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The Role of Virus Vectors in Orange Fleshed Sweetpotato Genotypes Infection - A Case Study

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Abstract- The economic importance of sweetpotato as a carbohydrate food and feed for man and animals is well known in the world all over. Sweetpotatoes are crops vegetatively propagated from vines, root slips (sprouts), and farmers often take vines for propagation from their own field year after year. Therefore, virus diseases are inevitably transmitted with propagation materials to newly planted field, resulting often in a marked decrease in yield. A virus infection is often spread by insects that pierce and suck. As is the case, no living organism such as sweetpotato is absolutely virus-free in its system. Sweetpotato genotypes accumulate viruses and the virus load is the major problem. Sweetpotato virus disease (SPVD) occurs after infection of two viruses: the sweetpotato feathery mottle virus (SPFMV) and the sweetpotato chlorotic stunt virus (SPCSV). These two viruses distort, stunted, cause chlorosis and narrowing of leaves leading to photosynthetic disturbance. SPCSV is the more problematic component of SPVD, because yield losses due to SPFMV - without SPCSV infection – are relatively low and SPFMV resistance of sweetpotato breaks down after the plant is infected by SPCSV. Of all the sweetpotato genotypes, the ones mostly affected by viruses are the orange fleshed sweetpotato genotypes.

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The Role of Virus Vectors in Orange Fleshed Sweetpotato Genotypes Infection - A Case Study

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Abstract- The economic importance of sweetpotato as a carbohydrate food and feed for man and animals is well known in the world all over. Sweetpotatoes are crops vegetatively propagated from vines, root slips (sprouts), and farmers often take vines for propagation from their own field year after year. Therefore, virus diseases are inevitably transmitted with propagation materials to newly planted field, resulting often in a marked decrease in yield. A virus infection is often spread by insects that pierce and suck. As is the case, no living organism such as sweetpotato is absolutely virus-free in its system. Sweetpotato genotypes accumulate viruses and the virus load is the major problem. Sweetpotato virus disease (SPVD) occurs after infection of two viruses: the sweetpotato feathery mottle virus (SPFMV) and the sweetpotato chlorotic stunt virus (SPCSV). These two viruses distort, stunted, cause chlorosis and narrowing of leaves leading to photosynthetic disturbance. SPCSV is the more problematic component of SPVD, because yield losses due to SPFMV - without SPCSV infection – are relatively low and SPFMV resistance of sweetpotato breaks down after the plant is infected by SPCSV. Of all the sweetpotato genotypes, the ones mostly affected by viruses are the orange fleshed sweetpotato genotypes. The slow sweetpotato breeding progress in Nigeria and Africa in general can be attributed to low investments into breeding virus resistant genotypes and selection procedures to farmer needs by formal plant breeding. Control measures in addition to others are put in place to avoid virus disease epidemic: planting of virus free materials, land and crop rotation, thermotherapy, meristem culture, control of vectors by chemical application, soaking of botanical seeds before planting in hot water and others.

Keywords: orange fleshed sweetpotato, virus disease, yield, SPVD and symptoms.

I. Introduction

Sweetpotato [*Ipomoea batatas* (L.)Lam] is cultivated in more than one hundred countries around the globe. It is a dicotyledonous plant belonging to the *Convolvulaceae* family of about 45 genera and 1000 species, with only *I. batatas* of economic importance as food (**Woolfe, 1992**). There are over 1402 varieties of sweetpotato which skin and flesh colour may be almost white, cream, yellow, orange, pink, or deep purple depending on the variety. However, the white/cream and yellow-orange fleshed colours are most common (**Ahmad *et al.*, (2006).**). It is an important staple food crop in some states in Nigeria such as Ebonyi, Nassarawa, Benue and Kaduna where most of the crop is produced.

Wolfgang and his co-workers (2012) reported that the Food and Agriculture Organization (FAO) statistics annual sweetpotato production in Africa has increased moderately from 11.6 million tonnes in 2002 to 12.9 million tonnes in 2006. This production was mainly by smallholders (the majority of whom are women) for home consumption. Typically less than 20% of production is traded in rural and urban markets. Although data on piecemeal harvested sweetpotato crop are difficult to collect.

