

Chapter 2

A review of viruses infecting sweet potatoes

2.1 Introduction

Diseases are of great importance in sweet potato production, viruses being the biggest problem worldwide because sweet potatoes are very susceptible to viral diseases (Bolton and du Plooy, 1984; Owour, 2000). Since the early 1946-1950's, viral diseases have been recognised as causing deterioration in the quality and yield of sweet potatoes in South Africa in Brits, Nelspruit, Mpumalanga, Pretoria and Rustenburg (McClellan and Klessner, 1947). During a baseline survey on cassava and sweet potato which was conducted by ARC-Roodeplaat Vegetable and Ornamental Plant Institute in 1997, viral diseases were ranked high based on symptom expression in the field and results of indexing the collected vines to the indicator plant, *Ipomoea setosa* Kerr. (van der Mescht *et al.*, 1997). Production of sweet potato is greatly constrained by diseases that cause yield reduction by up to 98% (Mukasa, 2001), and viruses severely limit sweet potato production in the tropics and in Africa, accounting for over 50% of yield reduction in Nigeria and Uganda (Mukiibi, 1977). In South Africa the crop is sensitive to viral diseases in hot tropical regions such as the Lowveld of Mpumalanga and the Limpopo province (Coertze *et al.*, 1996). Data on yield loss caused by viruses is generally lacking in South Africa and in some other areas where the crop is grown due to the practice of piece-meal harvesting which makes it difficult to measure yield (Jericho, 1999).

A suspected virus disease of sweet potato was first reported in 1944, in South Carolina (Steinbauer and Kushman, 1971). Presently, more than 14 virus diseases of sweet potato have been reported (Moyer and Salazar, 1989; Brunt *et al.*, 1996; Di Feo *et al.*, 2000). Studies indicate that there are five major potyviruses that affect sweet potato production: sweet potato feathery mottle potyvirus (SPFMV), sweet potato mild mottle ipomovirus (SPMMV), sweet potato latent potyvirus (SPLV), sweet potato vein mosaic potyvirus (SPVMV) and sweet potato yellow dwarf

ipomovirus (SPYDV) (Moyer and Salazar, 1989; Winter *et al.*, 1992; Chavi *et al.*, 1997; Di Feo *et al.*, 2000). In addition to these, viruses belonging to other taxonomic groups have been found to infect sweet potatoes such as sweet potato caulimovirus (SPCaLV), sweet potato chlorotic stunt crinivirus (SPCSV), sweet potato ring spot nepovirus (SPRSV), sweet potato leaf speckling luteovirus (SPLSV), and sweet potato chlorotic fleck potyvirus (SPCFV) (Winter *et al.*, 1992; Brunt *et al.*, 1996). SPFMV and SPMMV, and possibly SPLV have been reported in South Africa as the most important viruses of sweet potatoes (Thompson and Mynhardt, 1986; Jericho and Thompson, 2000). SPFMV in combination with SPCSV causes a devastating sweet potato virus disease (SPVD), which has been shown to be the principal virus disease of sweet potato in East Africa (Aritua *et al.*, 1998b; Gibson *et al.*, 1998, Karyeija *et al.*, 2000). A recently reported virus disease of sweet potato is sweet potato chlorotic dwarf disease (SPCDD), a synergistic combination of three viruses (SPFMV, SPMMV and SPCSV) found in Argentina (Di Feo *et al.*, 2000).

Although there are many sweet potato virus diseases described in the literature, the aetiology of many of these diseases has not been determined and reliable detection and characterisation procedures have not been documented (Moyer and Salazar, 1989). A summary of sweet potato viruses, their distribution and vectors is given in Table 2.1.

2.2 Aetiology of sweet potato viruses

2.2.1 Sweet potato feathery mottle potyvirus (SPFMV)

SPFMV is the most important and common virus infecting sweet potato and is found wherever sweet potato is grown, including South Africa (Clark and Moyer, 1988; Moyer and Salazar, 1989; Karyeija *et al.*, 1998a; Cipriani *et al.*, 2000; Jericho and Thompson, 2000; Kreuze *et al.*, 2000). Many strains have been identified and it has been referred to as russet crack virus, sweet potato virus A, sweet potato ring spot virus, sweet potato leaf spot virus and internal cork virus (Sheffield, 1957; 1958; Clark and Moyer, 1988; Moyer and Salazar, 1989;

Karyeija *et al.*, 1998a;). It has been detected in association with other viruses (Moyer and Salazar, 1989).

SPFMV is a member of the family Potyviridae, genus Potyvirus, the largest family of plant viruses (Clark and Moyer, 1988; Moyer and Salazar, 1989). Like other potyviruses, the virions are elongate, flexuous rods with a monopartite, single-stranded, positive sense RNA molecule (Karyeija *et al.*, 1998a; 1998b; Fauquet and Mayo, 1999), with a particle length of 830-850nm (Cohen *et al.*, 1988; 1997).

SPFMV is readily transmitted by aphids in a non-persistent manner through brief feeds of only 20-30 seconds (Schaefers and Terry, 1976; Clark and Moyer, 1988; Ames *et al.*, 1997; Owour, 2000). Aphids such as the green peach aphid (*Myzus persicae* Sulz.), cotton aphid (*Aphis gossipii* Glover) and groundnut aphid (*A. craccivora* Koch.) are reported to be the most efficient vectors of SPFMV (Schaefers and Terry, 1976; Karyeija *et al.*, 1998a). The virus is not seed-borne, but like many viruses infecting vegetatively propagated plants, it is also disseminated in tubers and vine cuttings (Cadena-Hinojosa and Campbell, 1981).

Symptoms on sweet potato leaves appear as faint to distinct, irregular chlorotic spots occasionally bordered by purplish pigmentation (Clark and Moyer, 1988; Cadena-Hinojosa and Campbell, 1981; Moyer and Salazar, 1989; Ames *et al.*, 1997). Diffuse mottle along the main veins and vein clearing can also be seen on infected leaves (Clark and Moyer, 1988; Moyer and Salazar, 1989; Karyeija *et al.*, 1998a; 1998b). Leaf symptoms vary with cultivar susceptibility, climatic condition, plant age and strain virulence (Thompson and Mynhardt, 1986; Clark and Moyer, 1988; Ames *et al.*, 1997). Some genotypes also exhibit external and internal root symptoms which include external cracking and internal necrosis depending on the cultivar and virus isolate (Cali and Moyer, 1981; Karyeija *et al.*, 1998a).

