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The mosaic diseases of cassava in Africa and India caused by whitefly-borne geminiviruses

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Abstract

The main features of the mosaic virus diseases of cassava in sub-Saharan Africa and India are summarised in this review. The diseases in the two regions share important features and they are transmitted similarly by the whitefly *Bemisia tabaci*. Consequently, it is argued the diseases should be referred to collectively as cassava mosaic disease (CMD), although they are caused by different viruses of the family *Geminiviridae*. Information is presented on the prevalence, effects and epidemiology of cassava mosaic disease in Africa and India and on the substantial benefits to be gained from effective control by adopting CMD-resistant varieties and phytosanitation. The latter involves the use of healthy planting material and the removal (roguing) of any diseased plants that occur.

Introduction

Mosaic diseases of cassava (*Manihot esculenta*) have been reported from South and Central America, Africa and the Indian sub-continent. The diseases in Africa, India and Sri Lanka are caused by whitefly-borne geminiviruses (Family: *Geminiviridae*, Sub-family III). They are the subject of this paper and it is argued that they should be referred to collectively as cassava mosaic disease. The mosaic disease of cassava in South/Central America that is usually referred to as cassava common mosaic is caused by a completely different virus of the genus *Potexvirus* (Costa & Kitajima, 1972). It has no known vector and is not considered further here. An early report of a mosaic disease of cassava in Indonesia (Muller, 1931) has not been confirmed and the symptoms were later attributed to causes other than a virus (Bolhuis, 1949).

The aetiology of the cassava mosaic diseases in Africa and India

Africa

The disease in Africa originally referred to as cassava mosaic (CMD), or more recently as African cassava mosaic (ACMD), was first reported in 1894, in what is now Tanzania. It has since been recorded in all the main cassava growing areas of sub-Saharan Africa and on several islands including Madagascar, Mauritius, Bioko (Fernando Poo), São Tomé and Cape Verde.

Early studies in several of the then colonial territories showed that CMD was transmissible by grafts and by adult whitefly of the species now known as *Bemisia tabaci* Gennadius. In the absence of any visible pathogen the disease was assumed to be caused by a virus, but no particles were detected until 1975 when sap inoculations from cassava to cassava and 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 because it could be obtained from CMD-affected plants in western Kenya, western Tanzania and Uganda, but not from similarly diseased plants in coastal Kenya (Bock *et al*., 1978). For this reason it was at first not considered to be the cause of CMD and was referred to as cassava latent virus (Bock *et al.*, 1977, 1981; Adejare & Coutts, 1982; Sequeira & Harrison, 1982).

The uncertainty was resolved when successful transmissions were made to *Nicotiana benthamiana* using sap from the leaves of CMD-affected plants in both western and coastal Kenya (Bock & Woods, 1983). The virus isolates obtained and characterised had geminate particles and were similar to those from CMD-affected cassava sampled elsewhere in Africa and also in India and caused typical symptoms of the disease when returned from herbaceous hosts to cassava, so fulfilling Koch's Postulates. Consequently, the name cassava latent virus was regarded as no longer valid and the virus was renamed African cassava mosaic virus (Bock & Woods, 1983; Robinson *et al*., 1984; Bock & Harrison, 1985). The original name was retained in some later publications (Pacumbaba, 1985; Townsend *et al.*, 1986; Etessami *et al.*, 1988).

The various isolates from CMD-affected cassava in different parts of Africa and also from India (see following section) were initially regarded as strains of African cassava mosaic geminivirus. However, serological differences were reported in tests with polyclonal antisera using the 'type strain' from western Kenya, the Kenya coastal strain and a strain from India (Bock & Harrison, 1985). The African and Indian isolates were later ascribed to separate West African ('A'), East African ('B') and Indian ('C') strain groups or 'clusters' based on their serological

properties in tests using a panel of monoclonal antibodies (Harrison *et al*., 1987, 1997a; Harrison & Robinson, 1988). However, on serological evidence and nucleotide sequencing the three groups of isolates were later distinguished as separate 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 & Harrison, 1994). All three viruses are members of the recently recognized family *Geminiviridae*: Sub-Family III: *Begomovirus*.

There have been no systematic or comprehensive surveys of the distribution of ACMV and EACMV throughout the African cassava belt. Nevertheless, Harrison *et al*. (1991, 1995) carried out serological tests on isolates from many different areas. Their initial results indicated that the two viruses have separate and largely non-overlapping geographic distributions. EACMV was detected only once in West Africa, in a sample collected in Côte d'Ivoire from a variety introduced from Madagascar (Aiton & Harrison, 1988). Otherwise it was detected initially only in Malawi, Madagascar, Zimbabwe and the coastal regions of Kenya and Tanzania (Swanson & Harrison, 1994). ACMV was not detected in these areas but occurred in several West African countries (Benin, Burkina Faso, Ghana, Côte d'Ivoire, Nigeria and Senegal) and also in Angola, Burundi, Cameroon, Chad, Congo, Mozambique, South Africa, Uganda, Zambia, western Kenya and western Tanzania.

These findings are consistent with the results of earlier studies in Kenya where the viruses later referred to as EACMV and ACMV were found together in only one locality, east of Mount Kenya, where the varieties being grown were from different parts of East Africa (Bock *et al*., 1981). Such an apparently clear distinction in the distribution of the two viruses within Kenya and Tanzania was somewhat unexpected and difficult to explain given the mobility of *B. tabaci* and the extensive traffic in vegetative material that must have occurred within and between these countries over many decades. There is also likely to have been considerable movement of cuttings between coastal and inland countries of eastern and southern Africa along the roads, railways, waterways and other communication routes. However, in assessing the results it is important to appreciate that the monoclonal antibodies available detect EACMV only when present alone and not in dual infection with ACMV (Harrison *et al.*, 1997a).

