R. F. Karyeija
Kawanda Agricultural Research Institute, Kampala, Uganda

R. W. Gibson
Namulonge Agricultural and Animal Production Research Institute (NAARI), Kampala, Uganda

J. P. T. Valkonen
University of Helsinki, Finland
Swedish University of Agricultural Sciences, Uppsala, Sweden

The Significance of Sweet Potato Feathery Mottle Virus in Subsistence Sweet Potato Production in Africa

Sweet potato (*Ipomoea batatas* L.; Convolvulaceae (=Morning Glory)), with an annual world production of 122 million metric tons (21), ranks fourth in importance in the developing world after rice, wheat, and corn (maize). The bulk of production is in China, which produces 102 million metric tons annually (21). Sweet potato originated from Central and/or South America. The International Potato Center (CIP) in Lima, Peru, has the international mandate for research on sweet potatoes in developing countries.

Production of sweet potato in Africa, 7.5 million metric tons, amounts to 6% of world production. About 75% of sweet potato production is in East Africa, mainly around Lake Victoria (Fig. 1). Uganda, the largest African producer and the third largest producer in the world (21), grows approximately 2.2 million metric tons, equivalent to the combined production of the Americas. Countries neighboring Uganda also rank high in production, and Rwanda has the greatest per capita production. However, during periods of hardship in Africa, the importance of root crops, particularly cassava and sweet potato, far outweighs the tonnage of production. Even where corn predominates, root crops provide food security during famine caused by drought, against which they are more resistant than corn. In periods of strife, root crops can again be vital because the tuberous roots are too bulky to be stolen on a large scale, and a new crop of sweet potato can be available within 3 to 4 months of planting. In Africa, sweet potato is predominantly the food of poor sectors of the population. Rural women grow it near their homes to feed their families (5,40), and its sale can provide women an entry to the cash economy (Fig. 2). Sweet potato requires few inputs, making it appropriate for subsistence farmers with limited resources. Among the major starch staple crops, it has one of the highest rates of production per unit area per unit time (79), making it attractive to farmers with little land. Other advantages are that its short growing season allows it to fit into

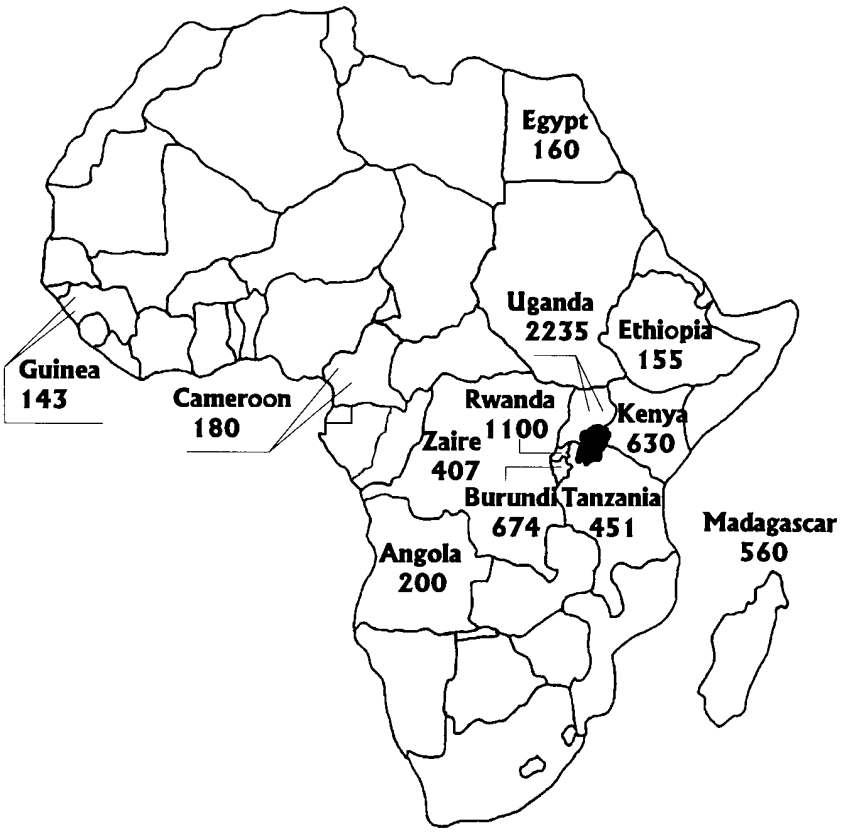


Fig. 1. The 12 African countries with an annual sweet potato production (×10^3) exceeding 100,000 t (21). Note the main production area concentrated around Lake Victoria (in black).

Dr. Gibson's address is: Natural Resources Institute (NRI), University of Greenwich, Central Avenue, Chatham Maritime, Kent, ME4 4TB, UK
E-mail: richard.gibson@nri.org

Publication no. D-1997-1107-01F

many different cropping systems, and it can be harvested piecemeal to provide fresh daily food for a family. Production is increasing in Uganda because cassava, with which it shares second place as a starch staple, is being severely affected by an epidemic of African cassava mosaic geminivirus (25).

Sweet potato feathery mottle potyvirus (SPFMV) occurs everywhere sweet potato is grown, including Africa. Economic loss may be associated with external cracking and internal corkiness, making the tuberous roots unmarketable, but there are few reports of this form of loss from Africa. Instead, SPFMV has economic impact as a component cause of sweet potato virus disease (SPVD), the most important disease of the crop (23). Furthermore, the titer of SPFMV is raised in SPVD-affected plants, and SPFMV is readily acquired by aphid vectors only from such plants (65). This unique impact and epidemiology of SPFMV in Africa, and the importance of sweet potato as a lifeline for vulnerable populations, are the reasons for focusing this review solely on Africa. The review focuses especially on Uganda, both because it is the main producer in Africa, and because this allows the authors to draw on personal experiences.

SPFMV and SPVD

SPFMV is one of only five viruses that have been detected and confirmed independently in sweet potato in Africa (Table 1). This probably reflects lack of surveys and confirmatory work rather than a lack of virus diversity, as an additional eight (7) or 13 (47) sweet potato viruses have been reported elsewhere. Of the five viruses, only sweet potato mild mottle virus has not been found elsewhere. SPFMV was first described in 1945 by Doolittle and Harter (19) in the United States. SPFMV was first reported in East Africa in 1957 in Kenya, Tanzania, and Uganda (67) under the name sweet potato virus A. It was not reported in West Africa until 1976 (65), when it was called sweet potato veinclearing virus.