II. Uses of Sweetpotato

Odebode (2004), reported on the many uses of Sweetpotatoes. He noted that sweetpotato have been consumed as carbohydrate food by man and animals. Many parts of sweetpotato plant, (leaves, roots and vines), are edible. Sweetpotato roots can be boiled, steamed, baked, and fried. Traditionally, sweetpotatoes can be boiled and eaten, fried into chips and eaten, pounded or mixed with yam and eaten with vegetable soup, roasted and eaten with red palm oil or sauce, or made into porridge. Use in preparing "kunnu" drink, processed into fufu and eaten with vegetable soup. Sweetpotato can be processed into flour or mixed with cassava flour to make *amala.* Processed sweetpotato flour can be used as sweetener, eaten boiled with rice, processed into kunuzaki which is oiled and eaten with groundnut cake. The leaves contain up to 4% protein and the fresh Sweetpotato leaves can be used in making vegetable soup and served with Pounded yam, *Eba or Amala*.

In China, sweetpotatoes are also canned or dried and made into flour, cereal, and noodles or sealed in cellophane bags and sold in supermarkets. Sweetpotato roots are often used in making biscuits, cakes, and desserts or processed into products such as chips, Sweetpotato 'Sparri' (toasted 'sparri' granules) soaked in water and eaten with groundnut as a snack or put in boiled water and served with soup, Sweetpotato

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Cake eaten as snacks or main dish to entertain visitors, puff-puff, buns, bread, crisps, chin-chin served as snacks, ketchup (chopped boiled sweetpotato tuber mixed with tomato sugar, onions, vinegar, salt, water) served with bread for breakfast, sweetpotato chips (deep fried sun-dried chips) eaten as snacks, Sweetpotato Jam served with bread for breakfast (ACC/SCN. 1998). To increase more yield for product value additions, breeding work are going on in National and international research centres across the globe.

In the short term, crossing and combining parents with medium to high genetic values across all objectives and traits is all that is required to produce high quality clones for various quality uses. In pre-breeding and medium to long term population improvement, parents are developed by incorporation of new attributes from sources which often only have a high genetic value in one or very few attributes (e.g. excellent disease resistance but poor yield performance and other traits). Within the sweetpotato gene pool, there is an enormous amount of genetic variation for quality attributes. A renowned example is the concentration of pro-vitamin A in storage roots, which ranges from 0 to nearly 1000 ppm on a storage root dry weight basis (dwb). This corresponds to 0 to 20 mg β-carotene in 100 g of fresh sweetpotato storage roots (about 5 mg β-carotene meets the daily requirement of a pre-school child (400 µg/day RAE). Similar magnitudes of genetic variation are found for starch, sugars and probably for dietary fiber. Moderate genetic variation is found for protein and minerals such as iron and zinc. It is quite convenient, however, that the attributes, proteins and minerals are positively correlated genetically with β-carotene in sweetpotato storage roots, so that improvement in pro-vitamin A is linked with an improvement in iron, zinc and other minerals such as calcium and magnesium. Breeders nearly always want to select for several traits concurrently. In practical terms, quality breeding often means to improve quality (three to five traits) and simultaneously maintain sufficient genetic variation for yield, yield stability, adaptability and resistant to major diseases such as viruses.

III. Virus Disease in Sweetpotato

A virus is a microscopic pathogen with a protein structure that is not visible with the naked eye. By rapidly multiplying itself in living plant cells, the virus can damage the sweetpotato and considerably reduce its yield, to the great detriment of the farmer. A virus infection is often spread by insects that pierce and suck. The damage caused by the virus is then usually much greater than the mechanical injury caused by the insect. Of all the sweetpotato genotypes, the ones mostly affected by viruses are the orange fleshed sweetpotato genotypes.

