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Sweet Potato Diseases: Diagnosis and Management

Vinayaka Hegde* • **R. S. Misra, M. L. Jeeva**

Central Tuber Crops Research Institute, Thiruvananthapuram-695 017, India

Corresponding author: * **vhegde18@yahoo.com**

ABSTRACT

Yield of sweet potato cultivars have appeared to gradually decline over the years in most of the sweet potato growing areas. This decline in yield and quality may be caused by a combination of several factors, including mutation, viruses and other pathogens. Several pathogens are known to cause diseases in sweet potato. Among them, the diseases caused by viruses are of worldwide economic importance. However, fungi, bacteria, viruses, nematodes and phytoplasma are known to attack sweet potato. Pathogenic bacteria, although not very common, are responsible for important economic losses. They affect vascular tissue as well as storage and fibrous roots, thus causing vine wilting and rots. Fungal pathogens are classified according to the type of disease they cause, such as foliar, stem, storage root and post harvest diseases. Even though the specific management practices have not been developed for various sweet potato diseases since the crop is propagated through vine cuttings, most of the viral and fungal diseases could be avoided by selecting healthy planting materials and sanitation. In the present chapter, an attempt has been made to review the worldwide diseases of sweet potato and the available management practices. This would be highly useful to take precaution to avoid spread and loss and for identifying the occurrence of new diseases.

Keywords: fungal disease, PCR, serological methods, *Sweet potato feathery mottle virus*, viral and phytoplasma disease
Abbreviations: CLD, chlorotic leaf distortion; **CIP**, International Potato Centre; **CMV**, *Cucumber mosaic virus*; **CP gene**, coat protein gene; **CTCRI**, Central Tuber Crops Research Institute; **ICTV**, International Committee of Taxonomy for Virus; **IYVV**, *Ipomoea yellow vein virus*; **NCM-ELISA**, nitro cellulose membrane-enzyme linked immunosorbent assay; **SPCSV**, *Sweet potato chlorotic stunt virus*; **SPLCV**, *Sweet potato leaf curl virus*; **SPLV**, *Sweet potato latent virus*; **SPFMV**, *Sweet potato feathery mottle virus*; **SPVD**, sweet potato virus disease; **ssRNA**, single stranded RNA; **RT-PCR**, reverse transcription polymerase chain reaction

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INTRODUCTION

Sweet potato (*Ipomoea batatas* L.) is the third most important root crop after potato and cassava and is ranked seventh in global food crop production with an annual production of over 127 million tonnes (Kays 2005). It is generally regarded as a food security crop which can withstand adverse climatic or soil conditions. Though sweet potato originated

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Invited Review

in Central/South America, the world production is centred in Asia, with China as the major producer with 109 million tonnes that counts for over 86% of the sweet potato world production. More than 95% of the global sweet potato crop is grown in the developing countries and provides sustainable food supply when other crops fail. In India, sweet potato is grown in 0.12 million ha, with an annual production of 1.12 million tonnes resulting in an average productivity of 9 t ha^{-1} (FAO 2011). Due to its nutritional qualities (rich in carbohydrates, dietary fiber, beta carotene, vitamin C, and vitamin B6), sweet potato is considered as a crop with great potential, not only for human consumption but also for animal feeding and industrial use. Sweet potato is the crop that produces highest biomass per unit area. Yields of sweet potato cultivars have appeared to gradually decline over the years in most of the sweet potato growing areas. This decline in yield and quality may be caused by a combination of several factors, including mutation, viruses and other pathogens (Clark *et al*. 2002, 2003). The major biotic problems in sweet potato cultivation worldwide are sweet potato weevil and viral diseases. However, fungi, bacteria, viruses, nematodes and phytoplasma are known to attack sweet potato (Clark and Moyer 1988). Pathogenic bacteria, although not very common, are responsible for important economic losses. They affect vascular tissue as well as storage and fibrous roots, thus causing vine wilting and rots. Fungal pathogens are classified according to the type of disease they cause, such as foliar, stem, storage root and post harvest diseases. In general, foliar and stem diseases are mild and cause little damage, except for scab, which is a very important disease in Southeast Asia. These diseases contribute to lower yields by reducing photosynthetic area and transport of nutrients and products to the storage roots. In some countries, storage rots do not cause much damage because sweet potatoes are consumed shortly after harvest. Tuber rot pathogens, however, are present in the field and can cause significant losses. Plant parasitic nematodes are included as the cause of serious damage to storage roots both in the field and during storage. Of all the sweet potato pathogens, viruses appear to contribute the most to yield losses (Hahn 1979).

In India, sweet potato is grown in almost all states; however, the major growing areas are in the states of Orissa, Bihar, Uttar Pradesh, West Bengal and Jharkhand. It is cultivated mainly as a rainfed crop in these states and accounts for 82% of sweet potato produced in India. The productivity of sweet potato in India is very low (9 t ha^{-1}) compared to many other Asian countries like China, Japan, Israel, Philippines, etc. One of the major reasons of low yield is the incidence of diseases and pests. Sweet potato weevil is the major constraint and no resistant varieties are available for the same. Next to weevil, the viral diseases are the major problems (Edison *et al*. 2009). Though the sweet potato virus disease (SPVD) complex reported in many African countries is not yet seen in India, the wide prevalence of *Sweet potato feathery mottle virus* (SPFMV) is reported. Sweet potato leaf curl caused by a geminivirus is also emerging. Due to no systematic, virus free planting materials production programme, accumulation of viruses has increased and resulted in cultivar decline.

Sweet potato diseases are reviewed by several workers in the recent years (Valverede *et al*. 2007; Clark *et al*. 2009; Loebenstein *et al*. 2009; Wang *et al*. 2010; Clark *et al*. 2012; Loebenstein 2012). In the present review, the diseases of sweet potato and the possible strategies for management are discussed.

FUNGAL DISEASES OF SWEET POTATO

Sweet potato is affected by many fungal diseases and more than 40 pathogens have been reported to attack sweet potato both in field and storage in different sweet potato growing areas all over the world. Among them, leaf spots caused by *Cercospora*, *Alternaria* and *Helminthosporium*, stem rots, white rust and storage rots are the major diseases (Thankappan 1994).

Cercospora leaf spot

The disease was first reported from Bihar in 1976 (Sihna and Mehta 1982) and a yield loss of 15-20% has been estimated (Sinha and Yadav 1977). The disease is characterized by yellowish brown spots in the beginning and gradually turning to deep brown, with circular ovoid or irregular margin. These spots coalesce and form larger patches covering most portion of the leaf. Shot hole and defoliation usually can be seen in severe cases under favourable conditions. These symptoms can be found on leaves of different ages throughout the plant, although more spots are found on older leaves. The disease is dispersed by wind or splashing rain. It is most prevalent in the hot and humid tropics and is seldom observed during dry season. The primary host of *C. bataticola* is sweet potato. Although no other hosts have been reported, it is suggested that this pathogen can overwinter on some weed species. The peculiar appearance of the spots with very sharp differences between the centre and the border is one way to recognize the disease in the field. Another way of identifying the disease is to observe the fruiting structures under a compound microscope. For this, it is advisable to collect the samples from the field, early in the morning and to scrape the surface of the spot to observe the conidiophores and conidia.

Two species of *Cercospor*a namely *C. batatae* and and *Cercospora ipomoeae* have been reported to cause leaf spot disease on sweet potato (Salam and Rao 1957; Sinha and Mehta 1982). *Cercospora batatae* differs from *C. ipomoeae* in having short conidiophores and fruiting mostly hypophyllus conidia, linear, tapering above, flexuous 3-4 septate, hyaline 50 × 10 µm in size. Conidiophores of *C. ipomoeae* generally emerge through stomata in fascicles, straight or curved apex, smooth, olivaceous brown in colour and multiseptate. Cultural requirements for pathogens have been studied (Mehta and Sinha 1983).

There are no reports on the epidemiology or management of this disease using fungicides. Control is not usually needed. However, it is suggested that only healthy material should be used for planting. Differences in susceptibility have been found among sweet potato accessions. During late 1980's, some of the sweet potato lines have been identified having field resistance to *Cercospora* leaf spot (Thankappan 1990).

Alternaria leaf spot/stem blight

Leaf spots caused by several species of *Alternaria* may affect sweet potato time to time in many parts of the world (Lenne 1991). The most common are *A. alternata*, *A. brassicae* and *A. solani.* There are some differences among these three species but they all have the same shape and colour of conidia. They all produce ellipsoidal or oblong conidia with transverse and longitudinal septae with *A. solani* and *A. porri* having a long peak while *A. alternata* has a short one. In this last species, conidia are somewhat short, with fewer septae and are formed in chains of five or more conidia. In most cases the disease develops on older leaves to a limited extent and do not affect production. In India, a severe premature defoliation due to *Alternaria* leaf spot caused by *A. capsici* has been reported (Sivaprakasam *et al*. 1977). The leaf spots of gray to black in appearance are found on mature and old leaves. They have concentric rings and well defined margins. Several lesions can fuse and cover a great area of the leaf. When this occurs, the leaf drops. Usually spots are surrounded by a chlorotic halo. As the spots become old, infected tissue may crack and fall off.

