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A REVIEW OF THE BREEDING OF CLONALLY PROPAGATED SWEETPOTATO GENOTYPES FOR ACQUIRED RESISTANCE TO SWEETPOTATO PATHOGEN COMPLEX

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ABSTRACT: *The vegetative propagation of sweetpotato crop by farmers involves taking of vine cuttings, from a previous year's crop which increases the risk of a buildup of viruses and other pathogens. The importance of virus diseases and their buildup in farmers' planting materials has been shown convincingly to drastically reduce yield of sweetpotato varieties. This has contributed to slow sweetpotato breeding process as a result of genotype decline, flower abortion and malformation of leaves. Chemical control measures to prevent virus insect vectors are no longer useful as chemical sprays destroy insects' pollinators. However, resistant sweetpotato varieties have made considerable contribution to plant breeding in terms of yield and provision of planting material. As a result of the merits of disease resistant sweetpotato varieties, this review discussed the "Breeding of clonally propagated sweetpotato genotypes for acquired resistance to sweetpotato pathogen complex". The review indicated that sweetpotato plants that failed to inherit resistance genes could be made to acquire defense mechanisms in field evaluation in hotspot fields and agro-ecologies through adaptation breeding to incorporate resistance to pathogens in the clonally bred sweetpotato genotypes. This enable the sweetpotato genotypes acquires natural resistance to major diseases attacking the sweetpotato crop in the field. Resistance sweetpotato genotypes eliminates losses from diseases and are environmentally friendly.*

KEYWORDS: Sweetpotato, Pathogens, Resistance, Vegetative Propagation, Acquired Defense, Ipomoea Batatas (L.) Lam

INTRODUCTION

Sweetpotato [*Ipomoea batatas* (L.) Lam] is a dicotyledonous plant, belonging to the *Convolvulaceae* family of about 45 *genera* and 1000 *species*, with only *Ipomoea. batatas* of economic importance as food for man and feed and fodder for livestock (Woolfe, 1992)**.** There are over 1402 varieties of sweetpotato which skin and flesh color vary from white, cream, and yellow, orange, pink, to deep purple depending on the variety. However, the white/cream and yellow-orange fleshed colors are most common **(**Ahmad *et al.*, 2006) and almost all are affected by the same pathogens in the field. Wolfgang and his co-workers (2012) reported that the annual sweetpotato production in Africa increased moderately from 11.6 million tonnes in 2002 to 12.9 million tonnes in 2006. Of which less than 20% of production is traded in rural and urban markets. Although, data on piecemeal harvested sweetpotato crop are difficult to collect. Sweetpotato performs well in relatively poor soils, with few inputs, and has a short growing period of between 3 to 4 months to harvest if tuberous roots are the cultivation objective. However, the clonal propagation of Sweetpotato and the use of re-growth encourage transmission of diseases from generation to generation.

Sweetpotato large tuberous roots can be consumed in various forms such as: boiled, steamed, baked, and fried. Traditionally, sweetpotato tuberous roots can be pounded or mixed with yam and eaten with vegetable soup, roasted and eaten with red palm oil or sauce, or made into porridge. However, disease infection affects the yield quantity and quality of the food products (Gibson et al., 2000). Within the sweetpotato gene pool, there is an enormous amount of genetic variation for quality attributes. The combining of parents with medium to high genetic values is all that is needed to produce high quality clones for various quality uses (Islam *et al*., 2002) such as excellent disease resistance and high yield performance and other traits. Although, Breeders always want to select for several traits concurrently, in practical terms, quality breeding often means to improve quality and maintain genetic variation for yield, yield stability, adaptability and resistant to major diseases such as viruses. Farmers grow sweetpotato landraces by selecting planting materials from seemingly symptomless parents. The sweetpotato crop is also grown in small plots that are interspersed among other crops (intercropping) which may be a host plant for vectors. In most cases, sweetpotato is grown continuously under rain fed or under irrigation and as such, mature and at times, newly planted sweetpotato crop fields overlap, leading to perpetuation of sweetpotato virus disease (SPVD). Sweetpotato plants affected by SPVD are low yielding (Karyeija *et al.,* 1998). However, the yield of the sweetpotato plants are curtailed when over 50% of the plants are affected (Gibson *et al.,* 2000). According to Gibson *et al* (2000), such high incidences are rarely observed because, in areas of high population of whitefly vectors, and where SPCSV are common, farmers grow SPVD-resistant sweetpotato landraces. These tend to be less productive since they produce storage roots of a lower yield and quality than some SPVD - susceptible exotic sweetpotato varieties (Gibson et al., 2000). The major challenge of SPVD is not to reduce the incidences of SPVD but to enable farmers to grow more productive varieties with little or no increase in the incidences of SPVD and other pathogens. This means that acquired immune system of the crop can protect desirable varieties of both landraces and hybrid genotypes released as varieties. Resistant sweetpotato varieties have made considerable contribution to plant breeding in terms of yield. As a result of the merits of disease resistant varieties, this review discussed "the breeding of clonally propagated sweetpotato genotypes to acquired resistance to sweetpotato pathogen complex".

