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Virus diseases of root crops in Africa: An overview

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Abstract. Virus diseases are some of the most economically important constraints to the production of the root crops: cassava, sweetpotato and yams in sub-Saharan Africa. Cassava is affected primarily by two virus diseases: cassava mosaic disease (CMD) and cassava brown streak disease (CBSD). CMD, caused by a group of cassava mosaic begomoviruses, is present in all cassava-growing areas of Africa, on average affects half of all plants, and is responsible for continent-wide losses in excess of US$ 1 billion annually, arguably making it globally the most important plant virus disease. This impact is currently being exacerbated by a pandemic of an unusually severe form of the disease. CBSD, caused by cassava brown streak virus, an ipomovirus, is most important in coastal East Africa where it leads to lost production through producing a brown necrotic root rot. Recent reports suggest it may also now be damaging cassava in parts of central Africa. Sweetpotato in Africa is attacked by a large number of viruses, although only two are of major economic importance. Sweetpotato chlorotic stunt virus, a crinivirus, and Sweetpotato feathery mottle virus, a potyvirus, commonly occur in sweetpotato plants in mixed infection, giving rise to sweetpotato virus disease (SPVD). In farmers' fields, incidences of greater than 30% are frequent, particularly in the Lake Victoria zone of East Africa, where the disease is most damaging. However, perhaps SPVD's most pernicious effect is to prevent farmers growing superior high-yielding genotypes, particularly ones from outside Africa, as these are generally extremely susceptible to it. Yam is similarly affected by a broad range of viruses, but the most widespread and economically damaging is Yam mosaic virus (YMV), a potyvirus, which occurs throughout the major yam-producing zone of West Africa, commonly at incidences in excess of 50%. Other potyviruses, a badnavirus (*Dioscorea alata* virus), and other viruses (including a cucumovirus and a comovirus) cause leaf symptoms that result in chronic and sometimes severe tuber losses. Whilst the virus threat to African root crops appears to be as great as ever, substantial progress has been made in developing control methods, most notably through the development of virus resistant germplasm and by identifying appropriate cultural controls. The challenge for the future will be to combine these approaches into integrated management strategies and ensure that they reach the producers for whose livelihoods these root crops are so essential.

Introduction

Root Crops in Africa. Tropical root crops are a vital component of the food systems of sub-Saharan Africa, most importantly within the humid and semi-humid zones lying between the Tropics of Capricorn and Cancer. A diversity of root crops is cultivated, but the three of greatest importance in terms of their distribution and overall production are cassava (*Manihot esculenta* Crantz), sweetpotato (*Ipomoea batatas* L.) and yams (*Dioscorea* spp.). Cassava is grown virtually

throughout the tropical belt of sub-Saharan Africa, running from Senegal in the north-west to Madagascar in the south-east. Total fresh weight production is greater than 100 million tonnes (FAO, 2003), and whilst cassava is a semi-perennial, typically maturing in 12 months, much of its success lies in its tolerance of drought and poor soils. Sweetpotato matures in four to six months, is also relatively tolerant of drought, but is primarily cultivated in the Great Lakes region of East and Central Africa. Continental production is slightly over 11 million tonnes (FAO, 2003). A number of yam species are commonly cultivated. These include: white yam, *Dioscorea rotundata* Poir., water yam *Dioscorea alata* L. and yellow yam *Dioscorea cayenensis* (Lam.). The species have different geographical distributions, but the main production zone is in the central-southern part of West Africa running from Nigeria to Ivory Coast. White yam is preferred in Nigeria, Benin and Togo but there is wider cultivation of yellow yam further west in Ghana and Ivory Coast. Total production of yams in Africa is > 38 million tonnes (FAO, 2003). Yams require higher potential growing conditions than either cassava or sweetpotato and have a complex set of cultivation and harvesting systems. Significantly, they also have important functions within the social systems of the major producer countries in West Africa, particularly Nigeria. As a result, they have a very high local commercial value. All three of the crops are nevertheless primarily grown for subsistence, and there is limited development of processing and utilization options that might provide enhanced commercial opportunities.