For these reasons it is not surprising that the latest findings suggest that the distributions of ACMV and EACMV are not as distinct as originally suggested. There have been three main developments. Firstly, Ogbe *et al*. (1996, 1997) surveyed parts of eastern and southern Africa and used monoclonal antibodies in serological tests to determine the viruses present. EACMV was detected in some of the samples from western districts of Kenya and Tanzania where previously only ACMV had been reported. Secondly, EACMV was detected together with ACMV in a severely diseased plant in western Tanzania on the island of Ukerewe in the south of Lake Victoria (Harrison *et al*., 1997b). Thirdly, a novel geminivirus variant designated UgV has been isolated and found to be associated with the very severe CMD pandemic now affecting large areas of Uganda and parts of western Kenya (Harrison *et al*., 1997a, 1997b; Zhou *et al*., 1997). The variant has also been detected in southern Sudan. It causes severe symptoms, especially when it super-infects plants already infected with ACMV. UgV has the serological properties of ACMV, but from nucleotide sequencing data it also has features of the EACMV genome (Harrison *et al*., 1997a, 1997b; Deng *et al*., 1997; Zhou *et al*., 1997). Consequently, UgV is considered to be a recombinant between EACMV and ACMV, although its exact status and origin are uncertain.

These results and the earlier finding of an apparently defective isolate from Angola that was not mechanically transmissible and not detected serologically (Sequeira & Harrison, 1982; Robinson *et al*., 1984) suggest that the geminiviruses causing CMD in Africa are more diverse than previously suggested. This emphasises the need for further studies on a much wider range of isolates and especially from plants showing the severe symptoms of the type associated with the Ugandan pandemic. Such plants are to be found in many parts of Africa and also in India (as discussed later), but whether they are infected with a novel virus, a mixture of strains, or with a particularly virulent strain of the usual virus occurring in the locality has not been investigated.

The serological and/or biochemical differences between the cassava mosaic geminiviruses found in Africa may, in at least some instances, reflect innate biological differences, as suggested by differences in their temperature requirements for optimum replication in herbaceous hosts and in experimental host range (Robinson *et al*., 1984) and also by the most recent results from Uganda (Harrison *et al*., 1997a, 1997b; Zhou *et al*., 1997). However, the different viruses cause similar diseases and seem to be transmitted similarly by *B. tabaci*. Possible differences between the viruses in such features as virulence to different cassava genotypes, natural host range, effects on yield, transmissibility by vectors and ability to infect resistant cassava varieties have not been sought and are important topics for study. Additional information is also required on possible interactions between the different African cassava mosaic geminiviruses and the frequency with which recombinants arise.

India

A mosaic disease of cassava in India was first noted in 1956 (Abraham, 1956), although it is likely to have occurred much earlier. The disease was described later (Alagianagalingam & Ramakrishnan, 1966) and it has since received considerable attention in the southern states of Kerala and Tamil Nadu, which are the main cassava growing areas of India. A similar disease has also been reported in Sri Lanka (Austin, 1986).

The symptoms in India are similar to those reported in Africa and the name cassava mosaic disease (CMD) was adopted in some publications and Indian cassava mosaic disease (ICMD) in others. Malathi & Sreenivasan (1983) isolated a geminivirus from CMD-affected plants, as in the earlier studies in Africa. Four Indian isolates were included in serological tests with isolates from coastal and western Kenya using polyclonal antisera prepared against African and Indian isolates (Malathi *et al*., 1985, 1987). Three of the Indian isolates reacted positively with African antisera, but they were distinguishable serologically from African isolates and so were regarded as being of a separate strain of ACMV. In subsequent tests using a panel of monoclonal antibodies, Indian and Sri Lankan isolates were distinguished from those from East and West Africa and later distinguished as a separate virus as described previously.

Nomenclature

The distinction between Indian cassava mosaic virus and the two African cassava mosaic viruses has led to considerable uncertainty as to the most appropriate designation of the disease(s) they cause. A distinction is made between Indian and African cassava mosaic diseases in some publications and not in others. Moreover, a recent paper seeks to distinguish between African and East African cassava mosaic diseases (Chikaunga *et al*., 1996), whereas others consider such a distinction to be unwarranted and inappropriate (Otim-Nape & Thresh, 1998). Further difficulties could arise following the recent description of the Ugandan variant (UgV) and its association with the severe form of mosaic disease that has recently spread into Kenya (Harrison *et al*., 1997a, 1997b; Zhou *et al*., 1997). These difficulties can be avoided by using

the term cassava mosaic disease in a broad sense for the mosaic diseases of cassava in India and in the different parts of Africa caused by whitefly-borne geminiviruses. This usage is adopted here and is discussed further in a later section.

The transmission of cassava mosaic viruses by the whitefly *Bemisia tabaci*

Studies in Africa

The putative virus assumed to cause CMD in Africa was one of the first pathogens to be transmitted experimentally by whiteflies, and studies began in the 1920s when it became evident that the virus was spreading naturally and that whiteflies were the only insects on cassava likely to be vectors. The first transmissions were reported from Congo using adults of a species referred to as *Bemisia mosaicivecta* (Ghesquière, 1932), which was later stated to be a misprint for *B. mosaicivectura* (Storey & Nichols, 1938). The species was later referred to as *B. gossypiperda* Misra & Lamba var. m*osaicivectura* (Kufferath & Ghesquière, 1932) The same or a closely related species referred to as *B. nigeriensis* Corbett was used in successful transmission experiments in Nigeria (Golding, 1936) and Tanzania (Storey & Nichols, 1938), where infection was achieved by transferring infective whiteflies to the youngest leaves and shoots, but not to older ones.

