THE VIRUSES AND VIRUS DISEASES OF CASSAVA IN AFRICA

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ABSTRACT

This review summarises the available information on the viruses and virus diseases of cassava in Africa. It then considers the distribution, prevalence and epidemiology of the two most important of these diseases: African cassava mosaic, which occurs in all the main cassava-growing areas, and cassava brown streak, which seems to be restricted to parts of eastern and southern Africa. The epidemiology of African cassava mosaic has been studied in few of the very diverse agro-ecological zones in which the disease occurs and there is even less information on brown streak. These are serious constraints in attempts to develop effective control measures. Information is also lacking on possible interactions between the two diseases and on their effects on the response of cassava to bacterial blight and other diseases and also to arthropod pests, nematodes and weeds. This emphasises the scope for a multi-disciplinary holistic ecological approach in any further studies aimed at developing fully integrated control measures to combat the whole range of cassava pests, diseases and weeds.

Key Words: African cassava mosaic, cassava brown streak, whitefly vector, *Bemisia tabaci*

RÉSUMÉ

Ce texte résume l'information disponible sur les virus et maladies virales du manioc en Afrique. Il traite ainsi de la répartition, de l'incidence et de l'édémiologie des deux principales maladies virales du manioc: la mosaïque africaine du manioc présente dans toutes les principales zones de culture du manioc, et le cassava brown streak qui semble être limité à certaines parties de l'Afrique de l'Est et d'Afrique australe. L'épidémiologie de la mosaïque africaine du manioc été étudiée dans quelques unes seulement des diverses zones agro-écologique où se trouve la maladie et il y a encore moins d'information sur brown streak. Les tentatives de contrôle de la maladie se heurtent ainsi à de sérieuses difficultés. Il n'y a pas suffisamment d'information non plus sur les interactions possibles entre les deux maladies et sur la réaction du manioc vis à vis d'autres maladies, arthropodes, nématodes et mauvaises herbes ennemis du manioc. Ceci souligne la nécessité d'une étude écologique multidisciplinaire de la biocénose parasitaire du manioc afin de développer une lutte intégrée contre l'ensenble des ennemis de la culture.

Mots Clés: Mosaïque africaine du manioc, cassava brown streak, aleurode, *Bemisia tabaci*

INTRODUCTION

Crops that are propagated vegetatively are particularly prone to virus disease problems because infection tends to build up in clones that are selected and used for repeated cycles of crop production. Cassava (*Manihot esculenta* Crantz) is no exception to this generalisation and 18 viruses or putative viruses have been isolated (Table 1). This paper summarises the information available on the eight viruses known to infect cassava in Africa and on the diseases they cause.

It is suggested that these diseases are more numerous, more prevalent and even more important than the present limited evidence suggests. Particular attention is given to African cassava mosaic and cassava brown streak diseases, the possibility of interactions with other pathogens, pests and weeds of cassava, and the need for a multi-disciplinary ecological approach.

THE VIRUSES OF CASSAVA

Cassava mosaic geminiviruses. What is now known as African cassava mosaic disease (ACMD) was first reported in 1894, in coastal Tanzania. The disease was later shown to be transmissible by grafts and by the whitefly now known as *Bemisia tabaci* Gennadius. In the absence of any visible pathogen, ACMD was at first assumed to be caused by a virus, but no particles were detected until 1975 when sap inoculations from cassava to the herbaceous host *Nicotiana clevelandii* Gray were successful (Bock, 1975). However, there was initial uncertainty as to the role of the geminivirus that was isolated in this way and it

TABLE 1: The viruses of cassava

Africa (9.60 mill. ha.*)

    African cassava mosaic geminivirus
    East African cassava mosaic geminivirus
    Cassava brown streak virus(es)
    Cassava Ivorian bacilliform virus
    Cassava Kumi viruses (2)
    Cassava 'Q' virus (= 'C' virus)
    Cassava common mosaic potexvirus

South/Central America (2.51 mill. ha.*)

    Cassava common mosaic potexvirus
    Cassava X potexvirus
    Cassava vein mosaic pararetrovirus
    Cassava Colombian symptomless potexvirus
    Cassava latent rhabdovirus
    Cassava American latent nepovirus
    Cassava Caribbean mosaic (potex)virus
    Cassava frogskin 'virus'

Asia/Pacific (3.89 mill. ha.*)

    Indian cassava mosaic geminivirus
    Cassava green mottle nepovirus

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* Areas of cassava from latest official estimates FAO, 1994).

was at first named cassava latent. This was because the virus was isolated from ACMD-affected plants in western Kenya, western Tanzania, Uganda, Nigeria and Ivory Coast, but *not* from similarly diseased plants in coastal Kenya (Bock *et al.*, 1978).

The uncertainty was resolved when successful inoculations were made to *Nicotiana benthamiana* from ACMD-affected plants in both western and coastal Kenya (Bock and Woods, 1983). The isolates obtained and characterised were related serologically to those from ACMD-affected cassava elsewhere in Africa and caused typical symptoms of the disease when returned to cassava, so fulfilling Koch's postulates.

The various isolates from mosaic-affected cassava in different parts of Africa and India were initially regarded as strains of the same virus. This was referred to as African cassava mosaic and serological differences were reported in tests with polyclonal antisera using the 'type strain' from western Kenya, the Kenya coast strain and a strain from India (Bock and Harrison, 1985). These and other isolates were later ascribed to separate West African ('A'), East African ('B') and Indian ('C') strain groups based on their serological properties in tests with a panel of monoclonal antibodies (Harrison *et al.*, 1987). However, on serological evidence and nucleotide sequencing, the three groups of isolates are now regarded as distinct viruses and referred to as African cassava mosaic virus (ACMV), East African cassava mosaic virus (EACMV) and Indian cassava mosaic virus (ICMV), respectively (Hong *et al.*, 1993; Swanson and Harrison, 1994). None of these three geminiviruses has been reported in South or Central America, and the mosaic disease of cassava that occurs there is caused by a completely different potexvirus.

Distribution. There have been no systematic surveys of the distribution of ACMV, EACMV and ICMV. Nevertheless, serological tests have been made on more than 150 isolates from mosaic-affected cassava in 22 countries of Africa or the Indian sub-continent. The results indicate that each of the three viruses has a separate and largely non-overlapping geographic distribution (Harrison *et al.*, 1995). Indian Cassava Mosaic Virus was detected only from India and Sri Lanka,

whereas the other two viruses were not reported outside Africa. East African Cassava Mosaic Virus has been detected only once in West Africa, in a sample collected in Ivory Coast from a variety introduced from Madagascar (Aiton *et al.*, 1988b). Otherwise it seems restricted to Malawi, Madagascar, Zimbabwe and the coastal regions of Kenya and Tanzania (Swanson and Harrison, 1994). African Cassava Mosaic Virus has not been detected in these areas, but it occurs in many West African countries (Benin, Burkina Faso, Ghana, Ivory Coast, Nigeria and Senegal) and also in Angola, Burundi, Cameroon, Chad, Congo, Mozambique, South Africa, Uganda, Zambia, western Kenya and Tanzania.

Such a clear distinction in the distribution of ACMV and EACMV within Kenya and Tanzania is unexpected and difficult to explain given the mobility of *B. tabaci* and the extensive traffic in vegetative material that must have occurred within these countries over many years. There is also likely to have been considerable movement of cuttings between coastal and inland countries of eastern and southern Africa along the railways, waterways and other communication routes. The difference between the viruses in eastern and western Africa is more readily explained by the greater distances and geographic separation and by the limited movement between the two regions until the extensive use of aircraft, by which time quarantine procedures had been introduced.

The separate geographical distribution of the three cassava geminiviruses is explained by Harrison *et al.* (1987, 1991, 1995) on the assumption that each virus spread from different indigenous plant species to cassava after the crop was introduced to Africa and later to the Indian sub-continent (Carter *et al.*, 1992). Harrison *et al.* (1987, 1991, 1995) further suggested that the current distribution of the three viruses reflects the geographic expansion of cassava cultivation following the three main points of introduction of the crop (a) to coastal West Africa and then to the north and east, (b) to Madagascar and East Africa then inland towards the west, and (c) to India and Sri Lanka.

The serological and other biochemical differences between the three cassava mosaic geminiviruses may reflect innate biological differences, even though they cause similar diseases and are transmitted similarly by *B. tabaci*. However, possible differences in such features as virulence to casava, effects on yield, host range, transmissibility by vectors and ability to infect resistant varieties have not been sought and this is an important topic for further research. Information is also required on possible interactions between the two African cassava mosaic viruses and whether they can occur together and interact in the same plant.

Cassava brown streak virus(es). Cassava brown streak disease (CBSD) was first noted in the early 1930s in the Amani district of what is now Tanzania. By 1935 the disease was known to be graft transmissible and the characteristic leaf, stem and root symptoms were recognised as distinct from those of ACMD (Storey, 1936). A virus was assumed to be responsible and additional evidence of this was obtained by successful sap inoculations from cassava to herbaceous test plants and back to cassava (Lister, 1959) and also by electron microscopy (Kitajima and Costa, 1964). Virus isolates in herbaceous hosts were later shown to have elongate particles 650-690 nm long (Lennon *et al.*, 1986). They resembled those of carlaviruses, but a relationship to definitive members of the group has not been demonstrated serologically or by other means (S. Seal, unpublished).