SPFMV is a member of the *Potyvirus* genus of the *Potyviridae*, the largest family of plant viruses. Like other definitive potyviruses, the virions are elongate, flexuous rods with a monopartite, single-stranded, positive-sense RNA molecule. SPFMV is transmitted in the nonpersistent manner by aphids (46,68). Leaf symptoms in sweet potato are generally mild and transient, but may include veinclearing, vein feathering, and chlorotic spots, particularly on older leaves. Symptoms on roots may include external cracking and internal necrosis, depending on cultivar and virus isolate. In other *Ipomoea* species, including the indicator species *I. setosa* (Fig. 3) and *I. nil*, symptoms are more pronounced and again include veinclearing, mosaic, leaf stunting, and distortion. SPFMV has a more limited host range than typical potyviruses: some isolates infect *Chenopodium amaranticolor*, *C. quinoa*, or *Nicotiana benthamiana*, but others seem to be restricted to *Ipomoea*. SPFMV also has an atypically long virion (810 to 865 nm) and a large RNA genome with a molecular weight of 3.65×10^6 (about 10.6 kbp). These sizes are about 10 to 15% larger than those of most potyviruses (1,44). SPFMV is not seed-transmitted in sweet potato (78).

SPFMV has evolved into strains that can be differentiated on the basis of their symptoms in different hosts. Much work on this has been done in the United States, where the main division is into the so-called common (C) and russet crack (RC) strains. Isolates of the C strain cause no necrosis or root cracking in sweet potato, whereas isolates of the RC strain may do so but only in cultivars susceptible to this injury. Useful indicators include the sweet potato cultivars Goldrush, Porto Rico, and Jersey: the RC strain in Porto Rico causes leaf spots, in Jersey roots develop russet crack and necrotic lesions, and in Goldrush roots develop internal cork (11,18,43). The C and RC strains can also be distinguished by their symptoms in indicator plants, with only the RC strain causing local lesions on sap-inoculated *C. amaranticolor* and *C. quinoa* (9).

No work has been reported on strains of SPFMV in Africa. Furthermore, cracking or necrosis in roots, as would typically be

Fig. 2. A family selling sweet potato and other surplus produce: Gayaza, Mpigi District, Uganda.

Table 1. Sweet potato viruses identified in Africa

Virus	Vector	Reference
Confirmed identifications		
Sweet potato feathery mottle virus	Aphids	65,67
Sweet potato mild mottle virus	Whiteflies	31,75
Sweet potato chlorotic stunt virus (Synonymous to sweet potato virus disease–associated closterovirus and sweet potato sunken vein virus)	Whiteflies	32,65,77
Sweet potato latent virus	Unknown	12,75
Cucumber mosaic virus	Aphids	47,75
Unconfirmed identifications		
Sweet potato caulimo-like virus	Unknown	75
Sweet potato ringspot virus	Unknown	75
Sweet potato chlorotic fleck virus	Unknown	12,34

Fig. 3. *Ipomoea setosa* infected with sweet potato feathery mottle virus.

Table 2. Virus incidence in sweet potato cuttings obtained from symptomless field plants in Uganda (modified from 27)

Detection method	Plants infected[a]			No virus detected	Cuttings tested
	SPFMV	SPMMV	SPCFV		
NCM-ELISA[b]	15	2	1	310 (97%)	328
Grafting to *I. setosa*	17	0	0	99 (84%)	116

[a] SPFMV, sweet potato feathery mottle virus; SPMMV, sweet potato mild mottle virus; SPCFV, sweet potato chlorotic fleck virus.
[b] Nitro-cellulose membrane enzyme-linked immunosorbent assay.

Fig. 4. A comparison of disease-free and sweet potato virus disease–affected sweet potato plant showing stunting, overall plant pallor, and leaf strapping.

Fig. 5. A close-up of a plant with sweet potato virus disease showing the veinclearing.

caused by the RC strain, has not been reported, and we have never observed such symptoms on tuberous roots for sale in marketplaces in Uganda. This may be either because the RC strain does not occur here or because the cultivars commonly grown do not express this symptom. However, some exotic cultivars tested in Kenya have exhibited cracking on tubers, which could be caused by RC isolates (E. E. Carey, *personal communication*); and external grooves, possibly symptomatic of infection with the RC strain, have also been reported on roots of virus-infected plants in South Africa (37). Perhaps more importantly, some African isolates may be unusually virulent. When the SPFMV-resistant CIP clone 420026 was infected with an isolate of SPFMV collected in Uganda in our experiments, graft-inoculated plants gave a positive reaction to SPFMV in nitro-cellulose membrane enzyme-linked immunosorbent assay (NCM-ELISA) and lower leaves developed a chlorotic mottle.

Economic Loss Caused by SPFMV

Data on yield losses caused by SPFMV are few and mostly unsatisfactory, because most experiments set out to determine the benefit of using virus-free planting material rather than to assess the yield effect of SPFMV. Consequently, identification of SPFMV as the virus most likely to be causing yield loss has sometimes had to be inferred. Thus, in South Africa, a virus-free clone of cv. Impala yielded three times more than so-called ordinary plants (37). In Nigeria, virus-free cuttings of five genotypes yielded about 30% more than field-derived, apparently symptomless cuttings (33), which often carry SPFMV (65). In a small screenhouse trial in Uganda, a virus-free clone of Tanzania had about twice the root yield of the same clone graft-inoculated with SPFMV (27). However, root yields were very poor in the screenhouse, and foliage production on virus-free and inoculated plants was similar. In the United States, plants with the RC strain of SPFMV yielded similarly to symptomless plants (38). In New Zealand, a virus-free clone of Owairaka Red yielded about 50% more than plants infected with what appears to have been SPFMV from the symptoms described in indicator plants (56). In Venezuela, symptomless plants of cv. USR.P1-20 yielded twice as much as plants with symptoms identified as caused by SPFMV by grafting to *I. nil* (58). Overall, these results suggest that SPFMV alone diminishes tuberous root yields in at least some cultivars. They cannot, however, be directly translated into massive yield losses in Africa attributable to SPFMV. In addition to frequently inadequate virus identification, incidences of infection in planting stocks may be much less than 100% (Table

2), and virus-free neighbors may compensate for any decreased yield of infected plants.