Later experiments on the mode of transmission were carried out in Nigeria (Chant, 1958), Côte d'Ivoire (Dubern, 1979, 1994) and Kenya (Seif, 1981) using what seems to have been the whitefly species used earlier, but referred to as *Bemisia tabaci* Gennadius, as in all subsequent studies. Based on current knowledge it is likely that the transmission studies in coastal East Africa (Storey & Nichols, 1938; Seif, 1981) were with EACMV and those in Congo and West Africa with ACMV (Ghesquière, 1932; Golding, 1936; Chant, 1958; Dubern, 1979, 1994). There have been no published reports of vector transmission studies with the recently distinguished Ugandan variant (UgV). The East and West African isolates are transmitted in a persistent manner and the minimum (and optimum) acquisition access, inoculation access and latent periods for successful transmission are 3 h (5 h), 10 min and 3-4 h (6 h), respectively. Virus is retained by adults for at least 9 days. It persists during moulting, but it is not transmitted transovarially (Dubern, 1979, 1994). Nymphs can transmit, but they are not of epidemiological importance because of their immobility. Up to 1.7% of the adult whiteflies were shown to be infective when collected in heavily infected cassava fields in Côte d'Ivoire and transferred to young test seedlings of cassava (Fargette *et al*., 1990).

Studies in India

CMD spreads naturally in India and following earlier experience in Africa the main attention has been on *B. tabaci* in the search for an insect vector. Successful transmissions have been reported using whiteflies transferred from infected to healthy cassava, from infected cassava to herbaceous hosts and between herbaceous hosts. High rates of transmission were achieved in some experiments, as between cassava (19%) and from cassava to *Nicotiana tabacum* cv. Jayasri (100%), *N. rosulata* (67%) and 11 other *Nicotiana* spp. (20-50%) using 50 whiteflies per test plant (Mathew & Muniyappa, 1993). However, such high rates of transmission seem to be exceptional and not readily reproducible. Much lower rates of transmission were reported in other studies (eg Nair, 1975), some of which were completely unsuccessful (Malathi *et al*., 1985; Palaniswami *et al*., 1996). Another inconsistency is that transmissions from cassava to cucumber were achieved in some trials (Menon & Raychaudhuri, 1970) but not in others (Mathew & Muniyappa, 1993). The reasons for this and the apparent difficulty experienced in transmitting Indian isolates by whiteflies compared with those in Africa have not been determined. One possibility is that the whiteflies on cassava in India are less well adapted to their host than those in at least some parts of Africa where a cassava biotype of *B. tabaci* has been distinguished (Burban *et al*., 1992) It certainly seems particularly difficult to transmit Indian isolates to cassava and similar difficulties have been recorded with other isolates in studies in glasshouses in temperate conditions (B.D. Harrison & P.J. Markham, personal communication). Despite these difficulties there is no reason to doubt that *B. tabaci* is the vector of ICMV and studies on epidemiology, control and whitefly population dynamics have proceeded on this assumption (eg Mahto & Sinha, 1978).

The incidence of cassava mosaic disease in Africa and India

Africa

There has until recently been little quantitative information on the incidence of CMD in the many different countries of sub-Saharan Africa where the disease occurs. Nevertheless, various estimates of disease prevalence have been made, based on general experience or limited surveys (Thresh *et al*., 1994c, 1997). For example, Bock (1983) noted that the incidence of CMD was generally high in coastal and western Kenya, where it exceeded 80% in some districts and approached 100% in some individual plantings. Infection was also high in a sample of 10 farms assessed in Ghana, where the mean incidence was 96% (Walker *et al*., 1985). There was a similar situation at the time in Côte d'Ivoire where virtually all cassava plants were affected by CMD, except those specially selected and propagated at an up-country site for use in experiments (Fargette *et al*., 1985; Fauquet *et al*., 1987, 1988). In Ghana, Nigeria, Côte d'Ivoire and Congo Democratic Republic, mean CMD incidences of 85%, 82%, 82% and 87%, respectively, were recorded in 1989 during the limited assessments made in the Collaborative Study of Cassava in Africa (F.I. Nweke, personal communication).

The results of recent more detailed surveys of the incidence of CMD in eight African countries and in India are presented in Table 1, which also gives details of the number of fields surveyed in relation to estimates of the areas of cassava grown in each country. A total of 1900 plants were assessed in 93 plantings at 63 sites in different agro-ecological regions of Nigeria (Table 1). The overall incidence of infection was 55% and the incidence was less in the southern forest zone (36%) than in the drier savannah areas to the north (64%) and at mid-elevations on the Jos plateau (64%).

Further data on the incidence of CMD in Nigeria and Ghana and in also in Benin and Cameroon were obtained during the systematic surveys carried out in 1994 as part of the Ecologically Sustainable Cassava Plant Protection Project (Table 1). The detailed results are not yet available, but a preliminary report (Wydra & Msikita, 1998) provides additional evidence of the high overall incidence of CMD in Nigeria (82%) and Ghana (72%) and the somewhat lower incidences in Cameroon (67%) and Benin (53%).

Detailed data on the incidence of CMD were also obtained recently in Uganda where representative plantings were assessed between 1990 and 1992 and again in 1994 in three counties in each of the districts where much cassava is grown (Otim-Nape, 1993; Otim-Nape *et al*., 1998). The disease was recorded in 1276 (92%) of the 1350 farmers' plantings sampled in 1990-92, and overall, the mean incidence was 57%. The incidence was lowest in southern districts bordering Lake Victoria and highest in the drier grassland savannah areas of central and northern Uganda where serious epidemics of CMD have caused severe food shortages and hardship in recent years (Otim-Nape, 1993; Thresh *et al*., 1994d; Otim-Nape *et al*., 1997b). The epidemic had spread to additional areas by 1994, but cassava production in some of the first affected areas had begun to recover and the overall incidence in the country as a whole was little greater than in 1990-92.

