

Viruses and Virus Disease Complexes of Sweetpotato

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ABSTRACT

Sweetpotato is an important crop for food security in many developing countries. Surveys have consistently listed virus diseases, especially sweetpotato virus disease (SPVD), as the most important diseases of this crop, yet they remain the most difficult diseases to manage. Much has been learned about sweetpotato viruses from independent research programs in different countries in recent years. Although there are indications that some viruses are yet to be isolated and characterized, there are at least 15 well characterized viruses now known from sweetpotato. It has become evident that sweetpotatoes are often infected by complexes of viruses and that interactions among these viruses influence the symptoms and yield losses. The crinivirus, *Sweet potato chlorotic stunt virus* (SPCSV), can greatly enhance the activity not only of the other key component of *Sweet potato feathery mottle virus* (SPFMV, a potyvirus), but also a number of other unrelated viruses. SPCSV will be the primary focus of future research to understand and control sweetpotato virus disease complexes. Sweetpotato begomoviruses are more widely distributed than previously recognized and may also require attention. International scientific exchange and collaboration could help determine why SPVD occurs in some countries but not others and provide insight to controlling this disease in the future.

Keywords: *Ipomoea batatas*, mixed infections, virus detection, host resistance

Abbreviations: CMV, *Cucumber mosaic virus*; ELISA, enzyme-linked immunosorbent assay; HCPro, helper component proteinase; HSP70, heat shock protein 70; IVMV, *Ipomoea vein mosaic virus*; IYVV, *Ipomoea yellow vein virus*; NCM-ELISA, nitrocellulose membrane ELISA; PCR, polymerase chain reaction; PTGS, posttranscriptional gene silencing; PVX, *Potato virus X*; PVY, *Potato virus Y*; QTL, quantitative trait loci; RT-PCR, reverse transcription PCR; SSA, sub-Saharan Africa; SPVD, sweetpotato virus disease; SPCSV, *Sweet potato chlorotic stunt virus*; SPFMV, *Sweet potato feathery mottle virus*; SwPLV, *Sweet potato latent virus*; SPLCV, *Sweet potato leaf curl virus*; SPLCGV, *Sweet potato leaf curl Georgia virus*; SPLSV, *Sweet potato leaf speckling virus*; SPMMV, *Sweet potato mild mottle virus*; SPV2, *Sweet potato virus 2*; SPVY, *Sweet potato virus Y*; SPMSV, *Sweet potato mild speckling virus*; SPVG, *Sweet potato virus G*; SPYD, *Sweet potato yellow dwarf virus*; TSWV, *Tomato spotted wilt virus*

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INTRODUCTION

Among the food crops worldwide, sweetpotato (*Ipomoea batatas*) is the third most important root crop after potato and cassava and is ranked seventh in global food crop production. Sweetpotato ranks fourth in importance in the developing world after rice, wheat, and corn (Kays 2005). The crop is grown primarily in tropical and subtropical regions usually with low input and can produce high yield under marginal conditions. In tropical regions, the crop can be grown year round. Vine cuttings from mature crops are

used to plant new crops. In temperate and subtropical regions, storage roots are stored over winter and used to initiate the next season's crop.

Viral diseases occur wherever sweetpotato is cultivated. Because it is a vegetatively propagated crop, accumulation and perpetuation of viruses can become a major constraint for production. Virus diseases often cause reduction in yield and quality of storage roots (Clark and Moyer 1988; Loebenstein *et al.* 2004). Studies have demonstrated yield losses of up to 30-50% in farmers' fields in the US (Clark and Hoy 2006), but losses of 80-90% have been recorded in

Table 1 Viruses reported to infect sweetpotato.^a

| Viruses | Family/Genus | Vector | Ref. ^b |
|--|-----------------------------------|------------|-------------------|
| <i>Cucumber mosaic virus</i> (CMV) | <i>Bromoviridae/Cucumovirus</i> | Aphids | 1 |
| <i>Ipomoea yellow vein virus</i> (IYVV) | <i>Geminiviridae/Begomovirus</i> | Whiteflies | 2 |
| <i>Sweet potato chlorotic stunt virus</i> (SPCSV) | <i>Closteroviridae/Crinivirus</i> | Whiteflies | 3, 4 |
| <i>Sweet potato feathery mottle virus</i> (SPFMV) | <i>Potyviridae/Potyvirus</i> | Aphids | 5, 6 |
| <i>Sweet potato latent virus</i> (SwPLV) | <i>Potyviridae/Potyvirus</i> | Aphids | 7 |
| <i>Sweet potato virus G</i> (SPVG) | <i>Potyviridae/Potyvirus</i> | Aphids | 8 |
| <i>Sweet potato leaf curl virus</i> (SPLCV) | <i>Geminiviridae/Begomovirus</i> | Whiteflies | 9 |
| <i>Sweet potato leaf curl Georgia virus</i> (SPLCGV) | <i>Geminiviridae/Begomovirus</i> | Whiteflies | 10 |
| <i>Sweet potato leaf speckling virus</i> (SPLSV) | <i>Luteoviridae/Enamovirus</i> | Aphids | 11 |
| <i>Sweet potato mild mottle virus</i> (SPMMV) | <i>Potyviridae/Ipomovirus</i> | ? | 12 |
| <i>Sweet potato mild speckling virus</i> (SPMSV) | <i>Potyviridae/Potyvirus</i> | Aphids | 13 |
| <i>Tomato spotted wilt virus</i> (TSWV) | <i>Bunyaviridae/Tospovirus</i> | Thrips? | 14 |
| Tentative species | Family/Putative genus | | |
| Sweet potato C-6 virus | ? | ? | 15 |
| Sweet potato caulimo-like virus | <i>Caulimoviridae</i> | ? | 16 |
| Sweet potato chlorotic fleck virus (SPCFV) | <i>Flexiviridae/Carlavirus</i> | ? | 17, 18 |
| <i>Ipomoea crinkle leaf curl virus</i> (ICLCV) | <i>Geminiviridae/Begomovirus</i> | ? | 19 |
| Sweet potato ringspot virus | <i>Comoviridae/Nepovirus</i> | ? | 20 |
| Sweet potato vein mosaic virus | <i>Potyviridae</i> | Aphids | 21 |
| Sweet potato virus 2 (SPV2) | <i>Potyviridae/Potyvirus</i> | Aphids | 22 |
| <i>Sweet potato yellow dwarf virus</i> (SPYDV) | <i>Potyviridae/Ipomovirus</i> | ? | 23 |

^a Nomenclature according to the International Committee on Taxonomy of Viruses (Fauquet *et al.* 2005).