Virus Buildup in Sweetpotato Plants

The virus disease complex of sweetpotato rapidly multiply itself in living sweetpotato plant cells thereby causing damage to sweetpotato plant and considerably reduce its yield. Kreuze and Fuentes, (2008) reported that more than 20 different viruses have been described infecting sweetpotato worldwide, but only 15 of these are currently recognized by the International Committee on Taxonomy of Viruses. A virus infection is spread by especially aphids, which are piercing sucking insects which move from plant to plant and by plant hopers and whiteflies causing leaf curl, leaf chlorosis, leaf mosaic, vein clearing, leaf deformation and stunting of plants. Viruses are the most important pathogens in terms of their ability to cause sudden and widespread epidemics (Ames ,2002). These insects can come from the direct vicinity or from far away fields with other types of crops such as *manihot species* and *Telferia occidentalis*. Viruses can also be spread by human hands that have come in contact with an infected crop or crop products. Vegetative propagated plant material can spread viruses when they are used as planting materials in the field. However, the only proven vectors of sweetpotato viruses are aphids and whiteflies (Stathers *et al*., 2005). Sweetpotato varieties infected by viruses cannot be treated with chemicals, however, chemicals such as pesticides could be used to control the insect vectors. The danger is that beneficial insects that engage in inter-mating of parents during open pollination may be destroyed and when this happens, it is a bad news to plant breeders that depends on open pollination to generate genetic variability of the sweetpotato crop. Clonal propagation which is the taking of vine cuttings from a previous crop increases the risk of a buildup of viruses.

Sweetpotato is a climbing, sprawling and crippling plant. As they grow, they are infected with viruses. The virus in the sweetpotato plant is systemic Nwankwo and Opara, 2015). Kreuze and Fuentes (2008) had reported that virus diseases buildup in farmers' planting materials has been shown convincingly in various experiments that it leads to yield reduction (Jeude (2004). It was observed that sweetpotato cultivars planted using pathogen-tested materials yielded 30–40% more, on average, then those grown from farm-derived planting materials. Virus diseases form the most important biotic production constraint in sweetpotato and are regarded second to sweetpotato root weevil in destructiveness. Most sweetpotato-infecting viruses, however, show only mild or no symptoms when in single infection and the damages caused by sweetpotato viruses are mostly through synergistic mixed infections (Jeude (2004). According to Gibson *et al* (1997), SPFMV alone causes no symptom in sweetpotato whereas SPCSV causes stunt growth, yellowing or purpling of lower leaves. The prevalence of SPVD is closely related to abundance of whiteflies (Aritua *et al.*, 1999). Symptoms include severe stunting of the plant and small malformed leaves such as leaf curling and dwarfing, leaf mottling and yellowing of veins sometimes with either a *chlorotic mottle* or vein clearing. These symptoms are most apparent in young sweetpotato plants as they get established, although plants can be infected at any age thereby reducing the plants' vigour and yield. It was observed by SASHA (2012) that the virus diseases spread by insect vectors such as whiteflies and aphids build up over time in the sweetpotato plants with each passing season. Some sweetpotato varieties are more resistant to the virus infection than others and produce less yield loss while susceptible varieties may disappear completely within one or two seasons in areas with high virus pressure Kreuze, (2008) observed.