There is evidence that two different elongate viruses occur in CBSD-affected plants (Lennon *et al.*, 1986; Brunt, 1990) and isolates in herbaceous hosts induce 'pin-wheel' inclusions of the type caused by potyviruses (Harrison *et al.*, 1995). The possible role of a potyvirus in causing CBSD has not been determined and the aetiology of the disease remains uncertain (Bock, 1994a). Moreover, there seems to have been no attempt to confirm the suggestion by Storey (1939) that a species of whitefly is the vector. The whitefly *Bemisia afer* Priesner-Hosny (formerly known as *B. hancockii*) is certainly common on cassava in coastal areas of eastern Africa and Malawi where CBSD is most prevalent (Munthali, 1992; Legg, 1994). The greatest population densities of *B. afer* tend to occur on the lowest leaves of cassava, which are those that show the most conspicuous symptoms of CBSD (J.P. Legg, personal communication).

Distribution. Nichols (1950) reported CBSD in lowland coastal areas of East Africa from the north-east border of Kenya to the southern border of what is now Tanzania, up to an altitude of *c.*1100 m above sea level. He also reported the disease in Uganda and in Nyasaland (now Malawi), where it was widely distributed at lower altitudes in the southern areas, particularly towards what is now Mozambique, where CBSD was considered likely to occur. Cassava brown streak disease was assumed to have been introduced to Uganda in vegetative material brought from Amani, Tanzania in 1934 (Jameson, 1964).

Cassava brown streak disease was studied in Kenya during the 1980s (Bock, 1994a), but seems to have been largely ignored elsewhere until reports from Malawi and Tanzania in 1993 and from Uganda in 1994. The disease was recorded at only one site in southern Uganda (G.W. Otim-Nape and J.M. Thresh, unpublished), but it was prevalent in many parts of Malawi during 1993-1994 surveys when it was noted that symptoms tended to be ephemeral and inconspicuous (H.W. Rossel, R.F.N. Sauti, M. Soko, A. Sweetmore and J.M. Thresh, unpublished information). Cassava brown streak disease was also prevalent at the time in lowland coastal areas of Tanzania, where symptoms were conspicuous and sometimes associated with severe root necrosis (M.D. Raya, S.C. Jeremiah and J. P. Legg, unpublished reports).

Cassava Ivorian bacilliform virus. This virus was detected fortuitously during a survey made in 1987 to assess variation amongst isolates of ACMV occurring in the north-west of Ivory Coast (Aiton *et al.*, 1988a). It has not been reported elsewhere. The virus infects herbaceous species of at least six plant families and it has been purified and shown to have bacilliform particles with predominant lengths of 42, 49 and 76 nm that contain three species of RNA. An antiserum is available and the virus has been tentatively ascribed to the alfalfa mosaic virus group (Fargette *et al.*, 1991). It is not known if the virus causes symptoms and substantial yield loss in cassava, or whether it is transmitted by vectors.

Cassava virus Q. This virus has not been fully characterised or named and it is currently known by the accession code 'Q' (Harrison *et al.*, 1995),

although it was referred to as cassava virus 'C' in an earlier abstract (Aiton *et al.*,1988a). The virus has squat elongate particles (20 x 30 nm) and it has been transmitted mechanically to herbaceous test plants (*Nicotiana benthamiana* and *N. occidentalis*). Isolates of the virus have been made in UK from cassava plants introduced under import licence from Cameroon, Ivory Coast, Malawi and Zimbabwe, which suggests that it is widely distributed in Africa. The source plants from which the Cameroon isolates were obtained showed pronounced leaf fleck symptoms that were distinct from those of ACMD (J.M. Thresh, unpublished). The vigour of affected plants in Cameroon was not obviously decreased, but effects on yield have not been assessed and it is not known whether the disease is spreading or whether there is a vector.

Kumi viruses A and B. These two viruses have been transmitted mechanically to herbaceous hosts, but they have not been fully characterised or named (Harrison *et al.*, 1995). Kumi virus A has bacilliform particles (50 x 18 nm) and Kumi virus B has isometric particles 30 nm in diameter. Both viruses were isolated in 1991 from plants in the Kumi district of eastern Uganda. The plants showed a pronounced leaf mottle that was readily distinguishable from the symptoms of ACMD (G.W. Otim-Nape and J.M. Thresh, unpublished). The vigour of affected plants was not obviously affected; possible effects on yield were not assessed. It is not known whether the disease spreads naturally, but infection was common in two local varieties which suggests that it does. The area from which the Kumi viruses were obtained has since been severely affected by ACMD (Thresh *et al.*, 1994b). Consequently, cassava production almost ceased for a time throughout the whole district and the Kumi viruses are unlikely to have survived in the area unless they have hosts other than cassava.

Cassava common mosaic potexvirus. This virus causes mild mosaic symptoms and chlorotic areas that are often limited by the leaf veins (Silberschmid, 1938). The virus has elongate particles (495 x 15 nm) and is a typical member of the potexvirus group. Costa and Kitajima (1972) present additional details of host range, properties

and effects on yield. Cassava common mosaic virus was first isolated in Brazil but it is now known to occur throughout the Neotropics from Mexico to Paraguay. It has been isolated only once in Africa from a plant in Ivory Coast assumed to have been introduced from South or Central America (Aiton *et al.*, 1988b). No vector has been identified but the virus is highly infectious and it is transmitted on contaminated implements.

THE PREVALENCE AND IMPORTANCE OF VIRUS DISEASES OF CASSAVA IN AFRICA

In order to assess the overall importance of virus diseases within a crop such as cassava, information is required on the incidence of each disease and on their effects on yield. Both requirements are difficult to meet for cassava in Africa as the crop is grown so widely and under such diverse conditions. Moreover, many different varieties are grown and they differ greatly in their susceptibility and response to infection.

The following account is restricted to ACMD and CBSD because there is so little information on the diseases caused by the other five viruses of cassava known to occur in Africa.

African cassava mosaic disease. There is general agreement that ACMD is the most important disease of cassava in Africa and it was rated as the most important vector-borne disease of any African food crop in a recent economic assessment (Geddes, 1990). However, there are few data to support these assertions, even though the incidence of ACMD can be assessed quickly by inspecting a representative sample of plants within a stand and noting the proportion with symptoms. Reliable estimates can be obtained in this way provided that the plants examined are not severely affected by insects or mites, mineral deficiency or drought. It is also important to appreciate that results based on a single visual assessment underestimate the incidence of infection because some virus-infected plants may be symptomless at the time of inspection. An even greater limitation is the enormous size of the area to be surveyed as cassava occupies an estimated 9.6 million ha. in Africa and a 0.1% sample represents *c.* 10,000 ha

or *c.* 1.0 million individual plants if 100 plants are assessed per ha.

Uganda. The most detailed data on the incidence of ACMD were obtained recently in Uganda (Otim-Nape, 1993). Representative plantings were examined between 1990 and 1992 in three counties in each of the thirty-two districts where cassava is grown. There were large differences between plantings in the prevalence of ACMD, as shown by the frequency distribution of the data for the entire survey (Fig. 1a). The incidence of infection exceeded 50% in 583 (43%) of the 1350 plantings sampled and 57% of all plants were affected. The incidence was lowest in southern districts bordering Lake Victoria and highest in the drier mainly grassland savanna areas of central and northern Uganda where serious epidemics of ACMD have occurred in recent years and caused severe food shortages and hardship (Otim-Nape, 1993; Thresh *et al.*, 1994b).

Tanzania. In a similar but less comprehensive survey of Tanzania in 1993 and 1994 (M.D. Raya, J.C. Jeremiah and J.P. Legg, unpublished information) there was a generally lower incidence of infection compared with Uganda. African cassava mosaic disease was recorded in 259 (74%) of the 325 plantings sampled but the incidence exceeded 50% in only 17% of the plantings (Fig. 1b). The mean incidence of infection for the country as a whole was 26%. Infection was most prevalent in the lowland coastal districts, especially in the Tanga coastal area north of Dar-es-Salaam.

Chad. The incidence of ACMD has been assessed in 21 wadis of the Ngouri Sous-Prefecture where cassava is grown under semi-arid conditions using irrigation from bore holes (Johnson, 1992). The disease occurred in 44 (92%) of the 48 plantings surveyed and the overall incidence of infection was 40%. However, there was great variation between plantings, even within the same locality and some stands were totally infected (Figure 1c).

Malawi. The only other comprehensive survey of the prevalence of ACMD has been in Malawi (Nyirenda *et al.*, 1993) The incidence of infection

seldom exceeded 20% in upland areas at altitudes above 800 m, whereas infection was more prevalent at lower elevations where conditions are warmer and cassava is more widely grown. Infection exceeded 90% in some plantings near the western shore of Lake Malawi (773 m).

Kenya. Bock (1983) noted that the incidence of ACMD was generally high in coastal and western Kenya where it exceeded 80% in some districts and approached 100% in individual farms.

Ghana. The mean incidence of ACMD was 96% in a limited sample of ten farms (Walker *et al.*, 1985) and 85% in the 120 plants assessed in various parts of the country during Phase I of the later Rockefeller-funded Collaborative Survey of Cassava in Africa (COSCA) described by Nweke (1994). Additional information is now being obtained in the initial diagnosis stage of the Ecologically Sustainable Cassava Plant Protection Project (ESCaPP) which operates in Ghana, Benin, Nigeria and Cameroon as described by Yaninek *et al.* (1994).

Ivory Coast. It was noted in the 1980s that almost

all the plants examined were affected by ACMD, except those specially selected and propagated at isolated sites for experiments (D. Fargette, unpublished information). This observation is consistent with the later findings of the COSCA survey in which the overall incidence of ACMD was 82%.