^bReferences: 1, Cohen *et al.* 1988; 2, Lotrakul *et al.* 2003; 3, Winter *et al.* 1992; 4, Kreuze *et al.* 2002; 5, Moyer and Kennedy 1978; 6, Sakai *et al.* 1997; 7, Colinet *et al.* 1997; 8, Colinet *et al.* 1994; 9, Lotrakul and Valverde 1999; 10, Lotrakul *et al.* 2003; 11, Fuentes *et al.* 1996; 12, Colinet *et al.* 1996; 13, Alvarez *et al.* 1997; 14, Clark and Hoy 2007; 15, Fuentes 1994; 16, Atkey and Brown 1987; 17, Fuentes and Salazar 1992; 18, Aritua and Adipala 2004; 19, Cohen *et al.* 1997; 20, Brunt *et al.* 1996; 21, Nome 1973; 22, Rossel and Thottapilly 1988; 23, Liao *et al.* 1979. *Ipomoea vein mosaic virus* (Souto *et al.* 2003) and *Sweet potato virus Y* (Ateka *et al.* 2004) are synonymous to SPV2 (Tairo *et al.* 2006; Ateka *et al.* 2007).

areas affected by virus complexes that include *Sweet potato chlorotic stunt virus* (SPCSV, genus *Crinivirus*) and potyviruses (Hahn *et al.* 1981; Milgram *et al.* 1996; Aritua *et al.* 2000; DiFeo *et al.* 2000; Mukasa *et al.* 2006). The use of infected planting material such as vine cuttings or storage roots is the most common source of sweetpotato viruses, but clean planting material can be quickly reinfected by some viruses, especially those transmitted by aphids and whiteflies (Moyer and Salazar 1989; Valverde *et al.* 2004d).

Sweetpotato cultivars gradually decline in performance over years after they are released, and are often replaced within 20 years (Clark *et al.* 2002). This is in part due to the accumulation of viruses and other pathogens in the propagating material. The viruses that cause decline have not been fully determined and may vary from one part of the world to another. In Africa, SPCSV is commonly found in complexes with other viruses such as *Sweet potato feathery mottle virus* (SPFMV, genus *Potyvirus*), and the Sweet Potato Virus Disease (SPVD) causes rapid decline (Gibson *et al.* 1998; Karyeija *et al.* 2000).

Ca. 20 viruses have been isolated, described, and/or characterized from sweetpotato in the past 25 years (Table 1). There have been several reviews describing the viruses found in sweetpotato, the most recent of which are Loebenstein *et al.* (2004) and Tairo *et al.* (2005). A great deal has been learned about SPVD in sub-Saharan Africa and there have been important findings on sweetpotato viruses in other regions of the world. However, these studies have mostly focused on local situations. For example, in Asia most of the sweet potato virus research has been conducted in Japan and mainly with SPFMV (Nishiguchi *et al.* 1998; Onuki and Handa 1998; Sonoda *et al.* 1999). This review will concentrate on recent developments in our understanding of viruses that infect sweetpotato. We hope our interpretation of recent research will clearly indicate the need for international exchange and potential areas for future research and collaboration.

ETIOLOGY OF SWEETPOTATO VIRUS DISEASES

The areas with the most detailed and updated knowledge on currently prevailing viruses in sweetpotato include southern parts of the United States, Peru, Israel, Japan, Australia and East and South Africa (Loebenstein *et al.* 2004; Tairo *et al.*

2005; Clark and Hoy 2006; Tairo *et al.* 2006; Ateka *et al.* 2007; Clark and Hoy 2007). A concerted effort is being made in several laboratories to elucidate the etiology of sweetpotato virus diseases as the cause of some of the diseases has not yet been determined. For example, in East Africa many sweetpotato plants which exhibit virus-like disease symptoms in the field are found to be sero-negative when tested with antibodies to the common viruses (Mukasa *et al.* 2003; Ateka *et al.* 2004; Tairo *et al.* 2004). In Africa, the severe symptoms and very high yield losses associated with SPVD and similar diseases resulting from mixed infections of several viruses have attracted most attention. Plants with mild symptoms or symptomless infections have been less studied. It is likely that new viruses not presented in Table 1 will be described in the future.

In the United States, virus symptoms are common in the field, but the effects on the crop are more moderate than in Africa. The typical syndrome consists of chlorotic spotting, ringspotting, and vein banding with or without purple borders, depending on the pigmentation of the sweetpotato cultivar. SPFMV is universal, but two other potyviruses, SPVG and Sweet potato virus 2 (SPV2 is synonymous with *Ipomoea vein mosaic virus* [IVMV]) also are common. Recent reports (Carroll *et al.* 2004; Clark and Hoy 2006) show that US sweetpotato yields can be reduced by as much as 30-40%, but that these three potyviruses do not reproduce either the typical syndrome seen commonly in the field, or the effect on yield. Furthermore, in naturally infected plants exposed in the field for seven years, the titers of SPFMV, SPVG, and SPV2 are several hundred fold greater than in virus-tested plants artificially infected with the same three viruses (Clark *et al.* unpublished). This has led to the hypothesis that other yet unidentified viruses are involved in the US chlorotic spotting syndrome and they enhance replication of the potyviruses. Renewed efforts to identify unknown viruses revealed the presence of *Tomato spotted wilt virus* (TSWV) (Clark and Hoy 2007). Studies of TSWV in sweetpotato are in their infancy, and it is not known how widespread this virus is or what effect it has on yield, if any. However, it appears that TSWV is not responsible for the enhanced potyvirus replication and thus the search continues for the potyvirus enhancer. In the US, certain sweetpotato cultivars are grown as ornamental plantings because of their attractive foliage. Interestingly, these sweetpotatoes are