Biotic Constraints to Root Crop Production. Yams are considered to be indigenous to Africa, whilst both cassava and sweetpotato have been introduced from Latin America. Partly as a consequence of this external origin, cassava had been relatively unaffected by major pests and diseases for much of its history in Africa following its introduction in the 16th century. Three of the most important pests/disease problems have arisen from recent (1970s) accidental introductions. These include the cassava mealybug (*Phenacoccus manihoti* Matt.-Ferr.), the cassava green mite [*Mononychellus tanajoa* (Bondar)] and cassava bacterial blight (*Xanthomonas axonopodis* pv. *manihotis*). The first two became continent-wide problems causing major losses in virtually all cassava-growing areas, but both are now under effective management following the implementation of classical biological control programmes (Herren and Neuenschwander, 1991). The two other major biotic constraints to cassava production are the virus diseases, cassava mosaic virus disease (CMD) and cassava brown streak virus disease (CBSD). Sweetpotato is attacked by a diverse range of pests and diseases, the most widely occurring and economically important of which are: sweetpotato weevils (*Cylas* spp.), sweetpotato butterfly (*Acraea acerata* Hew.), *Alternaria* leaf blight and sweetpotato virus disease (SPVD). Yams similarly have a wide range of pest and disease constraints. Nematodes, including *Scutellonema bradys* (Steiner and LeHew) and *Meloidogyne* spp. cause major losses and anthracnose disease, caused by the fungal pathogen *Colletotrichum gloeosporioides* Penz., is also a key production constraint. Yams are affected by a number of virus species, the most important of which is *Yam mosaic virus* (YMV), which occurs throughout the major yam-producing zone of West Africa.

Cassava, sweetpotato and yams are all propagated through vegetative material, although there is some limited use of seed by farmers. The use of vegetative propagation means that viruses that infect parental material are carried with vegetative propagules into the new crop. This is a key facet of the biology of the interaction between viruses and their root crop hosts. It helps to explain why virus infection can be found in virtually all fields of the three crops yet also why, under normal circumstances, symptoms are chronic and mild rather than acute and severe, since viruses that have limited effects on their hosts

are more likely to be propagated than those that have more drastic and noticeable effects. Vegetative propagation, whilst perpetuating systemic virus infections, does not facilitate spread. Adaptation to transmission by insect vectors, however, allows severe virus variants or virus combinations to be carried rapidly and over large distances in epidemics.

Virus Diseases of Cassava

Introduction. Although eight virus species have been reported from cassava in Africa (Calvert and Thresh, 2002), only two groups occur widely and cause economic damage. These are the cassava mosaic geminiviruses (CMGs) (*Geminiviridae*: *Begomovirus*) that cause CMD and cassava brown streak virus (CBSV) (*Potyviridae*: *Ipomovirus*) that causes CBSD.

CMD was first described more than a century ago (Warburg, 1894) and CBSD has also been recognised for many years, having first been reported from what is now Tanzania in the 1930s by Storey (1936). Symptoms comprise a chlorotic yellow or yellow-green mosaic on the leaves, reduction in size and deformation of leaves, and general plant stunting, all leading to reductions in tuberous root production. CMD is arguably Africa's most damaging pathogen. It occurs wherever cassava is grown in Africa (Fauquet and Fargette, 1990), affects approximately half of all plants (Legg and Thresh, 2003) and causes losses estimated at in excess of US$ 1,200 annually (Thresh *et al.,* 1997). During the 1990s, a pandemic of unusually severe CMD, first reported in Uganda (Gibson *et al.,* 1996; Otim-Nape *et al.,* 1997) was subsequently recorded as spreading widely through East and Central Africa (Legg, 1999) devastating cassava production and commonly leading to the abandonment of the crop. Most recent spread has been documented from north-eastern Burundi and eastern Gabon (Legg, unpublished data). As a result of this further spread of the pandemic, the economic importance of CMD continues to increase.

CBSD has been reported from Kenya, Tanzania, Mozambique, Zambia, Malawi and Uganda, but the incidence and effects of the disease are greatest in lowland coastal areas of Kenya, Tanzania and Mozambique where incidences commonly exceed 50% (Thresh and Mbwana, 1998; Hillocks *et al.*, 2002; Munga and Thresh, 2002). Symptoms include a blotchy chlorosis on the lower leaves of affected plants, brown streaky lesions on green stem portions and a sepia to brown dry corky rot in tuberous roots (Nichols, 1950). Losses occur primarily from the spoilage of roots resulting from the dry rot, but in severe cases, dieback of the stems may also occur leading to reduced tuberous root production (Hillocks *et al.,* 2001). There have been recent reports of the occurrence of CBSD further west in western Democratic Republic of Congo (Mahungu, unpublished data), which if verified, would represent a substantial extension of the known distribution, and significantly raise the economic significance of the disease.