The major economic loss in Africa due to SPFMV is as a component of sweet potato virus disease (SPVD). SPVD is used in Africa to describe a range of severe symptoms on sweet potato generally attributed to virus infection (Figs. 4 and 5). Probably the first report of SPVD in Africa was by Stayaert in 1939 (referred to in 66) in Ituri in what is now the Democratic Republic of Congo. Within a few years, crops of 30 t/year were reduced to 4 t/year and then abandoned. SPVD was subsequently reported in Rwanda, Burundi, and Uganda. SPVD seems to occur throughout sub-Saharan Africa (61) and also occurs in Ghana (15), Nigeria (60), Kenya, Tanzania (67), Gabon, and Zimbabwe (R. W. Gibson, *unpublished data*). It has been reported more rarely outside Africa, e.g., in Israel (16) and the United States (57). Symptoms of SPVD on sweet potato typically include plant stunting, and leaf distortion, and strapping coupled with a mosaic or veinclearing (Fig. 4). In Nigeria in 1976, SPVD was first shown to be caused by a combined infection of the whitefly-borne sweet potato chlorotic stunt closterovirus (SPCSV) and the aphid-borne potyvirus then called sweet potato veinclearing virus (65) but now recognized to be SPFMV (14). Interestingly, the symptoms of mosaic and leaf distortion are those typical of a potyvirus and suggest that SPFMV dominates within SPVD. Inclusion bodies characteristic of potyviruses as well as membrane-enclosed vesicles characteristic of closteroviruses can both be observed in SPVD-affected plants (77). We have confirmed this association of SPFMV and SPCSV with SPVD in Uganda (26). SPCSV has also been called SPVD-associated closterovirus (77), and serology has shown that it is also synonymous with sweet potato sunken vein closterovirus isolated from sweet potato in Israel (16,32).

SPVD is the most destructive disease of sweet potato throughout Africa (23). Yield of affected plants is generally reduced severely (Table 3). Indeed, the yield losses in Table 3 may be underestimates because the symptomless controls were in most cases not necessarily virus-free, and latent infections may have decreased their yields. We have found mean frequencies of SPVD-affected plants in crops in different areas of Uganda varying between 0 and 30%, and an even larger range has been reported in Kenya (66,75).

Spread of SPFMV in Africa

Natural spread of SPFMV in the field. SPFMV is aphid-borne in the nonpersistent manner (41,67,68) and so is carried efficiently only short distances. Nevertheless, SPFMV can spread rapidly in crops. In Kenya, SPFMV infected 25% of previously virus-free plants of exotic cultivars within one crop cycle and infected 80 to 90% within three crop cycles (75). Similarly rapid spread has been observed in Brazil (58) and the United States (38). As in the United States (41), we have rarely observed aphids infesting sweet potato crops in Uganda, although the peach-potato aphid, *Myzus persicae*, is occasionally a pest on screenhouse plants. By contrast, the whitefly *Bemisia tabaci*, which transmits SPCSV, is seasonally very abundant. The cotton aphid (*Aphis gossypii*), the groundnut aphid (*A. craccivora*), and *M. persicae* have been shown experimentally to be efficient vectors of SPFMV (46,65) and were commonly caught in yellow water traps in sweet potato fields in Kenya (75). Presumably, the alates of these and other noncolonizing species spread SPFMV while making test visits to crops, as occurs for dissemination of potyviruses in some other crops (30).

SPFMV is at a low titer in sweet potato when it infects by itself. Infections can be detected with difficulty by ELISA (2,20), and aphids acquire SPFMV infrequently (41) or not at all (65). A significant aspect of plants infected with both SPFMV and SPCSV is that the titer of SPFMV is increased, so that it becomes readily detected by both ELISA and electron microscopy (62). The exact mechanism of this synergy is unknown. Whether infection with SPCSV increases the susceptibility of sweet potato plants to infection with SPFMV is also unknown.

Perpetuation of SPFMV during vegetative propagation. In cultivation, sweet potato is usually propagated vegetatively by using cuttings either produced as

Table 3. Some examples of tuberous root yield losses associated with sweet potato virus disease in Africa

Country	Yield loss	Cultivar	Comments	Reference
Uganda	66%	Bitambi	Severely diseased plants	3
Uganda	98%	Tanzania	Virus-free clone as a negative control	26
Uganda	57%	Kyebandula	Some controls became diseased	49
Nigeria	76-78%	TIS 1499		28
Nigeria	60%	TIB 4	Low level of resistance to sweet potato virus disease	28
Cameroon	0-90%	Eight clones	Losses varied between clones and field trials	53

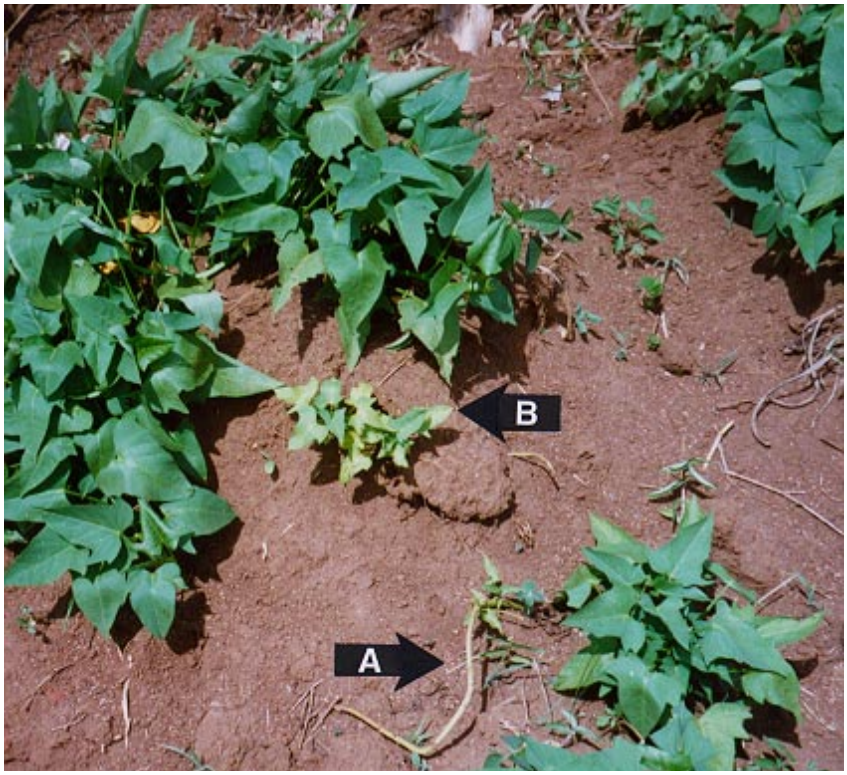


Fig 6. A sweet potato virus disease (SPVD)-affected cutting growing from a previous crop (arrow A) and a planted SPVD-affected cutting (arrow B), both growing among a young sweet potato crop.