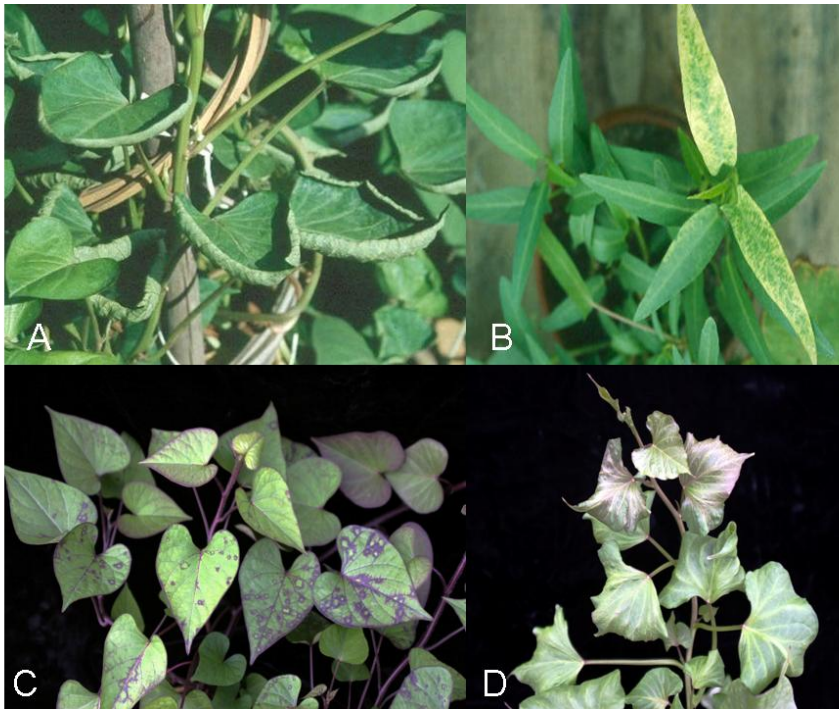


Fig. 1 Symptoms caused by sweetpotato viruses on *Ipomoea* spp. (A) Upward curling at the margin of young leaves of sweetpotato (*Ipomoea batatas*) caused by *Sweet potato leaf curl virus* (SPLCV). Many sweetpotato genotypes remain symptomless when infected with (SPLCV). (B) Yellow mottle on *I. aquatica* caused by SPLCV. (C) Sweetpotato (NC-1554) showing purple ringspots and vein banding typical of those commonly seen in the field in cultivars that normally have purple pigmentation on the leaves. (D) Sweetpotato cv. 'Beauregard' infected with the russet crack strain of *Sweet potato feathery mottle virus* and the White Bunch strain of *Sweet potato chlorotic stunt virus*. The fan-shaped and stunted leaves represent part of the syndrome of sweet potato virus disease.

affected by a completely different complex of viruses than the sweetpotatoes grown for human consumption. The purple-leaved cultivars of ornamental sweetpotatoes are commonly infected with the geminivirus *Sweet potato leaf curl virus* (SPLCV) (Fig. 1A) and the uncharacterized C-6 virus.

There are several unresolved problems in the taxonomy of sweetpotato viruses. Many viruses associated with important symptoms or diseases, such as internal cork, have unfortunately never been isolated or characterized. Some viruses have been described and named, but isolates are no longer available for direct comparison, such as Sweet potato vein mosaic virus (Nome 1973). Thus, it is not possible to determine their relatedness to other sweetpotato viruses. Although SPFMV is common wherever sweetpotatoes are grown, it is evident that there are several distinct phylogenetic clusters within this nomenclature (Karyeija *et al.* 2000b; Kreuze *et al.* 2000; Karyeija *et al.* 2001), and it has been suggested that some of these clusters, such as strain C, be treated as distinct species (Tairo *et al.* 2005). In some cases, these also correlate with important biological properties. For example, the russet crack strain of SPFMV interacts synergistically with SPCSV to induce typical SPVD symptoms (Fig. 1D), but the common strain of SPFMV does not (Souto *et al.* 2003; Kokkinos and Clark 2006b). The time has come for a systematic analysis of the taxonomy of the sweetpotato potyviruses utilizing complete sequence information, establishing priority names and identifying synonymy. A repository with information on the availability of antisera and virus cultures will be helpful to accomplishing this.

DIAGNOSIS AND DETECTION

The development of techniques for virus detection and identification should be a priority of any research program aiming to control viral diseases. Once the virus has been identified, one can develop indexing procedures, search for sources of resistance or develop other control methods. In recent years, progress has been made in developing sensitive techniques for several sweetpotato viruses (Abad and Moyer 1992; Colinet *et al.* 1998; Kokkinos and Clark 2006a; Mukasa *et al.* 2006, Tairo *et al.* 2006). The difficulty in detecting sweetpotato viruses in sweet potato is in some cases due to low virus titers rather than inhibitors or problems with the assays (Karyeija *et al.* 2000b; Kokkinos and Clark 2006a). Nevertheless, diagnosis of sweetpotato viruses is difficult due to the occurrence of mixed

infections, diverse viral strains, and uneven virus distribution within the plant. The universal presence of SPFMV has often masked the presence of other viruses in sweetpotato, especially potyviruses, and hindered efforts to isolate and identify them.

Indexing based on grafts to susceptible indicator plants such as *I. setosa* (Brazilian morning glory) is presumed to be a reliable method for detection of most sweetpotato viruses. Based on earlier observations it has been assumed that this plant was a host for all viruses infecting sweetpotato. However, some sweetpotato viruses such as TSWV do not cause visible symptoms on this host. *I. nil* 'Scarlet O Hara' is another host that produces symptoms in response to most sweetpotato viruses. Mechanical inoculation to other virus indicator hosts such as *Nicotiana benthamiana*, *N. clevelandii* and *Chenopodium quinoa* is also recommended (Moyer and Salazar 1989). In addition to grafting to *I. setosa* also grafting to *I. aquatica* a host that is not susceptible to SPFMV may reveal infections by SPLCV which induces vein yellowing on *I. aquatica* (Fig. 1B). The indexing procedures require considerable time, labor and greenhouse space.

Although biological properties remain very important in sweetpotato virus diagnosis, properties of the viral coat protein and nucleic acid are two categories widely exploited by diagnosticians. Several techniques have been developed and have been used in sweetpotato virus diagnosis. These include polymerase chain reaction (PCR), reverse transcription PCR (RT-PCR), molecular hybridization, electrophoretic analysis of dsRNA, western blot, enzyme-linked immunosorbent assay (ELISA), and serologically specific electron microscopy (Derrick 1973; Clark and Adams 1977; Abad and Moyer 1992; Colinet *et al.* 1998). The use of one or a combination of these techniques to complement indexing is recommended.

ELISA

Serology of virus particles and coat proteins has been widely used for the identification of plant viruses. ELISA has become the preferred test for plant viruses because of its simplicity, adaptability, sensitivity, and accuracy. Even though ELISA has been used for sweetpotato virus detection, there are some limitations. The low concentration and irregular distribution of viruses in sweetpotato are frequently cited as obstacles (Esbenshade and Moyer 1982). Another factor is the presence of phenolics, latex and inhib-

itors in sweetpotato tissue that adversely affect tests (Abad and Moyer 1992). However, many of these obstacles can be avoided by grafting scions of the tested sweet-potato plants to *I. setosa* and testing these indicator plants systematically infected with the viruses instead of the original, sampled sweetpotato plant. A membrane immuno-binding assay also known as nitrocellulose membrane ELISA (NCM-ELISA) has been used with success to detect several sweetpotato viruses (Abad and Moyer 1992; Gutierrez *et al.* 2003; Mukasa *et al.* 2003a; Souto *et al.* 2003; Tairo *et al.* 2004; Valverde and Moreira 2004). Detection kits using this technique have been developed by the International Potato Center. They are very practical, particularly in developing countries where the use of other methods is limited by the available resources.

Molecular hybridization

Nucleic acid hybridization, procedures have been developed for detection of several sweetpotato viruses. Probes consist of labeled viral DNA or RNA with radioactive or non-radioactive labels. A method using a SPLCV-specific probe direct-labeled with horseradish peroxidase and detected with chemiluminescence, has been tested with DNA samples from *I. batatas* and other *Ipomoea* species (Valverde *et al.* 2004a). Strong signals were obtained when tests were conducted using DNA from field (sweetpotato) and greenhouse (*Ipomoea* spp.) grown plants that were infected with SPLCV. Abad and Moyer (1992) developed *in vitro* transcribed RNA probes for SPFMV detection. The probes were developed using cDNA containing the 3'-terminal region of the capsid protein cistron. The riboprobe was more sensitive than immunological assays because it could overcome interference with host factors that compromise the reliability of immunodiagnostic assays.