Table 1. Surveys of the incidence of cassava mosaic disease (CMD) in India and eight African countries as discussed in the text and in more detail by Thresh *et al.* (1994c, 1997)

Ref.	Country (organisation)	Year	No. Fields assessed	Cassava area (mill. ha.)	Ha./field surveyed	CMD Incidence
1.	India (UAS, Bangalore)	1988	180	0.23	1,305	19
	Andhra Pradesh	1988	10	0	0	<1
	Karnataka	1988	40	0	0	5
2.	Uganda (NARO)	1990-92	1,350	0.36*	267	57
3.	Chad (AID)	1992	48	0.07	1,458	40
4.	Malawi (National)	1993	450	0.07	156	21
5.	Tanzania (National)	1993-94	325	0.69	2,123	26
6.	Ghana (ESCaPP)	1994	40	0.61	15,250	72
6.	Benin (ESCaPP)	1994	31	0.14	4,516	53
6.	Cameroon (ESCaPP)	1994	61	0.08	1,311	67
7.	Nigeria (IITA)	1994	93	2.00*	21,505	55
6.	Nigeria (ESCaPP)	1994	111	2.00*	18,018	82
8.	Uganda (NARO)	1994	1,200	0.38*	317	65
All African countries: total (mean)		**1990-1994**	**3,709**	**4.03***	**(1,087)**	**(54)+**

* Assuming single values for Uganda and Nigeria.

\+ In calculating the overall mean incidence the results of the first Uganda survey were omitted, the results of the two Nigerian surveys in 1994 were combined and the country values were weighted according to the area of cassava grown.

Sources:1. Mathew (1989), 2. Otim-Nape *et al.* (1998), 3. Johnson (1992), 4. Nyirenda *et al.* (1993), 5. Legg & Raya (1998), 6. ESCaPP (Yaninek *et al.*, 1994; Wydra & Msikita, 1998 and unpublished), 7. L.C. Dempster, unpublished, 8. G.W. Otim-Nape *et al.*, unpublished.

In a similar but less comprehensive survey of Tanzania in 1993 and 1994 (Legg & Raya (1998), CMD was recorded in 259 (74%) of the 325 farmers' plantings sampled, but the incidence exceeded 50% in only 17% of these. The mean overall incidence for the entire country was 26%. The disease was most prevalent in the lowland coastal districts, especially in the Tanga area north of Dar es Salaam.

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

In a survey of Malawi (Nyirenda *et al*., 1993), the incidence of CMD seldom exceeded 20% in upland areas at altitudes exceeding 800 m, whereas the disease was more prevalent at lower elevations where temperatures are higher and cassava is more widely grown. Almost total infection occurred in some farmers' plantings near the western shore of Lake Malawi, 773 metres above sea level.

India

There is little current information on the incidence of CMD in India and the only available data were obtained during a reconnaissance survey in 1988 (Mathew, 1989). Twenty fields were assessed in each of eighteen districts, including eleven districts of Kerala State. The overall incidence of CMD was higher in the two main cassava-growing states of Kerala (23%) and Tamil Nadu (30%) than in Andhra Pradesh (<1%) and Karnataka (5%), which are outside the main cassava-growing areas. However, the number of fields examined was limited, especially when considered in relation to the large area of cassava being grown (Table 1). There is a need for additional more comprehensive surveys, especially as CMD seems to have become more prevalent in recent years. This was evident on a recent tour of the main cassava-growing areas of Kerala and around Salem in Tamil Nadu. Many of the fields visited in the lowland areas were almost totally affected and in some localities the symptoms were unusually severe and associated with poor yields. The incidence was much less in the upland areas and in a lowland planting established with cuttings obtained from the hills (M. Thankappan and J.M. Thresh, unpublished observations, December 1996).

The effects of cassava mosaic disease on growth and yield

There is an extensive literature from Africa and India on the effects of CMD on growth and yield. Nevertheless, many uncertainties remain and there is a particular dearth of information on the effects of CMD on resistant varieties and in mixed stands of diseased and healthy plants in which there is an opportunity for compensatory growth to occur. Information is also lacking on the effects of intercropping and of the different viruses and virus strains.

Studies in Africa

Two main approaches have been adopted in studying yield loss, as discussed by Thresh *et al.* (1994a). One is to compare the growth and yield of plants raised from infected and uninfected cuttings set out in fully replicated experimental plots. The second is to compare the growth and yield of individual healthy plants and those infected naturally by whiteflies at different stages of growth. Both approaches have limitations but they have provided much important information on the effects of CMD on the growth and yield of different varieties in at least eleven countries. A striking feature of the results obtained is the great differences in the extent of the losses reported, which range from negligible to almost total. A mean loss of 54% compared with healthy controls was calculated from an analysis of the 94 comparisons made in the various studies listed by Thresh *et al.* (1994a). Much of the variation is associated with the sensitivity of the variety assessed; tolerant varieties that express inconspicuous symptoms are usually much less severely affected than sensitive varieties that develop conspicuous symptoms and grow poorly. Indeed, several studies have shown a general relationship between yield loss and symptom severity, as discussed by Thresh *et al.* (1997).

From observations on several varieties it is apparent that plants grown from infected cuttings sustain greater losses than plants infected later by whiteflies and that plants infected at a late stage of growth are even less affected and may sustain little or no yield loss (Fargette *et al*., 1988; Fauquet & Fargette, 1990). It is also likely that virulent strains have more severe effects than avirulent ones, but

evidence for this has not been presented. There is also a need for additional information on the effects of virus on yield in relation to soil fertility and intercropping and on the ability of healthy plants to compensate for the impaired growth of their diseased neighbours. In the only detailed study on compensation this was demonstrated with one variety but not with two other less sensitive ones and the overall significance of the phenomenon is uncertain (Otim-Nape, 1993; Otim-Nape *et al*., 1997c).