Nwankwo and Opara (2015) reported of a low investment into breeding virus resistant genotypes, and selection procedures to meet farmers' needs by formal plant breeding. Most imported exotic germplasm is highly susceptible to virus infection (Gibson *et al*., 2008) in the locality. This makes successful breeding for virus resistant progress long term in nature and complicated in designing parental breeding programmes. The cloning characteristic of sweetpotato plant permits rapid and wide dissemination of successful genotypes and varieties respectively and the exploitation of heterosis which is an important genetic factor for yield, yield stability, adaptability and a significant index for disease resistance. However, the multiplication and maintenance of vegetative propagules in virus loaded environment means multiplication, maintenance and dissemination of virus loaded materials. Viruses attack the leaves and floral parts of parents' plants for breeding purposes, cause flower abortion and render the whole plant un-reproductive.

To achieve medium to long term virus resistant sweetpotato varieties is a challenge to sweetpotato breeding because the performance of a parent in one generation is not a good indicator for the value of a parent for the next generation. This is because the next generation sweetpotato plants might accumulate enough virus inoculum for onward transmission to the next succeeding generations thereby continue the propagation of virus disease. At a point this might lead to genotype decline and a loss of the genotype. Since viruses in sweetpotato plant is systemic, any seemingly symptomless sweetpotato vine collected for propagation in virus endemic areas will eventually manifest the virus disease even in screen houses.

Adaptation Measures as a Means for Acquiring Disease Resistance

The adaptation of sweetpotato populations to new needs (environments, quality demands, tolerance to pests and diseases) will be achieved quite rapidly through crop evolution. This means evaluating the sweetpotato genotypes in hotspot disease areas to enable the genotypes acquire resistance through adaptation. Examples for this potential abound in the sweetpotato gene pool where it is possible to find genotypes which are specifically adapted to drought, heat, cold (in tropical highlands), mineral-stress (including acid soils) or extreme salinity. The yield, yield stability and adaptability (including genotype by environment of crops) are often associated with resistance to biotic and abiotic stresses. Newly bred sweetpotato genotypes succumb/broke down to viruses after a few years' trial as result of accumulation of the virus disease each year, being vegetative propagated. This is why breeding sweetpotato genotypes with long time virus resistance is still a big challenge. However, acquiring resistance through exposure in hotspot areas could confer long time resistance to certain sweetpotato parthogens.

It was noted by Nwankwo and Opara (2015) that sweetpotato SPCSV resistance has been found in germplasm screening programs and the resistance appears to be conferred by a recessive allele that occurs in low frequency in the sweetpotato gene pool. Field resistance to sweetpotato virus disease (SPVD) has been obtained in East Africa by screening large numbers of sweetpotato genotypes from mainly local germplasm, and open pollinated seed and limited controlled cross progenies, evaluated on-station and on-farm. It is certain that sweetpotato varieties with resistance to SPVD will result in significantly higher yields and yield stability in Nigeria. National breeding programmes in Nigeria should focus on selecting for resistant sweetpotato varieties since 'resistant' is a very important trait in sweetpotato breeding. This could be carried out by evaluating the sweetpotato germplasm in fields with virus hotspots. However, resistance goes a long way in protecting sweetpotato plants for losses due to diseases and pests. It is the cheapest way for disease control and does not add to cost of production of the crop. Acquired resistance is the only control measure against SPVD diseases of sweetpotato. Acquired resistance is environmentally friendly unlike pesticides and fungicides which are hazardous to humans and beneficial insects. Disease resistance could also be acquired by inheritance through hybridization and by incorporation through field evaluation in hotspot virus zones.

