

## Review

# Sweet potato breeding for resistance to sweet potato virus disease and improved yield: Progress and challenges

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Sweet potato is one of the main staple food crops for millions of subsistence farmers in Africa. Biotic and abiotic stresses and socio-economic challenges are the major production constraints of the crop. Amongst biotic constraints, the sweet potato virus disease (SPVD) is the most devastating causing yield reduction ranging from 50 to 98%. This paper highlights the progresses and challenges of breeding sweet potato towards improved yield and SPVD resistance. Further, the potential and limitations of non-conventional breeding techniques for sweet potato improvement have been reviewed. Both improved cultivars and landraces that are presently grown succumb to SPVD and several viral diseases. The yield losses caused by SPVD have significant negative impact on food security and income for the rural poor. Continued use of susceptible varieties, absence of high yielding and early maturing resistant varieties, and lack of effective control measures to SPVD contribute to low yields and disease build up, development and persistence. Both chemical and biological control methods are not effective against viral diseases. The use of resistant varieties remains the most effective and cheapest method for small-scale farmers. Breeding for resistance against SPVD is the most important component to improve yield and reduce the impact of SPVD. Reduced flowering and fertility, self- or cross-incompatibility are the major challenges of conventional breeding in sweet potato breeding. The use new breeding techniques such as marker-assisted selection and genetic engineering could have complementary roles in sweet potato breeding. This review provides theoretical bases on the progress and challenges for breeding sweet potato for SPVD resistance and improved yields.

**Key words:** Breeding, resistance, sweet potato virus disease (SPVD), sweet potato, viral disease, yield.

## INTRODUCTION

Sweet potato (*Ipomoea batatas* (L.) Lam.;  $2n=6x=90$ ) is a perennial plant cultivated as an annual crop. It is a dicotyledonous and belongs to morning glory family Convolvulaceae (Martin, 1970; Huaman, 1992; Reddy et al., 2007; Troung et al., 2011). Principally, sweet potato is grown for its storage roots for food security and income generation (Diaz et al., 1996; Tairo et al., 2004). It has

supported more people per square unit than any other crop (Okada et al., 2002). The genus *Ipomoea* consists of about 600 to 700 species including sweet potatoes (Vaeasey et al., 2008; Cao et al., 2009). The series Batatas consists of 13 species closely related to cultivated sweet potatoes (Orjeda et al., 1990; Diaz et al., 1996; Huang and Sun, 2000; Rajapakse et al., 2005;

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Srisuwan et al., 2006; Nimmakayala et al., 2011). Further, section *Batatas* consists of three cytogenetic groups, namely; group A, B and X; while A and X are self- and cross- compatible, group B where sweet potato belongs is self-incompatible but cross-compatible (Nishiyama et al., 1975; Kobayashi et al., 1993; Diaz et al., 1996; Kowyama et al., 2000). Central America has been documented as the origin and the primary centre of diversity of the currently cultivated sweet potatoes (Zhang et al., 2000; Gichuki et al., 2003; Srisuwan et al., 2006; Low et al., 2009). On the other hand, East Africa is one of the secondary centres for sweet potato diversity (Gichuki et al., 2003). Sweet potato is believed to be introduced to Africa by Portuguese during 16<sup>th</sup> and 17<sup>th</sup> century (Zhang et al., 2000; Gichuki et al., 2003).

Sweet potatoes are grown from 48°N to 40°S of the equator with altitudes ranging from 0 to 3000 m above sea level (Woolfe, 1992; Vaeasey et al., 2008; Low et al., 2009; Troung et al., 2011). The crop requires ambient day and night temperatures from 15 to 33°C for optimum growth and root development. Temperature above 25°C is considered optimal for maximum growth (Woolfe, 1992). However, temperatures below 12 and above 35°C retard sweet potato growth (Kuo, 1991). Dry matter production increases with increasing temperatures from 20 to 30°C, but declines at temperatures beyond 30°C (Kuo, 1991). The crop grows best with a well distributed annual rainfall of 600 to 1600 mm (Low et al., 2009). Excess rainfall at early stage of establishment may aggravate weed problem resulting in low yield (Harrison and Jackson, 2011). The crop is extensively grown under rainfed conditions and is relatively drought tolerant. However, prolonged and frequent dry spells or drought and erratic rainfall cause substantial yield reduction (Low et al., 2009; Schaffleitner et al., 2010). Sweet potato requires well-drained soil with a pH of 5.5 to 6.5 (Woolfe, 1992). It also requires full sunlight; however, it can tolerate a 30 to 50% reduction of full solar radiation (Troung et al., 2011).

Flowering ability is an essential aspect in sweet potato breeding and determines the potential for crop improvement through breeding (Gasura et al., 2010). Sweet potato flower contains both male and female reproductive organs for sexual reproduction (Jones, 1980). The flowers are born solitarily and grow vertically upward from the leaf axis (Huaman, 1992). Each flower has five united sepals and five petals joined together to form a funnel-shaped corolla tube (Jones, 1980; Huaman, 1992). The tube is usually lavender coloured and is the most conspicuous part of the flower (Jones, 1980). Five stamens with varying heights are attached to the base of the corolla tube (Jones, 1980). In most cultivars the two longest stamens are about the same length as the style. The filaments vary in length and are hairy and, anthers are either white or yellow or pink and contain numerous pollen grains on their surfaces (Huaman, 1992). The ovary consists of two carpel, each

containing one locule (Orjeda et al., 1991; Mont et al., 1993). Each locule contains either one or two ovules, with a maximum of four ovules per ovary (Jones, 1980; Huaman, 1992).

Sweet potato flowers mostly under short day length, however, long day and day neutral cultivars exist (Jones, 1980; Troung et al., 2011). However, most sweet potato cultivars are sensitive to daylength. Hence, some genotypes flower readily at any season while others only when days are short (Jones, 1980). Short days promote flowering and growth of storage root. Still others do not flower under any normal conditions. Those that do not flower readily can often be induced to flower by grafting on other *Ipomoea* species (Chiona, 2009). Sweet potato cultivars differ in their flowering ability, some do not flower, others produce very few flowers or flower profusely depending on the genotypes and environmental influences (Jones, 1980; Huaman, 1992). On the other hand, non-flowering genotypes pose challenges in exploiting their genes via the conventional breeding programmes.

The flowers open soon after daybreak and wither depending on prevailing environmental conditions (Jones, 1980). Flowers open longer on cool and cloudy days compared to hot and sunny days. Pollination can be facilitated either by insects or hand. In either case, the male pollen grain lands on the stigma, initiating fertilization. The pollen germinates few minutes to 3 or 4 h after pollination (Martin and Cabanillas, 1966; Jones, 1980; Kowyama et al., 2000). The pollen tube grows down the style until it meets the female gametophyte in 8 h after pollination (Martin and Cabanillas, 1966; Jones, 1980). Pollen may be rejected shortly after contacting the stigmatic surface resulting in pollen germination failure (Kowyama et al., 2000). With normal fertilization and embryo development up to four seeds can be produced per ovary (Jones, 1980; Mont et al., 1993). However, successful fertilization is uncertain, possibly due to embryo and fruit abortions (Mont et al., 1993). Gasura et al. (2010) reported higher fertilization successes for flowers pollinated in early than late hours of the day. Additionally, insect pollination produce more seeds compared to hand pollination (Nishiyama et al., 1975; Gasura et al., 2010). The low fertility or fertilization failure could be due to incompatibility contributed by hexaploid genome of the crop. Besides incompatibility, other environmental and management practices also affect the amount of seeds produced in the ovary. Weed management and controlled application of nitrogen fertilizer improve seed setting (Jones, 1980).

The sweet potato fruit is a capsule containing one to four seeds (Huaman, 1992). The seeds are black and about 3 mm long; also they are flat on one side and convex on the other (Huaman, 1992; Chiona, 2009). The seeds remain viable for many years with extended dormancy period probably due to thick, hard and impermeable testa (Huaman, 1992; Chiona, 2009). This

has implication on seed germination. Therefore, mechanical or chemical scarification is necessary for improved germination (Huaman, 1992; Ernest et al., 1994). Nevertheless, the production of sweet potato is constrained by several biotic, abiotic and socio-economic factors (Thottappilly and Loebenstein, 2009). Amongst the most important biotic constraints are sweet potato virus diseases. The objective of this paper is to highlight the progresses and challenges of breeding sweet towards improved yield and SPVD resistance. Further, the potential and limitations of non-conventional breeding techniques for sweet potato improvement have been reviewed.

## SWEET POTATO PRODUCTION CONSTRAINTS

### Biotic constraints

The production of sweet potato is affected by several biotic constraints such as viral diseases, insect pests and weeds (Ndunguru et al., 2009; Lou et al., 2010; Schafleitner et al., 2010; Harrison and Jackson, 2011). Diseases and insects of paramount importance are sweet potato virus diseases and sweet potato weevils, respectively. Sweet potato virus disease (SPVD) caused by the dual infection and synergistic interaction of sweet potato chlorotic stunt virus and sweet potato feathery mottle virus is distributed worldwide (Gibson et al., 1998; Mukasa et al., 2006). It is the most devastating disease causing reduction in plant growth and storage root yields (Gibson et al., 1997; Karyeija et al., 2000; Gibson et al., 2004; Gibson, 2005; Kapinga et al., 2009a). Also SPVD limits the length of time the roots can be kept in the ground and shorten the storage duration of the harvested crop (Engoru et al., 2005; Tsakama et al., 2010). The damage caused by SPVD ranges from 50 to 98% (Gibson et al., 1998; Njeru et al., 2004; Tairo et al., 2004). On the other hand, sweet potato weevils, *Cylas* spp., is another major sweet potato production constraint (Kapinga et al., 2003; Stathers et al., 2003; Munyiza et al., 2007; Korada et al., 2010). The weevils tunnel and feed on vines and storage roots thereby reducing the quality and yield of the crop (Mullen, 1984; Stathers et al., 1999). According to Stanthens et al. (1999), yield losses from weevils infestation can be as high as 100%. Moreover, infestation levels are highest under dry conditions due to many cracks which appear when the soil dries (Muyinza et al., 2007). Other biotic constraints such as millipedes, *Alternaria* leaf spot, stem blight, black rot, *Fusarium* rot, bacterial rot, nematodes and vertebrate pests such as rats are also a threat to sweet potato production (Kapinga et al., 1995; Johanson and Ives, 2001; Ebregt et al., 2004; Fugile, 2007). In addition, weeds may cause severe yield losses when high rainfall occurs early in the growing season (Harrison and Jackson, 2011).