SPFMV is mostly restricted to members of the *Ipomoea*, which include *I. nil* L. (Roth), *I. setosa* and sweet potatoes (Thompson and Mynhardt, 1986; Clark and Moyer, 1988; Karyeija *et al.*, 1998a; 1998b). Single infections of SPFMV have been reported to cause severe symptoms, which are variable in *I. nil*, and *I. setosa* (Schaefers and Terry, 1976). Some isolates infect *Chenopodium amaranticolor* Coste & Reyn, *C. quinoa* Willd, or *Nicotiana benthamiana* Gray, but others seem to be restricted to *Ipomoea* species (Thompson and Mynhardt, 1986; Clark and Moyer, 1988; Karyeija *et al.*, 1998a; 1998b).

2.2.2 Sweet potato chlorotic stunt crinivirus (SPCSV)

It was formerly known as sweet potato sunken vein virus (Hoyer *et al.*, 1996; Milgram *et al.*, 1996; Ames *et al.*, 1997; Alicai *et al.*, 1999) and SPVD-associated closterovirus (Winter *et al.*, 1992). It is a member of the family Closteroviridae, genus Crinivirus (Aritua *et al.*, 1998a; Fauquet and Mayo, 1999; Karyeija *et al.*, 2001) with a positive-stranded RNA genome (Karyeija *et al.*, 2000; Gibson and Aritua, 2002). Early classification was based on particle lengths, long types with particles from 1.200 to 2.000nm and short types with particles from 700 to 800nm (Liu *et al.*, 2000). Karyeija *et al.* (2000) showed that SPCSV remains confined to the phloem and at similar or slightly lower titer in the SPVD-affected plants. SPCSV is transmitted semi-persistently by whitefly, *Bemisia tabaci* Genn. and *Trialeurodes abutilonea* Haldeman, and not by mechanical means (Winter *et al.*, 1992; Hoyer *et al.*, 1996; Gibson *et al.*, 1998; Alicai *et al.*, 1999; Gibson and Aritua, 2002). Although SPCSV can infect plants by itself, it has been identified as a component of synergistic complexes with other viruses such as SPFMV and SPMSV (Schaefers and Terry, 1976; Gibson *et al.*, 1998; Di Feo *et al.*, 2000; Gibson and Aritua, 2002).

Symptoms vary with plant genotypes (Gibson *et al.*, 1998). Symptoms caused by SPCSV alone are relatively mild in sweet potato and *I. setosa* and plants may become mildly stunted, chlorotic and purpling of leaves can occur (Aritua *et al.*, 1998a; 1998b; Gibson *et al.*, 1998; Alicai *et al.*, 1999, Gibson and Aritua, 2002).

Affected plants commonly produce less than half the tuberous root yield of symptomless ones (Aritua *et al.*, 1998b). SPCSV infects sweet potato (Kaitisha and Gibson, 1999; Karyeija *et al.*, 2000) and *I. setosa* (Gibson *et al.*, 1997; 1998). It was reported by Cohen *et al.* (2001) that SPCSV was also found to infect *Lisianthus* (*Eustoma grandiflorum* Raf. Shinn.). SPCSV is known to be distributed in Nigeria, Zambia and Tanzania (Gibson *et al.*, 1998; Kaitisha and Gibson, 1999), and it is also found in Kenya and the Caribbean (Ames *et al.*, 1997).

2.2.3 Sweet potato virus disease (SPVD)

SPVD is a name used to describe plants affected by a range of severe symptoms associated with a dual infection of SPCSV and SPFMV (Schaefer and Terry, 1976; Carey *et al.*, 1997; Gibson *et al.*, 1997; Aritua *et al.*, 1998a; 1998b; Karyeija *et al.*, 1998a; 1998b; Kaitisha and Gibson, 1999; Gibson and Aritua, 2002). The first report of SPVD may have been in the eastern Belgian Congo (DR Congo) in 1939 (Carey *et al.*, 1997; 1999). It is also the most serious disease of sweet potato in Africa, especially in Uganda (Karyeija *et al.*, 1998b; Alicai *et al.*, 1999). Co-infection between SPFMV and SPCSV results in the development of SPVD characterised by severe leaf distortion including narrowing (strap-like), vein clearing and crinkled leaves, chlorosis, discolouration and stunting of plants (Ngeve and Boukamp, 1991; Carey *et al.*, 1997; 1999; Alicai *et al.*, 1999; Kreuze *et al.*, 2000; Ndunguru and Aloyce, 2000; Karyeija *et al.*, 2001; Gibson and Aritua, 2002).

SPVD infection caused yield depression of up to 90% in sweet potato cultivars tested in 1986 in Ekona, Cameroon (Ngeve and Boukamp, 1991). Gibson *et al.* (1998) showed that sweet potato infected with this virus-complex produce *c.* 2% of the yield of unaffected sweet potato cuttings. The incidence of SPVD was revealed to be higher in fields planted as monocrop than in other cropping patterns (Ndunguru and Aloyce, 2000). Although it has been reported in East and West

Africa, it has not been reported in southern Africa (Chavi *et al.*, 1997; Jericho and Thompson, 2000).

2.2.4 Sweet potato mild mottle ipomovirus (SPMMV)

SPMMV is a member of the family Potyviridae, genus Ipomovirus (Hollings *et al.*, 1976; Fauquet and Mayo, 1999). It was described as an RNA-containing virus with filamentous particles c. 950nm long, found in East Africa and it was referred to as SPV-T in preliminary reports (Hollings *et al.*, 1976; Clark and Moyer, 1988; Brunt *et al.*, 1996). Like other Potyviruses, SPMMV induces cytoplasmic inclusions (Hollings *et al.*, 1976). The virus is transmitted by whitefly, *Bemisia tabaci*, in a persistent manner and by grafting but not by contact between plants or by seeds (Hollings *et al.*, 1976; Moyer and Salazar, 1989; Brunt *et al.*, 1996). The virus is readily transmitted to a fairly wide range of herbaceous plant species (Hollings *et al.*, 1976; Moyer and Salazar, 1989; Brunt *et al.*, 1996). The host range has been demonstrated to include 45 species in 14 plant families (Hollings *et al.*, 1976; Brunt *et al.*, 1996). *Chenopodium quinoa*, *I. setosa*, *Nicotiana tabacum* L., *N. benthamiana*, *N. glutinosa* L. and *N. clevelandii* L. are good local lesion hosts and are highly sensitive to infection by SPMMV (Hollings *et al.*, 1976; Clark and Moyer, 1988). Symptoms include leaf mottling and stunting (Brunt *et al.*, 1996; Ames *et al.*, 1997). Symptoms of SPMMV-infected *I. setosa* are similar to those induced by SPFMV, which include vein clearing and distortion (Clark and Moyer, 1988; Ames *et al.*, 1997). Loss of yield also occurs in SPMMV-infected plants (Hollings *et al.*, 1976). Different sweet potato genotypes differ greatly in susceptibility and reaction to the virus, some being symptomless and others apparently immune (Brunt *et al.*, 1996). SPMMV has been identified in Kenya, Uganda, Tanzania and Burundi and South Africa but yield effects are unknown (Brunt *et al.*, 1996; Ames *et al.*, 1997; Jericho and Thompson, 2000). Taking into consideration the wide host range of the virus and the polyphagous nature of its vector (*Bemisia tabaci*), it is probable that the geographical spread of SPMMV is much wider than presently known (Jericho, 1999).