Landraces a Source of Resistance to Sweetpotato

Nwankwo and his co-workers in 2017 described sweetpotato landraces to include chance seedlings which have been selected and cultivated by farmers for millennium of years although has not been officially released as variety or varieties. Sharrma, (1980) acknowledged that crop species of landraces make significant contribution in the diet of the people or as varieties in the farming system of the people or as progenitors in breeding programme for the farmer preferred traits although this have not been scientifically evaluated and release as varieties. The genetic variability present in the unscientific bred sweetpotato population is not deliberately created by man but it is naturally present and therefore referred to as landraces (Teshome and Amenti 2010). Farmers in the sweetpotato growing States of Nigeria depend on the sweetpotato landraces for survival. Sweetpotato landraces are adapted to their local areas and have developed resistance to local pests and diseases. Sweetpotato landraces gained recognition from farmers as result of their good qualities and as such, could be used for genetic recombination (Nwankwo, *et al*., 2012). The landraces also contain valuable sources of resistance to important diseases and pests, capable of adaptation to environments where sweetpotato is grown, and other desirable characteristics such as high dry matter content which is associated with culinary qualities preferred by consumers (Huamán, *et al*., 1999). Huaman, *et al.,* (1999) also pointed out that landraces could be a source of resistance or immunity to sweetpotato virus diseases (SPVD) which has been a hindrance to sweetpotato cultivation.

Acquired Resistance/Tolerance to Sweetpotato Virus Diseases

Nwankwo and co-workers (2017) reported that to incorporate disease resistant genes to the sweetpotato genotypes, artificial high disease pressure condition (epiphytotics.) are created. Infection works well when there is an outbreak of a plant disease that suddenly and rapidly affects many plants in a specific area. Such an area is essentially designed in the field trial. Although, the procedure depends on the type of disease, nature of the crop and place of screening. However, field screening is usually done on hotspots. Hotspots are locations where disease occurs naturally every year with considerable intensity by growing in screening nurseries such diseases as viruses. Sick plots are developed by growing susceptible variety year after year and even adding inoculum in a plot in the evaluation field. If the screening sweetpotato plants are grown in such a plot, only the resistant ones will survive. Some viral diseases which spread through insect vectors are inoculated by feeding insects on diseased plants. Such insects acquire virus and become viruleferous and are then released on the screening sweetpotato plants. Some varieties in coming in contact with the disease become resistant. The disease resistance acquired in this way is not from nuclear genes. That is the disease is not passed through the DNA in the nucleus during meiosis to be incorporated into the genome of the yam plant. It is acquired. This leads to immunity of the sweetpotato plant against the viral infection.

Viral Transformation (**Transduction**)

This method packages the desired genetic material into a suitable sweetpotato plant virus and allows this modified virus to infect the sweetpotato plant. The genetic material which is the DNA can recombine with the chromosomes in the cytoplasm of the sweetpotato plant cell to produce transformant cells. The genomes of most viruses consist of single stranded RNA which replicates in the cytoplasm of infected cell of sweetpotato plant and form what is called transfection and not a real transformation. Transfection is the transfer of viral material or the infection of sweetpotato plant cell with viral DNA leading to the production of the virus in the cell. Since the inserted genes never reach the nucleus of the sweetpotato plant cell and do not integrate into the host genome, the progeny (that is the seeds) of the infected plants is virus-free and also free of the inserted gene. Sweetpotato plants which acquire resistance by being exposed to viral infection this way can remain viral free or resistant for a very long time (Balmori, et al. 2002). Acquired resistance is said to occur when a particular microorganism obtains the ability to resist the activity of a particular antimicrobial agent to which it was previously susceptible.

Acquired Resistance Through Cytoplasmic Defense

The plasmids in the cytoplasm of the sweetpotato plant cell contain extra chromosomal DNA. A plasmid is a small DNA molecule within a cell that is physically separated from a chromosomal DNA in the nucleus and can replicate independently. In nature, plasmids often carry genes that may benefit the survival of the sweetpotato plant. For example, plasmid can help the sweetpotato plant be antibiotic resistance. While the chromosomes are big and contain all the essential genetic information for living under normal conditions, plasmids usually are very small and contain only additional genes that may be useful to the sweetpotato under certain situations or particular condition. Plasmids may carry genes that provide resistance to naturally occurring antibiotics in a competitive environmental niche for the sweetpotato plant, or the proteins produced may act as toxins under similar circumstances, or allow the sweetpotato plant to utilize particular organic compounds that would be advantageous when nutrients are scarce in the soil environment (Marquis, 2009). Plasmids carry hereditary information in the form of genes, the basic units of inheritance (known as fertility plasmids) in order to transfer DNA to another bacterium. Marquis (2009) observed that plasmids generally carry fewer genes than do chromosomes, and that the genes that they carry are useful, but they are not essential, to the survival of the sweetpotato plant cell. For example, some plasmids help bacteria make use of unusual food sources, such as camphor or petroleum and could assist in disease prevention. Certain sweetpotato plasmids in the cytoplasm that is resistance enable bacteria to degrade or inactivate antibiotics used to halt bacterial growth, or to survive in the presence of heavy metals by converting the metals into less toxic forms. Other plasmids in the sweetpotato plant cell enable the bacteria to produce chemicals that are toxic to other organisms, including insects, humans, and other bacteria. According to Marquis (2009), plasmids are important tools that are used in genetic engineering. Marquis (2009) also noted that the structure of DNA is the same in all living cells, so DNA from almost any organism can be combined with plasmid DNA. Plasmids therefore serve as convenient vehicles for transferring genes from one organism to another and could be used in conferring disease resistance in sweetpotato plants.