IV. Origin and Spread of Viral Disease of Orange Fleshed Sweetpotato

Viruses are usually spread to orange fleshed sweetpotato crops by insects (vectors) that have sucking mouth parts, especially aphids, plant hopers and whiteflies, but other insect orders and families can also be responsible. These insects can come from the direct vicinity or from far away fields with other types of crops such as cassava, *Telferia occidentalis*. For instance the infamous tristeza virus in citrus trees is spread by an aphid that can be carried hundreds of kilometres by air currents. The infection can thus come from distant places, especially places where wind or tyhoons occurs. Viruses can also be spread by human hands that have come in contact with an infected crop or product. The tobacco mosaic virus is an example of a disease that can be spread in this way. This can sometimes be spread to sweetpotato or tobacco farms through the hands of workers who have rolled cigarettes with infected tobacco. Vegetatively propagated plant material can spread viruses. Soil viruses can be spread by nematodes and certain soil fungi. Orange fleshed sweetpotato field infested with nematodes can equally be attacked by viruses.

Some orange fleshed sweetpotato genotypes can carry a virus without being significantly damaged by it. The farmer may not even notice that the disease is present until it spread to different, more susceptible orange fleshed or white genotypes, where it does cause serious damage. Only then is the presence of the virus clearly evident. This indicated that not all orange fleshed sweetpotato genotypes are equally susceptible. Viruses affecting sweetpotato can be perpetuated and spread between cropping cycles by the use of foliar cuttings taken from infected plants. They are also transmitted from plant to plant by sap soaking insects. So far the only proven vectors of sweetpotato viruses are aphids and whiteflies (Stathers et al., 2005)

The sweetpotato mild mottle virus (SPMMV) and sweetpotato chlorotic stunt virus (SPCSV) are transmitted only by whitefly predominantly Bemisia spp, while sweetpotato feathering mottle virus (SPFMV) and the related sweetpotato virus 2 (SPV2) and sweetpotato virus G (SPVG) are transmitted by aphids. Some weeds such as Morning Glory harbor the viruses but the only economically important source of infection is other infected sweetpotato plants.

Stathers *et al.*, (2005) reported that SPFMV and SPCSV are together the most important viruses affecting sweetpotato in Africa and occur everywhere sweetpotato is growing. Sweetpotato can be infected by more than one virus species and when there is a build up, the viruses help each other to multiply with the result that the disease is even more severe. SPCSV in particular synergizing the multiplication of SPFMV and other viruses Stathers et al., (2005) observed. This severely

affects the growth of sweetpotato plant. Stathers et al., (2005) emphasized that it is the combination of SPCSV and SPFMV is the most important disease of sweetpotato worldwide and is known as sweetpotato virus disease (SPVD). The SPFMV is transmitted by a wide range of adult winged aphid species including those that do not colonize sweetpotato while SPCSV is transmitted by the mobile adult whiteflies especially *B. tabaci* as they fly from plant to plant. Therefore, since the spread of SPCSV by whiteflies that synergizes SPFMV, whiteflies are the force behind the spread of SPVD.

V. Yield Losses Caused by Viruses

SPMV by itself is sometimes symptomless in sweetpotato, however, Stathers et al., (2005) observed that it can cause vein clearing and purple ring spots on the leaves of susceptible varieties. SPCSV on its own cause dwarfing of sweetpotato plants and either purpling or yellowing of lower leaves. Both viruses cause yield loss when sweetpotato plants are affected. For instance, in South Africa and China yield increases of more than 30% occurred as a result of planting virus-free planting material.

When a sweetpotato plant is infected with both SPCSV and SPFMV, the symptoms become very severe SPVD. Symptoms include severe stunting of the plant and small malformed leaves such as leaf curling and dwarfing, leaf mottling, yellowing of veins. Sometimes with either a chlorotic mottle or vein clearing. These symptoms are most apparent in young sweetpotato plants as they get established although plants can be infected at any age. If the entire oranged fleshed sweetpotato plant is affected by SPVD, the plant produce small storage roots generally of unusable size, resulting in a massive reduction in the yield of individual plants. Stathers et al., (2005) gave the following figures in the Table of effect of viruses on sweetpotato plant:

