EFFECTS OF VIRUSES ON GROWTH AND YIELD OF LOCAL SWEET POTATO VARIETIES IN KENYA

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DECLARATION

I declare that the work presented in this thesis is from my own research and has not been submitted for a degree in any other university.

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We confirm that the work reported in this thesis was carried out by the candidate under our supervision and has been submitted for examination with our approval.

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DEDICATION

To my beloved mother

The late Councillor Mrs Sarah Towett

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ABSTRACT

Experiments to determine the effect of viruses on the vine and tuber yield of three popular Kenya sweet potato varieties were undertaken at the University of Nairobi Kabete farm. The sweet potato varieties were Kemb10, Bungoma and Ex-shimba hills which were either virus free or infected with *sweet potato feathery mottle potyvirus* (SPFMV), *sweet potato mild mottle ipomovirus* (SPMMV), *sweet potato chlorotic stunt crinivirus* (SPCSV) or SPFMV + SPCSV. The treatment combination was a split plot factorial structure in a randomized complete block design with three replicates. Disease severity and photosynthetically active radiation (PAR) interception by the plant canopy were determined. At harvest, leaf area index (LAI), specific leaf weight (SLW), fresh vine weight, total tuber yield, marketable and unmarketable tuber yield and harvest index (HI) were determined.

Dual infection resulted in severe symptoms characteristic of sweet potato virus disease complex (SPVD). SPFMV and SPMMV had no significant effect on PAR intercepted by the plant canopy. Infection with SPCSV and SPFMV + SPCSV had a reduced PAR interception by 18% and 75% respectively. In addition, SPCSV and dual infection reduced fresh vine yield by 40% and 75% respectively. Infection with single viruses caused an increase in SLW ranging from 5 to 40% while plants dually infected with SPFMV + SPCSV reduced SLW by 15%. Only dual infection with SPFMV + SPCSV caused 66% reduction in LAI. Plants dually infected with SPFMV + SPCSV caused a marketable tuber yield loss ranging from 82 - 98% in all varieties.

Infection with SPFMV, SPCSV and SPMMV on varieties Bungoma and Kemb10 reduced yields through hindered translocation of photosynthate as inferred by increased

SLW and reduced HI. Variety Ex-shimba hills was found to be tolerant to single virus infections but Bungoma and Kemb 10 were susceptible. All the three varieties were susceptible to infection with SPFMV + SPCSV causing reduced vine and tuber yield through reduced LAI, reduced photosynthetic organs due to stunting, chlorosis and consequently PAR interception by plant canopy. Dual infection also caused a reduction in tuber yield through harvest index.

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CHAPTER 1

INTRODUCTION

1.1 Importance of sweet potato

Sweet potato (*Ipomoea batatas* (L.) Lam.) is one of the major root crop grown in the tropical and sub-tropical regions of the world. Among the 50 genera and more than 1000 species of the *Convolvulaceae* family, only *Ipomoea batatas* (L.) Lam. is of major economic importance as food. Sweet potato is cultivated in more than 100 countries. On a world wide side, the crop ranks 7th in terms of total production and is ranked 4th in developing countries (FAO, 2000). In monetary terms, it is ranked 13th globally in production value of agricultural commodities (Woolfe, 1992). The world hectarage devoted to sweet potato is 9.1 million while those of potato (*Solanum tuberosum* L.) and cassava (*Manihot esculentum* Crantz) are 18.3 and 16.4 million hectares, respectively (FAO, 1997).

The bulk of sweet potato production is in China, which produces about 80 % of the total world production (FAO, 2000). In sub-Saharan Africa, sweet potato is grown over a wide range of environments, but mostly at mid elevations between 860 and 2000 metres above sea level, mainly by small holder farmers (Horton, 1988). It is a staple food in large parts of Uganda, Rwanda, Burundi, Kenya and eastern Zaire along with maize, beans, cassava and bananas (Carey *et al.*, 1996). In Kenya, sweet potato is grown all over the country and almost 75% of the total production is concentrated in the densely populated Lake Victoria basin in western Kenya (Rees *et al.*, 1997). Sweet potato is mainly intercropped with other staple food crops, especially maize (Low *et al.*, 1997).

Farmers predominantly plant sweet potato at the beginning of the rainy season, usually growing more than one variety in a single plot (CIP, 1991). Although sweet potato is traditionally regarded as a subsistence crop, a significant share of the harvest (60%) is sold to raise small amount of money to meet family needs (Qaim, 1998).

The crop is grown principally for its tuberous storage roots, which provide energy, high quality protein (e.g. containing lysine), vitamin B_1, B_2 niacin pyridoxine, folic acid and ascorbic acid. Orange-fleshed sweet potato also contains large quantities of β-carotene, which the human body can convert to vitamin A (Low *et al.*, 1997). Other important nutrients found in sweet potato include calcium, phosphorus, iron, sodium and potassium (Woolfe, 1992). Sweet potato can be utilized as a salad, staple or vegetable food, a sweet dessert, a fast food (French fries), an animal feed or a basic industrial raw material, for example, in the manufacture of alcohol (Ministry of Energy, Denmark 1981; Woolfe, 1992). The crop has a short growing season (Aldrich, 1963; Karyeija *et al.*, 1998), allowing it to fit into different cropping systems and it can be harvested piecemeal to provide fresh daily food for a family (Karyeija *et al.*, 1998). In addition, sweet potato yields the greatest amount of food per unit area and unit time. The storage root of sweet potato provides a reliable source of food during time of scarcity and in some densely populated areas, the foliage is an important supplementary source of fodder in livestock production systems (Ndolo *et al.*, 1997). Sweet potato is dependable because it can produce despite drought and in times of natural disasters, civil strife and economic hardship sweet potato has served well as a famine relief crop (CIP, 1998; Mutuura *et al.*, 1992). Sweet potato vines grow very rapidly and cover the ground within a few weeks of planting. Therefore, in areas where they are of major importance they are often grown

as the first crop after clearing fallow because their dense foliage can suppress weeds, making it a useful cover crop (Aldrich, 1963; Wambugu,1990).

1.2 Justification of the study

Although the crop can withstand adverse weather conditions better than most crops during growth, there are pests and diseases that affects its performance. A survey carried out in the year 2001 revealed that the viruses most commonly found in the major sweet potato growing areas in Kenya are *sweet potato feathery mottle potyvirus* (SPFMV), *sweet potato chlorotic stunt crinivirus* (SPCSV) and *sweet potato mild mottle ipomovirus* (SPMMV). Reports from other countries suggest that viruses diminish tuberous root yields in susceptible varieties and can constitute a serious constraint to sweet potato production. No reports have been documented on the effect of SPFMV, SPCSV or SPFMV + SPCSV on tuber yield of popular Kenyan varieties and neither is there any documented evidence on their effects on vine yield. In addition, the effects of SPMMV on yield have not been reported. There was therefore a need to determine the effect of SPCSV, SPFMV and SPMMV on the yield of local Kenyan sweet potato varieties. Because information on how viruses affect yield of sweet potato plants is scanty, there was also a need to determine the effect of virus infection on harvest index and light interception by plant canopy. Therefore, this study aims to determine the effect of virus infection on tuber and vine yield of three local and popular sweet potato varieties and attempts to show how virus affects sweet potato yield.

1.3 Objectives

The overall objective of the research was to determine the effect of viruses on the yield of popular local sweet potato varieties.

The specific objectives of the study were:

1) To determine the effect of SPFMV, SPMMV, SPCSV and SPFMV + SPCSV on tuber, harvest index and vine yield of three local sweet potato varieties

2) To understand how any yield effects might occur, through studying the effect of viruses on plant canopy cover.

CHAPTER 2

LITERATURE REVIEW

2.1 Origin of sweet potato

Sweet potato (*Ipomoea batatas* Lam. (L.)) originated from Central and/or South America. Its dissemination to Polynesia is associated with extraordinary voyage of early Peruvian and Polynesian explorers and traders. Christopher Columbus first brought sweet potato to Europe on his return from South America in the 16th century. Portuguese explorers introduced the crop to Africa, India and Eastern Asia (Onwueme, 1978; Bassett, 1986). Today, sweet potato is grown in nearly all parts of the tropical and sub tropical world and in the warmer areas of the temperate regions (Onwueme, 1978; Horton *et al.*, 1984).

2.2 Sweet potato production

Sweet potato is considered to be a warm season crop in spite of its wide adaptation to varying ecological zones. Optimal conditions for sweet potato growth are temperatures at or above 24^{o} C, well distributed rainfall of 750-1000 mm and a well drained soil of pH 5.6-6.6 as the crop does not withstand waterlogging (Onwueme, 1978). In tropical latitudes, it flowers readily but the plant usually set few viable seeds. However, many genotypes do not readily flower, others are sterile and most are self-incompatible (Basset, 1986). Sweet potato genotypes are broadly grouped into bush, intermediate and vining types and vary greatly in branching pattern and overall stem length. In selection of varieties, the important characteristics preferred by farmers in Kenya are high tuber yield, high dry matter content, extensive foliage growth and early maturity (Ateka *et al.*, 2001). The popular varieties include Kemb 10, Mugande, SPK 013, SPK 004, KSP 20, Bungoma and Ex-shimba Hills. Variety Kemb 10 has an erect growth habit, is early

maturing (90-120 days), tubers have cream skin colour and yellow flesh. Because it has a wide ecological adaptation it is grown in all major sweet potato producing areas. The Mugande variety has a red skin with white flesh and very good consumer acceptance. It is reported to originate from Rwanda (MOA, 2001) and is late maturing (120-150 days). Kenya sweet potato (KSP) 20 variety originated from International institute of Tropical Agriculture and has purple red skin with white flesh (MOA. 2001). It is early maturing and high yielding. Tubers of variety Bungoma have a yellow flesh with a red skin colour and long stolons. It has a semi-erect growth habit with petioles which are purple at the junction with lamina, is late maturing (120-150 days). Bungoma is widely grown in western Kenya. Tubers of variety Ex-shimba hills have a red skin colour and white flesh: it is early maturing (90-120 days). It has a spreading growth habit and anthocyanins pigmented purple stem. It is mainly grown in coastal Kenya.

2.2.1 Constraints to sweet potato production

Kenya's annual production of sweet potato has fluctuated over the years and the mean yield stands at 9.2 tonnes/ha, (FAO, 2000). Sweet potato production does not meet the demand (CIP 1998) and there is need to increase production. Farmers suffer significant yield loss and the yield levels are 20% of the crop's potential (50 tonnes/ha) observed under experimental conditions (Ndolo *et al.*, 1997; Qaim, 1999), so there is ample opportunity to increase yields. Constraints to increased production for sweet potato includes, lack of planting materials in some dry areas, lack of marketing prospects for the crop due to poor transport systems, poor soil fertility, poor agronomic practices and use of low yielding varieties (Moyer and; Salazar, 1989; Wambugu, 1991; Carey *et al* ., 1996; Ateka *et al.*, 2001). In addition biotic factors such as pests and diseases also cause

yield loss, Pests which include weevils, monkeys, moles, rats and porcupines. The crop is also attacked by a wide range of pathogens, which include fungi, bacteria, nematodes and viruses (Clark and Moyer, 1988; Moyer and Salazar, 1989; Ames *et al.*, 1996; Njuguna and Bridge; Geddes, 1990).

Viruses are major causes of diseases in sweet potatoes (Geddes, 1990; Wambugu 1991; Carey *et al.*, 1996). More than 20 viruses have been reported on sweet potatoes in the world and they include, *sweet potato chlorotic stunt crinivirus* (SPCSV), *sweet potato feathery mottle potyvirus* (SPFMV), *sweet potato mild mottle ipomovirus* (SPMMV), sweet potato chlorotic fleck potyvirus (SPCFV), sweet potato latent potyvirus (SPLV), C-6, sweet potato mild speckling potyvirus (SPMSV), *cucumber mosaic cucumovirus* (CMV), sweet potato leaf curling geminivirus (SPLCV) sweet potato ringsport nepovirus (SPRSV) and sweet potato caulimo like virus (SPCaLV) (Moyer and Salazar, 1989; Brunt *et al.*, 1996; Gibson *et al.*, 1998b). In Kenya SPFMV, SPCSV, SPMMV, SPCFV and CMV have been reported in farmers' fields and the most widely distributed in the major sweet potato production areas are SPCSV, SPFMV and SPMMV (Wambugu, 1990; Ateka *et al.*, 2001)

2.3 *Sweet potato chlorotic stunt crinivirus*

SPCSV is a member of the genus *Crinivirus* in the family *Closteroviridae*. The virus has flexuous, filamentous particle 850-920 nanometers (nm) long with a coat protein of molecular weight of 25-34 kilodaltons (kda). It has a positive single stranded RNA. SPCSV has been reported in Africa (Schaefers and Terry, 1976), Israel (Cohen *et al.*, 1992) and in South America (Di Feo *et al.*, 2000). In America, it occurs as a component of chlorotic dwarf disease complex (SPCSV, together with SPFMV and another potyvirus

Sweetpotato mild speckling virus (SPMSV)) The chlorotic dwarf disease has been reported in Argentina, Peru and Brazil (Gibson and Aritua, 2000; Di Feo *et al.*, 2000). In Africa, two serologically distinct strains currently known to infect sweet potato in East Africa ($SPCSV_{EA}$) and West Africa ($SPCSV_{WA}$) have been reported (Hoyer *et al.*, 1996; Gibson *et al.*, 1998a)

The main host of SPCSV in Africa seems to be sweet potato and no secondary hosts have been reported. The symptoms of SPCSV infection vary geographically. In East Africa the virus may cause no symptoms or induce colour changes (purpling or yellowing) of lower or middle leaves depending on the sweet potato variety (Gibson *et al.*, 1998a). On *Ipomoea setosa*, the virus causes mild chlorosis/mosaic and/or severe stunting of the plant and development of small brittle leaves. An inward curling of the leaves may also occur (Winter *et al*; 1992). On *I. nil*, SPCSV causes chlorosis and epinasty in new leaves followed by severe stunting and dwarfing of the entire leaf. Older leaves are bronze-coloured and brittle (Larsen *et al.*, 1991).