A similar type of disease causing severe stem and petiole blight by *A. bataticola* has been reported from many African countries like Kenya, Ethiopia and Uganda during 1984 (Bruggen 1984). Lopes and Boiteux (1994) reported the occurrence of severe leaf blight caused by *A. bataticola* from Brazil. Stem and petiole bilght has also been reported

from Papua New Guinea (Lenne 1991; Clark *et al*. 2009). The disease caused by *A. bataticola* is reported as more aggressive because it can attack whole vine (leaves, petioles and stems) at an early stage and through all the crop cycle. Girdling of stem due to severe necrotic spots may cause wilt or dieback of the terminal portion of the vine.

The fungus remains in plant debris on the soil as mycelium and conidia. The air borne conidia are spread through infected planting material by wind, splashing rain and water. High relative humidity or free water is necessary for infection and sporulation, conditions common in tropical regions due to continuous rain. The conidia germinate and directly enter the leaves through the epidermis of old leaves, killing the tissue in advance, probably by the toxins such as alternaric acid produced by the mycelia. During dry conditions the lesions get a silvery tone.

It has been suggested that red skinned varieties of sweet potato are more resistant than white skinned varieties for stem and petiole blight. Reaction of 648 sweet potato lines to *Alternaria* leaf spot is available, though none of these lines were resistant, 36 lines were comparatively less susceptible to *Alternaria* leaf spot (Sivaprakasam *et al*. 1980). Manage the disease by use of clean planting material, destroying and burning of crop residues after harvest and avoiding overhead irrigation.

Helminthosporium leaf spot

The disease is reported from India during 1977 (Mishra and Singh 1977). No information is available on the intensity, spread or damage due to the disease. The symptoms appear as small pale yellow, water soaked lesions on leaves which later develop into brown necrotic irregular spots surrounded by yellow halo. The matured spots are darker in colour and show a blighted appearance scattered over the leaves, petioles and vines. In severe cases, the entire leaves dry up and the affected area in the field can be noticed from a distance.

The pathogen involved has been identified as *Helminthosporium euphorbiae* Hans. The conidiophores are light brown or dark olivaceous in colour, simple or branched, arising mostly single, erect or slightly curved. Conidia are dark and olivaceous in colour. Except Mishra and Singh (1977), no other report on occurrence of *Helminthosporium* leaf spot in sweet potato has so far been reported. No information is currently available about the fungicidal control of this disease in sweet potato. There is no information or further reports about the occurrence of this disease on sweet potato.

White rust

White rust is a common disease distributed widely around the world on sweet potato and related species of family Convolvulaceae. It is a minor disease of sweet potato and there is no record about the importance of this disease on yield. A few studies that have investigated the effect on yield suggest that white rust does not affect yield of sweet potato storage roots (Clark *et al*. 2009).

The disease is caused by *Albugo ipomoea-panduratae* (Schw.) Swingle (Mukerji and Chrichet 1975). The disease was first reported in India during 1937 (Mitter and Tandon 1937). Except reports on host range of the pathogen by Bilgrami *et al*. (1979), no further information on the epidemicology or control of this disease is available. The most obvious symptom is the presence of chlorotic or yellowish blotches, initially roundish to angular where they are limited by veins, on the upper surface of leaves. On the lower surface, small pustules develop which later open and expose whitish masses of sporangial pustules. After sporulation, the infected tissue dies, forming irregular shaped brown lesions. In some cultivars or growing conditions, infection induces the development of galls or blisters of raised, thickened tissue. Galls may develop on leaves, petioles, stems and flowers. When pustules erupt, the galls become covered in the white spores. Diseased plants can also present general distortion, defoliation and flower abortion. In some very sensitive cultivars, symptoms resembling witches' broom, with shortening of internodes and bunchy growth habit, have been observed. The pathogen surviving in the form of oospores on crop refuge act as primary source of inoculam and air-borne sporangia acts as secondary source of inoculum during growing season. Specific control measures are considered not necessary for this disease, however in extreme cases copper fungicides recommended for other oomyceteous pathogens may be used.

Collar rot

Collar rot caused by *Sclerotium rolfsii* is an economically important disease of sweet potato causing severe damage in seed beds. The disease is known by different names like collar rot, stem rot, *Sclerotium* blight, *Sclerotium* wilt, bed rot, cottony rot, foot rot etc. known by several names. The infection on storage roots is named as separate disease as circular spots. In the field, the disease is present at any stage of growth, but mostly scattered in patches affecting several plants. Affected plants are girdled and usually die. Losses are 5-20% in tropical areas due to dead plants. The fungus attacks many hosts and is present worldwide. However, there were only few reports on sweet potato (Clark *et al*. 2009). In India, the disease was first reported from Tamil Nadu by Sivaprakasam and Kandaswamy (1983) and later observed from other sweet potato growing regions also (Thankappan 1994).

The symptoms of the disease are sudden wilting of the sprouts followed by rotting and death of the plants in patches. Plants initially show yellowing of the lower leaves and wilting. In the stems, depressed, water soaked, sunken lesions are usually observed at or below the soil surface (collar region), and they soon become covered with a white fan like mycelium that radiates from the lesion. Soon after, the stem is girdled and the plant eventually dies. The fungus continues to grow in dead plants in the field and when they start drying, numerous sclerotia are formed. They are initially white but become light brown, having the appearance of mustard seeds.

The pathogen is soil borne and can survive in soil for several years. Infected seed tubers also act as primary source of inoculum. Warm and humid weather favours the development of the pathogen and disease. The decaying seed roots in the beds or decaying leaves on the surface of the beds may stimulate disease development. It is present in tropical and subtropical areas of the world where temperature and relative humidity are high most of the time. The fungus is omnivorous and is recorded to live on more than 500 plant species.

An integrated approach involving cultural practices is essential for effective management of the disease. Selection of site which did not have sweet potato or other cops in which *S. rolfsii* was not a problem earlier for at least three years should be selected for production of sweet potato. Removal of crop debris and strict field sanitation is essential which otherwise would help the pathogen to multiply in the field. Drenching of soil around the plants with vitavax or plantvax (50 ppm) is also effective in controlling the disease. Application of biocontrol agents like *Trichoderma viride* to soil also help in control of the disease.

Fusarium wilt or stem rot

Fusarium wilt was once an important disease of sweet potato in many sweet potato growing countries. In India, severe wilt of sweet potato was noticed in 1974 (Sinha and Misra 1974). According to them about 30-40% of the crop was destroyed.

The symptoms first appear as yellowing of older leaves and transient wilting of the vines. Later on vines will be permanently wilted and die. Infected vascular elements become brown and in later stages the entire stem may rot. In some cases infected plant may survive and produce sto-

rage roots that are infected. If such tubers are used as seed tubers, they can transmit the fungus to the fresh sprouts which may wilt in plant beds. The disease is caused by *Fusarium oxysporium f. sp. batatas* (Holliday 1970). The fungus produces a white aerial mycelium and purple pigment characteristic of the species. Erect, hyaline conidiophores are formed successively producing conidia which accumulate into groups. It produces microconidia, macroconidia and chlamydospores forming bud cells in liquid medium (Brayford 1992).

The optimum temperature for infection is around 30°C, but the disease can develop at lower temperature and across a wide range of soil moisture from 28-75%. However, maximum damage occurs in fields where the moisture is low. The fungus is soil borne and can persist in the soil for many years. Infection is usually through vascular wounds such as those obtained when collecting cuttings for planting or when leaves are detached from the stems. The disease can affect vines at any stage of development, but when infected transplants (from mother roots) are used for propagation, plants die at an early stage. Once the soil has been infected, the infection persists in the plant refuse, because the fungus produces resistant structures known as chlamydospores that can survive in the soil for several years. Though the primary host is sweet potato, the fungus also attacks several *Ipomoea* species, and a number of other members of the family Convolvulaceae. *F. oxysporum* f.sp. *nicotianae*, the causal agent of *Fusarium* wilt in tobacco was found to cause wilt in sweet potato and conversely, *F. oxysporum* f. sp. *batatas* cause wilt in susceptible tobacco (Clark *et al*. 1998). The disease could be detected easily by taking transverse section of stem just above the soil surface. Brown to purple discolouration of the vascular system will confirm the presence of *F. oxysporum* f.sp. *batatas* as the disease develops in the vessels of stems and roots.

