

Weather

Climate variability affects temperature and precipitation patterns, mosquito abundance and survival, and therefore SLEV transmission. Annual temperature changes based on the El Niño/southern oscillation in the Pacific alter precipitation and temperature patterns over the Americas and cycle with varying intensity at 3–5 year intervals. These cycles alter storm tracks that affect mosquito and avian abundance, the intensity and frequency of rainfall events, and groundwater depth, all related to SLEV risk. Above-normal temperatures have been especially necessary for northern latitude SLEV epidemics, because elevated temperatures are required for effective SLEV replication within the mosquito host.

Prevention and Control

Effective vector control remains the only approach available to suppress summer virus amplification and prevent human infections. Best results are achieved using an integrated management approach that focuses on mosquito vector population suppression through habitat inspection and larviciding. Failure of larval management can be followed by emergency adult control focusing on reducing the force of transmission and preventing human infection. Protection of the human population by vaccination does not seem cost-effective or prudent, because there is no human-to-human transmission, few human infections produce disease, and infection rates remain relatively low, even during epidemics. However, if regional infection rates were to become high, thereby placing selected cohorts at high risk for disease, then selective

vaccination may be warranted. There currently is no approved commercial vaccine for SLEV, although vaccination against other flaviviruses such as JEV may impart some protection. Control of avian hosts such as house sparrows and pigeons in urban situations could be done, but this approach is not generally acceptable to the public. Notification of the public of infection risk through the media and the wide scale use of personal protection through changes in behavior (staying indoors after sunset) and/or repellent application were credited with reducing the number of infections during the 1990 epidemic in Florida.

See also: Japanese Encephalitis Virus; Tick-Borne Encephalitis Viruses.

Further Reading

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Sweetpotato Viruses

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Glossary

Cultivar decline A vegetatively propagated cultivar suffering from reduced vigor as a result of a chronic (but often symptomless) disease.

Differential hosts Special species of plants varying in susceptibility to a given disease agent, such that their distinctive symptoms facilitate a presumptive identification of the causal agent.

Indexing Any procedure for demonstrating the presence of a pathogen(s) in susceptible plants. The virus indexing combines information on

viruses with methodologies for their detection to assure effective safe movement of sweetpotato germplasm.

Introduction

Sweetpotato

Sweetpotato (*Ipomoea batatas* (L.) Lam.) is a dicotyledonous, perennial plant, producing edible tuberous roots. It belongs to the family Convolvulaceae, the morning glory.

This family contains about 55 genera. The genus *Ipomoea* is thought to contain over 500 species with ploidy levels ranging from $2x$ to $6x$. Sweetpotato is the only *Ipomoea* species of economic importance as a food crop, and has both $4x$ and $6x$ forms ($2n=4x=60$ or $2n=6x=90$). Thousands of cultivars of sweetpotato are grown throughout the tropics and subtropics. With an annual production of more than 133 million tons globally, sweetpotato currently ranks as the seventh most important food crop on a fresh-weight basis in the world, and fifth in developing countries after rice, wheat, maize, and cassava. The production is concentrated in East Asia, the Caribbean, and tropical Africa, with the bulk of the crop (88%) being grown in China (Figure 1). Sweetpotato performs well in relatively poor soils, with few inputs, and has a short growing period. Among the major starch staple crops, it has the largest rates of production per unit area per unit time: in some areas up to three harvests per year can be achieved. Sweetpotato roots are rich in vitamin C and essential mineral salts. Due to the high beta-carotene content of yellow and orange-fleshed storage roots, they are being promoted to alleviate vitamin A deficiency in East Africa and Eastern India.

Viruses of Sweetpotato

Until recently, viruses of sweetpotato have been relatively poorly studied as compared to viruses of other crops. Still,

more than 20 different viruses have been described infecting sweetpotato worldwide, but only 15 of these are currently recognized by the International Committee on Taxonomy of Viruses (ICTV; Table 1). This number, however, will most likely increase by additional surveys (Figure 2) and by indexing germplasm collection (Figure 3).

Vegetative propagation, usually by taking cuttings from a previous crop, increases the risk of a buildup of viruses. The importance of virus diseases and their buildup in farmers' planting materials has been shown convincingly in China, where sweetpotato cultivars planted using pathogen-tested materials yielded 30–40% more, on average, than those grown from farm-derived planting materials. Next to weevils, virus diseases form the most important biotic production constraint in sweetpotato. Most sweetpotato-infecting viruses, however, show only mild or no symptoms when in single infection and the damages caused by sweetpotato viruses are mostly through synergistic mixed infections. Viruses of the families *Potyviridae* and *Geminiviridae* as well as sweetpotato chlorotic stunt virus (SPCSV) are particularly significant in relation to sweetpotato cultivar decline.

Due to low virus titers and absence of symptoms from single infections in sweetpotato by most viruses, grafting and in some cases sap transmission onto indicator plants is often required to increase virus concentration and detect viruses reliably (Figure 3). Commonly used indicator plants are *Ipomoea setosa*, *I. nil*, *I. purpurea*, *I. aquatica*, and in some cases *Nicotiana benthamiana* and *N. clevelandii*.