Comparison of healthy and virus infected sweetpotatoes in some countries as noted by Stathers et al., (2005)

Country	Root yield loss	Cultivar	Comments
Uganda	66%	Bitambi	Severely diseased plants
Uganda	70-99%	7 clones	Virus free clones as negative control
Uganda	57%	Kyebandula	Some controls became diseased
Nigeria	76-78%	TIS 1499	Severe diseased plants
Nigeria	60%	TIB 4	Low level of resistance of SPVD
Cameroon	0-90%	8 clones	Losses varied between clones and field trials

Sweetpotatoes are vegetatively propagated from vines, root slips (sprouts), and farmers often take vines for propagation from their own field year after year. Therefore, if virus diseases are present in the field, they will inevitably be transmitted with propagation materials to the newly planted field, resulting often in a marked decrease in yield. Loebenstein *et al.*, (2009) reported that yields differ greatly in different areas or even fields in the same location. The average yield in African countries is about 7.02 t/ha, with yields of 9.4, 4.4, 2.5 and 3.2t/ha in Kenya, Uganda, Sierra Leone and Nigeria respectively. The yields in Asia are significantly higher, averaging 12.41t/ha. China, Japan, Korea and Israel have the highest yields with about 21.6, 25.8, 16.4 and 44.4t/ha respectively. In South America the average yield is 10.74t/ha with Agentina, Peru, and Uruguay in the lead with 17.2, 16.35, and 13.68t/ha, respectively. For comparison, the average yield in the USA is 20.1t/ha (all data are averaged for 2005 from the FAOSTAT, 2007).

Loebenstein and co-workers (2009) further observed that these differences in yields are mainly due to variation in quality of the propagation material often taken from the previous season of farmer's fields. Often these fields are infested with several viruses, thereby compounding the effect on yields. In China, on average, losses of over 20%, mainly due to sweetpotato feathery mottle virus (SPFMV) and sweetpotato latent virus (SPLV). The infection rate in the Shandong province reaches 5 to 41% . Loebenstein and co-workers (2009) noted that in countries were care is taken to provide virus - tested planting material as amongst others in the USA and Israel, yields increase markedly up to 7 times and more while in some countries, as in Uganda, Kenya and Tanzania virus diseases are a major constraint for sweetpotato production.

VI. Virus Disease, a Challenge to Sweetpotato Breeding Work

The main reason for slow sweetpotato breeding progress in Nigeria and Africa in general can be attributed to low investments into breeding virus resistant genotypes and selection procedures to farmer needs (Gibson *et al.,* 2008) by formal plant breeding. This makes successful breeding for yield progress long term in nature and complicated in designing crossing programs. Moreover, sweetpotato as a clonally

propagated crop can be easily multiplied and maintained, which is an advantage as well as a burden, because many diseases (especially viruses) are transmitted in planting material. The cloning characteristic permits rapid and wide dissemination of successful genotypes and varieties, respectively, and the exploitation of heterosis, an important genetic effect for yield, yield stability and adaptability. The multiplication and maintenance of vegetative propagules in virus loaded environment means multiplication, maintenance and dissemination of virus loaded materials.

Achieving medium to long term yield gains in sweetpotato is a challenge in sweetpotato breeding because the performance of a parent in one generation is not a good indicator for the value of a parent for the next sweetpotato generation. However, the genetic constitution of sweetpotato permits the adaptation of sweetpotato populations to new needs in the broad sense (environments, quality demands, tolerance to pests and diseases) to be achieved quite rapidly from the view point of crop evolution. Examples for this potential abound in the sweetpotato gene pool; it is possible to find many genotypes which are specifically adapted to drought, heat, cold (in tropical highlands), mineral-stress (including acid soils) or extreme salinity. Yield, yield stability and adaptability (including genotype by environment (G by E patterns) of crops are often associated with resistance to biotic and abiotic stress. However, most sweetpotato variety especially the orange fleshed genotypes succumb/broke down to viruses after a few years trial as result of accumulation of the virus disease each year being vegetatively propagated. As a result breeding orange fleshed sweetpotato genotypes with long time virus resistance is a challenge for now.