Use of resistant variety is the best option for controlling the disease. Sweet potato varieties resistant to stem rot/wilt have been identified in many countries (Clark 1988). Sinha and Mishra (1974) identified 16 sweet potato lines resistant to *F. oxysporum* f. sp. *batatas* in India. Seed tubers from wilt affected field should not be used for raising the new crop. If cuttings would be obtained from sprouts, cut them 5 cm above the soil surface. Crop rotation with non-host crops can reduce the pathogen population in soil. Dipping of seed tubers and vines in fungicides like carbendazim, benomyl or thiabendazole is very effective in reducing the disease incidence (Nielsen 1977).

Chlorotic leaf distortion

The chlorotic leaf distortion (CLD) of sweet potato is caused by *Fusarium denticulatum* (Nirenberg and O'Donnell 1998) which was originally identified as *F. lateritium* (Clark *et al*. 1990; Nelson *et al*. 1995). The disease has no effect on yield of storage roots regardless of the severity of the disease (Kim *et al*. 1996). Research need to be done on the potential effect of CLD on quality of vines for use as food or feed. The disease has been reported from USA, Brazil, Kenya, Peru and Venezuela (Gonzalez *et al*. 2003; Clark *et al*. 2009). However, the disease may be widely distributed in other sweet potato growing areas and it is likely that it has not yet been reported since there is no loss of tuber yield due to the disease. The causal organism has been isolated from botanical seed produced in many different countries (Clark and Hoy 1994).

Symptoms of the CLD are most pronounced exclusively at the growing tip of the vine. Leaves nearest the vine tip (1-2 youngest leaves) develop a bright general chlorosis and are often twisted or distorted. White waxes like substances (mycelia and conidia of the pathogen) are found on the upper surface of the young leaves that have just unfolded. As the leaves age, they regain normal green colour with only diffused chlorosis, however the newly emerged leaves may continue to show chlorosis. Symptoms are masked during cloudy weather, whereas warm humid weather with bright sunshine favours the development of symptoms.

The fungus primarily colonizes the surface of the growing vine tip without invading the plant (Clark 1992). Mycelia are found on apical meristems and between halves of developing leaves that have not yet opened. Once the leaves open and expose the fungal mycelia, the mycelia appear to stop growing. As a result, individual leaves appear to recover as they mature. Control measures are considered not necessary, since CLD has no effect on tuber yield.

BACTERIAL DISEASES

Bacterial diseases of sweet potato are reported from a few countries like USA, Japan and China (Clark *et al*. 1998; Ooshiro *et al*. 2006; Zhang *et al*. 2009). Root rot or stem rot caused by *Dickeya dadantii*, wilt caused by *Ralstonia solanaceraum* and soil rot caused by *Streptomyces ipomoeae* are the three bacterial diseases reported on sweet potato. Though the diseases are severe wherever they occur, their geographic distribution is limited. The bacterial wilt of sweet potato is confined to China whereas stem rot and root rot are mainly reported from USA.

Bacterial stem and root rot caused by *Dickeya dadantii* can be economically important because it destroys plants in the field and tubers after harvest. The pathogen was earlier identified as *Erwinia chrysanthemi,* which is reclassified into six new *Dickeya* species (Samson *et al*. 2005). The bacteria attacks several hosts in different regions of the world, however the disease on sweet potato has only been reported from USA (Martin and Dkes 1977; Clark *et al*. 2009).

This disease is more common in storage but may also affect plants in the field and in seed beds. The first symptom is the partial wilting of the plant; one or two branches may wilt, and eventually the entire plant may collapse and die. Discolouration of tissues inside the stem may also occur under some conditions. Water soaked sunken, brown to black lesions are observed at the base of stems and on petioles. On storage tubers small, sunken, brown lesions with black margins can be observed on the surface, but more frequently the rotting is internal with no evidence of external symptoms and the affected tissue becomes watery. The disease is more common in storage than in the field. The rotted tubers emit characteristic foul smell due to invasion of secondary microorganism.

The pathogen lives in plant debris and in the roots as well as on soil surrounding the roots of sweet potato and its other hosts. Dissemination is through infected planting material, irrigation water, tools, animal grazing, shoes of labourers etc. The vines and tubers may have latent infection of bacterial pathogen and later develop into active infection under favourable environment (Duarte and Clark 1992).

Wounding during any stage of crop production should be minimized. Disease free planting materials should be used and the cuttings obtained from the upper most portion or tips of vines are essential to avoid bacterial inoculums on the base of the stem. Cultivars varying in their susceptibility are identified and selection of resistant and less susceptible cultivars will also be useful in avoiding loss due to the disease.

VIRUS AND PHYTOPLASMA DISEASES

Several viruses on sweet potato were reported worldwide (Clark and Moyer 1988; Valverde 2007; Lozano *et al*. 2009), but only a few have been well studied and characterized (Salazar and Fuentes 2001; Loebenstein *et al*. 2009). The first report on viral infection in sweet potato was made by Hansford (1944) and later on by Stainbaeur and Kushman (1971). Worldwide, up to 20 different viruses have been described to infect sweet potato (Loebenstein *et al.* 2004; Valverde *et al*. 2007) and 12 of them are currently recognized by the International Committee of Taxonomy for Virus (ICTV). Viruses belonging to both RNA and DNA

Table 1 Viruses reported on sweet potato.

Virus	Family/Genus	Vector
Cucumber mosaic virus (CMV)	Bromoviridae / *Cucumovirus*	Aphids
Ipomoea yellow vein virus (IYVV)	Geminiviridae / *Begomovirus*	Whiteflies
Sweet potato chlorotic stunt virus (SPCSV)	Closteroviridae / *Crinivirus*	Whiteflies
Sweet potato feathery mottle virus (SPFMV)	Potyviridae / *Potyvirus*	Aphids
Sweet potato latent virus (SwPLV)	Potyviridae / *Potyvirus*	Aphids
Sweet potato virus G (SPVG)	Potyviridae / *Potyvirus*	Aphids
Sweet potato leaf curl virus (SPLCV)	Geminiviridae / *Begomovirus*	Whiteflies
Sweet potato leaf curl Georgia virus (SPLCGV)	Geminiviridae / *Begomovirus*	Whiteflies
Sweet potato leaf speckling virus (SPLSV)	Luteoviridae / *Enamovirus*	Aphids
Sweet potato mild mottle virus (SPMMV)	Potyviridae / *Ipomovirus*	?
Sweet potato mild speckling virus (SPMSV)	Potyviridae / *Potyvirus*	Aphids
Tomato spotted wilt virus (TSWV)	Bunyaviridae / *Tospovirus*	Thrips?
Tentative species	**Family / Putative genus**	
Sweet potato C6 virus	?	?
Sweet potato caulimolike virus	Caulimoviridae	?
Sweet potato chlorotic fleck virus (SPCFV)	Flexiviridae / *Carlavirus*	?
Ipomoea crinkle leaf curl virus (ICLCV)	Geminiviridae / *Begomovirus*	?
Sweet potato ring spot virus	Comoviridae / *Nepovirus*	?
Sweet potato vein mosaic virus	Potyviridae	Aphids
Sweet potato virus 2 (SPV2)	Potyviridae / *Potyvirus*	Aphids?
Sweet potato yellow dwarf virus (SPYDV)	Potyviridae / *Ipomovirus*	?

groups are known to infect sweet potato (**Table 1**). Mixed infection of viruses is known to cause more damage than the individual virus infection. Many viruses when present singly may not express visible symptoms, however, the symptom will be more severe when viruses occur in mixed infections.

Sweet potato is a vegetatively propagated crop, accumulation and perpetuation of viruses is a major potential constraint for crop production. Use of virus infected vines or tubers for propagation is the major reason for low yield of sweet potato in many parts of the world. Virus diseases often cause reduction in yield and quality of storage roots (Clark and Moyer 1988; Loebenstein *et al*. 2004). Studies have demonstrated yield losses up to 30-50% in farmers' fields in the US (Clark and Hoy 2006), but losses of 80-90% have also been recorded in areas affected by virus complexes that include *Sweet potato chlorotic stunt virus* (SPCSV, genus *Crinivirus*) and potyviruses (Mukasa *et al*. 2006). In India, occurrence of sweet potato virus disease syndrome was reported in 1990 (Thankappan and Nair 1990). Later on Kumar et *al*. (1991) reported the occurrence of *Sweet potato feathery mottle virus* (SPFMV) in sweet potato germplasm. Though SPFMV is the major virus prevalent in India, presence of another 3 viruses namely *Sweet potato chlorotic fleck virus*, *Sweet potato latent virus*, and *Sweet potato mild mottle virus* in sweet potato germplasm collection was reported based on detection using International Potato Centre's (CIP) NCM-ELISA kit (Makeshkumar *et al*. 2001). Occurrence of leaf curl disease has also been reported in recent years (Makeshkumar *et al*. 2007). Mixed infection of SPFMV and *Sweet potato leaf curl Georgia virus* (SPLCGV) on sweet potato was also recorded though synergetic effect on yield is not known (Prasanth and Hegde 2008). The survey carried out in India indicated about 10-80% viral disease incidence depending upon age of the crop, location and varieties (Makeshkumar *et al*. 2001).

