The Causes and Control of Virus Diseases of Sweetpotato in Developing Countries: Is Sweetpotato Virus Disease the Main Problem?

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Virus diseases can pose a significant constraint to sweetpotato production (Mukibii, 1977; Hahn 1979). As a result of the historical neglect of sweetpotato research in most developing countries, our knowledge of sweetpotato viruses, their importance, distribution, and control are still rather limited. However, recent studies on the occurrence of sweetpotato viruses in a number of countries are helping to clarify our understanding of the global importance of sweetpotato viruses, and to define control strategies. An array of complementary techniques are being used. They include diagnostic methods for the detection of specific viruses, pathogen-tested planting materials for the assessment of yield loss due to viruses, and host plant resistance to virus diseases.

More than 14 virus diseases of sweetpotato have been reported (Moyer and Salazar, 1990; Brunt, et al., 1996 onward). Study of several of these diseases has been hampered by the lack of simple detection techniques. Antisera are now available against a number of sweetpotato viruses including sweetpotato chlorotic fleck virus (SPCFV), sweetpotato latent virus (SPLV), sweetpotato mild mottle virus (SPMMV), C-6, sweetpotato mild speckling virus (SPMSV), sweetpotato feathery mottle virus (SPFMV), and sweetpotato chlorotic stunt virus (SPCSV) previously known as sweetpotato sunken vein virus (SPSVV).

Antisera for all but SPCSV are produced at CIP and are routinely used for nitrocellulose membrane-enzyme-linked immunosorbent assay (NCM-ELISA). Antisera (monoclonal antibodies) specific to SPCSV are produced by J. Vetten at the Institute for Biochemistry and Plant Virology, Braunschweig, Germany (Hoyer et al., 1996). These antisera have been used to survey the incidence of sweetpotato virus diseases at a number of sites.

Sweetpotato virus disease (SPVD) is the name that has become associated with a serious disease of sweetpotato, symptoms of which typically include leaf distortion, chlorosis, discoloration, and stunting. Work initially in West Africa and later in East Africa showed it to be caused by combined infection with aphid-borne SPFMV and whitefly-borne SPCSV (Schaefers and Terry, 1976; Winter et al., 1992; Hoyer et al., 1996; Gibson et al., 1998a) Despite the apparent broad meaning of the name SPVD, the symptoms are so characteristic that the name has become restricted to the disease with these symptoms and caused by these viruses.

The first report of SPVD may have been in eastern Belgian Congo (now DR Congo)

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in 1939. SPVD was first described in East Africa by Sheffield (1953).

SPVD can reduce yields of infected plants by up to 80% (Hahn, 1979; Mukiibi, 1977). It has recently been shown to be the principal virus disease of sweetpotato in East Africa where high disease incidences are often recorded in farmers' fields (Gibson et al., 1998b; Aritua et al., 1998a). In Uganda, disease incidence and severity in farmers' fields have been shown to be closely associated with prevalence of whiteflies (Aritua et al., 1998a, b, c). There are large varietal differences in susceptibility to SPVD. Good sources of resistance are present in local germplasm, but often are associated with later-maturing, lower-yielding genotypes (Aritua et al., 1998b, Carey et al., 1997). SPCSV isolates from eastern and southern Africa have been shown to be serologically different from those from West Africa, Brazil, USA, and Israel (Hoyer et al., 1996; Gibson et al., 1998a). These different isolates may have biological significance inasmuch as cultivars resistant to SPVD in Nigeria were susceptible to SPVD in Uganda (Mwanga et al., 1991).

This paper presents a brief overview of recent collaborative sweetpotato virus research conducted by our project. We start with evidence from surveys conducted in a number of countries using antisera available against sweetpotato viruses, including SPFVM and SPCSV, the components of SPVD. We then assess the effectiveness of the use of planting material free of known pathogens (pathogen-tested) as a virus control measure in China and Uganda. The implications of these results for virus control strategies are discussed.