2.2.5 Cucumber mosaic cucumovirus (CMV)

CMV was first observed in 1986 in Israel where it severely infected sweet potato fields (Cohen *et al.*, 1988). CMV was also found to infect sweet potatoes in Israel and the United States (Ames *et al.*, 1997). All CMV infected plants were also infected with SPFMV (Cohen *et al.*, 1988; Moyer *et al.*, 1989). For CMV to replicate in sweet potato a helper virus is required. CMV is easily transmitted to sweet potato plants mechanically and by aphid inoculation if the acceptor plant carries the whitefly-transmitted virus, which provides the necessary requirement for CMV replication (Cohen and Loebestein, 1991; Cohen *et al.*, 1992). Symptoms include stunting, chlorosis and yellowing (Cohen and Loebestein, 1991; Cohen *et al.*, 1992).

2.3 Other viral diseases of sweet potato

In addition to SPFMV, SPMMV, SPCSV and SPVD, there are other viruses reported to infect sweet potato: sweet potato leaf speckling luteovirus (SPLSV), sweet potato latent potyvirus (SPLV), sweet potato ring spot nepovirus (SPRSV), sweet potato caulimovirus (SPCaLV), sweet potato yellow dwarf ipomovirus (SPYDV), sweet potato vein mosaic potyvirus (SPVMV), sweet potato leaf curl badnavirus (SPLCV), sweet potato leaf curl geminivirus-US (SPLCV-US), *Ipomoea* crinkle leaf curl geminivirus (SPCLCV), and sweet potato phytoreovirus. Of all these viruses, only SPCFV, SPRSV, SPLV and SPCaLV have been reported to infect sweet potato in Africa (Atkey and Brunt, 1987; Brunt *et al.*, 1996; Carey *et al.*, 1997; Gibson *et al.*, 1997). Sweet potato chlorotic fleck virus (SPCFV) is a potyvirus, flexuous, with the length of 750-800nm and the vector for transmitting this virus is not yet known (Salazar and Fuentes, 2000).

Sweet potato chlorotic dwarf disease (SPCDD), a synergistic combination of SPFMV, SPMSV and SPCSV, is another virus complex, which was reported in Argentina (Di Feo *et al.*, 2000; Gibson and Aritua, 2002). SPCDD-affected plants are characterised by reduced leaf area, stunting and leaves show severe mosaic,

blisters, and distortion. The combination of three viruses and the different combinations of two of the three viruses account for the variability of SPCDD symptomatology observed in the field. This is very similar to the synergism of SPFMV and SPCSV causing the devastating SPVD-complex in Africa, except that in the case of SPCDD, three viruses are involved (Moyer and Salazar, 1989; Winter *et al.*, 1992). Sweet potato virus G (SPV G) has also been reported as another important virus of sweet potato (Colinet *et al.*, 1994a; Alvarez *et al.*, 1997). The length of the coat protein of SPVG is longer than that of SPFMV, but the virus is closely related to SPFMV and it is considered a potyvirus (Colinet *et al.*, 1994a; Brunt *et al.*, 1996). Sweet potato virus II (SPV II) has also been reported in Taiwan as another aphid-transmitted potyvirus that infects sweet potato (Salazar and Fuentes, 2000).

2.3.1 Sweet potato leaf speckling luteovirus (SPLSV)

SPLSV is a Luteovirus, which is transmitted only by the aphid, *Macrosiphum euphorbiae* Thomas (Fuentes *et al.*, 1996; Fauquet and Mayo, 1999). It infects sweet potato but it can infect 45 species of 14 plant families (Moyer and Salazar, 1989), including *I. nil* and *I. setosa* and symptoms include clear whitish flecks or specks on the leaves (Fuentes *et al.*, 1996).

2.3.2 Sweet potato latent potyvirus (SPLV)

SPLV is a member of the family Potyviridae, genus Potyvirus and was initially designated sweet potato virus N (Clark and Moyer, 1988; Moyer and Salazar, 1989; Brunt *et al.*, 1996; Fauquet and Mayo, 1999). It is a flexuous rod, 700-750nm long and it does not depend on another virus for replication (Clark and Moyer, 1988; Brunt *et al.*, 1990). It is transmitted by mechanical inoculation and no vector has been found to transmit the virus (Clark and Moyer, 1988; Moyer and Salazar, 1989; Brunt *et al.*, 1990; 1996). It is not transmitted by contact between plants nor by seed (Clark and Moyer, 1988; Brunt *et al.*, 1990; 1996). Infection of many sweet potato cultivars by SPLV does not result in obvious foliar symptoms (Clark and Moyer, 1988; Moyer and Salazar, 1989; Brunt *et al.*, 1996).

The host range includes many *Convolvulus*, *Chenopodium* and some *Nicotiana* spp. (Brunt *et al.*, 1996). Although SPLV induces mild symptoms in *I. setosa*, it is easily detected by serology (Clark and Moyer, 1988; Moyer and Salazar, 1989).

