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Identification and distribution of viruses infecting sweet potato in Kenya

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Summary

Four hundred and forty-eight symptomatic and 638 asymptomatic samples were collected from sweet potato fields throughout Kenya and analysed serologically using antibodies to *Sweet potato feathery mottle virus* (SPFMV), *Sweet potato chlorotic stunt virus* (SPCSV), *Sweet potato mild mottle virus* (SPMMV), *Cucumber mosaic virus* (CMV), Sweet potato chlorotic fleck virus (SPCFV), *Sweet potato latent virus* (SwPLV), Sweet potato caulimo-like virus (SPCaLV), Sweet potato mild speckling virus (SPMSV) and C-6 virus in enzyme-linked immunosorbent assays (ELISA). Only SPFMV, SPMMV, SPCSV, and SPCFV were detected. Ninety-two percent and 25% of the symptomatic and asymptomatic plants respectively tested positive for at least one of these viruses. Virus-infected plants were collected from 89% of the fields. SPFMV was the most common and the most widespread, detected in 74% of the symptomatic plants and 86% of fields surveyed. SPCSV was also very common, being detected in 38% of the symptomatic plants and in 50% of the fields surveyed. SPMMV and SPCFV were detected in only 11% and 3% of the symptomatic plant samples respectively. Eight different combinations of these four viruses were found in individual plants. The combination SPFMV and SPCSV was the most common, observed in 22% of symptomatic plants. Virus combinations were rare in the asymptomatic plants tested. Incidence of virus infection was highest (18%) in Kisii district of Nyanza province and lowest (1%) in Kilifi and Malindi districts of Coast province.

Key words: Sweet potato feathery mottle virus, survey, mixed infection, symptom, incidence

Introduction

Sweet potato (*Ipomoea batatas* L.) is an important food crop in Kenya, the fifth largest producer in Africa after Uganda, Nigeria, Rwanda and Burundi (Anon., 2002). The main sweet potato production areas in Kenya include Western, Nyanza, Central, Coast and Eastern provinces with about 75% of total production concentrated at mid altitudes (1000-1600 m) (Ndolo *et al.,* 1997). The crop is grown largely by smallholder farmers (Horton, 1988; Carey *et al.*, 1996). Although the area under sweet potato production in Kenya has increased over the years (Matin, 1999), yields, currently estimated at 8.2 t ha^{-1}, have declined over the last 5 yr (Anon., 2002) and are far below the crop's production potential, mainly due to pests and diseases (Ndolo *et al.,* 1997; Matin, 1999).

Viruses are the second most important biotic constraint [after insects (weevils)] of sweet potato production both in Africa (Geddes, 1990) and worldwide (Jannson & Raman, 1991), unaffected controls often yielding > 50% more than infected plants (Hahn, 1979; Ngeve & Bouwkamp, 1991; Milgram *et al.,* 1996; Gutiérrez *et al.,* 2003). Viruses reported to infect sweet potato in Africa include *Sweet potato feathery mottle virus* (SPFMV), *Sweet potato chlorotic stunt virus* (SPCSV), *Sweet potato mild mottle virus* (SPMMV), *Cucumber mosaic virus* (CMV), Sweet potato chlorotic fleck virus (SPCFV), *Sweet potato latent virus* (SwPLV) and Sweet potato caulimo-like virus (SPCaLV) (Mukiibi, 1977; Hahn, 1979; Geddes, 1990; Wambugu, 1991; Gibson *et al.,* 1998). Often, infection of sweet potato by two or more viruses leads to greater damage than by each virus alone. Sweet potato virus disease (SPVD) is the most damaging disease of sweet potato in many parts of Africa, in particular in East Africa (Geddes, 1990) and is caused by dual infection of sweet potato with SPCSV and SPFMV (Schaefers & Terry, 1976; Gibson *et al.,* 1998). Most sweet potato cultivars infected with SPFMV or SPCSV alone are respectively either symptomless or have only moderate plant stunting and purpling or chlorosis

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of middle and bottom leaves. Symptoms in plants affected by SPVD are, however, much more severe than those in plants infected with either virus alone (Gibson, *et al.,* 1997, 1998), SPCSV synergising the multiplication of SPFMV (Karyeija *et al.,* 2000), leading to production losses of > 90% (Gibson *et al.,* 1998; Karyeija *et al.,* 1998).

