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Review Article

Viruses Infecting Sweet Potato (*Ipomoea batatas* (L.) Lam.) in Nigeria

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Abstract
Sweet potato is the second most important root crop after cassava in Nigeria. Due to decline in Nigeria's economy in recent years, the production of sweet potato has significantly increased from 2.4 million metric tonnes in 2000 to 4.1 million metric tons in 2017 to meet up with its demand for local consumption. However, the profitable production of the crop is being threatened by virus diseases such as *Sweet potato feathery mottle virus* (SPFMV), *Sweet potato mild mottle virus* (SPMMV), *Sweet potato chlorotic stunt virus* (SPCSV), *Sweet potato leaf curl virus* (SPLCV), and *Cucumber mosaic virus* (CMV). Useful information about these viruses is instrumental in the effective management of the crop in Nigeria. This paper reviewed the major viruses that affect sweet potato production in Nigeria.

Keywords: *Infection, Management, Nigeria, Sweet potato, Viruses*

Introduction

Sweet potato (*Ipomoea batatas* (L.) Lam.) is a dicotyledonous perennial crop cultivated both in the warm temperate and tropical parts of the world but grown as an annual (Patil, 2020). It belongs to the family *Convolvulaceae* commonly known as Morning Glory, which comprises plants with funnel-shaped flowers (Muimba-Kankolongo, 2018). It ranks seventh in global food crop production and the third most important root crop after Irish potato and cassava (Loebenstein, 2015). Sweet potato is also the sixth most important food crop, based on dry matter, mainly cultivated for its storage root and leaves as vegetables globally (Tavva and Nedunchezhiyan, 2012). It has been reported to play a significant role in the food security for millions of people across South America, Africa and Asia and has had long historical importance in disaster relief (Muimba-Kankolongo, 2018). In Nigeria, sweet potato is produced virtually in every part of the country but predominantly in the Northern Guinea Savannah where many landraces abound (NAERLS *et al.*, 2007). It is a major cheap source of energy for more than 80% of Nigerians living below the poverty line especially in the northern part of the country. With the current decline in Nigeria's economy in recent years, the production of sweet potato has significantly increased from 2.4 million metric tons in 2000 to 4.1 million metric tonnes in 2017 (FAOSTAT, 2018). However, the production of the crop is now being threatened by the menace of viral diseases in Nigeria. Virus diseases of sweet potato cause significant yield loss ranging from 90-100% depending on the virus strains, plant cultivar, stage of infection and environmental conditions (Arkorful and Addae-

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Frimpomaah, 2015). Over thirty (30) viruses were reported on sweet potato worldwide (Barkessa, 2018). Based on their widespread and the nature of damage caused to a sweet potato plant, nine of these viruses are considered of global economic importance and they include *Sweet potato feathery mottle virus* (SPFMV), *Sweet potato chlorotic stunt virus, Sweet potato virus G* (SPVG), *Sweet potato mild mottle virus* (SPMMV), *Sweet potato chlorotic fleck virus* (SPCFV), *Sweet potato latent virus* (SPLV), *Cucumber mosaic virus* (CMV), *Sweet potato caulimo-like virus* (SPCaLV) *and Sweet potato leaf curl virus* (SPLCV). The reported viruses of sweet potato in Nigeria include *Sweet potato feathery mottle virus* (SPFMV), *Sweet potato chlorotic stunt virus* (SPCSV), *Sweet potato mild mottle virus* (SPMMV), *Cucumber mosaic virus* (CMV), and *Sweet potato leaf curl virus* (SPLCV) (Alegbejo, 2015; Mohammed *et al.*, 2019). Availing information about these viruses including their causative agents, properties, transmission, symptoms, yield losses as well as elucidating the effect of mixed infections of the viruses in sweet potato, will be instrumental for plant breeders, extension workers, national quarantine agency and policymakers for sustainable management of sweet potato viruses in the country. Therefore, we review the status of important viruses infecting sweet potato in Nigeria.