### Abiotic constraints

Abiotic constraints which significantly affect sweet potato production include low soil fertility and drought (Kapinga et al., 1995; Mwololo et al., 2007; Mihale et al., 2009; Pareek et al., 2010). Declining soil fertility constrains sweet potato production as its replenishment is limited by unaffordable high prices of inorganic fertilizers (Mudiope et al., 2000; Elliott and Hoffman, 2010). Moreover, degraded and depleted soils make applied fertilizers less effective. Continuous cropping without addition of organic and inorganic manures has led to a decline in soil fertility and consequently a decline in productivity (Saleh and Zahor, 2007).

Drought is a significant abiotic constraint that limits the productivity of not only sweet potato but also many other crops and affects both the quality and quantity of yield (Cattivelli et al., 2008; Collins et al., 2008; Balouchi, 2010). Although it is documented that sweet potato is drought tolerant, prolonged and frequent dry spells and erratic rainfall cause substantial yield reduction (Johanson and Ives, 2001; Liwenga and Kangalawe, 2009; Schafleitner et al., 2010). Drought not only affects crop growth and development, but also root yield, dry matter content and composition, and pests and disease incidences (Mcharo and Carey, 2001; Ekanayake and Collins, 2004; Masumba et al., 2005). Mwololo et al. (2007) reported an increased incidence and severity of sweet potato viral diseases in the event of drought. For instance, besides low dry matter content and susceptibility to viral diseases, the newly introduced orange fleshed sweet potatoes (OFSP) are unable to withstand drought, which leads to low productivity and unacceptability to farmers (Mwanga and Ssemakula, 2011). Sweet potato varieties less tolerant to drought significantly retard the efforts invested by farmers making them unpopular and subsequently rejected. Gibson (2005) reported that the participatory sweet potato breeding and selection trials were ruined by drought and farmers rejected the less drought tolerant varieties. Therefore, together with other constraints, the production of sweet potato is also significantly affected by drought leading to low productivity.

### Socio-economic constraints

There are several socio-economic constraints which affect sweet potato production. These include inadequate availability of high yielding, disease resistant planting materials, poor or no fertilization and weeding, and lack of post-harvest technologies (Rees et al., 1998; Mudiope et al., 2000; Mpagalile et al., 2003; Kulembeka et al., 2005; Tairo et al., 2005; Mwololo et al., 2007; Ndunguru et al., 2009; Schafleitner et al., 2010). The use of infected, low yielding planting materials contributes significantly to persistence of sweet potato viral diseases

(Mwololo et al., 2007; Opiyo et al., 2010). Inadequate extension services limits dissemination and adoption of improved husbandry practices. Consequently, farmers continue growing informally disseminated inferior planting materials, which lead not only to persistence of diseases but also negatively affect productivity and profit of the crop (Kapinga and Carey, 2003; Fugile, 2007). Similarly, poor linkage between farmers and other stakeholders coupled with undeveloped and fragmented infrastructures in rural areas significantly lowers the productivity of the crop (Kapinga and Carey, 2003; Waddington et al., 2010). Further, inadequate post-harvest technologies such as storage facilities and processing technologies severely affect investment, production and sustainability of the crop (Mpagalile et al., 2003; Fugile, 2007; Waddington et al., 2010; Hu et al., 2011).

Low production of sweet potato is also contributed by lack of high yielding varieties with farmers' preferred traits (Karuri et al., 2009). High yielding and farmers' preferred varieties are the bases for increased productivity and sustainable development of the crop. Presently, most farmers use local landraces. Though adapted to local agro-ecologies, the landraces are low yielding and late maturing (Gibson et al., 1998; Masumba et al., 2005). Likewise, sweet potato is one of the most under-exploited crop and breeding initiatives are at a relatively early stage compared to other crops such as maize, rice and cassava (Kriegner et al., 2003; Gasura et al., 2010). In the past, attempts were made to use exotic varieties in various agro-ecologies to address low productivity and circumvent pest and disease damages (Kapinga et al., 2009b; Gasura et al., 2010). Nevertheless, the exotic varieties have shown relatively poor performance compared to landraces which are well adapted to the farming systems (Abidin et al., 2005b; Gasura et al., 2010). Mwangi et al. (2007) and Mwangi and Ssemakula (2011) reported almost 100% failure of the newly introduced orange-fleshed sweet potatoes in Uganda. Similar studies in Tanzania indicated that, some of the introductions were rejected by farmers due to low root yields and dry matter content, and poor production of vines during recurrent droughts (Kulembeka et al., 2005). On the other hand, relatively similar performance of the local unimproved and introduced improved varieties for both yields and adaptability to different agro-ecologies has been reported (Mbwaga et al., 2007). This underpins the need for further sweet potato research and development.

## **SWEET POTATO VIRUS DISEASES**

Sweet potatoes are invariably affected by bacteria, fungal and viral diseases, and nematode (Clark et al., 2009; Thottappilly and Loebenstein, 2009). Different diseases attack the crop at different stages of growth, from pre-harvest to post harvest (Dje and Diallo, 2005). The levels

of damages due to diseases and pests depend on the causal agent, intensity of infestation, variety and prevailing environmental conditions (Thottappilly and Loebenstein, 2009). Viral diseases cause substantial yield losses in farmers' fields (Wambugu, 2003).

Viral diseases are amongst the important biotic constraints and severely affect sweet potato production (Gutiérrez et al., 2003; Wambugu, 2003). They are the most devastating and occur in all sweet potato growing areas (Tairo et al., 2004; Mwololo et al., 2007; Ndunguru et al., 2009). The most important sweet potato virus diseases include sweet potato feathery mottle virus (SPFMV), sweet potato chlorotic stunting virus (SPCSV), sweet potato mild mottle virus (SPMMV) and sweet potato chlorotic fleck virus (SPCFV) (Feng et al., 2000; Tairo et al., 2004). Sweetpotato mild speckling virus (SPMSV), sweet potato virus G (SPVG) and sweet potato latent virus (SPLV) have also been reported to affect sweet potato (Feng et al., 2000; Ndunguru and Kapinga, 2007). These viruses not only adversely affect sweet potato yields and quality but also decrease plant resistance to insect pests (Feng et al., 2000; Bryan et al., 2003; Yang, 2010). An infection by single virus strain causes little yield losses compared to co- or multiple-infections that cause the complex sweet potato virus disease (SPVD) (Ames de Icochea and Ames, 1997; Karyeija et al., 2000).

Sweet potato virus disease (SPVD) severely affects sweet potato production (Gutiérrez et al., 2003; Kokkinos et al., 2006). It is caused by dual infection and synergistic interaction of sweet potato chlorotic stunting virus (SPCSV); family *Closteroviridae*, genus *Crinivirus* and sweet potato feathery mottle virus (SPFMV); family *Potyviridae* genus *Potyvirus* (Karyeija et al., 1998; Untiveros et al., 2008; Kreuze et al., 2009). Sweet potato feathery mottle virus is non-persistently transmitted by aphids while sweet potato chlorotic stunting virus is semi-persistently transmitted by the whitefly [*Bemisia tabaci*] (IsHak et al., 2003; Kokkinos et al., 2006; Untiveros et al., 2008). In some incidences, co-infection of sweet potato chlorotic stunting virus and sweet potato mild mottle virus (SPMMV) occurs (IsHak et al., 2003; Mukasa et al., 2006). Further, not only dual co-infection but also triple infections occur resulting into most severe disease symptoms and yield losses (Gibson et al., 2004; Tairo et al., 2005; Mukasa et al., 2006; Kapinga et al., 2009a). The symptoms and damage of co-infection are more severe and devastating than individual viral disease (Feng et al., 2000; Karyeija et al., 2000; Mukasa et al., 2006). The SPVD symptoms and damages are subject to its incidences and severity.

The incidences and severity of SPVD are highly variable. They vary between and within agro-ecologies, between varieties and growth stages of plants (Gibson et al., 2000; Mwololo et al., 2007; Kapinga et al., 2009b; Gasura and Mukasa, 2010). The disease is characterized by stunted growth, chlorotic and malformed leaves, and

ultimately reduced yields (Gutiérrez et al., 2003; Gibson et al., 2004; Untiveros et al., 2008). The SPVD infection causes yield losses as high as 98% (Feng et al., 2000; Gibson et al., 2000; Gutiérrez et al., 2003; Mukasa et al., 2006). Bryan et al. (2003) observed a decrease in root diameter and yield due to presence of SPFMV and other potyviruses. The disease not only decreases yields, but also lowers quality and resistance to other pathogen (Bryan et al., 2003; Domola et al., 2008). In severe incidences, SPVD can lead to abandonment extinction of elite cultivars (Bryan et al., 2003; Gasura and Mukasa, 2010; Rukarwa et al., 2010).

The SPVD is persistent in farmers' fields due to several predisposing factors. Lack of knowledge among farmers, predominantly use of aged vegetative propagating materials, susceptible landraces, and high temperatures favour development, spread and expression of the disease (Kreuze, 2002; Ateka et al., 2004; Tairo et al., 2004; Mwololo et al., 2007; van den Bosch et al., 2007; Ndunguru et al., 2009). Also, the use of healthy-looking vines collected from the previous to the succeeding cropping cycles contributes to the spread of the disease (Opiyo et al., 2010; Rukarwa et al., 2010). Bryan et al. (2003) observed early development of disease symptoms from transplants infected with viruses compared to uninfected transplants which consequently led to decline in yield and root quality. Aritua et al. (2007) observed high virus incidences in bimodal rains compared to unimodal rain in a year. On the other hand, Nduguru et al. (2007) found lower incidences and severity of SPVD in cooler compared to warmer agro-ecologies and where the crop was grown in only one season per year. Further, prolonged, hot and dry spells provide natural breaks in the transfer of viruses between crop cycles. In endeavour to reduce the incidences and effects of SPVD, several strategies such as phytosanitation and breeding for resistant cultivars have been recommended (Tairo et al., 2004; Valverde et al., 2007).