Knowledge on the distribution of different sweet potato viruses in Kenya is still limited though this is essential for crop protection. There have been only two previously reported surveys and, whereas the second survey (Carey *et al.,* 1996) analysed relatively few samples, the first one (Wambugu, 1991) was conducted about a decade ago. Since then, changes in farming practices and population dynamics of virus vectors may have changed virus incidence (Wisler *et al.,* 1998). In addition, antisera to several sweet potato viruses, some previously unrecognised, have now become available. More emphasis also needs to be directed at the co-occurrence of viruses in sweet potato. Here we report on the identity and relative importance of viruses infecting sweet potato in all the major sweet potato growing regions of Kenya. The occurrence and distribution of multiple virus combinations are also presented.

Materials and Methods

A survey was conducted in the five major sweet potato-growing areas in Kenya, namely Western, Nyanza, Central, Coast and Eastern provinces (Fig. 1). Three- to 5-month-old sweet potato crops in 125 fields were surveyed in 16 districts between January and October 2001. Sweet potato fields in each growing area were inspected and sampled at approximately 5-km intervals while traveling along rural roads. The number of plants showing virus-like symptoms amongst 50 plants was recorded along X-shaped transects stretching between opposing corners of each field. A total of 448 symptomatic and 638 symptomless sweet potato cuttings were collected, established in an insect-proof screenhouse at the University of Nairobi and assayed by enzyme-linked immunosorbent assay (ELISA). SPFMV, SPMMV, SwPLV, SPMSV,