Materials and Methods

Surveys of sweetpotato viruses were conducted in China, Egypt, Indonesia, Peru, and Uganda. Viruses assayed using antisera and NCM-ELISA kits from CIP were SPCFV, SPLV, SPMMV, C-6, SPMSV and SPFMV. SPCSV was assayed by different means at different sites. In Uganda, triple antibody sandwich-ELISA was used as described by Gibson et al. (1998b). This method used two sets of monoclonal antibodies to discriminate between the East African and West African serotypes of SPCSV. In Indonesia and Peru, SPCSV was detected using NCM-ELISA. In Egypt, SPCSV was detected using polymerase chain reaction (PCR) assay and tests were conducted by Dr. S. Winter, Braunschweig, Germany. Details of sampling procedures at each survey location are given in the references cited in Table 1. In Kenya and Egypt, both symptomless and symptom-expressing plants were sampled. At other sites, only symptom-expressing plants were sampled.

Pathogen-tested planting material for trials in China and Uganda was obtained using standard techniques of thermotherapy and meristem tip culture, followed by indexing to assure virus-free status (Love et al, 1989). Field trials were conducted to compare yields of the pathogen-tested planting material with farm-derived planting material of the same cultivars. In China, trials were conducted during the spring cropping season of 1997 at Xuzhou, Jiangsu Province, and Jinan, Shangdong Province. Five cultivars were used. Trials consisted of 100-plant plots (5 rows of 20 cuttings each) planted in a randomized complete block design (RCBD) with three replications. Trials were harvested 170 d after planting.

In Uganda, three cultivars were used and trials were conducted at three sites: Kachwekano in the southwestern highlands, and Namulonge and Nakabango in the tall grasslands agoecological zone in the south-central part of the country. Trials were conducted at Kachwekano in 1995, and at Namulonge and Nakabango in 1996. Planting material from the 1995 trials was used to plant the 1996 trials. Each trial was planted using a RCBD with three replications. Harvest plot sizes were 16 plants for trials conducted in 1995 and 54 plants for the trials conducted in 1996.

Table 1. Frequencies of detection of sweetpotato viruses from surveys conducted at various sites.

Location	SPCFV	SPLV	SPMMV	C-6	SPMSV	SPFMV	SPCSV	SPFMV+SPCSV	Reference
Uganda (six sites sampled)	5/106	0/106	6/106	na[a]	na	58/106	105/106[b]	57/106	Gibson et al. 1998a
Kenya (four sites sampled)	0/182	6/182	13/182	0/182	na	40/182	na	—	Carey et al., 1998
Cañete, Peru	0/183	13/183	4/183	0/183	12/183	43/183	59/183[b]	34/183	Salazar, 1998
San Ramon, Peru	2/87	0/87	0/87	0/87	2/87	72/87	61/87[b]	59/87	Salazar, 1998
W., C. and E. Java, Indonesia (20 villages)	na	na	na	na	na	256/890	237/890[c]	155/890	Prain et al., 1998
Irian Jaya germplasm collection, Lembang, Indonesia	11/381	8/381	5/381	7/381	5/381	18/381	na	—	Prain et al., 1998
Kafr El Zayat, Egypt (6 cultivars from the Egyptian seed program)	4/382	5/382	6/106	na	na	67/382	+[d]	yes	Abo El-Abbas et al., 1998
Shangdong, China (5 infected farmers' cultivars)	0/115	15/115	0/115	0/115	0/115	93/115	na	—	Zhang and Ma, 1998

[a] na = not assayed.
[b] E. African strain detected by use of monoclonal antibodies.
[c] NCM-ELISA test, SPCSV strain not yet identified.
[d] + signifies detected by PCR, but frequency not quantified.

Results

Results of virus surveys conducted in China, Egypt, Indonesia, Kenya, Peru, and Uganda are presented in Table 1. SPFMV was assayed at all sites and was detected with high frequency. SPCSV was not assayed at all sites, but where assayed, it was detected at high frequencies, similar to SPFMV. In Uganda, 54% of the plants sampled had SPVD, being infected by both SPFMV and SPCSV. At sites in Java, Indonesia, about 65% of the SPCSV-infected samples were also infected with SPFMV. In Egypt, there was also combined infection, but the frequency of SPCSV was not quantified. In Peru, the frequency of combined infection by SPFMV and SPCSV was 34% of the sampled plants. The serotype of SPCSV was only determined during surveys in Uganda and Peru. In both countries, the so-called East African serotype was detected, not the West African serotype. The remaining viruses, except for C-6, which was only detected in the Irian Jaya germplasm collection at Lembang, Indonesia, were generally detected at a low frequency but on each of the continents surveyed.