For instance, plasmids are used in the genetic engineering of plants. It involves a plasmid known as a tumor inducing, or Ti, plasmid found in *Agrobacterium tumefaciens*, the bacterium that causes crown gall disease in plants. When the *Agrobacterium tumefaciens* bacteria enter a plant such as sweetpotato plants through a wound, they transfer the Ti plasmid into nearby sweetpotato plant cells. The presence of the plasmid changes the growth of the sweetpotato plant cells, resulting in the formation of a large tumor on the sweetpotato plant. This natural process is used by scientists to manipulate the bacteria *Agrobacterium* by removing the Ti plasmids from the *Agrobacterium tumefaciens* cells and to replace the plasmids' tumor-causing genes with genes that code for desirable characteristics. Nwankwo and Opara (2015) reported that recombinant plasmids are being introduced into plant cells to produce crop plants that are resistant to certain diseases, insect pests, or herbicides. This recombinant plasmid technique despite using it for inducing disease resistance in nature could also be used to improve the nutritional food value of sweetpotato plants.

Acquired Disease Resistance Through Genetic Exchange

Genetic exchange is a process through which a bacterium incorporates the DNA into a virus. When the virus infects a bacterial cell, it picks up part of the bacterial DNA. If the virus infects sweetpotato plant cell, it carries with it DNA from the first organism. This method of

DNA exchange is called transduction. Transformation and transduction generally transfer only small amounts of DNA. Marquis (2009) reported that many bacteria are also capable of transferring large amounts of DNA or even the entire genome (set of genes), through physical contact. The donor cell generally makes a copy of the DNA during the transfer process so that it is not killed. This method of exchange is called conjugation. DNA exchange enables bacteria that have developed antibiotic resistant genes to rapidly spread their resistance to other bacteria. Most of these processes take place within the cytoplasm of the cell and between the cytoplasm of cells. Therefore, bacteria causing pathogens could transfer large amounts of DNA through physical contact of the cells of sweetpotato plants thereby conferring resistance to certain major diseases attacking the crop in the field. That is why while breeding for disease resistance in sweetpotato genotypes, the diseased plants or host diseased plants are included in the evaluation plots to enable the new sweetpotato genotype acquire/develop resistance to the target pathogen(s).

Acquired Resistance Through Necrotic Defense Reaction

Sweetpotato plants during field evaluation acquire immunity/ resistance through Necrotic Defense Reaction. The Necrotic Defense Reaction work like hypersensitivity. For example, If the pathogen may penetrate the cell wall of the sweetpotato plant and establishes contact with the protoplast of the cell, the nucleus is triggered into moving toward the invading pathogen and disintegrate it resulting into brown, resin like granules forming in the cytoplasm. This brown resin like granules first form around the pathogen and then throughout the cytoplasm. As the browning discoloration of the cytoplasm of the plant cell continues, it led to the death of the invading hypha. It then begins to degenerate stopping further invasion of the cells and tissues of the sweetpotato plant. In bacterial infections of leaves of the sweetpotato plant, the hypersensitive reaction results in destruction of all cellular membranes of cells in contact with the bacteria, and that is followed by desiccation and necrosis of the leaf tissues invaded by the bacteria. Bauer *et al*. (1995), observed that necrotic type of defense is common, in diseases caused by obligate fungal parasites and by viruses and nematodes. He further reported that the necrotic tissue isolates the obligate parasite from the living cells and tissues of the plant on which it depends absolutely for its nutrition resulting in its starvation and death. The faster the sweetpotato cell dies after invasion, the more resistant to infection the sweetpotato plant seems to be. The isolated and death of the sweetpotato plant cells and tissues that fell out of the leaves form spots on the leaves causing leaf-spot disease of sweetpotato.