Sweet potato feathery mottle virus

Sweet potato feathery mottle virus (SPFMV, genus Potyvirus, family Potyviridae) is the most common sweet potato virus worldwide. The virus has many synonyms viz. *Sweet potato chlorotic leaf spot virus*, *Sweet potato internal cork virus*, *Sweet potato russet crack virus*, *Sweet potato vein mosaic virus*, *Sweet potato virus A*, *Sweet potato vein clearing virus* and *Sweet potato ring spot virus*. SPFMV is responsible for considerable yield reduction in sweet potato crops worldwide.

Some isolates of SPFMV cause economic losses by their effect on storage root quality by causing internal cork and russet crack. The SPFMV was first described in 1945 in the United States (Clark *et al*. 2002) and later on from various parts of the world (Arrendel and Collins 1986; Clark and Moyer 1988; Cedano *et al.* 1989; Ngeve and Boukamp 1991; Carey *et al.* 1999; Loebenstein *et al*. 2003; Prasanth and Hegde 2008).

Several types of symptom expression by SPFMV in sweet potato have been reported from different parts of the world which includes ring spot, feathering, chlorotic specks, leaf curl or leaf roll, yellow netting, mosaic and Witches' broom (Clark and Moyer 1988; Gibson *et al.* 1998; Makeshkumar *et al*. 2001; Hegde *et al*. 2007b). Most of these symptoms were best observed during the early growth period and they were generally mild and transient which are similar to the symptoms described by earlier workers (Thankappan and Nair 1990). The most distinctive symptom of the virus, irrespective of strain, present is the chlorotic feathering of the leaf midrib and, in some genotypes, the expression of chlorotic spots with purple rings (Moyer and Salazar 1989; Makeshkumar *et al*. 2001a, 2001b; Jeeva *et al*. 2004) (**Fig. 1A-E**).

Symptom visibility on foliage is influenced by cultivar susceptibility, degree of stress, growth stage, and strain virulence. Increased stress can lead to symptom expression, whereas rapid growth may result in symptom remission. Symptoms on storage roots depend on the strain of SPFMV and the sweet potato variety. The common strain causes no symptom on storage roots of any variety, but the 'russet crack' and 'internal cork' strains cause external and internal dark necrotic lesions respectively on certain varieties.

The SPFMV virions are filamentous; not enveloped; usually flexuous with the length ranging from 810 to 865 nm. The genome consists of a positive sense, single stranded linear RNA (ss RNA) of about 10.8 kb with a poly (A) region at the 3′ end (Sakai *et al*. 1997). The genome is larger than the average (9.7 kb) of a potyvirus genome (Shukla *et al*. 1994). The SPFMV coat protein is exceptionally large (38 kDa) as compared to other potyviruses (Abad *et al*. 1992). Like other potyviruses, the genome contains a single ORF, flanked by a UTR at both the 5′-end 3′-ends and encodes a large polyprotein *ca*. (3490 aa) that is processed to mature proteins by virus encoded proteases: P1, HC-Pro and NIa-Pro (Reichmann *et al*. 1992). The P1 and HC-Pro catalyse their own cleavage from the polyprotein (Carrington *et al*. 1989) while NIa-Pro is responsible for the cleavage of the C-terminal two-thirds of the polyprotein (Dougherty and Carrington 1998).

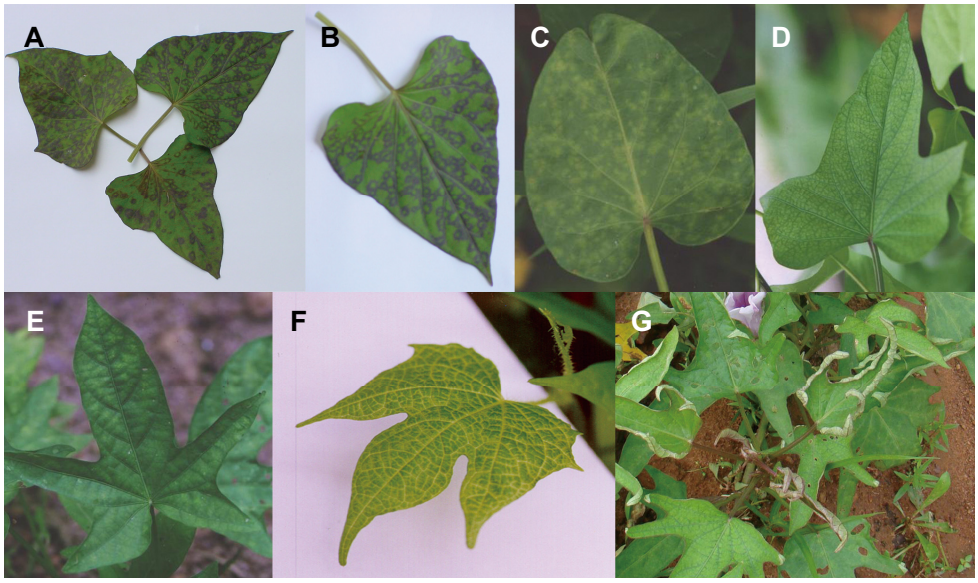

Fig. 1 Virus-associated symptoms in sweet potato observed in India. (**A**) Feathering; (**B**) ring spots; (**C**) chlorotic spots; (**D**) mottling symptoms; (**E**) yellow specks and serrated leaf margins. (**F**) *Ipomoea setosa* graft inoculated with SPFMV-infected sweet potato showing typical vein clearing symptom. (**G**) Upward leaf curl caused *by Sweet potato leaf curl virus*.

The SPFMV is sap transmissible when diluted 10-fold in 0.05M potassium phosphate buffer (pH 7.2) as well as graft transmissible from one host to another. The virus enters the host cell via a stylet of several aphid species (e.g. *Aphis gossypii*, *A. craccivora*, *Myzus persicae*) in a non-persistent manner. The transmission by aphids depends on the HC-Pro and an N-terminal amino acid motif Asp-Ala-Gly in the CP (DAG; Atreya *et al*. 1992). Their host range is narrow, limited to plants of the family Convolvulaceae (genus *Ipomoea*). Some strains have been reported to infect *Nicotiana benthamiana* and *Chenopodium amaranticolor* (Campbell *et al*. 1974; Moyer *et al*. 1980). The virus is not transmitted by seed, pollen or by contact between plants.

As other potyviruses, traditional criteria to discriminate between species and isolates are predominantly based on serology and biological criteria such as host range, cross-protection and symptomatology (Shukla *et al*. 1994). The SPFMV can routinely be diagnosed by grafting on a sensitive indicator host *Ipomoea setosa*, causing vein clearing (**Fig. 1F**) followed by remission or on *I. nil*, inducing systemic vein clearing, vein banding and ring spots (Green *et al*. 1988). The SPFMV can be diagnosed by ELISA and antisera are commercially available and NCM-ELISA kit has been developed by International Potato Centre, Peru to detect many of the common sweet potato viruses (Carey *et al*. 1999). Occurrence of SPFMV in Indian sweet potato cultivars was detected using antisera supplied by International Potato Centre (Makeshkumar *et al*. 2001). However, ELISA reliably detects SPFMV only in leaves with symptoms. The IPGRI has also recommended indexing the sweet potato by grafting on *I. setosa* for quarantine purpose (Moyer *et al*. 1989). Reliable detection and identification of SPFMV by RT-PCR and by subsequent cloning and sequencing of amplified product has also been reported by many workers. SPFMV has also been identified by RT-PCR utilizing degenerate genus specific primers, designed to amplify the variable 5′ terminal region of the potyvirus coat protein gene (Colinet *et al*. 1998). Nucleic acid spot hybridization using riboprobe which is highly sensitive and can detect up to 0.128 pg of RNA has also been developed (Abad and Moyer 1992). In India the detection of SPFMV using non radioactive biotinylated probe has been developed (Hegde *et al*. 2007b) using the CP gene of the SPFMV Trivandrum isolate.

The SPFMV can be purified from infected *I. nil* (Cali and Moyer 1981) or directly from infected sweet potato co-infected with SPCSV (Cohen *et al*. 1988). In India attempt was made to purify the virus directly from SPFMV affected sweet potato (Jeeva *et al*. 2004) and polyclonal antiserum was also produced. However, the antiserum titre was not satisfactory. Recently at CTCRI, India, recombinant polyclonal antiserum of SPFMV was produced using coat protein gene expressed in expression vector. The expressed protein was purified and injected into rabbit and the antiserum produced could able to detect SPFMV in sweet potato by DAC-ELISA. The antiserum at 1: 1000 dilutions could be used for detection of SPFMV. The SPFMV could also be successfully detected through Dot immunobinding assay or NCM-ELISA using the antiserum produced against the coat protein of SPFMV (Hegde *et al*. 2007a; Ganga 2009).