Several authors have used the results of yield loss studies to estimate the losses caused by CMD throughout Africa (Fargette *et al*., 1988; Fauquet & Fargette, 1990; Thresh *et al*., 1994c, 1994e, 1997). Different conclusions have been reached depending on the assumptions made. In the most recent assessment it was concluded that definitive estimates are not possible because of the inadequate information on the incidence of CMD and on the prevalence and sensitivity of the main varieties grown. Nevertheless, on plausible assumptions the losses in Africa were estimated to be 15-24% of total production (Thresh *et al.,* 1997). This amounts to 13-23 million tonnes compared with actual production in 1994 estimated as 73 million tonnes (FAO, 1995). Such losses amount to 1300-2300 million US dollars at a conservative valuation of US$100 per tonne.

Studies in India

There have been fewer yield loss studies on CMD in India than in Africa and no estimates have been made of overall losses in the sub-continent. Reductions in weight of tuberous roots of 84% were reported in the first experiments with a susceptible local variety (Narasimhan & Arjunan, 1974, 1976), but losses were only 19-26% in the hybrids tested and in the widely grown M4 from Malaysia (Thankappan & Chacko, 1976). In other experiments losses were 42% in the popular variety Kalikalan, ranged from 17 to 36% in nine selected hybrids and were 17% in M4 which was at the time considered to be tolerant of infection (Malathi *et al.,* 1985). Losses were even less in a later trial with M4 (7-10%) and four hybrid varieties (9-21%) and there was a positive relationship between yield loss and symptom severity scores (Nair & Malathi, 1987). These results and the relatively low incidence of disease suggest that CMD causes less severe losses in India than in Africa. Nevertheless, the disease is likely to have substantial effects in areas of India where CMD-sensitive varieties are grown and severe symptoms are prevalent.

The epidemiology of cassava mosaic disease in Africa and India

The viruses causing mosaic diseases of cassava are disseminated in the stem cuttings used routinely for propagation and they are also transmitted between plants by the whitefly vector (*Bemisia tabaci*). Consequently, the use of virus-free planting material is likely to provide an effective means of control in situations where there is little spread by whiteflies, but not where rapid spread occurs. This means that it is of crucial importance to distinguish between the two means of dispersal in order to understand the epidemiological situation and to determine the prospects for control by phytosanitation which involves the use of virus-free cuttings and the removal (roguing) of any additional diseased plants that occur.

Epidemiological studies have been done in some parts of Africa and in India, as discussed here. However, studies have been few when considered in relation to the large areas of cassava grown and the very diverse environments utilized (Fargette & Thresh, 1994). In many countries there are no stocks of healthy planting material for experiments and researchers seldom attempt to make the crucial distinction between plants grown from infected cuttings and those infected later by whiteflies. These are serious limitations and major obstacles to progress. There is an urgent need for additional studies and great advantages to be gained from an international, coordinated approach using a standard range of varieties, methodology and recording procedures (Thresh *et al*., 1994b; Legg *et al*., 1997). Otherwise there will be continuing difficulties in comparing the results obtained in different studies and in drawing valid conclusions on the main factors influencing vector populations, rates of disease spread and the prospects for control.

Studies in Africa

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 of Carter *et al*. (1992) and from more conventional zonations based on temperature and rainfall, or the mean duration of the growing season(s). It follows that there are likely to be big differences between regions in crop growth, vector populations, in the epidemiology of CMD and in the prospects for control, but there is inadequate information on which to confirm or refute this supposition.

The available evidence has been obtained at various times and in different countries, as discussed by Fargette & Thresh (1994) and Thresh *et al*. (1994c). The areas studied include both lowland and mid-altitude sites experiencing very different rainfall regimes. The most detailed studies have been in the lowland rain forest area of the Côte d'Ivoire near Abidjan, where temperatures and rainfall are generally favourable for crop growth and whitefly reproduction for much of the year (Fargette *et al*., 1994). In such conditions CMD usually spreads very rapidly, except to very resistant varieties and experiments were only possible by introducing virus-free cuttings from specially selected stocks raised at a site in the drier derived savannah area to the north where little spread occurred (Fauquet *et al*., 1987, 1988).

The Ivorian results are consistent with those of Bock (1983) who recorded more spread in the wetter areas of coastal Kenya, where the annual rainfall exceeded 1200 mm, than in the drier areas having a shorter growing season and where less cassava is grown. Moreover, Cours-Darne (1968) had earlier concluded from experience in Madagascar that *'the hotter and more humid the region the more virulent the disease. Coastal regions with high rainfall are therefore the ones most seriously affected'*. He also commented that virulence, in the sense of incidence of infection, *'diminished with increased altitude, so that at altitudes of c. 1300 m it was possible to grow varieties that had been abandoned on the coast because of their vulnerability to infection'*. Storey (1936) had earlier noted that the spread of CMD was less in upland areas of Tanzania (>1000 m a.s.l.) than at lower altitudes and that this was recognized by farmers in the lowlands who regularly introduced unaffected planting material from the hills.

Numerous epidemiology trials have been conducted in Uganda since severe problems due to CMD were reported in some parts of the country in 1988. Very rapid spread occurred to plantings of susceptible varieties in 1989-90 and 1990-91 in the worst affected areas. There was little or no spread at the time in the areas to the south bordering Lake Victoria that were relatively unaffected. Rapid spread was at first associated with the dry savannah conditions, where whitefly population densities and temperatures were higher and the dry season was longer and more intense than in the more humid former forest areas to the south (Otim-Nape, 1993). This view had to be revised when it became apparent that the severely affected area was expanding southwards at a rate of 20-30 km per year (Otim-Nape *et al.*, 1997b). The epidemic reached the northern shore of Lake Victoria and parts of western Kenya in 1995 and it is continuing to spread into previously unaffected areas to the east and west. The onset of the epidemic is associated with a big increase in the incidence and severity of CMD which has been attributed to the effects of the recently characterised UgV strain which

causes severe symptoms, especially in plants that also contain ACMV (Harrison *et al.*, 1997a, 1997b; Zhou *et al.*, 1997). However, this does not explain the big increase in whitefly population densities which appears to 'drive' the epidemic and a new or more aggressive strain of *B. tabaci*, or a change in populations of natural enemies, may be involved (Otim-Nape *et al.*, 1997b). These possibilities are now being investigated.