Acquired Resistance Through Cytoplasmic Defense Reaction

In cytoplasm defense reaction Agrios (2005) stated that most pathogens manage to penetrate the cells of the plant in the field through wounds and natural openings and to produce various degrees of infection. The sweet potato plants respond by forming structures that are more or less successful in defending the plant from the pathogen invasion. Then the sweetpotato plants cells under attack had to defend themselves through cytoplasmic defense reaction. According to Chaudhary (2001), hypersensitive reactions is a potential defense reaction which resulted as an increased sensitivity of hosts cells in the vicinity of infection site and is expressed in the necrotic area. However, Agrios (2005) reported that necrotic tissue separates the parasite from the living host and thereby stops further growth of the pathogen. This type of resistance is governed by cytoplasm factors.

Cao, and Dong, (1998), observed that hypersensitive response is a biochemical defense mechanism that took place in the cytoplasm of the cell of the plant. It happens as soon as the pathogen attack the sweetpotato plant and establishes contact with the cell of the host sweetpotato plant, the nucleus of the attacked cell moves toward the invading pathogen, attack and disintegrates it. This resulted into brown, resin-like granules to form in the cytoplasm, first around the point of penetration of the pathogen and then throughout the cytoplasm (Agrios, 2005). As the browning discoloration of the sweetpotato plant cell continues, death sets in, the invading hypha begins to degenerate and die. In most cases the hyphae do not grow out of such cells thereby stopping the invasion of the cells of the sweetpotato plant.

When there are bacterial infections of sweetpotato leaves, the hypersensitive response results in the destruction of all cellular membranes of cells in contact with the bacteria, which is followed by desiccation and necrosis of the leaf tissues invaded by the bacteria. The necrotic tissue of the sweetpotato leaf isolates the parasite from the living substance on which it depends for its nutrition resulting in the bacteria starvation and death (Loebenstein 2009). This inactivated the pathogen. The faster the sweetpotato plant cell dies after invasion, the more resistant to infection the sweetpotato plant seems to be. The compounds and pathways developed by the sweetpotato plant during the hypersensitive response latter serves as the springboard for localized and systemic acquired resistance for the sweetpotato plant. Agrios (2005) acknowledged that the cytoplasm plays a crucial part by surrounding the clump of hyphae, and disintegrates the mycelium of the pathogen. This stops the invasion of the pathogen thereby leading to the survival of the sweetpotato plant.

Pluses for Breeding Sweetpotato with Natural Defense Systems

One of the most effective means of controlling plant diseases in sweetpotato crops is the breeding of resistant varieties. This is because Sweetpotato plants' use their natural defense systems during disease development to protection themselves. Plant breeders select resistant sweetpotato cultivars with genes for countering harmful strains of pathogens. Diseases, caused by vascular pathogens and viruses that cannot be controlled adequately by other means, the use of resistant sweetpotato varieties provides the only means of producing yields without the use of pesticides {Gaudet, *et al*. 2000). According to Agrios (2005), there are pluses for using plants that acquired resistance. The use of resistant varieties is the least expensive, easiest, safest, and without polluting the environment using chemicals. Therefore, adaptation breeding to incorporate resistance to pathogens in clonally bred sweetpotato genotypes is a necessity in order to enable the sweetpotato genotypes acquire natural resistance. This will enable the variety or varieties acquired resistances to major pathogens attacking the sweetpotato crop before they are released as resistant varieties. After the release, sanitary management strategies are put in place to reduces the exposure of the sweetpotato variety to large pathogen populations (Agrios, 2005). The cultivation of resistant sweetpotato varieties not only eliminates losses from diseases but is also environmentally friendly (Agrios (2005).