Characterization and diagnostics. The first distribution map of CMGs in Africa was developed from virus diagnoses using serological techniques (Swanson and Harrison, 1994). Monoclonal antibodies were developed which when used with triple antibody sandwich enzyme-linked immunosorbent assay (TAS-ELISA) protocols could detect and differentiate between most isolates of African cassava mosaic virus (ACMV) and East African cassava mosaic virus (EACMV) (Ogbe *et al.,* 1996; 1997). The development and use of DNA-based diagnostic protocols using the polymerase chain reaction (PCR), however, was instrumental in showing the potential unreliability of serology-based diagnostics. Firstly, PCR techniques coupled with sequencing showed that the virus associated with the severe CMD epidemic in Uganda was a recombinant hybrid, in which a portion of the coat protein gene of the DNA A of ACMV was recombined with the rest of the DNA A

of EACMV (Zhou *et al.*, 1997). This strain became known as the Uganda variant of EACMV (EACMV-UG). Secondly, the new techniques also revealed the widespread occurrence of mixed virus infections, hitherto undistinguishable by serological methods. As the new diagnostics have become more widely practised, and sequencing has become quicker and cheaper, the amount of information available on the nature and organization of the bipartite genome of the CMGs has increased substantially. Six species of CMG are now recognised including ACMV, EACMV, South African cassava mosaic virus (SACMV) and three other EACMV-like species (Fauquet and Stanley, 2003). CMGs are revealed to be a highly complex, dynamic and adaptable group of viruses which would seem to explain their sustained and increasing success in infecting cassava in Africa.

Although there were unconfirmed reports of the aetiology of CBSD from various groups dating back to the 1970s, it was not until almost 70 years after the first description of the disease that the causal virus was definitively identified as cassava brown streak virus (Monger *et al.*, 2001a). Relatively insensitive serological diagnostic methods for the uncharacterized virus(es) associated with CBSD had been developed during the 1980s (Lennon *et al.*, 1986). However, following the characterization of the virus (Monger *et al.*, 2001a), which made use of reverse transcriptase-PCR (RT-PCR) and cloning technology in sequence determination, it became possible to develop much more sensitive RT-PCR-based diagnostic protocols (Monger *et al.*, 2001b). These have been used successfully to detect CBSV even in young symptom-free leaves of diseased plants, highlighting their sensitivity. Unlike CMGs, available sequence information on isolates of CBSV suggests that there is limited variability, and all isolates so far examined, albeit small in number, have had sequence homologies of greater than 90% (G. Foster, unpublished data). Very little information is currently available, however, and a much greater range of isolates from the diverse locations in which CBSV occurs will need to be characterized before more definitive statements can be made about CBSV variability.

Transmission and epidemiology. The whitefly *Bemisia tabaci* (Gennadius) was recognised as being the likely vector of CMGs from some of the earliest work on CMD, and this suspicion was confirmed experimentally as early as the 1930s (Kufferath and Ghesquière, 1932). Later studies provided more detail on the characteristics of the transmission process (Chant, 1958) and it was demonstrated that CMGs were transmitted persistently, with a minimum inoculation period of 10 minutes, and that the virus could be retained for at least nine days (Dubern 1979; 1994). Recent studies have compared transmission of different CMG species by different geographical populations of *B. tabaci* and shown that differences were relatively insignificant (Maruthi *et al.*, 2002). The corollary of this is that the most important determinants of patterns of virus spread in the field are *B. tabaci* abundance and the amount of virus inoculum. As a result of the persistent nature of CMG transmission by *B. tabaci*, external inoculum sources have the greatest bearing on the pattern of virus spread in initially CMD-free fields (Fargette *et al.*, 1990). A characteristic of the spreading 'front' of the CMD pandemic has been the co-occurrence of super-abundant populations of *B. tabaci* and a high frequency of severely diseased mixed ACMV+EACMV-UG infected plants (Harrison *et al.*, 1997). ACMV and EACMVs have been shown to interact synergistically in mixed infections (Harrison *et al.*, 1997; Fondong *et al.*, 2000; Pita *et al.*, 2001) and there is also preliminary evidence for similar positive interactions between *B. tabaci* and severely CMD-diseased cassava plants (Colvin *et al.*, 1999). A further key factor in the propagation of the CMD pandemic is the mobility of *B. tabaci* populations, and individual adults can fly distances of at least 7km (Byrne and Blackmer, 1996). The combination of an abundant and mobile vector, efficient transmission

mechanisms and the year round presence of the cassava crop in most areas where it is grown make for a very effective epidemiological system which lies at the heart of the success of the CMGs in Africa.