### **Strategies to control SPVD**

Adequate management of plant disease is amongst the prerequisite for stable and profitable crop production for ascertained food security. Plant viruses are a major problem in the cultivation of many crops. There is no effective and complete control method against the disease to date. The control of viral diseases remains difficult in subsistence cropping systems (Rukarwa et al., 2010). Both chemical and biological control methods are not effective against viral diseases (García-Arenal and McDonald, 2003; Dje and Diallo, 2005; Maule et al., 2007). Several strategies such as cultural practices, phytosanitary measures, control of vectors and deployment of genetic resistance to prevent or limit the extent of damage have been recommended (Maule et al., 2007; van den Bosch et al., 2007). On the other hand,

control of SPVD has been mainly by use of clean and virus-free planting materials and resistant varieties (Aritua et al., 1998; Gibson et al., 2000). The use of clean and disease free planting materials, sanitation and other cultural practices effectively contribute to the control of the disease (Tairo et al., 2005; Miano et al., 2008). Karyeija et al. (1998) and Thottappilly and Loebenstein (2009) suggested that, use of virus-free and certified planting materials are likely to significantly reduce the effects of SPVD. However, deployment of genetic resistance to virus disease is viewed as the most effective and sustainable approach for managing SPVD (García-Arenal and McDonald, 2003; Maule et al., 2007).

### ***Cultural practices to control SPVD***

Virus infected plants cannot be cured and the only way to adequately protect the crops is the use of resistant cultivars (Kreuze, 2002). The use of resistant varieties is cheap, easy, safe, effective and environmentally friendly (Okada et al., 2001; Byamukama et al., 2002; García-Arenal and McDonald, 2003; Valverde et al., 2007). The impact of SPVD in farmers' fields has been reduced by the use of resistant cultivars and landraces (Miano et al., 2008). However, the local landraces are highly variable in their resistance to SPVD. Most varieties are resistant to SPFMV but this resistance breaks down in the event of co-infection with SPCSV resulting in redundant resistance (Tairo et al., 2004; van den Bosch et al., 2007; Gasura and Mukasa, 2010).

Further, the sweet potato grown by farmers are landraces with build-up of viruses resulting from several generations of vegetative propagation (Fugile, 2007; Miano et al., 2008; Low et al., 2009). In general, resistant varieties are rarely available in addition to being low yielders and late maturing (Gibson et al., 2004; Abidin et al., 2005a; Miano et al., 2008). Therefore, improving virus resistance through development and deployment of SPVD resistant and high yielding varieties would improve production, productivity and ensure food security for subsistence farmers.

Improved phytosanitation offers considerable benefits for controlling SPVD (Muturi et al., 2007). Phytosanitary measures include quarantine, sanitation, use of virus-free vegetative propagules for all new plantings and roguing of diseased plants from within plantings (Thresh, 2003). Roguing, the removal of all plants showing disease symptoms has been reported to decrease the population of whitefly, a vector responsible in spreading SPVD (Karyeija et al., 1998; Muturi et al., 2007; Valverde et al., 2007). Also, Ndunguru et al. (2009) and van den Bosch et al. (2007) reported that, roguing of infected plants may form an effective way of minimizing SPVD incidence and its damage to sweet potato production. Gibson et al. (2000) and Gibson et al. (2004) found that, Tanzanian and Ugandan farmers controlled SPVD by using

symptomless plants to establish new crop and destroying all infected plants. On the other hand, control of vegetation closer to sweet potato fields is likely to significantly reduce vectors' population thereby reducing incidences of SPVD. Muturi et al. (2007) observed drastic increase in whitefly populations in experimental plots surrounded by maize plants. Contrastingly, Gutiérrez et al. (2003) used maize as an integrated pest management to control whitefly and aphid population to reduce virus transmission. Further, avoidance of introducing new infections in a new field by isolating new plots from SPVD-affected ones can effectively reduce spread and incidences of SPVD (Gibson et al., 2004; Domola et al., 2008). Moreover, Gibson et al. (1998) recommended enforcement of phytosanitary controls to prevent introduction of new and severe viral strains between regions.

Another approach to circumvent the damage caused by viral infection is the production and use virus-free plants through shoot tip culture (Feng et al., 2000; Okada et al., 2002; Rukarwa et al., 2010). The use of health planting materials contributes significantly to the control of viral diseases including SPVD. The approach is effective in eliminating sweet potato viruses. Hannington et al. (2002) reported that, an inadequate quantity of clean planting materials was amongst the causes of persistent low yields of sweet potato in farmers' fields in Kenya.

According to Feng et al. (2000) and Gutiérrez et al. (2003), the use of virus-free sweet potato is likely to restore cultivar's original yield, quality and improve resistance to other pathogens and insects. Further, the use of virus-free sweet potato planting materials has been recommended to be among the most effective way to circumvent the losses caused by viruses (Opiyo et al., 2010). Aritua et al. (2003) reported that farmers in Uganda selected cuttings from new unaffected crops to control SPVD thereby reducing disease incidences and yield losses. Nevertheless, the use of clean and virus-free planting materials is economically viable provided there is effective and efficient system for production, multiplication and distribution of planting materials (Carey et al., 1999; Feng et al., 2000). However, commercialization of sweet potato production is a major challenge in many developing countries particularly in Sub-Saharan Africa (SSA) as the crop is mainly grown for household subsistence (Valverde et al., 2007). The capacity of public institutes to sustainably produce and multiply clean and virus-free planting materials for low income farmers in these countries is uncertain. Research institutes are financially constrained and farmers lack purchasing power to multiply and distribute, and purchase improved healthy planting materials, respectively (Mtunda et al., 2003). Rukarwa et al. (2010) reported that, inadequacy of production, multiplication and distribution of certified virus-free planting materials was a major setback in sweet potato production in Uganda. Therefore, economic and infrastructure

constraints are likely to significantly limit establishment and development of clean and virus-free planting material schemes.

### ***Control of SPVD vectors***

viruses including SPFMV and SPCSV, the major components of SPVD are transmitted by aphids and whiteflies, respectively. The control of these vectors is likely to contribute significantly to the control of SPVD. The control of the vectors may involve varied practices such use of chemicals, eradication of weeds and other virus sources (Hull, 1994; Thresh, 2003). However, the control of vector populations under field conditions has proven to be difficult and seldom used in sweet potato (van den Bosch et al., 2007). Ames et al. (1997) pointed out that controlling whiteflies is not usually an effective means of limiting the incidence of the viruses they transmit. Also, Ndunguru et al. (2009) reported the absence of correlation between the number of whiteflies and severity of SPVD. Further, the control of insect vectors may not be economically viable as sweet potato is not well commercialized and is largely grown by subsistence farmers (Rukarwa et al., 2010).

### ***Deployment of sweet potato resistant germplasm to control SPVD***

Sweet potato is commercially propagated using stem cuttings. Botanically, true seeds have been exclusively used for breeding programmes (Sihachakr et al., 1997; Gaba and Singer, 2009). In farmers' fields sweet potato seeds or seedlings have not been considered as a source of diversity (Gibson et al., 2000). In sweet potato improvement programs genetic variation has largely been enhanced through conventional hybridization. The approach has some limitations due to biological nature of the crop (Yi et al., 2007; Shin et al., 2011). Genetic improvement of sweet potato has been challenging due to their heterozygous genetic constitution, polyploidy, self-incompatibility and cross-incompatibility (Zhang et al., 2000; Mwanga et al., 2002b; Okada et al., 2002). Sweet potato has hexaploid number of chromosomes ( $2n = 6x = 90$ ) (Martin and Ortiz, 1967; Magoon et al., 1970; Nishiyama et al., 1975; Orjeda et al., 1990; Kowiyama et al., 2000). This large number of chromosomes has implications on meiotic irregularity. Sexual compatibility barriers associated with hexaploidy nature restricts hybridization within the species (Jones, 1980; Diaz et al., 1996). The barriers are either genetic or cytogenetic or physiological and their interactions. Also, its genetic improvement is largely limited by sterility and incompatibility (Ting and Kehr, 1953; Martin, 1968, 1970; Jones, 1980; Kowiyama et al., 2000). Relatively few genetic studies on sweet potato could be largely be due

to reproductive barriers from self-incompatibility, high levels of cross-incompatibility, polyploidy and reduced or absence of flowering in some genotypes (Martin and Ortiz, 1967; Magoon et al., 1970; Okada et al., 2002; Cao et al., 2009; Chang et al., 2009; Shin et al., 2011). Incompatibility is caused by pre- and post-fertilization barriers (Martin and Ortiz, 1967; Martin, 1970; Kowyama et al., 1980; Kobayashi et al., 1993). The system of SI in sweet potato and other species in genus *Ipomoea* is homomorphic sporophytic incompatibility controlled by a single multiple alleles at S-locus (Martin, 1968; Kowyama et al., 1980; Diaz et al., 1996; Kowyama et al., 2000; Tomita et al., 2004). This system causes complete failure of pollen germination on the stigma after self-fertilization (Martin, 1970; Kowyama et al., 2000; Tomita et al., 2004). Martin (1968) suggested presence of duplicated incompatibility loci with epistatic interaction. On the other hand, Kowyama et al. (1980) suggested the presence of either dominance or independence or competitive relationships among multi-alleles controlling sporophytic incompatibility.

Self-incompatibility prevents self-fertilization while promoting cross-fertilization (Byers and Meagher, 1992; Tseng et al., 2002). However, cross-fertilization is not guaranteed due to cross-incompatibility (Martin, 1970; Tseng et al., 2002). According to Diaz et al. (1996), complex genetic, cytogenetic and physiological interactions, greatly influence interbreeding in the section *Batatas*. It plays a role in maintaining genetic diversity but limits genetic improvement due to cross-incompatibility (Nishiyama et al., 1975; Tomita et al., 2004). Despite the SI gene, high degree of cross-incompatibility and other barriers such as male sterility (Elameen et al., 2011; Liu, 2011), genetic improvement of sweet potato by either conventional or biotechnology means are necessary.

**Conventional sweet potato breeding for SPVD resistance:** Breeding for virus resistant cultivars has been recommended as the long-term solution to sustainably control SPVD and other viral diseases (Domola et al., 2008). However, breeding for SPVD resistance has not been an easy endeavour. Lack of resistant, high yielding and locally adapted varieties have given farmers limited alternatives to susceptible high yielding local varieties or landraces (Gibson et al., 2000). Therefore, incorporation of resistance genes into susceptible but high yielding landraces is a preferred strategy for managing crop diseases. This is the direct and effective strategy for long term control of viral diseases (Hull, 1994; Carey et al., 1999; Mihovilovich et al., 2000; Fraile et al., 2011). Jones et al. (1986) recommended that, “no matter which insect species infecting the plant, genetic resistance should be considered as the possible solution; even intermediate level of resistance can be of significant economic importance”. Efficient and effective breeding systems are likely to effectively contribute to the release of superior

and resistant cultivars to control SPVD (Gibson et al., 2000, 2004). Progress on breeding for SPVD resistance has been made in several countries including Uganda, United States, Japan, China, Taiwan and Peru (Carey et al., 1999; Mwangi et al., 2002b; Tairo et al., 2005; Lebot, 2010). For instance, in Uganda a number of sweet potato varieties namely NASPOT 1, 5, 6, 7, 8, 9, 10 and 11 which are resistant to SPVD have been released from 1999 to 2010 (Mwangi et al., 2009; Gibson et al., 2011; Mwangi et al., 2011). Some of the varieties are grown commercially and others are being used in breeding programmes in different countries such as Uganda, Tanzania, Kenya and Ethiopia.

