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Cassava mosaic virus disease in East Africa: a dynamic disease in a changing environment

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Abstract

Cassava mosaic disease (CMD), now known to be caused by cassava mosaic geminiviruses (Family *Geminiviridae*; Genus *Begomovirus*), was first reported in East Africa in 1894. Epidemics occurred in Madagascar and Uganda in the 1930s and 1940s, and more localised rapid spread of CMD was observed in parts of coastal Tanzania in the 1930s and coastal Kenya in the 1970s. During the 1990s, a major regional pandemic of an unusually severe form of CMD has expanded to affect parts of at least five countries, causing massive economic losses and destabilising food security. Mechanisms responsible for the development and progress of the pandemic have been described, and comparisons of epidemiological data for varieties grown throughout the period under review suggest that the recent pandemic has been characterised by rapid rates of CMD spread hitherto unknown in East Africa. A key factor in the genesis and spread of the pandemic has been the recombination between two distinct cassava mosaic geminiviruses to produce a novel and more virulent hybrid. Although such events may be common, the known history of CMD in East Africa suggests that the frequency with which they become epidemiologically significant is low. A corollary of this is that resistance, developed originally in Tanzania between 1934 and 1960, and utilized and supplemented at the International Institute of Tropical Agriculture, Nigeria, since 1971, is providing effective CMD control in current pandemic-affected areas of East Africa. Consequently, it is concluded that prospects for managing CMD in the 21st century are good, and that the approach adopted should build on the model of collaborative research and implementation that has been established in tackling the current CMD pandemic.

Keywords: Cassava; Cassava mosaic virus disease; Whitefly vector (*Bemisia tabaci*); East Africa; Epidemiology; Pandemic; Control

1. Cassava in Africa

Cassava, *Manihot esculenta* Crantz, has been cultivated in South America for millennia, but it is a relatively recent introduction to Africa (Carter et al., 1992). It was transported across the Atlantic by the Portuguese in the 16th century, and was initially grown in and around trading posts in the Gulf of Guinea in West Africa. From the earliest period of its cultivation in Africa, the key features of the crop of drought tolerance and a capacity to yield satisfactorily in marginal soils

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were recognised. Diffusion of the crop into the less accessible interior was slow, occurring mainly via trade along the major rivers of West and Central Africa. Cassava is thought to have been introduced to East Africa from Madagascar in the latter part of the 18th century, but was not grown in the Great Lakes region until almost a century later. Although cassava was grown virtually throughout much of sub-Saharan Africa by the 20th century, it remained subsidiary to other crops until colonial authorities began to promote cassava as a famine reserve and for use in times of drought. By the 1920/1930s, cassava had attained its present status as one of the major African staple food crops and currently the total annual production of approximately 85 million tonnes is greater than that of any other crop in the continent. Cassava at the end of the 20th century has a vital role in the economic life of sub-Saharan Africa, both as a reliable food source for rural and urban populations and as an important source of income through the sale of fresh and processed produce.

2. Cassava mosaic disease: origins and early spread

Cassava mosaic disease (CMD) is the most important disease of cassava in Africa. The symptoms typically include an irregular yellow or yellow–green chlorotic mosaic of the leaves, leaf distortion and stunted growth. They were first reported from the Usambara Mountains of north-eastern Tanzania towards the end of the last century (Warburg, 1894). It was assumed from the early years of the 20th century that CMD was likely to be caused by a virus, since no pathogen was visible and early tests showed that the condition was transmissible by grafts (Zimmerman, 1906). However, the viral aetiology was not confirmed until many years later, following the isolation and visualisation by electron microscopy of geminivirus particles and successful mechanical transmissions from cassava to the experimental herbaceous host *Nicotiana benthamiana* and back to a susceptible Brazilian cassava cultivar (Bock and Woods, 1983).