SPCSV alone may cause only small yield losses in the first year, but up to 30% in the second year (Hahn, 1979; Milgram *et al.*, 1996). In Argentina, the chlorotic dwarf disease can result in yield losses of up to 80% (Di Feo *et al.*, 1995). SPCSV is the second most prevalent virus with an incident rate of 62% in major sweet potato growing areas in Kenya. (Wambugu, 1990; Ateka *et al.*, 2003)

2.4 *Sweet potato feathery mottle potyvirus*

SPFMV belongs to the family *Potyviridae* and genus *Potyvirus*. The virus particles are elongate, flexous rods with a monopartite, single stranded, positive-sense RNA molecule. The positive RNA is approximately 10.6 kbp and is 10-15% larger than typical

potyviruses (Moyer and Cali, 1985). It has atypical long virion (810 x 865 nm-about 100nm longer than a typical potyvirus) and large RNA genome with a molecular weight of 3.65 x10, 000 kda (Moyer and Cali 1985; Abad *et al.*, 1992; Karyeija *et al.*, 1998). SPFMV occurs in all areas where sweet potato is grown (Karyeija *et al;* 1998).

Several isolates and strains of SPFMV have been characterized in different parts of the world. They include the ordinary (O) (Usugi *et al.*, 1991), russet crack (RC), (Moyer and Salazar, 1989) and severe (S) (Mori *et al.*, 1995) strains. Sweet potato is the main natural host of SPFMV although the virus occurs in wild *Ipomoea* species. The experimental host range of the virus is mainly restricted to the *Convolvulaceae* and *Chenopodiaceae*, but a few strains also infect species of the *Solanaceae* of which *Nicotiana benthamiana* is a good host (Clark and Moyer, 1988). Several strains cause local lesions on *Chenopodium amaranticolor, C. murale, C. amaranticolor, C. quinoa* and *Spinacia oleracea.* Also some strains cause lesions on Convolvulaceae species like *Calonyction aculeatun, Ipomoea hederacea, I. incarnata, I. lacunosa, I. purpurea, I. trichocarpa, I. tricolor, I. wrightii, Merremia sibirica and Quamoclit lobata* and on Solanaceae species (*Datura metel, Nicotiana benthamiana, N. clevelandii, N. occidentalis* and *N. tabaccum.* Some wild *Ipomoea* species are reservoirs of SPFMV (Clark *et al.*, 1986).

Symptoms of SPFMV on the foliage of sweet potato are either mild or absent. Leaf symptoms appear as faint, irregular chlorotic spots occasionally bordered by purplish pigment. The classic irregular chlorotic patterns (feathering) along midribs and faint to distinct chlorotic spots with or without purple margins occur in some cultivars. Symptom intensity on foliage is influenced by cultivar susceptibility, degree of

environmental stress, growth stage and strain virulence. Increased stress can lead to symptom expression, whereas rapid growth may result in symptom remission (Brunt *et al.*, 1996). Symptoms on storage roots depend on the strain of SPFMV and the sweet potato variety. The common strain causes no symptom, but the RC strain causes external necrotic lesions or internal cork on certain varieties (Clark and Moyer, 1988; Ames *et al.*, 1996). On *Ipomoea nil*, SPFMV induces vein clearing, vein banding, epinasty and crinkling of the leaf. Some severe strains induce, stunting and necrosis and eventually cause death of the plant. On *Ipomoea setosa*, the predominant symptoms are chlorotic vein-clearing, vein-banding and chlorotic spots. *I. setosa* plant is used as an indexing host when infected by grafting. On *Chenopodium amaranticolor* and *C. quinoa*, chlorotic lesions develop on inoculated leaves but no systemic infection occurs. SPFMV induces cytoplasmic inclusions characteristic of potyviruses (Campbell *et al;* 1974; Pozzer *et al;* 1995).

Campbell et al (1994) reported that some isolates of SPFMV cause 100% tuber yield loss on sensitive varieties. Similar results were obtained by Pozzer *et al* (1995) under field conditions in Venezuela. In a screenhouse experiment carried out in Uganda, plants inoculated with SPFMV had half the root yields of virus-free plants (Gibson *et al.*, 1997). In contrast, trials conducted in the United States indicated that plants inoculated with the RC strain of SPFMV had similar yield as symptomless ones (Kantack and Martin, 1958).. These reports show a lack of clarity as regards the effects of SPFMV on sweet potatoes varieties. SPFMV is the most prevalent virus an incident rate of 76% in the major sweet potato growing areas in Kenya (Ateka *et al.*, 2003).

2.5 Sweet potato virus disease

Sweet potato virus disease (SPVD) is caused by the synergistic interaction of SPFMV and SPCSV. The disease has been reported in Asia, South, North and Central America (Di Feo *et al.*, 2000). In sub-Saharan Africa, SPVD is common and it is recognized as the most devastating disease of sweet potato (Geddes, 1990). Recent survey carried out in Kenya has showed that the disease is widespread (Ateka *et al.*, 2001).

Synergism between viruses refers to situations where co-infection with two viruses causes more severe symptoms than the sum of the effects of infection with either virus alone and in which multiplication of at least one of the virus is enhanced by the other (Karyeija *et al.*, 1999). When SPFMV infects alone, mild or no symptoms are observed and in symptomless leaves the SPFMV titres are below the detection limits of most serological tests (Esbenshade and Moyer, 1982; Gibson *et al.*, 1998b). However, resistance to SPFMV is overcome in sweet potato co- infected with SPCSV (Karyeija *et al.*, 1999; Karyeija *et al.*, 2000). Sweet potato plants infected with both SPCSV and SPFMV have a higher titre of SPFMV, more severe symptoms and are a better source of aphid acquisition than plants infected with SPFMV alone (Aritua *et al.*, 1998). In addition, large amounts of SPFMV RNA are detected in plants dually infected with SPFMV and SPCSV but very small amounts are detected when SPFMV infects alone (Gibson *et al.*, 1998b; Karyeija *et al.*, 2000).

SPVD symptoms vary with plant genotype but typically include severe stunting of plants, the production of small leaves which are often distorted, narrow (strap-like) and crinkled and with a chlorotic mosaic and/or vein-clearing. On *Ipomoea setosa*, SPVD

causes stunting, severe chlorosis followed by necrosis of older infected leaves and a severe reduction of leaf laminae.

SPVD causes almost total yield loss of sweet potato infected plants of susceptible varieties (Mukiibi, 1977; Hahn, 1979). Studies conducted in Cameroon and Nigeria showed that SPVD reduces tuber yields in susceptible sweet potato varieties by 56-90% and 78% respectively (Hahn, 1979; Bouwkamp and Ngeve, 1991). In Taiwan, SPVD caused 25 -38% root yield loss (Chung *et al.*, 1981). In Uganda sweet potato plants of cultivars *Bitambi* and *Kyebandula* severely affected by SPVD produced only 33% of the tuber yield when compared to healthy plants (Aldrich, 1963; Mukiibi, 1977). About 62 - 97% root yield loss was reported on five non-indigenous sweet potato clones from International Potato Centre dually infected with SPFMV + SPCSV (Aritua *et al.*, 2000). Data on the effect of SPVD on the yield of popular sweet potato varieties grown in Kenya has not available while the disease is widespread (Ateka *et al.*, 2003).

2.6 *Sweet potato mild mottle ipomovirus*

The virus belongs to the family *Potyviridae*, genus *Ipomovirus*. The virus has flexous filamentous particles mostly of 950 nm long (Hollings *et al.*, 1976a, b). The viral coat protein consists of a single polypeptide with a molecular weight of 37.7 kda (Hollings *et al.*, 1976a, b). The particles contain single-stranded RNA. SPMMV has been found in Kenya (Sheffield, 1957; Hollings *et al.*, 1976a, b; Wambugu, 1991), Uganda (Sheffield, 1957; Hollings *et al.*, 1976a, b; Gibson *et al.*, 1997), Tanzania (Sheffield, 1957; Hollings *et al.*, 1976a, b), Argentina (Biderbost *et al.*, 1993), Asia (Nakano *et al.*, 1994), Oceania (Mason & Beetham, 1998) and South Africa (Jericho and Thompson, 2000).

The only known natural host of SPMMV is *Ipomoea batatas* but the virus is experimentally transmissible by mechanical inoculation to at least 45 species in 14 plant families (Hollings *et al.*, 1976a, b). SPMMV causes mild leaf mottling and stunting in plants of susceptible sweet potato cultivars, but less severe symptoms or even symptomless infection is reported in tolerant genotypes (Hollings *et al.*, 1976a, b). The virus induces the formation of cylindrical cytoplasmic inclusions (Edwardson, 1974; Hollings *et al.*, 1976b). The virus induces chlorotic lesions in sap-inoculated leaves of *Chenopodium amaranticolor* and *C. quinoa*, but no subsequent systemic infection occurs. On *Ipomoea setosa* and *Pharbitis nil*, SPMMV causes conspicuous chlorotic vein banding, mottling and distortion of systemically infected leaves. *Ipomoea. setosa* is also especially useful as an indexing host when infected by grafting. On *Nicotiana clevelandii*, *N. glutinosa*, *N. tabacum*, chlorotic or grey lesions develop in inoculated leaves, followed by vein banding, mottling and distortion of systemically infected leaves (Hollings *et al.*, 1976a, b).

Information on SPMMV effect on yield has been lacking although the virus is the third most commonly found in major sweet potato growing areas in Kenya with a prevalence rate of 42% (Ateka *et al.*, 2003).

2.7 Virus transmission

Different viruses may be transmitted mechanically, by grafting, through seed, by vectors or a combination of the above modes (Cadena-Hinojosa and Canpbell 1981). Diseases of sweet potatoes of viral etiology have been shown to be insect, graft and mechanical transmissible or combination of above modes (Clark and Moyer, 1988). However, some viruses mode of transmission have not been clearly understood because some viruses

share the same vectors and others occur in more than one serotype or strain (Winter *et al.*, 1992). C6, SPCaLV, SPLV, SPRSV, SPMSV and SPCFV are yet to been known whether there are transmitted by any vector, while CMV and SPFMV have been known to be transmitted by aphids and SPLCV, SPCSV and SPMMV are transmitted by white flies. (Clark and Moyer, 1988; Moyer and Larsen, 1991; Lenne, 1991; Carey *et al.*, 1996). Virus infected vines and insect vectors remain the major source of transmission, perpetuation and dissemination of viruses (Clark and Moyer, 1988)

SPCSV is limited to the phloem and is transmitted by the whitefly *Bemisia tabaci* in a semi-persistent manner (Karyeija *et al.*, 2000). It can acquire or transmitt SPCSV with access period of 1 hour or less and can remain viruliferous for 24 -28 hours (Larsen *et al.*, 1991). In Africa SPCSV can be transmitted by both the cassava specific biotype of *B.tabaci* (Burban *et al.*, 1992) and *B. tabaci* naturally colonising sweet potato (Cohen *et al* 1992; Gibson *et al.*, 1998a). *B. afer* has also been reported in Africa on sweet potato (Legg et al 1994) but no tests of it's ability to transmit SPCSV have been reported. SPCSV can also be transmitted by a whitefly in a second genus the banded winged whitefly *T. abutilonea*, although less efficiently than by B. tabaci (Gibson and Aritua 2002). The virus is not transmitted by mechanical inoculation or by contact between plants. It can also be transmitted by grafting (Brunt *et al.*, 1996). Because the virus infects sweet potato plants systemically, it is disseminated in infected vegetative cuttings used as propagules. Plants grown from such propagules are primary sources of infection in crops.

