lusaka, Zambia.

Ndunguru J & Kapinga R 2007. Viruses and virus-like diseases affecting sweet potato subsistence farming in southern Tanzania. *African Journal of Agricultural Research* **2**: 232–239.

Owour O 2000. Local trial for virus-free sweet potato. In: *KUZA* [newsletter], issue no. 1, p. 4 (Eds S Kandi & M Kinua). Monsanto Central Africa, Nairobi, Kenya.

Snedecor G W & Cochran W G 1980. *Statistical methods* (7th edition). Iowa State University Press, Ames.

Thompson G J & Mynhardt L 1986. A potyvirus infecting sweet potatoes. *Phytophylactica* **18**: 46.

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