2.3.3 Sweet potato ring spot nepovirus (SPRSV)

SPRSV is a member of the family Comoviridae, genus Nepovirus (Brunt *et al.*, 1996). Virions are isometric, not enveloped and 28nm in diameter with conspicuous capsomere arrangements and the genome consists of single-stranded RNA (Brunt *et al.*, 1996). It was first reported in sweet potato cultivars imported from Papua New Guinea (Brunt *et al.*, 1996) and Kenya (Ames *et al.*, 1997). Symptoms on sweet potato include occasional chlorotic ring spots when temperature reach 26-28°C, but plants can be symptomless (Brunt *et al.*, 1996). The virus is transmitted by aphids (*Myzus persicae*) and mechanical inoculation but not by contact between plants (Loebestein and Harpaz, 1959; Brunt *et al.*, 1990; 1996). Susceptible hosts include plants from 3-9 families including *I. setosa* (showing faint systemic chlorotic leaf mottling), *Chenopodium quinoa*, some *Nicotiana* spp. and *Glycine max* L. (Brunt *et al.*, 1990; 1996).

2.3.4 Sweet potato caulimovirus (SPCaLV)

SPCaLV is a caulimovirus with a genome consisting of double stranded DNA with isometric particles of 50nm in diameter (Atkey and Brunt, 1987; Moyer and Salazar, 1989). The virus is not transmitted by either *Aphis gossypii* or *Myzus persicae* (Moyer and Salazar, 1989), nor by mechanical inoculation, contact between plants or seed (Brunt *et al.*, 1990; 1996). It is transmitted by grafting only (Brunt *et al.*, 1990; 1996). SPCaLV is found in the U.S.A, Puerto Rico, New Zealand, Kenya, Australia, Papua New Guinea, Solomon Islands, Tonga and Madeira (Atkey and Brunt, 1987; Clark and Moyer, 1988; Moyer and Salazar, 1989; Brunt *et al.*, 1990; 1996). Host plants include *I. setosa*, which shows chlorotic veinal flecks or circular interveinal chlorotic spots (Atkey and Brunt, 1987; Clark and Moyer, 1988; Brunt *et al.*, 1990; 1996). Symptoms on host plants

also include chlorosis resulting in wilting and premature death of the leaves (Clark and Moyer, 1988; Moyer and Salazar, 1989).

2.3.5 Sweet potato yellow dwarf ipomovirus (SPYDV)

SPYDV is an Ipomovirus of the family Potyviridae (Clark and Moyer, 1988; Brunt *et al.*, 1996; Fauquet and Mayo, 1999). Virions are long, flexuous rods with a modal length of 750nm (Clark and Moyer, 1988; Brunt *et al.*, 1996). Virion morphology and the vector of SPYDV are similar to that of SPMMV (Clark and Moyer, 1988; Moyer and Salazar, 1989). The virus is transmitted by whitefly (*Bemisia tabaci*) in a persistent manner and by mechanical inoculation (Clark and Moyer, 1988; Brunt *et al.*, 1990; 1996). Symptoms on sweet potato include mottling, stunting (dwarfing), chlorosis, and poor roots and tubers (Steinbauer and Kushman, 1971; Clark and Moyer, 1988; Moyer and Salazar, 1989; Brunt *et al.*, 1996). Poor soil fertility and low temperatures favour expression of symptoms (Clark and Moyer, 1988; Moyer and Salazar, 1989). The root systems of infected plants are poorly developed and the fleshy roots are not marketable (Clark and Moyer, 1988; Moyer and Salazar, 1989). SPYDV frequently occurs in combination with SPFMV (Clark and Moyer, 1988; Moyer and Salazar, 1989).

2.3.6 Sweet potato vein mosaic potyvirus (SPVMV)

SPVMV was first found in sweet potato in Argentina and it has been classified as a Potyvirus within the family Potyviridae (Clark and Moyer, 1988; Brunt *et al.*, 1990; 1996; Fauquet and Mayo, 1999). A data comparison of the particle morphologies of SPVMV and SPFMV indicate that SPVMV has a modal length of 761nm, which is significantly shorter than that of SPFMV (Moyer and Salazar, 1989; Brunt *et al.*, 1996). It is transmitted non-persistently by aphids (*Myzus persicae*) and by mechanical inoculation (Clark and Moyer, 1988; Brunt *et al.*, 1996). Host plants include *I. batatas* and other *Ipomoea* spp. (Clark and Moyer, 1988). Sweet potatoes infected with SPVMV are severely stunted and produce fewer roots (Clark and Moyer, 1988; Moyer and Salazar, 1989; Brunt *et al.*, 1996). The virus also causes severe foliar symptoms similar to those of SPFMV in

I. setosa (vein clearing, mosaic, chlorosis and twisting of leaves) (Clark and Moyer, 1988; Moyer and Salazar, 1989; Brunt *et al.*, 1996).

2.3.7 Sweet potato leaf curl badnavirus (SPLCV)

SPLCV is considered a member of the Badnavirus group with short, rod-shaped particles found in the cytoplasm of phloem-cells (Clark and Moyer, 1988). Its geographical distribution is limited to Japan and Taiwan (Clark and Moyer, 1988; Brunt *et al.*, 1996). The virus is transmitted by whitefly (*Bemisia tabaci*) in a persistent manner and by grafting (Clark and Moyer, 1988; Brunt *et al.*, 1990; 1996). It is not transmitted by contact between plants, mechanical inoculation nor by seed (Clark and Moyer, 1988; Brunt *et al.*, 1990; 1996). Its symptoms are typical leaf curl (Thottapilly and Rossel, 1988). Host plants include *I. batatas*, and *I. nil* (Clark and Moyer, 1988; Brunt *et al.*, 1990; 1996).

2.3.8 *Ipomoea* crinkle leaf curl geminivirus (ICLCV)

ICLCV is a geminivirus, which is transmitted by a whitefly *Bemisia argentifolii* Bellows and Perring in a persistent manner and by grafting onto *Ipomoea* spp. (Cohen *et al.*, 1997). ICLCV is not transmitted mechanically (Cohen *et al.*, 1997). It is associated with geminated particles, inducing distinct symptoms on several *Ipomoea* spp. (Cohen *et al.*, 1997). Symptoms are leaf curling, crinkling and sunken veins on *I. setosa*, *I. hederacea* Jacq., *I. trifida* Kunth G., *I. littoralis* Blume and *I. batatas* (Cohen *et al.*, 1997). Vein clearing can also be observed on *I. hederacea*, *I. trifida* (Cohen *et al.*, 1997).