SPFMV is transmitted in a non-persistent manner (Pozzer *et al.*, 1993, Ames *et al.*, 1996) and most efficiently by the green peach-potato aphid (*Myzus persicae)*, the

cotton ahid (*Aphis Gossipii*) and less efficiently by the groundnut aphid (*A craccivora)* and *Lipaphis erusioni* (Wanbugu 1991; Schaefers and Terry 1976). The virus is also transmissible by stem and tuber core grafting, but the probability of seed transmission is low (Wolters et al 1990). It has been transmitted to *I.nil* by sap inoculation from symptomatic tissue (Campbell *et al.*, 1974; Moyer and Cali 1985). SPFMV is perpetuated from one cropping cycle to the next through planting materials (vines) which facilitates it's movement and multiplication, since it can also transmitted through vegetatively propagation.

SPMMV is transmitted by whiteflies (*Bemisia tabaci*) and biotypes of *Bemisia tabaci* may differ in their transmission ability (Hollings *et al.*, 1976a, b). The virus can be easily transmitted mechanically to susceptible sweet potato clones and test plants in the *Solanaceae* and *Convolvulaceae* families. It is also transmitted by grafting (Sheffield, 1957a,b; Hollings *et al.*, 1976a, b).

2.8 Management of the viruses that affect sweet potato

Virus management is based on the fact that virus diseases are not curable and virus infections must therefore be prevented. Use of chemicals to control the spread of vectors depends on the type of virus transmission involved. In non-persistent transmission of viruses such as SPFMV, aphids transmit the virus faster than the common aphicides can react to kill the vector hence insecticides can only slightly reduce non-persistent virus transmission but cannot prevent it. In persistent transmission of viruses such as with SPMMV, the inoculation periods is long enough to allow pesticides to control vector and therefore greatly reduce virus spread within the field though it cannot control infections

from outside by migrating insect (Walkey, 1991). However, over reliance on insecticide use has caused toxicity to the environment, destruction of natural balance, may result in insecticide resistance (Heathcote, 1973) and the cost of sprays may be uneconomical for use in subsistence farming. These have led to a change of concept supporting alternative more environmentally safe means of control.

Therefore management of virus that affects sweet potato production involves cultural practices such as selection of disease free planting material. Virus-free planting materials were evaluated for the control of sweet potato virus diseases in China and virus-free planting materials out yielded farmers' seed by at least a factor of 2 (Beetham and Mason, 1992). Efficient virus detection technology is essential to produce virus-free planting materials. Destruction and removal (roguing) of diseased plants especially in young crops, can reduce the development of virus population and/or inoculum sources (Dent, 1995). Crop rotation provides a cheap and effective means of crop protection against viruses in addition to its agronomic benefits. This is particularly against pests, which are relatively host specific. If infected tubers from previous crop sprout in the same field or in areas adjacent to a new crop they serve as sources of virus inoculum (Karyeija *et al.*, 1998). Since spread of some viruses such as SPCSV is closely related to the proximity of diseased sweet potato crop (Aritua *et al.*, 1998), even small increases in distance of new plantings from old crops and destroying crop residues should provide worthwhile benefits (Ateka *et al.*, 2001 Gibson and Aritua, 2002). Sweet potato may also be intercropped , often with fast-growing crops such as maize and beans (Kapinga *et al.*, 1995) intercropping especially with maize, may provide some control of viruses through decreasing vector number due to unfavourable micro climate (Ndunguru and Aloyce,

2000). Quarantine can also be employed to exclude new virus strains into a region or country. For instance quarantine can be a good measure for control when observed especially for viruses that occur in more than one geographically distinct serotypes as is the case with SPCSV

One of the main means of controlling virus diseases is through use of resistant cultivars (Gibson et al., 1997; Karyeija *et al.*, 1998). Since viruses cause the most important disease of sweet potato crop in sub-saharan Africa (Geddes 1990) breeding for resistance against viruses is a priority. SPVD resistant cultivars and those resistant to SPFMV have been identified in West Africa and Uganda respectively. There is currently great interest in the possibility of producing virus-resistant transgenic plants (Qaim, 1999) although none is yet available for sweet potato production in Africa. However virus resistance only slows down but does not prevent virus spread in vegetatively propagated crops (Fargette and vie, 1995). Vector resistance, other than resistance in sweet potato to viruses, which is an important component of resistance to both semi as well as non- persistent transmitted viruses, could be pursued.

CHAPTER 3

MATERIALS AND METHODS

3.1 Experimental site

The experiment was carried out at the University of Nairobi, Kabete Field Station located at latitude $1^0$15'S and longitude 36^0 46'E and at an altitude of 1814 m. The soils consist of well-drained, deep reddish brown to dark red friable clay with acidic humic top soil (humic nitosol) and a pH range of 5.0-5.4 (Siderius and Muchema, 1977). In Kabete, the mean maximum and minimum temperatures are 23°C and 13°C respectively. It receives an average rainfall of about 1000 mm per year. The rainfall is bimodal and long rains are received between March and June while the short rains fall between October and December. The rainfall and sunshine hours received at the experimental site during the investigation period are shown in Appendix 1.

3.2 Source of planting material and virus isolates

The experimental plant material comprised of three popular local sweet potato varieties namely Bungoma, Kemb 10, and Ex-shimba Hills. They were collected from farmers' fields during a national survey (Ateka *et al.*, 2003). Prior to this study, the materials were maintained in an insect-proof screenhouse at Kabete field station. The field-derived materials were assayed for SPFMV, SPMMV, SPCSV, SPMSV, SPCaLV, SPLV, SPCFV and C-6. Triple antibody sandwich enzyme linked immunosorbent assays (TAS-ELISA) technique described by Gibson *et al.*, (1997) was used to assay for SPCSV and SPMMV while the nitrocellulose membrane enzyme linked immunosorbent assay (NCM-ELISA) was used to assay for SPFMV, SPMSV, SPCaLV, SPLV, SPCFV and C-6. The

TAS- and NCM-ELISA techniques are shown in Figure 1a and 1b respectively. Scions were obtained from vines, which tested negative for all viruses and were grafted onto *I. setosa* indicator plants. Rootstocks were assayed for the viruses' four weeks later. Virus-free materials were those, which tested, negative for all viruses in ELISA and did not induce symptoms when grafted to *I. setosa* indicator plants.

Planting materials infected with SPFMV, SPCSV, SPMMV or a combination of SPFMV + SPCSV were obtained by side grafting (Beetham and Mason, 1992) virus-free plants with scions from plants infected with single virus as identified by ELISA. The graft union was then wrapped in parafilm and the whole plant was covered with a moistened plastic bag for seven days to minimize moisture loss through transpiration. All the planting materials were maintained in an insect proof screen house. Four weeks after grafting, rootstocks were assayed for viruses to confirm success of virus transmission. Positive plants were multiplied to provide planting material for the field experiments. Planting material consisting of leafy stem cuttings of approximately 30 cm were excised and kept in a cool screenhouse for three days for hardening off.

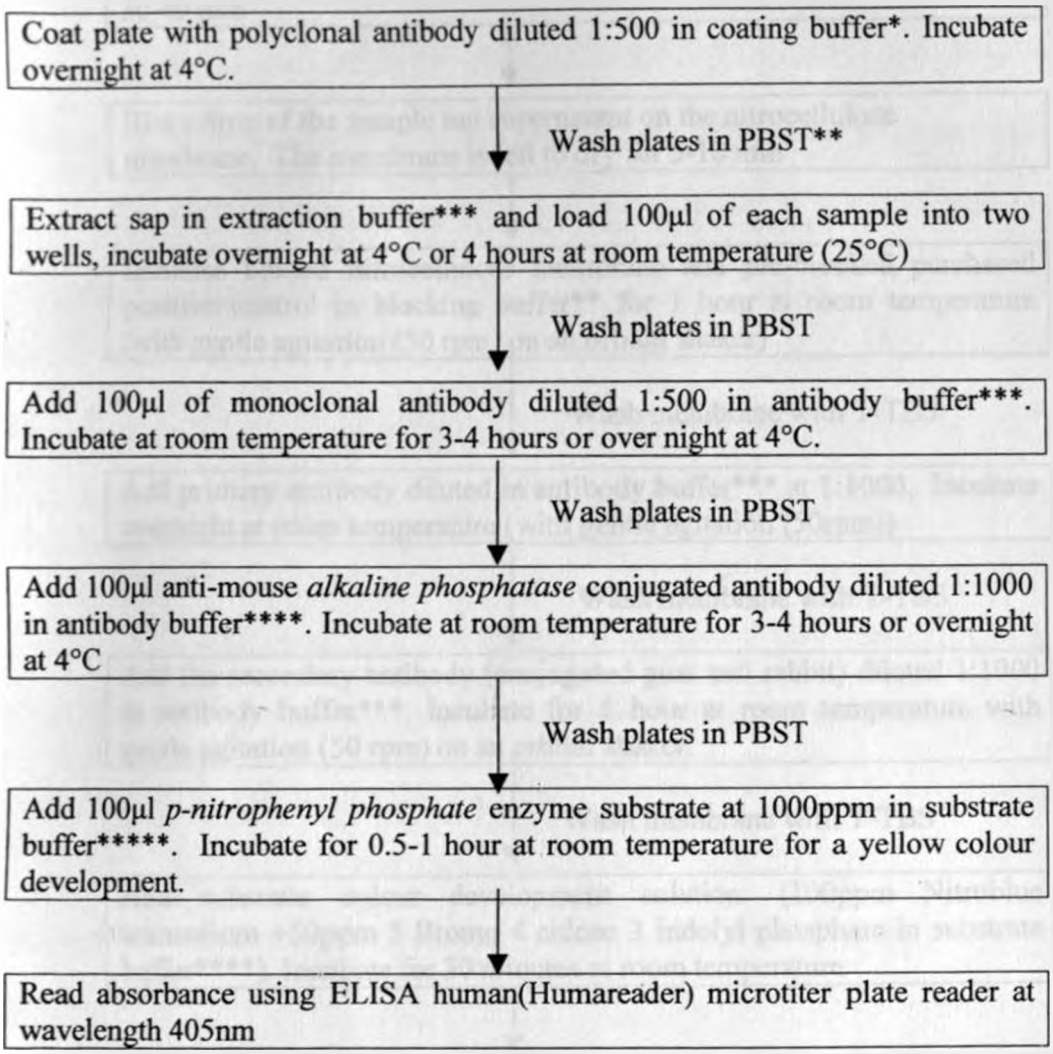


Figure 1a The steps followed in the Triple antibody sandwich enzyme linked immunosorbent assays.

*Coating buffer consisted of 1.6g sodium carbonate (Na_2C0_3) and 3g sodium hydrogen carbonate ($NaHC0_3$) in 1litre of distilled water.

**Phosphate -buffered saline (PBST) consisted of 8g sodium chloride (NaCl), 2.9g sodium hydrogen orthophosphate (Na_2HPO_4 $2H_2O$), 0.2g potassium chloride (KCl), 0.2g potasiun orthophosphate (KH_2P0_4) and 0.5ml tween-20 in 1 litre of distilled water.

