

## Chapter 8

# Virus and Phytoplasma Diseases

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Sweetpotatoes are vegetative propagated from vines, root slips (sprouts) or tubers, and farmers often take vines for propagation from their own fields year after year. Thus, if virus diseases are present in the field they will inevitably be transmitted with the propagation material to the newly planted field, resulting often in a marked decrease in yields. Yields differ greatly in different areas or even fields in the same location. Thus, the average yield in African countries is about 7.02 tons/ha, with yields of 9.4, 4.4, 2.5 and 3.2 ton/ha in Kenya, Uganda, Sierra Leone and Nigeria, respectively. The yields in Asia are significantly higher, averaging 12.41 tons/ha. China, Japan, Korea and Israel have the highest yields with about 21.6, 25.8, 16.4 and 44.4 tons/ha, respectively. In South America the average yield is 10.74 tons/ha, with Argentina, Peru and Uruguay in the lead with 17.2, 16.35 and 13.68 tons/ha, respectively. For comparison, the average yield in the USA is 20.1 tons/ha (all data are averages for 2005 from the FAOSTAT 2007).

These differences in yields are mainly due to variation in quality of the propagation material, often taken from the previous season of farmer's fields. Often these fields are infected with several viruses, thereby compounding the effect on yields. In China, on average, losses of over 20% due to sweetpotato virus diseases were observed (Gao et al. 2000), mainly due to *Sweetpotato feathery mottle virus* (SPFMV) and *Sweetpotato latent virus* (SPLV). The infection rate in the Shandong province reaches 5–41% (Shang et al. 1999). In countries where care is taken to provide virus-tested planting material as, amongst others in the USA and Israel, yields increase markedly, up to 7 times and more. In some countries, as in Uganda, Kenya and Tanzania virus diseases are a major constraint for sweetpotato production.

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## The Main Viruses

*Sweetpotato feathery mottle virus* Genus *Potyvirus* (SPFMV) is the most common sweetpotato virus worldwide. Certain isolates in the USA and Japan cause much economic damage by inducing cracking or internal corkiness in some cultivars (Fig. 8.1). In Africa, SPFMV causes a severe sweetpotato virus disease (SPVD) in a complex infection with the whitefly-transmitted *Sweetpotato chlorotic stunt virus* Genus *Crinivirus* (SPCSV) (Syn. *Sweetpotato sunken vein virus*, SPSVV).

Most sweetpotato cultivars infected by SPFMV alone show no or only mild circular spots on their leaves (Fig. 8.2) or light green patterns along veins. However, when infected together with the whitefly-transmitted SPCSV stunting of the plants, feathery vein clearing and yellowing of the plants are observed (Fig. 8.3). In controlled experiments, SPFMV-infection alone did not reduce yields compared to virus-free controls, while the complex infection with SPCSV reduced yields by 50% or more (Gutiérrez et al. 2003; Milgram et al. 1996). In Brazil, yield increases of 118% were observed when virus-tested cuttings derived from meristems of heat-treated plants were compared to yields from cuttings taken from fields (Pozzer et al. 1995). This is a unique report where SPFMV alone markedly decreased yields, and it might be that another virus could have been present.

SPFMV is transmitted in a nonpersistent manner by aphids, including *Aphis gossypii*, *Myzus persicae*, *A. craccivora* and *Lipaphis erysimi*. Aphid transmissibility is dependent on virus encoded helper proteinase and a DAG triplet in the protein coat. The virus can be transmitted mechanically to various *Ipomoea* spp. as *I. batatas*, *I. setosa*, *I. nil*, *I. incarnata* and *I. purpurea*, and some strains to *Nicotiana benthamiana*, *N. clevelandii*, *Chenopodium amaranticolor* and *C. quinoa* (Brunt

**Fig. 8.1** Internal cork disease caused by sweetpotato feathery mottle virus (Courtesy J.W. Moyer)



**Fig. 8.2** SPFMV circular spots



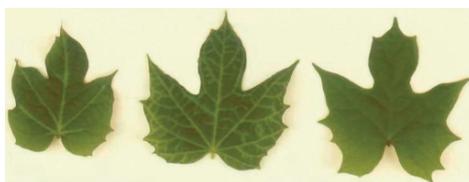
**Fig. 8.3** Sweetpotato infected by both SPFMV and SPSVV causing stunting of the plants, feathery vein clearing and yellowing of the plants



et al. 1996). The virus is transmitted by grafting but not by seed or pollen or by contact between plants. In Uganda SPFMV was found in 22 *Ipomoea* spp. *Hewittia sublobata* and *Lepistemon ovariensis* (Tugume et al. 2008).

The virus can best be diagnosed by grafting on *I. setosa*, causing vein clearing (Fig. 8.4) followed by remission, or on *I. incarnata* and *I. nil* inducing systemic vein clearing, vein banding and ringspots. SPFMV can be diagnosed by ELISA, and antisera are commercially available.

However, ELISA reliably detects SPFMV only in leaves with symptoms and when coinfecting with SPCSV (Gutiérrez et al. 2003). It is best to sample several leaves from a plant, as the virus seems to be unevenly distributed, especially for meristem-derived plantlets. This has to be followed up by indexing on a susceptible indicator (Green et al. 1988). SPFMV can also be detected by membrane immunobinding (MIBA, also termed NCM-ELISA) and by using a riboprobe (Abad and Moyer, 1992). The latter although being very sensitive, detecting 0.128 pg of RNA, compared with 179 pg of capsid protein, was still somewhat less sensitive than grafting on a sweetpotato indicator (cv. Jewel). The use of a riboprobe also requires a well-equipped laboratory and radioactive materials or digoxigenin for labeling the probe. SPFMV has also been identified by reverse transcription and polymerase chain reaction (RT-PCR), utilizing degenerate genus-specific primers,



**Fig. 8.4** *I. Setosa* infected with Sweetpotato feathery mottle virus (SPFMV), showing vein clearing

designed to amplify the variable 5' terminal region of the potyvirus coat protein gene (Colinet et al. 1998).

SPFMV can be purified from infected *I. nil* leaf material (Cali and Moyer, 1981), by rather complex purification procedures, and directly from sweetpotatoes (infected also with SPCSV), by a relatively simple method (Cohen et al. 1988). This method yielded 50–100 mg virus/kg tissue, 5–10 times more when compared with the previous methods. This high yield of virus is due to the synergistic interaction between SPFMV and SPCSV, where SPFMV titer attains more than 600-fold increase (Karyeija et al. 2000). It should be mentioned that virus yields from plants infected by SPFMV alone, without SPCSV, are markedly lower.

Virions are filamentous, not enveloped, usually flexuous, with a modal length of 830–850 nm. The genome consists of single stranded linear RNA, unipartite of 11.6 Kbp, with a poly(A) region (Moyer and Cali, 1985). The complete nucleotide sequence of a sweetpotato feathery mottle potyvirus severe strain (SPFMV-S) genomic RNA was determined (Sakai et al. 1997). The viral RNA genome (S strain) is 10,820 nucleotides (nts) long (GenBank accession D86371) excluding the poly(A) tail, and contained one open reading frame, potentially encoding 3,493 amino acids. Except in the regions of P1 and P3, the polyprotein has a high level of amino acid identity with those of other potyviruses (Sakai et al. 1997). The helper component-proteinase of SPFMV facilitates systemic infection spread of *Potato virus X* in *I. nil* (Sonoda et al. 2000).

Many strains of SPFMV (Moyer, 1986), isolates, variants and serotypes of SPFMV have been reported, as the russet crack (RC), C, S, C1 (from Peru), strain 835 (from Guatemala) and others (Colinet and Kummert, 1993; Karyeija et al. 2001). By comparing coat protein gene sequences of isolates it was shown that isolates from East Africa (EA) form a separate cluster (Kreuze et al. 2000). SPFMV can be divided into four phylogenetic groups (strains) according to the analysis of coat protein (CP) encoding sequences. Thus, strain EA contained the East African, Spanish, and Peruvian isolates of SPFMV. In contrast, strain RC contained isolates from Australia, Africa, Asia and North and South America. Strain O contained isolates from Africa, Asia and South America. The strain C isolates from Australia, Africa, Asia, and North and South America formed a group that was genetically distant from the other SPFMV strains (Tairo et al. 2005, Untiveros et al. 2007; Valverde et al. 2004b). It was suggested that SPFMV-C might be a distinct virus (Lyerly et al. 2003; Tairo et al. 2005). Evidences on recombination events between viruses from different strain groups of SPFMV have been recently reported (Untiveros et al. 2007).