There are few records of the spread of CMD during the early years of the 20th century, but a number of reports were made from countries of both East and West Africa of the increasing prevalence and geographic expansion of the disease during the 1920s/1930s, including those from Sierra Leone (Deighton, 1926), Uganda (Hall, 1928), Ghana (Dade, 1930) and Madagascar (François, 1937). Golding (1936) documented the northwards expansion of CMD into south-western Nigeria around the city of Ibadan and noted that, while the disease was absent between 1923 and 1925, the majority of fields contained infected plants by 1929. CMD was not recorded at this time from northern Nigeria. There was a clear pattern of northwards expansion of CMD within south-western Nigeria during this period, but the number of diseased plants in affected fields was 'not usually considerable'.

From some of the earliest reports of CMD, it was suspected that whiteflies (Homoptera: Aleyrodidae) were the most likely vectors, largely because these were the only insects commonly occurring on cassava (Dade, 1930). The first firm evidence of transmission by a whitefly of the genus *Bemisia* was obtained from what is now the Democratic Republic of Congo (Kufferath and Ghesquière, 1932).

Following a period of geographically extensive work during the 1930s on the newly emerging problem of CMD, research during the following two decades was concentrated within a smaller number of intensive and sustained programmes. Two of the most important, both in terms of CMD research and the development of the cassava crop, were those of Tanzania and Madagascar. These were followed by additional projects in Kenya during the 1970s–1980s and, more recently, in Uganda, following the onset of the current pandemic now affecting the region.

This paper considers the changing nature of CMD in East Africa from the 1930s–1950s to the present.

3. Early CMD epidemics and research in East Africa

3.1. Madagascar

Cassava cultivation seems to have been relatively sparse in the early part of the 20th century in Madagascar, although the situation changed abruptly in the 1920s following the development of a starch industry based on cassava. CMD was first reported from Madagascar in 1932 (François, 1937), when it was considered to be of relatively minor importance. This situation began to change in 1934 as an epidemic of CMD, apparently originating in the north-western part of the central plateau, spread to affect all cassava-growing areas of the country by the late 1930s (Cours, 1951; Cours et al., 1997). Symptoms of the disease that were associated with the epidemic became markedly more severe and were characterised by stunting and leaf abscission in some of the most sensitive cultivars. Losses were particularly great in the lower altitude coastal areas, where the high incidence and severity of CMD led farmers to abandon many of the cultivars being grown. In view of the important impact of the epidemic on cassava cultivation in Madagascar, and indirectly therefore on the developing starch industry, urgent action was demanded by the government (Frappa, 1938). It was recognised at an early stage that control would depend on the development of cultivars with a higher level of resistance to CMD than those being grown, and large-scale screening and breeding work began in 1935. Several factors favoured the development of resistance: first, significant variability was apparent among locally grown cultivars in their susceptibility and response to CMD; second, a wild relative of cassava, Ceara Rubber (*Manihot glaziovii* Muell.-Arg.), appeared to be less readily infected by CMD than cassava and, when affected, commonly expressed only transient symptoms. The first approach was to develop intra-specific crosses, both between locally available cultivars and introductions from other countries, notably Java, Indonesia. Resistance developed in this way, however, proved to be inadequate. Subsequently, inter-specific crosses were attempted between cultivated cassava and several wild relatives, followed by repeated cycles of backcrossing to cultivated cassava. Progeny with both acceptable levels of CMD resistance and good quality characteristics were obtained from crosses between cultivated cassava and Ceara Rubber, and distribution of the first resistant varieties began in the mid-1940s. Major multiplication programmes were set up to meet the demands for this material, and large blocks, tens of hectares in size, were established. By the latter part of the 1940s, it appears that CMD had become a minor problem, following the wide-scale adoption of resistant varieties.

3.2. Tanzania

The longest and most comprehensive programme of research on CMD during the first half of the 20th century was in what is now Tanzania. Initiated in 1934, the programme was sustained until 1960 and considered a wide range of key research issues. Surprisingly, however, there was little quantification of the scale of the CMD problem in Tanzania at the time and there is no published information on the prevalence of the disease on a countrywide or regional level. However, there was no reference to an epidemic on the scale of that described from Madagascar, so it is inferred that the status of CMD in Tanzania was relatively stable during this period.