***Sap extraction buffer: 20g polyvinylpyrrolidone was added to 1 litre of PBST

****Antibody buffer consisted of 20g polyvinylpyrrolidone, 25g dry milk powder in 1 litre PBST.

*****Substrate buffer consisted of 114ml Diethanolamine per litre of distilled water (pH was adjusted to 9.8 using HCl).

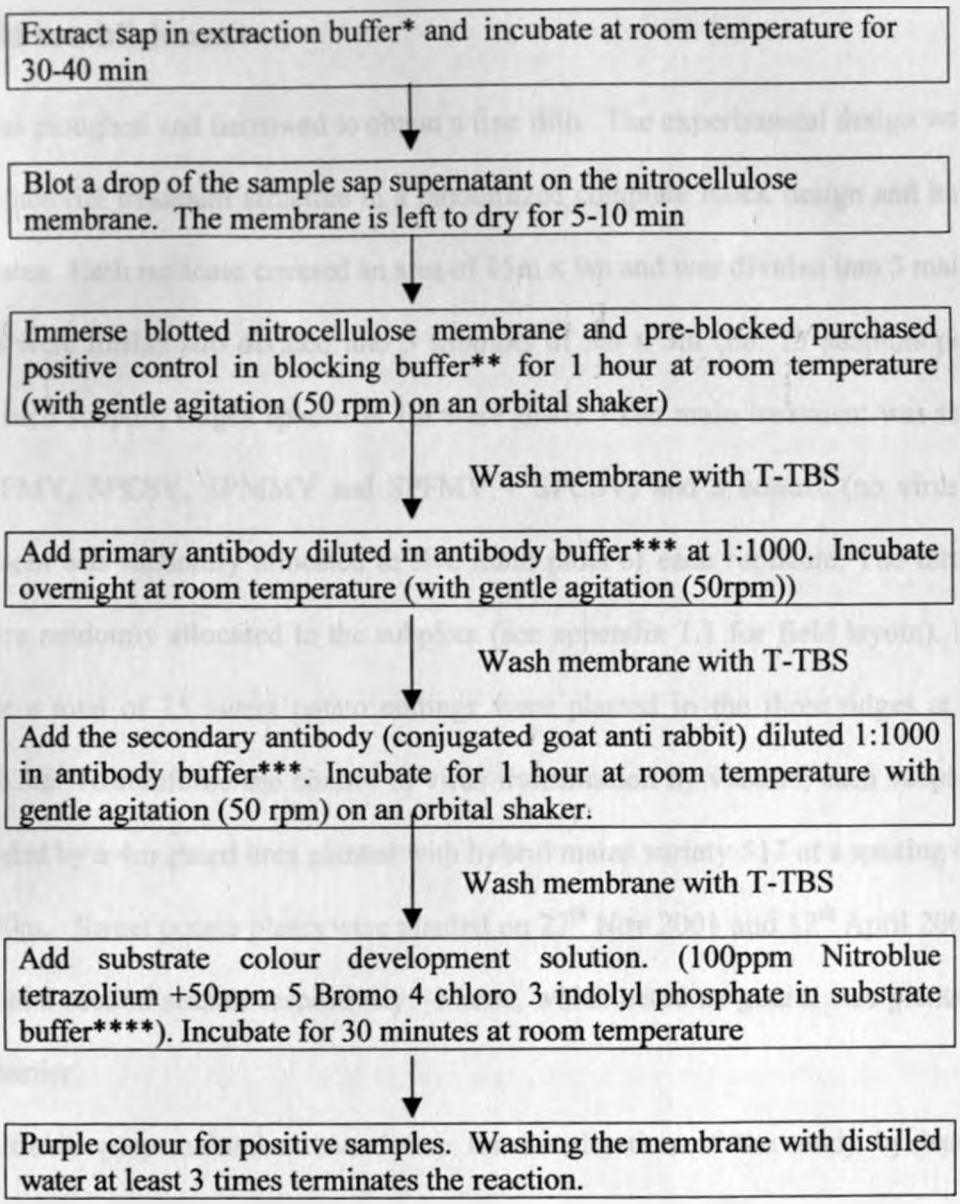


Figure 1b The steps followed in the Nitrocellulose membrane enzyme linked immunosorbent assays.

Tween-Tris buffered saline (T-TBS) consist of 2.42g Tris, 29.22g NaCl, 2.5ml HCl (18.5%) and 0.5ml Tween 20 in 1litre distilled water.

*Extraction buffer consist of 2g sodium sulfide (Na_2SO_3) per litre of TBS (T-TBS minus Tween 20

**Blocking buffer consist of 20g powdered cow milk and 20g triton in 1 litre TBS

***Antibody buffer consist of 20g powdered cow milk in 1 litre TBS

****Substrate buffer consist of 12.1g Tris, 5.8g NaCl, 1g Magnesiun chloride (MgCl) and 2ml HCl (18%) in 1 litre of distilled water

3.3 Field establishment

The field was ploughed and harrowed to obtain a fine tilth. The experimental design was a split plot factorial treatment structure in a randomized complete block design and had three replicates. Each replicate covered an area of 15m x 9m and was divided into 5 main plots which were further sub divided into 3 subplots of 3m x 3m (i.e. 15 subplots per block). In each subplot, ridges spaced at 1m were made. The main treatment was the viruses (SPFMV, SPCSV, SPMMV and SPFMV + SPCSV) and a control (no virus). Virus treatment was randomly allocated to five main plots of each replicate. The three varieties were randomly allocated to the subplots (see appendix 1.1 for field layout). In each subplot a total of 15 sweet potato cuttings were planted in the three ridges at a spacing of 0.5m. To reinforce the control of virus transmission by vectors, each subplot was surrounded by a 4m guard area planted with hybrid maize variety 513 at a spacing of 0.75m x 0.30m. Sweet potato plants were planted on 27th Nov 2001 and 12th April 2002 for the first and second season respectively. Maize, which acted as guard, was planted two weeks earlier.

The fields were maintained weed free for the duration of the study by hand hoeing. Malathion (Malathion 50 EC) and dimethoate (Degor 40 EC) insecticides were sprayed at recommended rates and intervals to control insect vectors. The plants were assayed for presence or absence of viruses using TAS-ELISA (for SPCSV and SPMMV) and NCM-ELISA (for SPFMV) two months after planting.

3.4 Data collection and analysis

Data on disease severity, photosynthetically active radiation (PAR) intercepted by the plant canopy, total fresh vine weight, fresh marketable and unmarketable tuber weight,

total number of marketable and unmarketable tubers, samples of leaf shoot and tuber dry weight were collected.

3.4.1 Disease severity

The disease severity assessment was made fortnightly starting from one and half months after planting and continued up to harvesting. A scale ranging from 1-5 was used in assessing disease severity (Ateka *et al.*, 2003). The ratings and corresponding symptoms are as shown below in Table 1. Ten randomly selected plants on each sub-plot were scored for disease severity and finally a mean score per sub-plot was calculated.

3.4.2 Photosynthetically active radiation intercepted by plant canopy

Photosynthetically active radiation (PAR) intercepted by the plant canopy was determined using sunfleck light ceptometer (SF-80 Decagon, Pulman. Washington). Readings were taken between 1200 and 1300 hrs every fortnight starting two months after planting. By holding the ceptometer horizontally, incident PAR reading was taken above the plant canopy. Then, by holding the ceptometer perpendicular to the sweet potato rows an average of ten readings were taken below the plant canopy of 10 randomly sampled plants. The difference between the PAR above the plant canopy and that below the canopy was the amount of solar radiation intercepted by the crop canopy. This was expressed as a percentage fraction (F) of radiation above the canopy and calculated using the formula below (Squire, 1990)

$$\% F = \frac{\text{Above canopy average PAR - below canopy average PAR X100}}{\text{Above canopy average PAR}}$$

Table 1 Scale for disease severity assessment on sweet potato plants infected with viruses.

Rating	Symptoms manifested
1	No visible symptoms, no purpling/yellowing, no mosaic, no distortion and no stunting.
2	Very mild symptoms on leaves, few leaves purpling/yellowing or mosaic, little distortion of leaf shape, apparent but negligible stunting.
3	Moderate symptoms of purpling/yellowing or mosaic on leaves, moderate distortion of leaves shape and moderate stunting.
4	Severe symptoms of purpling/yellowing or mosaic on leaves, severe distortion of leaves with reduced size, plant partially stunted (very short internodes) but still apparently growing.
5	Very severe symptoms of purpling/yellowing or mosaic on leaves, severe leaf distortion, reduced leaf size, plant severely stunted (stem extension more or less stopped).

Source: Ateka *et al.*, (2003)

3.4.3 Determination of leaf area and specific leaf weight

Specific leaf area (SLA), an index of leaf structures, was determined using 30 fully expanded leaves selected from a representative plant in each sub-plot. A cork borer, 1cm in diameter, was used to excise 120 discs from the leaves. The leaf discs were oven dried to a constant mass. The specific leaf area (SLA), which is leaf area to dry mass ratio, was then determined using the area of the discs divided by its dry mass. Specific leaf weight (SLW) which is weight per unit area of a leaf and is directly related to leaf thickness was calculated using the 120 leaf discs. SLW was then calculated by dividing leaf disc mass by leaf disc area. Leaf area index (LAI), which is the ratio of the total leaf area of the crop to the ground area, was calculated indirectly using SLA and total leaf mass. To calculate total dry leaf mass, one representative plant from each subplot was selected randomly and all its leaves detached, oven dried to a constant mass and weighed. The equation below was used for the determination of total leaf area (Mburu, 1996)

$$LA_{total} = LW_{total} * \frac{LA_{disc}}{LWt_{disc}}$$

Where:

LA_{total} = total leaf area (m^2)

LW_{total} = total plant leaf mass (g)

LA_{disc} = leaf disc area (m^2)

LWt_{disc} = leaf discs mass (g)

3.4.4 Harvest data

Both experiments were harvested four and half months after planting. At harvest, the weights of all fresh vines were recorded in each sub-plot. The tubers were placed into one of the two categories namely marketable tuber (above 50g) and unmarketable (50g or less). Numbers and weights of marketable and unmarketable tubers were recorded. Harvest index, which is a good indicator of economic yield was calculated. This is the weight of economical part of the plant (marketable tuber in this study) divided by total biomass. To calculate harvest index, one plant was randomly selected from each sub-plot and its shoots and tubers separated. The plant parts were then oven dried at 80^0C to a constant mass and weighed. Harvest index was then calculated as a ratio of total tuber mass (Evans, 1975) to total biomass as given below.

$$\text{Harvest index} = \frac{\text{Marketable tuber dry weight}}{\text{Total biomass}}$$

Where;

Total biomass =Total vine dry weight + total tuber dry weight

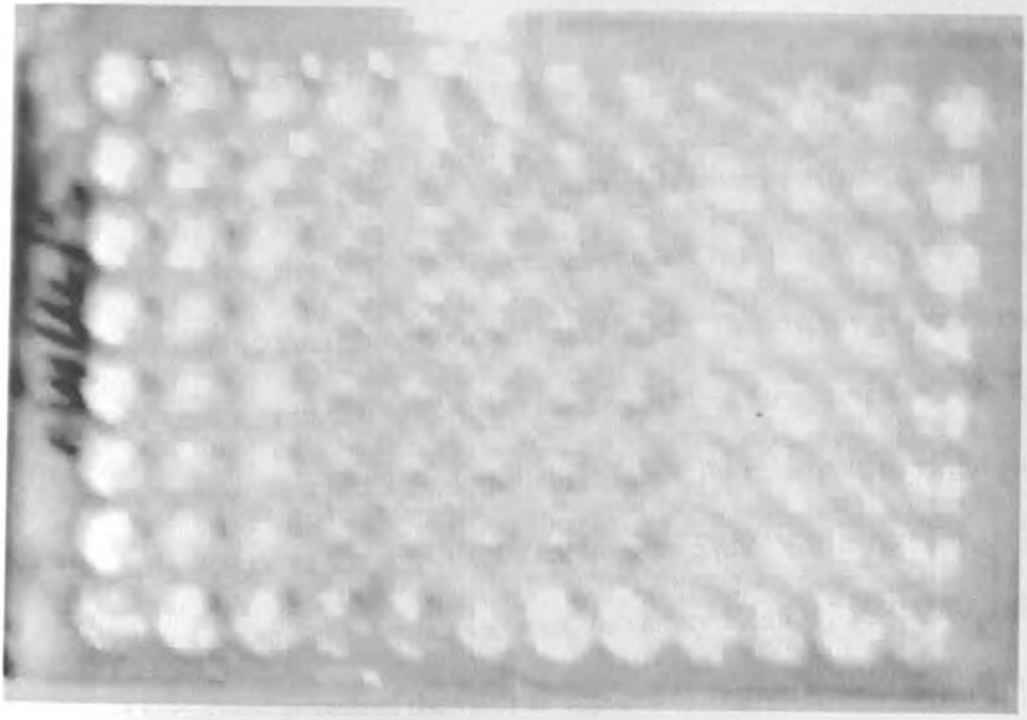
All data was subjected to analysis of variance using Genstat release 6.0.