Togo. Dengel (1978a, b) reported that ACMD occurred throughout all plantings of two widely grown local varieties.

Nigeria. Dorosch (1988) suggested that the losses caused by ACMD in Nigeria were equivalent to 16% of total production. This estimate was based on the assumption that losses were 40% and restricted to the unimproved varieties being grown in the south which accounted for 40% of the total area grown. No evidence was presented to support these assertions, or the assumption that ACMD was unimportant in the northern areas and in the improved varieties being grown in the south. Moreover, the assumptions are inconsistent with the results of the 1989 COSCA survey in which the overall incidence of infection was similar in
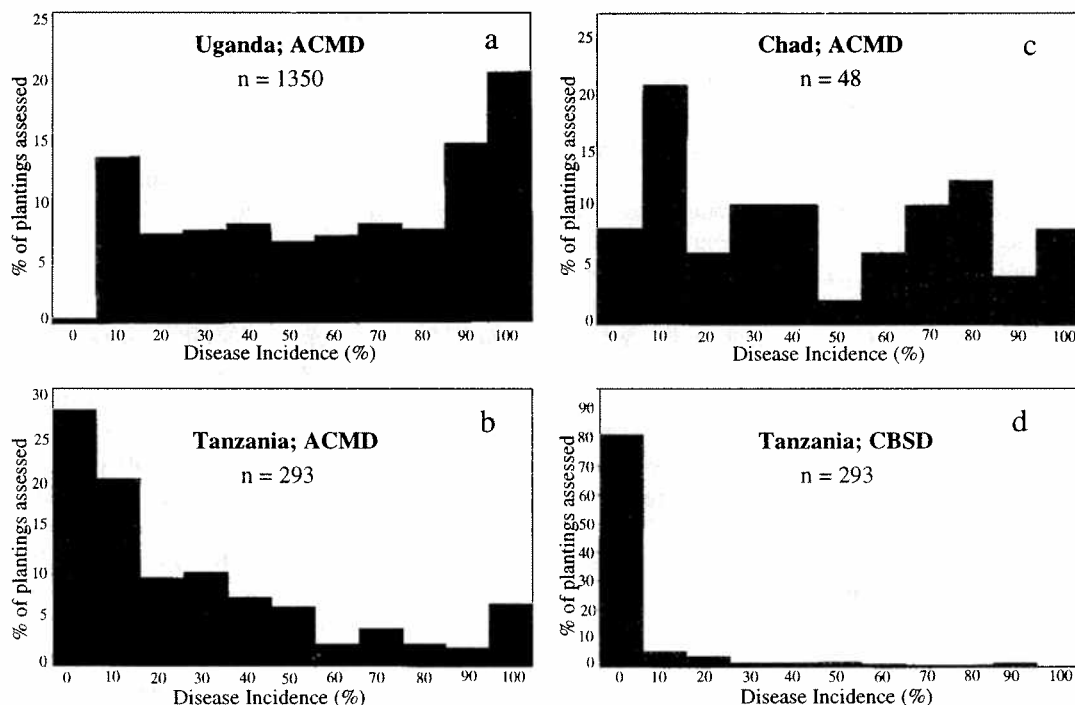

Figure 1. The prevalence of African cassava mosaic disease (ACMD) in Uganda (a), Tanzania (b) and Chad (c) and of cassava brown streak disease (CBSD) in Tanzania (d). Data for the percentage of plantings that were surveyed that fell in each disease incidence category (n=total number of farms surveyed). See text for sources of information. Note the difference in vertical scale used for the CBSD data.

the improved and unimproved varieties assessed (Nweke, 1994).

In a more comprehensive survey in 1994, a total of 1900 plants were assessed in 95 plantings at 63 sites in various agro-ecological regions (L. C. Dempster, unpublished information). The overall incidence of infection was 55% and the incidence was less in the southern forest zone (36%) than in the drier savanna areas to the north (64%) and at mid-elevations (1000-2000 m) on the Jos plateau (64%).

Elsewhere. Data are not available from other countries but it is clear that ACMD is not always prevalent and that unaffected or only slightly affected plantings can be found readily and in some areas predominate in Burundi, Cameroon, Zambia and Zimbabwe (J.M. Thresh, unpublished observations).

Cassava brown streak disease. The only detailed information on the prevalence of CBSD was obtained by Legg *et al.* during their 1993/1994 survey of ACMD in Tanzania referred to previously. Cassava brown streak disease was recorded in 62 (18.9%) of the 325 plantings assessed (Fig. 1d), and the overall incidence of disease for the country as a whole was only 6.0%. This is likely to be a considerable underestimate of the true incidence of infection because the symptoms of CBSD are much less conspicuous than those of ACMD and some varieties are extremely tolerant. Moreover, only the leaf and stem symptoms of CBSD were recorded and the tuberous roots were not examined for necrotic areas which sometimes occur in the absence of other symptoms.

The worst affected areas encountered in the survey were in the lowlands along the east coast from the Kenyan to Mozambique borders. Some plantings in the important cassava growing areas around the southern port of Mtwara were almost totally infected and the overall incidence of disease in the area was 28.5%. Farmers reported very severe losses due to severe necrosis of the tuberous roots which ultimately became unsaleable. In marked contrast, there was a generally low incidence of CBSD at uplands sites exceeding 1000 m above sea level and the disease was not

recorded on the Zanzibar islands of Pemba and Unguja.

The Tanzania survey confirmed the findings of earlier studies which established the importance of CBSD in some parts of the country. This emphasises the need for effective control measures and for similar surveys in Malawi, Kenya and other countries of southern and eastern Africa where CBSD is known or likely to occur.

ENVIRONMENTAL FACTORS INFLUENCING CASSAVA BROWN STREAK AND AFRICAN CASSAVA MOSAIC DISEASES

Cassava is grown in Africa in a wide range of agro-ecological conditions and in very diverse cropping systems (Dahniya, 1994). This is apparent from the maps and data presented by Carter *et al.* (1992) and from more conventional zonations based on temperature and rainfall, or the length of the growing season. It follows that there are likely to be big differences between regions in the epidemiology of the virus diseases of cassava and in the prospects for control. This proposition has received totally inadequate attention, but it is considered here with reference to ACMD and to the even more limited studies on CBSD. No relevant information has been obtained on any of the other virus diseases of cassava in Africa.

Cassava Brown Streak Disease. In early studies on CBSD it was noted that stem and leaf symptoms were very severe during the cool winter months at upland sites in Tanzania, whereas the plants tended to recover during the hot season when there was little stem necrosis (Storey, 1936). These observations were extended by Nichols (1950), who observed marked varietal and seasonal effects on symptom expression. He also noted that CBSD was most prevalent in lowland areas up to *c.* 670 m above sea level, and suggested that infected plants were so severely affected that they failed to survive the cool season at higher altitudes. Evidence was presented of 'natural control' at low altitudes due to the tendency of farmers to reject and discard varieties that developed severe symptoms and grew poorly. Relatively tolerant

TABLE 2. Mean incidence of ACMD in twenty-six plantings of Brazilian, local and hybrid cultivars established between 1981 and 1984 at fourteen sites in coastal Kenya grouped according to rainfall (Bock, 1994b).

Mean annual rainfall	Number of plantings	Mean incidence (%) of ACMD				
		Brazilian Cultivars	Local 1	Local 2	Hybrid 1	Hybrid 2
800-1000 mm	6	1	0	0	0	0
1000-1200 mm	12	9	12	1	2	<1
>1200 mm	8	40	23	4	11	1

varieties that develop inconspicuous symptoms and crop satisfactorily are selected by farmers for use in areas where CBSD is endemic. Jennings (1960) presented additional information on the incidence of CBSD and symptom expression at different altitudes in Tanzania and showed that the disease can persist at upland sites and does not invariably die out as Nichols (1950) had suggested.

AFRICAN CASSAVA MOSAIC DISEASE

Malawi. Nyirenda et al. (1993) established ACMD-free plantings of several local varieties at several experiment stations. They reported little spread of ACMD within 12-14 months of planting at the three upland sites (> 1000 m altitude), compared with rapid spread at lower altitude sites along the western shore of Lake Malawi (773 m), where cassava was more widely grown.

Tanzania. Storey (1936) noted that the spread of ACMD was less in upland areas (> 1000 m a.s.l.) than at lower altitudes and that this was recognised by farmers in the lowlands who regularly obtained unaffected planting material from the uplands. In later studies, monthly plantings were made for 2 years at a lowland site (c. 180 m) where there were marked seasonal differences in rates of spread. All plantings made between December and April were almost totally infected within 3 months, whereas plantings at other times were much less severely affected, especially in June (Storey and Nichols, 1938a). The results were later analysed in relation to contemporaneous meteorological data for a nearby site and a positive correlation was established between rates of spread and mean monthly temperatures (Fargette et al., 1993; Fargette and Thresh, 1994a).

Madagascar. Cours-Darne (1968) concluded from earlier experience on the island 'that the hotter and more humid the region the more virulent the disease. Coastal regions with high rainfall are therefore the most seriously affected'. He also commented that 'virulence', in the sense of incidence of infection, diminished with increasing altitude so that at c. 1300 m it was possible to grow varieties that had been abandoned on the coast because of their vulnerability to ACMD. Such varieties could also be grown successfully in those lowland areas where there was a prolonged dry season.