Sweet potato feathery mottle virus (SPFMV) genus Potyvirus

Sweet potato feathery mottle virus (SPFMV; family *Potyviridae*, genus *Potyvirus*) is a single-stranded linear, unipartite (11.6 kb) RNA virus with a poly(A) 3'-end region (Gibson *et al.*, 2000). Virions are filamentous, not enveloped, usually flexuous, with a modal length of 830 – 850 nm (Loebenstein, 2015). It has a thermal inactivation point of 60 – 90^{o}C, longevity *in-vitro* of 0.3 to 0.5 days or 7-12 hours and dilution endpoint 10^{-3} – $10^{-4.9}$ (Alegbejo, 2015). SPFMV is the most common sweet potato virus worldwide as well as the most important potyviruses infecting sweet potato in Africa and elsewhere (Barkessa, 2018). The virus has been reported to occur in all the sweet potato producing regions in Nigeria and recently reported in Kaduna State indicating a high incidence of the virus with yield losses ranging from 30 to 40% (Alegbejo, 2015; Mohammed *et al.*, 2019). Many strains, isolates, variants, and serotypes of SPFMV has been reported, but mainly from South America and Africa. By comparing coat protein gene sequences of the isolates, it was reported that a Spanish isolate was related to the East African cluster of SPFMV (Clark *et al.*, 2012). Symptoms of SPFMV are stunted growth, vein clearing in the younger leaves, and yellow chlorosis in older leaves with short internodes and reduced root number (Barkessa, 2018). During the co-infection of SPFMV with *Sweet potato chlorotic stunt virus* (SPCSV) extreme yield loss from 50% up to 90% has been reported (Patil, 2020). SPFMV is transmitted in a non-persistent manner by different species of aphids, including *Aphis gossypii*, *Myzus persicae*, *Aphis craccivora*, and *Lipaphis erysimi* (Muimba-Kankolongo, 2018). Aphid transmission of the virus depends on virus-encoded helper protein component and a DAG triplet in the protein coat (Loebenstein, 2015). The virus can also be transmitted by sap through mechanical inoculation (to various *Ipomoea* spp. as *I. batatas, I. setosa, I. nil, I. incarnata*, and *I. purpurea*), grafting and use of infected vines by farmers, but not pollen grains (Gutiérrez *et al.*, 2003; Alegbejo, 2015). Even though the single infection of SPFMV generally causes only minor damage, its control is imperative as in mixed infection with other viruses result in a significant yield loss (Loebenstein, 2015). Integrated management of SPFMV which include: the planting of only runners obtained from healthy plants, use of resistant/tolerant cultivars, maintaining weed-free fields, control of the virus vectors and early planting to minimize the incidence and severity of the disease have been reported to be an effective measure (Alegbejo, 2015).

Sweet potato mild mottle virus (SPMMV) genus Ipomovirus

Sweet potato mild mottle virus (SPMMV: family *Potyviridae*, genus *Ipomovirus*, synonym: *Sweet potato B virus* (Sheffield, 1957) is an economically important yield-reducing pathogen in sweet potato fields with reported prevalence in South Africa, Indonesia, China, Philippines, India, New Zealand, Egypt, Nigeria and some other West African countries (Alegbejo, 2015;

Loebenstein, 2015). Its virions are flexuous rod-shaped particles, measuring 800-950 nm in length, containing 5% ssRNA and 95% capsid (Loebenstein, 2015). The viral RNA is cloned and the assembled genomic sequence is 10,818nts in length with a polyadenylated tract at the 3'-end (Jones and Dwyer, 2007; Cuellar *et al.*, 2008). Sequences of several SPMMV isolates revealed nucleotide sequence identities of 88% of the coat protein (CP) encoding region and 3-UTR, respectively, while CP amino acid sequences were 93-100% identical. Analysis of the CP-encoding nucleotide sequences did not reveal phylogenetically distinguishable groups of SPMMV isolates. Rather, the analysis indicated high genetic variability (Tairo *et al.*, 2005). The virus is transmitted by whiteflies (*Bemisia tabaci* Genn.) in a semi-persistent manner, grafting, and mechanical inoculation but not transmitted by seed or by contact between plants. The virus has a wide host range with a report of transmission to plants in 14 families (McGregor *et al.*, 2009; Loebenstein, 2015). Disease caused by SPMMV in sweet potato express characteristic symptoms which include mottling of leaves, stunted plant growth, and loss of yield. However, different cultivars of sweet potato differ greatly in their reaction to the virus, some being severely infected or mild, some remain asymptomatic and others immune (Hollings *et al.*, 1976). Synergy was observed in sweet potato mixed infected by SPMMV and SPCSV (but not by SPFMV) (Untiveros *et al.*, 2007; Untiveros *et al.*, 2016) with SPMMV titres reported to increase by approximately 1000-fold (Mukasa *et al.*, 2006). Yield loss of 42 to 75% in mixed infection has been reported (Untiveros *et al.*, 2016). Integrated management approach as reported for SPFMV will also suffice for SPMMV.