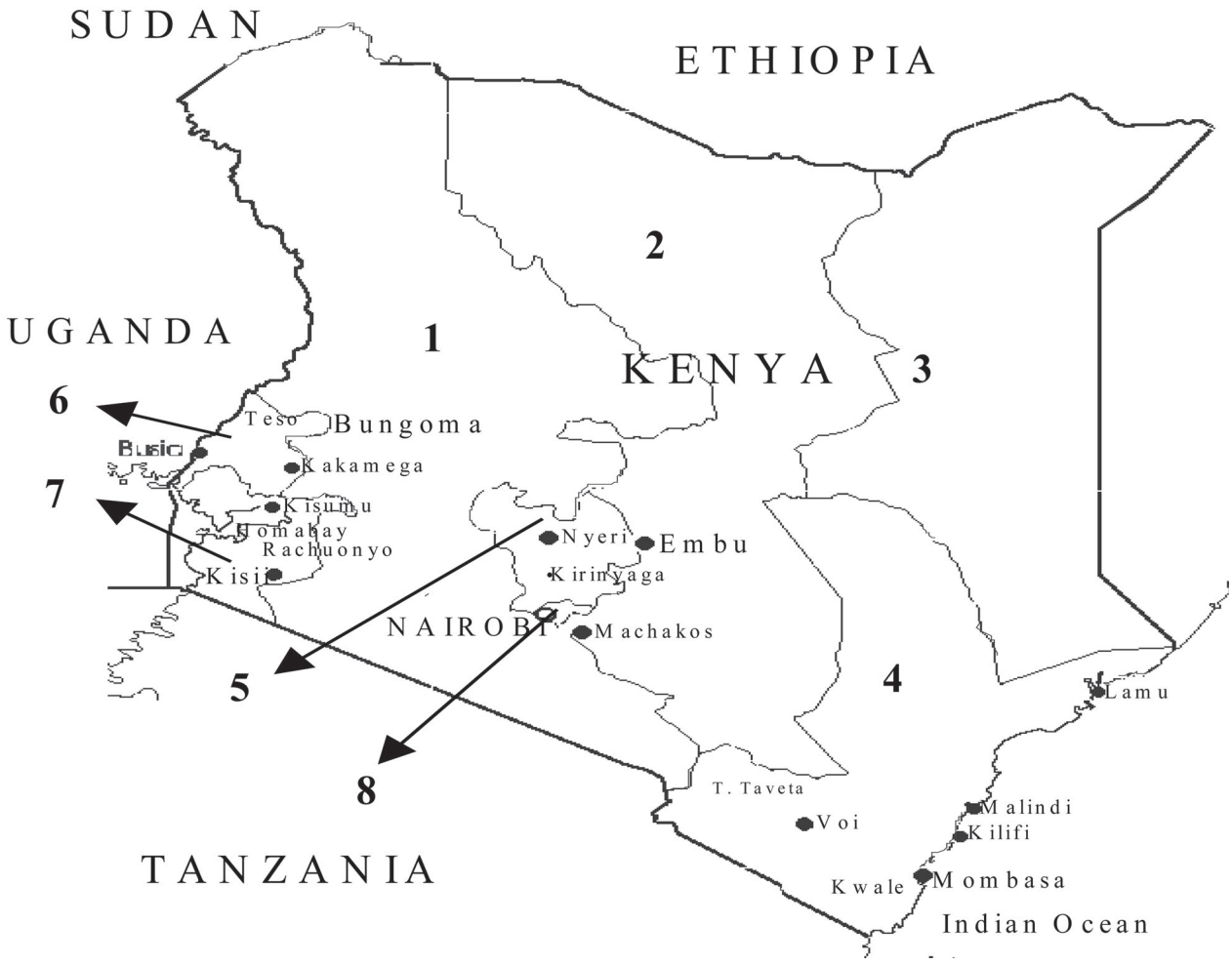

Fig. 1. Map of Kenya showing the provinces and the location of the surveyed districts and sweet potato production areas sampled. The Kenyan provinces are: 1 = Rift Valley, 2 = Eastern, 3 = North Eastern, 4 = Coast, 5 = Central, 6 = Western, 7 = Nyanza, 8 = Nairobi.

SPCaLV, SPCFV and C-6 virus were tested for by nitrocellulose membrane (NCM)-ELISA (Anon., 2001), utilising standard kits obtained from the International Potato Center (CIP). Two leaves (top and bottom) from each rooted cutting were tested by ELISA. The kits contained positive and healthy control sap pre-spotted on membrane strips. Positive and negative reactions were visually assessed, the degree of purple coloration determining those regarded as positive. Detection of SPCSV and CMV was by triple antibody sandwich (TAS)-ELISA in microtitre plates as described previously (Gibson *et al.,* 1998). For SPCSV, monoclonal antibodies which distinguish the serotype originally described from East Africa ($SPCSV_{EA}$; Mab mix 1) and the serotype originally described from West Africa ($SPCSV_{WA}$; Mab mix 2) were used (Vetten *et al.,* 1996; Gibson *et al.,* 1998). The coating antisera and detecting monoclonal antibodies to SPCSV and CMV were from the stock of BBA, Braunschweig, Germany. In TAS-ELISA, absorbancies were recorded at 405 nm (A_{405}) after 1 h substrate incubation using a microplate reader (Humareader Model 2106, Germany). Readings at least twice the values of the negative controls were considered positive. Samples of sweet potato that did not react with antisera to these viruses were re-indexed by grafting onto *Ipomoea setosa,* the universal indicator plant for sweet potato viruses. Symptoms were recorded and leaves of both symptomatic and asymptomic *I. setosa* were assayed serologically 4 wk after grafting.

Results

Virus disease incidence

Eighty-three percent (372) of the symptom-bearing plants reacted with antisera to at least one virus (Table 1). Sixty-five of those that tested negative were grafted onto *I. setosa* and, of these, 38 were positive to at least one of the viruses. Overall, 410 (92%) symptomatic plants tested positive to at least one virus. Only 28 (4%) the 638 asymptomatic plants reacted directly with any of the antisera. After grafting those that were negative, a further 133 plants

Table 1. *Number of diseased and asymptomatic sweet potato samples that tested positive for at least one virus when serologically assayed directly or following graft inoculation onto* I. setosa

		Samples from symptomatic plants			Samples from symptomless plants		
		Number of	Number testing positive for at least one virus†:		Number of	Number testing positive for at least one virus†:	
Province	District	plants tested	by ELISA	by grafting	plants tested	by ELISA	by grafting
Western	Kakamega	50	41/50	4/4	46	2/46	13/44
	Bungoma	61	54/61	1/6	42	0/42	11/42
	Teso	12	9/12	3/3	20	2/20	6/17
	Busia	36	36/36	0/0	22	1/22	5/20
	Total	159	140/159	8/13	130	5/130	35/123
Nyanza	Rachuonyo	35	34/35	1/1	34	6/34	3/28
	Kisii	55	53/55	0/2	34	6/34	5/26
	Homa bay	43	40/43	0/4	36	2/36	8/34
	Kisumu	35	31/35	2/3	31	1/31	3/30
	Total	168	158/168	3/10	135	15/135	19/118
Central	Nyeri	11	5/11	4/5	48	2/48	19/46
	Kirinyaga	29	12/29	12/14	46	2/46	10/45
	Total	40	17/40	16/19	94	4/94	29/91
Eastern	Machakos	16	9/16	4/7	97	4/97	19/93
	Embu	6	0/6	4/4	57	0/57	15/57
	Total	22	9/22	8/11	154	4/154	34/150
Coast	Kilifi	5	0/5	0/5	18	0/18	2/18
	Malindi	2	0/2	2/2	29	0/29	2/29
	Kwale	39	35/39	1/3	44	0/44	4/44
	Taita Taveta	13	13/13	0/0	34	0/34	8/34
	Total	59	48/59	3/10	125	0/125	16/125
Total		448	372/448	38/65	638	28/638	133/607

† Surviving diseased and asymptomatic plants in which no virus was detected by ELISA were grafted to *I. setosa* for re-indexing

tested positive for at least one of the viruses but mainly SPFMV. Thus, 161 (25%) of the asymptomatic plants tested were infected.