Kenya. Bock (1994b) established many plantings of ACMD-free material of different varieties in coastal Kenya at various lowland sites north and south of Mombasa and also in western districts at mid-altitudes (c. 1100 m) near Lake Victoria. There was usually little or no spread at any of the sites, even though sources of infection were established within some of the trials and ACMD was prevalent in nearby farmers' fields due to the widespread use of infected cuttings for new plantings. The varieties used for the first experiments were somewhat resistant to infection and greater spread occurred in later trials with susceptible Brazilian varieties, especially in coastal areas where the mean annual rainfall exceeded 1200 mm (Table 2). However, these are also the areas of Kenya where cassava is most widely grown and it is unclear whether the increased spread was due to climatic effects on crop growth and vector populations, or to the increased intensity of cassava production and consequent decrease in mean separation between plantings.

Uganda. Many variety trials and propagation plots have been established since 1990 at a wide

range of sites in different parts of Uganda (Otim-Nape, 1993; Otim-Nape *et al.*, 1994). Little or no spread of ACMD occurred to any of the varieties grown in southern areas near the northern shore of Lake Victoria, where whitefly vector population densities and incidence of ACMD were usually low. By contrast, whiteflies were more numerous and spread was usually rapid in eastern, central and northern areas where ACMD is prevalent. At some sites susceptible varieties were almost totally infected within 6 months of planting. This occurred even though many of the sites in both high and low spread areas were at similar altitudes of 1100-1300 m.

The big differences in rates of spread between the two regions of Uganda were at first associated with the generally better soils, somewhat higher rainfall, cooler temperatures and longer growing season near Lake Victoria, compared with areas to the north (Otim-Nape, 1993). However, it is now known that the area in which spread is rapid and ACMD has become prevalent is extending southwards towards Lake Victoria along a broad front that extends across much of Uganda and over-rides differences in soil type, altitude, climate and agro-ecology (Thresh *et al.*, 1994b). This suggests an invasion by new strains of vector and/or virus and these possibilities are now being investigated.

Ivory Coast. Many different epidemiological trials were carried out by ORSTOM staff in the 1980s, mainly using a standard local variety 'CB' and sites in the southern lowland tropical rainforest zone near Abidjan, where temperature and soil moisture conditions are generally favourable for crop growth throughout much of the year. African cassava mosaic disease usually spread rapidly, especially where infected plantings occurred near and up-wind of the experimental site (Fauquet *et al.*, 1988). Whitefly population densities and rates of virus spread as influenced by planting date and seasonal effects were studied in a series of monthly plantings between May 1981 and May 1986 (Fargette and Thresh 1994b; Fargette *et al.*, 1994a). Positive relationships were established between mean monthly temperatures and crop growth, populations of adult whiteflies and virus spread. Effects of rainfall and soil moisture on

whitefly populations and virus spread were relatively unimportant in the generally humid conditions prevailing at the trial site. Less spread occurred at two inland savanna locations, especially at the Toumodi site which was isolated from other cassava and used to raise ACMD - free planting material for further epidemiology experiments (Fauquet *et al.*, 1988).

In all trials, there were marked effects associated with the direction of the prevailing wind. Vector populations and spread of ACMD were greatest at the margins of plantings and especially on plants at the windward and leeward edges (Fargette *et al.*, 1985). There were marked environmental gradients, and effects of wind turbulence were also evident from the increased incidence of ACMD in one trial alongside a foot-path that caused a break in the canopy of foliage (Fargette *et al.*, 1993).

Nigeria. Much of the available information is from plantings at the International Institute of Tropical Agriculture (IITA), Ibadan at a lowland site at the transition between the forest and Guinea savanna zones. There was rapid spread of ACMD to susceptible varieties in the few experiments in which a distinction was made between disease incidence and severity (e.g. Anonymous, 1980; Hahn *et al.*, 1980). Rapid spread also occurred at Ibadan in monthly plantings of seedling progenies, especially those made in April, May and June (Leuschner, 1977). There were marked seasonal effects on whitefly populations and a general relationship was established between numbers of whiteflies caught in traps and rates of disease spread. In later plantings of seedling progenies from South America there was less spread at a lowland forest site to the south near Onne and even less at a cooler upland plateau site near Jos and also near Kano in the north, where conditions are much hotter and drier (Porto *et al.*, 1994)

Togo. Dengel (1978a, b) reported rapid spread of ACMD to disease-free plantings in lowland coastal areas. A very susceptible variety was almost totally infected within 7 months when planted alongside ACMD-affected material, whereas the incidence was 38% in a more resistant variety. At an inland site 15-25% infection occurred within 3

months, even though the plots were at least 800 m from the nearest cassava.

Overall. The disparate series of experiments on ACMD summarised here were done at various times, using different varieties and procedures and in only some of the many countries and agro-ecological environments in which cassava is grown. The results obtained provide very inadequate data on which to base valid generalisations on the various factors influencing populations of *B. tabaci* on cassava and the spread and control of ACMD. Nevertheless, there is considerable evidence from Ivory Coast (Fargette *et al.*, 1990) and Uganda (Otim-Nape, 1993) that spread of ACMD by whiteflies is mainly by infective adults moving *between* and not *within* plantings. This explains the limited effects of roguing or the health status of the planting material on subsequent rates of spread by whiteflies *within* the plantings studied.

It is also clear that spread is usually rapid in humid lowland areas where temperature and soil moisture conditions are favourable for crop growth and whitefly reproduction throughout much of the year, as in parts of Madagascar, Nigeria, Ivory Coast and coastal Tanzania. This is consistent with the generally high incidence of ACMD in these areas, as reported in the previous sections. Spread tends to be less rapid where there is a prolonged dry season, as in coastal Kenya and at mid-altitudes (1000-1,500 m) where temperatures are relatively cool, as in parts of Kenya, Malawi and Tanzania.

Uganda provides a marked exception to these generalisations because very rapid spread is now occurring at mid-altitudes of *c.* 1200 m where crop growth is restricted by one and sometimes two dry periods each year. This emphasises the importance of the results obtained by Legg (1994) and the need for additional studies in Uganda and elsewhere in order to gain a fuller understanding of the factors influencing vector populations and the spread of ACMD. Meanwhile, from the evidence already available, it is apparent that particular attention should be given to cropping intensity and the abiotic environmental factors of wind speed and direction, rainfall and temperature. It is also clear that the effects of these factors on the three components of the pathosystem (virus/host/vector) are complex, and that there are likely to be important interactions between them.

Cropping intensity. The extent to which cassava is grown and the disposition of plantings within a locality influence the mean separation between plantings and the opportunity for ACMV to spread between them. This is evident from experience in Ivory Coast (Fauquet *et al.*, 1988) and Kenya (Bock, 1994b) and is consistent with the generally rapid spread reported in areas where cassava is widely grown and forms an important feature of the agro-ecology. More recently there is evidence from Uganda that the reduction in the area of cassava grown which occurred as a consequence of the current severe epidemic has led to some relaxation of the inoculum pressure in the worst-affected areas. This facilitates successful replanting, albeit temporarily until the cropping intensity increases to such an extent that there is a resurgence of ACMD. Hence the importance of the attempts now being made to build up adequate stocks of resistant varieties for release to farmers (Otim-Nape *et al.*, 1994). The extent to which such varieties are grown and the intensity of cassava production in the area can be expected to influence the overall incidence of infection. Indeed, recent modelling studies suggest that a possible long-term outcome will be cyclical changes in the use of resistant varieties in response to the changes in inoculum pressure and/or the amount of cassava grown (Holt *et al.*, 1995).

Rainfall. The effects of rainfall are complex, because it influences soil moisture, atmospheric humidity and crop growth. These in turn influence the susceptibility and response of cassava to infection with ACMV and as a host of *B. tabaci.* This explains why seasonal changes in cassava growth, whitefly populations and spread of ACMV are related to the distribution of rainfall in areas such as coastal Kenya and central Uganda where there are prolonged periods of little or no rain (Robertson, 1987; Legg, 1994). No such effects were evident at sites in Tanzania or Ivory Coast where lack of soil moisture was not an important factor limiting crop growth (Fargette, 1994a, b; Fargette and Thresh, 1994). However, in all areas

TABLE 3. Possible first order interactions (+) between the main diseases, pests and weeds of cassava

Pest/ disease	1	2	3	4	5	6	7	8	9	10	11
1. ACMVD	•	++	++	+	+	++	++	++	+	+	++
2. CBSVD	•	•	+	+	+	+	+	+	+	+	+
3. CBBD	•	•	•	+	+	+	+	+	+	+	+
4. Fungi	•	•	•	•	+	+	+	+	+	+	+
5. Nematodes	•	•	•	•	•	+	+	+	+	+	+
6. CGM	•	•	•	•	•	•	+	+	+	+	+
7. B. tabaci	•	•	•	•	•	•	•	+	+	+	+
8. CMB	•	•	•	•	•	•	•	•	+	+	+
9. Zonocerus	•	•	•	•	•	•	•	•	•	+	+
10. Vertebrates	•	•	•	•	•	•	•	•	•	•	+
11. Weeds	•	•	•	•	•	•	•	•	•	•	•

ACMVD	African cassava mosaic virus disease
CBSVD	Cassava brown streak virus disease
CBBD	Cassava bacterial blight disease
B. tabaci	Bemisia tabaci
CGM	Cassava green mite: Mononychellus tanajoa
Zonocerus	Variegated grasshopper: Zonocerus variegatus
CMB	Cassava mealybug: Phenacoccus manihoti
++	The main interactions discussed in the text.

whitefly populations are likely to be influenced by periods of heavy rain which cause direct mortality of adults (Legg,1994).