PCR

PCR offers several advantages compared to traditional methods of plant virus detection. The sensitivity of PCR is an advantage for detection of viruses that are unevenly distributed and are present at very low levels in infected plants. SPLCV has been detected from both indicator hosts and sweetpotato by PCR using virus specific and degenerate primers (Onuki and Hanada 1998; Lotrakul 2000; Li *et al.* 2004). Although SPLCV and related begomoviruses can be detected by PCR, it is not yet possible to detect them inexpensively. An RT-PCR assay utilizing degenerate primers corresponding to the border sequences of the N1b and the coat protein genes has been used for detection of sweetpotato potyviruses (Colinet *et al.* 1998; Souto *et al.* 2003). Sequencing of PCR products has been used to determine the taxonomic status of these viruses (Colinet *et al.* 1998; Mukasa *et al.* 2003b; Souto *et al.* 2003). Degenerate primers have been developed that can be used to amplify a portion of the gene that encodes the heat shock protein 70 homologue (HSP70) present in all known members of the family *Closteroviridae*. SPCSV has been detected by reverse transcription PCR (RT-PCR) using HSP70 primers (Sim *et al.* 2000). Real-time PCR has been used to detect and quantify sweetpotato viruses (Kokkinos and Clark 2006a; Mukasa *et al.* 2006). Kokkinos and Clark (2006a) found using real-time PCR assays that titers of the potyviruses SPFMV, SPV2, and SPVG were near the threshold of detection in singly infected sweetpotato plants, but much greater in *I. setosa* and *I. nil* cv. 'Scarlet O'Hara'. The results of that study showed that real-time PCR was a more sensitive method for the detection and quantification of sweetpotato viruses directly from sweetpotato plants compared with traditional assays. Since real-time PCR is more sensitive than ELISA, this indicates that low titers of these viruses probably account for underestimation of the prevalence of these viruses in ELISA assays directly from sweetpotato (Gibson *et al.* 1997; Karyeija *et al.* 2000a, 2000b). This confirms the advantage of graft indexing on *I. setosa*

for sensitive detection.

PCR-based approaches may also be helpful for sorting out viruses that infect sweetpotato plants as a complex. The procedure devised for detecting and distinguishing SPV2 and two strains of SPFMV from sweetpotatoes in Australia included reverse transcription of viral RNA with oligoT₂₅ primer, PCR using a combination of degenerate primers, and restriction analysis of the 1.8-kb amplification products with *Hind*III and *Pvu*II endonucleases (Tairo *et al.* 2006). Similar procedures can be designed for viruses from which sequence data of several strains and isolates are available.

SWEETPOTATO VIRUS DISEASE COMPLEXES

SPVD can cause yield reductions of 80-90%. It is the result of a synergistic interaction between the whitefly-transmitted crinivirus (*Closteroviridae*), SPCSV and the aphid-transmitted potyvirus (*Potyviridae*) SPFMV. This disease was first noted in eastern Belgian Congo (now Democratic Republic of Congo) in 1939 and was for many years considered a regional problem of sub-Saharan Africa (Carey *et al.* 1999). While SPFMV is universally distributed, SPCSV was initially only recognized in Africa. However, it has become apparent in recent years that disease complexes involving SPCSV also occur in Spain, South America and Central America (Carey *et al.* 1999; Di Feo *et al.* 2000; Gutierrez *et al.* 2003; Valverde and Moreira 2004; Valverde *et al.* 2004).

Several studies have revealed interesting aspects of the interaction between SPCSV and SPFMV. Karyeija *et al.* (2000a) showed that SPCSV enhances the accumulation of SPFMV by approximately 600-fold. This is unusual in that while potyviruses are often involved in synergistic interactions, more commonly they are the enhancer, as opposed to SPVD where SPFMV is the enhanced virus. There are also indications that SPCSV may broadly enhance the replication of several other sweetpotato viruses. Mukasa *et al.* (2006) showed that *Sweet potato mild mottle virus* is also enhanced by SPCSV, with virus titers increased approximately 1000-fold. The combined infection caused severe symptoms, and the name sweetpotato severe mosaic disease was suggested for the resulting disease. Kokkinos and Clark (2006b) found that SPCSV enhances replication of SPV2 (IVMV), *Sweet potato virus G* (SPVG), and both the russet crack and common strains of SPFMV. Symptoms from the mixed infections differed qualitatively, but were commensurate in severity with the enhanced replication of the potyvirus component, except for the SPFMV-C/SPCSV combination. Even though the titer of SPFMV-C was enhanced, plants infected with SPFMV-C and SPCSV only developed mild symptoms typical of SPCSV infection by itself. This suggests that enhancing accumulation of the potyvirus component is alone not sufficient for SPVD development. Both studies (Kokkinos and Clark 2006; Mukasa *et al.* 2006) showed that titers of SPCSV were decreased in the mixed infections compared to single infections, suggesting an antagonistic effect. Untiveros *et al.* (2007) found synergistic interactions between SPCSV and carla- and cucumoviruses in addition to ipomo- and potyviruses. Thus, although there are numerous potential interactions among sweetpotato viruses, it has become evident that SPCSV is the key element causing enhancement of a broad array of other viruses.

Kokkinos *et al.* (2006) used microarray technology to compare the effects of single infections with SPFMV-RC and SPCSV with concomitant infection on expression of sweetpotato genes. Even though the array represented only a portion of the sweetpotato genome, there was a dramatic difference in the number of genes that were differentially expressed: SPFMV – 3 genes, SPCSV – 14 genes, and SPFMV + SPCSV – >200 genes.

Plant viruses are capable of rapid evolution to overcome the plant host defenses. Several plant viruses have been shown to encode proteins that are suppressors of the RNA silencing process (Carrington *et al.* 2001). These suppressors are diverse in sequence and structure and appear to be encoded by virtually any type of plant viruses (Moissiard

and Voinnet 2004). RNA silencing is a host defense mechanism targeted against invasive or mobile RNA elements such as viruses or transposable retro-elements, leading to sequence-specific degradation. In plants, this is known as post-transcriptional gene silencing (PTGS) (Vance and Vaucheret 2001). When a plant virus infects a host cell it activates an RNA-based defense that is targeted against the viral genome.

A synergistic interaction in which a coinfection of *Potato virus Y* (PVY, genus *Potyvirus*) causes an increase in titer of a second, unrelated virus, *Potato virus X* (PVX, genus *Potexvirus*), in tobacco plants has been reported (Vance 1991). The titers of PVX RNA and coat protein increase and more severe symptoms are induced, but the titer of PVY is not affected. This synergism was the result of suppression of a host defense mechanism by the multifunctional helper component-proteinase (HCPro) of potyviruses. Later it was established that HCPro was a suppressor of PTGS (reviewed in Vance and Vaucheret 2001).