Viral diseases were common in most of the provinces surveyed though there were considerable differences in incidence between the districts surveyed and even among fields within a district. In terms of provinces, incidence was highest in Nyanza and the closely adjacent Western province and lowest in the Eastern province (Table 2). The highest mean disease incidence (18%) in a district was observed in Kisii of Nyanza province (Table 2) and the lowest (1%) in Kilifi and Malindi districts of Coast province. The highest incidence of viral diseases in an individual field (48%) was observed in Kirinyaga district of Central province in a field in which sweet potato had been cultivated for several consecutive seasons. The visually-assessed incidences were consistently lower than those based on serological assays (Table 2), their closely correlation ($P = 0.05$; $r = 0.9$) (Fig. 2) indicating a fairly constant presence of some latently infected plants. However, there were also several instances of symptomatic sweet potato plants in which no virus could be detected even after grafting onto *I. setosa*. There was no significant ($P > 0.05$) correlation between virus disease incidence and altitude, mean rainfall or mean temperature.

Table 2. *The mean incidence (%) of virus diseases in surveyed sweet potato crops as assessed visually and serologically*

Province/ District	Number of fields surveyed	Incidence (%) of virus-infected plants based on Visual assessment	Serological detection
Western			
Kakamega	10	9 (0-32)*	13 (0-38)
Bungoma	10	16 (0-40)	17 (4-42)
Teso	5	4 (0-12)	8 (0-20)
Busia	5	11 (0-18)	15 (6-22)
Mean		10	13
Nyanza			
Rachuonyo	7	12 (0-38)	14 (0-42)
Kisii	7	18 (6-24)	18 (6-22)
Homa bay	7	13 (4-22)	15 (2-26)
Kisumu	7	10 (2-20)	10 (4-18)
Mean		13	14
Central			
Nyeri	8	2 (0-8)	6 (2-12)
Kirinyaga	10	14 (0-48)	16 (0-48)
Mean		8	11
Eastern			
Machakos	15	4 (0-20)	4 (0-12)
Embu	11	3 (0-8)	4 (0-8)
Mean		4	4
Coast			
Kilifi	4	2 (0-4)	1 (0-2)
Malindi	6	1 (0-4)	1 (0-4)
Kwale	7	15 (4-30)	13 (2-30)
Taita Taveta	6	4 (0-16)	4 (0-16)
Mean		6	6

*Figures in parentheses give the incidence range (in %)

Viruses detected

Four viruses, namely SPFMV, SPCSV, SPMMV, and SPCFV, were detected in samples from 448 symptom-bearing and asymptomatic sweet potato plants collected from the five provinces surveyed (Table 3). As already shown in Table 1, the majority (92%) of the symptomatic samples reacted with antisera to at least one of the viruses. SPFMV was detected in samples from all areas surveyed. A total of 334 (75%) and 123 (19%) symptomatic and asymptomatic samples, respectively, reacted with the SPFMV antibodies, making SPFMV the most frequently detected virus. SPCSV was the second most frequently detected virus, detected always as $SPCSV_{EA}$, in 39% of the symptomatic plants and 3% of the asymptomatic plants. Most of the SPCSV-infected plants exhibited distinct virus symptoms consistent with it being the most severe sweet potato-infecting virus. SPCSV was not detected in samples collected from Embu, Kilifi and Malindi districts. SPMMV was detected in 10% of symptomatic and 5% of asymptomatic plants but was not detected in plant samples collected from Malindi and Kilifi districts. SPCFV was the rarest virus, though detected in all provinces.