VII. Sweetpotato Virus Disease (SPVD) Tolerance Clones

The humid tropical low and mid-elevation States in Nigeria (0 to 1200 m.a.s.l.) with only very short dry seasons have high SPVD pressure, which is extreme in regions where sweetpotato is extensively cultivated. As is the case, no living organism is absolutely virus-free in its body system. The same applies to sweetpotato genotypes. Sweetpotato genotypes accumulate viruses in virus prone areas and the virus load is the major problem. SPVD occurs after infection of two viruses: the sweetpotato feathery mottle virus (SPFMV) and the sweetpotato chlorotic stunt virus (SPCSV). SPCSV is the more problematic component of SPVD, because yield losses due to SPFMV - without SPCSV infection – are relatively low and SPFMV resistance of sweetpotato breaks down after the plant is infected by SPCSV. SPCSV resistance has been found in germplasm screening programs and the resistance appears to be conferred by a recessive allele that occurs in low frequency in the sweetpotato gene pool. Field resistance to SPVD has been obtained in East Africa by screening large numbers of sweetpotato genotypes from mainly local germplasm, and open pollinated seed and limited controlled cross progenies, evaluated on-station and on-farm. However, this resistance, still needs to be proven in extensive controlled artificial inoculation with SPVD and field tests under high SPVD pressure locations in Nigeria. It is nearly certain that new sweetpotato varieties with resistance to SPVD will result in significantly higher yields and yield stability in Nigeria, at least for a period of 5 to 8 years. After this period new strains of the sweetpotato chlorotic stunt virus gene pool are expected to emerge. Viruses are serious on sweetpotato. Some of them are briefly discussed below.

VIII. Sweetpotato Viral Diseases in Nigeria

a) Sweetpotato feathery mottle potyvirus (SPFMV)

Sweetpotato feathery mottle potyvirus (SPFMV) This virus disease causes mild or no symptoms in sweetpotato (Nyiira, 1982). It is transmitted by Aphid. In sweetpotato plant or when indicator plant such as *Ipomoea setosa* or *Ipomoea nil* is grafted to the sweetpotato plant, the symptom appear mild and transient vein clearing, vein feathering and chlorotic spots, especially on older leaves, and in roots there is external cracking cork and internal necrosis depending on cultivar and isolate although the symptom is more severe on indicator plants. In indicator plants, the leaves are mosaic, vein clearing, leaf stunting, and distortion (Gibson *et al.,*1998).

b) Sweetpotato chlorotic stunt cloresterovirus (SPCSV)

Gibson *et al., (*1998) reported that in Nigeria, the SPCSV alone causes no symptoms in sweetpotato. He reported that this was proved from a screen house experiment where plants grown from SPCSV infected cuttings produced about 25% of uninfected foliage cuttings and storage root yield was only about 13% of uninfected cuttings. However, Karyeija *et al*., (1998) and Gibson *et al*., (1998), observed in a survey and in an experimental transmission in NAARI that it is the dual infection of SPCSV and SPFMV that is the main cause of Sweetpotato virus disease (SPVD). SPVD is the most destructive disease of sweetpotato in Nigeria and in Africa in general. Gibson *et al*., (1998) also reported that symptoms of SPVD vary with sweetpotato genotype but include stunted plants with small leaves often distorted, narrow strap-like and crinkled, with a chlorotic mosaic and/or vein clearing. Affected sweetpotato plants generally appear pale. Root yield losses due to SPVD vary from 57% - 98% depending on varietal susceptibility. Although Gibson *et al*., (1998) reported that root yield losses vary in other countries. SPVD is most severe in sweetpotato cultivating states in Nigeria

such as Ebonyi State, Nassarawa State, Benue State and in Umudike Abia State indicating that this disease is wide spread. This disease mostly attack orange fleshed sweetpotato genotypes including the recently released orange fleshed variety UMUSPO/3 by National Root Crops Research Institute, Umudike..