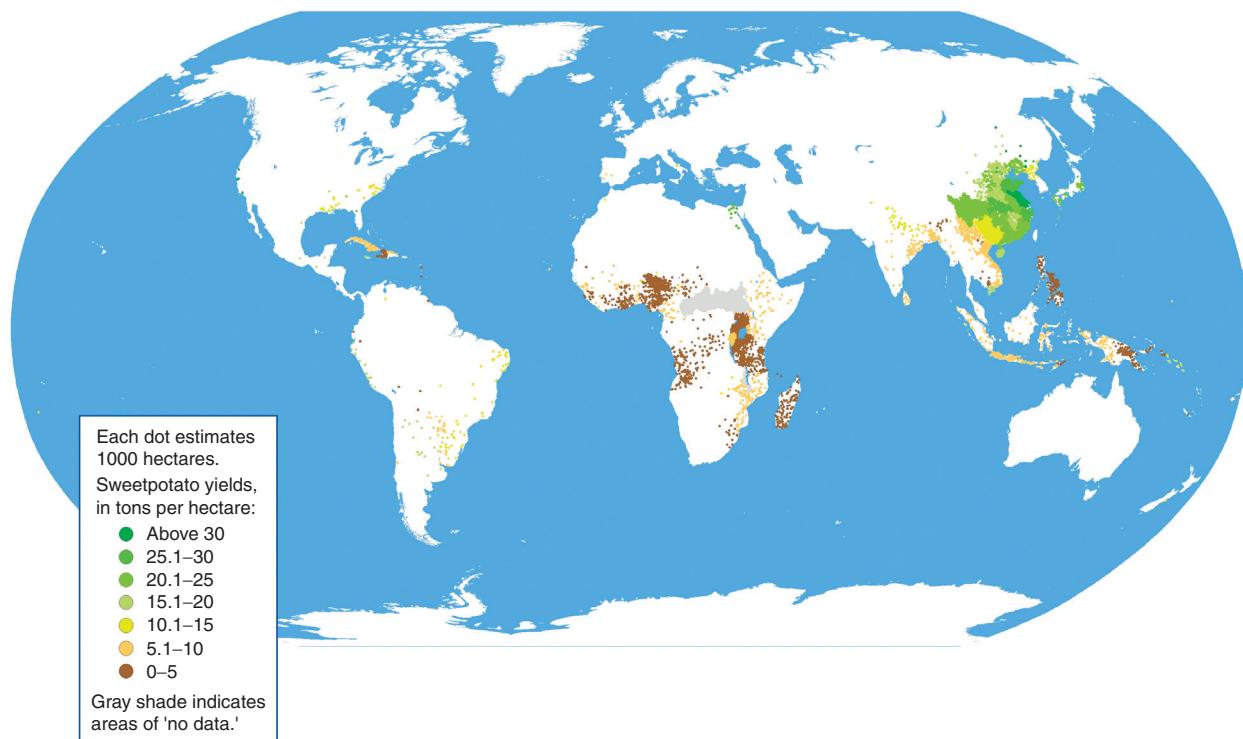


Figure 1 Sweetpotato cultivation: production areas and average yields.

Table 1 List of viruses that have been reported infecting sweetpotato

Genus/family	Species/virus	Reported distribution ^a	Vector	Detection methods ^b	Strains/serotypes	Component of synergistic disease ^c	Sequence from GenBank ^d
Begomovirus	<i>Ipomoea yellow vein virus</i> (IYVV)	Spain, Italy	Whiteflies?	PCR	None reported	AJ132548	
Begomovirus	<i>Sweetpotato leaf curl Georgia virus</i> (SPLCV)	USA, Puerto Rico	Whiteflies	NA hybridization, PCR, qPCR	None reported	AF326775	
Begomovirus	<i>Sweetpotato leaf curl virus</i> (SPLCV)	Far East, USA, China, Taiwan, Japan, Korea, Europe, Africa?, Peru	Whiteflies	NA hybridization, PCR, qPCR	None reported	AF104036	
Carlavirus	<i>Sweetpotato chlorotic fleck virus</i> (SPCFV)	Africa, South America, Asia, Cuba, Panama, China, Taiwan, Japan, Korea, New Zealand	Unknown	Serology	East Africa type, Asia type	Carrot kulot, unnamed (with SPCSV)	
Carlavirus?	C-6 virus	USA, Peru, Cuba, Dom, Rep, Indonesia, Philippines, P. Rico, Egypt, Uganda?, Kenya, South Africa, New Zealand	Unknown	Serology	None reported	Camote kulot, unnamed (with SPCSV)	
Caulimovirus?	<i>Sweetpotato caulimo-like virus</i> (SPCalV)	South Pacific Region, Madeira, China, Egypt, Puerto Rico, Uganda, Kenya?, Nigeria	Unknown	Serology, PCR	None reported	None available	
Crinivirus	<i>Sweetpotato chlorotic stunt virus</i> (SPCSV)	Worldwide	Whiteflies	Serology, NA hybridization, RT-PCR, qRT-PCR	EA, WA	SPVD, SPCD, Camote Kulot, SPSMD	
Cucumovirus	<i>Cucumber mosaic virus</i> (CMV)	Israel, Egypt, Kenya, Uganda?, Japan, South Africa, New Zealand	Aphids	Serology	None reported	Unnamed (with SPCSV)	
Geminiviridae	<i>Ipomoea crinkle leaf curl virus</i> (ICLCV)	Israel	Whiteflies	NA hybridization	None reported	RNA1: D00356, RNA2: D00355, RNA3: D10538	
Iaviridae	<i>Tobacco streak virus</i> (TSV)	Guatemala	Thrips	Serology	None reported	None available	
Ipomovirus	<i>Sweetpotato mild mottle virus</i> (SPMMV)	Africa, Indonesia, China, Philippines, Papua New Guinea, India, Egypt, New Zealand	Whiteflies?	Serology, RT-PCR	Different strains	RNA1: U80934, RNA2: U75538, RNA3: X00435	
Ipomovirus	<i>Sweetpotato yellow dwarf virus</i> (SPYDV)	Taiwan, Far East	Whiteflies	Serology	None reported	Z73124	
Luteoviridae	<i>Sweetpotato leaf speckling virus</i> (SPLSV)	Peru, Cuba	Aphids	NA hybridization, RT-PCR	None reported	DQ655700	