CBSD has been shown to spread naturally under field conditions (Nichols, 1950) and relatively steep gradients of spread have been described (Hillocks *et al.*, 1999). Attempts have been made to identify the vector, and most studies have tested the whiteflies, *B. tabaci* and *Bemisia afer* Priesner and Hosny. *B. tabaci* would seem to be the most likely candidate since other ipomoviruses are transmitted by this insect. All transmission efforts with *Bemisia* spp. have so far failed to give positive results, however, and a key facet of future research into CBSV will be to undertake comprehensive studies targeted at identifying the vector.

Management. Approaches developed for the management of the virus diseases of cassava are typical of those used for many virus diseases of crop plants. Greatest attention has been directed towards the development of virus resistance, although significant efforts have also been made to identify crop management and phytosanitation practices that provide control. Resistance breeding programmes for both CMD and CBSD were initiated in what is now eastern Tanzania in the 1930s (Jennings, 1957). The most successful approach for CMD involved interspecific crossing with the wild relative, *Manihot glaziovii* Muell.-Arg., followed by multiple backcrosses with cultivated cassava. Progeny had good resistance to CMD, although root quality required further improvement. Parent material obtained from Tanzania then formed the basis for a major and uninterrupted breeding effort at IITA, which ran from 1970 to the present day. The multigenic resistance provided by the interspecific crosses has subsequently been augmented by the incorporation of new sources of resistance identified from local West African landraces. Some of the most recently developed varieties combine virtual immunity to CMD with excellent agronomic and organoleptic characteristics. These now form the basis for CMD management programmes throughout the continent. Both *M. glaziovii* and *M. melanobasis* Muell.-Arg. were used in early efforts to develop resistance to CBSD (Jennings, 1957), with a certain amount of success, but following the termination of the Tanzania-based cassava breeding effort in the late 1950s, there was no further work on breeding for CBSD resistance until a major new IITA-led programme was initiated in 2001. CBSD resistance has also been identified as a trait for which there is good potential for the development of transgenic resistance, and new research initiated in 2003 aims to address this. In the current absence of good sources of CBSD resistance, on-going management programmes in coastal East Africa, Malawi and Mozambique are promoting the dissemination of local landraces identified as tolerant to CBSD (Hillocks *et al.*, 2002).

Phytosanitation has long been advocated as a means of controlling both CMD and CBSD, but the two principal elements, roguing and selection of disease-free planting material, are rarely practised, and clear guidelines on their use still remain to be developed. Crop management approaches such as intercropping, protecting susceptible with resistant varieties and isolation have all been proposed as possible additional control measures, but clearcut data on their effectiveness as CMD or CBSD control measure in the farm situation have yet to be presented. Studies of local landraces in Uganda have shown that cassava plants initially infected with mild strains of CMGs grow and yield better than initially CMD-free plants, suggesting the occurrence of a form of mild strain protection (Owor, 2002). Further research will be required, however, both to understand the mechanisms underlying this phenomenon and to identify ways in which it might be enhanced for the management of CMD in local landraces.

The development and deployment of virus resistance is likely to remain the primary tactic

used in the control of the two principal virus diseases of cassava for the foreseeable future. However, a range of alternative and potentially complementary methods do exist, and the challenge in the years ahead will be to develop an integrated approach to control that combine these elements into a sustainable strategy that is both effective and fits in with the requirements of farmers.