2.3.9 Sweet potato leaf curl virus-US

SPLCV-US is another geminivirus, which was reported in the United States to infect sweet potatoes (Lotrakul *et al.*, 1998). The disease is transmitted by whitefly (*Bemisia tabaci*) and symptoms on *Ipomoea aquatica* Forssk., *I. cordatotriloba* Dennst. and *I. fistulosa* Mart. ex Choisy consist of yellow vein mottling and leaf curling. Infected *I. setosa* shows symptoms such as mild leaf curl, interveinal chlorosis and stunting.

2.3.10 Sweet potato (?) phytoeovirus

It is a Reoviridae virus, which was first reported in *I. aquatica* from material tested in the U.K. (Brunt *et al.*, 1996). The virus induces mild leaf chlorosis symptoms (Brunt *et al.*, 1996). The virus is restricted to members of the Convolvulaceae, more especially *I. setosa* (Brunt *et al.*, 1996).

Table 2.1 A summary of sweet potato viruses, their distribution and vectors

Virus	Known distribution	Vectors	References
SPFMV	Nigeria, Uganda, Kenya, Zimbabwe, Louisiana, U.S.A and South Africa	<i>Myzus persicae</i>	Brunt <i>et al.</i> , 1996; Chavi <i>et al.</i> , 1997; Gibson <i>et al.</i> , 1998; Kaitisha and Gibson, 1999; Jericho and Thompson, 2000.
SPMMV	Burundi, Kenya, Tanzania, Uganda and South Africa	<i>Bemisia tabaci</i>	Hollings <i>et al.</i> , 1976; Brunt <i>et al.</i> , 1996; Jericho and Thompson, 2000.
SPCSV	Nigeria, Uganda, Kenya, Zambia, and Israel	<i>Bemisia tabaci</i>	Winter <i>et al.</i> , 1992; Cohen <i>et al.</i> , 1992; Hoyer <i>et al.</i> , 1996; Brunt <i>et al.</i> , 1996; Ames <i>et al.</i> , 1997; Gibson <i>et al.</i> , 1998; Kaitisha and Gibson, 1999.
SPCFV	South East Africa, Indonesia, China, Japan, Peru, Philippines, Brazil, Cuba, Colombia, Bolivia and Central and South America	?	Ames <i>et al.</i> , 1997; Carey <i>et al.</i> , 1997; Gibson <i>et al.</i> , 1997; Salazar and Fuentes, 2000.
SPRSV	Papua New Guinea and Kenya	?	Brunt <i>et al.</i> , 1996.
SPLV	Taiwan, Japan, China and Israel	?	Clark and Moyer, 1988; Brunt <i>et al.</i> , 1990; 1996; Ames <i>et al.</i> , 1997.
SPCaLV	U.S.A., Puerto Rico, New Zealand, Kenya, Papua New Guinea, Australia, Solomon Island and Madeira	?	Atkey and Brunt, 1987; Moyer and Salazar, 1989.
CMV	Israel, Kenya and the United States	<i>Aphis gossypii</i>	Cohen and Loebestein, 1991; Ames <i>et al.</i> , 1997.
SPLSV	Peru	<i>Macrosiphum euphorbiae</i>	Clark and Moyer, 1988; Moyer and Salazar, 1989; Fuentes <i>et al.</i> , 1996.
SPVMV	Argentina	Aphids	Clark and Moyer, 1988; Moyer and Salazar, 1989; Brunt <i>et al.</i> , 1996.
SPYDV	Taiwan	<i>Bemisia tabaci</i>	Brunt <i>et al.</i> , 1996.
SPCDD	Argentina	<i>Bemisia tabaci</i> & <i>Myzus persicae</i>	Di Feo <i>et al.</i> , 2000.
ICLCV	North America	<i>B. argentifolii</i>	Cohen <i>et al.</i> , 1997.
SPLCV	Taiwan and Japan	<i>B. tabaci</i>	Clark and Moyer, 1988; Brunt <i>et al.</i> , 1996.
SPV G	China	?	Colinet <i>et al.</i> , 1994a.
SPV II	Taiwan	Aphids	Salazar and Fuentes, 2000.

? = The vector has not yet been identified

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2.4 Sweet potato virus detection and diagnosis

Detection and characterisation of sweet potato viruses is crucial in the understanding of the epidemiology of the disease(s) caused by these viruses, development of infectivity-based forecasting systems and control strategies (Joubert *et al.*, 1979; Jericho, 1999). Study of several of these diseases has been hampered by lack of simple detection techniques (Carey *et al.*, 1999). Sweet potato viruses have been detected by observing symptom expression in the field and host range studies (Chavi *et al.*, 1997) and some by their vector relationship (Schaefer and Terry, 1976). The primary tests to detect sweet potato viruses are bioassays on indicator plants by observing symptoms, vector transmission procedures and serology using enzyme linked immunosorbent assay (ELISA) (Moyer and Salazar, 1989; Chavi *et al.*, 1997).

2.4.1 Biological methods

2.4.1.1 Mechanical inoculation

Mechanical inoculation is the application of virus-bearing fluids (sap) to the surface of the leaves of an indicator plant in such a way that the virus can enter the cells and elicit a response or symptom in that plant (Sheffield, 1957a; Noordam, 1973; Chavi *et al.*, 1997). Mechanical inoculation is done by grinding sweet potato leaf tissue using a mortar and pestle in a phosphate buffer of the pH 7.2 containing 0.01M sodium diethyl dithiocarbamate (Cohen *et al.*, 1988; Chavi *et al.*, 1997; Jericho and Thompson, 2000). An abrasive such as carborundum or diatomaceous earth is normally added to the leaf extracts during mechanical inoculation to enhance the process (Cohen *et al.*, 1988; Chavi *et al.*, 1997; Jericho and Thompson, 2000). This method was first used by Sheffield when he transmitted sweet potato virus A and B mechanically, as a mixed infection, from naturally infected plants to healthy sweet potato plants and *Ipomoea* test plants (Sheffield, 1957).

Mechanical inoculations have been used to establish the host range of the different viruses including viruses infecting sweet potatoes (Sheffield, 1957;

1958; Walkey, 1991; Chavi *et al.*, 1997; Jericho and Thompson, 2000). Species in the family Convolvulaceae such as *I. nil* and *I. purpurea* L. (Roth) are most frequently used as host plants for mechanical inoculations. Other plant species such as *Chenopodium amaranticolor*, *C. quinoa*, *Cucumis sativus* L., *Datura stramonium* L., and *N. benthamiana* have also been used to transmit sweet potato viruses mechanically (Cali and Moyer, 1981; Cohen *et al.*, 1988; Chavi *et al.*, 1997). *I. nil* is more sensitive than *I. setosa* (Cohen *et al.*, 1997). Symptoms such as leaf puckering, vein clearing, chlorotic vein slashing and leaf distortion have been seen following graft and mechanical inoculation of *I. nil* and *I. setosa* with sweet potato cuttings from small-scale farmers in South Africa (Thompson and Mynhardt, 1986; Jericho and Thompson, 2000).