CHAPTER 4

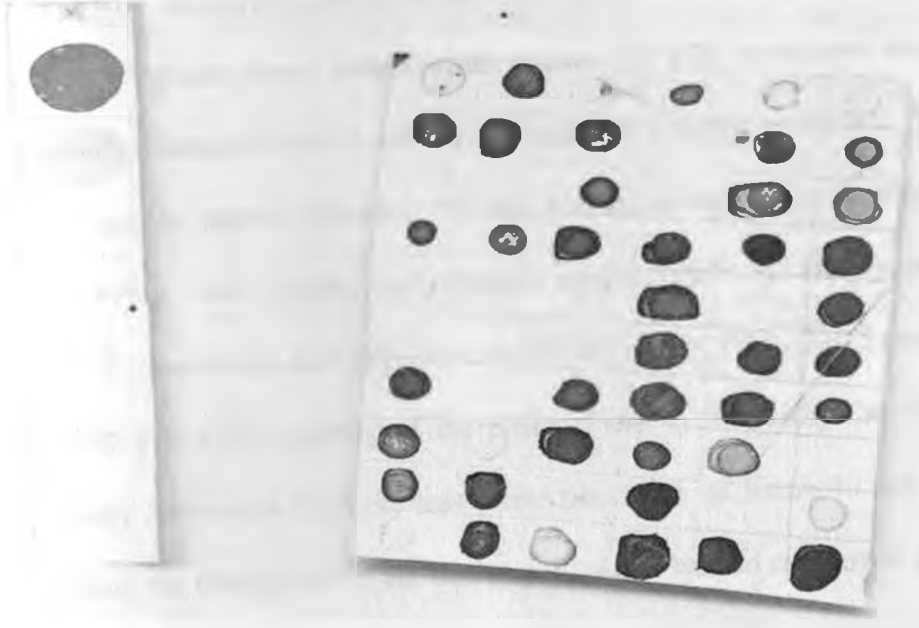
RESULTS

4.1 Planting materials and virus isolates

The viruses detected in the field-derived material in decreasing order of frequency of occurrence were SPFMV, SPCSV SPMMV SPCFV and SPCaLV. The viruses were found to occur as individual as well as in complexes. Symptoms on grafted *I.setosa* took 2 to 3 weeks to appear. Infection with SPCSV caused stunting of the *I.* setosa plant and mild mosaic (yellowing and/or vein yellowing in leaves). Infection with SPFMV on *I. setosa* caused typical symptoms of potyvirus, which include chlorotic mottle or vein clearing and distortion of leaves. SPMMV produced chlorotic vein banding, mottling and distortion of leaves. SPVD caused very severe stunting and chlorosis on leaves of *I.setosa.* The success of virus transmission on the grafted plants was quite high (60 %) and this gave sufficient planting material for the field trials. The ELISA assays results showed that the status of the viruses did not change throughout the field experiments and within varieties. Although there were few negligible cases (0.08%) such as block 1 Ex-shimba infected with SPMMV but contaminated with SPFMV (Appendix 3). Concentration of SPFMV when alone in the sweet potato plants was not within detectable levels (Appendix 3) but in combination with SPCSV and when grafted to *I. setosa* SPFMV concentration could be detected by NCM -ELISA test. All the plants with SPVD showed strong positive results for SPFMV. Samples of Elisa assay results are shown in plate 1



A



B

Plate 1:An enzyme linked immunosorbent assay results

(A) Test plate. The yellow colour indicate a positive reaction in TAS-ELISA reaction for *sweet potato mild mottle ipomovirus*

(B) Test nitrocellulose membrane and a positive strip (left). The purple colour indicate a positive reaction in NCM-ELISA for *sweet potato feathery mottle potyvirus*

4.2 Disease severity

Disease severity varied between virus treatments, within varieties and during the growth of the crop. Infection with SPCSV caused stunting and purpling of the middle leaves in variety Kemb 10 throughout both seasons (Plate 2). The severity of symptoms induced by SPCSV gradually increased up to the third month and then started to decline (Figure 2a). In variety Bungoma and Ex-shimba hills, SPCSV infection caused slight stunting during the early stages of the growth. Infection with SPFMV did not induce any apparent symptoms on any of the three varieties. In variety Kemb 10, SPMMV infection caused rapid horizontal growth of the vines as opposed to vertical growth typical of Kemb 10 but otherwise caused no apparent foliar symptoms on any of the varieties.

Typical sweet potato virus disease (SPVD) symptoms were manifested on varieties Bungoma and Kemb 10 infected with SPCSV + SPFMV. These were: severe stunting of plants, distortion of leaf lamina, narrowing of lamina (strap-like), leaf feathering, vein clearing and chlorotic mosaic (Plate 3). The symptoms observed on variety Ex-shimba hills infected with SPFMV + SPCSV included purpling of the middle leaves and slight stunting of the plants (Plate 4). Generally the SPVD symptoms on variety Ex-shimba hillswere less severe than those on Kemb 10 and Bungoma varieties. During the third month of the first season, plants seemed to recover, the SPVD symptoms disappearing in most of the plants as they started growing vigorously, especially those of variety Bungoma as depicted in Figure 2b. It is worth noting that during that period there was a favourable weather condition for the crop.

A

B

Plate 2: Variety Kemb 10 plants at 120 days after planting (A) healthy, (B) infected with SPCSV note the purpling of the middle leaves due to SPCSV infection.

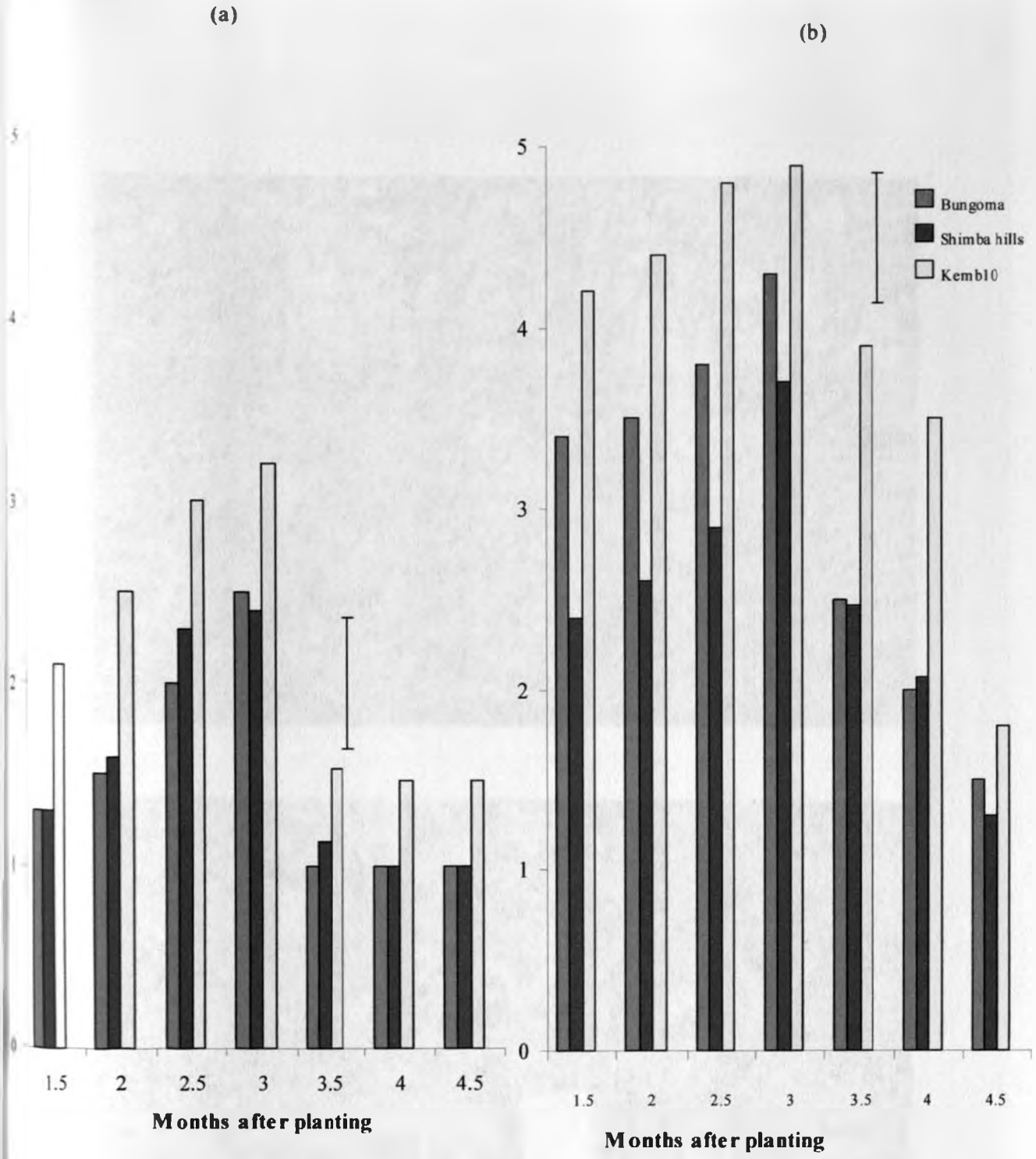


Fig 2 Mean disease severity score for the sweet potato varieties infected with:

(a) Sweet *potato chlorotic stunt crinivirus* and

(b) *Sweet potato chlorotic stunt crinivirus* plus *sweet potato feathery mottle potyvirus* for the three sweet potato varieties.

A

B

Plate 3:Variety Bungoma plants at 120 days after planting (A) healthy, (B) infected with SPFMV+SPCSV note the vein clearing, leaf strapping and chlorosis due to the dual infection.

A

B

Plate 4: Variety Ex-shimba hills plants at 120 days after planting (A), healthy (B) infected with SPFMV+SPCSV. Note the purpling of leaves.

4.3 Photosynthetically active radiation intercepted by sweet potato plant canopy.

Virus infection was the only treatment which had a significant effect on PAR interception by the plant canopy. Infection with SPFMV and SPMMV had no significant effect on PAR interception but SPCSV reduced PAR interception by 18% when compared to virus-free plants (Figure 3). Dual infection with SPCSV + SPFMV reduced PAR interception by 75%. The trend was similar throughout the sample periods and in both seasons.

4.4 Effect of viruses on vegetative plant growth

Data on specific leaf weight (SLW), specific leaf area (SLA), leaf area index (LAI) and vine yield was collected. The virus treatment effect was significant ($P<0.01$) for SLW in both seasons. During the second season, infection with SPMMV and SPFMV resulted in about 20% and 27% increase in SLW respectively when compared with virus-free plants. Infection with SPCSV resulted in a slight (5%) increase in SLW while dual infection caused a significant reduction (15%). This trend was observed in the both seasons (Table 2).

Virus infection had a significant ($P<0.05$) effect on SLA of the sweet potato varieties. Dual infection with SPFMV + SPCSV reduced SLA by 42% when compared to virus-free plants in season one. However no significant difference was detected in SLA between virus-free plants and those infected with single viruses (Table 2). This trend was observed in both seasons.

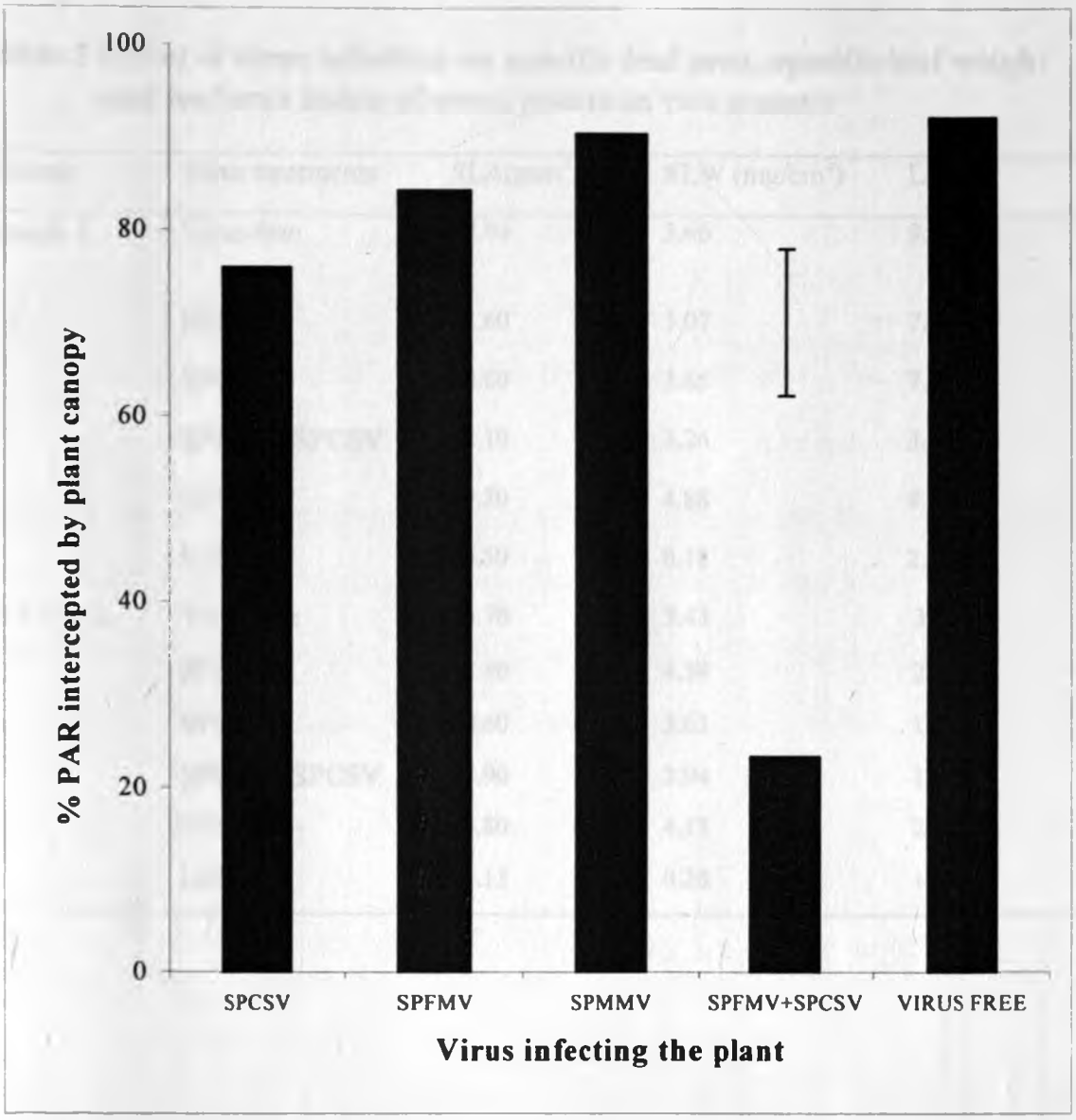


Figure 3 Percentage photosynthetically active radiation intercepted by sweet potato plant canopy when infected with three sweet potato viruses at 105 days after planting in season one.