Virus Diseases of Sweetpotato

Introduction. Although sweetpotato was, like cassava, introduced to Africa from the Americas in the sixteenth century, it appears (despite less scientific attention) to be affected by a wider range of viruses. The most common, occurring throughout Africa, is sweetpotato feathery mottle virus (SPFMV) (*Potyviridae*: *Potyvirus*). Like other potyviruses, SPFMV is transmitted in the non-persistent manner by a wide range of aphid species. It causes either mild (typically purple or yellow ringspots or mottling on older leaves) or no symptoms in sweetpotato when infecting alone. Both the common (C) and the russet crack (RC) strains of SPFMV have been identified in Africa (Karyeija *et al.,* 1998). The second most common yet economically most important virus is Sweetpotato chlorotic stunt virus (SPCSV) (*Closteroviridae*: *Crinivirus*). It is transmitted in the semi-persistent manner by *B. tabaci*, which is common on sweetpotato. It may cause mild stunting and yellowing when infecting sweetpotato alone (Gibson and Aritua, 2002). However, its main economic damage results from its ability to break resistance to SPFMV (Karyeija *et al*., 2000a), enabling SPFMV to spread and multiply in sweetpotato plants and reach titres up to a thousand-fold higher than when it infects alone (Karyeija *et al.,* 2000b). SPFMV does not, however, synergise SPCSV. The synergy of SPFMV by SPCSV also enables SPFMV to be acquired and transmitted more readily by aphids (Aritua *et al.,* 1998b) and results in the severe disease known as sweetpotato virus disease (SPVD), characterised by severe stunting and distortion, vein-clearing and/or chlorotic mosaic to the leaves. Affected plants generally have a storage root yield less than 70% that of unaffected plants and many of the roots produced are small and unmarketable. SPVD appears to occur throughout sub-Saharan Africa. Two strains of SPCSV distinguishable serologically as well as by nucleic acid analyses have been found in Africa: $SPCSV_{WA}$ occurring in countries along the Atlantic Ocean and $SPCSV_{EA}$ in countries along the Indian Ocean and in more central countries such as Uganda and Zambia (Hoyer *et al*., 1996; Gibson and Aritua, 2002). Other viruses found in Africa include further members of the Potyviridae, including sweetpotato mild mottle virus (SPMMV), sweetpotato virus Y and sweetpotato virus G. Sweetpotato chlorotic fleck virus and sweetpotato caulimo-like virus have also been found. Their distribution is unclear and, though they may be locally common, it is unclear whether any cause appreciable economic damage. Cucumber mosaic virus (CMV) has also been reported.

Diagnosis of sweetpotato viruses. Graft-inoculating scions of suspect sweetpotato to *Ipomoea setosa* Kerr. provides a detection system for most sweetpotato viruses (though not a specific diagnosis) as all except perhaps CMV cause symptoms in test plants of this species. It is perhaps the most sensitive technique available, though limited by the partial systemicity of viruses such as SPFMV when infecting alone. Panels of antisera against viruses infecting sweetpotato are also available from the International Potato Center in Lima, Peru, and enzyme-linked immunosorbent assays (ELISA) are available for relatively sensitive detection and precise diagnosis. Several of the viruses have also been sequenced allowing polymerase chain reaction (PCR) tests to be developed.

Epidemiology of SPVD. Sweetpotato is traditionally propagated in Africa by foliar cuttings taken from mature crops or from crops resprouting from storage roots following

rainfall after prolonged drought. Consequently, farmers have the opportunity to inspect the foliage of, and reject, abnormal plants including SPVD-affected ones as sources of cuttings. This process usually ensures that cuttings are free from SPCSV apart from those obtained from recently-infected, asymptomatic parent plants. SPFMV, having more-or-less unnoticeable symptoms, is not similarly restricted but it appears that it is only partially systemic in many cultivars so only a minority of cuttings may be infected (Gibson *et al.*, 1997).

There is no evidence that wild plants are a common source of either SPFMV or SPCSV although a few *Ipomoea* species growing wild in Africa have been shown to be infected rarely with SPCSV and SPFMV. Like CMD, the rate of spread of SPVD into unaffected crops has been shown experimentally to be determined by the numbers of adult *B. tabaci* infesting crops and the numbers of SPVD-affected sweetpotato plants in the vicinity (Aritua *et al.*, 1999). SPFMV is itself carried into new crops in many of the cuttings and, aided by the high titres it reaches in dually-infected plants, appears to spread rapidly in the presence of SPCSV. Spread of SPCSV is generally the factor determining the incidence of SPVD and, perhaps because whiteflies transmit SPCSV semi-persistently (for only a few hours or at most for a day following acquisition), spread is predominantly from nearby source plants.

Control of SPVD. Since resistance to SPFMV is broken by infection with SPCSV, resistance to SPVD is based on resistance to infection by SPCSV. Varieties of sweetpotato differ widely in their susceptibility to SPVD: cultivars imported from the Americas for other valuable genetic characters tend to be extremely susceptible whereas some African landraces are extremely resistant – though immunity has not been recorded. Resistance to SPVD generally seems to be inversely related in landraces to yielding ability or other beneficial attributes (Aritua *et al.*, 1998a). Consequently, farmers may have to make compromises between these beneficial attributes and adequate SPVD resistance – and such compromises are another important means by which SPVD causes economic loss. Modern breeding has, however, been able to identify genes for resistance (Mwanga *et al*., 2002a & b) and to select high-yielding resistant varieties such as NASPOT 1, released in Uganda by the Namulonge Agricultural and Animal Production Research Institute.