Based on serological differences and the distinct symptoms induced in sweet potato, isolates of SPFMV are divided into two strains: russet crack (RC), named after the characteristic symptoms it causes on the storage roots of susceptible cultivars, and the common (C) strain (Kennedy and Moyer 1982). Later, in Japan, strains O (ordinary) and S (severe) were distinguished based on their differential symptoms caused in sweet potato (Usugi *et al*. 1994). Since then, phylogenetic analysis of the coat protein (CP) sequences have shown that SPFMV isolates can be subdivided into four genetic strain groups: C, RC, O and EA (East Africa) (Kreuze *et al*. 2000). Strains RC, O and EA are closely related to each other but phylogenetically distant from C (Tairo *et al*. 2005), with CP sequence identity values around the species discrimination threshold recommended for potyviruses (Adams *et al*. 2005). These findings are concordant with biological and serological data that indicate that strain C is distantly related to other SPFMV strains (Mukasa *et al*. 2003; Tairo *et al*. 2006). On this basis, it has been proposed that isolates of strain group C should be regarded as belonging to a distinct potyvirus species whose members infect sweet potato (Tairo *et al*. 2005).

Though SPFMV alone generally causes only minor damage to sweet potato cultivars, the RC strain is associated with russet cracking of the tuberous roots in certain cultivars and has been reported from China (Chen *et al*. 2001), Japan (Sakai *et al*. 1997), Egypt (IsHak *et al*. 2003), Korea (Ryu *et al*. 1998) and the USA (Cali and Moyer 1981; Abad *et al*. 1992). Isolates of strain C deviate from RC by 82% aa and have been reported from Argentina, China and the USA (Abad *et al*. 1992; Colinet *et al*. 1998). The sequencing and phylogenetic analysis of complete coat protein gene of four SPFMV isolates in India showed the occurrence of SFPMV stains belonging to RC and EA strain groups (Ganga 2009). However, tuber crack symptoms were

not observed in any of the Indian cultivars or germplasm.

Though SPFMV alone generally causes only minor damage to sweet potato, control is essential as in combination with other viruses its effect on plant growth and yields may become substantial. Use of meristem derived virus free (indexed) sweet potato has shown 20-30% increase in tuber yield in countries like China and Israel (Fuglie *et al*. 1999; Lobenstein *et al*. 2003). Development of virus resistant cultivars has been the most effective means of reducing sweet potato losses due to virus infection (Hahn *et al*. 1981). Some sweet potato germplasm and many African sweet potato land races have resistance to SPFMV (Carey *et al*. 1997). Breeding of SPFMV resistant plants was initiated by International Potato Centre (CIP), Peru (Mihovilovich *et al*. 2000). Two CIP clones (420020 and 420026) have extreme resistance to SPFM-C (Fuentes and Salazar 1996). However, several clones resistant to SPFMV in CIP's test were found to be susceptible when exposed to Ugandan (Karyeija *et al*. 2000) and Israeli isolates (Lobenstein *et al*. 2009). Apparently strain diversity requires that breeding and selection have to be done in various locations. Transgenic sweet potato has also been developed with coat protein gene of SPFMV (Odame *et al*. 2001; Okada *et al*. 2001). However, these transgenic lines were not found resistant to the complex infection of SPCSV causing sweet potato virus disease (Wambugu 2004). Transgenic sweet potato plants were also developed with a rice cysteine inhibitor gene, which interfere with virus replication by inhibiting proteiolysis of viral polyprotein. However, these transgenic plants still contained some virus (Cipriani *et al*. 2001).

Sweet potato mild mottle virus (SPMMV)

The *Sweet potato mild mottle virus* (SPMMV), the second most predominant one, belongs to the genus Ipomovirus of the family *Potyviridae*, is transmitted semi-persistently by whitefly *Bemisia tabacai* (Hollings *et al*. 1976). The genome consists of single stranded RNA with a filamentous particle of 950 nm length. Symptoms associated with SPMMV are mottling, stunting and vein clearing.

The virus has a wide host range and has been successfully transmitted to 14 plant families (Hollings *et al.* 1976). It induces leaf mottling, vein chlorosis, dwarfing and poor growth on sweet potato plants. Morphologically and in size, the virion is similar to potyviruses. Cytoplasmic inclusions are also induced in SPMMV infected cells (Moyer and Salazar 1989). The difference between SPMMV and potyviruses is the DAG tripeptide that is associated with aphid-transmissibility of potyviruses (Atreya *et al.* 1992) but is missing from SPMMV (Mukasa *et al*. 2003). Serologically, SPMMV has no relationship with potyviruses. In spite of a wide genetic variability (82-100% aa) in the sequence of the CP-encoding region of SPMMV isolates from East Africa (Mukasa *et al.* 2003), diagnosis is fairly reliable using serology with available antibodies. Little is known about genetic diversity of this virus from outside EA though there have been reports on its occurrence in different countries outside Africa.

Sweet potato chlorotic stunt virus (SPCSV)

Symptoms caused by *Sweet potato chlorotic stunt virus* (SPCSV) on sweet potato are generally mild and the general symptoms are mild stunt, chlorotic and purpling of leaves (Gibson and Aritua 2002). Symptoms vary considerably depending on the host. In some cultivars, the virus can be symptomless, whereas in others symptoms such as mild vein yellowing, sunken secondary veins on abaxial leaf surfaces and swollen veins on abaxial leaf surfaces are observed. Very severe symptoms associated with SPVD such as stunting, leaf distortion, crinkling and blistering are observed in sweet potato plants when the virus co-infects with SPFMV. Symptoms on *Ipomoea nil*, a plant species used as an indicator plant as well as a propagation host for the virus, include chlorosis and epinasty in younger leaves followed by a general stunting and dwarfing of the mature plant (Salazar and Fuentes 2001). *Ipomoea setosa* plants exhibit symptoms such as stunting, smaller brittle leaves and occasional inward leaf rolling. The SPCSV is present in Asia (Taiwan and China), South America (Brazil, Argentina and Peru) and Africa (Uganda, Kenya, Nigeria and Zaire) (Karyeija *et al*. 1998; Salazar and Fuentes 2001; Zhang *et al*. 2006). Isolates from Argentina, Brazil, USA, Nigeria, Kenya, Israel and Taiwan were found to be serologically closely related (Salazar and Fuentes 2001).

The SPCSV is a member of the genus *Crinivirus* of the family Closteroviridae (Kreuze *et al.* 2002). It is an economically important pathogen of sweet potato transmitted by whiteflies (*Bemisia tabaci* and *Trialeurodes abutilonea*) in a semi-persistent non-circulative manner (Sim *et al.* 2000). Hosts of this virus are limited mainly to the genus *Ipomoea*, some species of *Nicotiana* and *Amaranthus palmeri* (Cohen *et al.* 1992) and wild species of lisiantus (*Eustoma randiflorium*) (Cohen *et al.* 2001). The virus is phloem limited and cannot be transmitted through sap. The SPCSV has flexuous particles of 850-950 nm length and 12 nm diameter. The genome consists of two RNA molecules (Kreuze *et al.* 2002). RNA1 (9407 nt) contains five putative ORFs for replication related proteins and RNA2 (8223 nt) contains seven putative ORFs. The virus encodes two types of CP proteins, the major CP of 33kDa and a minor CP. This virus encodes proteins that are not found in any other viruses. For instance, RNA1 contains an ORF for a putative RNaseIII and also 22 kDa protein (p22) that shows no significant similarity to any known proteins from any organism (Kreuze *et al.* 2002). The most striking is the evidence shown (Kreuze *et al.* 2005) that the two novel proteins (RNase III and p22) cooperatively control the RNA silencing suppressor function, which may elucidate the mechanisms employed by SPSCV to breakdown host resistance in favour of unrelated virus during dual infection on sweet potato which causes SPVD (Gibson *et al.* 1998; Karyeija *et al.* 2000).

The virus is best being diagnosed by biological assay on a pair of sweet potato plants, one healthy and another infected with SPFMV. On the healthy plants hardly any symptoms will appear, while severe symptoms of sweet potato virus disease (SPVD) will appear on infected plants. Virus can also be diagnosed by immunosorbent electron microscopy and ELISA. Strain specific monoclonal antibodies (MAbs) (Cohen *et al.* 1992; Hoyer *et al.* 1996) have been routinely used in many countries including Africa. The SPCSV can be serologically divided into two major serotypes, which correlate to two genetically distantly related strain groups based on coat protein and heat shock protein 70 homologue (hsp70h) gene similarities (IsHak *et al.* 2003).