2.4.1.2 Grafting (Indexing)

This method is useful for viruses that cannot be mechanically transmitted (Walkey, 1991). This is done by grafting a two-leaf shoot of sweet potato onto *I. setosa* and monitoring for the development of symptoms (Anonymous, 1978). Attempts to transmit sweet potato virus A, transmitted by aphids (*Myzus persicae*), and sweet potato virus B, transmitted by whiteflies (*Bemisia tabaci*), were successful by cleft grafting sweet potato cuttings with infected scions (Sheffield, 1957; 1958). Virus A and B seemed to be readily transmitted to sweet potato and even to other species of *Ipomoea* (Sheffield, 1957; 1958). Plants for virus testing are grown in the greenhouse to produce stems, which are later assayed by grafting to *I. setosa* and to sweet potato clones because nearly all known viruses of sweet potato plants also infect *I. setosa* (Esbenshade and Moyer, 1982; Moyer and Salazar, 1989). Side grafting of sweet potato plants (cuttings) onto *I. setosa* induces symptoms such as vein clearing, puckering, leaf deformation and chlorotic spotting which start showing in 3-5 weeks, depending on temperature, age of the plant and the virus concentration (Clark and Moyer, 1988; Moyer and Salazar, 1989; Winter *et al.*, 1992; Gibson *et al.*, 1998; Jericho and Thompson, 2000).

Although *I. setosa* is susceptible to many viruses infecting sweet potato and is a good assay host, it is of no diagnostic value for some, such as cucumber mosaic virus (CMV) and tobacco streak virus (TSV) (Moyer and Salazar, 1989; Winter *et al.*, 1992). Although graft transmission is a reliable method for detection, it requires greenhouse space, labour, good insect control, and several weeks for reliable diagnosis (Karyeija *et al.*, 1998a).

2.4.2 Insect transmission

The most common vectors of sweet potato viruses are *Myzus persicae*, *Aphis gossypii*, *Macrosiphum euphorbiae* and *Bemisia tabaci* (Brunt *et al.*, 1996). Only whiteflies transmitted sweet potato virus B when species such as leafhoppers (unidentified), froghoppers (unidentified), mealybugs (*Planococcus kenyae* Lepelley) and mites (*Aceria* spp.) were experimentally tested for transmitting the virus (Sheffield, 1957; 1958). Various aphid species were tested for sweet potato virus A, but *Myzus persicae* transmitted the virus non-persistently and symptoms appeared 3-4 weeks after inoculation (Sheffield, 1957a; 1958). Insect vectors are allowed an acquisition access feeding and are then kept on the acceptor plant for inoculation of the virus (Winter *et al.*, 1992). Results are based on symptom expression (Winter *et al.*, 1992). Aphids (*Aphis gossypii*) can transmit CMV to cucumbers, *I. nil*, and to sweet potato plants if the acceptor plant carries the whitefly transmitted agent (Cohen *et al.*, 1988).

2.4.3 Serological detection

Enzyme-linked immunosorbent assay (ELISA) has been used many times to detect plant viruses since its introduction in 1976 (Voller *et al.*, 1976; Clark and Adams, 1977; Bar-Joseph *et al.*, 1979). It is based on the covalent linkage of an enzyme to an antibody, registering the occurrence of an antigen-antibody complex by rapid enzymatic development of a distinctly coloured product (Burrows *et al.*, 1984; Converse and Martin, 1990). Together with bioassay on indicator plants, ELISA is the primary test to detect plant viruses with polyclonal or monoclonal

antibodies (Voller *et al.*, 1976; Bar-Joseph *et al.*, 1979; Ben-Ze'ev *et al.*, 1988; Converse and Martin, 1990; Singh and Barker, 1991; Walkey, 1991).

Heterologous precipitin tests were used previously for detecting severe russet crack (SRC) and mild russet crack (MRS) strains of SPFMV (Cali and Moyer, 1981). Due to lack of sensitivity required, ELISA, which detected SPFMV in partially purified and symptomatic leaves of *I. batatas* and *I. incarnata* Choisy, was developed and was found to be a faster, convenient and more sensitive method to confirm SPFMV in sweet potato foliages and other *Ipomoea* spp. (Cadena-Hinojosa and Campbell, 1981; Thottapilly and Rossel, 1988). The most common ELISA methods that are used are double antibody sandwich-ELISA (DAS-ELISA) and indirect ELISA using the antisera specific for each virus (Hammond *et al.*, 1992; Kaitisha and Gibson, 1999). They have been used to examine the relationship between SPFMV, sweet potato latent virus (SPLV), and sweet potato mild mottle virus (SPMMV) and confirming that they are three distinct potyviruses (Hammond *et al.*, 1992). Triple antibody sandwich-ELISA is preferably used to test for SPCSV (Gibson *et al.*, 1998). Sap extract is prepared in a standard sample buffer of phosphate-buffered saline containing Tween and polyvinyl pyrrolidone (PBS-Tween, + 2% PVP) (Abad and Moyer, 1991; Jericho and Thompson, 2000).

Nitrocellulose membrane-ELISA (NCM-ELISA) is also used for detecting viruses such as SPFMV and sweet potato chlorotic fleck virus (SPCFV) in sweet potato and *I. setosa* (Abad and Moyer, 1991; Karyeija *et al.*, 1998a; 2000; Jericho and Thompson, 2000). It produces results consistent to those obtained using triple antibody sandwich-ELISA (TAS-ELISA) (Gibson *et al.*, 1997). Monoclonal (Mabs) and polyclonal (Pabs) antibodies produced against purified sweet potato viruses have been used to detect sweet potato viruses (Cadena-Hinojosa and Campbell, 1981; Chavi *et al.*, 1997; Kaitisha and Gibson, 1999; Jericho and Thompson, 2000; Karyeija *et al.*, 2001). Results from naturally infected sweet potato and grafts indicated that SPFMV occurred in leaves of infected plants at

concentrations approaching the limits of ELISA (Esbenshade and Moyer, 1982). Thus, proper tissue selection and timing of the assay is critical (Esbenshade and Moyer, 1982). It is now widely accepted that SPFMV is most reliably detected by ELISA (Abad and Moyer, 1991). Advantages of ELISA are that it can detect viruses in small amounts or in low concentrations and speedy reaction, which is why ELISA is important in virus detection (Voller *et al.*, 1976; Clark and Bar-Joseph, 1984; Siitari and Kurppa, 1987; Walkey, 1991). But many serological methods such as most types of ELISA are not sensitive enough to detect antigens at low concentrations and those occurring in complexes as in the case of sweet potato (Voller *et al.*, 1976; Clark and Bar-Joseph, 1984; Siitari and Kurppa, 1987; Walkey, 1991). The high cost of good quality enzymes and their substrates can also prevent the widespread use of ELISA in developing countries (Singh and Barker, 1991).