Yield loss assessment trials in China (Table 2) demonstrated a significant benefit to using pathogen-tested planting material at both Jinan and Xuzhou. The effect of using pathogen-tested planting material was greater at Jinan, with a significantly higher yield from pathogen-tested planting material than from farm-derived material for each cultivar. At Xuzhou the pathogen-tested planting material only yielded significantly more for two of the cultivars evaluated. However, the mean yield from pathogen-tested planting material was consistently higher than from farm-derived material.

In the Ugandan trials (Table 3), no significant effect of using pathogen-tested planting material was detected. The mean yield of pathogen-tested planting material was not consistently higher. However, in three of four trials, the mean yield from pathogen-tested cultivar Tanzania was higher than the yield from farm-derived planting material. Tanzania was also the highest yielding cultivar in three out of four trials.

Discussion

The finding that SPFMV was predominant at each site sampled is in line with previous information about the cosmopolitan distribution of this virus. In the countries where it was assessed—Egypt, Indonesia, Peru, and Uganda—SPCSV was present in as high a proportion of virus symptom-expressing plants as SPFMV, often in association with the latter. This appears to indicate a broader distribution and importance of SPVD than has previously been reported. The detection of the East African strain of SPCSV as the predominant form in Peru also indicates a wider distribution of this serotype than was previously reported (Hoyer et al., 1996). Preliminary results have indicated the presence of SPCSV in China (L. Salazar, CIP, Lima, Peru, 1999, pers. comm.).

The results of the present study indicate that SPVD may be the most important virus disease of sweetpotato globally. Until now, its global impact has been overlooked. That is largely because diagnostic tools have not been readily available for SPCSV, so only SPFMV was ubiquitously detected. Further survey work is required to confirm the occurrence and prevalence of SPVD and its causal agents (SPFMV and SPCSV) around the world. Further work is also required to clarify the biological significance of different serotypes of SPCSV. Because of a lack of antisera against a number of additional sweetpotato viruses reported in the literature, these have not yet been widely surveyed. One or more of them could also be important.

Results from China of yield comparisons of sweetpotato varieties using pathogen-tested and farm-derived planting materials (Table 2) indicate the large yield gains that can be made using this technique. Indeed,

Table 2. Fresh storage root yields (t/ha) of five major sweetpotato cultivars grown from pathogen-tested and farm-derived planting materials at two sites in China in 1997.

Cultivar	Planting material source[a]	Yield and difference (%) between planting material sources for each cultivar[b]	
		Jinan	Xuzhou
Xushu 18	PT	43.1 (36%**)	41.5 (22% ns)
	F	31.6	34.0
Lashu No. 7	PT	46.4 (35%**)	39.0 (17% ns)
	F	34.6	33.3
Lushu No. 8	PT	39.9 (23%**)	41.6 (48% ns)
	F	32.4	28.0
Beijing 553	PT	39.4 (47%**)	39.0 (64%*)
	F	26.9	23.8
Fenghsoubai	PT	37.7 (75%**)	45.7 (92%*)
	F	21.5	23.8

[a] PT = Pathogen-tested, F = Farm-derived.
[b] Significant according to LSD (0.01) = ** or LSD (0.05) = *, not significant = ns.

Table 3. Fresh storage root yields (t/ha) of three sweetpotato cultivars grown from pathogen-tested and farm-derived planting materials at three sites in Uganda during two seasons.