Sweet potato virus disease complexes

Sweet potato is affected by several virus disease complexes. These complexes, which have been reported from different countries, have in all cases SPFMV as one of the viral components (Salazar and Fuentes 2001). The severe sweet potato disease occurring in Africa by synergistic interaction of white fly transmitted SPCSV and SPFMV is commonly referred in literature as sweet potato virus disease (SPVD). The SPVD can cause yield reductions of 80-90 per cent. Compared to apparently healthy plants, 43, 16, and 37% reduction of the total carotenoids content in orange fleshed sweet potato (OFSP) variety Resisto were observed in plants infected with SPCSV, SPFMV and co-infection of both viruses (Kapinga 2009). This disease was first noted in eastern Belgian Congo (now Democratic Republic of Congo) in 1939 and was, for many years, considered a regional problem of sub-Saharan Africa (Carey *et al*. 1999). While SPFMV is universally distributed, SPCSV was initially only recognized in Africa. Later on disease complexes involving SPCSV also occur in Spain, South America and Central America (Carey *et al*. 1999; Di Feo *et al*. 2000;

Gutierrez *et al*. 2003; Valverde and Moreira 2004; Valverde *et al*. 2004).

During co-infection by SPFMV and SPCSV, sweet potato plants exhibit severe symptoms such as leaf strapping, vein clearing, leaf distortion, chlorosis, puckering and stunting (Salazar and Fuentes 2001). Gibson *et al.* (1998), Clark and Moyer (1988) and Carey *et al.* (1999) reported SPVD as the most devastating syndromes of sweet potato which caused yield reduction of 56-96%. Karyeija *et al*. (2000) showed that SPCSV enhances the accumulation of SPFMV by approximately 600-fold. This is unusual in that while potyviruses are often involved in synergistic interactions, more commonly they are the enhancer, as opposed to SPVD where SPFMV is the enhanced virus. There are also indications that SPCSV may broadly enhance the replication of several other sweet potato viruses. Mukasa *et al*. (2006) showed that sweet potato mild mottle virus is also enhanced by SPCSV, with virus titers increasing approximately 1000-fold. The combined infection caused severe symptoms, and the name sweet potato severe mosaic disease was suggested for the resulting disease. Kokkinos and Clark (2006) found that SPCSV enhances replication of SPV2 (IVMV), S*weet potato virus G* (SPVG), and both the russet crack and common strains of SPFMV. Symptoms from the mixed infections differed qualitatively, but were commensurate in severity with the enhanced replication of the potyvirus component, except for the SPFMV-C/SPCSV combination. Even though the titer of SPFMV-C was enhanced, plants infected with SPFMV-C and SPCSV only developed mild symptoms typical of SPCSV infection by itself. This suggests that enhancing accumulation of the potyvirus component is alone not sufficient for SPVD development. Studies (Kokkinos and Clark 2006; Mukasa *et al*. 2006) showed that titers of SPCSV were decreased in the mixed infections compared to single infections, suggesting an antagonistic effect. Untiveros *et al*. (2007) found syngergistic interactions between SPCSV and carla and cucomoviruses in addition to ipomo and potyviruses. Thus, although there are numerous potential interactions among sweet potato viruses, it has become evident that SPCSV is the key element causing enhancement of a broad array of other viruses. Kokkinos *et al*. (2006) used microarray technology to compare the effects of single infections with SPFMV-RC and SPCSV with concomitant infection on expression of sweet potato genes. Even though the array represented only a portion of the sweet potato genome, there was a dramatic difference in the number of genes that were differentially expressed: SPFMV – 3 genes, SPCSV – 14 genes, and SPFMV + SPCSV – >200 genes.

Sweet potato chlorotic dwarf (CD) is another complex disease described in Argentina (Di Feo *et al.* 2000) and is caused by interaction between SPMSV, SPFMV and SPCSV. Besides these diseases complexes, cucumber mosaic virus (CMV) of the genus *Cucumovirus* and family Bromoviridae was found to infect sweet potato together with SPCSV and usually with SPFMV also, producing similar symptoms to SPVD, and a yield loss up to 80% in Israel (Moyer and Salzar 1989; Milgram *et al.* 1996). Mixed infection of SPFMV/SPMMV (*Sweet potato mild mottle virus*) and SPCSV were reported from Uganda and East Africa (Gibson *et al.* 1998; Untiveros 2007b). In China, a crop loss over 20 per cent was observed due to the mixed infection of SPFMV and *Sweet potato latent virus* (SPLV) (Gao *et al.* 2000). Sweet potato chlorotic dwarf disease is another complex disease due to the mixed infection of SPFMV, SPCSV and *Sweet potato mild speckling virus* (SPMSV) (Di Feo *et al*. 2000). 'Camote kulot' due to the mixed infections of several viruses, (Salazar and Fuentes 2001) and the sweet potato severe mosaic disease due to SPCSV and SPMMV are two other complex diseases (Mukasa *et al*. 2006). Untiveros *et al*. (2007a) found synergistic interactions between SPCSV and carla and cucumo viruses in addition to ipomo and potyviruses. Double or mixed infection of plants often result in one virus assisting second or co-infection leading to increased titer and intensified symptom severity. In U.S.A, mixed infection of SPFMV with SPLCGV (*Geminivirus* of the family Geminiviridae) was observed (Lotrakul *et al*. 1998; 2003), while in Peru, *Sweet potato leaf curl virus* (SPLCV) was found in mixed infection with SPCSV (Fuentes and Salazar 2003). In India the mixed infection of SPFMV and SPLCGV was observed on sweet potato showing feathering, chlorotic spot, leaf distortion and leaf curl (Prasanth and Hegde 2008). Experiments with single and mixed infections with russet crack (RC) strain of SPFMV and SPLCV resulted in higher titers of SPLCV in mixed infections, while that of SPFMV remained the same (Kokkinos 2006). Kokkinos and Clark (2006) reported that viral titers of the two sweet potato infecting potyviruses (SPFMV and SPMMV) are greatly enhanced in the presence of SPCSV resulting in severe symptom development.

Begomovirus diseases

1. Sweet potato leaf curl virus

Begomoviruses are known to infect large number of plants causing leaf curl symptoms. The occurrence of leaf curl disease on sweet potato was first reported in 1979 from Taiwan (Liao *et al*. 1979; Chung *et al*. 1985). Later on, the disease has been reported from other countries namely Israel (Cohen *et al*. 1997), Japan (Onuki and Hanada 1998), USA (Lotrakul *et al*. 1998), Spain (Bank *et al*. 1999), Peru (Fuentes and Salazar 2003), Italy (Briddon *et al*. 2005), China (Luan *et al*. 2006), Kenya (Miano *et al*. 2006), India (Makeshkumar *et al*. 2007; Prasanth and Hegde 2008) and Brazil (Paprotka *et al*. 2010). Three begomovirus species have been described as infecting *Ipomoe*a species and their genomes have been fully sequenced. These are: *Sweet potato leaf curl virus* (SPLCV) (Lotrakul *et al*. 1998; Lotrakul and Valverde 1999), *Ipomoea yellow vein virus* (IYVV) (Banks *et al*. 1999) and SPLCGV (Lotrakul *et al*. 2003, Prasanth and Hegde 2008). Begomoviruses are likely to be present in many regions where sweet potato is grown but their prevalence and distribution is still unknown (Lozano *et al*. 2009). The virus can cause up to 30% reduction in yield (Clark and Hoy 2006). Yield loss due to leaf curl disease in sweet potato is yet to be estimated in many of the countries.

The most common symptom is upward curling or rolling of leaves on young plants (**Fig. 1G**). The rolled edge tends to be crinkled and vein swelling may be apparent. An interveinal chlorotic mottle is sometimes observed. Symptoms may appear seasonally and often disappear with time. Storage roots of infected plants have been reported to develop longitudinal grooves or ribs. This appears more pronounced when SPFMV is also present.

The virus is transmitted by white fly *Bemisia tabaci* biotype B in a persistent manner and by grafting, but not mechanically or by seeds. The transmission efficiency of the vector was found very low under experimental conditions (Valverde *et al*. 2004). Various *Ipomoea* species were found to be susceptible to SPLCV. The virus induces typical leaf curl symptoms on *Ipomoea nil*, *I. setosa* and *Nicotiana benthamiana* (Lotrakul *et al*. 1998).

The SPLCV has a monopartite genome (DNA-A, 2,828 nucleotides) and its organization is typical of Old World begomoviruses, containing six open reading frames and an intergenic region containing a conserved stem loop motif (Lotrakul and Valverde 1999). In Japan, SPLCV was partially purified yielding typical geminate particles with a size of *ca*. 18 × 30 nm and Western blot analysis revealed serological relationships with bean golden mosaic virus (BGMV) and *Mungbean yellow mosaic virus* (MYMV) (Onuki *et al*. 2000). A complete sequence of SPLCV (AF 104036, 2828 nts) has been determined by Lotrakul and Valverde (1999). Its genomic DNA and organization is similar to that of monopartite begomoviruses. The phylogenetic analysis of partial sequence of different SPLCV isolates suggests that there may be more than one species of SPLCV.