sprouts from tuberous roots or obtained from the foliage of mature crops (34). Cuttings form roots readily, and SPFMV can be perpetuated from one cropping cycle to the next in cuttings obtained from infected plants (Fig. 6). Furthermore, both unharvested roots and discarded foliage can reestablish themselves as plants, especially when the land is left fallow. Such plants are often SPVD-infected (Fig. 6) and may also perpetuate SPFMV between cropping cycles. Sprouting roots discarded as food or transported specifically to provide planting material may similarly provide means of long-distance, including intercontinental, dissemination of SPFMV. When questioned, most farmers show or explain how they select cuttings for new crops only from plants unaffected by SPVD. However, by itself, SPFMV causes very mild or no symptoms in the foliage of most sweet potato cultivars (7), including those used in West (62,65) and East Africa (27). This lack of obvious symptoms implies that farmers cannot selectively avoid SPFMV-infected cuttings. Consequently, the additional spread of SPFMV by aphids should cause SPFMV gradually to increase in occurrence in farmers' stocks over succeeding generations, as occurs with many viruses in other vegetatively propagated crops (74). Surprisingly, symptomless cuttings obtained from farmers' fields in Uganda (Table 2) were largely virus-free (27), indicating that something is preventing this spread.

The cause of the lack of increase of SPFMV in planting stocks appears to be that cuttings taken from infected sweet potato do not all carry SPFMV (20,27,54). Indeed, a majority of symptomless cuttings of the East African cv. Tanzania graft-inoculated with SPFMV failed to graft-infect *I. setosa* when tested some months later (27). Sweet potato plants with SPVD in West Africa have also been observed to become symptomless and resume vigorous growth (70,71). Recovery from disease in plants infected with SPFMV also occurs in the indicator species *I. nil*: proteolytic activity, which breaks down the N-terminal region of potyvirus coat protein virus particles in vitro is raised in recovered *I. nil* (64). Whether this proteolysis has a role in virus elimination in sweet potato is unknown. In cassava, the ability to recover has been associated with genotypes in which the systemic spread of ACMV is incomplete (63), and it seems more than coincidental that resistance to SPFMV in sweet potato has also been associated with restricted virus movement (52). The value of a mechanism that enables infected plants to produce virus-free cuttings is potentially enormous, as also shown by the example of cassava in Africa, where natural recovery from ACMV leads to virus-free planting material remaining available to African farmers (22). We are currently examining the extent to which SPFMV elimination occurs in different cultivars in order to assess whether it suffices to explain the apparently low SPFMV incidence in planting materials used by African farmers.

Fig 7. (A) Sweet potato feathery mottle virus–infected plant of *Ipomoea tenuirostris* growing in a hedge in Uganda. (B) Healthy plant of *I. tenuirostris*.

Fig 8. A plot of sweet potato growing typically around a homestead in Kanoni, Mpigi, Uganda.

Wild plants as virus reservoirs. *Ipomoea* spp. and related genera occur throughout the Tropics, and native *Ipomoea* spp. have been reported to be natural hosts of SPFMV (8,13,24). Eighty-nine wild *Ipomoea* species occur in East Africa (6) alone, but there has been no report of an African *Ipomoea* host of SPFMV. Introduced species known to be hosts of SPFMV, such as *I. hederifolia*, have become naturalized in some areas, and we have identified SPFMV in a diseased *I. hederifolia* growing wild in Uganda. *I. tenuirostris* (Fig. 7) is a common native species in Uganda (42). It is a semiperennial with vines that twine through low-growing plants. Among 104 vines found at NAARI Farm, 46 had such symptoms as veinclearing, leaf stunting, and distortion (Fig. 6). In a sample of 25 symptomatic vines, 24 tested positive for SPFMV using NCM-ELISA, and two of 25 symptomless vines were also positive. Infection with SPFMV was confirmed by grafting a seedling of *I. setosa* with a symptomatic shoot

of *I. tenuirostris*, the *I. setosa* developing typical SPFMV symptoms. One of seven diseased plants of an unidentified *Ipomoea* sp. found at NAARI Farm also reacted positively in NCM-ELISA to SPFMV. Although most wild *Ipomoea* spp. are semiperennial climbers, some such as *I. spathulata* and *I. hildebrandtii* are erect, woody perennials. In areas of West Kenya, these two species are the dominant shrub, but whether they carry SPFMV is untested.

Cultivation practices affecting the epidemiology of SPFMV. The fact that sweet potato in Africa is a crop of the poor, and especially women, has many implications for the cultivation of the crop and consequently for the epidemiology of SPFMV. Fields are generally small (<1 ha) and are often planted near the home for security and easy access (Figs. 8 and 9). Homes are also generally grouped. Consequently, crops are close to each other. In a survey in Uganda in 1996, most crops were within sight of others, averaging only about 10 m apart (Table 4, Figs. 9 and 10). The small plots are interspersed among other crops, a practice sometimes called "patch intercropping" (40). Sweet potato is also grown over a longer period than most staple crops. This is because it is generally eaten fresh and also because farmers have to grow crops in a continual cycle because shoots for planting material are obtained from growing crops. Some Tanzanian farmers have two main planting seasons (40), and some Ugandan farmers even have three (5). Piecemeal harvesting also allows crops to continue yielding over many months. Consequently, mature and newly planted crops overlap.

Farmers generally obtain cuttings from a mature crop, either their own or a neighbor's, but in areas with a prolonged dry season, crops may be grown in swampy or shaded areas, especially for producing cuttings (Table 5). There are no formal systems of virus-free certified cuttings within much of Africa, although certified cuttings are available in southern Africa (37). Disease-free cuttings, about 30 cm long, are hand-planted, three to five cuttings per mound or approximately 30 cm apart on ridges. Sweet potato is grown either as a monocrop or intercropped with, for example, corn, beans, or cassava, the proportion intercropped differing enormously between districts. When grown as a monocrop, however, sweet potato is often a mixture of two or three cultivars, either planted as a patchwork or intermingled (Table 4, Fig. 11). Pesticides are used only rarely, largely to control attacks of caterpillars, which can completely defoliate crops. Tuberous roots usually develop within 3 to 6 months. Women generally harvest roots piecemeal using a small stick in order to leave the plant intact (Fig. 12), harvesting them as they mature and as required before completely harvesting individual hills sequentially to remove the last few roots. This practice prolongs the period over which a crop can yield to up to 12 months and favors cultivars that yield over a prolonged period (5,40,69).