Though SPFMV alone generally causes only minor damage, its control is imperative as in combination with other viruses its effect on plant growth and yields may become substantial. The proven approach is to prepare virus-tested plants from meristems. The plants are then grown under insect-proof conditions, and propagation is continued by cuttings. The farmer plants a certain number of vines in the open field and continues to propagate until the stand of the field is complete. The following year he will again start from virus-tested plants. This scheme has been in operation for more than 15 years in Israel, and presently it is difficult to find SPFMV in the country.

Breeding of SPFMV resistant plants was initiated by CIP (Mihovilovich et al. 2000). Several clones that were resistant to SPFMV in CIP's tests were found to be susceptible, when exposed to Israeli (unpublished) and Ugandan isolates (Karyeija et al. 1998). Apparently, strain diversity requires that breeding and selection have to be done in various locations. On the other hand a substantial number of African sweetpotato landraces have resistance to this virus (Carey et al. 1997).

Another approach was the development of transgenic sweetpotatoes with coat-mediated (CP) resistance to SPFMV (Okada et al. 2001). Also, CP-mediated resistance introduced into several African varieties and cultivar CPT-560 was evaluated in Kenya in a cooperative project between Monsanto Co., USA and Kenya Agriculture Research Institute (KARI) (Odame et al. 2001) However, these transgenic lines were not resistant to the 'complex' infection with SPCSV, causing the SPVD (Wambugu, 2004).

An interesting idea for controlling SPFMV involves the introduction of a rice cysteine-inhibitor gene. This gene inhibits the proteolysis of the viral polyprotein, thereby interfering with viral replication. Improved resistance to SPFMV was observed in 18 of the 25 transgenic lines after challenge-inoculation with the russet crack strain of SPFMV (Cipriani et al. 2001). However, when stem cuttings were prepared from the tolerant transgenic plants and grafted with healthy *I. setosa* scions, virus symptoms appeared on the scions. Apparently, the transgenic sweetpotato plants still contained some virus.

*Sweetpotato chlorotic stunt virus* Genus *Crinivirus* (SPCSV). {Possible synonym: *Sweetpotato sunken vein virus* (SPSVV)}. Although SPCSV is the name recognized by the International Committee of Taxonomy of Viruses (ICTV), they might be considered as separate viruses for the following reasons: (i) SPCSV and SPSVV differ in their nucleotide and protein sequences by more than 20% (Cuellar et al. 2008); (ii) comparing nucleotide sequences of the Israeli SPSVV with the Ugandan isolate of SPCSV in the heat shock 70-like protein region, revealed a homology of only 0.69 (Cuellar et al. 2008); (iii) symptoms induced by SPSVV on *I. setosa* differ markedly from those by SPCSV; (iv) infection of sweetpotato by SPSVV alone produced on cv. Georgia Jet mild symptoms consisting of slight yellowing of veins, with some sunken secondary veins on the upper sides of the leaves and swollen veins on their lower sides (Fig. 8.5) (at 28–29 °C). Upward rolling of the 3–5 distal leaves was also observed. Similar symptoms were also obtained on graft-inoculated plants of cv. Papel and Camote Negro (Cohen et al. 1992). The disease could easily be detected in the field, where plants had erect branches, with some upward rolling leaves. In some cultivars, SPCSV isolates cause no symptoms or mild stunting combined with slight yellowing or purpling of old leaves (Gibson et al. 1998; Gutiérrez et al. 2003).

Effects on yields by SPSVV or SPCSV alone are minor or close to 30% reduction, but in complex infection with SPFMV or other viruses yield losses of 50% and more are observed (Gutiérrez et al. 2003; Milgram et al. 1996; Untiveros et al. 2007).

SPCSV and/or SPSVV are transmitted by the whitefly *Bemisia tabaci* biotype B, *Trialeurodes abutilonea*, and *B. afer* (Gamarra et al. 2008; Ng and Falk, 2006; Schaefer and Terry, 1976; Sheffield, 1957; Sim et al. 2000; Valverde et al. 2004b)

**Fig. 8.5** *Sweetpotato sunken vein virus* – swollen veins on the lower side of the leaf; left leaf –healthy control



in a semipersistent manner, requiring at least one hour for acquisition and infection feeding and reaching a maximum after 24 h for both of them. The virus is graft transmissible, but not by mechanical inoculation. The virus was transmitted by whiteflies to *I. setosa*, *N. clevelandii*, *N. benthamiana* and *Amaranthus palmeri* from *I. setosa* and by grafting to other Ipomoeas.

The virus is best being diagnosed on a pair of sweetpotato plants- one healthy, the other infected by SPSMV. On the healthy plants hardly any symptoms will become apparent, while (if carrying SPSMV) severe symptoms of SPVD will appear. The virus can also be diagnosed by immunosorbent electron microscopy (ISEM) and by ELISA.

SPSVV can be purified from infected *I. nil* or *N. clevelandii*, maintained at 28 °C (Cohen et al. 1992). Normal length of virus particles from purified preparations was 850 nm, the diameter ~12 nm, with an open helical structure typical for closteroviruses. In leaf dip preparations from *I. setosa* infected with an isolate of SPCSV from Nigeria a modal length of 950 nm was obtained (Winter et al. 1992). The coat protein of the Israeli and Nigerian isolates had a MW of ~ 34, 000 and ~ 29, 000, respectively (Cohen et al. 1992; Hoyer et al. 1996). Double-stranded RNA from the Israeli isolate consisted of one major band of  $M_r$  10.5 Kbp, and 2 minor bands of 9.0 and 5.0 Kbp. Northern blot analysis of the Nigerian isolate revealed the presence of a large dsRNA with an estimated size of ~9.0 Kbp and several smaller ones. Several Kenyan cDNA clones revealed an open reading frame (ORF) of 774 nts coding for the coat protein (Hoyer et al. 1996).

SPCSV can be serologically divided into two major serotypes, which correlate to two genetically distantly related strains/groups based on the coat protein (CP) and the heat shock protein 70 homologue (Hsp70h) genes similarities. The East Africa (EA) group was first identified in East Africa, and also occurs in Peru, while the West Africa (WA) was first identified in West Africa and occurs additionally in the Americas and the Mediterranean, but not in East Africa (Hoyer et al. 1996; Tairo et al. 2005). In East Africa two serotypes ( $S_{EA1}$  and  $S_{EA2}$ ) were distinguished using a panel of monoclonal antibodies (MAbs) to a Kenyan isolate of SPCSV.  $S_{EA1}$  serologically resembled the Kenyan isolate of SPCSV whereas  $S_{EA2}$  has not previously been reported.  $S_{EA1}$  was predominant in eastern Uganda whereas  $S_{EA2}$  was predominant in southern and western Uganda. The Hsp70h and CP genes were generated by RT-PCR. Sequence analyses revealed substitutions at two nucleotide positions in the Hsp70h gene, although neither affected deduced amino-acid sequences. Nucleotide

substitutions in the CP gene region, which led to 11 amino-acid substitutions, revealed two major groupings plus other minor variants (Alicai et al. 1999). SPCSV isolates from East Africa were phylogenetically distant to SPCSV isolates from elsewhere (Tairo et al. 2005).

The complete nucleotide sequences of genomic RNA1 (9,407 nts) and RNA2 (8,223 nts) were determined, revealing that SPCSV possesses the second largest identified positive-strand single-stranded RNA genome among plant viruses after *Citrus tristeza virus* (Kreuze et al. 2002). RNA1 contains two overlapping open reading frames (ORFs) that encode the replication module, consisting of the putative papain-like cysteine proteinase, methyltransferase, helicase, and polymerase domains. RNA2 contains the *Closteroviridae* hallmark gene array represented by an Hsp70h, a protein of 50 to 60 kDa depending on the virus, the major coat protein, and a divergent copy of the coat protein. The two genomic RNAs of SPCSV contained nearly identical 208-nt-long 3' terminal sequences, and the ORF for a putative small hydrophobic protein was found in SPCSV RNA1. Furthermore, unlike any other plant or animal virus, SPCSV carried an ORF for a putative RNase III-like protein (ORF2 on RNA1). Several subgenomic RNAs (sgRNAs) were detected in SPCSV-infected plants, indicating that the sgRNAs formed from RNA1 accumulated earlier in infection than those of RNA2. Recently it was shown that the 3-proximal part of RNA 1 and the partial sequence of the Hsp70h gene of the Israeli SPSVV differ markedly in sequence from most of the SPCSV isolates (Cuellar et al. 2008; see also accession numbers EU124491 and EU124487). Additionally, the phylogenetic analysis of the partial Hsp70h, CP, and p60 genes of the Ea and the WA strains indicate that they may belong to different species in the genus *Crinivirus* (Abad et al. 2007; Tairo et al. 2005).