Temperature. The effects of temperature and the closely related parameter of solar radiation are also complex, because of their direct and indirect influences on host, virus and vector. For example, temperature is an important determinant of shoot growth and leaf production. This influences the availability of young tissue that is vulnerable to infection by vectors, or that can serve as a source of inoculum from which vectors acquire virus that can be transmitted to uninfected plants. There are also direct effects of temperature on vector populations and on the mobility and flight activity of adults, as described by Leuschner (1977) and Legg (1994).

The effects of temperature on the transmission of ACMV by vectors, and on the virus content of infected cassava have not been assessed, although they could be important in explaining seasonal differences in rates of virus spread (Fargette et al., 1994a), symptom expression (Gibson, 1994) and the extent of 'reversion'. This is the important but poorly understood phenomenon whereby uninfected cuttings are obtained from ACMV-infected plants and occurs because the virus is incompletely systemic, even within plants raised from infected cuttings (Storey and Nichols, 1938b; Fargette et al., 1994c). Reversion is a particularly important feature of ACMV-resistant varieties and explains why stocks of such varieties are not totally infected, even when grown for repeated cycles of crop production in areas of high inoculum pressure and where phyto-sanitation is not practised (Fargette et al., 1994c; Fargette and Thresh, 1994b). Temperature and other seasonal factors that influence crop growth, virus content and symptom expression are likely to influence the extent of reversion and the incidence of infection at the equilibrium values attained. However, this has not been demonstrated or modelled and is an important topic for further study.

INTERACTIONS BETWEEN ACMD AND OTHER DISEASES, PESTS AND WEEDS OF CASSAVA

African cassava mosaic disease is so widespread and can have such detrimental effects on the growth and yield of cassava that the disease is likely to influence the response of the crop to other biotic and abiotic stresses. Possible interactions of this type are but some of the many that may occur between the various pests, diseases and weeds of cassava. Table 3 sets out the 55 first-

order interactions between the main virus and other diseases, arthropod pests, nematodes and 'weeds' (which are treated here as a single group of species). Some of the the the main interactions involving ACMD are discussed in the following sections and it is suggested that such effects are likely to be far more important than the present limited evidence suggests.

ACMD and CBSD. These two virus diseases are so prevalent in lowland areas of Malawi and Tanzania that they often occur together and dual infection has also been seen in Kenya (Bock, 1994a) and Uganda (G.W. Otim-Nape and J.M. Thresh, unpublished). This could influence the concentration and effects of the viruses involved and the ease with which they are acquired and transmitted by vectors. These possibilities have not been investigated, although Harrison *et al.* (1995) suggest that the two diseases may have a mutually synergistic effect. If this is substantiated it would provide further justification for the efforts being made to achieve their control.

ACMD and bacterial blight caused by *Xanthomonas campestris* pv. *manihotis*. These diseases are so prevalent in many cassava growing areas of Africa that they frequently occur together and there is the possibility that each disease influences the behaviour and effects of the other. For example, the effects of bacterial blight in causing premature leaf abscission promotes the growth of axillary shoots which are susceptible to ACMD and which frequently show symptoms of mosaic that are more conspicuous than those on undamaged shoots. This effect may explain the apparent association between resistance to bacterial blight and to ACMD (Jennings, 1978; Hahn *et al.*, 1990).

ACMD and the whitefly vector (*Bemisia tabaci*). Considerable attention has been given to the role of *B. tabaci* as the vector of the viruses causing ACMD and recent reviews are available on the mode of transmission (Dubern, 1994) and vector ecology (Fishpool and Burban, 1994; Legg, 1994). However, little attention has been given to the possibility that ACMD influences cassava as a host of *B. tabaci* and on the sequence of colonisation and population development on the

crop. Such effects might be expected from the influence of ACMD on rates of leaf and stem production, leaf area, leaf structure and leaf composition. Recent observations in Uganda (R.W. Gibson and S. Ogwal, unpublished) suggest that any such effects are not great as similar numbers of adult whiteflies were recorded on leaves with symptoms of ACMD and those without. However, the immature whitefly 'scales' occurred at greater density in the green areas of mosaic-affected leaves than in the yellow areas.

ACMD and cassava green mite *Mononychellus tanajoa* (Bondar). The cassava green mite (CGM) was first reported in Africa in 1972 and it has since become prevalent in many cassava growing areas (Yaninek, 1994). Infestations cause damage to the leaves which can lead to drastic reductions in leaf area, number and longevity. The most severely affected plants have small malformed leaves or become virtually leafless, and crop growth and yields are seriously affected. This can make it difficult to diagnose the symptoms of ACMD and other virus diseases of cassava, as evident from experience in several countries. Extension and research staff, and also farmers, sometimes attribute the severe effects of CGM to ACMD and this can cause serious difficulties in attempts to introduce roguing as a virus disease control measure (Otim-Nape *et al.*,1994). However, the mistake is readily avoided because the damage caused by CGM is usually similar on the different leaflets of each individual leaf and on either side of the main vein, whereas the symptoms of ACMD are more variable and usually differ on either side of the main vein.

The deleterious effects of CGM are likely to have an important impact on the biology of other pests and pathogens of cassava including ACMD and the whitefly vector (*B. tabaci*). This is apparent from the inverse relationship established between populations of adult *B. tabaci* and the severity of the damage to leaves caused by CGM (Legg, 1994). It is also likely that the effect of CGM on the overall condition and growth rate of the shoot tips will influence their susceptibility to infection with ACMV by whiteflies, and their suitability and potency as sources of inoculum from which ACMV can be acquired and transmitted to uninfected plants. Moreover, ACMD and CGM

are likely to interact in their effects on growth and yield and their combined effects may be greater than either alone. This possibility has not been considered and merits attention because the control of ACMD through the use of virus-free or virus-tolerant planting material could decrease the damage caused by CGM and improve the ability of plants to withstand mite infestation and recover from even the most severe attack.

ACMD and the cassava mealybug *Phenacoccus manihoti* Mat.-Fer. Much of the discussion on ACMD and CGM is relevant also to ACMD and the cassava mealybug (CMB). This is another neotropical pest species that was introduced to Africa in the 1970s and now occurs in all the main cassava growing areas of the continent, except Madagascar (Neuenschwander, 1994). Cassava mealybug can cause very serious damage and severe infestations led to almost total crop failure in some countries until an effective parasitoid (*Epidinocarsis lopezi* De Santis) was introduced and became widely established to achieve a generally good degree of biological control (Neuenschwander, 1994).

Cassava growth, populations of CMB and its natural enemies and the effectiveness of the biological control achieved have been assessed in comprehensive surveys carried out in several countries of western and southern Africa (Neuenschwander *et al.*, 1989, 1990, 1991; Chakupurakal *et al.*, 1994). The effects of various cropping practices and a wide range of environmental conditions were considered in these studies, but not the role of ACMD. In Zambia this was because fields that were heavily damaged by ACMD or other diseases were avoided in the survey and there was also uncertainty over disease identification (Chakupurakal *et al.*, 1994). A different limitation was encountered in West Africa, where there were insufficient uninfected plants for valid comparisons to be made with those affected by ACMD (P. Neuenschwander, personal communication).

It may be significant that some of the worst damage caused by CMB in Uganda has been recorded at a site where the variety being grown was very sensitive to infection with ACMD and all plants were affected by the disease and showed conspicuous mosaic symptoms (G. W. Otim-

Nape, unpublished). However, ACMV-free plants were not available for comparison and there is obvious scope for additional studies on possible interactions between ACMD and CMB in different agro-ecological conditions. This is because ACMD is such an important determinant of plant growth that it is likely to influence not only populations of CMB and its natural enemies, but also the survival of the cuttings used to establish new plantings and their ability to withstand the effects of mealybug infestation. An important consequence of such an effect would be that the use of ACMV-free or ACMV-tolerant planting material would decrease the damage caused by CMB as well as that caused by ACMD, and possibly also CGM, as discussed in the previous section.

ACMD and Weeds. Weeds are important in cassava production and can severely decrease yields unless controlled effectively, especially during the early stages of crop growth when plants are most vulnerable to weed competition (Melifonwu, 1994). The ease with which weeds are controlled is greatly influenced by the rapidity with which cassava becomes established and forms a continuous canopy of foliage that suppresses weeds. This suggests that the detrimental effects of ACMD in decreasing the proportion of cuttings that establish and on subsequent rates of growth and canopy formation will facilitate weed growth and necessitate additional weed control measures if severe competition is to be avoided. Moreover, the effect is likely to be greater with erect varieties, and especially those sensitive to infection with ACMV, than with spreading varieties and those tolerant of infection. These possibilities seem not to have been investigated and yet the evidence obtained could be important in influencing the choice of variety, cultural practices and the use of ACMV-free planting material.

Some weed species and other components of the natural vegetation are known to be hosts of ACMV, which is assumed to have originated in indigenous species (p. 461). However, there is no evidence that weeds are of continuing epidemiological importance as sources of infection from which spread occurs to cassava (Fargette and Thresh, 1994a). Weeds are also considered to be unimportant as hosts of *B. tabaci* populations

that infest cassava, as the populations occurring on weeds seem to be of a distinct race or biotype that seems unable to infest cassava (Legg, 1994).