CONCLUSION

Sweetpotato plants in the fields are attacked by of pathogens. Most sweetpotato plants have developed/acquired natural defense strategies that successfully ward-off these pathogens and overcome them. Therefore, sweetpotato plants that failed to inherit resistance genes could be made to acquire defense mechanisms through field evaluation in hotspot fields and agro - ecologies through adaptation breeding to incorporate resistance to pathogens in the clonally bred sweetpotato genotypes. This is to enable the sweetpotato genotypes acquires natural resistance to major diseases attacking the sweetpotato crop in the field. Breeding resistance sweetpotato genotypes through field adaptation evaluation eliminates yield losses from diseases and is environmentally friendly.

REFERENCES

Agrios, G. N. (2005) Plant pathology 5th ed. Elsevier Academic Press 30 Corporate Drive, Suite 400, Burlington, MA 01803, USA 525 B Street, Suite 1900, San Diego, California 92101-4495, USA 84 Theobald's Road, London WC1X 8RR, UK.

Ahmad M. H., Morrison E. Y., Asemota H. N. (2006). "Food processing methods influence the glycermic indices of some commonly eaten West Indian carbohydrate-rich foods". Br. J. Nutr. 96(3):476

Ames T (ed.) (2002) Food and Health for the Future. Acta Horticulturae. ISHS: Proceeding of the First International Conference on Sweet potato. Brugge, Belgium: ISHS. p583.

Aritua, V., Legg, J.P., Smit, N.E.J.M., Gibson, R.W. (1999). Effect of inoculum on the spread of sweet potato virus disease: Limited infection of susceptible cultivars following widespread cultivation of a resistant sweet potato cultivar. Plant Pathology J. 48, pp 665-661.

Balmori, E., et al. (2002). Sequence, tissue, and delivery-specific targeting of RNA during posttranscriptional gene silencing. Mol. Plant-Microbe Interact 15, 753–763

Bauer, D. W., Tobia, G., Godson, N (1995). Erwinia chrysanthemi HarpinEch: An elicitor of the hypersensitive response that contributes to soft rot pathogenesis. Mol. Plant-Microbe Interact. 8, 484–491.

Cao, H., Li, X., and Dong, X. (1998). Generation of broad-spectrum disease resistance by over expression of an essential regulatory gene systemic acquired resistance. Proc. Natl. Acad. Sci. USA 95, 6531–6536.

Chaudhary, S.A. 2001. Flora of the Kingdom of Saudi Arabia illustrated. Vol. 3. National Agriculture and Water Research Centre, Riyadh, Saudi Arabia.

Gaudet, D. A., Kayoni F., Adams, S.O (2000). Expression of plant defense-related (PR_protein) transcripts during hardening and dehardening of winter wheat. Physiol. Mol. Plant Pathol. 57, 15–24.

Gibson, R.W., Aritua, V., Byamkama, j., Mpembe, I., and Kayongo, J (1997). Control Strategies for sweet potato virus diseases in Africa. ESARC - IITA and Namulonge, Agricultural and animal production Research Institute. P.O Box 7084, Kampala, Uganda (r.wgibson@gre.ac.uk).

Gibson, R.W., Byamukama, E., Mpembe, I., Kayongo, J., and Mwanga, R. (2008). "Working with farmer groups in Uganda to develop new sweet potato cultivars: decentralization and building on traditional approaches". Euphytica 159: 217 – 228.

Gibson, R.W., Jeremiah, S.C., Aritua, V., Msabaha, R.P., Mpembe, I. Ndunguru, J. (2000)." Sweet potao virus disease in sub-sahara Africa; Evidence that neglect of seedlings in the traditional farming system hinders the development of superior resistant landraces." Phytopathology 148, PP441-447.

Huamán, Z., Aguilar C., Ortiz R. 1999. Selecting a Peruvian sweet potato core collection on the basis of morphological, Eco-geographical and disease and pest reaction data. Theoretical and Applied Genetics 98:840-845

Islam, M.J., Haque, M.Z., Majunder, U.K., Haque, M.M. and Hossain, M.F. (2002). "Growth and yield potentials of nine genotypes of sweet potato". Pakistan J. Biol. Sci., 5(5): 537-538.