2.4.4 Electron microscopy (EM) and immunosorbent electron microscope (ISEM)

EM is done to assign viruses according to a particular group and to confirm the presence of virus particles (Cohen *et al.*, 1992; Winter *et al.*, 1992). Viruses can be viewed either in leaf dip preparations or in ultra-thin sections of embedded material (Cali and Moyer, 1981; Cohen *et al.*, 1992; Winter *et al.*, 1992; Jericho and Thompson, 2000). Negative stains such as uranyl acetate have been used to observe long filamentous rod-shaped virus-like particles of SPCSV and other virus particles such as those of SPFMV in *I. setosa* and sweet potato plants (Cohen *et al.*, 1992; Winter *et al.*, 1992; Jericho and Thompson, 2000).

In order to be able to detect and determine the relationships among viruses, serological specific-electron microscope or immunosorbent electron microscope (ISEM) is used (Cadena-Hinojosa and Campbell, 1981; Hoyer *et al.*, 1996). It refers to the trapping of virus particles onto grids, which have been coated with specific virus antisera, to decorate the trapped virus particles, and observation using an electron microscope (Di Feo *et al.*, 2000; van der Merwe, 2001). Polyclonal antisera to viruses such as SPFMV-RC, SPFMV, SPMMV, SPLV,

SPCV and SPCSV have been used to detect these viruses (Hoyer *et al.*, 1996; Chavi *et al.*, 1997).

2.4.5 Polymerase chain reaction (PCR) and hybridisation

PCR is the use of synthetic nucleic acid probes or the *in vitro* amplification of the specific nucleic acid sequences (Chavi *et al.*, 1997). It involves making multiple copies of a particular sequence in a genome (virus genome) that are then used to identify the presence of a particular disease (Rosa, 2001). Lack of progress in virus identification and classification and due to the frequent occurrence of mixed infections and synergistic complexes in sweet potatoes (Clark and Moyer, 1988; Moyer and Salazar, 1989), PCR technology has been used for identifying and characterising members of the potyviruses infecting sweet potato (Chavi *et al.*, 1997; Colinet *et al.*, 1998). This method provides a convenient way of detecting mixed infections and unknown viruses without preliminary separation and purification of the components of the viral complexes (Colinet *et al.*, 1994b; 1998). Genus-specific PCR and subsequent molecular analysis of amplified regions thus comprises a powerful method for the rapid identification and differentiation of potyviruses infecting sweet potato and is the most suitable method for viruses which are difficult to purify or which occur in mixed infections (Colinet *et al.*, 1994b). Specific primers for detecting and differentiating SPFMV (-CHH and -CH2), SPLV, SPMMV and other viruses have been designed from nucleotide sequences of these viruses (Colinet *et al.*, 1994b; 1998; Chavi *et al.*, 1997). Antigen detection can be considerably enhanced by coupling serological trapping of viruses with PCR, the so-called immunocapture-PCR (Mumford *et al.*, 1994; Rosa, 2001). This provides sensitivity, rapidness and the ability to assay many samples.

Hybridisation is performed by extracting DNA or RNA from the samples, base-denaturing in NaOH, and directly blotting onto nylon membranes (Colinet *et al.*, 1994b; Cohen *et al.*, 1997; Lotrakul *et al.*, 1998). Membranes are then fixed with heat or ultraviolet (UV) radiation and then hybridised with a labelled probe

consisting of cDNA sequence of a particular virus (Colinet *et al.*, 1994b; Cohen *et al.*, 1997). If the virus is present, the probe will hybridise with its DNA extracts from the sample and if not, the probe is lost during the washing process of the membranes (Lotrakul *et al.*, 1998; van der Merwe, 2001). Western and Southern blottings have been successfully used to detect sweet potato sunken vein virus (SPSVV), which is now called SPCSV and SPLCV-US (Hoyer *et al.*, 1996; Lotrakul *et al.*, 1998). Southern blotting uses the DNA extracts to hybridise with the complimentary DNA probe whereas Western blotting refers to the transfer of proteins to the membranes and detecting with the antibody probes (Memelink *et al.*, 1994). The use of riboprobes complementary to the RNA or DNA of the virus was found to be more sensitive than serological assays, labelled cDNA and immunobinding assays due to specificity and the removal of non-hybridised probe to minimise non-specific background (Abad *et al.*, 1992). Non-radioactive methods such as horseradish peroxidase (HRP), dioxigenin-anti-dioxigenin (DIG) and the biotin-streptavidin systems to label nucleic acids that are used as probes are also used to detect plant pathogens (Karcher, 1994). Their use is due to the advantage that the probes generated are stable and require shorter exposure time to detect hybridised material (Karcher, 1994).

2.5 Management of sweet potato viruses

The fact that sweet potato in Africa is perceived as a crop for the poor, mainly grown by women, has many implications for cultivation of the crop (Karyeija *et al.*, 1998a; Owour, 2000). Traditional cultivation practices such as piecemeal harvesting and exchanging planting material freely between neighbouring farmers provides for the spread and perpetuation of virus infected material (Karyeija *et al.*, 1998a). Farmers obtain planting materials from mature crops which are normally not virus-free and on which pesticides are rarely used (Karyeija *et al.*, 1998a; Owour, 2000). These practices favour the survival and spread of SPFMV and other viruses (Karyeija *et al.*, 1998a; Owour, 2000).