A possible explanation of the synergism between SPFMV and SPCSV is that the host actively inhibits optimum multiplication of SPFMV and that SPCSV suppresses the resistance mechanism. Kreuzer *et al.* (2005) reported an RNase III with dsRNA-specific endonuclease activity that enhances the RNA-silencing suppression activity of another protein (p22) encoded by SPCSV. These two independent proteins are cooperatively involved in RNA silencing suppression.

Both RNA and DNA plant viruses encode suppressor proteins of silencing. It has been shown that geminiviruses both induce and probably also are targets of PTGS (Voinnet *et al.* 1999). Geminivirus infection does not include dsRNA replicative forms of the virus, although the mRNAs for the expression of the geminiviral genes could be subject to PTGS. Chellappan *et al.* (2004) suggest that in the case of geminiviruses, dsRNA could be formed by the virion-sense and complementary-sense transcripts annealing to each other due to a short overlap at their 3' ends.

Experiments with single and mixed infections of SPFMV-95-2 and SPLCV yielded surprising results since SPLCV DNA titer increased in mixed infections, while that of SPFMV-95-2 remained the same (Kokkinos and Clark 2006). These results suggest that the HCPro of SPFMV-95-2 is acting as the suppressor of gene silencing for SPLCV. Although, symptoms on sweetpotato were not dramatically affected by mixed infections of SPFMV-95-2 and SPLCV, preliminary results on *I. setosa* indicate that mixed infections of these two viruses cause more severe symptoms than either alone.

In addition to SPVD, other viral disease complexes of sweetpotato have also been described, which invariably seem to involve SPCSV. In Israel, CMV was found infecting sweetpotato together with SPCSV (also known as Sweet potato sunken vein virus) and usually also SPFMV, producing symptoms similar to SPVD and causing up to 80% reduction in yield (Cohen and Loebenstein 1991). It was shown that CMV could only infect sweetpotato if the plants were first infected with SPCSV (Cohen *et al.* 1988). Interestingly, this seems not to be the case for CMV in Egypt, where it is found infecting sweetpotato with or without SPCSV (IsHak *et al.* 2003). CMV has not been detected infecting sweetpotatoes in the field in the United States although there are old reports of diseases of sweetpotato involving mosaic symptoms (Martin 1962) and there have been many reports of many strains of this virus infecting other crops in the United States. In preliminary experiments, we have found pepper isolates of CMV that could infect *I. nil* (single infections) but failed to infect sweetpotato.

In the US, the situation with SPCSV has been confusing. SPVD symptoms have not been observed in the field in recent years. Nevertheless, SPCSV (West African strain) was found in a single tissue culture accession in a germplasm collection (Pio-Ribeiro *et al.* 1996). The origins of the sweetpotato accession and its infection are unclear, making it difficult to trace back possible origins of the

SPCSV. Abad *et al.* (2007) reported finding SPCSV in two fields and provided additional indications that the virus was not a recent introduction to the US. It is interesting to speculate that 'Georgia Mosaic' episodes that were reported in the 1950s and subsequently reported to be eradicated (Borders and Ratcliffe 1954; Girardeau and Ratcliffe 1963) represent a prior introduction of SPCSV, but it will be difficult to ever establish the connection. Regardless, it now appears that SPCSV has been present in the US, and it has long been known that SPFMV is universally associated with sweetpotato in the US (Clark and Moyer 1988), yet SPVD symptoms are rare, at best. Perhaps if we can learn why SPVD is rare in the US despite the fact that the causal viruses, their vectors, and susceptible cultivars are all present, a basis would be found to develop strategies to mitigate SPVD in regions where it is a serious threat.

SWEETPOTATO BEGOMOVIRUSES – AN OVERLOOKED COMPONENT

Until recently, most surveys of sweetpotato viruses did not mention begomoviruses, even though leaf curl symptoms caused by unknown viruses have been observed for many years (Clark and Moyer 1988). SPLCV was first reported in the USA by Lotrakul *et al.* (1998) and in Japan by Onuki and Hanada (1998). A molecular characterization of the US isolate of SPLCV was conducted by Lotrakul and Valverde (1999). An apparently distinct geminivirus (*Ipomoea* crinkle leaf curl virus), was also found in sweetpotato in Israel (Cohen *et al.* 1997). *Ipomoea yellow vein virus* formerly known as SPLCV-Ipo, has been found in *I. indica* in Spain and Sicily (Banks *et al.* 1999; Briddon *et al.* 2006). SPLCV or related begomoviruses have been reported from sweetpotato in South America (Fuentes and Salazar 2003), the US (Lotrakul *et al.* 2003), East Africa (Miano *et al.* 2006), Spain (Lozano *et al.* 2004), and China (Luan *et al.* 2006).

It is now evident that begomoviruses are associated with sweetpotato in most, if not all, geographic regions where sweetpotatoes are grown, but the prevalence and distribution of the viruses within these regions is not known. Although these reports are recent, there is evidence that there is considerable variability among the strains of begomovirus represented in these reports (Lotrakul and Valverde 1999; Lotrakul *et al.* 2002) and prior observations of leaf curl symptoms suggest that these viruses were present long before they were reported. Some of the strains either do not induce symptoms or induce very mild, transient symptoms in the standard indicator host, *I. setosa*. Some genotypes of sweetpotato, such as cv. 'Beauregard', do not show any symptoms when infected with SPLCV (Clark and Hoy 2006). Genotypes that do develop the characteristic upward curling at the margins of young leaves generally do so only during warm periods of the year and may require the presence of other viruses for symptom development (Clark *et al.* 2002). Recent development of sensitive PCR (Li *et al.* 2004) and real-time PCR (Kokkinos and Clark 2006a) assays have helped us to recognize that SPLCV and related begomoviruses have sometimes escaped detection prior to the advent of these technologies. The diversity among sweetpotato begomoviruses reported (Cohen *et al.* 1997; Onuki and Hanada 1998; Banks *et al.* 1999; Onuki *et al.* 2000; Lotrakul *et al.* 2002; Briddon *et al.* 2006), may also indicate that these viruses undergo a high rate of recombination, similar to reports for other geminiviruses (Seal *et al.* 2006).

The potential importance of begomoviruses is indicated in part by the study of Clark and Hoy (2006) in which they found that yields of 'Beauregard' sweetpotato were reduced by 25-30% by SPLCV despite the fact that no symptoms were observed on the plants. Some farmers may believe that selecting symptomless vine cuttings for propagating material from genotypes that have virus resistance (Gibson *et al.* 1997), and/or that roguing symptomatic plants from production fields can be used as an effective means of managing SPVD (Gibson *et al.* 2004). Begomoviruses have the poten-

tial to compromise these efforts as they are capable of causing significant yield effects without inducing apparent symptoms. There is a need therefore, to incorporate specific begomovirus test procedures into sweetpotato virus indexing protocols and to determine the prevalence and effects of this group of viruses in those regions where begomoviruses have recently been reported.