Emphasis in developing resistance to SPVD has largely focused on resistance to SPFMV, an important component of SPVD (Mwangi et al., 2002b; Valverde et al., 2007). This resistance breaks down in co- or multi-infections with SPCSV and SPMNV. Breakdown of resistance by different strains or highly virulent viruses leaves the resistance redundant (Miano et al., 2008; Kreuze et al., 2009). This implies that, resistant cultivars developed such as in Peru and other parts of the world might be of little value in other environments due to presence of different viral strains. The international potato center (CIP) identified some clones resistant to SPFMV after exhaustive germplasm screening; however the selections succumbed to the SPVD in Uganda (Karyeija et al., 1998). Further, Gibson et al. (1998), Karyeija et al. (1998) and Mwangi et al. (2002b) reported that, resistant varieties in West Africa and Peru succumbed viral diseases in East Africa, possibly due to different strains of viruses. Even in the same region, resistant cultivars still succumbed to SPVD (Tairo et al., 2005). Therefore, this underpins the use of local germplasm in breeding for SPVD resistant varieties than heavily depending on exotic introductions (Gasura et al., 2010). The resistance of landraces could have been attributed by co-evolutionary processes which led to accumulation of resistance genes in the host population due to dynamic pathogen population (host-parasite co-evolution) (Ghazvini and Tekauz, 2007; Anderson et al., 2011; Fraile et al., 2011). Plants have diverse mechanisms to survive and adapt to broad range of biotic and abiotic stresses. Ulukan (2009) pointed out that most field crops have in-built protection mechanism against diseases, pests and vermin. Therefore, there is a need to identify and use local germplasm in breeding for SPVD resistant varieties (Gibson et al., 1998; Gasura et al., 2010). Despite its contribution in genetic deployment for disease resistance, conventional hybridization in sweet potato is constrained by its sterility, incompatibility and hexaploidy nature. Biotechnology or genetic engineering offers great potential for improving disease, pest or stress resistance in sweet potato (Liu, 2011).

**Marker-assisted breeding and genetic engineering:** Efficient methods to control the sweet potato virus

disease are not available and conventional breeding for resistance has limited success. Breeding for resistance through genetic engineering offers an alternative solution for the control of SPVD. For more than two decades non-conventional approaches have shown the potential to accelerate crop improvement. Plant tissue culture, regeneration techniques and development of transgenic plants are valuable tools for sweet potato improvement and development (Yi et al., 2007; Nyaboga et al., 2008; Yang, 2010; Liu, 2011). Some of the value added traits through genetic engineering include plant resistance to viral diseases (Jauhar, 2006). Feng et al. (2000) pointed out the potential of genetic engineering in virus-resistance breeding. Also, Chang et al. (2009) pointed the value of marker-assisted selection (MAS) to breeders for rapid determination of superior genotypes prior field maturity. For instance, Prakash and Varadarajan (1992) reported successful introduction of foreign marker genes into the genome of sweet potato through particle bombardment. Otani et al. (2003) and Yi et al. (2007) successfully introduced herbicide resistant *bar* gene in sweet potato cells and pointed the potential of combining it with other agronomically important traits for improvement of new sweet potato cultivars. Anwar et al. (2011) successfully produced transgenic plants from a diverse group of sweet potato cultivars that were tolerant to herbicide and indicated the possibility of generating transgenic plants for economically important groups of sweet potato. Therefore the use of transgenic technology appears to be an excellent option to protect crops against the devastating viral diseases including SPVD via pathogen-derived resistance or non-conventional protection to viral diseases (Hull, 1994; Kreuzer, 2002; Jauhar, 2006). Transgenic sweet potato resistant to SPVD through resistance to SPFMV have been developed in Kenya, China and other parts of the world (Okada et al., 2001; Hannington et al., 2002; Wambugu, 2003). However, the Kenyan transgenic sweet potato resistant to SPFMV is controversial. While Wambugu (2003) reported success; Ching (2004) reports "Broken promises; GM sweet potato turns project sour as the transgenic material did not quite withstand virus challenge in the field all lines tested were susceptible to viral attacks". Further, Hannington et al. (2002) reported that, despite a decade of transgenic sweet potato farmers did not receive the virus resistance transgenic sweet potato due to underdeveloped biosafety systems.

The transgenic resistance uses the viral coat protein (CP) gene to achieve resistance to SPFMV (Kreuzer, 2002). The international potato center (CIP) has used cysteine proteinase inhibitor to develop transgenic resistance to both SPFMV and SPCSV (Kreuzer, 2002). Nishiguchi et al. (1998) observed no significant difference in transgenic and non-transgenic sweet potato with regard to morphological and biological characters. Further, observed no significant differences of ELISA values between the inoculated-transgenic and the non-

inoculated-virus free plants to SPFMV. Nyaboga et al. (2008) observed increased resistance with less severe symptoms in transgenic plants than the non-transformed lines inoculated with a combination of SPFMV and SPCSV. Also, Okada and Saito (2008) reported that CP gene provided long term protection to transgenic sweet potato against SPFMV complex infection compared to the control and suggested that the same are likely to acquire resistance to SPFMV in the field. The technology shades some light as the CP gene is likely to be transmitted from one generation to the next (Okada and Saito, 2008). Despite the appropriateness of transgenic resistance in addressing sweet potato farmers' priorities is doubtful as low productivity is attributed not only by diseases but also several other factors (Clark et al., 2002; Hannington et al., 2002). Further, transgenic approach is useful for a single gene trait while most of economically important traits including disease resistance in sweet potato are quantitatively inherited (Mwanga et al., 2002a, b; Cervantes-Flores et al., 2011; Jain, 2010). Working with Kenyan sweet potato genotypes, Miano et al. (2008) identified molecular markers associated with SPVD resistance which could be used in breeding. Yang (2010) recommended that, *in vitro* shoot tip tissue culture could contribute significantly to the production of virus-free plantlets for farmers. The use of tissue culture in generating clean propagating materials should be an integral component of any management programme as it offers the possibility of managing not only virus diseases but also other pathogens and control genetic stability (Clark et al., 2002).

Despite the low transformation efficiency which has limited the successful application of genetic engineering in sweet potato (Liu, 2011), still the technology has attractive potential of contributing to sweet potato improvement not only disease resistance but also other agronomically important traits. Further, marker-assisted selection techniques are effective tools for improving disease resistance and quality in sweet potato (Liu, 2011). Therefore, the identification and development of improved cultivars is one of the strategies for increasing productivity and food security; however, this depends on the availability of diverse germplasm coupled with improved and efficient technologies.

**Mutation breeding:** For more than half a century, mutation breeding, specifically induced mutation has contributed significantly in the development of superior crop varieties (Jain, 2010). Since sweet potato is clonally propagated, mutation breeding is an effective approach for crop improvement and breeding for disease resistance (Wang et al., 2007; Liu, 2011; Shin et al., 2011). Maluszynski et al. (1995) and Wang et al. (2007) pointed out the application of *invitro* mutagenesis techniques in improvement of vegetatively propagated crops. By *in vitro* selection, desirable mutants with useful agronomic traits such as disease resistance can be

isolated within a relatively short period of time (Jain, 2010). Contrary to transgenic approach which is for single gene traits, mutants with multiple traits are possible. Further, mutation breeding in conjunction with genetic engineering is likely to enhance the improvement of sweet potato not only for disease resistance but also other important agronomic traits (Wang et al., 2007). Further, Jain (2010) commended mutation induction as being flexible, workable and a low-cost alternative to genetically modified organisms (GMOs).

### The genetics of SPVD

Most of economically important traits in sweet potato are quantitatively inherited (Lin et al., 2007; Cervantes-Flores et al., 2010). Knowledge on heritability of quantitative traits is necessary for an efficient genetic improvement in breeding programmes. Unlike resistance to other plant pathogens, resistance to plant viruses is inherited quantitatively (Diaz-Pendon et al., 2004). Studies on inheritance of SPVD resistance are limited due to its hexaploidy characterized by high genetic variability and complex segregation ratios of sweet potato progeny genotypes (Nishiyama et al., 1975; Mwanga et al., 2002b). Previous studies have indicated the potential of improving resistance to SPVD despite limited knowledge on its inheritance which hinders its efficient utilization in breeding programmes. Hahn et al. (1981) and Mwanga et al. (2002b), reported broad-sense heritability of resistance to SPVD ranging from 0.48 to 0.98 and narrow-sense heritability of 0.31-0.41. Therefore, with these levels of heritability there are potentials for sweet potato improvement for SPVD resistance through population improvement techniques.

The breeding of vegetatively reproducing crops differs from sexually reproducing crops. In sweet potato, once the seedlings are established from the true seeds following hybridization, the integrity of its genotype is maintained by vegetative propagation (Tai, 1974). Hence the genetic effects, either additive or dominance are inherited as whole. Genetic effect can either be additive or dominant or epistatic and in rare cases over dominance. According to Griffing (1956), general combining ability (GCA) and specific combining ability (SCA) are used to estimate gene effects. The GCA is used to estimate the additive genetic effect while SCA estimates the non-additive components.

Mihovilovich et al. (2000) pointed out that where virus resistance was controlled by more than one gene, additive effects were found. Similarly, using a diallel mating design, Mwanga et al. (2002b) found significant proportion of GCA effect compared to SCA implying the presence of additive gene action with regard to inheritance to SPVD resistance. Also, Mihovilovich et al. (2000) reported the predominance of additive genetic effect on the inheritance of resistance to SPFMV, a major

component of SPVD. In addition to additive effects, dominance genetic effect also contributed significantly in the inheritance of SPVD (Mwanga et al., 2002b). Despite the efforts made in developing resistant varieties, lack of knowledge and limited information on the nature of inheritance of the resistance hinders its application in sweet potato breeding (Mihovilovich et al., 2000; Mwanga et al., 2002b; Valverde et al., 2007) necessitating further investigations. Valverde et al. (2007) pointed the need for comprehensive resistance for protection against local strains in the breeding programmes.