In addition to the use of resistant varieties, the main way by which African farmers control SPVD is through careful selection of unaffected plants as sources of cuttings for their new crops. Careful questioning has, however, revealed that most farmers in East Africa do not realise that SPVD is a disease caused by viruses. In particular, they are not aware that whiteflies spread the virus that triggers it – although farmers that have received training in the control of CMD, especially those affected by the CMD pandemic, quickly appreciate it. Because most spread of SPCSV by whiteflies occurs over a short distance, roguing diseased plants in young crops to remove infection sources and planting new crops isolated from older ones have been shown experimentally to reduce spread considerably (Gibson *et al*., 2003). Consequently, following careful explanation and demonstration of how SPVD is spread, local phytosanitation is being evaluated together with East African farmers as a practical method of controlling SPVD.

Virus Diseases of Yam

Introduction. Most edible yam species are vegetatively propagated using planting setts, tuber pieces, aerial tubers, bulbils and, to a lesser extent, tissue culture. Where viruses infect the mother plants, they will be transmitted in the vegetative propagules and there will therefore be a tendency for build-up of virus diseases in the germplasm. The symptoms caused by viruses infecting yams range from almost symptomless infection or mild chlorosis / mottling to severe mosaics, leaf distortion, stunting and occasionally

dieback, depending on the virus. Infection by more than one virus can induce more severe symptoms. Host genotype also influences symptom expression and, where the growing conditions are sub-optimal, symptom expression is usually more severe.

Characterization and diagnostics. Yam mosaic virus (YMV) (*Potyviridae*: *Potyvirus*) is found in almost all regions throughout the world where yams are grown. YMV induces chlorotic flecking, green-banding, severe leaf chlorosis and leaf distortion. In *D. rotundata-cayenensis* and *D. esculenta*, the virus is widespread in West Africa and has been detected occasionally in *D. alata* in Nigeria (Hughes, unpublished data). YMV is mechanically transmissible to indicator plant species (*e.g. N. benthamiana*), which can be used for diagnosis of virus infection where other tools are not available (Brunt *et al.*, 1997). Both monoclonal and polyclonal antibodies are available which can be used in either TAS-ELISA or protein A-sandwich (PAS)-ELISA for identification of YMV in Africa. YMV can also be detected by tissue blotting of cut tubers, PCR using specific primers as well as the more broad-spectrum primers against the *Potyvirus* genus.

Dioscorea alata virus (DaPV), (*Potyviridae*: *Potyvirus*) is probably coincident with the culture of *D. alata* (Hughes, 1986; Mumford and Seal, 1997; Odu *et al.*, 1999; Olatunde, 1999), and is commonly found in West Africa. It causes mild or inconspicuous mottling, veinal necrosis, occasional vein-banding, leaf distortion and severe chlorosis (Odu *et al.*, 1999). DaPV can be detected using polyclonal antisera (Hughes, 1986; Odu *et al.*, 1999).

Other potyviruses have also been identified infecting *Dioscorea* species. Dioscorea dumetorum virus (DDV), (*Potyviridae*: *Potyvirus*) has been detected by ELISA in *D. alata* from Nigeria (Hughes *et al.*, 1997). 'Universal' potyvirus monoclonal antibodies are not appropriate for detecting potyviruses in yam sap extracts. False values are obtained in ELISA which may be a result of the composition of the host sap. However, it appears that the 'universal' antibodies can be effective when the virus has been transmitted to alternative herbaceous plant species.

A badnavirus, probably Dioscorea alata virus (DaBV), reported by Phillips *et al.* (1999) and Briddon *et al.* (1999) is associated with severe leaf distortion, crinkling and mottling in *D. alata* in Nigeria. The virus is mechanically transmissible to several *Dioscorea* species. ELISA using polyclonal antibodies can be used to detect DaBV. Immunosorbent electron microscopy (ISEM) can also be a useful tool to detect and identify the virus in its natural host. While specific primers are available for a West African strain of the virus, because of the high level of virus variability, the primers only work with some isolates (Briddon *et al.*, 1999).

Cucumber mosaic virus (CMV) (*Bromoviridae*: *Cucumovirus*) infects *D. alata*, *D. trifida* (Fauquet and Thouvenel, 1987; Migliori and Cadilhac, 1976) and *D. rotundata* (Dongo, pers. com.) in West Africa. Symptoms caused by CMV can include severe mosaic, leaf chlorosis, stunting and leaf distortion. ELISA can be used for detection of CMV using polyclonal antisera. Immunocapture (IC)-PCR can also be used and primer pairs are available which can detect CMV isolates that infect *Dioscorea* spp. (Brunt *et al*., 1997).