Sweet potato chlorotic stunt virus (SPCSV) genus Crinivirus

Sweet potato chlorotic stunt virus (SPCSV: family *Closteroviridae*, genus *Crinivirus*) is one of the global most economically important pathogens of sweet potato reported to cause up to 50% yield reduction (Mukasa *et al.*, 2006). The occurrence of the virus has been documented in Argentina, Brazil, Egypt, East and Southern Africa, Indonesia, Israel, Nigeria, Niger, Spain, Peru (Karyeija *et al.*, 1998), and in the United States (Abad *et al.*, 2007). SPCSV has flexuous particles of 850-950 nm length and 12 nm diameter (Hedge *et al.*, 2012). The genome consists of two RNA molecules, RNA1 (9407 nt) contains five putative ORFs for replication related proteins and RNA2 (8223 nt) contains seven putative ORFs. The virus encodes two types of CP proteins, the major CP of 33kDa and a minor CP (Kreuze *et al.*, 2002). Qin *et al.* (2013) have documented the complete genome sequences of two SPCSV isolates from China. SPCSV is transmitted by species of whiteflies (*Bemisia tabaci* Genn. and *Trialeurodes abutilonea* Westwood) in a semi-persistent non-circulative manner (Hedge *et al.*, 2012). The virus is phloem-limited and cannot be transmitted through sap and mechanically. Hosts of this virus are limited mainly to the genus *Ipomoea*, some species of *Nicotiana* and *Amaranthus palmeri* and wild species of lisianthus (*Eustoma randiflorium*) (Chen *et al.*, 2002; Byamukama *et al.*, 2004). Symptoms caused by the virus on sweet potato plant include mild stunt, chlorotic and purpling of leaves; and the symptoms varies considerably depending on the host. In some cultivars, the virus can be symptomless, whereas, in other symptoms such as mild vein yellowing, sunken secondary veins on abaxial leaf surfaces are observed (Hedge *et al.*, 2012). Coinfections of SPCSV with SPMMV or SPFMV have been reported to cause a break down of moderate and high level of resistance in sweet potato cultivars against SPMMV and SPFMV respectively (Mukasa *et al.*, 2006).

Sweet potato leaf curl virus (SPLCV) genus Begomovirus

Sweet potato leaf curl virus (SPLCV: family *Geminiviridae*, genus *Begomovirus*) is a DNA virus with geminate particles (18 x 30 nm) with infected cells having crystalline inclusions (Alegbejo, 2015). It has a monopartite genome (DNA-A, 2,828 nucleotides) and its organization is typical of Old World *Begomoviruses*, containing six open reading frames and an intergenic region containing a conserved stem-loop motif (Lotrakul and Valverde, 1999). Lotrakul and Valverde, (1999) determined the complete sequence of SPLCV (AF 104036, 2828 nts) its genomic DNA and organization have been reported to be similar to monopartite

Begomoviruses (Hedge *et al.*, 2012). The phylogenetic analysis of the partial sequence of different SPLCV isolates suggests that there may be more than one species of SPLCV (Hedge *et al.*, 2012). The occurrence of SPLCV has been reported in the United States, Taiwan, Korea, Argentina, Japan, Brazil, China (Loebenstein, 2015) and Ghana (Arkorful and Addae-Frimpomaah, 2015). In Nigeria, the virus occurrence is distributed in the Northern and South-Western States of the country where up to 30% yield reduction may occur if the infection is severe (Alegbejo, 2015). It is transmitted by whitefly (*Bemisia tabaci* Genn.) biotype B in a persistent manner and by grafting, but not mechanically or by seeds (ICTVdB Management, 2006). Various *Ipomoea* species were found to be susceptible to SPLCV. The virus induces typical leaf curl symptoms on *Ipomoea nil*, *I. setosa* and *Nicotiana benthamiana* (Hedge *et al.*, 2012). The most common symptom is upward curling or rolling of leaves on young plants. The rolled edge tends to be crinkled and vein swelling may be apparent. An interveinal chlorotic mottle is sometimes observed (Hedge *et al.*, 2012). Symptoms may appear seasonally and often disappear with time. Storage roots of infected plants have been reported to develop longitudinal grooves or ribs and this appears more pronounced when mixed infected with yields of virus-free plants of the cultivars ranged from 10 to 80% greater than the yields of SPLCV-infected plants in mixed infection (Ling *et al*., 2010; Hedge *et al.*, 2012).