Continued

Table 1 Continued

Genus/family	Species/virus	Reported distribution ^a	Vector	Detection methods ^b	Strains/serotypes	Component of synergistic disease ^c	Sequence from GenBank ^d
<i>Nepovirus</i>	Sweetpotato ringspot virus (SPRSV)	Papua New Guinea, Kenya?	Unknown	Serology	None reported		None available
	Sweetpotato featherly mottle virus (SPFMV)	Worldwide	Aphids	Serology, NA hybridization, RT-PCR, qRT-PCR	EA, RC, O, C	SPVD, SPCD, Camote Kulot	D86371
<i>Potyvirus</i>	Sweetpotato latent virus (SPLV)	Africa, Taiwan, China, Japan, India, Philippines, Indonesia, Egypt	Aphids	Serology, NA hybridization	None reported	Camote Kulot	X84011*, X84012*
	Sweetpotato mild speckling virus (SPMSV)	Argentina, Peru, Indonesia, Philippines, China, Egypt, Uganda?, Kenya?, South Africa, Nigeria, New Zealand	Aphids	Serology	None reported	SPCD, Camote Kulot	U61228*
<i>Potyvirus</i>	Sweetpotato vein mosaic virus (SPVMV)	Argentina	Aphids	No available	None reported		None available
	Sweetpotato virus G (SPV(G))	China, Japan, USA, Egypt, Ethiopia, Nigeria, Barbados, Peru	Aphids	Serology, RT-PCR, qRT-PCR	None reported		Z83314*, AJ515380*
<i>Potyvirus</i>	Sweetpotato virus 2	Taiwan, USA, China, South Africa, Portugal, Australia, Barbados	Aphids	Serology, RT-PCR, qRT-PCR	Geographical/genetical lineages	Unnamed (with SPCSV)	AY178992*, AY232437*
	Tobamovirus	Tobacco mosaic virus (TMV)	USA	None	Serology	None reported	X02144, AJ132845

^a? signifies unconfirmed.^bNA, nucleic acid; RT-PCR, reverse transcription PCR; qPCR, quantitative real-time PCR; qRT-PCR, quantitative real-time reverse transcription PCR.^cSPVD, sweetpotato virus disease (Africa, Peru); SPCD, sweetpotato chlorotic dwarf (Argentina); SPSMD, sweetpotato severe mosaic disease (Africa); Camote kulot (Philippines).^dComplete genome sequence except those marked with (*).