Dioscorea mottle virus (DMoV) infects *D. alata* in Nigeria (Hughes *et al*., 1997). Symptoms include mild chlorosis, mottling and necrosis. This virus is only partially characterised but has been found in samples from the yam belt of Nigeria (Hughes, unpublished data). DMoV is mechanically transmissible from *D. alata* (Hughes *et al.*, 1998) and therefore transmission to indicator species (*Vigna unguiculata*, *Glycine max*, *Chenopodium muralae*, *C. amaranticolor* and *C. quinoa*) can be used for diagnosis. Polyclonal antibodies are also available and these can be used in PAS-ELISA to detect the virus.

Transmission and epidemiology. Aphids are the most important insect vectors of yam viruses. A range of aphids (*Aphis fabae*, *A. craccivora*, *Rhopalosiphum maidis*, *Toxoptera citricidus*, *Myzus persicae* and *A. gossypii*) transmits YMV (Brunt *et al*., 1997) although the relative importance of individual aphid species as vectors in the field is unknown. *A. craccivora* and *R. maidis* also transmit DaPV (Odu *et al*., 1999). CMV is efficiently transmitted in nature in the non-persistent manner by a wide range of aphid species (including *A. craccivora* and *M. persicae*). Mealybugs may play an important role in the transmission of DaBV. *Planococcus citri* (Briddon *et al*., 1999) transmits DaBV and several other mealybug species have been found in association with yams in the field, but their role in transmitting this group of viruses is unknown. The natural insect vector of DMoV is not known.

Although vector transmission occurs and may play an important role in the epidemiology of yam viruses, one of the most important means of transmission is through vegetative propagation. As yams are traditionally multiplied through tuber setts or pieces, if the mother plant was virus infected, any tubers that are produced will also be infected with the same viruses, and consequently so will any progenies derived from these tubers. Observations in farmers' fields suggest that the larger tubers (most likely derived from healthy mother plants) are more likely to be sold or consumed, while the smaller tubers, which may have been derived from infected mother plants, may be used for propagation. As with all vegetatively propagated species, virus infection of the propagules often leads to slower germination and reduced vigour in the young plants. Continued propagation of virus-infected germplasm can lead to a build-up of more than one virus in the plants as a result of vector transmission in the field (or perhaps as a result of mechanical transmission when the planting setts are cut) thus causing significantly reduced vigour and reduced yields.

Management. Yam viruses are transmitted vegetatively from one generation to the next and from one season to the next using conventional vegetative propagation methods. An important method for controlling the diseases is therefore to use healthy planting materials. Farmers can improve the health status of their crops by ensuring that only propagules taken from yams that looked healthy during the previous growing season are used. Where resources are available it may be possible to use virus-tested plantlets produced in tissue culture. Where it is not feasible to use these plantlets because of their requirements for transplanting and 'hardening' before they can be planted in the field, mini-tubers can be produced in vector-proof conditions and distributed to seed yam producers for further multiplication and distribution.

Discussion and Conclusions

Viruses of the three principal root and tuber crops grown in sub-Saharan Africa comprise some of the most important constraints to their production. They are relatively more important than other groups of pests and diseases in part as a result of the importance of vegetative propagation which typically represents the most important source of infection in new plantings of cassava, sweetpotato and yams. All three crops are infected by a diversity of viruses, although cassava has only two groups that cause economic damage (Table 1). The two virus groups that affect cassava are largely confined to Africa, in contrast to sweetpotato and yams where most of the viruses have worldwide distributions. The importance of Nigeria in the distribution of yam viruses reflects the fact that it produces siginificantly more yams than any other country in the world.

Recent research has revealed a range of mechanisms that are helping the cassava mosaic geminiviruses to evolve, as evidenced by the frequent occurrence of recombinants and pseudorecombinants. Variability

Table 1: Viruses identified as affecting cassava, sweetpotato or yams in Africa.