The disease has been reported from East and Southern Africa, Nigeria, Niger, Indonesia, Israel, Egypt, Spain, Argentina, Brazil, Peru (Gutiérrez et al. 2003) and recently from the USA (Abad et al. 2007).

In a recent research a Peruvian sweetpotato landrace was transformed with an introduced hairpin construct targeting the replicase encoding sequences of SPFMV and SPCSV (Kreuze et al. 2008). A high level of resistance to SPCSV was observed in the transgenes, which however did not prevent development of SPVD when plant became infected also with SPFMV.

For control of SPCSV/SPSVV it is best to start from meristems and test the plants on a pair of sweetpotato plants as described above.

**Sweetpotato virus disease (SPVD)** is caused by the interaction of SPFMV and SPCSV/SPSVV. Characteristic symptoms of the disease include vein-clearing (Fig. 8.3), chlorosis and stunting. The disease was described by Schaefer and Terry (1976) in Nigeria and is the most important virus (complex) disease in East Africa, where sweetpotato is often the main food staple (Karyeija et al. 1998). The disease was described in Israel by Loebenstein and Harpaz (1960), Peru (Gutiérrez et al. 2003), the USA (Abad et al. 2007), Spain (Trenado et al. 2007), and occurs probably in Italy (Parrella et al. 2006).

It can cause losses over 50%, especially in Uganda and Kenya, though in another study from Uganda, losses were much smaller, probably due to relatively high levels

of virus resistance in their landraces (Gibson et al 1997). In a 3-year field study in Cameroon SPVD reduced root yields by 56–90% in susceptible varieties (Ngeve and Bouwkamp, 1991); and yield reductions of 78% due to SPVD were reported from field trials in Nigeria (Hahn, 1979). In Israel in a 2-year field experiment yield reductions of ~50% were observed in plots planted with SPVD-infected cuttings (Milgram et al. 1996). In Peru in a field study, SPVD reduced root yield by 65–72% (Gutiérrez et al. 2003).

As stated above the optimal approach for controlling SPVD is to supply virus-tested planting material. In practice, however, in Uganda and other African countries this has so far not been achieved. In Uganda, where sweetpotatoes are grown on a large scale the most important control practice, as perceived by farmers, is to plant cuttings from symptomless parents, and destroying diseased plants (Gibson et al. 2000). Although SPVD-resistant sweetpotato landraces are available, these were perceived by farmers to have poor and late yields. Cultivars Tanzania and New Kawogo were relatively resistant to SPVD in low pressure of whiteflies. SPFMV in these cultivars was difficult to detect, because of low virus titer, and was seldom acquired by aphids. However, resistance to SPFMV was not apparent once infected also with SPCSV (Aritua et al. 1998). In SPVD-infected plants SPFMV titers were much higher than in sweetpotato plants infected with SPFMV alone (Cohen et al. 1988; Karyeija et al. 2000), facilitating the spread of SPFMV by aphids (Rossel and Thottappilly 1988). This increase is not associated with enhanced virus movement but is due to a synergistic interaction with the crinivirus (Karyeija et al. 2000).

**Sweetpotato mild mottle virus** Genus *Ipomovirus*, (SPMMV). Synonyms – sweetpotato B virus (Sheffield, 1957). SPMMV has so far been reported from West Africa (Kenya, Uganda, Burundi and Tanzania), South Africa, Indonesia, China, Philippines, Papua New Guinea, India, New Zealand, and Egypt.

SPMMV can cause leaf mottling, stunting and loss of yields. Cultivars differ greatly in their reaction to the virus, some being symptomlessly infected, others apparently immune. The virus is transmitted semipersistently by *B. tabaci*, by grafting and by mechanical inoculation. It is not transmitted by seed or by contact between plants. The virus was transmitted to plants in 14 families (Hollings et al. 1976). Diagnostic species: *N. tabacum*, *N. glutinosa* – vein clearing, leaf puckering, mottling and distortion; *C. quinoa* – local lesions, not systemic; *I. setosa* – conspicuous systemic vein chlorosis (Fig. 8.6). Diagnosis can be confirmed by serological tests of *I. setosa*. Commercial antisera are available. After 3–4 weeks, new growth is almost symptomless. Sap transmission from sweetpotato to test plants is often difficult. The virus is best maintained in *N. glutinosa*, *N. clevelandii* and *N. tabacum*, and can be assayed quantitatively on *C. quinoa*, where SPMMV induces local lesions.

The virus can be purified from systemically infected *N. tabacum* (Hollings et al. 1976). Virions are flexuous rod shaped particles, 800–950 nm in length, containing 5% RNA and 95% protein. The genome consists of single stranded RNA. The viral RNA was cloned and the assembled genomic sequence was 10,818 nts in length with a polyadenylated tract at the 3' terminus. The sequence accession code is Z73124. Almost all known potyvirus motifs are present in the polyprotein of SPMMV, except some motifs in the putative helper-component and coat protein, which

**Fig. 8.6** Symptoms of *Sweetpotato mild mottle virus* on *I. setosa* leaves. Healthy leaf is at top left (Courtesy Dr. J. Moyer)



are incomplete or missing. This may account for its vector relations (Colinet et al. 1998). The coat protein has a MW of 37,700.

SPMMV isolates showed a high level of variability with no discrete strain grouping. Sequences of several SPMMV isolates revealed nt sequence identities of 88.0% and 89.9% or higher for the CP-encoding region and 3'-UTR, respectively, while CP aminoacid (aa) sequences were 93.0–100% identical. Analysis of the CP-encoding nt sequences did not reveal phylogenetically distinguishable groups of SPMMV isolates. Rather, analysis indicated high genetic variability (Tairo et al. 2005).

A synergism was observed in sweetpotato doubly infected by SPMMV and SPCSV (but not by SPFMV) (Untiveros et al. 2007).

**Sweetpotato latent virus** Genus *Potyvirus* (SPLV) is widespread in China and has been reported also from Taiwan, Korea, Indonesia, Japan, Philippines, Uganda, Kenya, South Africa, India, Egypt and New Zealand.

SPLV may cause mild chlorosis but in most cultivars the infection is symptomless. Symptoms often disappear after infection, but the plants remain infected. Crystal inclusions are observed in the nucleus and pinwheels in the cytoplasm.

SPLV isolates from Japan and China were transmitted by the aphid *Myzus persicae* (Usugi et al., 1991) and the virus can be transmitted by mechanical inoculation and by grafting. It is not transmitted by seed.

Diagnostic species: *N. benthamiana* – systemic mosaic and stunting; *N. clevelandii* – systemic pin-prick chlorotic lesions; *C. quinoa*, *C. amaranticolor* – brown necrotic local lesions, no systemic infection; *I. setosa* – systemic mottle. Diagnosis can be confirmed by serological tests of *I. setosa*.

The virus is best maintained in *N. benthamiana* or *N. clevelandii* and can be assayed on *C. quinoa* or *C. amaranticolor*.

The virus can be purified according to Liao et al. (1979). Virus particles are flexuous rods, 750–790 nm in length. The capsid protein has a MW of 36,000. By using MAbs and polyclonal antibodies some epitopes common to SPLV and SPFMV are

found. These can easily be differentiated when potyvirus cross-reactive MAbs are used, indicating a distant relationship (Hammond et al. 1992).

Combining RT-PCR with degenerate oligonucleotide primers derived from the conserved regions of potyviruses, it was possible to identify SPLV, as well as SPFMV and *Sweetpotato G virus* (Colinet et al. 1998), and to differentiate between 2 strains of SPLV (Colinet et al. 1997).