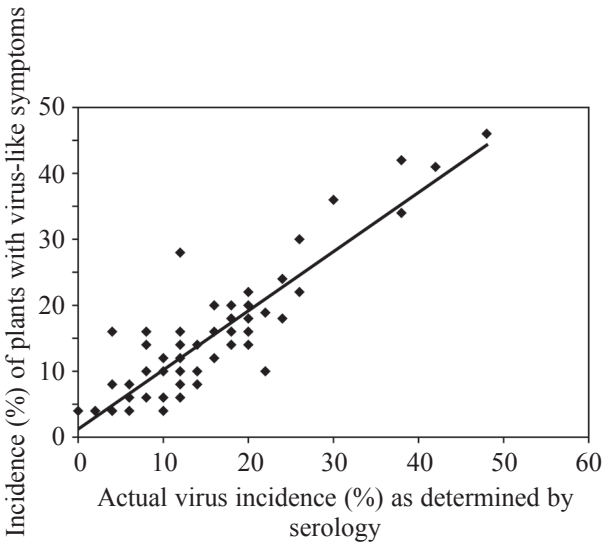

Fig. 2. Graph showing the close relationship between the incidence (%) of plants in each field showing virus-like symptoms and their incidence as estimated by ELISA.

Single and mixed virus infections

A high proportion (34%) of symptomatic samples was infected with two or more viruses whereas only 2% of asymptomatic samples were. By contrast, high proportions of both symptomatic (58%) and asymptomatic (23%) samples were infected by a single virus (Fig. 3). SPFMV was the most common single infection in both symptomatic (42%) and asymptomatic plants (17%). SPCSV, the second most commonly detected virus, singly infected 14% of symptomatic plants but only 2% of asymptomatic plants (Fig. 3). Virtually all plants infected with SPCSV + SPFMV were diseased, whereas a high proportion of plants infected with SPFMV, SPMMV or SPCFV, either singly or in combination, had no obvious symptom when inspected in the field. Indeed, SPMMV was unusual in that more single infections of it were detected in asymptomatic than in symptomatic samples (Fig. 3).

Seven different combinations of viruses were detected in the symptomatic plants tested but only four different virus complexes were detected in the asymptomatic plants (Fig. 3). SPFMV + SPCSV (= SPVD) was the most common combination, occurring in 22% of the symptomatic samples (Fig. 3), though only in a single asymptomatic sample. SPVD was most frequent in samples from Western and Nyanza provinces though also present in Coast, Central and Eastern provinces. SPFMV + SPMMV was the second most prevalent dual infection in symptomatic plants, detected in 7% of samples, and was the most prevalent dual infection in asymptomatic plants, detected in 3% of plants. Indeed, SPMMV was overall detected more often in combination with SPFMV than by itself. Triple infections were observed in only eight plants and a mixed infection involving four viruses (SPFMV, SPCFV, SPMMV and SPCFV) was detected in only one symptomatic plant collected from Homa bay district, Nyanza province.

The correlation between the number of plants infected with SPCSV and SPFMV was highly significant ($P = 0.01$; $r = 0.8$). Similarly, the correlation between the number of plants infected with SPFMV and SPMMV was also significant ($P = 0.01$; $r = 0.6$). No significant correlation was observed between the number of plants infected with (i) SPFMV and SPCFV, (ii) SPCSV and SPMMV, (iii) SPCSV and SPCFV, and (iv) SPMMV and SPCFV.

Distribution and relative importance of individual viruses

SPFMV was the commonest virus infecting sweet potato in all the provinces surveyed, detected in plants collected from 86% of all fields sampled and in all districts (Fig. 4). SPCSV was the second most common virus, detected in plants collected from 50%

Table 3. *Number and types of virus infections of the symptomatic (S) and asymptomatic (A) plants collected in five provinces of Kenya*

		No. of samples		SPFMV		SPCSV		SPMMV		SPCFV	
Province	District	S	A	S	A	S	A	S	A	S	A
Western	Kakagema	50	46	33 (66)*	13 (28)	20 (40)	1 (2)	5 (10)	3 (7)	1 (2)	0 (0)
	Bungoma	61	42	43 (70)	9 (21)	29 (48)	1 (2)	9 (15)	2 (5)	0 (0)	0 (0)
	Teso	12	20	9 (75)	3 (25)	4 (33)	0 (0)	3 (25)	5 (25)	0 (0)	1 (5)
	Busia	36	22	27 (75)	3 (14)	27 (75)	0 (0)	6 (17)	4 (18)	0 (0)	0 (0)
Nyanza	Rachuonyo	35	34	27 (77)	5 (15)	9 (26)	4 (12)	7 (20)	0 (0)	0 (0)	0 (0)
	Kisii	55	34	49 (89)	8 (24)	18 (33)	2 (6)	0 (0)	1 (3)	0 (0)	0 (0)
	Homa bay	43	36	29 (67)	7 (19)	16 (37)	0 (0)	6 (14)	3 (8)	4 (9)	0 (0)
	Kisumu	35	31	23 (66)	1 (3)	15 (43)	1 (3)	8 (23)	3 (10)	2 (6)	0 (0)
Central	Nyeri	11	48	8 (73)	19 (40)	3 (27)	0 (0)	0 (0)	2 (4)	2 (18)	1 (4)
	Kirinyaga	29	46	23 (79)	12 (26)	6 (21)	1 (2)	0 (0)	1 (2)	0 (0)	0 (0)
Eastern	Machakos	16	97	12 (75)	15 (15)	2 (13)	6 (6)	1 (6)	3 (3)	1 (6)	0 (0)
	Embu	6	57	4 (67)	13 (23)	0 (0)	0 (0)	0 (0)	4 (7)	0 (0)	0 (0)
Coast	Kilifi	5	18	0 (0)	2 (11)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
	Malindi	2	29	2 (100)	2 (7)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
	Kwale	39	44	33 (85)	4 (10)	20 (51)	0 (0)	1 (3)	0 (0)	0 (0)	0 (0)
	Taita Taveta	13	34	12 (92)	7 (21)	4 (31)	0 (0)	1 (8)	1 (3)	4 (31)	2 (6)
Total		448	638	334	123	173	16	47	32	14	4