DISCUSSION

From this assessment, it is apparent that few of the viruses of cassava in Africa have been studied adequately. Several have been isolated quite fortuitously, they have not been well characterised, and there are serious deficiencies in the information available on their distribution, effects on yield and mode of spread. This unsatisfactory situation reflects the inadequate attention given to viruses of cassava, especially in Africa where there is a particular dearth of trained virologists and facilities (Thresh, 1991). However, the situation is little different in other regions and cassava has received far less attention than comparable staple food crops such as *Solanum* potato. This is apparent from the CD-ROM data base which lists a total of 1984 references on viruses of potato published in the 10 years to 1995, compared with only 162 on cassava. Comparable figures for cassava and potato in the previous 10 year period were 157 and 2812, respectively (personal communication, J. Brunt, CAB International). An important difference between the two crops is that cassava is grown almost entirely in the tropics and mainly in Africa, whereas potato is widely grown in the developed countries where much of the research on viruses is done.

It seems inevitable that further studies will show viruses of cassava to be more numerous, more prevalent and even more important than the present limited evidence suggests. Meanwhile, the available results indicate that, with the exception of cassava common mosaic potexvirus, each of the viruses reported from cassava has a distribution that is largely or entirely confined to one continent and perhaps to only certain countries or regions. This emphasises the importance of quarantine measures in regulating the movement of plant material between and also within continents. It is particularly important to prevent the introduction of the two African cassava mosaic geminiviruses to Asia or South/Central America. There is also a need to maintain the present limited distribution of cassava brown streak virus(es) and of the two African cassava mosaic geminiviruses within Africa.

The importance of quarantine and other restrictions on the movement of cassava is well recognised (Lozano, 1978; Calvert, 1994) and guidelines are available on the most appropriate procedures to adopt (Frison and Feliu, 1991). Much use has been made by IITA of *in vitro* cultures to transfer genotypes from and within Africa (Ng, 1983) and 'third country' facilities in Scotland have been used to provide additional safeguards in transferring cassava clones from Africa to South America (Lennon *et al.*, 1986). Moreover, seeds rather than vegatative propagules are used wherever possible to decrease the risks involved, although it is appreciated that some viruses of cassava may be seed-borne (Lennon *et al.*, 1987; Porto and Asiedu, 1992). It is important to maintain and improve current quarantine procedures by revising the techniques used as new viruses are discovered and more sensitive methods of detection become available.

The selection of symptomless plants together with the use of whatever virus detection techniques are available, and the possible use of heat and/or meristem-tip therapy (Kaiser and Teemba, 1979; Frison, 1994), can be used to improve the health status of the vegetative planting material of cassava that is available to farmers. This is a basic approach to virus disease control that is widely adopted with many other vegetatively-propagated crops to improve productivity and profitability. However, it is one that has seldom been adopted with cassava in Africa, where the use of simple selection procedures has received only limited attention (Terry and Hahn, 1982; Porto and Asiedu, 1993). Consequently, many of the stocks being grown by farmers are seriously affected by ACMD and other viruses may also be present, including the one(s) causing CBSD. There is a similar unsatisfactory situation with many of the stocks being propagated officially in many African countries for field experiments or for release to farmers.

The yield penalty incurred by the failure to adopt virus-free cassava planting material is likely to be substantial but cannot be estimated because of the limited information available on the incidence of the different viruses and on their

effects on the growth and yield of even the most widely grown varieties (Thresh *et al.*, 1994a). There is also inadequate information on the long-term performance of virus-free stocks in different agro-ecological conditions and on the rapidity with which they are infected with ACMV and other viruses. Nevertheless, it is clear that viruses are being introduced inadvertently in planting material to areas where they do not occur, or where they are not prevalent and are not spread rapidly by vectors. Viruses introduced from the outset in cuttings have the most deleterious effects on growth and yield and have ample opportunity to spread to other plants. This suggests that there are likely to be substantial benefits from adopting virus-free stocks, but the benefits will be difficult to achieve because the present unsatisfactory attitudes and practices are so well entrenched.

There is a tendency in at least some countries for farmers and even extensionists and researchers to accept ACMD as a largely inevitable and unavoidable problem. Moreover, CBSD and other virus diseases tend to be unrecognised or completely ignored because they cause inconspicuous or ephemeral symptoms that may disappear as the plants age. These attitudes are to some extent justified because there is considerable variation between varieties in their suceptibility or response to virus-infection, and farmers and those concerned with crop improvement tend to adopt varieties that perform satisfactorily despite the effects of any viruses present. The factors influencing the adoption and rejection of varieties by farmers and the high rates of turnover reported in recent surveys are complex (Otim-Nape *et al.*, 1994). It is unclear if the response of farmers is conscious or intuitive and whether resistant varieties are sought or vulnerable ones that are found to be unsatisfactory are simply discarded.

Whatever the practice adopted, the outcome is likely to be similar in achieving a switch to ACMD-resistant varieties in areas where the overall infection pressure is high. Conversely, ACMD-resistance becomes unimportant where the infection pressure is low and the farmers' choice of variety is then largely determined by other criteria. In some circumstances this can be expected to lead to cyclical changes in the prevalence of the varieties grown, as the use of resistant varieties has the effect of decreasing the overall inoculum pressure and the incidence of infection. Less resistant varieties may then become increasingly important until there is a resurgence of disease (Thresh *et al.*, 1994b). There is certainly evidence of a rapid turn-over of varieties from the COSCA and other surveys of farmers' attitudes to varieties and the main attributes sought (Nweke, 1994; Otim-Nape *et al.*, 1994). The turn-over in varieties is facilitated by the frequent appearance within plantings of self-sown seedlings, some of which are propagated and grown by farmers and may eventually be widely adopted as varieties. This practice is common and has the effect of releasing the considerable genetic diversity that is present within the genome of a polyploid crop such as cassava (Jennings, 1994, 1995).

The concept of a dynamic continually changing situation as host populations respond to fluctuations in infection pressure influenced by biotic and abiotic factors is discussed by Buddenhagen (1977). It is consistent with the history of ACMD in Madagascar in the 1930s and 1940s and more recently in Uganda. African cassava mosaic disease was first reported in Madagascar in 1930 and until 1935 it was considered unimportant. A serious epidemic then occurred and, within two years, the widely grown local varieties referred to as *'manihoc du pays'* were almost completely destroyed and production was greatly decreased. The whole future of the crop on the island was in doubt until resistant varieties were introduced from elsewhere and highly resistant ones were developed from crosses between cassava and *Manihot glaziovii*. Such varieties were widely adopted and production was fully restored within 10-15 years (Cours-Darne, 1968).

There is currently a crisis situation in Uganda similar to the earlier one in Madagascar, as a very severe epidemic of ACMD is extending southwards across the country and the most vulnerable local varieties are being eliminated. Cassava production has almost ceased for a time in areas where such varieties were virtually the only ones being grown and, there have been serious food shortages and many famine-related deaths. Between 1989 and 1993 various attempts were made to supply these areas with planting material obtained from less severely affected areas. However, the varieties deployed in this way were

not sufficiently resistant to infection and the only long-term solution is to switch to more resistant types. Suitable ones are now being selected and made available in quantity (Otim-Nape *et al.*, 1994; Thresh *et al.*, 1994b).

These experiences, and those of Nichols (1950) with CBSD and Nolt *et al.*, (1992) with Caribbean mosaic disease in Colombia, emphasise the crucial importance of the varieties being grown in determining the losses caused by viruses of cassava. The use of resistant varieties is likely to remain the key element of virus disease management strategies, but there is an urgent need to clarify the role of phytosanitation in the control of ACMD (Thresh and Otim-Nape, 1994). One argument is that the use of ACMD-free planting material, selection and roguing are unnecessary if the varieties grown are sufficiently resistant to infection. The alternative view is that such measures complement the use of resistant varieties and render these even more effective. The paradox is that under conditions of high inoculum pressure phytosanitation is only feasible when resistant varieties are adopted and yet phytosanitation may be unnecessary if such varieties are used. However, the issue is likely to be even more complicated because there are considerable differences in the resistance to ACMD of the improved varieties originating from IITA that are now available and being promoted in Uganda and elsewhere in Africa. The productivity of at least some of these varieties is likely to benefit from phytosanitation, as demonstrated recently by Ogbe *et al.* (1995), but whether the benefits are sufficient to justify the considerable effort required to achieve them remains uncertain.

Further research is needed on this topic and on the most appropriate means of deploying virus-resistant varieties. These are but some of the many aspects of research on the virus and virus diseases of cassava that require attention. There is also an obvious need for studies that are more comprehensive and involve a wider range of countries and agro-ecological conditions than any yet undertaken. Moreover, the studies should be sustained for a suitably long period and be closely associated with technology-transfer projects to ensure that the research findings are utilised more effectively than previously (Otim-Nape *et al.*,

1994). Another requirement is that any further studies should be mounted in close association with other crop protection personnel and also with breeders, agronomists and socio-economists to achieve an integrated multi-disciplinary approach to crop protection and production.

The time is appropriate for new multi-disciplinary initiatives of this type, as indicated by the many other contributors to this volume who also stress the need for an holistic, ecological approach. There is undoubtedly great scope for rapid progress and for big increases in productivity through the control of virus diseases. However, these benefits will not be easy to achieve without a greatly increased commitment of funds and resources to sustain, strengthen and enhance the current limited, inadequately-funded, desultory and fragmented effort. The success of the multi-million dollar Africa-wide Biocontrol Project against cassava mealybug (Neuenschwander, 1994) and the great potential of the recently initiated ESCaPP Project in Ghana, Benin, Nigeria and Cameroon (Yaninek *et al.*, 1994) indicate possible approaches. A comparable and equally ambitious international effort is required on the viruses and virus diseases of cassava if substantial progress is to be made and virologists are to make a full contribution to improvement of the crop.