Sequence data accession codes are: X84011 SPLV (Chinese) mRNA for coat protein; X84012 SPLV (Taiwan) mRNA for coat protein. According to Nishiguchi et al. (2001), SPLV has 58% homology to SPFMV-S.

The best way to control this virus, as well as other viruses infecting sweetpotato is by establishing propagation nurseries derived from virus-tested mother plants.

***Sweetpotato virus G*** Genus *Potyvirus* (SPVG) is widespread in China, (Colinet et al. 1994, 1998), and was reported also from Egypt (IsHak et al. 2003), the USA (Souto et al. 2003; Kokkinos and Clark, 2006), Peru (Untiveros et al. 2007), Spain (Trenado et al. 2007), Tanzania (Ndunguru and Kapinga, 2007), Peru (Untiveros et al. 2007), Spain (Trenado et al. 2007), Japan, Ethiopia, Nigeria, and Barbados. Recently, the virus was also found in areas of the Pacific Ocean and their molecular characteristics compared to other isolates (Rannalli et al. 2008). The virus is transmitted mechanically and by aphids *Myzus persicae* and *Aphis gossypii* in a non-persistent manner (Souto et al. 2003). SPVG causes mottling in *I. nil* and chlorotic spotting in *I. setosa* and *I. tricolor* (Souto et al. 2003). Cylindrical inclusions bodies, which consisted of pinwheels and scrolls, were observed in the cytoplasm of epidermal, mesophyll, and vascular cells of infected *I. nil* and *I. setosa* (Souto et al. 2003). Isolates LSU-1 and -3 obtained from sweetpotato plants in Louisiana, USA (Souto et al. 2003) reacted with MAb PTY-1 (Jordan and Hammond, 1991).

A partial sequence of SPVG (X76944) has been obtained after RT-PCR, showing an identity of around 70% and 80% in the amino acid sequence between the complete and conserved core of the coat protein of SPFMV, respectively (Colinet et al. 1998). The SPVG coat protein has 355 amino acids while that of SPFMV has 316 amino acids (Colinet et al. 1994). Comparison with coat protein sequences of known potyviruses indicates that SPVG is a member of the genus *Potyvirus*. Strains of SPVG seem to occur in China (Colinet et al. 1998). SPVG-CH2 shared 89.2% and 90.6% amino acid sequence identities with SPVG-CH in the N-CP and the N-terminal region of the coat protein core (N-CP core), respectively. Sequence identity was much lower with SPFMV and even more so with SPLV and other potyviruses. No biological properties (including host range and symptomatology) or viral characteristics have come to our attention.

***Sweetpotato virus 2*** Tentative member Genus *Potyvirus* (SPV2). Synonyms: Sweetpotato virus II, Ipomoea vein mosaic virus and Sweetpotato virus Y (Moyer et al. 1989; Souto et al. 2003; Ateka et al. 2004).

The virus was first isolated from sweetpotato plants from Taiwan (Rossel and Thottappilly, 1988), then isolates were obtained from sweetpotato clones from China, Portugal, South Africa, China, USA (Souto et al. 2003), Spain (Trenado et al. 2007), Australia (Tairo et al. 2006), and Peru (Untiveros et al. 2007). SPV2 has filamentous particles of 850 nm in length and induces cytoplasmic cylindrical

inclusions consisting of pinwheels and scrolls (Souto et al. 2003; Ateka et al. 2004). The virus was nonpersistently transmitted by *M. persicae* and mechanically transmitted to several species of genera *Chenopodium*, *Datura*, *Nicotiana*, and *Ipomoea*. SPV2 causes vein clearing and leaf distortion on *N. benthamian*, chlorotic local lesions on *Chenopodium* spp., vein mosaic on *I. nil*, *I. setosa* and *I. tricolor* (Souto et al. 2003; Ateka et al. 2007). Isolates LSU-2 and -5 obtained from sweetpotato plants in Louisiana, USA (Souto et al. 2003) reacted with MAb PTY-1 (Jordan and Hammond, 1991).

Although SPV2 has been isolated from sweetpotato plants showing mild symptoms consisting of leaf mottle, vein yellowing and / or ringspots (Rossel and Thottappilly, 1988; Tairo et al. 2006) the significance of this virus to sweetpotato production is not clear as similar symptoms may be caused by other viruses, and sweetpotato cultivars inoculated with SPV2 under greenhouse conditions failed to produce obvious symptoms (Ateka et al. 2004; Souto et al. 2003). However, SPV2 interacts synergistically with SPCSV, increasing symptoms severity on sweetpotato (Kokkinos and Clark, 2006; Tairo et al. 2006), suggesting that SPV2 might be economically important in areas where SPCSV occurs.

It seems that biologically and genetically diverse strains of SPV2 occur. Some differences in test plant reactions and host range appeared to correlate to some extent with the geographic origin and molecular distinctness of the SPV2 isolates (Ateka et al. 2007). Comparison of the CP gene sequences of several isolates revealed nt and aa sequences identities ranging from 81 to 99% and from 86 to 99%, respectively. Phylogenetic analysis of sequences distinguished several groups, which partially correlated with the geographic origin of the isolates (Ateka et al. 2007).

**Sweetpotato mild speckling virus** Genus *Potyvirus* (SPMSV) was isolated from plants cv. Morada INTA from Argentina showing symptoms of “sweetpotato chlorotic dwarf disease” (Di Feo et al. 2000). These plants show chlorosis, dwarfing, vein clearing, and leaf distortion (Alvarez et al. 1997; Di Feo et al. 2000). The severity of the disease depends on the presence of SPMSV in the complex. Symptoms in plants infected with SPFMV and SPCSV were milder than those in plants infected with the three viruses (Di Feo et al. 2000). SPMSV is synergistic in plants infected with SPCSV but not to SPFMV (Untiveros et al. 2006).

SPMSV has been found in Peru, Argentina, Philippines, China, Indonesia, Egypt, South Africa, Nigeria, and New Zealand. SPMSV is transmitted mechanically and by *M. persicae* in a nonpersistent manner. However, it does not react with the MAb PTY-1 that recognises a cryptotope conserved in most aphid-transmitted potyviruses (Di Feo et al. 2000; Jordan and Hammond, 1991). Its host range is restricted to *Convolvulaceae*, *Chenopodiaceae*, and *Solanaceae*. *I. setosa* and *I. nil* react with vein clearing, blistering, leaf deformation and mosaic; *N. benthamiana* with vein clearing, and reduction, deformation and down rolling of leaves, and *C. quinoa*, and *N. tabacum* “Samsun” with local infections (Fuentes et al. 1997). SPMSV can easily be detected by ELISA.

This virus has flexuous particles of c. 800 nm. The coat protein sequences of SPMSV showed 63% identity with SPFMV, 68 to 70% with SPLV, 57% with SPVG, and 73% with *Potato virus Y* (Alvarez et al., 1997). Cylindrical inclusion (bundles,

lamine aggregates, and pinwheels, neither circles nor scrolls) were observed in the cytoplasm of infected *I. setosa* and *I. batatas* (Nome et al. 2006). This high resemblance favors inclusion of SPMSV within the *Potyvirus* genus.

**Sweetpotato leaf speckling virus** Possible Genus *Polerovirus* (SPLSV) was isolated from a sweetpotato accession DLP 1541 from CIP's germplasm collection, showing slight leaf curl and whitish speckling symptoms (Fuentes et al. 1996) (Fig. 8.7). It is found occasionally in farmers' materials. The virus was also found in Cuba. Infected plants of cv. Jewel yielded c. 20% less under greenhouse conditions. Virus causes dwarfing and leaf curling in addition to chlorotic and necrotic spotting in *I. nil* and *I. setosa*. SPLSV is transmitted by grafting and by *Macrosiphon euphorbiae* in a persistent manner, but not by *M. persicae*, *A. gossypii* or mechanically. Diagnosis can be confirmed by nucleic acid spot hybridization (NASH) from *I. setosa* or *I. nil* (Fuentes et al. 1977). Concentration of SPLSV increases towards the bottom of the plant. On leaves, the virus was detected in the basal part of the leaves and petioles, but not in the apical halves.