Most of these common cultivation practices favor the survival and spread of the nonpersistently transmitted SPFMV, particularly:

- the close proximity of sweet potato crops;
- sequential planting, resulting in new crops overlapping with mature crops;
- small fields, which ensure that most plants are close to an edge;
- use of vegetative propagation without organized virus-free planting schemes;
- lack of SPFMV symptoms in local cultivars when the virus infects alone, preventing SPFMV-free plants being selected as sources of cuttings;
- piecemeal harvesting followed by sequential harvesting, which prolongs the period crops are retained, during which virus disease can build up and diseased crops are available as a source of inoculum; and
- the general poverty of farmers growing sweet potato, allowing few resources to be allocated for crop protection measures.

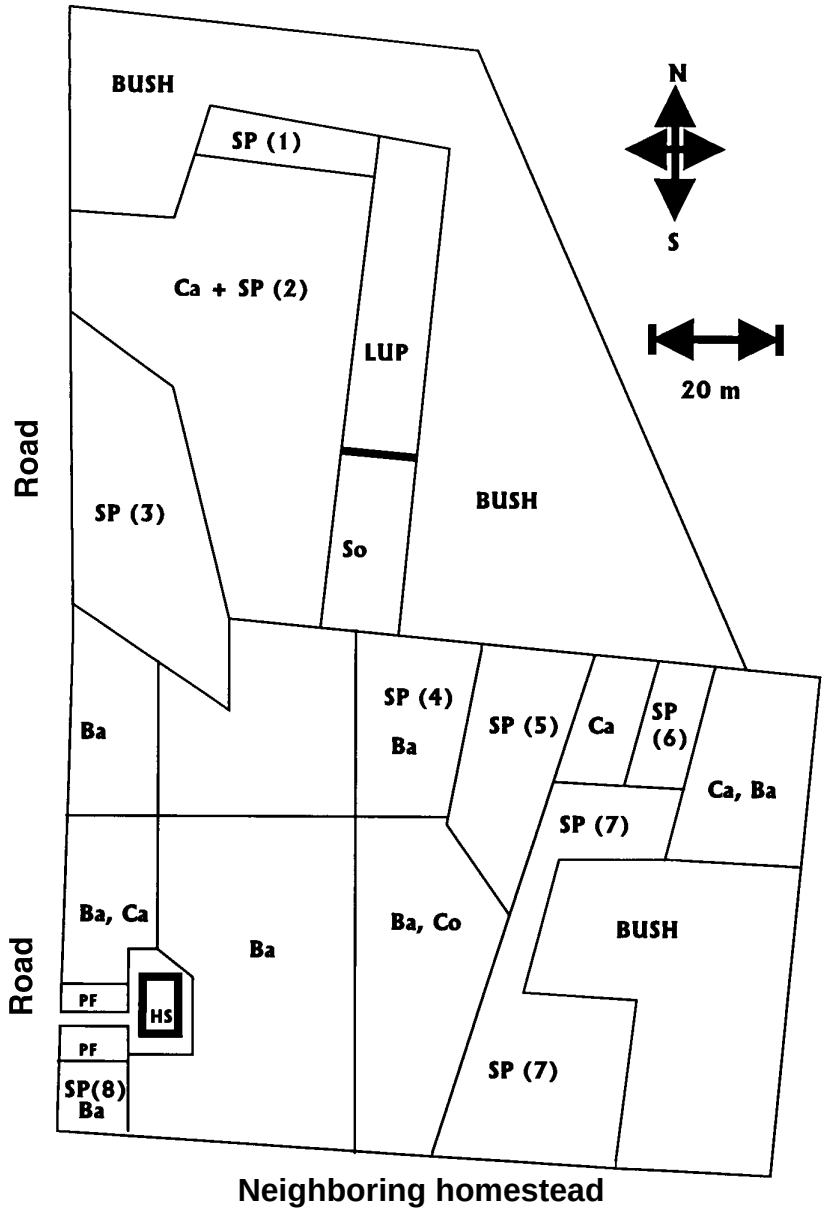


Fig 9. A sketch map of a typical homestead in southern Mpigi District, Uganda, showing the "patch intercropping" of large numbers of small plots of sweet potato of differing ages and mixed cultivars. Sweet potato plots (SP): SP (1), four cultivars, 2-month-old crop; SP (2), harvested sweet potato (groundskeepers) and cassava; SP (3), six cultivars, 4-month-old crop; SP (4), four cultivars of sweet potato (3-month-old crop) grown with bananas; SP (5), five cultivars, 6-month-old crop; SP (6), three cultivars, 6-month-old crop; SP (7), five cultivars, 6-month-old crop; SP (8), four cultivars of sweet potato (3-month-old crop) grown with bananas. Other plots: Ba, banana; Ca, cassava; Co, coffee; LUP, land under preparation; PF, passionfruit; So, sorghum. HS, homestead.

Table 4. Cultural practices for sweet potato in different districts in Uganda

	Occurrence of intercropping[a]		Harvesting[b]		Proximity of neighboring crops			
	Mixed cvs. (%)	Mixed species (%)	Sequential (%)	Piecemeal (%)	Another crop visible (%)	Distance (m)	Sex of farmer (% females)	No. of farmers interviewed
Soroti	24	3	100	100	59	7.2±2.4	31[c]	29
Tororo/Busia	54	15	100	100	21	6.8±2.6	77	26
North Mpigi	83	24	100	100	86	13.6±5.1	62	29
Southwest Mpigi	85	35	100	100	54	6.4±2.7	77	26
Masaka/Rakai	87	23	100	100	50	14.0±5.4	70	30

[a] Some farmers, especially in Soroti, Busia, and Tororo, grew single cultivar crops; whereas elsewhere some farmers grew crops comprising mixed sweet potato cultivars and mixed crop species, so rows do not add up to 100%.
[b] All farmers initially piecemeal-harvested and then sequentially harvested the remainder.
[c] Sweet potato production in Soroti is largely commercial for the Kampala market.

Fig. 10. A young crop growing directly adjacent to an old crop of sweet potato in Kanoni, Mpigi District, Uganda.