### Effects of genotype by environment interaction on resistance to SPVD

Several important and common traits are a composite reflection of multiple genetic and environmental factors (Vuylsteke and van Eeuwijk, 2008). Sweet potato is grown in diverse environments across the world (Grüneberg et al., 2005; Caliskan et al., 2007; Haldavanekar et al., 2011). Despite its adaptability to diverse and harsh growing conditions, the crop is very sensitive to environmental variation (Bryan et al., 2003). This influences most of economically important traits which are largely quantitatively inherited and delays selection process in breeding programmes (Ngeve, 1993; Lebot, 2010). Nakitandwe et al. (2005) found that, sweet potato genotypes grown in multi-location trials performed differently with regard to yield and disease resistance. The G x E interactions could have largely contributed to break down of resistance in improved varieties grown in agro-ecologies with high SPVD pressure (Gibson et al., 1998; Karyeija et al., 1998). Osiru et al. (2009) suggested that, knowledge of genotype performance in different agro-ecologies is critical in cultivar development. Since there are differences in virus strains between agro-ecologies or regions, this could cause resistant genotypes in one region to be susceptible in others (Gibson et al., 1998; Carey et al., 1999). Therefore, newly developed cultivars need to be evaluated across target agro-ecologies to ascertain their reaction to SPVD (Caliskan et al., 2007; Mwololo et al., 2009). Further, selecting genotypes that interact less with the environments in which they are grown would be beneficial though not an easy endeavour.

### CONCLUSION AND RECOMMENDATIONS

Sweet potato is a vital staple food crop for most communities in developing world. Unfortunately, the crop is not researched in detail and underexploited compared to other crops despite its contribution. Hence its productivity is not encouraging. The low productivity is aggravated by biotic, abiotic and socio-economic factors.

Amongst the biotic factors, SPVD is the most important. The effects of SPVD in sweet potato production are real and devastating. Breeding for resistant cultivars is indispensable to control the disease for ascertained food security and incomes of rural and marginalized communities depending on this subsector. Sweet potato breeding is a difficult endeavour and challenging. Conventional breeding in combination with non-conventional techniques such as biotechnology, mutation breeding and genetic engineering have significant role in developing new sweet potato cultivars that are high yielding and resistant to SPVD. Despite the potential of genetic engineering in crop improvement, its application is not promising in developing countries (Jain, 2010). Presently, combination of conventional breeding, mutation breeding and tissue culture has the role in new cultivar development while waiting for institutionalization of transgenic crops and other GMOs. Lastly, phytosanitary practices have a role in maintaining the newly developed cultivars.

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## REFERENCES

- Abidin PE, Eeuwijk FA, Stam P, Struik PC, Malosetti M, Mwangi ROM, Odongo B, Hermann M, Carey EE (2005a). Adaptation and stability analysis of sweet potato varieties for low-input systems in Uganda. *Plant Breed.* 124:491-497.
- Abidin PE, Van Eeuwijk FA, Stam P; Struik PC, Malosetti M, Mwangi ROM, Odongo B, Hermann M, Carey EE (2005b). Adaptation and stability analysis of sweet potato varieties for low-input systems in Uganda. *Plant Breed.* 124:491-497.
- Ames De Icochea T, Ames T (1997). Sweetpotato: major pests, diseases, and nutritional disorders, International Potato Center.
- Ames T, Smit NEJM, Braun AR, O'sullivan JN, Skoglund LG (1997). Sweetpotato: Major pests, diseases and nutritional disorders, Lima, Peru, International Potato Center. P. 153.
- Anderson KM, Kang Q, Reber J, Harris MO (2011). No fitness cost for wheat's H gene-mediated resistance to Hessian fly (Diptera: Cecidomyiidae). *J. Econ. Entomol.* 104:1393-1405.
- Anwar N, Watanabe K, Watanabe J (2011). Transgenic sweet potato expressing mammalian cytochrome P450. *Plant Cell, Tiss. Organ Cult.* 105:219-231.
- ARITUA V, BUA B, GIBSON RW, MWANGA ROM, ADIPALA E (2003). Toward integrated management of sweet potato virus disease: Lessons from Uganda. *African Crop Science Conference Proceedings.*, 6:307-314.
- Aritua V, Bua B, Barg E, Vetten HJ, Adipala E, Gibson RW (2007). Incidence of five viruses infecting sweet potatoes in Uganda; the first evidence of Sweet potato caulimovirus-like virus in Africa. *Plant Pathology* 56:324-331.
- Aritua V, Alicai T, Adipala E, Carey EE, Gibson RW (1998). Aspects of resistance to sweet potato virus disease in sweet potato. *Ann. Appl. Biol.* 132:387-398.
- Ateka EM, Njeru RW, Kibaru AG, Kimenju JW, Barg E, Gibson RW, Vetten HJ (2004). Identification and distribution of viruses infecting sweet potato in Kenya. *Ann. Appl. Biol.* 144:371-379.
- Balouchi HR (2010). Screening wheat parents of mapping population for heat and drought tolerance, detection of wheat genetic variation. *Intern. J. Biol. Life Sci.* 6:56-66.
- Bryan AD, Schultheis JR, Pesic-Vanesbroeck Z, Yencho GC (2003). Cultivar decline in sweetpotato: II. Impact of virus infection on yield and storage root quality in 'Beauregard' and 'Hernandez'. *J. Am. Soc. Hort. Sci.* 128:856-863.
- Byamukama E, Adipala E, Gibson R, Aritua V (2002). Reaction of sweet potato clones to virus disease and their yield performance in Uganda. *Afr. Crop Sci. J.* 10:317-324.
- Byers DL, Meagher TR (1992). Mate availability in small populations of plant species with homomorphic sporophytic self-incompatibility. *Heredity* 68:353-359.
- Caliskan ME; Erturk E; Sogut T; Boydak E; Arioglu H (2007). genotype x environment interaction and stability analysis of sweet potato (*Ipomoea batatas*) genotypes. *New Zealand J. Crop Hort. Sci.* 35:87-99.
- Cao Q, Zhang A, Ma D, Li H, Li Q, Li P (2009). Novel interspecific hybridization between sweetpotato (*Ipomoea batatas* (L.) Lam.) and its two diploid wild relatives. *Euphytica* 169:345-352.
- Carey E, Gibson R, Fuentes S, Machmud M, Mwangi R, Turyamureeba G, Zhang L, Ma D, El-Abbas FA, El-Bedewy R (1999). The causes and control of virus diseases of sweetpotato in developing countries: is sweetpotato virus disease the main problem. *CIP Program Report 1997-1998:241-248.*
- Cattivelli L, Rizza F, Badeck FW, Mazzucotelli E, Mastrangelo AM, Francia E, Mare` C, Tondelli A, Stanca AM (2008). Drought tolerance improvement in crop plants: An integrated view from breeding to genomics. *Field Crops Res.* 105:1-14.
- Cervantes-Flores J, Sosinski B, Pecota K, Mwangi R, Catignani G, Truong V, Watkins R, Ulmer M, Yencho G (2011). Identification of quantitative trait loci for dry-matter, starch, and  $\beta$ -carotene content in sweetpotato. *Mol. Breed.* 28:201-216.
- Chang K, Lo H, Lai Y, Yao P, Lin K, Hwang S (2009). Identification of quantitative trait loci associated with yield-related traits in sweet potato (*Ipomoea batatas*). *Bot. Stud.* 50:43-55.
- Ching LL (2004). Broken promises: GM sweet potato project turns sour. *A magazine of Green Social Thoughts* St. Louis, MO 63130 WD Press, Synthesis/Regeneration
- Chiona M (2009). Towards enhancement of  $\beta$ -carotene content of high drymass sweetpotato genotypes in Zambia. PhD, University of KwaZulu Natal.
- Clark C, Valverde R, Fuentes S, Salazar L, Moyer J (2002). Research for improved management of sweetpotato pests and diseases: cultivar decline. *Acta Hort.* 583:103-112.
- Clark CA, Holmes GJ, Ferrin DM (2009). Major fungal and bacterial diseases. *In: Loebenstein, G Thottappilly, G (eds.) The sweetpotato.* Springer Netherlands. pp. 81-103.
- Collins NC, Tardieu F, Tuberosa R (2008). Quantitative trait loci and crop performance under abiotic stress: where do we stand. *Plant Physiol.* 147:469-486.
- Diaz-Pendon JA, Truniger V, Nieto C, Garcia-Mas J, Bendahmane A, Aranda M (2004). Advances in understanding recessive resistance to plant viruses. *Mol. Plant Pathol.* 5:223-233.
- Diaz J, Schmiediche P, Austin DF (1996). Polygon of crossability between eleven species of *Ipomoea*: section *Batatas* (Convolvulaceae). *Euphytica* 88:189-200.
- Dje Y, Diallo HA (2005). Detection of sweet potato feathery mottle virus in sweet potato using membrane immunobinding assay. *Afr. J. Biotech.* 4:717-723.
- Domela MJ, Thompson GJ, Aveling TaS, Laurie SM, Strydom H, Van Den Berg AA (2008). Sweet potato viruses in South Africa and the effect of viral infection on storage root yield. *Afr. Plant Protec.* 14:15-23.
- Ebregt E, Struik PC, Abidin PE, Odongo B (2004). Farmers' information on sweet potato production and millipede infestation in north-eastern Uganda. II. Pest incidence and indigenous control strategies. *Njas-Wageningen J. Life Sci.* 54:69-84.
- Ekanayake IJ, Collins W (2004). Effect of irrigation on sweet potato root