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REFERENCES

Aiton, M.M., Lennon A.M., Roberts I.M. and Harrison, B.D. 1988a. Two new cassava viruses from Africa. p. 43. *Abstracts: International Congress of Plant Pathology*, Kyoto, Japan.

Aiton, M.M., Roberts, I.M. and Harrison, B.D. 1988b. Cassava common mosaic potexvirus from mosaic-affected cassava in the Ivory Coast. *Report of the Scottish Crop Research Institute for 1987*, p. 191.

Anonymous, 1980. Cassava Pathology: Cassava mosaic disease (CMD). *Annual Report for*

1979. Root and Tuber Improvement Program. International Institute of Tropical Agriculture, Ibadan. pp. 58-61.

Bock, K.R. 1975. Maize streak, cassava latent and other similar viruses. In: *Abstracts of the Third Congress of Virology*, Madrid, p. 93.

Bock, K.R. 1983. Epidemiology of cassava mosaic disease in Kenya. In: *Plant Virus Epidemiology*. Plumb, R.T. and Thresh, J.M. (Eds.), pp.337-347. Blackwell Scientific Publications, Oxford.

Bock, K.R. 1994a. Studies on cassava brown streak virus disease in Kenya. *Tropical Science* 34:134-145.

Bock, K.R. 1994b. The spread of African cassava mosaic geminivirus in coastal and western Kenya. *Tropical Science* 34:92-101.

Bock, K.R. and Harrison, B.D. 1985. African cassava mosaic virus. *AAB. Descriptions of Plant Viruses*: No. 297, 6 pp.

Bock, K.R. and Woods, R.D. 1983. Etiology of African cassava mosaic disease. *Plant Disease* 67:994-995.

Bock, K.R., Guthrie, E.J. and Meredith, G. 1978. Distribution, host range, properties and purification of cassava latent virus, a geminivirus. *Annals of Applied Biology* 90: 361-367.

Brunt, A.A. 1990. Cassava brown streak 'carlavirus'. Cassava brown streak potyvirus. In: *Viruses of Tropical Plants*. Brunt, A., Crabtree, K. and Gibbs, A. (Eds.), pp. 157-158. CAB International, Wallingford, UK.

Buddenhagen, I.W. 1977. Resistance and vulnerability of tropical crops in relation to their evolution and breeding. *Annals of the New York Academy of Sciences* 278: 309-326.

Calvert, L.A. 1994. The safe movement of cassava germplasm. In: *Report of the First Meeting of the International Network for Cassava Genetic Resources*. International Crop Network Series, pp. 163-165. International Plant Genetic Resource Institute, Rome, Italy.

Carter, S.E., Fresco, L.O., Jones, P.G. and Fairbairn, J.N. 1992. *An Atlas of Cassava in Africa: Historical, Agroecological and Demographic Aspects of Crop Distribution*. Centro Internacional de Agricultura Tropical, Cali, Colombia.

Chakupurakal, J., Markham, R.H., Neuenschwander, P., Sakala, M., Malombo, C., Mulwanda, D., Banda, A., Chalabero, A., Bird, T. and Haug, T. 1994. Biological control of the cassava mealybug, *Phenacoccus manihoti* (Homoptera:Pseudococcidae) in Zambia. *Biological Control* 4: 254-262.

Costa, A.S. and Kitajima, E.W. 1972. Cassava common mosaic virus. *CMI/AAB Descriptions of Plant Viruses* No. 90. Commonwealth Agricultural Bureaux, Slough, U.K. 4 pp.

Cours-Darne, G. 1968. Improving cassava in Africa. The Abidjan Conference. *Agricultural Research Priorities for Economic Development in Africa* 2:330-339.

Dahniya, M.T. 1994. Cassava in Africa. *African Crop Science Journal* 2:337-343.

Dengel, H-J. 1978a. Untersuchungen über das Maniokmosaik in Süden Togos. Inaugural Dissertation zur Enlargung des Doktorgrades in Fachbereich Angewandte Biologie und Umweltsicherung der Justus-Liebig-Universität. Giessen, Germany. 142 pp.

Dengel, H-J. 1978b. Methoden und Vorschläge für die Bekämpfung der Maniokmosaikkrankheit in Südost-Togo. In: *Dynamik der Landnutzung in den Wechselfeuchten Tropen*. Grenzebached, K. (Ed.). *Gieener Beiträge zur Entwicklungsforschung* 1 (4): 97-106.

Dorosch, P. 1988. *The Economics of Root and Tuber Crops in Africa*. RCMP Research Monograph No. 1. Ibadan, International Institute of Tropical Agriculture.

Dubern, J. 1994. Transmission of African cassava mosaic geminivirus by the whitefly (*Bemisia tabaci*). *Tropical Science* 34:82-91.

FAO, 1994. Production Year Book: 1993; 47. *FAO Statistical Series No. 117.*

Fargette, D. and Thresh, J.M. 1994a. The ecology of African cassava mosaic geminivirus. In: *Ecology of Plant Pathogens*. Blakeman, J.A. and Williamson, B. (Eds.), pp. 269-282. CABI International Walllingford.

Fargette, D. and Thresh, J.M. 1994b. Analysis and modelling of the temporal spread of African cassava mosaic virus and implications for disease control. *African Crop Science Journal* 2:449-458.

Fargette, D., Fauquet, C. and Thouvenel, J.- C. 1985. Field studies on the spread of African cassava mosaic. *Annals of Applied Biology* 106:285-294.

Fargette, D., Fauquet, C., Grenier, E. and Thresh, J.M. 1990. The spread of African cassava mosaic virus into and within cassava fields. *Journal of Phytopathology* 130:289-302.

Fargette, D., Jeger, M., Fauquet, C. and Fishpool, L. D.C. 1994a. Analysis of temporal disease progress of African cassava mosaic virus. *Phytopathology* 84:91-98.

Fargette, D., Muniyappa, V., Fauquet, C., N'Guessan, P. and Thouvenel, J.- C. 1993. Comparative epidemiology of three tropical whitefly-transmitted geminiviruses. *Biochemie* 75:547-554.

Fargette, D., Roberts, I.M. and Harrison, B.D. 1991. Particle purification and properties of cassava Ivorian bacilliform virus. *Annals of Applied Biology* 119:303-312.

Fargette, D., Thresh, J.M. and Otim-Nape, G.W. 1994b. The epidemiology of African cassava mosaic geminivirus: reversion and the concept of equilibrium. *Tropical Science* 34:123-133.

Fauquet, C., Fargette, D. and Thouvenel, J.- C. 1988. Some aspects of the epidemiology of African cassava mosaic virus in Ivory Coast. *Tropical Pest Management* 34:92-96.

Fishpool, L.D.C. and Burban, C. 1994. *Bemisia tabaci*: the whitefly vector of African cassava mosaic geminivirus. *Tropical Science* 34: 55-72.

Frison, E.A. 1994. Sanitation techniques for cassava. *Tropical Science* 34:146-153.

Frison, E.A. and Feliu, E. 1991. *FAO/IBPGR Technical Guidelines for the Safe Movement of Cassava Germplasm*. Food and Agriculture Organization of the United Nations and International Board for Plant Genetic Resources, Rome. 47 pp.

Geddes A.M.W. 1990. *The Relative Importance of Crop Pests in Sub-Saharan Africa*. Bulletin No. 36. Natural Resources Institute, Chatham UK.

Gibson, R.W. 1994. Long-term absence of symptoms in heat-treated African cassava mosaic geminivirus-infected resistant cassava plants. *Tropical Agriculture* 34: 154-158.

Hahn, S.K., Howland, A.K. and Terry, E.R. 1990.

Correlated resistance of cassava to mosaic and bacterial blight diseases. *Euphytica* 29: 305-311.

Hahn, S.K., Terry, E.R. and Leuschner, K. 1980. Breeding cassava for resistance to cassava mosaic disease. *Euphytica* 29:673-683.

Harrison, B.D., Lennon, A.M., Massalski, P.R., Robinson, D.J. and Thomas, J.E. (1987). Geographical variation in geminivirus isolates associated with cassava mosaic disease. *Annual Report 1986, Scottish Crop Research Institute*, pp. 179-180.

Harrison, B.D., Swanson, M.M., McGrath, P. and Fargette, D. 1991. Patterns of antigenic variation in whitefly-transmitted geminiviruses. *Annual Report 1990, Scottish Crop Research Institute*, pp.88-90.

Harrison, B.D., Swanson, M.M. and Robinson, D.J. 1995. Cassava viruses in the Old World. *Proceedings Second International Scientific Meeting of the Cassava Biotechnology Network*, Bogor, Indonesia, 22-26 August 1994 (In press).

Holt, J., Jeger, M.J. and Thresh, J.M. 1995. Theoretical model of African cassava mosaic virus dynamics to investigate possible virulence shifts in host-virus interactions. Abstract. *Sixth International Plant Virus Epidemiology Symposium*, Jerusalem, April 1995.

Hong, Y.G., Robinson, D.J. and Harrison, B.D. 1993. Nucleotide sequence evidence for the occurrence of three distinct whitefly transmitted geminiviruses in cassava. *Journal of General Virology* 74:2437-2443.

Jameson, J.D. 1964. Cassava mosaic disease in Uganda. *East African Agricultural and Forestry Journal* 29:208-213.