Table 5. Sources of planting material in different districts of Uganda

	Proportions of different sources (%)[a]				No. of farmers interviewed
District	Swamp	Shade	Own old crop	Neighbors	
Soroti[b]	55	48	40	24	29
Tororo/Busia	0	4	85	35	26
North Mpigi	0	0	66	83	29
Southwest Mpigi	4	0	85	38	26
Masaka/Rakai	0	10	97	50	30

[a] Most farmers used more than one source of planting material, so percentages add up to >100%.
[b] Soroti has a long dry season.

As almost every aspect of cultivation of sweet potato in Africa seems to favor SPFMV, it is surprising that SPFMV by itself does not seem to be a serious problem. Although average crop yields are low, there is little evidence that chronic infection of planting material with SPFMV is involved (Table 2). Why SPFMV's impact remains low is a key question, and much of the answer seems to be that the cultivars grown by farmers are resistant and have an inherent ability to become SPFMV-free.

Selection of SPVD-Resistant Cultivars by African Farmers

Most sweet potato cultivars grown in Africa are local landraces derived from chance seedlings, and only a few derive from conventional plant breeding done by national or international agencies (29). Several factors may favor selection for resistance, notably:

- the traditional cultivation practices and the presence of SPVD-affected plants provide a high inoculum–selection pressure;
- individual farmers often grow several cultivars, often in the same crop, allowing direct competition, comparison, and selection among cultivars;
- farmers select asymptomatic plants—and therefore relatively virus-resistant genotypes—from which to obtain cuttings;
- piecemeal harvesting allows the crop to be retained for several months so that plants can seed;
- an enormous diversity of cultivars may be grown in an area, 257 named cultivars in the Lake Zone of Tanzania alone (40); and
- neighboring farmers exchange planting material freely.

Evidence for this selection is the rapid turnover in local cultivars. In Uganda, the nine main cultivars grown in the Kingdom of Buganda in 1937 were all superseded by 1963 (3), and cultivars Namajuna, Bitambi, and Kalebe became dominant. By 1989 to 1992 (5), these cultivars had themselves been replaced. In Mpigi, one of the main districts in Buganda, most farmers had changed by 1990 to 1992 to growing New Kawogo, a high-yielding, virus-resistant cultivar. Furthermore, the two most widespread cultivars in Uganda in 1963, Magabali and Kawungezi, are now seldom grown either in Buganda or elsewhere in Uganda (5). A similar dynamic situation has been recorded in Tanzania (40).

A suggested cause of sweet potato cultivars being superseded is a chronic buildup of viruses because of prolonged vegetative propagation (3,37). There seems to be little evidence for this, however, since some local cultivars have been maintained for decades with no apparent difficulty (27). Indeed, new cultivars may simply be superior in yield, rate of maturing, palatability, or other characteristics (40) in addition to possessing virus resistance. In Uganda, the

Fig 11. Three different cultivars of sweet potato growing intermingled together.

Fig 12. A woman piecemeal harvesting sweet potato in Uganda.

value of virus resistance is confirmed by the present dominance of the very resistant cultivar New Kawogo in Mpigi District and by the occurrence of virus-resistant cultivars among local landraces (50,51). Resistance to infection by SPFMV alone is unlikely to be selected by farmers, as the infection is largely latent. Instead, clones that are relatively unaffected by SPVD are being selected. In Uganda, several officially released cultivars have also been obtained by screening local cultivars for resistance to SPVD and superior yield characteristics. One of these cultivars, called Tanzania, appears to have been selected originally in Tanzania from among local cultivars. It has become perhaps the most widely grown cultivar in sub-Saharan Africa, occurring in Uganda, Kenya, Tanzania, Malawi, and Zambia (E. E. Carey, *personal communication*).

Additional Strategies to Control SPFMV in Africa

Two main control practices are currently used by African farmers to limit the effects of SPFMV. These are: (i) selection of SPVD-resistant cultivars and (ii) use of disease-free planting material. African farmers have been successful in both these practices, but other approaches may provide opportunities to improve control.

Conventional breeding. Farmers can exploit only the local gene pool, which may lack sources of adequate resistance or other desirable traits. However, this potential disadvantage may be more theoretical than real for SPFMV, as sources of better resistance are not clear. In our tests, some apparently SPFMV-resistant clones, identified at CIP after exhaustive screening of their germ plasm (48), succumbed to Ugandan SPFMV either alone or in complex with SPCSV. We are currently examining the ability of local African cultivars to eliminate SPFMV to determine if it occurs at a sufficient rate to have practical significance. This could be a target for conventional breeding, but the lack of symptoms on SPFMV-infected sweet potato means there is no easy visual marker for plants in which virus elimination has occurred. If the ability to eliminate SPFMV is linked with extremely low virus titers in infected plants, improving the sensitivity of ELISA might provide an effective approach.

Field trials of 23 sweet potato cultivars at four locations in Nigeria identified resistance to SPVD in several (29), and a rapid method of screening by core-grafting roots was developed (70), but only resistant rather than immune genotypes were identified. A different serotype of SPCSV occurs in East Africa to that found in West Africa (32), and sweet potato cultivars selected for resistance in West Africa were severely diseased in Uganda (50). Unlike in most African countries, an extensive conventional breeding program is established in Uganda and led to the release of the cultivar Sowola. Seed from Ugandan crosses has also been provided to national programs of neighboring countries. Again, however, benefits of such a combined approach are potentially negated by a lack of knowledge of the strains of SPFMV and SPCSV in different countries. The International Center for Tropical Agriculture (CIAT) is coordinating a worldwide integrated pest management initiative on whiteflies and whitefly-borne viruses: CIP, NRI, and other partners are working on SPVD. One outcome should be increased knowledge of the variability of SPCSV within and between regions of Africa.

Genetic engineering. Transgenic sweet potato plants, in which NPT-II and GUS marker genes were expressed, were reported in 1993 to have been created using *Agrobacterium rhizogenes* (55). In 1995, introduction of the SPFMV coat protein-encoding region in sense or antisense orientation into the genome of sweet potato was also achieved (35,59), and these transgenic plants are being tested for resistance to SPFMV. If successful, one challenge for developing-country governments will be to determine whether to accept genetically transformed sweet potato plants for agricultural use. For Africa, it will also be important to test whether such engineered resistance is effective against the combination of SPFMV and SPCSV.