- carbohydrates and nitrogenous compounds. *Food Agric. Environ.* 2:243-248.
- Elameen A, Larsen A, Klemsdal S, Fjellheim S, Sundheim L, Msolla S, Masumba E, Roggli O (2011). Phenotypic diversity of plant morphological and root descriptor traits within a sweet potato germplasm collection from Tanzania. *Genet. Resour. Crop Evol.* 58:397-407.
- Elliott KA, Hoffman M (2010). Pulling Agricultural Innovation into the Market together: Working paper. Massachusetts, Washington, DC: Center for Global Development.
- Engoru P, Mugisha J, Bashaasha B (2005). Tuber utilization options among sweet potato producers in eastern Uganda. *Afr. Crop Sci. J.* 7:715-719.
- Ernest J, Wagih IE, Kesavan V (1994). Genetic evaluation of polycross hybrids of sweet potatoes. *Afr. Crop Sci. J.* 2:29-34.
- Feng G, Yifu G, Pinbo Z (2000). Production and deployment of virus-free sweetpotato in China. *Crop Protec.* 19:105-111.
- Fraile A, Pagan I, Anastasio G, Saez E, Garcia-Arenal F (2011). Rapid genetic diversification and high fitness penalties associated with pathogenicity evolution in a plant virus. *Mol. Biol. Evol.* 28:1425-1437.
- Fugile KO (2007). Priorities for sweetpotato research in developing countries: Results of a survey. *HortiScience* 42:1200-1206.
- Gaba V, Singer S (2009). Propagation of sweet potatoes: *In situ* germplasm conservation and conservation by tissue culture. In: Loebenstein, G. and Thottappilly, G (eds.) *The Sweetpotato*. Springer Science+Business Media B.V. pp. 65-80.
- García-Arenal F, McDonald BA (2003). An analysis of the durability of resistance to plant viruses. *Anal. Theoret. Plant Pathol.* 93:941-952.
- Gasura E, Mashingaidze AB, Mukasa SB (2010). Genetic variability for tuber yield, quality, and virus disease complex traits in Uganda sweetpotato germplasm. *Afr. Crop Sci. J.* 16:147-160.
- Gasura E, Mukasa SB (2010). Prevalence and implication of sweet potato recovery from sweet potato virus disease in Uganda. *Afr. Crop Sci. J.* 18:195-205.
- Ghazvini H, Tekauz A (2007). Reactions of Iranian barley accessions to three predominant pathogens in Manitoba. *Canadian J. Plant Pathol.* 29:69-78.
- Gibson R, Mpenbe I, Mwangi R (2011). Benefits of participatory plant breeding (PPB) as exemplified by the first-ever officially released PPB-bred sweet potato cultivar. *J. Agric. Sci.* 149:625-632.
- Gibson RW (2005). Working with farmers to control sweet potato virus disease in East Africa: Crop protection programme. UK: Natural resource institute.
- Gibson RW, Aritua V, Byamukama E, Mpenbe I, Kayongo J (2004). Control strategies for sweet potato virus disease in Africa. *Virus Res.* 100:115-122.
- Gibson RW, Jeremiah SC, Aritua V, Msabaha RP, Mpenbe I, Ndunguru J (2000). Sweet potato virus disease in Sub Saharan Africa: Evidence that neglect of seedlings in the traditional farming system hinders the development of superior resistant landraces. *J. Phytopathol.* 148:441-447.
- Gibson RW, Mpenbe I, Alicai T, Carey EE, Mwangi ROM, Seal SE, Vetten HJ (1998). Symptoms, aetiology and serological analysis of sweet potato virus disease in Uganda. *Plant Pathol.* 47:95-102.
- Gibson RW, Mwangi ROM, Kasule S, Mpenbe I, Carey EE (1997). Apparent absence of viruses in most symptomless field-grown sweet potato in Uganda. *Ann. Appl. Biol.* 130:481-490.
- Gichuki ST, Berenyi M, Zhang D, Hermann M, Schmidt J, Glössl J, Burg K (2003). Genetic diversity in sweetpotato [*Ipomoea batatas* (L.) Lam.] in relationship to geographic sources as assessed with RAPD markers. *Genet. Resour. Crop Evol.* 50:429-437.
- Griffing B (1956). Concept of general and specific combining ability in relation to diallel crossing systems. *Australian J. Biol. Sci.* 9:463-493.
- Grüneberg WJM, Zhang K, Dapeng Hermann M (2005). Genotypic environment interactions for a diverse set of sweetpotato clones evaluated across varying ecogeographic conditions in Peru. *Crop Sci.* 45:2160.
- Gutiérrez DL, Fuentes S, Salazar LF (2003). Sweetpotato Virus Disease (SPVD): Distribution, incidence and effect on sweetpotato yield in Peru. *Plant Dis.* 87:297-302.
- Hahn S, Terry E, Leuschner K (1981). Resistance of sweet potato to virus complex. *HortScience* 16:535-537.
- Haldavaneekar PC, Bhavé SG, Kahandekar RG, Kadam SG, Sawant SS (2011). Stability analysis in sweet potato (*Ipomoea batatas* L.). *Karnataka J. Agric. Sci.* 24:358-361.
- Hannington O, Kameri-Mbote P, Wafula D (2002). Innovation and policy process: Case of transgenic sweet potato in Kenya. *Econ. Political Weekly* 37:2770-2777.
- Harrison HF, Jr., Jackson DM (2011). Response of two sweet potato cultivars to weed interference. *Crop Protec.* 30:1291-1296.
- Hu W, Jiang A, Jin L, Liu C; Tian M, Wang Y (2011). Effect of heat treatment on quality, thermal and pasting properties of sweet potato starch during yearlong storage. *J. Sci. Food. Agric.* 91:1499-1504.
- Huaman Z (1992). Systematic botany and morphology of the sweetpotato plant. CIP. P. 25.
- Huang JC, Sun M (2000). Genetic diversity and relationships of sweetpotato and its wild relatives in *Ipomoea* series *Batatas* (Convolvulaceae) as revealed by inter-simple sequence repeat (ISSR) and restriction analysis of chloroplast DNA. *Theor. Appl. Genet.* 100:1050-1060.
- Hull R (1994). Resistance to plant viruses: Obtaining genes by non-conventional approaches. *Euphytica* 75:195-205.
- Ishak JA, Kreuze JF, Johansson A, Mukasa SB, Tairo F, Abo El-Abbas FM, Valkonen JPT (2003). Some molecular characteristics of three viruses from SPVD-affected sweet potato plants in Egypt. *Arch. Virol.* 148:2449-2460.
- Jain SM (2010). Mutagenesis in crop improvement under the climate change. *Romanian Biotech. Lett.* 15:89-106.
- Jauhar PP (2006). Modern biotechnology as an integral supplement to conventional plant breeding: The prospects and challenges. *Crop Sci.* 46:1841-1859.
- Johanson A, Ives CL (2001). An inventory of agricultural biotechnology for the Eastern and Central Africa region. Michigan, USA: Institute of International Agriculture.
- Jones A (1980). Sweet potato. *Am. Soc. Agron.* 46:645-655.
- Jones A, Dukes PD, Schalk JM (eds.) (1986). Sweet potato breeding, Westport, CT, USA: AVI Publishing Co. P. 35.
- Kapinga R, Ndunguru J, Mulokozi G, Tumwegamire S (2009a). Impact of common sweetpotato viruses on total carotenoids and root yields of an orange-fleshed sweetpotato in Tanzania. *Sci.Hortic.* 122:1-5.
- Kapinga R, Zhang D, Lemaga B, Andrade M, Mwangi ROM, Laurie S, Ndoho P, Kanju E (2009b). Sweet potato improvement in sub-Saharan Africa. In: Kapinga, RR, Kingamkono M, Msabaha J, Ndunguru B, Lemaga G, Tusiime (eds.). *International Society of Trop. Root Crops Conference Proc.* 13:82-94.
- Kapinga RE, Carey EE (2003). Present status of sweet potato breeding for eastern and southern Africa. In: Rees, DOQ and R. Kapinga (eds.) *Sweet potato post-harvest assessment: Experience from East Africa*. London, UK: Natural Resource Institute, University of Greenwich. pp. 1-8.
- Kapinga RE, Ewell PT, Jeremiah SC; Kileo R (1995). Sweetpotato in Tanzanian farming and food systems: Implications for research, International potato Center (CIP) Sub-Saharan Africa Regional Office, Nairobi and Ministry of Agriculture, Dar es Salaam, Tanzania. P. 45.
- Karuri HW, Ateka EM, Amata R, Nyende AB, Muigai AWT (2009). Characterization of Kenyan sweet potato genotypes for SPVD resistance and high dry matter content using simple sequence repeat markers. *Afr. J. Biotech.* 8:2169-2175.
- Karyeija R, Gibson R, Valkonen J (1998). The significance of sweet potato feathery mottle virus in subsistence sweet potato production in Africa. *Plant. Dis.* 82:4-15.
- Karyeija RF, Kreuze JF, Gibson RW, Valkonen JPT (2000). Synergistic interactions of a potyvirus and a phloem-limited crinivirus in sweet potato plants. *Virology* 269:26-36.
- Kobayashi RS, Sinden SL, Bouwkamp JC (1993). Ovule culture of sweet potato (*Ipomoea batatas*) and closely related species. *Plant Cell Tiss. Organ Cult.* 32:77-82.
- Kokkinos CD, Clark CA, Mcgregor CE, Labonte DR (2006). The effect of sweet potato virus disease and its viral components on gene expression levels in sweetpotato. *J. Am. Soc. Hort. Sci.* 131:657-666.
- Korada RR, Naskar SK, Prasad AR, Prasanna AL, Jyothi KN (2010). Differential volatile emission from sweet potato plant: mechanism of resistance in sweet potato for weevil *Cylas forficarius* (Fab). *Curr. Sci.* 99:1597-1601.