SPLSV can be purified from leaves or roots of infected plants (Fuentes et al. 1996). Virions are isometric c. 30 nm in diameter. RT-PCR amplification of RNA from SPLSV-infected plants, using degenerate primers designed to amplify sequences in Luteovirus- or PLRV-RNA, yielded c. 500 bp and c. 600 bp DNA fragments, respectively. The nucleotide sequence of these fragments encoded two polypeptides (coat protein and 17K) characteristic of luteo- and poleroviruses sequences (GenBank accession DQ655700). The coat- and the 17K protein of SPLSV have similarities to of 70% and 63% those of PLRV, respectively. NASH using a PLRV probe can therefore detect SPLSV in sap. However, the heterologous probe gives a lower signal than the homologous reaction with PLRV. SPLSV was not detected in ISEM or ELISA tests using PLRV antibodies.

**Sweetpotato chlorotic flecks virus** (SPCFV) (tentatively member in Genus *Carlavirus*) was isolated from sweetpotato accession DLP 942 from the CIP's germplasm collection, showing fine chlorotic spots (flecks) (Fuentes and Salazar, 1992). SPCFV was detected in samples from Peru, Japan, China, Korea, Taiwan,



**Fig. 8.7** Sweet potato cv Jewel infected with SPLSV showing whitish speckling

Cuba, Panama, Bolivia, Colombia, Brazil, Uganda, Philippines, India, Indonesia, Australia and New Zealand (Aritua et al. 2007; Fuentes and Salazar, 1992; Gibson et al. 1997; Usugi et al. 1991; Jones and Dwyer 2007; Fletcher et al. 2000). This virus is transmitted mechanically but not by aphids and not by seed and has a limited host range in the families *Convolvulaceae* and *Chenopodiaceae*. SPCFV induces in *I. nil* fine chlorotic spots and vein clearing on the first and second true leaf. The virus can be recovered and detected from symptomatic leaves, but not from symptomless ones. No clear symptoms are observed on infected *I. setosa* (only transient vein clearing on 1–2 leaves). In *C. murale* and *C. quinoa* react with local necrotic lesions. Graft inoculated cvs. Nemañete, Jewel, Luby 3074, IITA TIS 2498 did not show symptoms. Infectivity of SPCFV is lost at dilution between  $10^{-2}$  and  $10^{-3}$ , and after storage between 1h and 1 day. The thermal inactivation point is between 50 and 60 °C. Some cytological alterations (hypertrophy of chloroplasts) were observed in *I. nil* but no inclusion bodies (“pinwheels”).

Synergism was observed in sweetpotato doubly infected by SPCFV and SPCSV (Untiveros et al. 2006).

SPCFV seems to be serologically related to an uncharacterised Japanese isolate (sweetpotato symptomless virus – SPSV). Although SPCFV and SPSV are serologically related, they differ in some characteristics. SPSV has flexuous short and long particles c. 710 to 760 nm and 1,430 to 1,510 nm in length, and it infects only *Ipomoea* spp., while SPCFV particles are flexuous rod c. 750–800 in length. Also, *I. nil* infected with SPSV showed stronger symptoms (vein clearing, curling and vein necrosis) than those caused by SPCFV. The thermal inactivation point of SPSV (70–80 °C) is higher than that of SPCFV.

Virus can be purified from cotyledons and leaves of infected *I. nil* (12 day after inoculation). The virus particles are flexuous rods c. 750–800 in length with a capsid polypeptide of Mr 34.5 kDa (Fuentes and Salazar, 1992). Its genome (GenBank accession NC\_006550) is 9,104 nts long (excluding the poly(A) tail) and potentially includes six open reading frames (ORFs) (Aritua et al. 2007). Its genomic organization and similarity resembles that of genus *Carlavirus* but is the least related to typical carlaviruses. SPCFV genome differed from that of typical carlaviruses in being comparatively large (9,104 vs. 6,480–8,739 nts). The remarkable size of the SPCFV genome results from a large ORF1 (237.5 vs. 200–235 kDa) and the presence of an abnormally long untranslated region (233 vs. <50 nts) between ORF4 and ORF5. Its close relative is melon yellowing-associated virus, a proposed carlavirus from Brazil, which is transmitted by whiteflies (*B. tabaci* biotype B) (Aritua et al. 2007). Comparison of the CP gene of geographically diverse SPCFV isolates showed a wide variation both in their CP nucleotide and the corresponding aa sequences ranging from 75.1–99% and 88.3–99.7%, respectively. At aa level, the East African isolates were 94.0–99.7% similar while isolates of the non-East African origin were more divergent (88.3–96.7%). The phylogenetic analysis of the CP aa sequences separated the SPCFV isolates into various clusters that corresponded to their geographical origin (Aritua et al. 2007).

**Sweetpotato caulimo-like virus** (SPCaLV) has been found together with SPFMV in widely scattered geographical locations – Madeira, New Zealand, Papua New

Guinea and Solomon Islands (Atkey and Brunt, 1987), China (Gao et al. 2000) and Uganda (WISARD Project Information, 1991 Aritua et al. 2007). Also reported from Kingdom of Tonga, Kenya, Nigeria, Egypt, Philippines, Australia, and Puerto Rico.

Sweetpotato plants infected by SPCaLV are usually without symptoms, though a few leaves occasionally have chlorotic or purple spots, probably induced by SPFMV (Atkey and Brunt, 1987). Virus is not transmitted by mechanical inoculation, not by contact between plants or by seed.

Graft inoculation of *I. setosa* seedlings with SPCaLV resulted in appearance of chlorotic flecks along minor veins or circular interveinal spots on several leaves. Such leaves sometimes become completely chlorotic, wilt and die. Fifty nm caulimolike particles can be detected in negatively stained sap of *I. setosa* (but not from *I. batatas*), and c. 10 times more by ISEM. In the cytoplasm of infected *I. setosa* leaf cells numerous isometric particles and intracellular inclusions are readily detected. The inclusions are spherical or ovoid, up to 4  $\mu$ m in diameter with a large central lacuna and usually several smaller peripheral lacunae; they do not contain virions (Atkey and Brunt, 1987).

The viral genome consists of double-stranded DNA and the coat protein has a Mr 42–44 kDa (Brunt et al. 1996).

**Sweetpotato leaf curl virus** Genus *Begomovirus* (SPLCV) was first reported by Shinkai (1979) and Liao et al. (1979) from Japan and Taiwan. The virus has also been reported from USA, Far East and samples from Brazil, Mexico, China, Korea, Puerto Rico (Lotrakul et al. 2002), Kenya (Miamo et al. 2006), and Peru (Fuentes and Salazar 2003). In infected young sweetpotato plants upward curling of leaves and vein swelling on young plants are observed, though later only few symptoms remain and plants become symptomless. SPLCV can cause up to 30% reductions in yield. The virus is transmitted by *B. tabaci* biotype B in a persistent manner and by grafting, but not mechanically or by seeds. Under experimental conditions, the virus is transmitted by its vector at relatively low levels (Valverde et al. 2004a). Various *Ipomoea* species were susceptible to SPLCV, as *I. purpurea* causing leaf curl and stunt (Osaki and Inouye, 1991), *I. aquatica* – yellow vein symptoms, *I. nil*, *I. setosa* and *N. benthamiana* – leaf curl symptoms (Lotrakul et al. 1998). The disease is caused by a gemini virus belonging to the *Begomovirus* group (Onuki et al. 2000). Cytopathic changes in the nucleoplasm, as fibrillar rings and crystalline arrays of virus-like particles, typical of gemini-infected tissue were also observed (Osaki and Inouye, 1991). The virus was partially purified yielding typical geminate particles with a size of c. 18  $\times$  30 nm, and Western blot analysis revealed serological relationships between SPLCV and *Bean golden mosaic virus* (Onuki et al. 2000). A complete sequence of SPLCV (AF104036, 2,828 nts) has been determined by Lotrakul and Valverde (1999). Its genomic DNA and organization is similar to that of monopartite begomoviruses. The partial nucleotide sequence identity (from 87% to nearly 100%) of the AC1 gene of different SPLCV isolates and the phylogenetic analysis of them suggest that there may be more than one species of the SPLCV (Lotrakul et al. 2002).