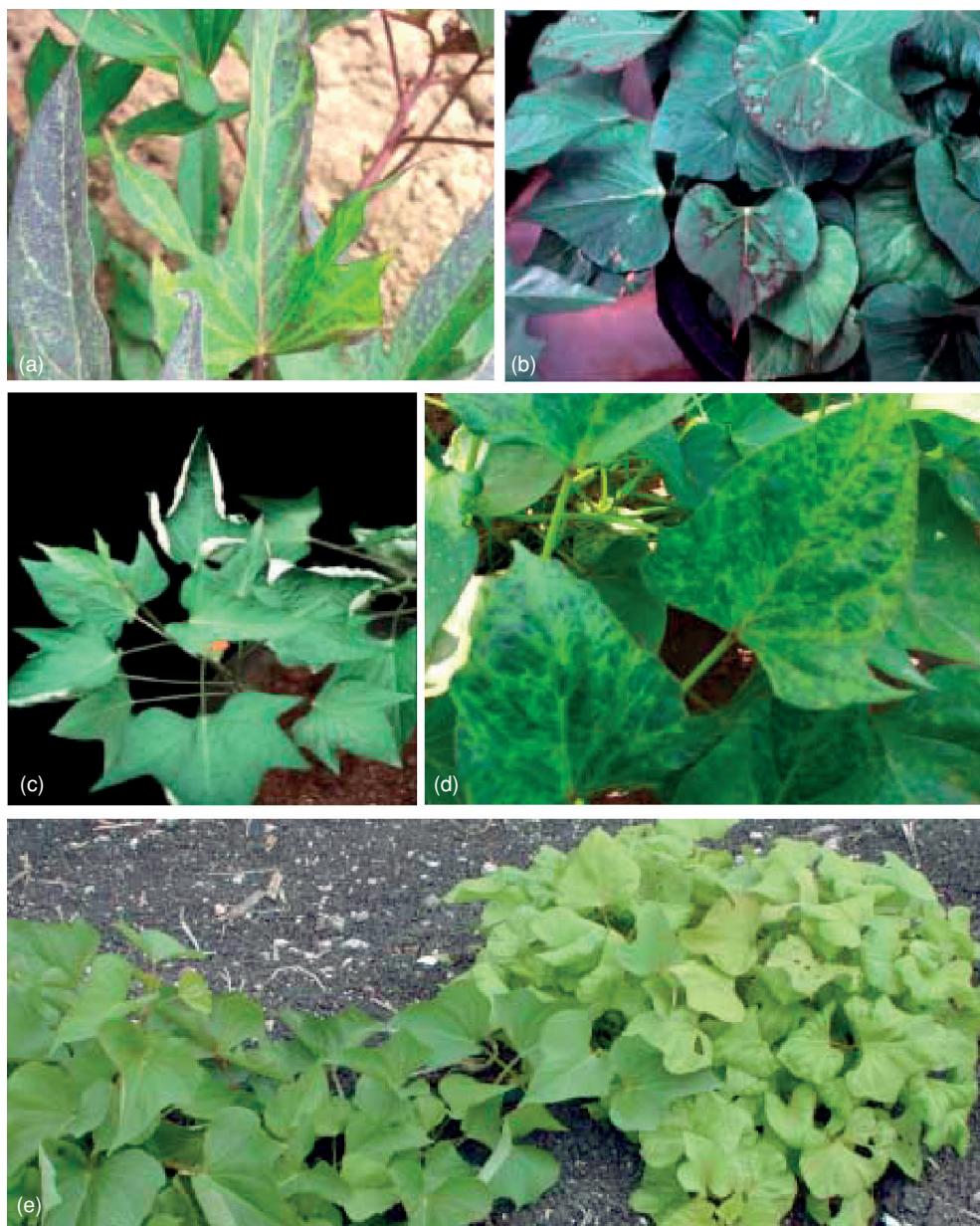


Figure 2 Sweetpotato plants showing symptoms of vein clearing (a and b), leaf curling (c), mosaic (d), and chlorosis, stunting, and leaf deformation. Some times the vein clearing is surrounded by purple pigmentation.

Potyviruses

Several potyviruses infect sweetpotato and usually only cause transient, mild, or no symptoms when infecting sweetpotato by themselves. The most widespread of these, and the one studied in most detail is sweetpotato feathery mottle virus (SPFMV, genus *Potyvirus*, family *Potyviridae*) that occurs wherever sweetpotato is grown. In many cases infection of sweetpotato plants with SPFMV causes mild or no symptoms, although certain strains can cause qualitative damage due to internal cork

or cracking of the tuberous roots. However, quantitative losses due to reduced plant vigor associated with chronic infection with SPFMV have also been experienced. Yet, it is as a component of complex virus diseases that SPFMV probably causes the greatest damage.

Sweetpotato Feathery Mottle Virus

SPFMV has flexuous filamentous particles measuring 830–850 nm in length. They contain a single-stranded, positive-sense RNA genome of about 10.6 kb, which is