Virus	Acronym	Genus	Family	Vector	Distribution
Cassava*					
Cassava mosaic geminiviruses	CMGs	*Begomovirus*	*Geminiviridae*	Whiteflies	Mainly Africa
Cassava brown streak virus	CBSV	*Ipomovirus*	*Potyviridae*	Unknown	Africa
Sweetpotato					
Sweetpotato feathery mottle virus	SPFMV	*Potyvirus*	*Potyviridae*	Aphids	Worldwide
Sweetpotato chlorotic stunt virus	SPCSV	*Crinivirus*	*Closteroviridae*	Whiteflies	Worldwide
Sweetpotato mild mottle virus	SPMMV	*Ipomovirus*	*Potyviridae*	Whiteflies	Mainly Africa
Sweetpotato chlorotic fleck virus	SPCFV	Unassigned	Unassigned	Unknown	Worldwide
Sweetpotato virus Y	SPVY	*Potyvirus*	*Potyviridae*	Aphids	Worldwide
Sweetpotato virus G	SPVG	*Potyvirus*	*Potyviridae*	Aphids	Worldwide
Sweetpotato caulimovirus	SPCaV	Unassigned	Unassigned	Unknown	Worldwide
Cucumber mosaic virus	CMV	*Cucumovirus*	*Bromoviridae*	Aphids	Worldwide
Yams					
Yam mosaic virus	YMV	*Potyvirus*	*Potyviridae*	Aphids	Worldwide
Dioscorea alata virus	DaV	*Potyvirus*	*Potyviridae*	Aphids	Worldwide
Dioscorea dumetorum virus	DDV	*Potyvirus*	*Potyviridae*	?aphids?	Nigeria
Dioscorea alata bacilliform virus	DaBV	*Badnavirus*	*Caulimoviridae*	Mealybugs	Nigeria
Cucumber mosaic virus	CMV	*Cucumovirus*	*Bromoviridae*	Aphids	Worldwide
Dioscorea mottle virus	DMoV	Unassigned	Unassigned	Unknown	Nigeria

*Additional unassigned viruses recorded from cassava in one or a few locations only in Africa include: *Cassava Ivorian bacilliform virus*, Cassava Kumi viruses, Cassava 'Q' virus and *Cassava common mosaic virus* (Calvert and Thresh, 2002).

amongst viruses of both sweetpotato and yams suggests that these pathosystems are similarly dynamic. Sweetpotato is host to viruses transmitted both by aphids and the whitefly, *B. tabaci*, and significantly, the whitefly-borne crinivirus, SPCSV, and the aphid-borne potyvirus, SPFMV, interact synergistically to give rise to the severe disease condition of SPVD. Although similar intergenus interactions do not seem to occur in cassava or yams, synergism is an important feature of the interaction of different CMG species.

The similar environmental requirements of both cassava and sweetpotato mean that they are commonly grown together. Although *B. tabaci* occurs on both crops, it is significant that evidence suggests that these are distinct biotypes that do not cross colonize (Legg, 1996). This has important implications for the development of 'banker crop' biocontrol approaches which use one or other of the crops to build up natural enemy populations to infest the other, since whilst natural enemies will move between the crops, the *B. tabaci* populations will not. Another important consequence of the frequent co-cultivation of these two crops is that where major losses occur to one, the other can be multiplied up as a substitute, a phenomenon that was an important feature of the management of the CMD pandemic.

All three of the root/tuber crops face major management challenges as a result of virus disease infection, and in all cases, host plant resistance has been developed as a key

control tactic. High levels of resistance to CMD have been developed through interspecific and local landrace crossing for cassava. Resistance will remain the key component in integrated control programmes set up to manage the acute epidemics associated with CMD and CBSD. The greater virus diversity, less acute damage and stronger reliance on vegetative propagation that characterize virus disease problems in sweetpotato and yams mean that a stronger emphasis is being placed on developing mixed management systems combining resistance with cultural methods, phytosanitation and seed health systems for yams. An additional challenge that faces those trying to develop and implement control programmes targeting virus diseases of root and tuber crops is the lack of awareness and understanding of these problems amongst farmers and agricultural extensionists. In the future, researchers will need to work closely with these groups in strengthening knowledge both through field-based training and in enhancing the quality and availability of training materials. Important synergies can be achieved in this work by considering the crops together, most particularly for cassava and sweetpotato where there are many commonalities in the virus problems and cropping environments.

Substantial progress has been made over the last decade in researching the viruses and virus diseases of the African root and tuber crops, and the benefits of the increased knowledge accrued are already being realised in countries where efforts have been targeted such as Nigeria, Ghana, Uganda and Tanzania. Much still remains to be done, however, and major research for development efforts will need to be sustained and strengthened over the next decade, if the virus diseases of these crops are to be effectively managed. Only if this is achieved will it be possible to realize the full potentials of these crops for the benefit of the hundreds of millions of sub-Saharan Africans who depend on them for their livelihoods.

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