2. Sweet potato leaf curl Georgia virus

This virus was formerly known as *Ipomoea* leaf curl virus (Lotrakul *et al*. 2003). It is transmitted by *Bemisia tabaci* biotype B. Unlike SPLCV, SPLCGV does not cause yellow vein symptoms in *I. aquatica* and *I. cordatotriloba* (Lotrakul *et al*. 2003). Based on sequence analysis (76.5% DNA-A nucleotide sequence identity) and on differential host range (*I. aquatica* and *I. cordatotriloba*), SPLCGV is considered a distinct species of SPLCV (Fauquet *et al*. 2003; Lotrakul *et al*. 2003). Although the coat protein of SPLCGV was nearly identical to that of SPLCV, the sequence of common region and the AC1, AC2, AC3 and AC4 ORFs were different (Lotrakul *et al*. 2003). In India, leaf curl symptoms was observed on sweet potato plants with feathery mottle symptoms and such plants were found positive for geminivirus when subjected to PCR with Geminivirus group specific primers. Cloning, sequencing and phylogenetic analysis of the partial AV1, AC1 and AC 3 regions of the SPLCV Indian isolate indicated close identity with SPLGV isolates. The SPLCGV probe was also prepared from CP gene and SPLCGV was detected through NASH. The CP gene of the virus was cloned in bacterial expression vector and polyclonal antiserum specific to SPLCGV recombinant protein was also produced (Ganga 2009). The complete genome sequence of SPLCV occurring in West Bengal was reported (Kumar and Tarafdar 2009). Sequence comparison analysis of SPLCV isolates in Kenya, Peru and China suggests that the isolates from Peru and Kenya are closely related to the US isolate of SPLCV (Fuentes and Salazar 2003; Miano *et al*. 2006), while the isolate from China is more closely related to SPLCGV (Luan *et al*. 2007).

3. Ipomoea yellow vein virus

Ipomoea yellow vein virus (IYVV) was first found in Spain infecting *I. indica* plants with yellow vein symptoms (Banks *et al*. 1999) and in Italy (Briddon *et al*. 2005). Later on infection was reported on sweet potato plants (Lozano *et al*. 2004). Complete genome sequences of the IYPV have confirmed its begomovirus nature. The virus is not transmitted by *B. tabaci* biotype B. Phylogenetic analysis of three *Ipomoea* infecting begomovirus species (SPLCV, SPLCGV and IYVV) recognized by ICTV revealed that these viruses form a separate cluster that place them apart from all other begomovirus.

Other viral diseases of sweet potato

In addition to the viral diseases discussed earlier, viruses belonging to other taxonomic group also have been found to infect sweet potato. *Sweet potato ring spot virus* (SPRSV) a *Comovirus* of the genus *Nepovirus* has been reported from Kenya (Brunt *et al*. 1996). The general symptoms associated with this virus are stunting and chlorotic spot. Sweet potato leaf speckling luteovirus (SPLSV) is a sweet potato infecting luteovirus, causing mild whitish specking on leaves (Fuentes *et al.* 1996). The association of SPFMV or SPCSV and *Cucumber mosaic virus* (CMV) in sweet potato has been reported from Israel (Milgram *et al*. 1996). Moyer and Salzar (1989) reported a caulimovirus namely sweet potato caulimovirus (SPCaLV) infecting sweet potato from U.S.A. Aritua *et al.* (2009) reported a sweet potato chlorotic fleck virus (SPCFV), a carlavirus of the family Flexiviridae infecting sweet potato from South Africa, the general symptoms associated with this virus are fine chlorotic spot and leaf distortion. A *Potyvirus* of the family Potyviridae, *Sweet potato virus-G* (SPV-G) has been reported from Egypt (IsHak *et al.* 2003) and the symptoms associated with SPV-G are purple rings and vein clearing. *Sweet potato virus 2* (SPV2), a tentative member of the genus *Potyvirus* has been reported by Souto *et al.* (2003) and Ateka *et al.* (2004). The virus was first isolated from Taiwan and the general symptoms associated with SPV2 are leaf distortion and vein mosaic. Recently, Cuellar *et al*. (2011) reported synergistic interaction of two distinct cavemoviruses with SPCSV in cultivated sweet potato in East Africa, Central America and the Caribbean islands, but not in samples from South America.

Little leaf phytoplasma disease also known as Witches' broom was first described by van Velson (1967) from Papua New Guinea. The disease affects yield primarily by reducing the number of roots. Yield reductions of more than 50% have been recorded (Pearson *et al.* 1984). The disease is widespread in Asia (Bangladesh, China, Taiwan, India, Indonesia, Japan, Korea, Malaysia, Philippines, Australia and the western Pacific (Chen 1972; Kahn *et al*. 1972; Dabek and Sagar 1978; Jackson and Zettler 1983).

Symptoms of vein clearing, small leaves (little leaf) frequently chlorotic with a more rounded shape, curling of the leaf margins, stunting of plants, growth habit tending to be more erect, proliferation of auxiliary shoots, together with a greatly reduced root system, result in weak plants with a compressed or bushy appearance. The number and quality of tubers are reduced and production of latex in vines and roots is also reduced. The host range of the pathogen include *Ipomoea pes-caprae, Pharbitis nil*, *Pharbitis purpurea*, *Vigna unguiculata* var. *sesquipedalis* and *Lycopersicon esculentum* which act as reservoir of inoculums (Saqib *et al.* 2006). Experimentally, the phytoplasma can be transmitted to *Ipomoea setosa*, *I. triloba*, *I. indica*, *I. ericolor* and *Catharanthus roseus*. The little leaf phytoplasma can be transmitted by the leaf hopper *Orosius lotophagorum ryukyuensis* and *Nesophrosyne ryukyuensis* in a persistent manner. Low annual rainfall and prolonged dry seasons favour the vector and under these conditions, the disease can reach epidemic proportions. Infected planting material is also important in the dissemination of the disease. As the disease has an exceptionally long incubation period in sweet potato (up to 283 days) following graft transmission, infected planting material can appear healthy (Jackson and Zettler 1983). The phytoplasma could be diagnosed by PCR, and the full length chromosome was determined as 600 Kbp which is one of the smallest phytoplasm genome sizes (Gibbs *et al*. 1995). Tairo *et al*. (2006) identified the phytoplasma causing little leaf of sweet potato in Australia as *Candidatus Phytoplasma aurantifolia* species belonging to 16SrII group based on sequencing and phylogenetic analysis of 16srRNA gene sequences. Symptoms on graft inoculated *I. setosa* may take six months or more to develop (Moyer *et al*. 1989). The disease can be diagnosed on graft inoculation on to *I. ericolor* with relatively short incubation period of 35 to 49 days (Clark and Moyer 1988). In seed certification schemes, no phytoplasm infections must be tolerated during the growing season. Stocks of *in vitro* cultures used for propagation should be from pathogen free plants and maintained under conditions designed to prevent infection and contamination. Use of healthy planting material, field sanitation by removing old crop debris and weeds and roguing of symptomatic sweet potato and other hosts are highly useful for management of the disease. Meristem culture and thermotherapy was found effective in elimination of phytoplasma from affected sweet potato (Green *et al*. 1989).

Identification and detection of sweet potato viruses

Detection and characterization of sweet potato virus is crucial in understanding the epidemiology of the disease caused by viruses and development of control strategies (Jubert *et al.* 1979; Hollings and Brunt 1981; Chatterjee *et al.* 2007). Sweet potato viruses have been detected by observing symptom expression in the field and host range studies (Chavi *et al.* 1997) and by their vector relationship (Schaefers and Terry 1976). The primary test to detect sweet potato viruses is bioassay on indicator plants by observing symptoms, vector transmission procedures and serological detection (Moyer and Salzar 1989). Based on

biological, serological and nucleic acid properties of plant viruses, several diagnostic methods have been developed for sweet potato virus detection (Valverde *et al*. 2008). However, detection and identification of sweet potato viruses is not an easy task due to the low concentration and the uneven distribution of some viruses within the plant (Esbenshade and Moyer 1982) and the presence of phenolic compounds, latex and inhibitors in sweet potato tissue (Abad and Moyer 1992). Difficulties in detection have also been attributed to the occurrence of mixed infections and strain variations (Valverde *et al*. 2008).

1. Biological methods

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

Though biological methods are very important in diagnosis of sweet potato viruses, in recent years, progress has been made in developing sensitive techniques for several sweet potato viruses (Abad and Moyer 1992; Colinet *et al*. 1998; Kokkinos and Clark 2006; Mukasa *et al*. 2006; Tairo *et al*. 2006). Hence use of one or combination of different molecular and serological techniques is recommended for indexing of sweet potato for viruses.

2. Serological methods

Serological methods have been widely used for the detection of different sweet potato viruses including SPFMV (Esbanshade and Moyer 1982; Gibson *et al.* 1998; Gutierrez *et al*. 2003). Moyer and Kennedy (1978) performed double diffusion tests using SPFMV polyclonal antiserum. A precipitin line was observed in between SPFMV infected extract and polyclonal antiserum, where as no precipitin line was found when healthy extract was used. The presence of precipitin line was considered as positive for SPFMV.