- Koyama Y, Shimano N, Kawase T (1980). Genetic analysis of incompatibility in the diploid *Ipomoea* species closely related to the sweet potato. *Theor. Appl. Genet.* 58:149-155.
- Koyama Y, Tsuchiya T, Kakeda K (2000). Sporophytic self-incompatibility in *Ipomoea trifida*, a close relative of sweet potato. *Ann. Bot.* 85:191.
- Kreuze JF (2002). Molecular studies on the sweet potato virus disease and its two causal agents. PhD Thesis, Swedish University of Agricultural Sciences.
- Kreuze JF, Valkonen JPT, Ghislain M (2009). Genetic engineering. In: Loebenstein, G and G Thottappilly (eds.) *The sweet potato*. Netherlands: Springer pp. 41-63.
- Kriegner A, Cervantes JC, Burg K, Mwanga ROM, Zhang D (2003). A genetic linkage map of sweetpotato based on AFLP markers. *Mol. Breed.* 11:169-185.
- Kulembeka H, Rugutu C, Kanju E, Chirimi B, Rwiza E, Amour R (2005). The agronomic performance and acceptability of orange fleshed sweetpotato varieties in the lake zone of Tanzania. *Afr. Crop Sci. J.* 12:229-240.
- Kuo CG (1991). Conservation and distribution of sweet potato germplasm. In: DODDS, JH (ed.) *In-vitro methods for conservation of plant genetic resources*. London, UK: Chapman and Hall. p. 123-144.
- Lebot V (2010). Sweet potato. In: Bradshaw, JE (ed.) *Root and tuber crops: Handbook of plant breeding*. London: Springer. p. 97-125.
- Lin K, Lai YC, Chang K, Chen Y, Hwang SY, Lo HF (2007). Improving breeding efficiency for quality and yield of sweet potato. *Bot. Studies* 48:283-292.
- Liu Q (2011). Sweet potato omics and biotechnology in China. *Plant Omics* 4:295-301.
- Liwenga ET, Kangalawe RYM (2009). Climate change/variability and implications on agricultural production and livelihoods in the southern highlands of Tanzania. In: Paul, SM and AE. Majule (eds.) *Strengthening local agricultural innovations to adapt to climate change in Botswana, Malawi, South Africa and Tanzania*. Dar-es-Salaam, Tanzania: Institute of Resource Assessment, University of Dar es Salaam. pp. 124-135.
- Lou HR, Maria MS, Benavides J, Zhang DP, Zhang Y, Ghislain M (2010). Rapid genetic transformation of sweet potato (*Ipomoea batatas* (L.) Lam) via organogenesis. *Afr. J. Biotech.* 5:1851-1857.
- Low J, Lynam J, Lemaga B, Crissman C, Barker I, Thiele G, Namanda S, Wheatley C, Andrade M (2009). Sweetpotato in Sub-Saharan Africa. In: Loebenstein, G. and G Thottappilly (eds.) *The sweet potato*. Springer Netherlands. pp. 359-390.
- Magoon ML, Krishnan R, Vijaya Bai K (1970). Cytological evidence on the origin of sweet potato. *Theor. Appl. Genet.* 40:360-366.
- Maluszynski M, Ahloowalia B, Sigurbjörnsson B (1995). Application of *in vivo* and *in vitro* mutation techniques for crop improvement. *Euphytica* 85:303-315.
- Martin FW (1968). The system of self-incompatibility in *Ipomoea*. *J. Hered.* 59:263.
- Martin FW (1970). Self-and interspecific incompatibility in the Convolvulaceae. *Bot. Gaz.* 131:139-144.
- Martin FW, Cabanillas E (1966). Post-pollen-germination barriers to seed set in sweet-potato. *Euphytica* 15:404-411.
- Martin FW, Ortiz S (1967). Anatomy of stigma and style of sweet potato. *New Phytologist* 66:109-113.
- Masumba E, Kulembeka H, Tollano S, Yongolo M (2005). Participatory evaluation of improved sweetpotato varieties in Eastern Tanzania. *Afr. Crop Sci. J.* 12:259-265.
- Maule AJ, Caranta C, Boulton MI (2007). Sources of natural resistance to plant viruses: Status and prospects. *Mol. Plant Pathol.* 8:223-231.
- Mbwaga Z, Mataa M, Msabaha M (2007). Quality and yield stability of orange fleshed sweet potato (*Ipomoea batatas*) varieties grown in different agro-ecologies. *Afri. Crop Sci. Conf. Proc.* 8:339-345.
- Mcharo M, Carey EE (2001). Performance of selected sweet potato varieties in Kenya. *Afr. Crop Sci. J.* 9:49-57.
- Miano D, Labonte D, Clark C (2008). Identification of molecular markers associated with sweet potato resistance to sweet potato virus disease in Kenya. *Euphytica* 160:15-24.
- Mihale M, Deng A, Selemeni H, Kamatenesi M, Kidukuli A, O'endo J (2009). Use of indigenous knowledge in the management of field and storage pests around Lake Victoria basin in Tanzania. *Afr. J. Environ. Sci. Tech.* 3:251-259.
- Mihovilovich E, Mendoza HA, Salazar LF (2000). Combining ability for resistance to sweetpotato feathery mottle virus. *HortScience* 35:1319-1320.
- Mont J, Iwanaga M, Orjeda G, Watanabe K (1993). Abortion and determination of stages for embryo rescue in crosses between sweet-potato, *Ipomoea batatas* Lam. (2n=6x=90) and its wild relative, *I. trifida* (H. B. K.) G. Don. (2n=2x=30). *Sexual Plant Rep.* 6:176-182.
- Mpagalile J, Silayo V, Laswai H, Ballegu W, Kikuu C (2003). Effect of different storage methods on the shelf-life of fresh sweetpotatoes in Gairo, Tanzania. *Trop. Root. Tuber Crops* 10:500-505.
- Mtunda KJ, Kanju E, Chilosa D Kilima M (2003). Increasing sweet potato production in Tanzania through multiplication and distribution of improved farmer selected varieties. Technical Annual Report. Kibaha: Sugarcane Research Institute.
- Mudioppe J, Kindness H Haigenemana V (2000). Socio-economic constraints to the production, processing and marketing of Sweetpotato in Kumi district, Uganda. UK: DFID.
- Mukasa SB, Rubaihayo PR, Valkonen JPT (2006). Interactions between a crinivirus, an ipomovirus and a potyvirus in co-infected sweetpotato plants. *Plant Pathol.* 55:458-467.
- Mullen MA (1984). Influence of sweet potato weevil infestation on the yields of twelve sweet potato lines. *J. Agric. Entomol.* 1:227-230.
- Munyiza H, Stevenson P, Mwanga R, Talwana H, Murumu J Odongo B (2007). The relationship between stem base and root damage by *Cylas* spp. on sweet potato. *Afr. Crop Sci. J.* 8:955-957.
- Muturi PW, Mwololo JK, Mburu MWK, Njeru RW, Kiarie N, Munyua JK, Ateka EM, Muinga RW, Kapinga RE, Lemaga B (2007). Strategies of maintaining sweet potato nurseries free from insect vectors that spread sweet potato virus disease. *Afri. Crop Sci. Confer. Proc.* 8:2071-2074.
- Muyinza H, Stevenson P, Mwanga R, Talwana H, Murumu J, Odongo B (2007). The relationship between stem base and root damage by *Cylas* spp. on sweet potato. *Afr. Crop Sci. J.* 8:955-957.
- Mwanga R, Carey EE, Moyer JW, Zhang DP, Yencho GC (2002a). Nature of resistance of sweet potato to sweet potato virus disease. *Acta Hort.* 583:113-119.
- Mwanga R, Ssemakula G (2011). Orange-fleshed sweet potatoes for food, health and wealth in Uganda. *Intern. J. Agric. Sust.* 9:42-49.
- Mwanga R, Yencho G, Moyer JW (2002b). Diallel analysis of sweetpotatoes for resistance to sweetpotato virus disease. *Euphytica* 128:237-248.
- Mwanga RO, Niringiye C, Alajo A, Kigozi B, Namukula J, Mpenbe I, Tumwegamire S, Gibson RW, Yencho GC (2011). 'NASPOT 11', a Sweetpotato Cultivar Bred by a Participatory Plant-breeding Approach in Uganda. *HortScience* 46:317-321.
- Mwanga RO, Odongo B, Niringiye C, Alajo A, Kigozi B, Makumbi R, Lugwana E, Namukula J, Mpenbe I, Kapinga R (2009). 'NASPOT 7', 'NASPOT 8', 'NASPOT 9 O', 'NASPOT 10 O', and 'Dimbuka-Bukulula' Sweetpotato. *HortScience* 44:828-832.
- Mwanga ROM, Odongo B, Niringiye C, Kapinga R, Tumwegamire S (2007). Sweet potato selection releases: Lessons learnt from Uganda. *Afr. Crop Sci. J.* 15:11-23.
- Mwololo JK, Mburu MWK, Njeru RW, Ateka EM, Kiarie N, Munyua JK, Muinga RW, Kapinga R, Lemaga B (2007). Resistance of sweet potato genotypes to sweet potato virus disease in Coastal Kenya. *Afr. Crop Sci. Confer. Proc.* 8:2083-2086.
- Mwololo JK, Muturi PW, Mburu MWK, Njeru RW, Kiarie N, Munyua JK, Ateka EM, Muinga RW, Kapinga RE, Lemaga B (2009). Additive main effects and multiplicative interaction analysis of genotype x environmental interaction among sweet potato genotypes. *J. Anim. Plant Sci.* 2:148-155.
- Nakitandwe J, Adipala E, El-Bedewy R, Wagoire W, Lemaga B (2005). Adaptability of SIFT potato genotypes in different agro-ecologies of Uganda. *Afr. Crop Sci. J.* 13:107-116.
- Ndunguru J, Kapinga R (2007). Viruses and virus-like diseases affecting sweet potato subsistence farming in southern Tanzania. *Afr. J. AgricRes.* 2:232-239.
- Ndunguru J, Kapinga R, Sseruwagi P, Sayi B, Mwanga R, Tumwegamire S, Rugutu C (2009). Assessing the sweetpotato virus disease and its associated vectors in northwestern Tanzania and central Uganda. *Afr. J. Agric. Res.* 4:334-343.