* Figures in parentheses are percentages

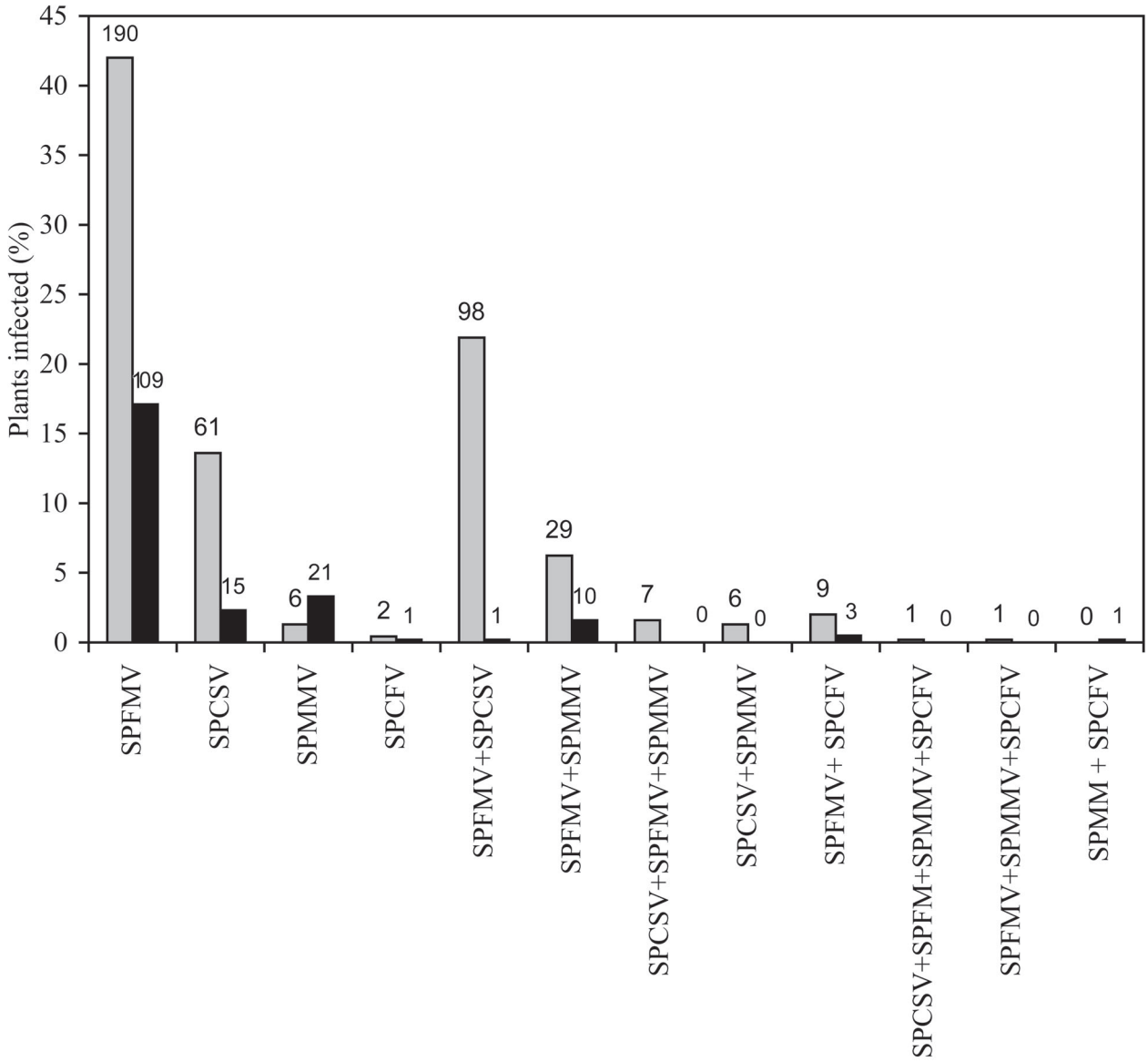

Fig. 3. Proportion (%) of single and multiple infections of different viruses in symptomatic and asymptomatic sweet potato plants collected from 16 districts of Kenya. The numbers above each bar indicate the number of plants infected with single or multiple viruses. Grey bars = diseased; black bars = symptomless.

of the fields in 13 of the 16 districts. Although SPMMV and SPCFV seemed similarly widespread, being detected in samples collected from all the five provinces surveyed, these two viruses were rarer, detected in samples collected from only 36% and 10% of the fields, respectively. SPMMV was most prevalent in Western province, especially in Teso and Busia districts. Of the 125 fields inspected, 112 (90%) had sweet potato plants infected by at least one virus.

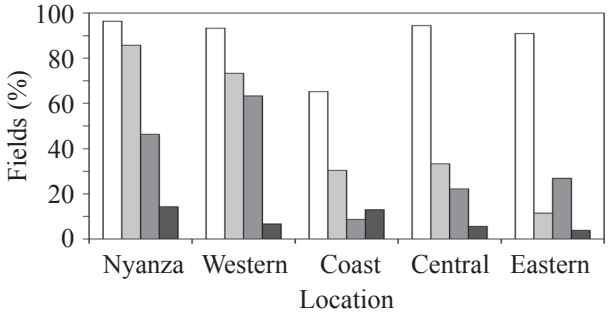

Fig. 4. Proportion (%) of fields with plants infected with SPFMV, SPCSV, SPMMV and SPCFV in five provinces of Kenya. Unshaded bars = SPFMV; light grey bars = SPCSV; dark grey bars = SPMMV; black bars = SPCFV.

Discussion

Four viruses, SPFMV, SPCSV, SPMMV and SPCFV were detected in a comprehensive survey of both symptomatic and asymptomatic sweet potato plants collected in farmers' fields in the major growing areas in Kenya. SPFMV was the commonest and was widely distributed, consistent with previous reports that SPFMV occurs wherever sweet potato is grown (Moyer & Cali, 1985; Moyer

& Salazar, 1989; Sakai *et al.,* 1997; Colinet *et al.,* 1998). SPCSV was the second most prevalent virus. All the SPCSV-positive samples reacted only with monoclonal antibodies specific to the East African serotype, indicating that, as in Uganda (Gibson *et al.,* 1998), only the East African serotype occurs in Kenya. SPMMV was the third most common virus. Although an earlier survey (Carey *et al.,* 1996) failed to detect SPCFV in Kenya, our data indicate a low incidence of this virus. Our survey was the first in Kenya to include SPMSV antibodies but no sample reacted with antiserum to this virus and so far, SPMSV