Cucumber mosaic virus (CMD) genus Cucumovirus

Cucumber mosaic virus (CMV: family *Bromoviridae, genus Cucumovirus*) is considered the most widespread plant virus with the largest host range, infecting more than a thousand host species and limiting the profitable yield production of valuable crops (Zitter and Murphy, 2009; Jacquemond, 2012). The virus occurs in all ecological zones of Nigeria (Alegbejo, 2015) with infection of several vegetable (Arogundade *et al.*, 2010; Aliyu *et al.*, 2014; Ayo-John and Hughes, 2014; Odedara and Kumar, 2017; Adediji, 2019; Abraham *et al.*, 2019) legume and tuber (Alegbejo, 2015) crops been reported. CMV virions are icosahedral particles measuring 29 nm in diameter and contain 18% RNA and 82% single coat protein (Jacquemond, 2012). The RNA constitutes a tripartite single-stranded positive-sense biomolecules (RNA-1, -2 and -3) encoding five proteins which include the 1a, 2a, 2b, movement and coat proteins (Bujarski *et al.*, 2012). The thermal inactivation point is 55-70^{o}C; longevity *in-vitro* is 1-20 days while the dilution endpoint is 10^{-3}-10^{-6}. Particles are found in all parts of the host plant and specifically in the cytoplasm while the crystalline inclusion bodies are found in infected cells (Alegbejo, 2015). CMV is reported to be transmitted to sweet potato plants mechanically and by several species of aphids in a non-persistent manner (Dafalla, 2000). Conditions that favour the virus, host and aphid vectors will readily lead to an epidemic because several hosts harbour the virus (Alegbejo, 2015). *Cucumber mosaic virus* has been reported to severely infect sweet potato and the study indicated that elimination of the virus can lead to an increase in yield of up to 80% (Domola *et al*., 2008). Patil (2020) reported that a complete potato crop failure can occur following mixed infections by SPCSV and CMV.

Sweet potato virus disease (SPVD)

Sweet potato plant is known to be affected by various virus disease complexes. These complexes of viruses have been reported from different countries with SPFMV as one of the viral components in all cases (Kokkinos *et al.* 2006a; Mukasa *et al.* 2006). In Africa, severe sweet potato viruses occur by synergistic interaction of SPCSV transmitted by whitefly and SPFMV transmitted by aphids is commonly referred to as *Sweet potato virus disease* (SPVD) in literature. The yield loss caused by SPVD was reported to range from 80 – 90% and 37- 43% reduction of total carotenoids content in orange-fleshed sweet potato (OFSP) variety as compared with healthy plants (Kokkinos *et al.*, 2006a; Opiyo *et al.*, 2010). More recently, up to 78% yield reductions have been reported from field trials in Nigeria due to SPVD (Patil, 2020). The characteristic symptoms exhibit by sweet potato plants as a result of co-infection by SPFMV and SPCSV include leaf strapping, leaf distortion, puckering, vein clearing, chlorosis, and stunting (Kokkinos *et al.*, 2006b). SPCSV may broadly enhance the replication of several other sweet

potato viruses. (Mukasa *et al.*, 2006) reported that *Sweet potato mild mottle virus* is also enhanced by SPCSV, with virus titres increasing approximately 1000-fold. The combined infection caused severe symptoms, and the name sweet potato severe mosaic disease was suggested for the resulting disease.