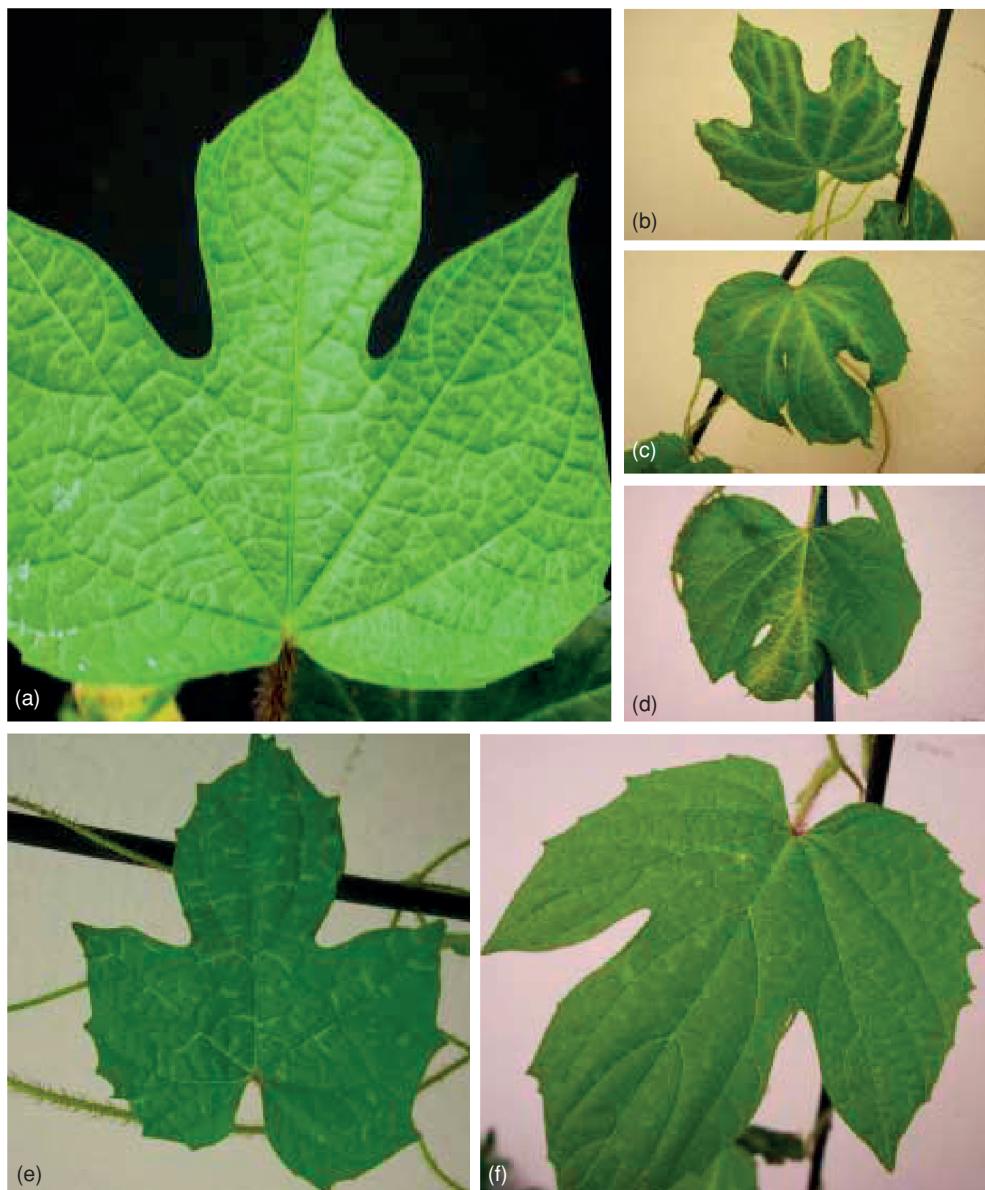


Figure 3 *Ipomoea setosa* graft-inoculated with scions from sweetpotato plants collected in farmer's fields in Peru. Infected with sweetpotato feathery mottle virus (a-d) and sweetpotato virus G (e). Faint chlorotic spots (f) induced by an unknown virus. The causal virus tested negatively to available antisera in NCM-ELISA.

larger than the average size (9.7 kb) of a potyvirus genome. The coat protein (CP) of SPFMV is also exceptionally large (38 kDa) as compared to other potyviruses, which is largely due to the insertion of a contiguous sequence at the 5'-end of the CP cistron. SPF MV is transmitted by several aphid species (i.e., *Aphis gossypii*, *A. craccivora*, *Lipaphis erysimi*, *Myzus persicae*) in a non-persistent manner. However, these aphids rarely colonize sweetpotato under field conditions and therefore itinerant alate aphids are probably the most efficient vectors of SPF MV. The experimental host range of SPF MV is narrow and mostly limited to plants of the family Convolvulaceae,

and especially to the genus *Ipomoea*, although some strains have been reported to infect *N. benthamiana* and *Chenopodium* spp. Symptoms, host range, and serology have been used to group SPF MV isolates into two strains, the common strain (C) and the more severe russet crack (RC) strain. However, based on phylogenetic analysis of 3' nt sequences of an extensive number of isolates it is now clear that SPF MV can be distinguished in four phylogenetic lineages RC, O (ordinary), EA (East Africa), and C (**Figure 4**). Based on molecular data the C strain is rather distantly related to the remaining strains and may in the future be classified as a separate virus.

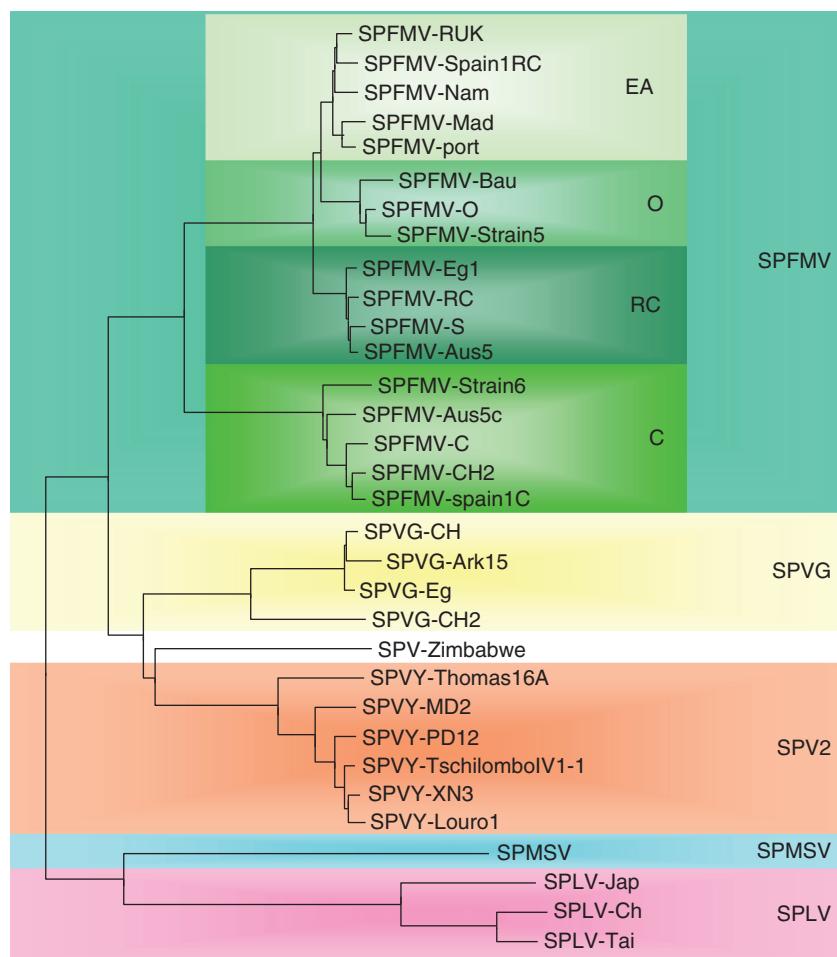


Figure 4 Phylogeny of representative isolates of sweetpotato-infecting potyviruses based on complete CP sequences. Clades corresponding to described virus species are shaded in different colors. The four recognized strain groups of SPFMV are also indicated and shaded in different colors of green. One sequence, originally described as SPFMV from sweetpotato in Zimbabwe, is distinct from all other named species.

Other Potyviruses

Several other potyviruses are known to infect sweetpotato, some of which are distributed widely. They have in common that they are mostly symptomless and in low titers when infecting sweetpotato by themselves, but can be distinguished by their symptoms induced in indicator plants such as *I. nil* and *I. setosa* (Figure 3), or by symptoms induced through synergism with SPCSV. With the exception of sweetpotato vein mosaic virus (SPVMV) sequence information is available for these viruses and their phylogenetic relationships are depicted in Figure 4.