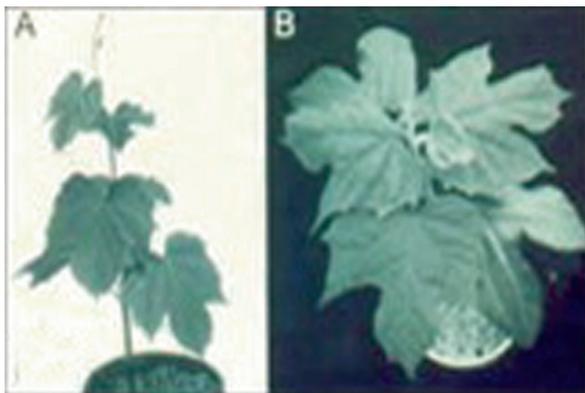
Coinfection of SPLCV and SPFMV in *I. nil* and *I. setosa* induce severe leaf distortion, general chlorosis and stunting. SPLCV titers/replication were signifi-

cantly greater in plants co-infected with SPFMV compared to plants inoculated with SPLCV alone (Kokkinos and Clark, 2004).

**Sweetpotato leaf curl Georgia virus** Genus *Begomovirus* (SPLCGV). This virus, formerly known as *Ipomoea leaf curl virus* (Lotrakul et al. 2003), is transmitted by *B. tabaci* biotype B. It occurs in USA, Puerto Rico, and India (Prasanth and Hegde, 2008). The virus caused leaf curl symptoms in several *Ipomoea* species. Unlike SPLCV, SPLCGV did not cause yellow vein symptoms on *I. aquatica* and *I. cordatotriloba*. Its genome of 2,773 nts (AF326775) has an organization typical of other Old World monopartite begomoviruses. Sequence comparisons showed that SPLCGV was similar to SPLCV and to another *Ipomoea*-infecting begomovirus, *Ipomoea yellow vein virus* (IYVV) from Spain with about 76 and 78% nucleotide sequence identity, respectively. Although the coat protein of SPLCGV was nearly identical to that of SPLCV and IYVV, the sequence of the common region and the AC1, AC2, AC3 and AC4 ORFs were different (Lotrakul et al. 2003).

**Ipomoea yellow vein virus** Genus *Begomovirus* (IYVV) was first found infecting *I. indica* plants showing yellow vein symptoms in Spain (Banks et al. 1999) and Italy (Bridson et al. 2005), then found infecting cultivated sweetpotato plants. The complete nucleotide sequence (AJ132548) confirmed its begomovirus nature. Contrary to SPLCV, IYVV was not transmitted by *B. tabaci* biotype B (or Q and S). Phylogenetic analysis of the three *Ipomoea*-infecting begomoviruses species (SPLCV, SPLCGV, and IYVV) recognised by the ICTV revealed that these viruses form a separate cluster that place them apart from all other begomovirus.

**Ipomoea crinkle leaf curl virus** (ICLCV). In 1992, Cohen et al. (1997) observed on *I. setosa*, grafted with scions from sweetpotato cv. Georgia Jet plants, introduced from an unknown source in North America, atypical symptoms of little leaf and crinkle symptoms (Fig. 8.8). Geminata particles were observed in crude sap preparations. The virus was transmitted by *B. tabaci* in a persistent manner and by grafting, but not mechanically. The virus was transmitted to several *Ipomoea* species, including *I. hederacea*, *I. trifida*, *I. nil*, *I. littoralis* and *I. setosa* and induced symptoms on them, including on some cultivars of *I. batatas*, but not on cv. Georgia



**Fig. 8.8** Symptoms of *Ipomoea crinkle leaf virus* on *I. setosa*. Left- healthy plant

Jet. It might be mentioned that SPLCV did not infect *I. hederacea* and symptoms induced by ICLCV on *I. setosa* and *I. nil* differed from those described for SPLCV (Chung et al. 1985). Based on the host range ICLCV is considered to be distinct from SPLCV, but its exact relationship to other identified viruses remains unclear.

**Cucumber mosaic virus** Genus *Cucumovirus* (CMV) is one of the most widespread plant viruses, recorded in more than 190 species, belonging to more than 40 families (Francki et al. 1979). CMV has been isolated from *I. setifera* (Migliori et al. 1978) and Martin (1962) succeeded in transmitting CMV by mechanical inoculation to *I. nil*, *I. purpurea*, *I. lacunosa*, and *I. trichocarpa* but not to *I. batatas* cv. Puerto Rico. Cohen and Loebenstein (1991) failed in transmitting CMV to healthy sweetpotato plants. However, sweetpotatoes carrying the whitefly-transmitted SPSVV can easily be infected by CMV by aphid, mechanical or graft inoculations (Cohen and Loebenstein, 1991). Untiveros et al. (2007) found that CMV was able to infect sweetpotatoes without the assistance of SPCSV. It appears that CMV strains are nonspecific for infection in sweetpotato. CMV isolated from cucumber (Cohen and Loebenstein, 1991) or *Arracacia xanthorrhiza* (Untiveros et al. 2007) were able to infect sweetpotato plants assisted by SPSVV or no, respectively. In some fields in Israel during the 80es heavy infections together with SPFMV and SPSVV caused severe yellowing and stunting (Fig. 8.9). Later, when farmers used certified planting material such symptoms were hardly found. Apparently, the presence of another virus (SPSVV) facilitates replication or translocation of some CMV strains in sweetpotato. It may be that there is a gene silencing mechanism that inhibits replication of such CMV strains in healthy sweetpotato and is suppressed by SPSVV, allowing CMV to replicate and/or move in the sweetpotato plant.

It is interesting to note that although CMV occurs worldwide, in sweetpotato it has been reported so far only from Israel, Japan, New Zealand (Fletcher et al. 2000), Spain, West Africa (Clark and Moyer, 1988) and Egypt. CMV was not found in Kenya (Ateka et al. 2004) and Tanzania (Ndunguru and Kapinga, 2007) even though SPCSV is very widespread SPCSV strains do not support infection of sweetpotatoes with *Cucumber mosaic virus* (CMV), while SPSVV is needed to infect sweetpotatoes with CMV.



**Fig. 8.9** Sweet potato plants (cv. Georgia Jet) severely affected by yellowing and stunting caused by double infection with *Cucumber mosaic virus* and *Sweet potato sunken vein virus*

**Sweetpotato yellow dwarf virus** Genus *Ipomovirus*, Family *Potyviridae* (SPYDV) in *I. batatas* was first reported from Taiwan by Liao et al. (1979), Far East, and Brazil ([www.abam.com.br/livros/cargil/Capitulo%202/Capitulo%2022.pdf](http://www.abam.com.br/livros/cargil/Capitulo%202/Capitulo%2022.pdf)). The virus in sweetpotato causes severe systemic leaf chlorosis and stunting. The virus was transmitted by *B. tabaci* in a persistent manner, as well as by grafting and by mechanical inoculation. Susceptible hosts include in addition to sweetpotato, *Chenopodium* spp., *Gomphrena globosa*, *Datura stramonium*, *Cassia occidentalis*, *Sesamum indicum* (Chung et al. 1986). SPYDV induces in *I. setosa* stunting, general leaf chlorosis, small distinct chlorotic spot, and vein chlorosis. Virions are filamentous, not enveloped, flexuous, with a clear modal length of 750 nm. The viral coat protein has a MW of 33,000. Pinwheel inclusions are present in the cytoplasm of infected cells.

The virus can be eliminated by meristem tip culture. Stemtips up to 2.5 cm below the meristems of infected plants which had received a one or two-month treatment of heat (37 °C) were found to be free of SPYDV (Green and Luo, 1989).

**Sweetpotato vein mosaic virus** Possible Genus *Potyvirus* (SPVMV) was first reported from Argentina by Nome (1973). In sweetpotato, the virus causes a disease called “batata crespa” and infected plants shows vein clearing, mosaic, twisting of leaves, stunting, and noticeable reduction in number and size of the fleshy roots. The virus can be transmitted in a nonpersistent manner by *M. persicae*, as well as by grafting and by mechanical inoculation (Nome et al. 1974). The virus was transmitted to several *Ipomoea* species, including *I. alba*, *I. nil*, *I. setosa*, *I. lacunosa*, *I. hederacea*, *I. tricolor*, *I. trichocarpa*, *I. kurtziana*, *I. fistulosa*, and *I. angulata*, generally inducing systemic vein clearing and mosaic. Twisting, chlorosis and size reduction of leaves were marked in *I. setosa*, *I. nil*, *I. hederacea*, and *I. batatas*. Virus particles are flexuous rod, 761 nm in length. Virions are found in the cytoplasm of all parts of the host plant. Pinwheels and scrolls and laminated aggregates inclusions are present in the cytoplasm of infected cells (Brunt et al. 1996). The virus could be eliminated by heat treatment (Nome and Salvadores, 1979). Unfortunately the original culture and antiserum are not available.