Managing viruses infecting sweet potato will require knowledge on aetiology and ecology of the viruses. Information on the control of viruses and how viruses infect plants is lacking among resource-poor farmers in South Africa (Jericho, 1999). Three main control practices are used by African farmers to limit the effects of SPFMV and these are (a) selection of SPVD-resistant cultivars, (b) use of disease-free planting material and (c) removing all infected plants (Clark and Moyer, 1988; Karyeija *et al.*, 1998a). Efforts to control the spread of sweet potato viruses by controlling the vectors have not been successful (Clark and Moyer, 1988). Both SPFMV and SPCSV and other sweet potato viruses can be controlled using virus-free material and controlling weeds, which may serve as alternative hosts of insects and viruses, especially wild *Ipomoea* spp. in and around fields (Joubert *et al.*, 1974; Bolton and du Plooy, 1984; Thompson and Mynhardt, 1986; Karyeija *et al.*, 1998a). Isolating new crops a distance from the old mature crops will reduce virus incidence and result in high yielding crops (Gibson and Aritua, 2002). The use of intercropping to reduce the numbers of infectious vectors attacking the sweet potato crop can help reduce SPVD incidence by delaying SPVD vectors onset and build-up (Ndunguru and Alyoce, 2000). A sweet potato/maize cropping pattern was found to have a lower SPVD incidence and it can be an option to reduce SPVD damage in the traditional sweet potato farming system (Ndunguru and Alyoce, 2000). If volunteer sweet potato plants, which may have survived from previous crops, are removed and resistant varieties planted, viral diseases can be minimised (Joubert *et al.*, 1974; Bolton and du Plooy, 1984; Thompson and Mynhardt, 1986; Karyeija *et al.*, 1998a).

The development of transgenic sweet potato plants can be another method of controlling sweet potato viruses (Owour, 2000). The use of cysteine proteinase inhibitor gene (oryzacystatin I) proved to make some sweet potato cultivars tolerant to SPFMV-RC (Cipriani *et al.*, 2000). Although the transgenic lines can still be affected by SPFMV-RC through grafting with SPFMV-RC infected *I. setosa*, it was proven that the multiplication rate of the virus is reduced and the virus cannot be detected directly by either visual observation or by NCM-ELISA (Cipriani *et al.*,

2000). Genetic engineering of sweet potato may be possible but its extended application will be limited by resources, multiplication of viruses and their strains, and virus complexes that may alter virus-plant interactions and result in disease development (Mukasa, 2001).

Meristem-tip culture is a method for eliminating viruses from sweet potato cultivars and is based on the discovery that virus concentrations are lower in plant apices (Chiu *et al.*, 1982; Moyer *et al.*, 1989). Together with thermotherapy whereby sweet potatoes are grown at 38-40°C for four to 12 weeks, can possibly give rise to virus-free plants (Terry, 1982). Due to its importance, sweet potato germplasm free from known viruses is needed for commercial production (Moyer and Salazar, 1989), and cultivation practices neglected by many small-scale farmers need to be taken into consideration to prevent further spread.

In South Africa, sweet potato varieties are cleaned from viruses through virus-elimination and a sweet potato plant improvement scheme, which was initiated in the early seventies (Joubert *et al.*, 1974; Laurie and Stork, 1997). The scheme involves maintenance of disease-free mother stock of the varieties in an insect-free greenhouse and obtaining disease-free plantlets, which are supplied to registered sweet potato vine growers and sweet potato producers (Laurie and Stork, 1997). The scheme takes place at ARC-Roodeplaat and it plays an important role in securing the profitability of sweet potato in South Africa (Laurie and Stork, 1997).

2.6 Conclusion

Due to the increase in population growth in sub-Saharan Africa where economic growth is slow and poverty being the biggest problem, root crops such as cassava and sweet potato have drawn much attention. Sweet potato, especially in parts of central, eastern and southern Africa is important as a seasonal source of food, food security and cash (Scott *et al.*, 2000). Some sweet potato varieties contain high carbohydrate and vitamin contents (Ewell and Mutuura, 1991; Owour, 2000), and will thus continue to provide an affordable diet to rural households in many parts of

the continent (Karyeija *et al.*, 1998a; Madibela *et al.*, 1999; Owour, 2000). Improving sweet potato production and utilisation is often considered as a means to improve incomes and food security among the poorer of the rural populations (Anonymous, 2002). Unfortunately the crop is very susceptible to viral diseases, especially SPVD, resulting in deterioration of the quality and yield of many sweet potato cultivars (Joubert *et al.*, 1974; Karyeija *et al.*, 2000; Owour, 2000). Even though many of the sweet potato viruses are insect transmitted, wild plants such as *Ipomoea* spp. and their related genera, which act as host plants, contribute to making virus control difficult (Cadena-Hinojosa and Campbell, 1981; Karyeija *et al.*, 1998a).

Biological transmission of viruses to healthy plants, using vectors and grafting onto indicator plants has proven to be the most successful and efficient method of detecting viruses (Cohen *et al.*, 1992; 1997; Karyeija *et al.*, 1998a). It can be used more efficiently in places where other viral detection techniques are limited or inaccessible. Biological assays of sweet potato viruses have specific limitations because of co-infection by SPFMV as well as restricted host range (Moyer and Salazar, 1989), low concentration, uneven distribution in test plants and possible inhibitors of virus inoculation by plant tissue extracts (Chavi *et al.*, 1997). Although grafting is convenient, it requires time, greenhouse space, labour and large quantities of *I. setosa* (Cadena-Hinojosa and Campbell, 1981; Esbenshade and Moyer, 1982; Karyeija *et al.*, 1998a).

Although ELISA techniques has also proven to provide satisfactory results in detection of sweet potato viruses, the sensitivity and specificity depend on the antibodies used (Clark and Bar-Joseph, 1984).

Because of its ability to rapidly identify and differentiate potyviruses infecting sweet potato, PCR has become a powerful tool in detecting sweet potato viruses (Colinet *et al.*, 1994b; 1998). But in Africa, neither riboprobes nor PCR-based methods were reported to be used to detect SPFMV due to the fact that facilities and

materials required are rare (Karyeija *et al.*, 1998a). Although there could be some financial constraints in other cases, all methods described above are useful in order to obtain satisfactory results.

Management of sweet potato viruses should be directed towards the analysis of the constraints that farmers encounter. Lack of knowledge results in farmers using the same planting material year after year (without any crop protection measures) and ignoring other important agronomical aspects such as weeding and plant protection. Supplying farmers with the necessary training can help minimise virus infections.

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