Studies in India

The area of cassava in India is considerably less than in Africa. Nevertheless, the crop is grown in diverse environments including the lowland humid forest areas of coastal Kerala, the upland foothills of the Western Ghats and the irrigated areas of Tamil Nadu where there is a prolonged dry season.

Epidemiological studies have used virus-free stocks of selected planting material or clones derived from meristem-tip cultures. Several cultivars were included in experiments done in three successive seasons at a site near Trivandrum, Kerala State (Nair, 1985). The final incidence of CMD did not exceed 1.3% in plots containing initial disease foci and was even less in plots without sources. There was also little or no spread in a later study where monthly plantings were made at a site near Bangalore in Karnataka State which is outside the main cassava-growing area (Mathew, 1989).

In a further trial at a site near Trivandrum, six cultivars were established in plots which contained initial sources of inoculum and CMD was also prevalent in the surrounding plantings. There was substantial spread to the susceptible cv. Kalikalan (50%), but not to the five more resistant cultivars (1-10%) (Nair, 1988). In a later more comprehensive study, there was more spread to plots which contained initial sources of inoculum (overall incidence 5.7%) than to those without (2.8%). However, the source effect was not consistent at each of the four sites or in the five cultivars and was largely due to the big difference in incidence in cv. Kalikalan at the site where most spread occurred (Nair & Thankappan, 1990).

It is not appropriate to make broad generalizations on the basis of these few experiments, but they suggest that there is considerable scope for exploiting the benefits of virus-free planting material, especially of resistant varieties and in areas of low infection pressure. Moreover, the results indicate that the high incidence of CMD in Tamil Nadu is due to the use of infected cuttings and not to rapid spread by whiteflies. Further studies are required to substantiate these conclusions and to establish whether they are of wide general validity. Additional evidence is also required on the importance of spread from sources within plantings and on the suggestion that this occurs more frequently in India than in Africa, where experience in Côte d'Ivoire, Kenya and Uganda has shown that much of the spread is by infective whiteflies moving between rather than within plantings (Fargette *et al.*, 1990; Bock, 1983; Otim-Nape, 1993).

Control measures

Experience in Africa

CMD is so prevalent in Africa and has such detrimental effects on growth and yield that there are substantial benefits to be gained from adopting effective control measures. Nevertheless, specific measures are seldom used and in many areas CMD is widespread but largely ignored and regarded as an inevitable feature of cassava production (Thresh *et al.*, 1994e, 1997). The reasons for this unsatisfactory situation are undoubtedly complex and they have received inadequate attention from socio-economists and those concerned with crop protection.

One reason for the limited adoption of control measures is that there has been inadequate research on the various options discussed by Thresh & Otim-Nape (1994). The main emphasis has been on the use of resistant varieties and phytosanitation, which involves the adoption of CMD-free planting material and the removal (roguing) of any diseased plants that occur later (Thresh *et al.*, 1998a). However, the most effective means of deploying resistant varieties and of practising phytosanitation have not been determined and there is a need to develop simple but durable and effective measures that farmers are prepared to accept and adopt on a sufficiently large scale.

Another difficulty is that extension services in Africa are generally so inadequately resourced that there is only limited dissemination of any resistant varieties or of information on any other measures that are developed. This is a particular problem because of the need to contact and influence millions of mainly small-scale farmers, many of whom are illiterate, poorly educated and belong to the poorest sectors of society. Moreover, farmers and consumers are understandably reluctant to accept virus-resistant or otherwise improved varieties unless they also meet other exacting criteria. These include the taste and palatability of the tuberous roots, growth habit and overall suitability for use in the cropping system.

These considerations explain why African farmers are sometimes unwilling to adopt virus-resistant varieties, even if they are made available. There is also a failure to recognize the advantages of selecting cuttings only from plants that are symptomless and unlikely to be infected (Thresh *et al.*, 1994c, 1998a). Inevitably there is much use of infected planting material and diseased plants are seldom removed from within plantings unless they grow so badly that they will produce little yield.

Such attitudes and practices are deeply entrenched and will be difficult to change. Nevertheless, controlling CMD would lead to substantially increased production, or release land for other crops, or permit longer periods of fallow. There would also be indirect benefits arising from the enhanced vigour of uninfected stands. These are likely to establish better, require less weeding and are better able to withstand the effects of cassava brown streak virus, cassava green mite (*Mononychellus tanajoa*), cassava mealybug (*Phenacoccus manihoti*) and other arthropod pests (Thresh *et al.*, 1994c). Important features of any new initiative to control CMD would be the development and release of a wide range of virus-resistant varieties that are well adapted to the needs of farmers and consumers and that are also satisfactory in all other respects. There is also a need to promote the use of simple selection procedures and to assess the possible benefits of roguing and the circumstances in which it should be adopted.

Experience in several parts of Africa has shown that researchers, extensionists, farmers and even administrators can, in exceptional circumstances, be impelled to mount substantial control campaigns to combat CMD and to alleviate the food shortages and hardship caused by particularly severe epidemics. Such epidemics occurred in several countries in the 1920s and 1930s when CMD first became prevalent in East and West Africa and many of the varieties being grown at the time were extremely vulnerable to infection (Cours *et al.*, 1997). This led to the first efforts to select local varieties or breed new ones that were resistant to infection, as discussed later by Jennings (1994).

Resistance breeding programmes continue in many African countries but the largest and most influential is at the International Institute of Tropical Agriculture (IITA), Ibadan. Improved varieties raised at IITA and having at least some degree of resistance to CMD are now widely grown in Nigeria, Uganda and several other African countries (Mahungu *et al.*, 1994). Parental lines and seed stocks have also been widely distributed for use in national breeding programmes in these countries and elsewhere.