Sweetpotato virus G (SPVG) was first reported from China, but is now known to occur also in the Americas and Africa. Another virus, originally identified as sweetpotato virus 2 in Taiwan, was recently further characterized simultaneously by two groups suggesting the species names sweetpotato virus Y and ipomoea vein mosaic virus, respectively, and has been reported from Taiwan,

China, USA, South Africa, Australia, and Barbados. The name SPV2 is currently being considered for this virus by the ICTV. Sweetpotato latent virus (SPLV) was also first reported from China, but is now known to occur in most major sweetpotato-growing areas in Asia. The two remaining potyviruses reported were first described from Argentina and are SPVMV and sweetpotato mild speckling virus (SPMSV). Whereas SPMSV has also been detected in Peru and Indonesia, neither antibodies nor sequence information are available for SPVMV, and the virus has not been reported from elsewhere. The number of sweetpotato-infecting potyvirus species will certainly still increase through future surveys, for example, a potyvirus distinct from any of the above mentioned was isolated from infected sweetpotato in Zimbabwe and remains to be characterized in more detail (Figure 4).

Sweetpotato mild mottle virus (SPMMV, genus *Ipomovirus*) is transmitted by whiteflies in a nonpersistent

manner. It should be noted, however, that since the initial report, it has not been possible to confirm independently the whitefly transmissibility of SPMMV. A study of the variability of SPMMV in Uganda using 3' nt sequences showed the virus consisted of a population of distinct sequence variants (>85.9% nt identity in the CP), which however did not show any particular clustering. A distinct feature of SPMMV as compared to other sweetpotato-infecting viruses is its exceptionally broad host range including species in 14 families. SPMMV has serologically been detected throughout Africa, Indonesia, China, Philippines, Papua New Guinea, India, Egypt, and New Zealand. Another whitefly-transmitted potyvirus with properties distinct from SPMMV was described in Taiwan and named sweetpotato yellow dwarf virus (SPYDV). The relationship between these two viruses is however unclear as no sequence information is available for SPYDV.

Begomoviruses

Sweetpotato leaf curl diseases typical of geminivirus infection (**Figure 2(c)**) have been reported from Peru, Japan, Taiwan, Korea, China, Puerto Rico, Costa Rica, Spain, United States, Africa, and other countries. The host range of the *Ipomoea*-infecting begomoviruses (family *Geminiviridae*) is narrow and mostly restricted to species in the family Convolvulaceae (especially to the genus *Ipomoea*), but *N. benthamiana* (Solanaceae) can also be infected. They cause leaf curl on some hosts, and yellow vein or leaf distortion and chlorosis symptoms on others. Some sweetpotato cultivars are symptomless. As they induce mild, transient symptoms in the standard virus indicator, *I. setosa*, a universally reliable biological indicator is lacking. Some *Ipomoea* species (*I. aquatica*, *I. cordatotriloba*, *I. purpurea*) can however be used as differential hosts to distinguish *Ipomoea*-infecting begomoviruses.

Only three sweetpotato-infecting begomovirus species have been officially recognized by the ICTV (**Table 1**): *Sweetpotato leaf curl virus* (SPLCV), *Sweetpotato leaf curl Georgia virus* (SPLCGV), and *Ipomoea yellow vein virus* (IYVV). DNA B components have not been identified for any of these viruses indicating they are monopartite begomoviruses. Sequences of AC1 gene fragments from isolates obtained from infected *Ipomoea* spp. from USA, China, Taiwan, Korea, Puerto Rico, Spain, and Italy all fall into two groups corresponding to SPLCV/IYVV and SPLCGV. A third phylogenetically distinct group of begomoviruses was identified from infected sweetpotato in Spain, representing a putative fourth species. Similarly the sequence of a SPLCV isolate from China shows <83% identity to other published sequences indicating yet another virus species. Another virus, proposed to

be named ipomoea crinkle leaf curl virus (ICLCV) and reported from Israel, differed in host range from SPLCV but its exact relationship to other identified viruses remains unclear.

From a taxonomic point of view, the *Ipomoea*-infecting begomoviruses are a curiosity; phylogenetic analysis of IYVV and several strains of SPLCV and SPLCGV revealed that these viruses form a separate unique cluster within the genus *Begomovirus*, dissimilar to both the New World and Old World begomoviruses. Additionally, the relatively poor transmission rates of the *Ipomoea*-infecting begomovirus by *Bemisia tabaci* may be a reflection of the low CP amino acid sequence identity (46%) between them and the other begomoviruses.

Sweetpotato Leaf Curl Virus

The virus has geminate particles of c. 18 × 30 nm with a genome of 2828 nt. Its genomic DNA and organization is similar to that of monopartite begomoviruses. SPLCV virus was first reported from Japan and Taiwan but now it is known to occur in several countries on different continents. SPLCV can cause up to 30% reductions in yield of storage roots. Various *Ipomoea* species are susceptible to SPLCV, such as *I. purpurea* causing leaf curl and stunt, *I. aquatica* causing yellow vein symptoms, *I. nil*, *I. setosa*, and *N. benthamiana* causing leaf curl symptoms. Co-infections of SPF MV and SPLCV in *I. setosa* and *I. nil* induce severe leaf distortion, general chlorosis, and stunting.