c) Sweetpotato mild mottle ipomovirus (SPMMV)

SPMMV is a virus disease transmitted by whitefly and aphids. Carey *et al*., (1998), noted that SPMMV causes relatively mild symptoms in sweetpotato plants. Carey et al., (1998) and Gibson *et al.,* (1998) noted that no yield loss assessment on *Sweetpotato mild mottle ipomovirus (SPMMV)* has been carried out in sweetpotato in Uganda but the disease was detected in sweetpotato samples in some districts in Uganda such as Mbale, Mpigi, Masindi, Kabale, Tororo and Iganga.

d) Sweetpotato chlorotic fleck virus (SPCFV)

Carey *et al*., (1998) reported that SPCFV have been detected in sweetpotato samples from some districts in Uganda such as Mbale, Mpigi, Masindi, Kabale, Tororo and Iganga. Wambugu (1991) also reported cucumber mosaic virus CMV and sweetpotato latent virus SPLV as common sweetpotato virus disease in sweetpotato field.

IX. Symptoms on Orange Fleshed Sweetpotato

The tissue of Orange fleshed sweetpotato plant damaged by a viral disease does not die off immediately. It does not display any necrotic spots or areas. The most important symptom of viral infections is the light (white and yellow) colour of the leaves or a mosaic pattern of light and darker shades of green on the leaves. Larger spots (sometimes in an oak-leaf pattern) can also appear within which a 'rain-stripe' pattern (with multiple yellow or pale green, narrow, parallel lines and bands) is visible. The spots that form the mosaic pattern can be angular (bordered by the leaf's veins) or rounded and sometimes even ring shaped. The latter example usually involves a soil virus. The leaf veins often also become lighter in colour, appear waxy and have a thin, darker-coloured streak on either side (i.e vein - clearing). Wabungu (1991) observed that the *Psorosis* virus in citrus trees causes their bark to die off and separate above the bud union with the lower trunk. Gummossis then often occurs as well.

In many cases, virus disease leads to dwarfed growth, rosette formation or other strange and leaf abnormalities. Rice, for example, can take on a grassy appearance as its leaves become small and thin. The same in sweetpotato and sometimes with flattened vines with numerous tiny narrow leaves. Cocoa can develop a type of 'witches' broom appearance, in which many small branches grow closely together. Leaf curl in cotton causes deformation of the edges of the leaves, which become curled, wavy or contorted as some parts of the leaf grow faster than others. The same effect can be seen on fruits (e.g. Citrus fruits), which develop shallow grooves, bulges, or other irregularities on their surface (Joep van Lidth de Jeude(2004)..

The symptom of viral infection on sweetpotato genotypes are often not found everywhere in a cultivated sweetpotato field as is usually the case with with fungal or bacterial diseases. However, in orange fleshed sweetpotato genotypes, a whole field can show the symptom of viral infection. It is almost always possible to find a number of sweetpotato plants that show no signs of the disease. Surprisingly, even a plant that is thoroughly infected with a viral disease may only show symptoms on one part, such as one half of a leaf.

Abnormal (lighter) leaf colour, abnormal leaf and stem shape, dwarfed growth and mosaic patterns on leaves can, however, be signs of a nutrient deficiency as well as a viral infection or nematode infestation. A viral disease cannot be diagnosed with any certainty at first glance or without laboratory tests.

X. Tests to Detect Viral Infection in Orange Fleshed Sweetpotato Genotypes

The best approach in this case is probably to conduct a few simple tests to determine whether the anomaly could be caused by a deficiency or nematode infestation (Joep van Lidth de Jeude (2004). This can be done by spraying a nutrient solution of micro- and macro-elements on the affected oranged fleshed sweetpotato plants and applying a nematicide to see if this brings about any improvement in the sweetpotato plant's condition. If not, then it is indeed likely that the damage is caused by a viral infection.