Table 2 Effect of virus infection on specific leaf area, specific leaf weight and leaf area index of sweet potato in two seasons

Season	Virus treatments	SLA(mm^2/g)	SLW (mg/cm^2)	LAI
Season 1	Virus-free	58.90	3.66	9.96
	SPFMV	53.60	5.07	7.29
	SPCSV	50.60	3.85	7.54
	SPFMV+SPCSV	34.10	3.26	3.44
	SPMMV	59.30	4.88	8.18
	LSD	16.50	0.18	2.95
Season 2	Virus-free	55.70	3.43	3.21
	SPFMV	53.40	4.39	2.96
	SPCSV	50.60	3.63	1.76
	SPFMV+SPCSV	24.90	2.94	1.07
	SPMMV	54.80	4.13	2.42
	LSD	17.13	0.20	1.26

Virus infection had a significant ($P<0.05$) effect on the leaf area index (LAI) of the sweet potato varieties. Dual infection with SPFMV + SPCSV reduced LAI by 66% when compared to virus-free plants during the first season. However, no significant difference was detected in LAI between virus-free plants and those infected with single viruses (Table 2). This trend was observed in both seasons.

Virus treatment had a significant effect ($P<0.01$) on vine yield. The greatest vine yield (120 tonnes/ha) was observed in virus-free plants. Infection with SPCSV significantly reduced vine yield by 40% (during the first season) but SPFMV and SPMMV had no significant effect. During the first season, the least vine yield (30.7 tonnes/ha) was observed in plants dually infected with SPFMV + SPCSV (Figure 4). A similar trend was observed during the second season though yields were less.

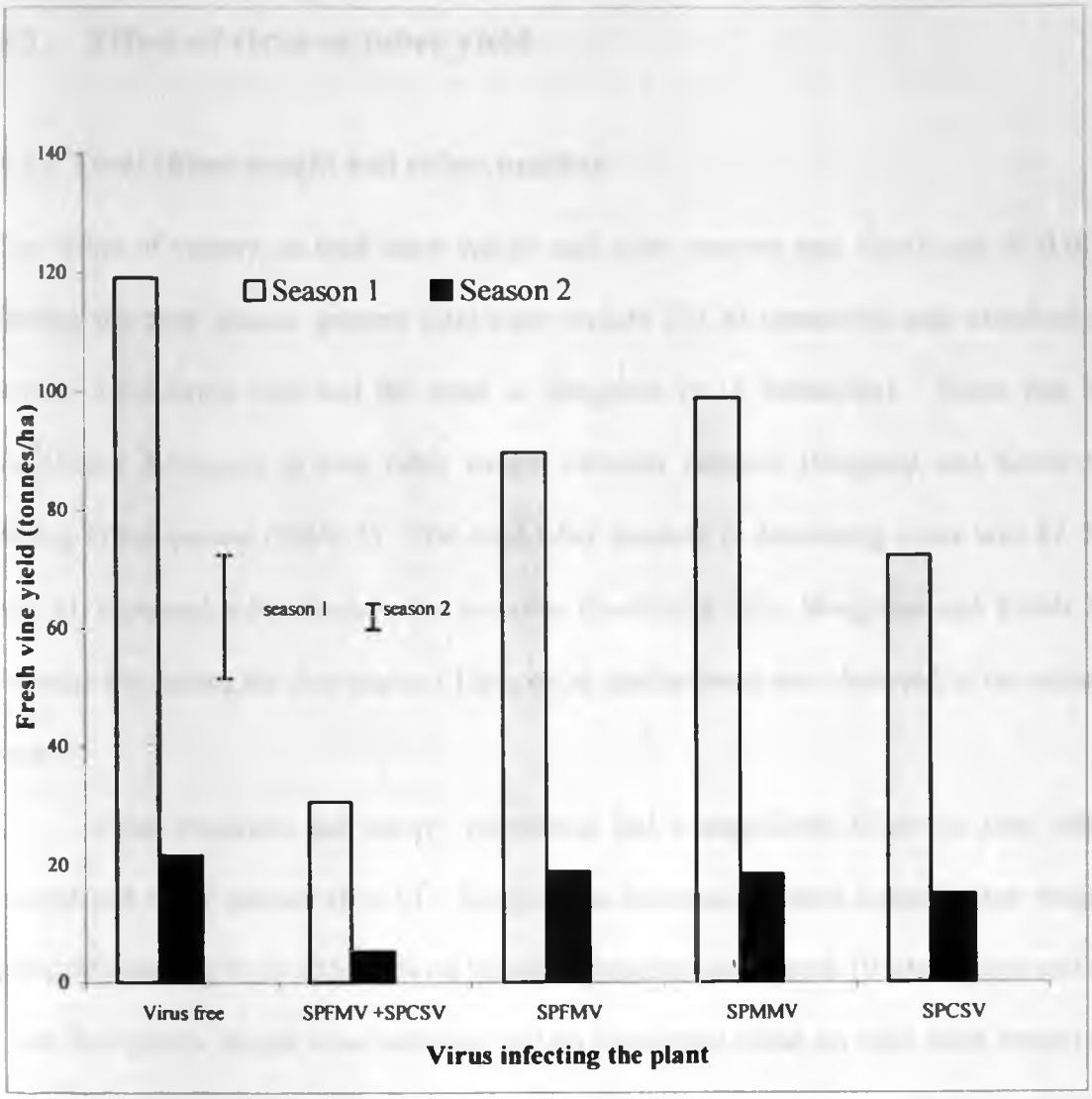


Figure 4 Effect of three viruses on fresh vine yield of sweet potato in Kenya

4.5 Effect of virus on tuber yield

4.5.1 Total tuber weight and tuber number

The effect of variety on total tuber weight and tuber number was significant ($P<0.01$). During the first season, greatest total tuber weight (23.30 tonnes/ha) was observed in variety Ex-shimba hills and the least in Bungoma (6.16 tonnes/ha). There was no significant difference in total tuber weight between varieties Bungoma and Kemb 10 during either season (Table 3). The total tuber number in decreasing order was 87, 57 and 41 thousand tubers/hectare for varieties Ex-shimba hills, Bungoma and Kemb 10 respectively during the first season (Table 4). A similar trend was observed in the second season.

Virus treatment and variety interaction had a significant effect on total tuber weight and tuber number ($P<0.01$). Single virus infection resulted in total tuber weight reduction ranging from 50 to 95% on varieties Bungoma and Kemb 10 when compared to virus-free plants. Single virus infection had no significant effect on total tuber weight in variety Ex-shimba hills during both seasons. Dual infection with SPFMV + SPCSV significantly reduced total tuber weight (85-96%) in all the three varieties. A similar trend was observed during the two seasons but a lower total tuber weight was obtained in the second season (Table 3).

Single virus infection resulted in total tuber number reduction ranging from 40 to 90% on varieties Bungoma and Kemb 10 when compared to virus-free plants. Single virus infection had no significant effect on total tuber number in variety Ex-shimba hills during both seasons. Dual infection with SPFMV + SPCSV significantly reduced total

Table 3 Virus infection and variety effects on total tuber yield (tonnes/ha) of three sweet potato varieties in two seasons

Season	Virus Variety	Total tuber yield (tonnes/ha) Bungoma	Kemb 10	Ex-shimba hills
Season 1	Virus-free	13.9	23.3	30.5
	SPFMV	5.3	2.1	26.3
	SPCSV	3.5	6.3	27.8
	SPMMV	7.0	1.2	27.4
	SPFMV + SPCSV	1.2	0.9	4.5
	Variety mean	6.2	6.7	23.3
	$LSD_{0.05}$ variety	2.4		
	$LSD_{0.05}$ virus x variety	4.9		
Season 2	Virus-free	7.0	6.4	14.4
	SPFMV	4.6	1.6	11.7
	SPCSV	2.7	2.3	9.2
	SPMMV	5.1	1.8	9.6
	SPFMV+SPCSV	0.8	0.5	2.4
	Variety means	4.1	2.5	9.5
	$LSD_{0.05}$ for variety	2.0		
	$LSD_{0.05}$ for virus x variety	2.2		

Table 4 Effect of virus treatment and variety on number (in '000/ha) of total tubers of three sweet potato varieties in two seasons

Season	Variety / Virus	Number of total tubers		
		Bungoma	Kemb 10	Ex-shimba hills
Season 1	Virus free	105	105	120
	SPFMV	67	38	96
	SPCSV	37	40	97
	SPMMV	61	12	96
	SPFMV+SPCSV	18	10	29
	Variety means	57	41	87
	$LSD_{0.05}$Variety	9.89		
	$LSD_{0.05}$virus x variety	24.53		
Season 2	Virus free	50	56	79
	SPFMV	46	32	73
	SPCSV	27	24	66
	SPMMV	49	21	70
	SPFMV+SPCSV	11	10	23
	Variety means	37	29	62
	$LSD_{0.05}$variety	4.99		
	$LSD_{0.05}$Virus x variety	13.28		

tuber number (76-92%) in all the three varieties. A similar trend was observed during the two seasons but a lower total tuber weight was obtained in the second season (Table 4).

4.5.2 Marketable tuber weight and marketable tuber number

The effect of variety on marketable tuber weight and marketable tuber number was significant ($P<0.01$). Greatest marketable tuber weight (21.25tonnes/ha) was observed in variety Ex-shimba hills but was least in variety Kemb 10 (1.73 tonnes/ha) (Table 5). The highest number of marketable tubers was observed in variety Ex-shimba hills and there was no significant difference in number of marketable tubers on varieties Kemb 10 and Bungoma in both season (Table 6).

Virus treatment and variety interaction had a significant effect on marketable tuber weight and number. Single virus infection resulted in a 58- 98% reduction in marketable tuber weight on varieties Bungoma and Kemb 10 when compared to those of virus-free plants. Infection with single viruses had no significant effect on weight of marketable tubers on variety Ex-shimba hills during either season. Marketable tuber weight loss ranging from (85-98%) was noted in plants dually infected with SPFMV+ SPCSV (in all the three varieties) when compared to virus free plants during both seasons (Table 5). This same trend was observed in both seasons although the marketable tuber weight was lower in the second season (Table 5)

Single virus infection resulted in a reduction in number of marketable tubers on varieties Bungoma and Kemb 10 when compared to those of virus-free plants. Infection with single viruses had no significant effect on number of marketable tubers on variety Ex-shimba hills during either season. The number of marketable tubers in dually infected plants was reduced in all the three varieties (Table 6).

Table 5 Virus infection and variety effects on marketable tuber yield (tonnes/ha) of three sweet potato varieties in two seasons

Season	Virus / Variety	Marketable tuber yield (tonnes/ha) Bungoma	Kemb 10	Ex-shimba Hills
Season 1	Virus-free	11.9	21.4	27.8
	SPFMV	3.9	0.7	24.4
	SPCSV	2.5	5.4	25.7
	SPMMV	5.0	0.5	25.0
	SPFMV + SPCSV	0.5	0.5	4.3
	Variety mean	4.8	5.7	21.3
	$LSD_{0.05}$ variety	2.5		
	$LSD_{0.05}$ virus x variety	5.3		
Season 2	Virus-free	5.4	5.4	12.4
	SPFMV	3.2	0.8	10.0
	SPCSV	1.9	1.6	8.4
	SPMMV	3.3	0.8	8.5
	SPFMV + SPCSV	0.3	0.2	2.2
	Variety means	2.8	1.7	8.3
	$LSD_{0.05}$ variety	0.9		
	$LSD_{0.05}$ virus x variety interaction	2.0		

Table 6 Effect of virus treatment and variety on number (in '000/ha) of marketable tubers of three sweet potato varieties in two seasons.

Season	Virus / Vareity	Number of marketable tubers		
		Bungoma	Kemb 10	Ex-shimba hills
Season 1	Virus free	63	47	65
	SPFMV	22	3	51
	SPCSV	13	18	47
	SPMMV	22	4	48
	SPFMV + SPCSV	4	2	13
	Variety means	25	17	45
	$LSD_{0.05}$ variety	9		
	$LSD_{0.05}$ virus x variety	21		
Season 2	Virus free	29	37	43
	SPFMV	10	8	37
	SPCSV	12	8	36
	SPMMV	16	7	42
	SPFMV + SPCSV	3	2	9
	Variety means	13	12	34
	$LSD_{0.05}$ variety	5.99		
	$LSD_{0.05}$ virus x variety	9.75		

a

b

c

Plate 5: Tubers harvested from
a) Variety Bungoma infected with SPCSV + SPFMV(left)and control(right)
b) Variety Shimba hills infected with SPCSV + SPFMV (left)and control(right)
c) Variety Kemb 10 infected with SPCSV + SPFMV(left)and control(right)

Most tubers harvested from dually infected plants were small in size, mainly unmarketable (Plate 5a,b & c). The same trend was observed in both seasons although the number of marketable tubers were less in the second season than in the first (Table 6)

4.5.3 Unmarketable tuber weight and unmarketable tuber number

Single virus infection with SPFMV, SPMMV and SPCSV on varieties Kemb 10 and Bungoma resulted in a higher proportion of unmarketable number tubers, about 60% of the total tubers number were unmarketable during the first season. A similar trend was observed during the second season. In plants dually infected with SPFMV + SPCSV, the average size of tubers were half that of virus-free plants and a higher proportion of unmarketable tuber number was observed, 65% of total number of tubers were unmarketable during the first season. A similar trend was observed in the second season.