Cultivar	Planting material source[a]	Yield and difference (%) between planting material sources for each cultivar[b]			
		Kachwekano	Nakabango		Namulonge
		(1995)	(1995)	(1996)	(1995)
Tanzania	PT	15.0 (19%)	39.8 (39%)	11.5 (-14%)	13.8 (50%)
	F	12.6	28.7	13.4	9.2
Bwanjule	PT	5.9 (-40%)	28.2 (28%)	19.3 (7%)	11.0 (29%)
	F	9.9	22.1	18.0	8.5
Wagabolige	PT	6.3 (-40%)	20.8 (-13%)	20.4 (16%)	9.4 (-8%)
	F	10.5	23.8	17.6	10.2

[a] PT = Pathogen-tested; F = Farm-derived, symptomless.
[b] No significant effect of planting material source was detected.

in Shangdong, the use of pathogen-tested planting material has been widely adopted in the past few years and extended to all parts of the Province (Fuglie et al., 1999). There has been a very high payoff to this research and extension effort. Increased production has supplied the increasing demand for sweetpotato as an industrial and feed source, while farmers' production costs have actually been reduced due to improved seed health. Following initial investment of public money in the clean-seed system, the costs of operation of the seed system are borne by the farmers who

purchase the planting material, resulting in an economically viable business. The experience in Shangdong is being extended to other provinces in China, and may continue to have a large impact on sweetpotato production there. A successful clean planting material program has also been established in Egypt, made highly effective by the high virus pressure there and the susceptibility to viruses of the major commercial cultivars (Abo EL-Abbas et al., 1998).

Results of our assessment of the potential for using pathogen-tested planting material in Uganda were in marked contrast to the results from China. In Uganda, no benefit to using pathogen-tested planting material was demonstrated. That was probably due to the relatively high levels of virus resistance in the landrace cultivars that we used for our test in Uganda. Also, we followed the normal farming practice of using only symptomless (essentially healthy) plants as sources of planting material (Gibson et al., 1997).

Had we chosen more susceptible genotypes for our comparison in Uganda, or had we chosen to use symptom-expressing farm-derived planting material for our experiments, it is likely that we would have detected differences. Indeed, for cultivar Tanzania, the most virus-susceptible of the genotypes evaluated (rated moderately resistant by Mwanga et al. (1995)), there was a tendency for pathogen-tested planting material to yield higher than farm-derived planting material. We will continue to investigate the potential for using pathogen-tested planting material in Uganda and elsewhere in eastern and southern Africa, particularly for more virus-susceptible but high yielding cultivars. It should be noted, however, that sweetpotato production systems in Sub-Saharan Africa are less commercially oriented than systems in China and elsewhere, with sweetpotato primarily a food security crop for low-income farmers. That might pose a constraint to the widespread implementation of pathogen-tested planting material schemes as viable enterprises.

Given the relatively unpromising prospects for pathogen-tested seed schemes in Uganda, and the high levels of resistance to SPVD that exist in the African germplasm (Carey et al., 1997), breeding resistant varieties will continue to be a principal component of SPVD control here. As already mentioned, there is a tendency for local farmers' cultivars with high levels of resistance to be rather low yielding and late maturing compared with earlier maturing, high yielding, yet susceptible local cultivars or exotic introductions (Aritua et al., 1998b; Carey et al., 1997). The higher mean yield of the more virus susceptible cultivar Tanzania compared with the other two cultivars in our trial is in line with this observation.

A principal aim of the sweetpotato breeding program at Namulonge, Uganda, where the Ugandan program and CIP are concentrating breeding efforts, is to combine SPVD resistance with desirable agronomic traits such as yield, earliness, and acceptable culinary quality. Considerable progress has already been made, and the program has released 12 cultivars combining good levels of field resistance with high yields and other desirable traits (Mwanga et al., 1995; Turyamureeba et al., 1998). However, improvements in breeding efficiency are required, including the development of screening techniques to rapidly identify resistant genotypes. Currently, very large progeny populations (as many as 100,000 seedlings) are required to identify a handful of resistant, agronomically superior genotypes. The inheritance of host plant resistance is under study. It is anticipated that considerable progress will soon be made in breeding for resistance.

The international value of resistance breeding will depend on the degree to which resistant genotypes from one site maintain their resistance at other sites. Recent evidence of the widespread global

occurrence of SPVD leads us to anticipate that resistance from Africa may hold up in other environments where viruses are a problem. However, differences in viruses or virus strains between sites could cause resistant genotypes from one site to be susceptible in others. International studies are underway to investigate this, using standard sets of genotypes with various levels of resistance to SPVD.

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