Ipomoea Yellow Vein Virus

IYVV was first found in Spain infecting *I. indica* plants showing yellow vein symptoms, but has since then been found infecting cultivated sweetpotato as well. Its properties, including typical geminivirus symptoms, detection of geminate particles by electron microscopy, and complete nucleotide sequence confirmed its begomovirus nature. Based on nucleotide sequence similarity of >89% over the entire genome, IYVV should be considered a strain of SPLCV, and will probably be revised accordingly in the future. However, contrary to SPLCV which is transmitted by *B. tabaci* biotype B, IYVV-[Spain] was not transmitted by biotypes Q, S, or B of *B. tabaci*.

Sweetpotato Leaf Curl Georgia Virus

SPLCGV, previously called ipomoea leaf curl virus, has a genome of 2773 nt with an organization typical of other Old World monopartite begomoviruses. The complete nucleotide sequence of SPLCGV is 82–85% identical to those of SPLCV and IYVV, which is below the species threshold of 89% nucleotide sequence identity for begomoviruses. Like SPLCV, SPLCGV is transmitted by *B. tabaci* biotype

B and induces similar symptoms in most *Ipomoea* species (various degrees of leaf curling). The means to distinguish SPLCGV from SPLCV are the use of *I. aquatica* and *I. cordatotiloba* as differential hosts and a combination of polymerase chain reaction (PCR) and restriction enzyme digestion.

Sweetpotato Chlorotic Stunt Virus

Due to its ability to mediate severe synergistic viral diseases with several other sweetpotato-infecting viruses including potyviruses, cucumoviruses, and carlavirus, SPCSV is probably the most devastating virus of sweetpotato worldwide. In single infection, this virus causes no symptoms at all or usually only mild stunting combined with slight yellowing or purpling of older leaves, symptoms which are easily confused with nutritional deficiencies.

SPCSV belongs to the genus *Crinivirus* of the family *Closteroviridae*. The particles of SPCSV are 850–950 nm in length and 12 nm in diameter. The size of the major coat protein is 33 kDa, which is similar to other criniviruses. SPCSV is transmitted by whiteflies (e.g., *B. tabaci*, *B. afer*, and *Trialeurodes abutilonea*) in a semipersistent, noncirculative manner, and is not mechanically transmissible. Similar to most other sweetpotato-infecting viruses, the host range of SPCSV is limited mainly to the family Convolvulaceae and the genus *Ipomoea*, although *Nicotiana* spp. and *Amaranthus palmeri* are reportedly susceptible. SPCSV has also been detected in the ornamental species *Lisianthus* (*Eustoma grandiflorum*). SPCSV can be serologically divided into two major serotypes. One of the serotypes (EA for East Africa) was first identified in East Africa, and also occurs in Peru, while the other serotype (WA for West Africa) was first identified in West Africa and occurs additionally in the Americas, and the Mediterranean, but not in East Africa. The two serotypes correlate to two genetically distantly related strain groups based on coat protein as well as Hsp70h gene similarities.

The genome of an SPCSV isolate from Uganda has been entirely sequenced and consists of two RNA segments of 9407 and 8223 nt. The SPCSV genome encodes two unique proteins, p22 and RNase3, not known to occur in any other RNA viruses. mRNAs corresponding to these proteins are transcribed early during infection and they are cooperatively able to suppress RNA silencing, a process that requires the RNA binding and double-stranded RNase activity of the RNase3 protein.

Other Viruses

Some viruses recognized by ICTV that affect other crops (i.e., cucumber mosaic virus, tobacco mosaic virus, and

tobacco streak virus), are sporadically found infecting sweetpotato. Information on these viruses is extensively available. Other sweetpotato-specific viruses have not yet being assigned to a genus (i.e., sweetpotato leaf speckling virus – SPLSV) or recognized as a species (i.e., sweetpotato chlorotic fleck virus – SPCFV; C-6 virus; sweetpotato caulimo-like virus – SPCaLV) by the ICTV (Table 1).

Sweetpotato Leaf Speckling Virus

SPLSV has isometric particles c. 30 nm in diameter. The virus is transmitted by *Macrosiphon euphorbiae* in a persistent manner. It has a restricted geographical distribution. The sequence of the CP and 17K encoding region has been determined and is characteristic of luteo- and poleroviruses sequences, with highest similarity to potato leafroll virus (PLRV). Although these two viruses can be detected with heterologous CP probes, they are not serologically related.

Sweetpotato Chlorotic Fleck Virus

SPCFV has filamentous particles measuring about 800 nm in length consisting of its genome encapsidated by polypeptide subunits of 33.5 kDa. The virus appears to have a wide geographic distribution in sweetpotato crops in South America, Africa, and Asia. SPCFV has a narrow host range in the families Convolvulaceae and Chenopodiaceae, but some strains/isolates infect *N. occidentalis*. SPCFV is mostly symptomless in its natural host; hence, it was also referred to as sweetpotato symptomless virus in Japan.

Sequence analysis of the entire genome of SPCFV (9104 nt) provides unambiguous evidence for the assignment of SPCFV as a distinct species in the genus *Carlavirus*. The RNA of SPCFV is larger than that of the other carlavirus due to its considerably larger replicase (238 vs. 200–223 kDa) and a long untranslated region of 236 nt between ORF4 and ORF5. Phylogenetic analysis of CP sequences suggests that there is some geographically associated variation among SPCFV isolates.