CONTROL OF SWEETPOTATO VIRUS DISEASES

Attempts at controlling sweetpotato viruses are relatively recent, and generally involve either use of resistant cultivars or 'clean seed' programs. The relative merits of these two approaches are viewed quite differently in various countries with different production systems.

Approaches to using virus resistance

Resistance is an attractive option for disease management as it generally does not require significant expenditures by the grower. In the case of sweetpotato, there have likely been unintended gains in development of virus resistance as breeders and farmers have both selected for high yield, and/or mild symptoms, in plantings that were exposed to natural virus infection, but this has not been documented. There have also been notable successes in producing cultivars that were resistant to development of certain symptoms, such as internal cork or russet crack, despite a lack of knowledge of the etiology of these disorders (Clark and Moyer 1988). However, efforts targeted at true resistance to specific viruses or virus complexes are relatively recent.

Initial attempts to develop virus resistance in sweetpotato focused on SPFMV because of its universal distribution. Genotypes have been described as resistant to SPFMV in different countries and Mihovilovich *et al.* (2000) used graft inoculation techniques to study resistance to SPFMV. They found significant general combining abilities but no significant specific combining abilities and suggested that additive gene action is important in resistance to SPFMV. However, genotypes described as resistant to SPFMV in Peru were found to be susceptible in East Africa (Mwanga *et al.* 1991; Gibson *et al.* 1998; Karyeija *et al.* 1998b; Mwanga *et al.* 2002). Subsequent studies have demonstrated that there is not only a diversity of potyviruses infecting sweetpotato worldwide, but considerable diversity within what has been called SPFMV (Kreuze *et al.* 2000), which greatly complicates efforts at developing resistance. In the case of SPCSV, there are also different serotypes, and sweetpotato genotypes found resistant to SPVD in Nigeria where the West African serotype of SPCSV predominates were susceptible in Uganda where the East African serotype is predominant (Mwanga *et al.* 1991; Alicai *et al.* 1999b; Carey *et al.* 1999). There can also be considerable diversity of viruses within a location, as has been found for SPFMV, SPCSV, and SPMMV (Mukasa *et al.* 2003b, 2003c; Tairo *et al.* 2005). Tairo *et al.* (2005) discuss the implications of diversity of sweetpotato viruses for efforts to breed for resistance. The important point is that it is essential to assure that resistance is sufficiently comprehensive to provide protection from local strains. Furthermore, Karyeija *et al.* (2000a) demonstrated that infection with SPCSV overcomes resistance to SPFMV.

In East Africa, many farmers grow landraces that have been shared through generations. Although SPVD is a limiting factor to sweetpotato production in sub-Saharan Africa (SSA), the impact of the disease is mitigated by use of resistant cultivars and landraces selected from local germplasm (Aritua *et al.* 1998b; Karyeija *et al.* 1998a). When non-indigenous genotypes, such as high-yielding North American cultivars are grown in SSA, most plants quickly develop SPVD symptoms and their yield is dramatically reduced (Aritua *et al.* 2000). By contrast, plantings of locally adapted cultivars generally have a lower proportion of plants with SPVD symptoms (25-30% reported by

Aritua *et al.* 1998a), the symptoms appear later, plants may recover from SPVD, and the yield reduction attributable to SPVD is not as great. Sweetpotato is an indeterminate plant without a defined physiological maturity, and as such, storage roots may continue to enlarge for a long time. Unfortunately, many of these cultivars are slow yielding (Gibson *et al.* 2000), producing an acceptable yield only after very long growth periods, and it has been said that "Widespread use of less productive SPVD-resistant landraces may be the most damaging consequence of SPVD" (Gibson *et al.* 2004). Gibson *et al.* (2000) lamented the lack of attention to sweetpotato seedlings by farmers and the consequent lack of development of SPVD-resistant landraces with improved yielding ability. There thus remains a need for improved SPVD-resistant cultivars that produce acceptable yields in a shorter time, combined with other desired characteristics, and this is the focus of ongoing research.

Several characteristics of sweetpotato make it a difficult crop in which to breed for resistance (Mwanga *et al.* 2002; Mcharo *et al.* 2005). It is a highly heterozygous allohexaploid ($2n = 90$) with complex segregation ratios and many traits are inherited quantitatively. Self-incompatibility makes it time consuming to introgress desirable traits. Nevertheless, Hahn *et al.* (1981) used a core graft transmission technique to screen sweetpotato clones for resistance to SPVD and found a high broad sense heritability for resistance.

In recent years, public institutions have become more active in developing and releasing SPVD-resistant cultivars (Mwanga *et al.* 2001, 2003). A series of publications by Mwanga and co-workers has provided a foundation for studies of genetics of resistance to SPVD. Ten parental clones were originally selected for resistance to SPVD based on reactions observed in the field under conditions of natural inoculation (Mwanga *et al.* 2001). Unfortunately, only two were considered resistant to SPVD and the limited availability of resistant genotypes has limited heritability studies on SPVD. Nevertheless, these parents were crossed in a half diallel mating design and progeny were challenged by simultaneous graft inoculation with SPFMV and SPCSV (Mwanga *et al.* 2002a). All progeny initially developed severe SPVD symptoms and the frequency distribution of SPVD severity scores among diallel families was skewed to the susceptible ratings, but there were significant differences in severity ratings among parents. Full sib families all differed significantly for recovery from SPVD, indicating that recovery is an important attribute in assessing resistance to SPVD. Almost all of the inoculated plants tested positive for SPFMV by DAS-ELISA and SPCSV by TAS-ELISA and those that tested negative could be infected by repeated graft inoculation and were positive by indexing on *I. setosa*. Although the resistance to SPVD observed in these progeny was considerably less than the high level of resistance that has been observed in other *Ipomoea* species (Karyeija *et al.* 1998a), Mwanga *et al.* (2002b) found moderate narrow-sense heritability and high broad-sense heritability for resistance to SPVD and suggested that rapid gains in SPVD resistance could be made by mass selection techniques. They identified cvs. New 'Kawago' and 'Sowola' as promising parents for improving resistance to SPVD (Mwanga *et al.* 2002a, 2002c). In another study, Mwanga *et al.* (2002b) found two amplified fragment length polymorphism (AFLP) markers, one that explained 70% of total variation for resistance to SPCSV and another that explained 66% of total variation for resistance to SPFMV. They also suggested "...that additional genes mediate oligogenic or multigenic horizontal (quantitative) resistance to both viruses." (Mwanga *et al.* 2002b). Mcharo *et al.* (2005) extended the findings in a study in which they used three analytical procedures: quantitative trait loci (QTL) analysis, discriminant analysis, and logistic regression to separate DNA marker profiles. The latter two techniques revealed one important marker for SPCSV and two for SPFMV that had not been identified by QTL analysis and they found that prediction accuracy could be elevated

to 96% using as few as four markers for SPFMV and six for SPCSV.