Cross-protection. In sweet potato, the common SPFMV strain has been observed to protect against the RC strain (11). In apparent contradiction, however, mixed infections have been reported to occur naturally (9). Although vegetative propagation of sweet potato might allow the easy maintenance of a cross-protecting strain in sweet potato stocks, the approach seems inappropriate if SPVD still develops when SPCSV co-infects. However, such an approach might be useful if an isolate of SPFMV can be found that does not contribute to SPVD (62).

Phytosanitation, Quarantine, and the Need for Virus Detection

Introduction of more germ plasm may be necessary to provide the diversity to improve the African crop, since sweet potato originates in the Americas and the bulk of the crop is grown in Asia. International movement of sweet potato germ plasm risks the importation of viruses, many of which may not occur in

Africa (Table 1) or may have a restricted distribution there. For SPFMV specifically, there is a need to exclude the movement of nonindigenous strains of SPFMV, particularly isolates of the RC strain. However, it is equally important to control the movement of SPCSV if effects on SPFMV via SPVD are to be minimized. In this respect, the fact that the SPCSV serotype found in East Africa differs from that in West Africa (32) emphasizes the value of quarantine within Africa as well as against external threats.

Absence of visual symptoms in sweet potato is an inadequate test for sweet potato viruses, including SPFMV, since most viruses cause slight or no symptoms when infecting alone (7). Serological detection methods such as most types of ELISA lack the sensitivity required for the detection of SPFMV in sweet potato tissue (20) with the certainty required for quarantine. However, NCM-ELISA is more sensitive than microplate ELISA (2,24), and NCM-ELISA kits requiring limited laboratory facilities have been developed by CIP (34). Nevertheless, even NCM-ELISA is insufficiently sensitive for quarantine purposes. An in vitro–transcribed RNA probe (riboprobe) can detect SPFMV with greater sensitivity than ELISA methods (2). SPFMV can also be detected by the reverse transcription polymerase chain reaction (RT-PCR) using primers derived from conserved regions of the SPFMV genome (17). In Africa, neither riboprobes nor PCR-based methods were reported to be used to detect SPFMV, probably because the facilities and materials required for these assays are rare. Another disadvantage of these and serological methods for quarantine is that they can be too specific, missing atypical isolates.

The most appropriate means of detecting SPFMV (and most other sweet potato viruses) in Africa for quarantine purposes remains graft-inoculation to the indicator plant *I. setosa* (Fig. 3). It is essential, however, to graft several scions onto seedlings because of possible restricted movement of viruses such as SPFMV (45). The sensitivity of graft-inoculation of *I. setosa* can be further increased for detecting SPFMV by preinoculating with SPCSV, as the symptoms are then more severe (62). This can provide a very practical method for Africa. Although graft-transmission is a reliable method of detection, it requires greenhouse space for indicator plants, labor, good insect control, and several weeks for a reliable diagnosis. In some countries, pathogen-tested sweet potatoes can be imported only in tissue culture. However, it is even safer if seeds are obtained from pathogen-tested mother plants, since SPFMV and perhaps other sweet potato viruses may not be seed-borne in sweet potato (78).

Crop Hygiene

Certified virus-free sweet potato planting material is available to commercial growers in southern Africa (37). It is mostly unavailable elsewhere. There are few signs that this will change in the near future, since it would be difficult to distribute planting material to subsistence farmers with a sufficient frequency to ensure it remained largely virus-free. It therefore seems likely that in most of Africa, planting materials will continue to be obtained from symptomless parents. Fortunately, this may not have large yield penalties, as such materials may be mostly virus-free, or may carry only SPFMV. Such planting material occasionally may have been infected with SPFMV and/or SPCSV just before being collected. This risk could be reduced if surplus cuttings are planted and diseased ones rogued out, or if planting material is taken from relatively disease-free crops growing sufficiently rapidly to express symptoms on new foliage. Because SPFMV is nonpersistently transmitted, aphids lose it rapidly, little spread occurs between fields more than 100 m apart (39), and isolation from diseased crops should in theory be relatively easy to achieve. However, in practice even such minimal changes may be difficult to introduce because of the tradition of growing crops in small plots around homesteads, and because of an unwillingness of farmers to take cuttings from immature crops in case their yield is adversely affected. No chemicals are applied to control virus vectors in sweet potato, and the returns would probably not justify them.

Conclusions

By itself, SPFMV does not seem to infect sweet potato stocks of African subsistence farmers chronically, apparently because local cultivars are very resistant and because infected plants of these cultivars may be able naturally to become virus-free. The most important aspect of SPFMV in sweet potato in Africa is its close association with SPCSV to produce SPVD. Little work has been done on the synergy between SPFMV and SPCSV, but it is clearly important in the epidemiology of SPFMV and a key to developing SPVD-resistant sweet potato for Africa.

CIP identified Africa as the only continent where breeding sweet potatoes for virus resistance should be a priority (36). SPVD-resistant cultivars have been selected in Africa in three ways. The most important has been the informal selection of resistant cultivars by farmers, as shown by the dominance of such cultivars throughout Africa. The other two approaches are the collection of local germ plasm followed by its formal screening for superior genotypes to release as official national varieties, and the conventional production of seed from superior parents, followed by selection among seedlings for superior genotypes.

Advantages of farmer selection include:

- it involves no government expense;
- it is done in all agro-ecosystems where sweet potato is grown; and
- selected genotypes are automatically acceptable to farmers, since they do the selecting.

Disadvantages include:

- the possibly narrow genetic base of seedlings available to farmers;
- that superior seedlings may be weeded out before their superiority is obvious;
- that few seedlings may join the local pool of germ plasm each season; and
- the possible slowness of distribution of selected genotypes through informal networks.

The main advantages of formal selection within a local germ plasm collection are that:

- it is again relatively cheap, involving only collection costs and selection among perhaps a few hundred genotypes; and
- all genotypes are likely to be acceptable to farmers as they have already selected them.

The main disadvantage is again that the local gene pool is restricted and may lack valuable characters.

The main advantages of formal seedling selection are that:

- many seedlings can be tested each year (more than 300,000 seedlings were screened between 1989 and 1994 by the Ugandan Sweet potato Program (51); and
- exotic germ plasm (provided largely by CIP) can be accessed.

The main disadvantage is the expense of the breeding program and of off-station field trials of superior genotypes. Additionally, this may preclude screening seedlings in all but a few locations–agro-ecosystems.