Complex Virus Diseases of Sweetpotato

Multiple virus infections are common in sweetpotato and synergistic interactions are often involved. The most common of these disease complexes, known as sweetpotato virus disease (SPVD), is caused by simultaneous infection with SPFMV and SPCSV (Figures 5 and 6). This disease is characterized by chlorosis, small, deformed leaves, and severe stunting, and can reduce yields of infected plants by up to 99%. Despite the apparent broad meaning of the name SPVD, the symptoms are so characteristic that the name has become restricted to the disease



Figure 5 Sweetpotato plants affected by viruses in Peru. Sweetpotato field with a large number of plants affected by sweetpotato virus disease complex (a) and a close up of an affected plant showing stunting, mosaic, and leaf deformation (b).

with these symptoms and caused by these viruses. SPVD is the most serious disease of sweetpotato in Africa and Peru, and may be the most important virus disease of sweetpotato globally.

Other viral disease complexes have also been described, which invariably seem to involve SPCSV. In Israel and Egypt cucumber mosaic virus (CMV, genus *Cucumovirus*, family *Bromoviridae*) is found infecting sweetpotato together with SPCSV and usually also SPFMV, producing symptoms similar to SPVD and causing up to 80% reduction in yield. It was shown that CMV could only infect sweetpotato if the plants were first infected with SPCSV. Interestingly, this seems not to be the case for CMV in Egypt, where it is found infecting sweetpotato with or without SPCSV. In Argentina, a disease locally known as chlorotic dwarf is caused by infection with SPCSV and SPFMV and/or SPMSV, and is the most important disease of sweetpotato in the country. Once again, the symptoms resemble those of SPVD and are most severe when all three viruses infect sweetpotato simultaneously. In the Philippines

SPCSV together with several other viruses causes a disease locally known as Camote Kulot.

In all the mentioned disease complexes, infection with each virus separately causes only mild or no symptoms in sweetpotato. They are thus caused by a synergistic interaction between the viruses. As both SPFMV and SPCSV are involved in all these diseases, the variation in the strains of these viruses should be important factors determining the disease severity.

Experimentally SPCSV can induce synergism with all tested potyviruses (including SPMMV), CMV as well as carlaviruses, and is always associated with an increase in the titers of the co-infecting virus and reduced yield of storage roots. For instance, the dual infection of SPCSV and SPMMV has been named ‘sweetpotato severe mosaic disease (SPSMD)’. Yet there are reports indicating that strains of SPFMV and SPV2 may differ in their ability to cause synergism with SPCSV. Triple infections involving SPCSV, SPFMV, and an additional virus are even more severe, leading to further increase of the SPFMV

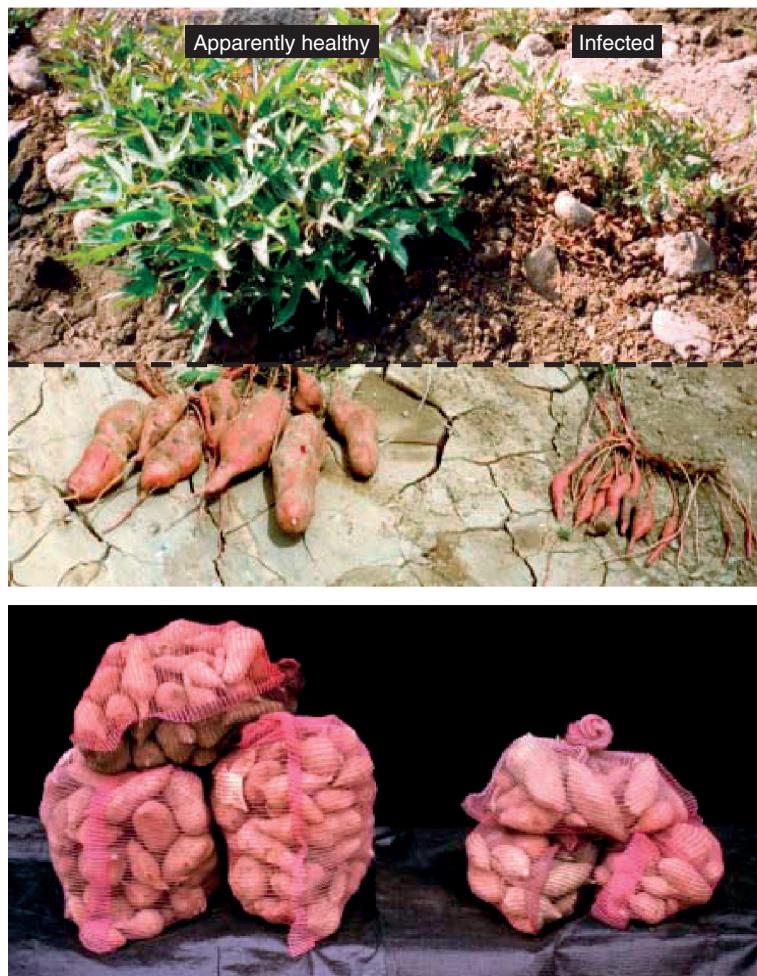


Figure 6 Effect of viruses on sweetpotato plants (top) and yield of storage roots (bottom). Observe yield reduction on sweetpotato variety Costanero (30 plants each treatment).

titors, whereas the titers of the third virus may either increase or decrease. It is thought the synergistic effects of SPCSV on other viruses may be due to interference with RNA silencing, because they are associated with substantially increased accumulation of co-infecting viruses.

See also: *Carlavirus*; Citrus Tristeza Virus; *Iilarvirus*; Luteoviruses; Plant Antiviral Defense: Gene Silencing Pathway; Plant Resistance to Viruses: Geminiviruses; Potyviruses; Viral Suppressors of Gene Silencing.

Further Reading

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