Symptoms and yield effects of sweet potato viruses

Several viruses infecting sweet potato have been reported worldwide, but only a few have been shown to affect yield and especially when they occur in co-infections. Most of their damage is on tuber quality, size of leaves and storage roots. Economically important viruses infecting sweet potato worldwide are SPFMV, SPCSV, SPMMV and SPLCV. Globally, SPFMV is found wherever sweet potato is grown and some strains can induce root cracking (Cipriani *et al.*, 1999; Heisswolf *et al.*, 1994) and/or internal corkiness in susceptible cultivars (Gibson *et al.*, 2000). Yield losses of up to 50% as a result of SPFMV infection have been reported (Ateka *et al.*, 2007; Njeru *et al.*, 2004). Yield losses of up to 90% have been reported in plants affected with SPVD (Opiyo *et al.*, 2010). SPVD is caused by a synergistic interaction between a *Potyvirus*, *Sweet potato feathery mottle virus* (SPFMV), and a *Crinivirus*, *Sweet potato chlorotic stunt virus* (SPCSV). Sweet potato plants co-infected with SPFMV or other sweet potato *Potyviruses* and SPCSV exhibit severe symptoms such as leaf strapping, vein clearing, leaf distortion, chlorosis, puckering, and stunting. Symptoms on infected plants as a result of SPFMV infection alone are rare except when first infecting (Opiyo *et al.*, 2010) when symptoms can include leaf mottling, vein chlorosis, dwarfing and poor growth. Symptoms caused by SPMMV are rare and include leaf chlorosis and rugosity in susceptible sweet potato plants when it occurs as a single infection and it seems to not affect yield. The virus is, like SPFMV, also synergized by SPCSV; leaf symptoms then include chlorosis, rugosity, leaf strapping and dark green island, and the disease is called sweet potato severe mosaic disease (SPSMD) and it reduces the storage root yield by up to 80% (Tugume *et al.*, 2010). SPCSV as the name indicated, it caused stunts of infected plant and can also cause reddening, purpling or chlorotic yellowing of leaves (Njeru *et al.*, 2004). Although these symptoms can be mild or absent, however, yield loss of up to 50% has been reported (Tugume *et al.*, 2010). Yield reduction of up to 26% of affected Beauregard cultivar in the U.S.A has been reported as a result of SPLCV infection and caused grooving and skin darkening (Paprotka *et al.*, 2010) but the disease rarely causes interveinal chlorosis and leaf curling symptoms in infected plants (Cuellar *et al.*, 2015).

Management of sweet potato viruses

Virus diseases cause severe sweet potato yield losses worldwide and their control is further complicated under subsistence cropping systems (Rukarwa *et al.*, 2010). As there are no practical therapeutic measures for plant viruses, thus, plant virus management focuses on preventative measures (Ownley and Trigiano, 2016). Integrated disease management strategies such as good cultural practices, phytosanitary measures, planting of virus-free vines, control of vectors and deployment of genetic resistance to prevent or limit the extent of damage have been considered the best line action (Maule *et al.*, 2007; Van den Bosch *et al.*, 2007, Patil, 2020). Among these, the use of disease-resistant cultivars and control of virus vectors are ideal for effective and sustainable management of sweet potato virus diseases (Barkessa, 2018). Potyviruses of sweet potato are managed through reducing virus inoculum by using limited generation ‘seed’ that was initially virus free, continually flushing out the diseased material (Clark *et al*., 2012). For the SPLCV, the management of whitefly is a critical element for its management (Clark *et al*., 2012). Planting cuttings taken from symptomless parent plants which mostly excludes planting SPVD-affected material is considered by the farmers to be one of their most effective means of managing SPVD (Clark *et al*., 2012). Three commonly cultivated varieties (*Dan Madakala* cultivar (CV1) *Dan gwaranyo* cultivar (CV3) and *Dan Ayi* cultivar (CV4)) in Kebbi State, Nigeria were reported to show mild symptoms after being inoculated with SPFMV and were recommended for farmers in Kebbi State (Mohammed *et al.*, 2019).

Conclusion

Although over thirty (30) viruses were known to infect sweet potato worldwide, only five of these viruses (SPFMV, SPMMV, SPCSV, SPLCV, CMV) have been reported in Nigeria. These viruses are, however, among those considered to be of major global economic importance and are therefore of economic concern to sweet potato farmers in Nigeria. There is a need for diagnosis of other economically important viruses of sweet potato in the country as a prerequisite for their management. Further research on the epidemiology, yield loss assessment, molecular characterization and other functional studies of the prevalent viruses are areas that also need to be explored. Integrating the use of resistant varieties, management of the *virus* vectors and tactical manipulation of the routine cultural practices of sweet potato against viruses or their vectors are worthwhile for effective management of virus diseases of sweet potato. Sweet potato farmers in the country need to be aware of these viruses, the menace they cause on sweet potato production and their management strategies.

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