Interestingly, the respective advantages of the formal selection of seedlings in a breeding program (many seedlings tested, exotic germ plasm available) seem largely to reflect the disadvantages of selection among local cultivars, and the disadvantages of formal selection are largely the advantages of farmer selection. This perhaps explains why the combination of local cultivars coupled with formal screening provided five of six cultivars recently released officially in Uganda (51). This combination approach might be further refined if geographical areas where particular problems (for example, SPVD) are known to be prevalent are targeted for collection of local cultivars. The international success of cultivar Tanzania also suggests that farmer selections should be tested internationally within Africa in order to obtain maximum benefit. Furthermore, the elimination of susceptible types during formal screening might be achieved more quickly by artificial inoculation, perhaps using core-grafting as pioneered

by IITA (70), or by mass infestation with viruliferous aphids and whiteflies.

The close relationship of SPFMV with SPCSV suggests a possible co-evolution, and Sheffield's observation that SPVD appears to have been long-established in Africa (66) implies that both viruses may have originated in Africa. An African origin of SPFMV is made plausible by our discovery of a native host, and the occurrence of at least two serotypes of SPCSV within Africa (26,32) is also consistent with SPCSV originating there. Isolates of SPCSV found in Israel and the United States are of the serotype found in West Africa (32) and may have spread through trade with West Africa. This is also consistent with an African origin of SPCSV's insect vector, *B. tabaci* (10). If SPCSV did spread to the Americas from West Africa, SPFMV could presumably have spread by a similar route. A non-American origin of SPFMV (and SPCSV) might explain the apparent lack of immunity genes to SPFMV (and SPCSV) within sweet potato, unlike the situation with the Irish potato for several of its viruses (73). However, the genus *Ipomoea* occurs throughout the Tropics, and naturally infected native *Ipomoea* spp. have also been reported in Australia (24). Consequently, SPFMV need not have originated in Africa for it not to have evolved alongside sweet potato.

Despite the importance of viruses in African sweet potato crops, relatively little work has been done investigating them. The first report of SPFMV in Africa was in 1957 by Sheffield (67), contrasting with the first description of ACMV, itself an under-researched virus (72), more than 60 years earlier (76). Little additional work was done in East Africa, where the bulk of the crop has been grown for more than 30 years (75). This is confirmed by the finding that CIP's database contains only six references to SPFMV for Africa from 1985 to 1996; whereas 59 references were found for the same period for the Americas despite their much smaller production of the crop. The small number of viruses with confirmed identification in Africa again seems likely to reflect a lack of research. Areas particularly in need of research include:

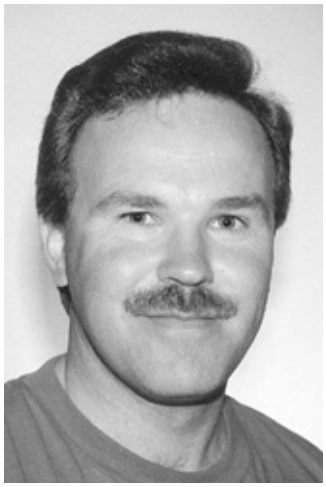
1. assessing the variability of SPFMV (and SPCSV) within Africa;
2. investigating how SPCSV and SPFMV act synergistically in sweet potato and whether there are sources of resistance to SPFMV that are unaffected by this synergism;
3. confirming that SPFMV is naturally eliminated in some genotypes and assessing its importance in preventing sweet potato stocks becoming chronically infected;
4. screening for resistance to SPCSV and, if necessary, genetically engineering it;
5. epidemiology of SPFMV and SPCSV

R. F. Karyeija

Mr. Karyeija is a plant pathologist for the Plant Quarantine Department of the Ministry of Agriculture, Animal Industry and Fisheries, Uganda, and is based at Kawanda Agricultural Research Institute. He earned his B.S. in agriculture from Makerere University, Kampala, Uganda, and his M.S. in plant disease management at the University of Wales, Aberystwyth, UK. He is currently studying the variability of sweet potato feathery mottle potyvirus in Africa as part of his Ph.D. training at the University of Helsinki, Finland.

R. W. Gibson

Dr. Gibson is a virologist now based at the Natural Resources Institute in the University of Greenwich, UK. He recently returned from a 3-year secondment to NAARI, Uganda. He received his B.S. from the Department of Zoology in 1968 and his Ph.D. from the Department of Agriculture and Horticulture in 1971 at the University of Bristol, doing his research work for the latter on the biology of aphids of potatoes. His research career initially continued this focus on the control of aphids and aphid-borne viruses of potatoes but has recently expanded into viruses of tropical root crops including sweet potato and cassava.



J. P. T. Valkonen

Dr. Valkonen is professor of virology at the Swedish University of Agricultural Sciences in Uppsala, Sweden. He earned his M.S. and Ph.D. in plant pathology at the University of Helsinki, Finland, in 1989 and 1993. While pursuing his doctoral studies on virus resistance of wild potato species, he worked at Rothamsted Experimental Station, UK, in 1989, and the International Potato Center, Peru, in 1990 to 1991. He carried out postdoctoral studies at the Department of Plant Pathology, Cornell University, in 1993 to 1994. He was a Fellow of the Academy of Finland from 1993 to 1997 and became a research group leader at the Institute of Biotechnology, University of Helsinki. His current studies are on molecular virus–host interactions, with particular emphasis on resistance to potyviruses in cultivated and wild potato and sweet potato.

both in crops and in wild hosts;
6. more accurate assessment of yield effects; and
7. improved tools for SPFMV diagnosis.

We are currently working on only 1, 2, and 3. We would also like to emphasize the need for more support to national sweet potato programs both from national governments and donors, since both research and extension staff working on sweet potato seem, like most cultivators of the crop, to belong to the poorest funded sector.

Acknowledgments

We particularly thank E. Pehu, Department of Plant Production, University of Helsinki, for her strong backing during the development of this project. We also thank I. Mpembe for technical assistance, R. O. M. Mwanga and G. Turyamureeba (NAARI) for valuable insights into sweet potato breeding, and S. Fuentes and L. F. Salazar (CIP, Lima, Peru), E. E. Carey (CIP, Nairobi, Kenya), and J. M. Thresh (NRI, Kent, UK) for critically reading the manuscript. R. F. Kayeija thanks the Academy of Finland and FINNIDA for financial support (grant 34268). This publication is also an output from a research project funded by the British Department for International Development. The DFID can accept no responsibility for any information provided or views expressed.

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