**Sweetpotato C-6 virus.** The virus was isolated from sweetpotato Sosa 29, from the Dominican Republic, showing chlorotic spots (Fuentes, 1994). In Louisiana it was found in most of the black ornamental sweetpotatoes (Blackie, Ace of Spades, and Black Beauty) (Clark and Valverde, 2000) Its host range is restricted to *Convolvulaceae*. The virus induces on *I. nil* and *I. setosa* fine chlorotic spots and vein clearing (Fig. 8.10) but on sweetpotato cv. Paramanguino, Costanero, and Jonathan chlorotic spots. This is followed by yellowing and leaf drop. The virus is transmitted by grafting and by mechanical inoculation (with a low efficiency), using sap from *I. nil* roots but not from leaves. Attempts to transmit the virus with *M. persicae* were unsuccessful. The virus was detected in samples from Peru, Uganda, Cuba, USA, Dominican Republic, Philippines, Indonesia, Egypt, Kenya, South Africa, New Zealand, and Puerto Rico. C-6 virus gave a mild reaction with Potato virus S (PVS) antiserum.

C-6 virus was purified from infected *I. setosa* plants (Fuentes, 1994). Virus particles are flexuous rods, 750–800 nm in length. No “pinwheels” were observed in infected *I. nil*, but chloroplasts and mitochondria showed hypertrophy.

**Fig. 8.10** *Ipomoea setosa* infected with C-6 virus showing fine chlorotic spots and vein clearing



**Sweetpotato C-3 virus**, a suspected flexuous virus, was isolated from a sweetpotato co-infected with SPFMV and showing leaf deformation, vein clearing and mosaic, from Brazil (Fuentes and Salazar, 1989). The virus is transmitted by grafting but neither mechanically nor by aphid *M. persicae*. It induces mosaic, leaf deformation and vein clearing in *I. setosa*. Infected sweetpotato cv. Paramonguino showed mottle symptoms and cv. Georgia Red interveinal mottling. *N. benthamiana* graft-inoculated with C-3 showed yellowing vein, mosaic and leaf deformation, but *I. nil* did not show symptoms.

**Sweetpotato ringspot virus** Genus *Nepovirus* (SPRSV) was observed in *I. batatas*, imported from Papua New Guinea into the UK. (Brown et al. 1988). The virus on sweetpotato occasionally causes chlorotic ringspots, which disappear soon after infection. The virus can be transmitted by grafting and mechanical inoculation. The virus infects a wide range of experimental hosts, including *I. setosa* – faint systemic chlorotic leaf mottling; *N. megalosiphon* – necrotic rings and systemic mottling; *N. benthamiana* – systemic leaf deformations; *C. quinoa*, *C. murale* – transient systemic chlorosis ([www.ncbi.nlm.nih.gov/ICTVdb/](http://www.ncbi.nlm.nih.gov/ICTVdb/)). Virions are not enveloped, 28 nm in diameter, which sediment as three components (T, M and B) with sedimentation coefficients of 47, 81 and 130 S and buoyant densities in cesium chloride of 1.32, 1.45 and 1.52 g cm<sup>-3</sup>, respectively. All components contain a single polypeptide of Mr 56.6 × 10<sup>3</sup>. T component particles contain only protein but those of M and B components contain ss-RNA 8,848 nt (Mr 2.93 × 10<sup>6</sup>) and 6,670 nt (Mr 2.31 × 10<sup>6</sup>), respectively. Virions can be found in leaves and roots. Although the virus has physico-chemical properties resembling those of nepoviruses, it showed no serological relationship to any of 13 recognised nepoviruses (including *Arabidopsis mosaic virus*, *Cassava green mottle virus*, *Cherry leaf roll virus*, *Raspberry ringspot virus*, *Strawberry latent ringspot virus* and *Tobacco ringspot virus*) (Brunt and Brown, 1990).

## ***New Potyviruses***

Two previously undescribed viruses affecting sweetpotato in North Carolina, USA have been reported by Moyer et al. (2002). These two isolates gave no reaction with antiserum for SPFMV, SPMMV, SPLV, SPCFV, SPMSV, C-6, SPCaLV, and SPCSV, but showed novel dsRNA profiles. The first is a 9–10 Kbp single species and may correspond to a potyvirus, the second is a 7.5 Kbp dsRNA with two putative sub-genomic fragments. Both isolates tested negative in ELISA with a universal antiserum for potyviruses. From the first profile one isolate, GW Beauregard, was selected for dsRNA purification, cloning and sequencing. This isolate when graft inoculated on cv. Tanzania induced severe mosaic, vein banding and leaf deformation. The CP gene sequences were used for the phylogenetic analysis. Sequence of the putative potyvirus was more closely related to SPMMV than SPLV, SPVG, and SPFMV.

*Sweetpotato chlorotic stunt virus* is a wrongly name given to a tentatively *Potyvirus* isolated from sweetpotatoes from the Caribbean area showing stunting and chlorotic leaves (Brown et al. 1988). This virus should not be confused with SPCSV (genus *Crinivirus*) previously described here. We are including some information on this virus, which could be useful when characterizing new potyviruses. This virus is not transmitted by aphids, but is readily sap-transmissible to a wide range of herbaceous species of which *N. benthamiana* is a good propagation host and *C. amaranticolor* and *C. quinoa* are convenient local lesion assay hosts. The virus was also detected in Kenya, Uganda (Wambugu, 1991), and Zimbabwe (Chavi et al. 1997). Virus particles are flexuous rod, 850–950 nm in length, which contain ss-RNA and a single capsid polypeptide of Mr 43 kDa. The virus induces “pinwheels”.

## ***Viruses Infecting Wild Ipomoeas or Sporadically Sweetpotato***

A virus infecting *I. aquatica* was reported by Brunt et al. (1996). *Tobacco streak virus* and *Tobacco mosaic virus* (Clark and Moyer, 1988) have also been reported infecting sweetpotato.

## ***Little-leaf Phytoplasma Disease***

Little-leaf Phytoplasma Disease also referred to as witches’ broom, was first described by Van Velsen (1967) from Papua and New Guinea. It has been reported also from the Ryuku islands (Yang, 1969), Tonga (Kahn and Monroe, 1969), Solomon Islands Dabek and Sagar, (1978), Taiwan (Yang, 1969) and Northern Australia (Gibb et al. 1995). This disease has an exceptionally long incubation period in sweetpotato, ranging from 50 to 186 days following graft transmission (Clark and Moyer, 1988). First symptoms are veinclearing of otherwise normal leaves. Subsequent symptoms include large reductions in the size of leaves and internodes and an erect habit of

the tips of the vines. Plants which become infected at an early stage yield poorly, producing only one or two pencil-thin tubers (Pearson et al. 1984). Typical phytoplasma bodies were observed in phloem sieve cells. Meristem tip culture was found to be effective in eliminating the phytoplasma from the infected sweetpotato tissue (Green et al. 1989). The phytoplasma could be diagnosed by PCR, and the full-length chromosome was determined as 600 Kbp, which is one of the smallest phytoplasma genome sizes (Gibb et al. 1995). Symptoms on graft-inoculated *I. setosa* may take 6 months or more to develop (Moyer et al. 1989). The disease is lethal in *I. ericolor*, with a relatively short incubation period of 35 to 49 days, making this species a candidate for an indexing host (Clark and Moyer, 1988).

## ***Control***

At present the best way to control virus diseases in sweetpotato is to supply the grower with virus-indexed propagation material. Such plantlets can be obtained from meristems shoot tip cultures, also in combination with cryotherapy (Wang and Valkonen, 2008). Such programs are operating in Israel and in the Shandong province of China (Gao et al. 2000). In Israel, as a result of planting virus-tested material, yields increased at least by 100%, while in China increases ranged between 22–92%. The payoff to the farmer has been high and in Israel use of certified material is common practice, while in China the use of pathogen-free material is being extended. In African countries such programs are operating only on a limited scale, because sweetpotatoes are grown mainly as a food security crop, and not as a commercial one.