seems to be geographically confined to Argentina or the Americas (Alvarez *et al.,* 1997). CMV and SwPLV were not detected although an earlier survey (Wambugu, 1991) indicated that these viruses were common in East Africa. In Uganda, the same range of viruses, namely SPFMV, SPCSV, SPMMV and SPCFV, was detected in a similar survey (Mukasa *et al.,* 2003). Also as Uganda, SPFMV and SPCSV often occurred together to produce the severe disease SPVD (Gibson *et al*., 1998; Mukasa *et al.,* 2003).

Virus incidences were overall highest in Nyanza and Western provinces, consistent with a survey undertaken several decades earlier (Sheffield, 1953). In their relatively high rainfall and warm climate, farmers can grow sweet potato throughout the year, ensuring a continuous availability of host plants for the aphid and whitefly vectors of SPFMV, SPCSV and SPMMV whilst overlap of old and newly-planted crops allows easy transfer of virus inoculum between cropping cycles. Disease incidence was low in Eastern, Central and Coast provinces probably because the crop is less widely grown due to unfavourable climatic conditions. Furthermore, most herbaceous crops including sweet potato lose their leaves or otherwise die back during the long and intense dry season, particularly in Kilifi and Malindi districts, so providing a break in the food supply of the virus vectors. Several of the varieties grown in these areas, for instance, cvs Ex-Shimba Hills, Kemb 10, Ex-Diani, Muibai and Mtwapa 8, were found to be infected in other locations, suggesting that the low infection rates in these provinces was due to low numbers of the insect vectors and virus source plants rather than the growing of especially virus-resistant sweet potato varieties. Data of Sheffield (1953) suggested that virus incidence is negatively correlated with altitude. In our study there was no significant correlation between virus incidence and altitude or temperature as there was less SPVD in both the cooler locations highlands of Nyeri, Embu and Kirinyaga districts of central Kenya and the low-lying, hotter coastal locations.