Another test is Sweetpotato virus indexing. This is accomplished by grafting sweetpotato cuttings onto indicator plants (*Ipomoea setosa and Ipomoea nil*), and symptoms are evaluated after 4 weeks (Panta *et al*., 2007). Positive symptom observation is followed by Nitro-cellulose membrane Enzyme Linked Immunosorbent Assay (NCMELISA) with available antisera (sweetpotato feathery mottle virus (SPFMV), sweetpotato latent virus (SPLV), sweetpotato mild mottle virus (SPMMV), sweetpotato chloric fleck virus (SPCFV), C-6 virus sweetpotato chlorotic stunted-virus (SPCSV), sweetpotato caulimo-like virus (SPCaLV) and Cucumber mosaic virus (CMV).

According to Loebenstein and co-workers (2009, Nucleic acid spot hybridization and PCR are optionally used to confirm the presence of some viruses for which antisera are not available, and that after the initial plant health check, infected accessions are

submitted to virus elimination process and subsequently re-checked.

XI. Control Measures of Viruses in Orange Sweetpotato Field

Viruses spread very fast through the vascular system of a plant to entire plant and plant population. As a result, sweetpotato plants that show symptoms of a viral disease have to be removed from the crop and destroyed as soon as possible. If a virus spread through seeds especially during seedlings evaluation in Breeding Programme, the seeds can sometimes be neutralized by soaking the seeds in warm water before planting. Viruses cannot be treated with chemical agents. The most important way to prevent a viral infection is to use virus-free seeds and vine plant material. It is possible, however, to control the vectors (insects, nematodes) by applying chemicals, or often by adhering to strict periods during which a susceptible crop, or another botanically related crop, is not allowed to be cultivated on a particular field or during a particular period. Burning infected plants, isolation and planting of new fields far away (more than 100m) from old sweetpotato production in the control of viral diseases to maintain or increase production (Panta *et al*., 2007).

Joep van Lidth de Jeude (2004) observed that it is very difficult to disinfect soil that has been infected by a virus. The best approach is to cultivate sweetpotato genotypes that are not susceptible to that particular virus or to initiate a fallow period during which the soil can receive a great deal of sun exposure. Improved, virus resistant orange fleshed sweetpotato are available at the National Root Crops Research Institute, Umudike, Nigeria. Using these resistant orange fleshed sweetpotato genotypes is the easiest way to prevent viral infection.

The production of virus free sweetpotato is achieved almost exclusively by meristem culture *in vitro.* In meristem culture, the essence is to take as large a meristem as possible, while excluding virus infected tissue. Therefore, smaller apical (meristematic) explant might be clean from virus but not a longer apex subtending some leaf primordia (Henderson *et al*., 1984). Virus cleaning of sweetpotato by meristem culture is considered much more effective than by thermotherapy as Kuo (1991) noted and that with an 80% rate of virus clean shoots. Dangler *et al* (1994) reported that virus free sweetpotato propagation material has been produced by heat therapy alone. However, virus cleaning by meristem culture was the basis of the California State programme for sweetpotato improvement (Dangler *et al* (1994).

Therefore for establishing orange flesh sweetpotato field, collect cuttings for new crops from healthy plants. Avoid collecting cuttings for new plantings from very old crops because SPVD may have built up in these crops and SPVD is less easy to see in old plants than in vigorous-growing crops. The following are a summary of control measures in orange fleshed sweetpotato field Stathers *et al*., (2005):

- Remove any diseased plants as soon as they appear.
- Avoid planting new crops where sweetpotato was grown in the last season. This is because roots and cuttings from old surviving diseased plants in the soil will produce disease plants which act as source of inoculums to the new crop.
- Plant new crops far away from old crops so that it is difficult for whiteflies and aphids to reach the new crop.
- Other measures include crop hygiene such as ensuring that crop debris, leaves and roots is completely destroyed by fire or fed to livestock. Viruses attacking plants do not infect animals.
- Plant resistant varieties, which is the most convenient means of controlling SPVD. High yielding resistant varieties have been bred; farmers can use these varieties to produce a crop in areas of high SPVD pressure.
- Farming communities can work together to eradicate SPVD by applying all the control measures since all will benefit from it.

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