Some cultural/phytosanitation practices may facilitate control of viral diseases. Examples of such cultural practices include selection of disease-free planting material, destroying (roguing) of diseased plants and wild *Ipomoea* spp, especially in young crops, isolating new crops (15–20 m far) from old diseased crops, destroying crop residues, and protecting crops with barriers or intercropping with maize (Gibson and Aritua, 2002).

Breeding programs might be a future answer and such programs are in operation in Uganda, combining SPVD resistance with desirable agronomic traits such as yield, earliness and acceptable culinary quality (Karyeija et al. 2000; Mwanga et al. 2002). Progress has been made and several cultivars were released (Turyamureeba et al. 1998). It will have to be seen if these cultivars will retain their resistance in other places where different strains of virus components of SPVD may be present. Thus, several clones that were resistant to SPFMV in CIP's tests were found to be susceptible, when Israeli (unpublished) and Ugandan isolates were tested (Karyeija et al. 1998).

So far transgenic approaches did not result in cultivars resistant to SPVD. Apparently the small residues of SPCSV were in the plant were sufficient to cause together with SPFMV the severe SPVD.

**Table 8.1** A list of recognized viruses known to infect sweetpotato

Virus	Shape and size	Vector	Distribution	References
<b>SwPLV-<i>Polyvirus</i></b>	Flexuous, 750–790 nm	Aphid	Uganda, Kenya, Taiwan, Japan, China, Indonesia, Philippines, India	10, 11, 25
<b>SPMSV-<i>Potyvirus</i></b>	Flexuous, 800 nm	Aphid	Argentina, Peru, Indonesia, Philippines, Egypt	1, 12, 18
<b>SPLSV-<i>Polerovirus?</i></b>	Isometric, 30 nm	Aphid	Peru, Cuba	17
<b>CMV-<i>Cucumovirus</i></b>	Isometric, 30 nm	Aphid	Israel, Uganda, Kenya, Japan, Egypt.	7, 13
<b>SPVMV-<i>Potyvirus?</i></b>	Flexuous, 761 nm	Aphid	Argentina	29, 30
<b>SPSVV-<i>Crimivirus</i></b>	Flexuous, 850–950 nm	Whitefly	Africa (Nigeria, Uganda, Kenya, Zaire, Zambia, Madagascar), Asia (Taiwan, China, Indonesia, Philippines), America (USA, Brazil, Argentina, Peru), Israel, Egypt	6
<b>SPMMV-<i>Ipomovirus</i></b>	Flexuous, 800–950 nm	Whitefly	West Africa (Kenya, Uganda, Burundi, Tanzania), Indonesia, Papua New Guinea, India, Egypt	11, 21, 32
<b>SPYDV-<i>Ipomovirus</i></b>	Flexuous, 750 nm	Whitefly	Taiwan	25
<b>SPLCV-<i>Begomovirus</i></b>	Geminate	Whitefly	Taiwan, Japan, Korea, China, USA, Puerto Rico, Peru	25, 26, 33
<b>ICLCV-<i>Geminivirus?</i></b>	Geminate	Whitefly	USA?	8

Table 8.1 (continued)

Virus	Shape and size	Vector	Distribution	References
<b>SPVG-Potyvirus</b>	Flexuous	Unknown	China	9, 11
<b>SPCFV</b>	Flexuous, 750–800 nm	Unknown	Peru, Japan, Brazil, China, Cuba, Panama, Colombia, Bolivia, Indonesia, Philippines, Uganda, India, Austria	16, 20, 35
<b>C-6</b>	Flexuous 750–800 nm	Unknown	Peru, Uganda, Cuba, Dominican Republic, Indonesia, Philippines, USA, Puerto Rico, Egypt	14
<b>SPCV-Caulimo-like</b>	Isometric, 50 nm	Unknown	South Pacific region ( Kingdom of Tonga, Papua New Guinea, New Zealand, Salomon Islands, Austria), Madeira, Kenya, Uganda, China, Egypt, Puerto Rico,	2, 19
<b>SPCSV-Potyvirus</b>	Flexuous, 850–950 nm	Unknown	Caribbean Region, Kenya, Uganda, Zimbabwe	3
<b>SPRSV-Nepovirus</b>	Isometric, 28 nm	Unknown	Papua New Guinea, Kenya	3, 4
<b>TSV-Iilar-like</b>	Isometric, 30 nm	Unknown	Guatemala	5
<b>C-3</b>	Flexuous?	Unknown	Brazil. Unknown in other countries	15
<b>TMV-Tobamovirus</b>	Rod, 300 nm	None	USA	5

1) Alvarez *et al.* 1997; 2) Atkey and Brunt, 1987; 3) Brown *et al.* 1988; 4) Brunt *et al.* 1996; 5) Clark and Moyer, 1988; 6) Cohen *et al.* 1992; 7) Cohen and Loebenstein, 1991; 8) Cohen *et al.* 1997; 9) Colinet *et al.* 1994; 10) Colinet *et al.* 1997; 11) Colinet *et al.* 1998; 12) Di Feo *et al.* 2000; 13) Francki *et al.* 1979; 14) Fuentes, 1994; 15) Fuentes and Salazar, 1989; 16) Fuentes and Salazar, 1992; 17) Fuentes *et al.* 1996; 18) Fuentes *et al.* 1997; 19) Gao *et al.* 2000; 20) Gibson *et al.* 1997; 21) Hollings *et al.* 1976; 22) Karyeija *et al.* 1998; 23) Karyeija *et al.* 2001; 24) Kreuzer *et al.* 2000; 25) Liao *et al.* 1979; 26) Lotrakul *et al.* 2002; 27) Moyer, 1986; 28) Moyer *et al.* 1989; 29) Nome, 1973; 30) Nome *et al.* 1974; 31) Pozzer *et al.* 1995; 32) Sheffield, 1957; 33) Shinkai, 1979; 34) Sim, 2001; 35) Usugi *et al.* 1991.

As *Ipomoea* spp. are susceptible to most sweetpotato viruses it is of importance to survey weeds as hosts and potential reservoirs of viruses.

## Concluding Remarks

The demand for root and tuber crops in developing countries to ensure a sustainable food supply is predicted to grow steadily over the next decades. Sweetpotato is one of the major crops, and much of it is grown in developing countries, for supplying carbohydrates and beta-carotene. In Sub-Saharan Africa it serves as a food security crop, and is important also in relief famine. As sweetpotato is propagated vegetatively control of virus diseases is an important factor in obtaining high yields. Over 20 viruses have so far been reported to infect sweetpotato (Table 8.1), but several of them are still not characterised. Antisera are available only for a limited number of viruses. Broad-spectrum PCR for detecting new uncharacterised viruses has been demonstrated especially with potyviruses and geminiviruses. It is important to determine whether the new isolated viruses are strains of previously reported viruses or represent a new virus species. Their identification, economic impact and effect in mixed infections, especially with Poty-, Geminiviruses and SPCSV/SPSVV are an important task for the future.

Knowledge of sweetpotato viruses and their genetic variability that provides information on the components of virus population is the base for designing appropriate control measures that deploy host resistance in sweetpotato crops. Damages caused by sweetpotato viruses are mostly through synergistic mixed infections. SPCSV can synergise with viruses member in potyvirus, ipomovirus, carlavirus, and cucumovirus. It means that development of durable resistance to synergistic diseases, as SPVD, should be resistance to SPCSV/SPSVV.

It was demonstrated that transgenic sweetpotato containing the CP gene from SPFMV or RdRp genes for both SPFMV and SPCSV did not confer resistance (Kreuze et al. 2008) to 'complex' infection with SPCSV, causing the SPVD. SPCSV encodes two unique proteins: RNase 3 and p22, which are expressed early in infection and are co-operatively able to suppress RNA silencing. As not all isolates of SPCSV contains the p22 gene (Cuellar et al. 2008) but are able to synergise with different viruses, it is hoped that transgenes containing the RNase3 gene will achieve resistance to the SPVD.

Recently, a landrace sweetpotato variety 'Huachano' that is extremely resistant to SPFMV was genetically engineered for resistance to SPCSV (Kreuze et al. 2008). In this cultivar as in many others (Karyeija et al. 2000) the high levels of resistance to SPFMV breaks down following infection with SPCSV and the plants succumb to the severe SPVD.

Research on sweetpotato viruses has not received the attention it deserves and the international community and its organizations should increase their efforts to strengthen sweetpotato research in general and sweetpotato virus focused research in particular.

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