It was confirmed that SPFMV on its own causes mild or no symptoms in East African sweet potato cultivars (Gibson *et al.,* 1997), a substantial proportion of samples from asymptomatic plants reacting with antiserum to SPFMV. In such sweet potato samples, SPFMV generally occurred at concentrations too low to be detected by ELISA (Aritua *et al.,* 1998; Karyeija *et al.,* 2000) and was detected serologically only in *I. setosa* following graft inoculation. The higher incidence and wider distribution of SPFMV as compared to the other three viruses could be due to the relative abundance of its aphid vectors over the whitefly vectors of SPCSV (Schaefers & Terry, 1976) and SPMMV (Hollings *et al.*, 1976) and the (unknown) vector(s) of SPCFV. An alternative/additional explanation might be that because sweet potato singly infected with SPFMV exhibit no symptoms, farmers inadvertently select symptomless SPFMV-infected cuttings as planting material for the next crop (Ateka *et al.,* 2002), so maintaining this virus. However, the much rarer SPMMV and SPCFV also commonly infected some sweet potato latently. Indeed, about three-quarters of the asymptomatic plants were virus-free, consistent with many East African sweet potato cultivars possessing resistance capable of limiting the multiplication and eliminating viruses not only SPFMV (Gibson *et al.,* 1997; Karyeija *et al.,* 1998) but perhaps also these other sometimes latently-infecting viruses.

Most combinations of viruses resulted in plants showing virus-like symptoms. The observation that SPVD, the disease associated with SPCSV's synergistic effect on SPFMV (Schaefers & Terry, 1976; Gibson *et al.,* 1998; Karyeija *et al.,* 2000; Gibson & Aritua, 2002), was severe and is widely distributed in Kenya, supports SPVD being the most serious disease of sweet potato in Africa (Geddes, 1990). The correlation between the proportion of plants infected by SPCSV and those infected by SPFMV was highly significant, consistent with previous reports from neighbouring Uganda (Mukasa *et al.,* 2003). Since SPCSV is transmitted by whiteflies and SPFMV by aphids the cause of this association is not immediately obvious, despite the synergism of SPFMV by SPCSV in mixed infections. Possible explanations are:

- Since the titre of SPFMV increases markedly when plants are co-infected with SPCSV (Karyeija *et al*., 2000), SPFMV becomes more easily detected leading to an apparent rather than a real association.
- The inoculation of sweet potato with SPFMV by viruliferous aphids results in a higher transmission rate in plants pre-infected with SPSCV than in plants uninfected by SPCSV.
- Some sweet potato cultivars have an inherent resistance mechanism, which enables them to eliminate an infection by SPFMV alone (Aritua *et al.,* 1998). This would lead to a high proportion of plants infected with SPFMV + SPCSV than can be attributed to chance.

Our current data fail to distinguish these possible explanations. SPCSV was not linked with any other virus, not even with SPMMV with which SPCSV shares its whitefly vector (Hollings et al., 1976; Schaefers & Terry, 1976). Instead, SPMMV occurred more frequently in mixed infection with SPFMV than either by itself or mixed with any other virus: similar results have also been obtained in Uganda (Mukasa *et al*., 2003). Both SPFMV and SPMMV are members of the *Potyviridae*, though belonging to different genera, and may benefit each other, perhaps through some shared function. Further investigations need to be conducted to understand the epidemiological and economic significance of mixed infections in sweet potato.

No virus was detected in about 8% of the apparently virus-diseased sweet potato plants, even following grafting to *I. setosa*. The significance of these observations is currently unclear as they may indicate either genetic abnormalties in the plants or the presence of unknown virus(es).

This study describes the identity and relative importance of viruses and virus complexes infecting sweet potato in Kenya. The determination of the areas with high virus disease incidences indicates where resistance breeding and other control strategies are urgently needed**.** Since SPFMV and SPCSV were the most commonly detected viruses and are known to interact synergistically (Gibson *et al.,* 1998; Karyeija *et al.,* 2000), future resistance breeding including pathogen derived resistance should focus on these two viruses as a priority.

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