Jennings, D.L. 1960. Observations on virus diseases of cassava in resistant and susceptible varieties II. Brown streak disease. *Empire Journal of Experimental Agriculture* 28:261-270.

Jennings, D.L. 1978. Inheritance of linked resistance to African cassava mosaic and bacterial blight disease. In: *Proceedings: Cassava Protection Workshop*. Brekelbaum, T., Bellotti, A. and Lozano, J.C. (Eds.), pp. 45-50. CIAT, Cali, Colombia.

Jennings, D.L. 1994. Breeding for resistance to

African cassava mosaic geminivirus in East Africa. *Tropical Agriculture* 34: 110-122.

Jennings, D.L. 1995. Cassava *Manihot esculenta*: (Euphorbiaceae). In: *Evolution of Crop Plants*. Second Edition. Smartt and Simmonds, N.W. (Eds.) Longmans. (In press).

Johnson, A. 1992. Report: *Lake Chad Farmer Training and Agricultural Development Project:* American Organizations for Rehabilitation Through Training, US Agency for International Development.

Kaiser, W.J. and Teemba, L.R. 1979. Use of tissue culture and thermotheropy to free East African cassava cultivars of African cassava mosaic and cassava brown streak viruses. *Plant Disease Reporter* 63:780-784.

Kitajima, E.W. and Costa, A.S. 1964. Elongated particles found associated with cassava brown streak. *East African Agricultural and Forestry Journal* 29:28-30.

Legg, J.P. 1994. *Bemisia tabaci:* the whitefly vector of African cassava mosaic geminiviruses in Africa: an ecological perspective. *African Crop Science Journal* 2:437-448.

Lennon, A.M., Aiton, M.M. and Harrison, B.D. 1986. Cassava viruses from South America. Cassava viruses from Africa third country quarantine of cassava. *Scottish Crop Research Institute Annual Report for 1985*, pp. 167-169.

Lennon, A.M., Aiton, M.M. and Harrison, B.D. 1987. Purification and properties of cassava green mottle, a previously undescribed virus from the Solomon Islands. *Annals of Applied Biology* 110:545-555.

Leuschner, K. 1977. Whiteflies: biology and transmission of African mosaic disease. In *Cassava Protection Workshop.* Brekelbaum,T., Bellotti, A. and Lozano, J. (Eds.), pp. 51-58. CIAT, Colombia.

Lister, R.M. (1959). Mechanical transmission of cassava brown streak virus. *Nature (London)* 183:1588-1589.

Lozano, J.C. 1978. General considerations on cassava pathology. In: *Proceedings Cassava Protection Workshop.* Brekelbaum, T., Bellotti, A. and Lozano, J. (Eds.), pp. 17-27. CIAT Cali, Colombia.

Melifonwu, A. A. 1994. Weeds and their control in Africa. *African Crop Science Journal* 2: 519-530.

Munthali, D.C. 1992. Effect of cassava variety on the biology of *Bemisia afer* (Priesner and Hosny) (Hemiptera: Aleyrodidae). *Insect Science and its Application* 13:459-465.

Neuenschwander, P. 1994. Control of the cassava mealybug in Africa: lessons from a biological control project. *African Crop Science Journal* 2:369-383.

Neuenschwander, P., Borowka, R., Phiri, G., Hammans, H., Niyirenda, S., Kapeya, E.H. and Gadabu, A. 1991. Biological control of the cassava mealybug *Phenacoccus manihoti* (Hom., Pseudococcidae) by *Epidinocarsis lopezi* (Hom., Encyrtidae) in Malawi. *Biocontrol Science and Technology* 1:297-310.

Neuenschwander, P., Hammond, W.N.O., Gutierrez, A.P., Cudjoe, A.R., Adjakloer, and Baumgärtner, J.U. 1989. Impact assessment of the biological control of the cassava mealybug *Phenacoccus manihotis* Matile-Ferrero (Hemiptera: Pseudococcidae), by the introduced parasitoid *Epidinocarsis lopezi* (De Santis) Hymenoptera: Encyrtidae). *Bulletin of Entomological Research* 79:579-594.

Neuenschwander, P., Hammond, W.N.O., Ajuonu, O., Gado, A., Echendu, N., Bokonon-Ganta, A.H., Allomasso, R. and Okon, I. 1990. Biological control of the cassava mealybug *Phenacoccus manihoti* (Hom., Pseudococcidae) by *Epidinocarsis lopezi* (Hym., Encyrtidae) in West Africa, as influenced by climate and soil. *Agriculture, Ecosystems and Environment* 22:39-55.

Ng, S.Y. 1983. Tissue culture: Cassava. *Annual Report for 1982, Root and Tuber Improvement Program*, pp. 113-114. International Institute of Tropical Agriculture, Ibadan.

Nichols, R.F.W. 1950. The brown streak disease of cassava: distribution, climatic effects and diagnostic symptoms. *East African Agricultural Journal* 15:154-160.

Nolt, B.L., Pineda, B. and Velasco, A.C. 1992. Surveys of cassava plantations in Colombia for virus and virus-like diseases. *Plant Pathology* 41:348-354.

Nweke, F. I. 1994. Farm level practices relevant to cassava plant protection. *African Crop Science Journal* 2:563-581.

Nyirenda, G.K.C., Munthali, D.C., Phiri, G.S.N, Sauti, R.F.N. and Gerling, D. 1993. Integrated pest management of *Bemisia* spp. whiteflies in Malawi. *Report Makoka Research Station, Thordwe, Malawi.*

Ogbe, F.O., Nnodu, E.C. and Odurukwe, S.O. 1995. The control of African cassava mosaic disease incidence and severity. *Tropical Science.* (In press).

Otim-Nape, G.W. 1993. *The Epidemiology of the African Cassava Mosaic Geminivirus Disease in Uganda.* PhD. Thesis, University of Reading, U.K. 252 pp.

Otim-Nape, G. W., Bua, A. and Baguma, Y. 1994. Accelerating the transfer of improved production technologies: Controlling African cassava mosaic virus disease epidemics in Uganda. *African Crop Science Journal* 2:479-495.

Porto, M.C.M. and Asiedu, R. 1992. IITA-CIAT initiative for the safe movement of germplasm. *Cassava Newsletter* 16:2-4.

Porto, M.C.M. and Asiedu, R. 1993. Production hints: selection and preparation of cassava planting materials. *Tropical Root and Tuber Crops Bulletin* 7:3-4.

Porto, M.C.M., Asiedu, R., Dixon, A. and Hahn, S.K. 1994. An agro-ecologically-oriented introduction of cassava germplasm from Latin America into Africa. In: *Tropical Root Crops in a Developing Economy.* Ofori, F. and Hahn, S.K. (Eds.), pp. 118-129. Proceedings 9th Symposium International Society for Tropical Root Crops, 20-26 October 1991, Accra, Ghana.

Robertson, I.A.D. 1987. The whitefly *Bemisia tabaci* (Gennadius) as a vector of African cassava mosiac virus at the Kenya coast and ways in which the yield losses in cassava *Manihot esculenta* Crantz caused by the virus can be reduced. *Insect Science and its Application* 8:797-801.

Silberschmid, K. L. 1938. O mosaico a mandioca. *O Biologico* 4:177-182.

Storey, H.H. 1936. Virus diseases of East African plants: VI. A progress report on studies of the disease of cassava. *East African Agricultural Journal* 2:34-39.

Storey, H.H. 1939. Report of the Plant Pathologist. *Report of the East African Agricultural Research Station for 1939,* p.9.

Storey, H.H. and Nichols, R.F.W. 1938a. Virus diseases of East African Plants VII. A field experiment in the transmission of cassava mosaic. *East African Agricultural Journal* 3: 446-449.

Storey, H.H. and Nichols, R.F.W. 1938b. Studies of the mosaic diseases of cassava. *Annals of Applied Biology* 25:790-806.

Swanson, M.M. and Harrison, B.D. 1994. Properties, relationships and distribution of cassava mosaic geminiviruses. *Tropical Science* 34:15-25.

Terry, E.R. and Hahn, S.K. 1982. Increasing and stabilizing cassava and sweet-potato productivity by disease resistance and crop hygiene. In: *Root Crops in Eastern Africa,* pp. 47-52. Proceedings Workshop Kigali, Rwanda 23-27 Nov. 1980. IDRC, Ottawa, Canada.

Thresh, J.M. 1991. The ecology of tropical plant viruses. *Plant Pathology* 40:324-339.

Thresh, J.M. and Otim-Nape, G.W. 1994. Strategies for controlling African cassava mosaic geminivirus. *Advances in Disease Vector Research* 10:215-236.

Thresh, J.M., Fargette, D. and Otim-Nape, G.W. 1994a. Effects of African cassava mosaic virus on the yield of cassava. *Tropical Science* 34:26-42.

Thresh, J.M., Otim-Nape, G.W. and Jennings, D.L. 1994b. Exploiting resistance to African cassava mosaic virus. *Aspects of Applied Biology* 39:51-60.

Walker, P.T., Heydon, D.L. and Guthrie, E.J. 1985. *Report of a Survey of Cassava Yield Losses in Ghana.* Tropical Development Research Institute Report CO/479, 9pp.

Yaninek, S. 1994. Cassava green mite intervention technologies. *African Crop Science Journal* 2:361-367.

Yaninek, J.S., James, B.D. and Bieler, P. 1994. Ecologically sustainable cassava plant protection (ESCaPP): a model for environmentally sound pest management *African Crop Science Journal* 2:553-562.