Jeude, J.L.(2004). Irene, K (ed) "Identification of crop damage caused by pests or mineral deficiencies". Agrodok, 28 CTA, P.O.Box 6700AJ, Wageningen. The Netherlands pp35 - 37.

Karyeija, R.F., R.W. Gibson and J.P.T. Valkonen., (1998). "The significant of sweet potato feathery mottle virus in subsistence sweet potato production in Africa". Plant diseases 82 (1).

Kreuze, J and S. Fuentes (2008). Sweet potato Viruses. International Potato Center (CIP), Lima, Peru. ã Elsevier Publishers Ltd.

Kreuze, J. (2008) Acta Universitatis agriculturae Sueciae. Agraria, vol. 335: Molecular studies on the Sweet Potato Virus Disease and Its Two Causal Agents. Doctoral Dissertation, Dept. of Plant Biology, SLU (ISBN 91–576–6180–4; ISSN 1401–6249).

Loebenstein, G. Fuentes, S. Cohen, J. and Salazar, L.F (2009). Sweet potato. In: Loebenstein G and Thottappilly G (eds.) Virus and Virus-Like Diseases of Major Crops in Developing Countries, pp. 223–248. Dordrecht, The Netherlands: Kluwer Academic Publishers. P108.

Marquis, Robert E. "Bacteria." Microsoft® Encarta® 2009 [DVD]. Redmond, WA: Microsoft Corporation, 2008.

Nwankwo, I. I. M., Nwaigwe, G.O and Njoku, J.C (2014). Farmers' Varietal Assessment of Advanced Sweetpotato Genotypes Evaluated On-farm.IOSR Journal of Agriculture and Veternary Science Volume 7, Issue 1 Ver. 1 (Jan.2014), pp86 - 92.

Nwankwo, I.I.M (2012). Performance of Introduced and Locally Bred Orange Fleshed Sweet potato Varieties in the Rainforest Ecology of Southeastern NigeriaInternational Journal of Applied Research and Technology; Published by Esxon Publishers 2 (6): 48 - 54

Nwankwo, I.I.M., Ikoro, A.I., Akinbo, O.K.1, Nwaigwe, G.O., Okereke, A.C and Davids, E.C.F (2018) Overview of the importance of existing yam (*Dioscorea species*) diversity in farmers' fields their implications for breeding programmes Agricultural Science Research Journal April - 2018. Volume (8) Issue (4): pp 71 – 82.

Nwankwo. I.I.M & Opara, E.U (2015). The Role of Virus Vectors in Orange Fleshed Sweet potato Genotypes Infection - A Case Study. Global Journal of Science Frontier Research D Agriculture and Veterinary. Volume 15, Issue 2 Version 1.0 Year 2015. Global Journal Inc.(USA) Online ISSN:2249 - 4626 & Print ISSN: 0975 - 5896.

SASHA (2012). " Net Tunnel to production sweet potato planting material from Disease". Sweet potato Action for Security and Health in Africa. A Guide to Construct and Maintain Tunnels. pp 1 -2

Sharrma, A.K (1980). The Principles and Practice of plant breeding. Oxford University press. New Delhi.IPSIO. p345

Stathers, T., Namanda, S., Mwanga, R.D.M., Khisa, G., Kapinga, R. (2005). "Manual for Sweet potato Integrated Production and Pest Management Farmer Field Schools in Sub-

Saharan Africa". International Potato Center Kampale, Uganda pp. 61-62. ISBN 9970-895-01-X.

Teshome A, Amenti C (2010). On farm participatory evaluation and selection of sweet potato (Early, Medium and Late set) varieties at Adami Tulu Jiddo Kombolcha District. Int. J. Agric., 2(4): 1-5

Wolfgang, G., Robert, M., Maria, A. and Harrison, D. (2012). "Unleashing the potential of sweet potato in Sub-Saharan Africa: Current challenges and way forward". Challenge Theme Paper 1: Sweet potato breeding. CIP Social Sciences Working Paper 2009– 1.

Woolfe, J.A. (1992)." Sweet potato: An Untapped Food Resource". Cambridge Univ. Press, Cambridge, UK.