4.6 Effect of virus on harvest index

The effect of variety on harvest index (HI) was significant ($P<0.01$) in both seasons. The HI for the three varieties in virus-free plants in decreasing order was 0.48, 0.34 and 0.24 for varieties Ex-shimba hills, Bungoma and Kemb 10 respectively. Both virus treatment and virus x variety interaction had a significant ($P<0.01$) effect on HI in both seasons. Single virus infection with SPFMV SPMMV and SPCSV significantly reduced HI in varieties Bungoma and Kemb 10 by 49-79% while single virus infection had no significant effect on HI in variety Ex-shimba hills when compared with virus-free plants in season one (Table 7). Dual infection with SPFMV + SPCSV reduced HI in all the three varieties by 63-88% when compared with virus-free plants. A similar trend was observed during the second season (Table 7).

Table 7 Effect of virus treatment and variety interaction on harvest index of three sweet potato varieties in two seasons

Variety		Bungoma	Kemb 10	Ex-shimba hills
Season	Virus			
Season 1	Virus-free	0.34	0.24	0.48
	SPFMV	0.10	0.06	0.42
	SPCSV	0.07	0.11	0.42
	SPVD	0.07	0.09	0.06
	SPMMV	0.20	0.06	0.36
	LSD	0.13	0.13	0.13
Season 2	Virus-free	0.43	0.38	0.61
	SPFMV	0.25	0.19	0.53
	SPCSV	0.23	0.12	0.60
	SPVD	0.07	0.16	0.33
	SPMMV	0.18	0.18	0.50
	LSD	0.17	0.17	0.17

CHAPTER 5

DISCUSSION

5.1 Symptoms

Infection with the different viruses caused a range of symptoms on the different sweet potato varieties and on *Ipomoea setosa.* The symptoms induced by SPCSV in this study concurs with reports that the virus may cause purpling or yellowing of lower and middle leaves (Gibson *et al.*, 1998a) or may cause no symptoms depending on the sweet potato variety (Cohen *et al.*, 1992). Other reported symptoms include mild vein yellowing, some sunken secondary adaxial leaf surfaces and swollen veins on adaxial surface (Cohen *et al.*, 1992) but these were not observed in this study. SPCSV causing stunting and chlorosis on *I.Setosa* has also been reported by Winter *et al* (1992). The symptoms observed in I. *setosa* infected with SPFMV concurs with observation that the virus causes typical potyvirus symptoms of vein clearing and leaf distortion (Gibson et al 1997; Karyeija et al 1998). Infection with SPFMV induced no observable symptoms in the three sweet potato varieties. This conforms to the observations made in Uganda (Gibson *et al.*, 1997). The mild or no symptoms in most African varieties have been attributed to the restricted cell to cell movement and/or replication of the virus within the plants (Schaefers and Terry, 1976; Gibson *et al.*, 1998b). The ELISA test results concurs with reports that SPFMV is at low titer in sweet potato when it's infects by itself, infections can be detected with difficulty (Esbershade and Moyer 1982; Abad, 1992) but when both SPCSV + SPFMV infect , SPFMV becomes readily detected by ELISA (Karyeija *et al.*, 1998). The SPMMV symptoms observed in *I. setosa* in this study has also been

reported by Hollings et al (1976a & b). Infection with SPMMV induced no symptoms in the sweet potato varieties in this study, and is consistent with reports that SPMMV may cause symptomless infections in some sweet potato clones (Clark and Moyer, 1988).

The severe symptoms manifested on plants dually infected with SPFMV + SPCSV were similar to those observed by Schaefers and Terry (1979) and Gibson *et al.*, (1997). The decline in disease severity observed in this study when conditions were favourable has been reported and was associated with reduced environmental stress on the plant. There is documented evidence that increased host plant stress leads to symptom expression, whereas rapid growth may result in symptom remission (Brunt *et al.*, 1996).

5.2 Effects of viruses on vine yield and leaf laminar.

In this study the foliage yield of plants infected with SPFMV was not significantly different from those of virus-free cuttings. This concurs with observation made in Uganda (Gibson *et al.*, 1997). Likewise the foliage yields of plants infected with SPMMV was similar in weight to that of virus-free cuttings. In areas where sweet potato is grown for fodder infection with SPFMV and SPMMV may not be of dire economic importance but there is danger of infected plants acting as reservoir for viruses and the virus could be disseminated over long distance by vectors and man. Infection with SPFMV and SPMMV did not significantly reduce the LAI and PAR intercepted by plant canopy but there was an increase in leaf dry matter. This was reflected by the16-40% increase in SLW, which was associated with thickening of the leaves. This may imply that in plants infected with SPFMV and SPMMV, most of the assimilate produced was allocated/retained in the leaves and less allocated to the storage roots when compared to

the virus-free plants. This can be attributed to impaired translocation of assimilates, among other metabolic activities as reported for cassava infected with cassava mosaic geminiviruses (Chant *et al.*, 1971). Infection with SPCSV and SPFMV + SPCSV reduced PAR interception and the vine yield of the sweet potato plants. The reduction may be attributed to reduced photosynthetic activity due to reduced photosynthetic organs caused by stunting. In addition the low PAR must also have contributed to low vine yield. There is documented evidence that the rate of photosynthesis (dry matter production) is largely dependent on incoming solar radiation and is proportional to the amount of solar intercepted and the efficiency to which it is converted to dry matter (Squire, 1990). With dual infection the pronounced chlorosis and leaf strapping observed may have contributed to the poor vine yield. The change in leaf structure (leaf strapping) reflected in the 45% reduction in SLA, which summed up to 66% reduction in LAI might have contributed to reduced vine yield. The reduced assimilate production, which contributed to vine yield loss is also reflected in the 15% reduction on SLW and this was associated with the thinning of the leaves. The poor (75%) vine yield due to SPVD concurs with previous reports where 72% loss was recorded (Hahn, 1979). For livestock farmers the reduction in vine yield means less fodder for their animals. Sweet potato vines are easily digestible and are known to be a rich source of protein (10 to 15%) and carbohydrates (Lusweti 1995; Woolfe 1992). The important role of sweet potato vines as a protein supplement in dairy feed in Kenya will be compromised by the virus effect on vine yield.

5.3. Effect of viruses on total and marketable tuber yield and number

In this study infection with SPFMV and SPMMV reduced the total and marketable tuber weight in addition to total and marketable tuber number in varieties Bungoma and Kemb 10. Similar effects of SPFMV were observed in Uganda in a greenhouse trial using clone Tanzania (Gibson *et al.*, 1997). SPMMV infection reduced marketable tuber weight on varieties Bungoma and Kemb 10 by up to 40% and 98%, respectively. Effects of SPMMV on tuber yield have not been reported before and the 40-90% marketable tuber yield loss on varieties Bungoma and Kemb 10 show the huge economic impact of this virus on susceptible varieties. It appears that infection with SPFMV and SPMMV interferes with the sink activities probably through impaired translocation of assimilate from the leaves to the roots. The impaired translocation of assimilate which caused a higher allocation of dry matter to the vegetative parts than to the storage roots of the plants, may have contributed to the reduced tuber weight. This is reflected by the increased SLW and the 41-75% reduction in harvest index (HI). Since SPFMV is the most prevalent virus occurring in Kenya (Ateka *et al.*, 2003), it is probably contributing to the low yield attained (about 20% of the crop potential) in farmers' fields (Ndolo *et al.*, 1997; Qaim 1999). SPFMV and SPMMV had no significant effect in total tuber yield and tuber number in variety Ex-shimba hills meaning that the farmer will get better yields despite threat with single virus infection in this variety.

In this study SPCSV infection resulted in a reduction in total tuber weight and total tuber number on varieties Kemb 10 and Bungoma. Hahn (1979) and Milgram *et al* (1996) have reported tuber yield loss due to infection with SPCSV. The reduction in total tuber weight and number may be attributed to reduced photosynthetic activity due to

reduced photosynthetic organs caused by stunting. The low PAR intercepted must also have contributed to tuber yield loss. Therefore the constraining effects on root yield is most likely to have been the reduction in photosynthetic area and hence reduced photosynthetic activity and assimilate production. In addition, the partitioning of more assimilate to the aerial parts than to the storage roots as reflected by the low HI could have contributed to the tuber yield loss. Variety Kemb 10 infected with SPCSV had smaller tubers compared to those of variety Bungoma. This can be attributed to the purple pigmentation observed on Kemb 10, which may have reduced the rate of photosynthesis further. Variety Ex-shimba hills is tolerant to the single virus infection with SPFMV, SPMMV and SPCSV as is reflected in the high tuber yield and HI. Therefore it is important that this variety be incorporated in the pest management programme for increased sweet potato productivity.

Tuber yield during the second season was only 30% of that realized in the first season possibly because of the low rainfall received during the tuber growth period in the second season. There is documented evidence that water stress at tuber maturation (around 90-230 days after planting) is most detrimental to the final tuberous root yield (Bok *et al.*, 2000). Fewer sunshine hours prevailing during the second season may also have contributed to the low yields as less solar energy was available for assimilates production. Therefore, the seasonal differences in yields can be attributed to inter-seasonal differences in rainfall distribution and solar radiation supply.

The total tuber yield loss of 85-98% due to SPFMV+SPCSV observed in this study concurs with results from other countries. Studies conducted in Cameroon and Nigeria showed that SPVD reduces yields in susceptible sweet potato varieties by 56-

90% and 78% respectively (Hahn, 1979; Bouwkamp and Ngeve, 1991). Similarly, in Taiwan, SPVD caused 25-38% reduction in root yield (Chung *et al.*, 1981). In Uganda sweet potato plants of cultivar Bitambi and Kyebandula severely affected by SPVD produced only 33% of the yield of healthy plants (Aldrich, 1963; Mukiibi, 1977). Also, SPVD caused a 62-97% yield losses on five non-indigenous sweet potato clones from International Potato Centre (Aritua *et al.*, 2000). Karyeija *et al* (1998) attributed the negative effect of SPVD on root yields to have been probably caused by a reduction of leaf area, which would result in a poorer solar energy utilization for root development. The total mass of the crop depends on the size of the leaf area developed, the rate at which the leaf produce assimilates and the length of time the leaf persist. Therefore, the 66% reduction in LAI and subsequent 60% reduction in PAR interception due to SPVD observed in this study confirms that the negative effects on root yield is most likely to have been caused by a reduction in leaf area. The leaf strapping and stunting observed in plants dually infected with SPFMV + SPCSV caused the reduced leaf area and reduced photosynthetic organs. In addition, the pronounced chlorosis may have contributed to the reduced photosynthesis in the leaves by having a negative effect on plant conversion efficiency,(rate at which the leaf produce assimilate). A higher percentage of total tuber yield loss was due to a higher number of unmarketable tubers. Perhaps translocation of assimilates to the storage roots was hindered as this is reflected in the low HI and the small size of marketable tubers.

5.4 Effects of virus on unmarketable tubers

Infection with single virus resulted in a high percentage of unmarketable tubers compared to marketable tuber number (up to 76% of total tubers) on susceptible varieties. The high percentage of small sized (unmarketable) tubers is an indication of hindered translocation of assimilates available for tuber enlargement. Dual infection with SPFMV + SPCSV increased the number of small unmarketable tubers and the average tuber size was 120g compared to 210 grams of tubers harvested from virus-free plants.

The partitioning of dry matter from the leaves to the storage root appears to be one of the most important tuber yield determinants among varieties and with variety x virus interaction in this study and this is reflected by the trends in tuber yield and HI. In this study the partitioning of dry matter to storage root (HI) was greater (1.5 times) for variety Ex-shimba hills than for Bungoma and Kemb 10 and total tuber yield for variety Ex-shimba hills was almost twice the yield of varieties Bungoma and Kemb 10. This shows that the difference in tuber yields observed among varieties was due to the influence of HI. This relation between tuber yield and HI among varieties conforms to observation made by Brown (1991). The tuber yield and HI in the tolerant variety Ex-shimba hills, when infected with single viruses, remained high while in the susceptible varieties low tuber yield and reduced HI was observed. This shows that, the proportion of dry matter partitioning to the tuberous roots in relation to total biomass (HI) appears to be one of the most important tuber yield determinant among the popular sweet potato varieties in Kenya. It also implies that a variety with high harvest index despite virus infection can be identified as a tolerant variety.