There has naturally been interest in developing transgenic forms of resistance to sweetpotato viruses. One highly publicized effort (Wambugu 2003) involved transformation of some East African cultivars with the coat protein gene from the russet crack strain of SPFMV. When finally evaluated in the field, these transformants were no more resistant to SPVD than the nontransformed lines (New Scientist, 7 Feb 2004, p 7), possibly because the East African and common strains of SPFMV are the common strains in SSA (Tairo *et al.* 2005). Because SPCSV can cause yield loss on its own, and because it synergises with most other sweetpotato viruses, it has been suggested that efforts to develop SPVD resistance in sweetpotato should target SPCSV (Tairo *et al.* 2005; Aritua *et al.* 2007).

Aritua *et al.* (1998) studied SPVD development on three sweetpotato clones in the field in Uganda and found that aphids were absent and whiteflies were equally abundant on all three clones and they concluded that differences in susceptibility among these three clones to SPVD were not attributable to differences in susceptibility to the virus vectors, but likely due to differences in susceptibility to infection by one of the component viruses. Beyond this study, there is little published information to indicate whether resistance to aphids or whiteflies exists in sweetpotato germplasm.

Progress has been made on developing resistance to sweetpotato viruses, but little is known about the nature of tolerance and/or resistance. For SPFMV, SPCSV, or SPVD, genotypes have been characterized as resistant if they fail to develop symptoms under natural inoculum pressure in the field, or in other cases following graft inoculation. However, these two approaches may actually measure different phenomena. For example, Beauregard, which has been called susceptible to SPFMV because there is often high incidence of infection in the field (Clark and Hoy 2006), develops a very low titer of this virus (Kokkinos and Clark 2006), is nearly symptomless when infected with SPFMV, and yield is not greatly affected when plants are artificially inoculated with SPFMV (Clark and Hoy 2006). Given these observations, apparent susceptibility to SPFMV may be because of increased attractiveness to aphid vectors rather than a specific reaction of the host to the virus. SPLCV does not induce symptoms in the foliage of most genotypes examined, but may present the simplest interaction with cultivars of sweetpotato. Clark *et al.* (2004) studied seven genotypes and found dramatic differences in the effect of SPLCV infection on yield. Cvs. 'Bienville' and 'Jonathan' were especially sensitive and yields were reduced as much as 75% while yields of cvs. 'Xushu-18' and 'Picadito' were unaffected. Estimates of SPLCV titer by real-time quantitative PCR indicated that titers were very high in 'Bienville' and very low in 'Xushu-18' and 'Picadito' (Kokkinos 2006). Thus, each virus may be different in how it interacts with sweetpotato genotype and how virus titer correlates with symptom development and effects on yield. While it will be valuable to know whether resistance affects virus titer, suitability of plants as sources of virus acquisition, and/or systemic movement of the virus, we recommend that the terms resistance/susceptibility be used only to describe the performance of the virus within the sweetpotato host plant and that tolerance/susceptibility be used to describe the difference in performance between an infected host and an uninfected plant (Carr 2006).

There has been an impressive expansion of knowledge about sweetpotato viruses in Africa and resistance to these diseases, however, it also illustrates our lack of understanding of the etiology of virus diseases in other parts of the world. The concern is especially great in the Americas, which includes the putative center of origin of sweetpotato, and the South Pacific which is a secondary center of diversity in sweetpotato (Tairo *et al.* 2005). There are also indications that there may be unidentified viruses still in SSA

(Mukasa *et al.* 2003a). This deficiency retards not only development of virus resistance adapted for these regions, but also poses a risk of inadvertent introduction of unknown viruses to other areas where they might contribute to overcoming existing resistance (Tairo *et al.* 2005).

Seed programs and phytosanitary measures

Technologies for eliminating viruses from plants by meristem-tip culture and for indexing sweetpotatoes for viruses are not new. However, most programs for providing farmers with propagating material that is relatively free of viruses were only deployed in the last 20 years. While there are a number of published reports that describe the effects sweetpotato viruses can have on yield and quality of the crop (Ma *et al.* 2002; Bryan *et al.* 2003b; Njeru *et al.* 2004; Clark and Hoy 2006; Mukasa *et al.* 2006), we are only aware of one in-depth evaluation of the impact of a clean seed program. Fuglie *et al.* (1999) studied the impact of the "virus-free" production system in Shandong province, China. They estimated that from its inception in 1994, this program was extended to approximately 80% of the province's 500,000+ hectares by 1998. Farmers' responses to surveys and data from demonstration plots indicated yield increases of over 30%. They found the virus-free seed was adopted in both poor and richer villages, regardless of whether sweetpotato was an important crop for the village. The program in China may have been successful because at the time it started, the cost of virus-free seed was low relative to the benefits gained from higher yields. The benefits were enhanced by demand from the food processing industry which kept prices from falling in response to increased supply. Except for California, sweetpotato producing states in the US did not start virus-tested seed programs until about 1999. Bryan *et al.* (2003a) suggested that returns to farmers were potentially greater when farmers grew their crop from seed 1-2 years away from the original virus-tested seed because of the lower cost of producing the seed even though yields gradually declined through this period. Actual impact of the US programs has not been investigated in the depth that was done for the Shandong program.

The success of and necessity for seed programs to manage sweetpotato viruses may depend on a number of factors in addition to the economic factors considered by Fuglie *et al.* (1999). In a study in East Africa, no benefit of using 'virus-free' planting material was seen (Gibson *et al.* 1997; Carey *et al.* 1999). However, many of the cultivars grown there are slow-yielding and have some level of resistance to SPVD, and are less likely to show a similar yield response from clean seed. In addition, these studies compared 'virus-free' seed with traditional planting material, which in East Africa usually consists of plants selected as free of symptoms and therefore less likely to be infected than the crop overall. In most of SSA, production is by small-scale farmers for home consumption, and economic constraints and limitations of infrastructure make establishment of a clean seed program especially difficult. Without data on how high-yielding SPVD susceptible cultivars perform in this environment using clean seed technology, it is difficult to judge the potential value of a clean seed program.

Since SPVD induces dramatic symptoms, it is possible to readily identify affected plants in the field, unlike other virus diseases of sweetpotato. Furthermore, some of the resistant landraces in Africa often produce branches or shoots on the plant that are free of symptoms, and in many cases, also free of viruses for which they have been tested (Gibson *et al.* 1997). This provides farmers both an opportunity to select planting material that is free of symptoms and also to rogue the crop after planting to remove plants with SPVD. It has been reported by several workers that virus distribution can be very uneven in sweetpotato plants, but there remains a real need to evaluate how factors such as rate of plant growth, mixed infections with multiple viruses, environmental variables, host resistance and others affect dis-

tribution of viruses and especially their systemic movement toward the shoot tip.