- Ngeve JM (1993). Regression analysis of genotype  $\times$  environment interaction in sweet potato. *Euphytica* 71:231-238.
- Nimmakayala P, Vajja G, Reddy UK (2011). *Ipomoea*. Wild crop relatives: Genomic and Breeding Resources. Berlin: Springer. pp. 123-132.
- Nishiguchi M, Mori M, Okada Y, Murata T, Kimura T, Sakai J, Hanada K, Miyazaki C, Saito A (1998). Virus resistant transgenic sweet potato with the CP gene: Current Challenge and perspective of its use. *Phytoprotection* 79:112-116.
- Nishiyama I, Miyazaki T, Sakamoto S (1975). Evolutionary autopolyploidy in the sweet potato (*Ipomoea batatas* (L.) Lam.) and its progenitors. *Euphytica* 24:197-208.
- Njeru RW, Mburu MWK, Cheramgoi E, Gibson RW, Kiburi ZM, Obudho E, Yobera D (2004). Studies on the physiological effects of viruses on sweet potato yield in Kenya. *Ann. Appl. Biol.* 145:71-76.
- Nyaboga EN, Ateka EM, Gichuki ST, Bulimo WD (2008). Reaction of transgenic sweet potato (*Ipomoea batatas* L.) lines to virus challenge in the glasshouse. *J. Appl. Biosci.* 9:362-371.
- Okada Y, Nishiguchi M, Saito A, Kimura T, Mori M, Hanada K, Sakai J, Matsuda Y, Murata T (2002). Inheritance and stability of the virus-resistant gene in the progeny of transgenic sweet potato. *Plant Breed.* 121:249-253.
- Okada Y, Saito A (2008). Evaluation of resistance to complex infections of SPFMVs in transgenic sweet potato. *Breeding Sci.* 58:243-250.
- Okada Y, Saito A, Nishiguchi M, Kimura T, Mori M, Hanada K, Sakai J, Miyazaki C, Matsuda Y, Murata T (2001). Virus resistance in transgenic sweetpotato [*Ipomoea batatas* L. (Lam)] expressing the coat protein gene of sweet potato feathery mottle virus. *Theor. Appl. Genet.* 103:743-751.
- Opiyo SA, Ateka EM, Owuor PO, Manguro LOA, Miano DW (2010). Development of a multiplex PCR techniques for simultaneous detection of Sweet potato feathery mottle virus and Sweet potato chlorotic stunt virus. *J. Plant Pathol.* 92:363-366.
- Orjeda G, Freyre R, Iwanaga M (1990). Production of 2n pollen in diploid *Ipomoea trifida*, a putative wild ancestor of sweet potato. *J. Hered.* 81:462.
- Orjeda G, Freyre R, Iwanaga M (1991). Use of *Ipomoea trifida* germplasm for sweet potato improvement. 3. Development of 4x interspecific hybrids between *Ipomoea batatas* (L.) Lam. (2n=6x=90) and *I. trifida* (H.B.K) G. Don. (2n=2x=30) as storage-root initiators for wild species. *Theor. Appl. Genet.* 83:159-163.
- Osiru MO, Olanya OM, Adipala E, Kapinga R, Lemaga B (2009). Yield stability analysis of *Ipomoea batatas* L. cultivars in diverse environments. *Australian Journal of Crop Science* 3(4):213-220.
- Otani M, Wakita V, Shimada T (2003). Production of herbicide-resistant sweet potato (*Ipomoea batatas* (L.) Lam) plants by Agrobacterium tumefaciens-mediated transformation. *Breeding Sci.* 53:145-148.
- Pareek A, Sopory SK, Bohnert HJ, Govindjee (eds.) (2010). *Abiotic stress adaptation in plants: Physiological, Molecular and Genomic Foundation.*, Dordrecht, The Netherlands: Springer. pp. 91-102.
- Prakash CS, Varadarajan U (1992). Genetic transformation of sweet potato by particle bombardment. *Plant Cell Reports* 11:53-57.
- Rajapakse S, Ryan-Bohac J, Nirmalgoda S, Ballard R, Austin DF (2005). The Use of DNA Sequences from the  $\beta$ -Amylase Gene to Determine the Genetic Relationship among Sweetpotato, *I. batatas* and Other *Ipomoea* Species in Series *Batatas* (Convolvulaceae). *HortScience* 40:1121-1122.
- Reddy UK, Bates GT, Ryan-Bohac J, Nimmakayala P (2007). Sweetpotato. In: KOLE, C (ed.) *Genome mapping and molecular breeding in plants*. New York: Springer. pp. 237-239.
- Rees D, Kapinga R, Rwiza E, Mohammed R, Van Oirschot Q, Carey E, Westby A (1998). The potential for extending the shelf-life of sweet potato in East Africa through cultivar selection. *Trop. J. Agric.* 75:208-211.
- Rukarwa RJ, Mashingaidze AB, Kyamanywa S, Mukasa SB (2010). Detection and elimination of sweet potato viruses. *Afr. Crop Sci. J.* 18:223-233.
- Saleh H, Zahor O (2007). Farmers' perception and varieties acceptability of orange-fleshed sweetpotato in Zanzibar. In: Kapinga RR, Kingamkono M, Msabaha J, Ndunguru B, Lemaga G Tusiime (eds). *Int. Soc. Trop. Root Crops Conf. Proc.* 13:801-806.
- Schafleitner R, Tincopa LR, Palomino O, Rossel G, Robles RF, Alagon R, Rivera C, Quispe C, Rojas L, Pacheco JA (2010). A sweet potato gene index established by de novo assembly of pyrosequencing and Sanger sequences and mining for gene-based microsatellite markers. *BMC Genomics* DOI:10.1186/1471-2164-11-604.
- Shin JM, Kim BK, Seo SG, Jeon SB, Kim JS, Jun B, Kang SY, Lee JS, Chung MN, Kim SH (2011). Mutation breeding of sweet potato by gamma-ray radiation. *Afr. J. Agric. Res.* 6:1447-1454.
- Sihachakr D, Haïcour R, Cavalcante Alves JM; Umboh I; Nzoghé D, Servaes A, Ducreux G (1997). Plant regeneration in sweet potato (*Ipomoea batatas* L., Convolvulaceae). *Euphytica* 96:143-152.
- Srisuwan S, Sihachakr D, Siljak-Yakovlev S (2006). The origin and evolution of sweet potato (*Ipomoea batatas* Lam.) and its wild relatives through the cytogenetic approaches. *Plant Sci.* 171:424-433.
- Stathers TE, Rees D, Jeffries D, Kabi S, Smith N, Mbilinyi L, Kiozya H, Jeremiah S, Nyango M, Moss C (1999). Investigating the potential of cultivar differences in susceptibility to sweet potato weevil as a means of control. *Crop Post-harvest Programme*. UK. DFID.
- Stathers TE, Rees D, Kabi S, Mbilinyi L, Smith N, Kiozya H, Jeremiah S, Nyango A, Jeffries D (2003). Sweet potato infestation by *Cylas* spp. in East Africa: I. Cultivar differences in field infestation and the role of plant factors. *Int. J. Pest Manage.* 49:131-140.
- Tai GCC (1974). A method for quantitative genetic analysis of early clonal generation seedlings for asexual crops with special application to a breeding population of the potato (*Solanum tuberosum* L.). *Theor. Appl. Genet.* 45:150-156.
- Tairo F, Kullaya A, Valkonen JPT (2004). Incidence of viruses infecting sweetpotato in Tanzania. *Plant Dis.* 88:916-920.
- Tairo F, Mukasa SB, Jones RaC, Kullaya A, Rubaihayo PR, Valkonen JPT (2005). Unravelling the genetic diversity of the three main viruses involved in Sweet potato virus disease (SPVD), and its practical implications. *Mol. Plant Pathol.* 6:199-211.
- Thottappilly G, Loebenstein G (2009). Concluding Remarks. In: Loebenstein, G. and G. Thottappilly (eds.) *The Sweetpotato*. Springer Netherlands. pp. 503-513.
- Thresh JM (2003). Control of plant virus diseases in sub-Saharan Africa: The possibility and feasibility of an integrated approach. *Afr. Crop Sci. J.* 11:199-223.
- Ting YUC, Kehr AE (1953). Meiotic studies in the sweet potato. *J. Hered.* 44:207.
- Tomita RN, Suzuki G, Yoshida K, Yano Y, Tsuchiya T, Kakeda K, Mukai Y, Koyama Y (2004). Molecular characterization of a 313-kb genomic region containing the self-incompatibility locus of *Ipomoea trifida*, a diploid relative of sweet potato. *Breed. Sci.* 54:165-175.
- Troung VD, Avula RY, Pecota K, Yencho CG (2011). Sweet potatoes. In: SINHA, NK (ed.) *Handbook of vegetables and vegetable processing*. New Jersey: Wiley-Blackwell. pp. 717-737.
- Tsakama M, Mwangwela AM, Manani TA, Mahungu NM (2010). Physicochemical and pasting properties of starch extracted from eleven sweet potato varieties. *Afr. J. Food Sci. Tech.* 1:90-98.
- Tseng YT, Lo HF, Hwang SY (2002). Genotyping and assessment of genetic relationships in elite polycross breeding cultivars of sweet potato in Taiwan based on SAMPL polymorphisms. *Bot. Bull. Acad. Sinica* 43:99-105.
- Ulukan H (2009). The evolution of cultivated plant species: Classical plant breeding versus genetic engineering. *Plant Syst. Evol.* 280:133-142.
- Untiveros M, Fuentes S, Kreuze J (2008). Molecular variability of sweet potato feathery mottle virus and other potyvirus infecting sweet potato in Peru. *Arch. Virol.* 153:473-483.
- Vaeasey EA, Borges A, Rosa MS, Queiroz-Silva JR, Bressan EA, Peroni N (2008). Genetic diversity in Brazilian sweet potato (*Ipomoea batatas* (L.) Lam., Solanales, Convolvulaceae) landraces assessed within microsatellite markers. *Genet. Mol. Biol.* 31:725-733.
- Valverde RA, Clark CA, Valkonen JPT (2007). Viruses and virus disease complexes of sweetpotato. *Plant Vir.* 1:116-126.
- Van Den Bosch F, Jeger M, Gilligan C (2007). Disease control and its selection for damaging plant virus strains in vegetatively propagated staple food crops; a theoretical assessment. *Proc. Royal Soc. B: Biol. Sci.* 274:11-18.
- Vuylsteke M, Van Eeuwijk F (2008). The use of general and specific combining abilities in a context of gene expression relevant to plant

- breeding. *Euphytica* 161:115-122.
- Waddington S, Li X, Dixon J, Hyman G, De Vicente M (2010). Getting the focus right: production constraints for six major food crops in Asian and African farming systems. *Food Sec.* 2:27-48.
- Wambugu FM (2003). Development and transfer of genetically modified virus-resistant sweet potato for subsistence farmers in Kenya. *Nutr. Rev.* 61:S110-S113.
- Wang Y, Wang F, Zhai H, Liu Q (2007). Production of a useful mutant by chronic irradiation in sweet potato. *Science* 35:24-34.
- Woolfe JA (1992). *Sweet potato: An untapped food resource*, Cambridge, England, Cambridge University Press. P. 643.
- Yang X (2010). Rapid production of virus-free plantlets by shoot tip culture in vitro of purple sweet potato (*Ipomoea batatas* (L.) Lam). *Pakistan J. Bot.* 42:2069-2075.
- Yi G, Shin Y-M, Choe G, Shin B, Kim Y, Kim KM (2007). Production of herbicide-resistant sweet potato plants transformed with the bar gene. *Biotech. Lett.* 29:669-675.
- Zhang D, Cervantes J, Huamán Z, Carey E, Ghislain M (2000). Assessing genetic diversity of sweet potato (*Ipomoea batatas* (L.) Lam.) cultivars from tropical America using AFLP. *Genet. Resour. Crop Evol.* 47:659-665.