CMD-resistant varieties have several important features (Fargette *et al*., 1996; Thresh *et al*., 1994d, 1998a, 1998b):-

- They are much less readily infectible than susceptible varieties;
- They develop inconspicuous symptoms, especially at the later stages of crop growth and may even become symptomless. Consequently, they grow and yield satisfactorily, even if infected at an early stage;
- There is a localized distribution of virus and the overall concentration tends to be low. This suggests that resistant plants are a poor source of inoculum from which spread can occur, but evidence for this has not been sought;
- Uninfected cuttings can be obtained from infected plants because of the localized distribution of virus. This is the 'reversion' phenomenon that has important epidemiological consequences in restricting the deterioration in health status that would otherwise occur during successive cycles of propagation.

The effectiveness of resistant varieties as a means of controlling CMD has become apparent in several countries. For example, a particularly serious epidemic occurred in Madagascar in the 1940s when the whole future of the crop on the island was in doubt until CMD-resistant varieties were developed and distributed widely (Cours, 1951; Cours-Darne, 1968). Additional details of the approach adopted have become available only recently (Cours *et al*., 1997), but there is a detailed account of the measures promoted in Uganda in the 1940s and 1950s in response to the severe epidemic that occurred then over much of the country (Jameson, 1964). CMD-resistant varieties were introduced from Tanzania and elsewhere and several were selected as being suitable for release to farmers. Moreover, roguing was strictly enforced by local government statute and the measures were so successful that CMD was brought under control and it was not a problem for several decades.

More recently there has been a resurgence of CMD in Uganda where the latest epidemic was first reported in 1988 (Otim-Nape, 1993; Otim-Nape *et al*., 1996, 1997b). It has since spread across much of the country and into western Kenya, causing severe food shortages and hardship, especially in areas where the varieties being grown were extremely vulnerable to infection. Sensitive varieties of this type have been virtually eliminated and the consequences have been particularly severe in districts where suitable resistant varieties were not available. There has been a huge largely unfulfilled demand for CMD-resistant varieties and several Government and Non-Governmental Organizations have mounted emergency relief or rehabilitation projects in some of the most severely affected districts of Uganda (Otim-Nape *et al*., 1994, 1997a, 1997b). Cassava production is now recovering in some of the first affected areas. These have received considerable external support from donors, but little has been done in Kenya or in the southern and eastern districts of Uganda affected more recently. On even the most optimistic assumptions it will be several years before production has fully recovered.

Experience in Uganda and elsewhere in Africa is that farmers' practices and particularly their choice of variety can be greatly influenced by CMD (Thresh *et al*., 1994d; Otim-Nape *et al*., 1997b). Where the incidence is high there is a tendency to discard varieties that are vulnerable and react so severely to infection that they produce little or no yield. Conversely, varieties that are somewhat tolerant are at a selective advantage and eventually predominate. It has also become evident from observations during the current pandemic in Uganda that farmers may accelerate this process of adaptation by culling out the most severely affected plants from within stands of mixed varieties and by seeking less vulnerable varieties from within the locality or elsewhere. Another important factor influencing the proportion of severely affected plants within a stand is that such plants produce little stem material for use as cuttings, even if farmers do not actively discriminate in favour of healthy plants.

These considerations are consistent with the findings of the Collaborative Study of Cassava in Africa (COSCA) which has shown a dynamic continually changing situation. Farmers have been found to adopt and abandon varieties with surprising rapidity, according to their productivity and in response to abiotic and biotic factors including pests and diseases (Nweke *et al*., 1994; Otim-Nape *et al*., 1994; Thresh *et al.,* 1994d).

It is also understandable why vulnerable varieties can be grown successfully and may even predominate where CMD is not a problem, but not where the disease is prevalent. Indeed, this could lead to cyclical changes in the cultivation of resistant varieties, which can be expected to increase in areas where CMD is prevalent until the disease is brought under control. The cultivation of vulnerable varieties then becomes feasible and may increase until conditions again facilitate spread, there is a further resurgence of CMD and more resistant varieties are again required (Thresh *et al.,* 1994d). Such changes have been modelled (Holt *et al*., 1997) and there is evidence that they have occurred in Uganda in recent decades as the incidence of CMD and the losses sustained have waxed and waned and there have been alternating periods of stable production and acute instability.

Experience in India

Cassava in India is grown under very different conditions from those in Africa. The relatively high productivity achieved in India is associated with the limited use of intercropping and with generally good husbandry practices. These include effective weed control, the establishment of uniform stands, the routine application of fertilizers and in some areas the use of irrigation. Moreover, the Indian crop is unaffected by either the cassava green mite or the cassava mealybug which have had such damaging effects in many parts of Africa.

In these favourable circumstances Indian farmers might be expected to give considerable attention to the health status of the planting material used and to other means of controlling CMD so as to further enhance yields and optimize production. However, their attitude towards the disease seems to be similar to that in many parts of Africa in that it is largely ignored. Little attempt is made to select cuttings from healthy plants, or to remove diseased plants from within partially infected stands. Moreover, considerable use is made of susceptible varieties even though resistant ones are available. This attitude can be explained in part by the high yields obtained, even from stands in which CMD is prevalent. Nevertheless, the disease is so widespread and has such detrimental effects on yield in some areas that productivity is affected and would be increased substantially by adopting effective control measures.

As in Africa, the main possible approaches to control are through phytosanitation and resistant varieties. Some attention has also been given in India to the use of insecticides to control the whitefly vector in attempts to reduce the spread of CMD. However, the results have been unsatisfactory and the routine use of insecticides is inappropriate on health and environmental grounds and not recommended (Malathi *et al*., 1985).