Epidemiology and management strategies

Understanding the epidemiology of diseases is critical to implementing and evaluating strategies for their management. Gradually, the deficiencies in our understanding of etiology of sweetpotato virus diseases is being overcome and control tactics are being developed and deployed, as outlined above. As these necessary prerequisites are attained, emphasis is shifting to using this knowledge to develop comprehensive management strategies for each region that are based on a sound understanding of the epidemiology of the locally important viruses.

Study of the epidemiology of sweetpotato viruses is particularly difficult because of the lack of tools to enable inexpensive, sensitive detection of the viruses directly from sweetpotato. While some studies have used ELISA or PCR methods, it is clear that false negatives can result as compared to indexing on *I. setosa*, especially if single infections by SPFMV are involved (Gibson *et al.* 1997; Kokkinos and Clark 2006b; Aritua *et al.* 2007). The necessity of biological indexing presents a great bottleneck to epidemiological studies and a real boon would be realized if a sensitive, inexpensive alternative could be devised. There also has been very little effort to study the relationships of sweetpotato virus vectors.

As a few studies related to epidemiology of sweetpotato viruses have come out, it appears that there are some fundamental differences in virus spread between different geographic regions and cropping systems. In Brazil in the cvs. 'Brazlândia Branca', 'Brazlândia Roxa', and 'Coquinho', and in the US in the cv. 'Beauregard', SPFMV rapidly infects sweetpotato plants in the field and can infect 100% of plants within the first season (Pozzer *et al.* 1994; Bryan *et al.* 2003a; Clark *et al.* 2004), and in one report, as early as nine weeks after planting (Bryan *et al.* 2003b). Even though reinfection with SPFMV occurs rapidly, decline in yield is gradual over several years (Clark *et al.* 2002; Bryan *et al.* 2003a; LaBonte *et al.* 2004), suggesting the involvement of other viruses. In the cultivar Georgia Jet in Israel, only SPCSV was found in a crop planted entirely from healthy stock, but when SPFMV was present in the planting material, SPFMV spread very rapidly (Milgram *et al.* 1996). On the other hand, surveys in Africa, although they report SPFMV to be the predominant virus, often report much lower incidences of SPFMV (Gibson *et al.* 1997; Aritua *et al.* 2007). There could be many reasons for the apparent difference, including differences in wild hosts as sources of viral inoculum, vector abundance and/or movements, and differences in cultivar resistance. However, Clark *et al.* (2004) found high rates of infection in the US in 'Tanzania', a cultivar with resistance to SPFMV that is grown commonly in East Africa (Mwanga *et al.* 2002b). Another factor may be the abundance of high-titer source plants from which the aphid vectors acquire SPFMV in the field. Aritua *et al.* (1998) found that SPFMV became very difficult to detect in singly infected plants and aphids did not readily acquire the virus from such plants but readily transmitted the virus from plants with SPVD. Since co-infection with SPCSV causes a dramatic increase in titer of SPFMV (Karyeija *et al.* 2000a; Kokkinos and Clark 2006b), it is not surprising that acquisition of SPFMV is much greater. However, some of the greatest rates of spread of SPFMV in the field seem to occur in locations where SPCSV is not detectable (Bryan *et al.* 2003b; Clark *et al.* 2004). There is a critical need in these situations to determine what other factors are favoring rapid spread of SPFMV.

Byamukama *et al.* (2004) studied populations of whiteflies and aphids and spread of SPVD from an infected block at the center of a field planted to the cultivar 'Tanzania', which is resistant to SPFMV but moderately susceptible to SPVD. They trapped whiteflies within and outside

the crop, but in greater numbers close to the crop canopy. Aphids did not colonize the crop, but were trapped near and above the canopy in equal numbers and it was suggested that these were itinerant alate aphids originating from other hosts. SPVD spread only a short distance from the central infected block. Spread was much greater from the central block than from outside the crop, and SPVD spread only short distances. Thus, the authors inferred that older infected fields closely neighboring new fields, or infected plants inadvertently planted by farmers were the most likely sources of new infections. They surmised that roguing would be an effective means of limiting spread of SPVD. These observations were borne out in another study in which Gibson *et al.* (2004) found that roguing out plants with SPVD symptoms at one month after planting and isolating healthy plantings from SPVD-affected plants by 15 m greatly decreased spread of SPVD to a susceptible cv. ('Tanzania'). Furthermore, SPVD-resistant cultivars out yielded some local SPVD-resistant landraces. Widespread planting of SPVD-resistant cultivars may also lead to a reduction of incidence of SPVD in susceptible cultivars grown nearby (Aritua *et al.* 1999). Strategies combining these tactics may allow farmers to grow better yielding cultivars with lesser degrees of SPVD resistance (Gibson *et al.* 2004). SPVD is most prevalent in drier regions of East Africa where whiteflies are also more abundant, but fluctuations in whitefly populations within a given region are not necessarily correlated with SPVD incidence (Aritua *et al.* 1998a; Alicai *et al.* 1999; Aritua *et al.* 2007).

The role of wild hosts in the epidemiology of sweetpotato virus diseases has not been critically evaluated. Clark *et al.* (1986) found that certain perennial species could serve as overwintering reservoirs of SPFMV in a temperate environment in the US. Tugume *et al.* (2005) also surveyed wild *Ipomoea* species in Uganda and found that about 36% of the plants were infected with SPFMV, SPMNV, SPCSV, or SPCFV or a combination of these viruses. While it is clear that some of these hosts may serve as virus reservoirs, it is not clear to what extent they may affect epidemic development in sweetpotato. Since titers of some viruses, such as SPFMV, SPV2 (IVMV), and SPVG, are very low in sweetpotato but much higher in some other *Ipomoea* species (Kokkinos and Clark 2006a), it is possible that some may serve as more efficient hosts for virus acquisition by vectors in the field, but this has not been examined.

It is tempting to extrapolate the knowledge of sweetpotato virus diseases gained in Africa to other locations. However, SPVD lends itself more readily to roguing not only because the disease spreads short distances, but also because symptoms of SPVD are so dramatic that affected plants are readily identified in the field. It also appears that at least one of the component viruses, SPFMV, spreads more slowly in East Africa than in other locations.

CONCLUSIONS

Past research on sweetpotato viruses has not only been limited by lack of financial support, but also has focused on local or regional interests. It has become obvious that there is little reliable information on geographic distribution of sweetpotato diseases, and some important viruses, such as SPLCV and SPCSV, are more widely distributed than was previously recognized. Many other questions remain: How do mixed infections that enhance the titer of SPFMV affect virus acquisition by vectors and spread in the field? What roles do wild hosts, some of which may support greater replication of SPFMV play? Most important, however, is why does SPVD not occur in some places where SPFMV is universal, SPCSV has been detected, aphids and whiteflies are present, and the dominant cultivars are highly susceptible?

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