There are many ways of controlling viral diseases and planting of virus-free cuttings coupled with vector control is one of the best control measure but this may be quite uneconomical for subsistence farmers who cannot afford insecticides or other costly inputs. Resistant varieties should be priority option for controlling viral diseases because resistant varieties do not require high input cost. Therefore, there is a need to screen more local varieties for the combination of superior yield attributes and resistance to sweet potato viruses and also focus on sweet potato virus resistance in breeding programs taking into account the HI mechanisms behind virus infections observed in this study.

This study has shown that virus infection may be seriously affecting the yield of sweet potato in the country. Therefore, for increase in sweet potato yield, it is imperative that farmers be encouraged to recognize and manage viral disease within their means. In this connection, efforts should be directed towards educating farmers on SPVD and its management.

5.5 Conclusions.

The following conclusion can be made from the study:

Infection with SPFMV and SPMMV had no significant effect on vine yield in the three varieties. SPCSV significantly reduced vine yield and infection with SPFMV + SPCSV resulted in severely constraining vine yield

Infection with SPFMV, SPCSV and SPMMV reduced tuber yield in varieties Bungoma and Kemb 10. Ex-shimba hills variety was tolerant to single virus infection.

Dual infection with SPFMV + SPCSV reduced tuber yield in all the three varieties

SPVD affects tuber and vine yield through reduced LAI, reduced PAR intercepted by the plant canopy, reduced photosynthetic organs and reduced harvest index.

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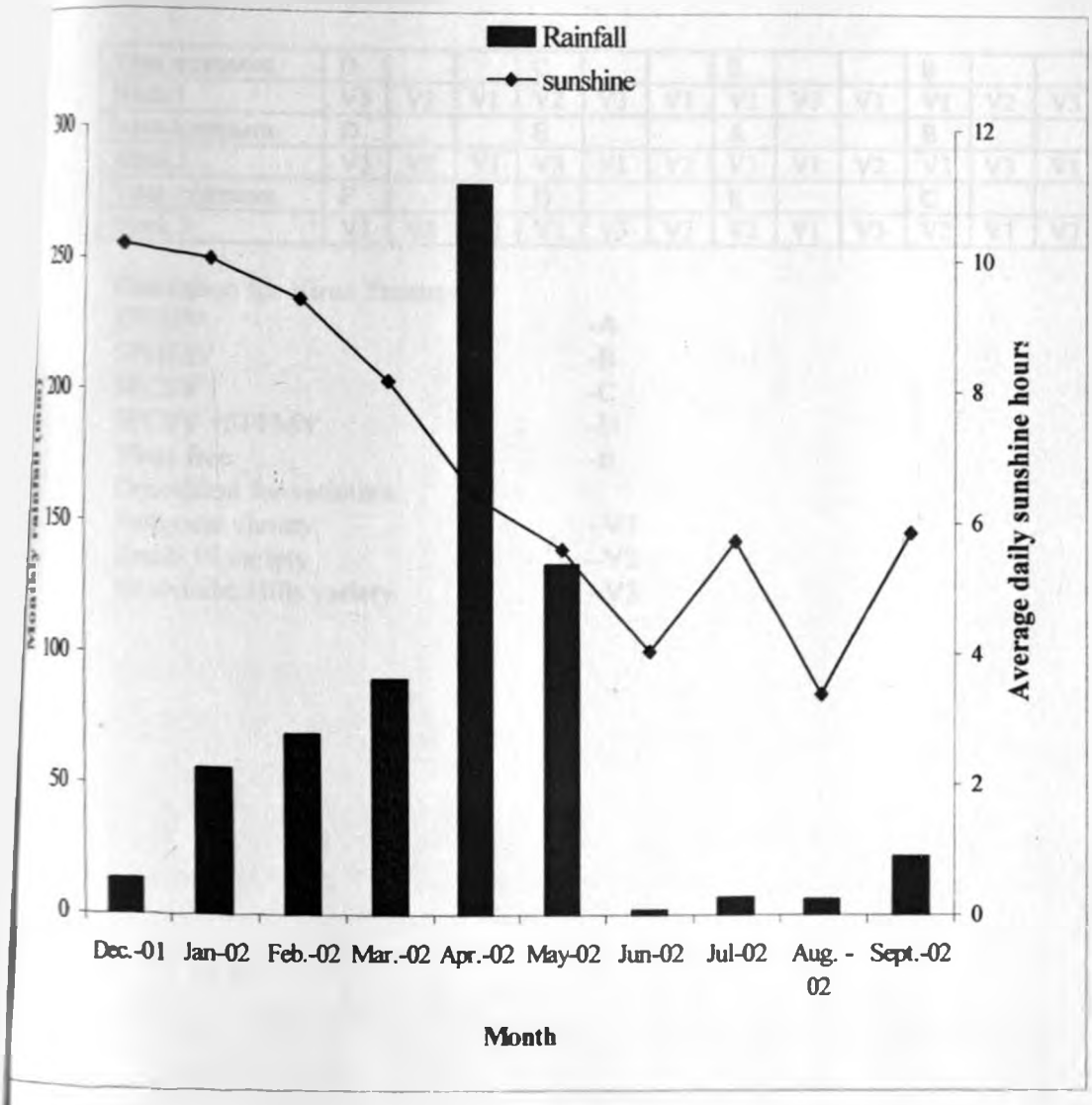
APPENDICES



ppendix 1 Average daily sunshine hours and total monthly rainfall (mm) received at Kabete Field Station during the entire study period, December 2001 to September 2002 (Source, Kenya Meteorological Department, Kabete Station)

Appendix 2 The layout of the randomized complete block design with factorial treatments.

Virus treatment	D			C			E			B			A		
Block 1	V3	V2	V1	V2	V3	V1	V2	V3	V1	V1	V2	V3	V1	V2	V3
Virus treatment	D			E			A			B			C		
Block 2	V3	V2	V1	V3	V1	V2	V3	V1	V2	V2	V3	V1	V2	V1	V3
Virus treatment	F			D			E			C			A		
Block 3	V1	V3	V2	V1	V3	V2	V2	V1	V3	V2	V1	V3	V1	V2	V3

Denotation for Virus Treatments

SPFMV	-A
SPMMV	-B
SPCSV	-C
SPCSV +SPFMV	-D
Virus-free	-E

Denotation for varieties

Bungoma variety	–V1
Kemb 10 variety	–V2
Ex-shimba Hills variety	–V3

Appendix 3 ELISA tests results from the sweet potato field experiments

Variety/virus	Block	SPFMV	SPMMV	SPCSV
Bungoma/Virus free	1	-	-	-
	2	-	-	-
	3	-	-	-
Kemb 10 Virus free	1	-	-	-
	2	-	-	-
	3	-	-	-
Ex-shimba Virus free	1	-	-	-
	2	-	-	-
	3	-	-	-
Bungoma/SPFMV	1	-	-	-
	2	-	-	-
	3	-	-	-
Kemb 10/SPFMV	1	-	-	-
	2	-	-	-
	3	-	-	-
Ex-shimba /SPFMV	1	-	-	-
	2	-	-	-
	3	-	-	-
Bungoma/SPMMV	1	-	+	-
	2	-	+	-
	3	-	+	-
Kemb 10 SPMMV	1	-	+	-
	2	+	+	-
	3	-	+	-
Ex-shimba /SPMMV	1	+	+	-
	2	+	+	-
	3	-	+	-
Bungoma//SPCSV	1	-	-	+
	2	-	-	+
	3	-	-	+
Kemb 10/SPCSV	1	-	-	+
	2	-	-	+
	3	-	-	+
Ex-shimba /SPCSV	1	-	-	+
	2	-	-	+
	3	-	-	+
Bungoma/SPVD	1	+	-	+
	2	+	-	+
	3	+	-	+
Kemb 10 SPVD	1	+	-	+
	2	+	-	+
	3	+	-	+
Ex-shimba /SPVD	1	+	-	+
	2	+	-	+
	3	+	-	+

Appendix 4 Analysis of variance

Appendix 4.1 Analysis of variance table showing mean sum of squares for disease severity scores

		Mean sum of squares			
Months after planting		1	2	3	4
Source of variation	Df				
Blocks	2	0.1556	0.23674	0.30957	0.0022
Virus treatment	4	10.2556**	6.01208**	6.29448**	5.0054**
Residual	8	0.0722	0.08297	0.15400	0.0501
Variety	2	0.6889	2.20297**	1.70582**	0.9329*
Treatment X Variety	8	0.4389	0.55017	0.48327*	0.1931*
Residuals	20	0.2222	0.05636	0.09679	0.2233
Total	44				

* -significant at p =0.05, *** -significant at p =0.01

Appendix 4.2 Analysis of variance table showing mean sum of squares for photosynthetically active radiation intercepted by sweet potato plant canopy

		Mean sun of squares
Days after planting		105 days
Source of variation	df	
Blocks	2	160.70
Virus treatment	4	7338.30**
Residual	8	141.90
Variety	2	346.80
Treatment X Variety	8	338.80
Residuals	20	243.80
Total	44	

* -significant at p =0.05, ** -significant at p =0.01

Appendix 4.3 Analysis of variance table showing mean sum of squares for specific leaf weight

		Mean sum of squares	
Season		1	2
Source of variation	Df		
Blocks	2	1.1550	0.7936
Virus treatment	4	5.6768**	2.9582**
Residual	8	0.3273	1.4804
Variety	2	0.2835	1.9190
Treatment X Variety	8	0.5715	0.3323
Residuals	20	0.1668	0.9738
Total	44		

* -significant at p =0.05, ** -significant at p =0.01

Appendix 4.4 Analysis of variance table showing mean sum of squares for specific leaf area

		Mean sum of squares	
Season		1	2
Source of variation	df		
Blocks	2	71.9	451.6
Virus treatment	4	1514.7**	948.6**
Residual	8	91.8	151.3
Variety	2	787.6	474
Treatment X Variety	8	184.4	57
Residuals	20	108.2	187.5
Total	44		

* -significant at p =0.05, ** -significant at p =0.01

Appendix 4.5 Analysis of variance table showing mean sum of squares for Leaf area index

		Mean sum of squares	
Season		1	2
Source of variation	df		
Blocks	2	2.724	1.483
Virus treatment	4	51.386**	6.919*
Residual	8	7.362	1.351
Variety	2	6.331	1.519
Treatment X Variety	8	5.223	0.743
Residuals	20	8.407	1.025
Total	44		

* -significant at p =0.05, ** -significant at p =0.01

Appendix 4.6 Analysis of variance table showing mean sum of squares for fresh vine yield.

		Mean sum of squares	
Season		1	2
Source of variation	Df		
Blocks	2	1437	7.964
Virus treatment	4	5886**	358.370**
Residual	8	766	15.201
Variety	2	1760	8.376
Treatment X Variety	8	2780	20.364
Residuals	20	1223	7.717
Total	44		

* -significant at p =0.05, ** -significant at p =0.01

Appendix 4.7 Analysis of variance table showing mean sum squares for total tuber number and yield

Season	Source of variation	df	Mean sun of squares Total tuber weight	Total tuber number
1	Blocks	2	60.88	177.30
	Virus treatment	4	456.31**	9606.40**
	Residual	8	21.25	298.50
	Variety	2	1369.40**	8139.70**
	Treatment X Variety	8	108.83**	786.60**
	Residuals	20	29.56	168.7
	Total	45		
2	Blocks	2	1.100	143.27
	Virus treatment	4	63.321**	2793.53**
	Residual	8	1.819	98.24
	Variety	2	193.97**	4872.52**
	Treatment X Variety	8	8.558**	278.09**
	Residuals	20	30.375	42.91
	Total	45		

* -significant at p =0.05, ** -significant at p =0.01

Appendix 4.8 Analysis of variance table showing mean sum of squares for number and yield of marketable tubers

		Mean sum of squares		
Season	Source of variation	Df	Number of marketable tubers	Marketable tuber yield
	Blocks	2	144.9	32713
	Virus treatment	4	3179.7**	483168**
	Residual	8	198.7	11617
1	Variety	2	2538.9**	1244405**
	Treatment X Variety	8	466.2**	94826**
	Residuals	20	168.4	2223
	Total	45		
	Blocks	2	42	1980266
	Virus treatment	4	1078.36**	53140303**
	Residual	8	30.36	1606083**
2	Variety	2	2201.89**	187327691**
	Treatment X Variety	8	165.34**	6712230
	Residuals	20	34.97	1344277
	Total	45		

*-significant at p =0.05, ** -significant at p =0.01

Appendix 4.9 Analysis of variance table showing mean sum of squares for Harvest index

		Mean sum of squares	
Season		1	2
Source of variation	df		
Blocks	2	0.06845	0.08927
Virus treatment	4	0.086229**	0.10945*
Residual	8	0.008536	0.02638
Variety	2	0.222360**	0.23046**
Treatment X Variety	8	0.025922**	0.01679*
Residuals	20	0.005528	0.01487
Total	44		

* -significant at p =0.05, ** -significant at p =0.01