Virus-free stocks have been obtained by rigorous selection and through the use of meristem-tip therapy. They have been used in experiments and shown to remain largely free of CMD in areas where there is limited spread by whiteflies. Substantial increases in yield have been achieved in this way (Nair, 1990; Nair & Thankappen, 1990), but only limited attempts have been made to encourage the widespread adoption of such material.

Discussion

The mosaic diseases of cassava in Africa and India have received much attention from researchers, but there has been no consistent attitude towards the way in which they should be designated. They are both referred to as CMD in some publications, including a recent comprehensive review (Mathew, 1991). The Indian disease was also considered within the context of the *International Symposium on African Cassava Mosaic Disease* held in Côte d'Ivoire in 1987 (Malathi *et al.*, 1987; Fauquet & Fargette, 1987). However, there has been only limited contact between researchers in Africa and India and little cross-representation at the regular meetings of the International Society for Tropical Root Crops:- Africa Branch, and the Indian Society for Tropical Root Crops.

This lack of interchange has undoubtedly been to the disadvantage of both regions and partly explains why the diseases in Africa and India are treated as separate in many publications, especially those written since the distinction was made between Indian cassava mosaic virus and the two African cassava mosaic viruses. We consider this distinction inappropriate and unhelpful in that it overlooks the many similarities between the African and Indian diseases. They are caused by very similar geminiviruses that are transmitted similarly by the same whitefly vector (*B. tabaci*). Moreover, they have similar epidemiologies and can be controlled in the same way. It is also possible that further research may establish that there are even more geminiviruses of cassava than those now distinguished and it is already apparent that no clear distinction can be made visually between the diseases caused by EACMV and ACMV, or between these diseases and the one caused by ICMV. They can only be distinguished biochemically by using PCR assays to determine the virus(es) present. This requires equipment and expertise and it is not feasible to test individually the many mosaic-diseased plants encountered in epidemiology trials or routine surveys. For these reasons it is considered appropriate to regard the mosaic diseases of cassava in Africa and in India as the same disease, which it is convenient to refer to by the original name of cassava mosaic disease. This usage is commended as a means of avoiding the present difficulties and uncertainties and to prevent further problems arising should other cassava mosaic geminiviruses be distinguished.

The argument for regarding the African and Indian CMDs as the same disease would become even stronger if it can be shown that genotypes resistant to ICMV are also resistant to ACMV and EACMV and *vice versa*. Such evidence is not available, but it has not been sought. This is a serious omission, not least because of the opportunity to extend the range of resistant types that can be used in resistance breeding programmes in Africa and in India. The practical benefits to be gained are apparent from previous experience in Africa where progenies from breeding lines found to be resistant to CMD at Amani in eastern Tanzania were raised in Nigeria and a clone was selected and used as the resistant parent in the IITA breeding programme based at Ibadan in Nigeria (Beck, 1982). From the the information now available it can be inferred that the virus at Amani was EACMV, whereas that at Ibadan was ACMV, which suggests cross-resistance to each of the two viruses. Moreover, IITA clones with different levels of resistance to ACMV rank similarly in their resistance to the virulent UgV strain or virus associated with the current pandemic in Uganda (Otim-Nape, 1993).

There has been an understandable reluctance to exchange cassava genotypes between Africa and India because of the risks of transporting pests or pathogens between the two regions. It is certainly important to avoid introducing any of the African cassava mosaic geminiviruses to India or ICMV to Africa and the introduction of cassava mealybug or cassava green mite to India would have serious consequences. However, these risks can be avoided by enforcing strict quarantine requirements and by using *in vitro* cultures derived from meristem tips as a means of transporting germplasm between different regions. This technique requires expertise and inevitably leads to delays before the introduced material is established and made available for breeding purposes or field experiments. Consequently, *in vitro* cultures are not always used when moving material between neighbouring countries or within the same region of Africa and 'open quarantine' agreements have been negotiated to allow the movement of conventional hardwood cuttings. This carries inherent risks of facilitating an extension of the present distribution of EACMV and ACMV and of extending the even more limited geographic distribution of the damaging Ugandan variant (UgV). Those involved with quarantine and the movement of germplasm should be aware of these risks which should be considered in reaching decisions on the most effective policy to adopt in disseminating germplasm and planting material. It is particularly important to appreciate that, from recent experience with UgV, it can no longer be assumed to be safe to move infected material between countries which from serological tests seem to have the same serotype of virus.

Although there are many differences between cassava cultivation in India and in Africa there are also similarities in the problems posed by CMD and in achieving a satisfactory degree of control. In Africa three distinct situations have been recognized (Thresh *et al.*, 1997) and designated *'epidemic'*, *'endemic'* and *'benign'*. The main features of each are set out in Table 2. The epidemic situation currently seems restricted to parts of Uganda and the adjacent region of western Kenya, although it may be more widespread elsewhere in Africa than the available evidence suggests. Such a damaging situation has not been reported in India, but both the endemic and benign situations occur there and benefits are to be gained from adopting control measures. However, the reasons for using these are not compelling, especially where CMD is benign and yields are already high by African standards. This explains why Indian farmers are even more reluctant to implement phytosanitation than those in Africa and in both regions only limited use is being made of the resistant varieties already available. Such attitudes will be difficult to change, but there are advantages to be gained from a greater exchange of information between the two regions on the various possible approaches to the research and extension problems encountered.

Table 2. The main features of cassava mosaic virus disease (CMD) and its control in contrasting situations

Feature	Epidemic	Endemic	Benign
CMD Incidence	Prevalent	Prevalent	Not prevalent
CMD Severity	Severe	Less severe	Not severe
Yields	Drastically reduced	Impaired	Largely unaffected
Production	Unstable	Stable	Stable
Control	Essential	Optional	Unnecessary
Funding	Essential	Justified	Contingency Plans
Emergency relief	Required	Unnecessary	Unnecessary

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