A membrane immune binding assay, also known as nitrocellulose membrane ELISA (NCM-ELISA) has been used with success to detect several sweet potato viruses (Abad and Moyer 1992; Makeshkumar *et al*. 2001; Gutierrez *et al*. 2003; Mukasa *et al*. 2003a; Souto *et al*. 2003; Tairo *et al*. 2004; Valverde and Moreira 2004). Detection kits using this technique have been developed by the International Potato Center, Peru. Ganga (2009) detected SPFMV in sweet potato germplasm collections maintained at CTCRI, India by NCM–ELISA using the polyclonal antiserum produced against the recombinant coat protein of SPFMV. The protocols for detection of SPFMV infecting sweet potato in India by grafting on *I. setosa*, NCM-ELISA and RT-PCR have been brought out in the form of technical bulletin by CTCRI, India (Hegde *et al*. 2010). Most of the ELISA methods can detect sweet potato viruses mainly from symptomatic plants of sweet potato. Hence grafting of sweet potato on to *I. setosa* or *I. nil* and indexing of the resulting *Ipomoea* samples with NCM-ELISA is recommended.

3. Nucleic acid spot hybridization

Abad and Moyer (1992) reported the use of Nucleic Acid Spot Hybridization (NASH) for the detection of SPFMV using CP gene specific riboprobe. Relative sensitivity of MIBA and NASH assay were compared to the sensitivity of graft transmission assay. The study also identified the sensitivity of riboprobe in detection of SPFMV even from asymptomatic plants. Detection of SPLCV in sweet potato samples collected from the field, through molecular hybridization using SPLCVCP probe was reported by Valverde *et al*. (2004a). Müller *et al.* (2001) developed a non-radioactive CP gene specific probe to detect SPCSV through Nucleic Acid Spot Hybridization. The non-radioactive probe was labeled by a random priming labeling technique with a fluoresceinated nucleotide and assayed by NASH. The probe developed has been used for the detection of SPCV and was more sensitive than ELISA. The SPFMV in India was also detected through NASH using biotinylated CP gene probe (Makeshkumar *et al*. 2006; Hegde *et al*. 2007b).

4. Polymerase chain reaction

The polymerase chain reaction (PCR) is one of the most sensitive and reliable techniques for the detection of sweet potato virus (Colinet and Kummeret 1993; Colinet *et al.* 1998; Lotrakul *et al.* 1998; Alicai *et al.* 1999; Lyerely *et al.* 2003). Grisoni *et al.* (2006) reported the use of RT-PCR for the detection of sweet potato infecting potyviruses, using degenerate genus specific primers designed to amplify the variable 3′ untranslated region (UTR) and coat protein gene of the *Potyvirus.* The genus specific PCR and subsequent molecular analysis of amplified regions can be used as a powerful method for the rapid identification and differentiation of potyviruses infecting sweet potato and appeared as the most suitable method for viruses that are difficult to be purified and/or occurring in mixed infections (Colinet *et al.* 1998). Specific primers for detecting and differentiating SPFMV, SPMMV, SPCSV, SPLCV and other sweet potato viruses have been designed from the nucleotide sequence of these viruses (Colinet *et al.* 1994; Lotrakul *et al.* 1998; Alicai *et al*. 1999).

Joeng *et al.* (2003) reported the detection of SPFMV using gene specific primers flanking to the core region of SPFMV CP gene. The use of RT-PCR for the detection of SPFMV using various plant parts was reported by Takeshi (2002). The study identified high virus concentration at the proximal end of a tuberous root or at the lower leaf than any other parts of the plant. It was also observed that RT-PCR demonstrated higher detection sensitivity than bioassay and ELISA. Alicai *et al.* (1999) reported the identification of SPCSV by amplification of HSP70 homologue gene and CP gene through RT-PCR. Kokkinos and Clark (2006) reported a quick and quantitative RT-PCR assay for the detection of SPFMV and SPCSV from infected sweet potato as well as from the indicator host. Using this real time quantitative polymerase chain reaction, an increase in titer levels of SPFMV was observed in the presence of SPCSV (Kokkinos and Clark 2006; Mukasa *et al*. 2006). PCR was standardized for the detection of sweet potato leaf curl virus also (Li *et al.* 2004). The PCR based detection using specific and degenerate primers have been successfully used to detect SPLCV from indicator hosts and sweet potato plants (Lotrakul and Valverde 1999; Li *et al*. 2004; Valverde *et al*. 2008). Sweet potato leaf curl virus was detected from samples collected from USA using specific and degenerate primers for SPLCV (Valverde *et al*. 2008). Kokkinos and Clark (2006) standardized a RT-PCR for the quick and reliable detection of sweet potato leaf curl virus.

Management of sweet potato viruses

Traditional cultivation practices such as the piece meal harvesting and exchanging the planting material freely between neighbouring farmers are the major factors favouring the spread and survival of SPFMV and other sweet potato viruses. Information on the control of virus and the method of infection through planting material is lacking in resource

poor farmers of India. The control of virus relies mainly on preventing the establishment, development and dispersal of the viruses. Prevention of sweet potato viral diseases involves a wide variety of measures such as eradication of sources of infection, elimination of alternate hosts as well as vectors (Clark and Moyer 1988). At present, the best way to control virus diseases in sweet potato is to supply the farmer with virus indexed propagation material. Such plantlets can be obtained from meristem, shoot tip cultures in combination with cryotherapy (Wang and Valkonen 2008) or nodal cultures with thermotherapy (Jeeva *et al*. 2004). Such programmes adopted in Israel increased the yield by 100% (Loebenstein *et al*. 2009) and in China increase in yield was 22-92% (Gao *et al*. 2000). In other countries in Africa or Asia, such programmes are used only on limited scale where sweet potato is grown only a food security crop and not on a commercial scale. However, cultural/phytosanitation methods including destroyal of freshly infected plants and weeds and isolation of new crops from the old infected crops with barriers like maize (15-20 m) (Gibson and Aritua 2002) are highly effective in controlling these virus diseases. The virus free plants were found to get infected under field conditions, however, if strict phytosanitation and cultural practices are adopted, the planting materials can be used by farmers for at least 3 years. Several cultivars resistant to SPVD have been developed for African countries (Mwanga *et al*. 2002). However, their resistance need to be seen in places where different strains of viruses of the SPVD are present. Genotypes described as resistant to SPFMV in Peru were found to be susceptible in East Africa (Gibson *et al*. 1998; Karyeija *et al*. 1998b; Mwanga *et al*. 2002). Development of transgenic sweet potato plant can be another method of controlling the sweet potato virus (Owour 2001). The use of cysteine proteinase inhibitor gene (oryzacystine1) has proved to make some sweet potato cultivar tolerant to SPFMV-RC (Cipriani *et al*. 2000). However, Karyeija *et al*. (2000a) demonstrated that infection with SPCSV overcomes resistance to SPFMV.

CONCLUSION

It is clear from the review that sweet potato diseases are one of the major constraints in sweet potato production throughout the world though the disease incidence and severity varies in different countries depending on the type of cultivar used and management practices adopted. Most of the work on identification of pathogens and management of the diseases has been undertaken in a few countries like USA, Israel, China and Japan. In most of the developing countries, though diseases are known to cause significant damage, no systematic efforts are being undertaken to manage these diseases. Sweet potato is grown mainly by resource poor, small and marginal farmers and the crop is not being exploited for its commercial uses in many other sweet potato growing countries. Productivity of sweet potato is also far below world average in many countries. There are many reasons for the low productivity and one of the important reasons is the lack of awareness among farmers about sweet potato diseases and continuous use of diseased planting materials year after year. Moreover no private firms are coming forward to produce disease free planting materials. Hence, a systematic disease free planting materials production and distribution to farmers through government agencies and non government organizations (NGOs) is essential. Educating the farmers about the benefits of healthy planting materials through trainings and demonstrations are needed. Identification and development of seed villages for production of disease free quality planting materials may be highly useful to supply healthy planting materials. A constant survey and monitoring of the diseases prevailing on sweet potato is also required. Research on variability of the pathogens infecting sweet potato and ready to use diagnostics for all the diseases need to be developed for indexing of planting materials. The new biotechnological approaches may enable scientists to rapidly develop superior disease resistant cultivars. There are several fungal, bacterial and viral diseases which infect the sweet potato crop. As sweet potato is grown primarily as a subsistence crop in most developing countries, chemical control of these diseases is not widely practised. Frequent replanting with virus free stock is also no enduring solution as warm climates lead to a high reinfection rate. SPFMV is a major problem that causes 'russet crack' disease and affects sweet potato production, particularly in Africa. Efforts are under way to develop resistance to the SPFMV using the coat protein gene and antisense RNA genes.

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