The CMD research programme was initiated at Amani (approximately 900 m above sea level), in the Usambara Mountains of north-east Tanzania, a location close to an area of the coastal lowlands in which CMD was prevalent. Within the first few years, significant progress was made in describing symptom variants, confirming whitefly vector transmission and identifying potential control approaches (Storey, 1936). It was realised from an early stage that mild and severe forms of CMD occurred, and, through graft inoculation into a standard susceptible cultivar, '*Mbarika*', the stability of these symptom variants was demonstrated over successive cycles of vegetative propagation (Storey and Nichols, 1938a). It was also observed that the severe strain was much more readily transmitted by the whitefly vector

than the mild strain. Failure of prior infection with either 'strain' to provide cross-protection against subsequent infection by the other indicated that the mild and severe 'strains' were unlikely to be related.

Extensive whitefly transmission tests confirmed that the putative virus causing CMD was transmissible by a *Bemisia* species now known as *Bemisia tabaci*, and that infection only occurred through inoculating young newly emerging leaves (Storey and Nichols, 1938a). Evidence was also adduced for the incomplete systemicity of the causal virus, a phenomenon that was subsequently exploited in the development of improved germplasm, and that is now regarded as one of the most important components of resistance to CMD (Fargette et al., 1996).

Epidemiological investigations revealed that spread of CMD to initially healthy cassava plantings was much more rapid at lowland locations than at the upland Amani site. Successive monthly plantings of a susceptible cultivar at a lowland location (approximately 150 m above sea level) suggested that, while the age of the cassava crop had little effect on the rate of CMD spread, there were important seasonal differences. Spread was rapid during the hottest months of February–May and much less during the cooler months of July–November (Storey and Nichols, 1938b).

Due to the relatively rapid spread of CMD in the important cassava-producing area of the coastal lowlands, it was realised that phytosanitation measures would be only partially effective in controlling the disease and would be difficult to teach to growers. Consequently, great emphasis was given to resistance breeding. The approach was similar to that in Madagascar but, although the two research teams worked concurrently, there seems to have been little or no contact between them.

Early germplasm development work involved the evaluation of many local and introduced cultivars. Selections were made at a number of sites in East Africa including Malindi in coastal Kenya, Bukalasa in Uganda, Morogoro and Amani in Tanzania, and Zanzibar (Jennings, 1994). Three regionally important products of this work were cv. *Malindi* from Kenya (Storey, 1936), cv. *Aipin Valenca* originally from Brazil (Jennings, 1994) and *Msitu* from Zanzibar (Briant and Johns, 1940), all of which were considered to be moderately resistant to infection yet sensitive to CMD. Inter-specific crosses were attempted with a number of wild cassava relatives, but only crosses with Ceara Rubber provided progeny that both exhibited a degree of CMD resistance and grew vigorously. F1 hybrids had woody roots and three backcrosses were required to obtain material that combined resistance with acceptable tuberous root quality. Intercrosses between third backcross selections were then used to 'concentrate' putative recessive resistance genes. Open-pollinated seed obtained subsequently from intercrossed parents was sent to Uganda, Kenya and many other African countries, and this formed the basis for the major breeding programme initiated at the International Institute of Tropical Agriculture (IITA), Nigeria, in 1971 (Jennings, 1994). Of the many achievements of the Amani programme, this was perhaps the most significant in view of the great strategic importance for sub-Saharan Africa assumed by the IITA germplasm development programme since the 1970s.

3.3. Uganda

CMD was recorded from Uganda as early as the late 1920s (Hall, 1928), but it was not of major economic importance until after substantial increases in the intensity of cassava cultivation that followed the Migratory Locust outbreaks and food shortages of 1931–1933 (Jameson, 1964). Attempts were made to establish healthy plots of cassava in some of the areas worst affected by CMD, but all plants became infected and so a small number of reputedly resistant cultivars were imported from the Amani programme in Tanzania. CMD seems to have become less prominent between 1934 and 1938, but resurgences in incidence were again recorded following drought years in 1939 and from 1941 to 1944. In response, a second and more extensive introduction of resistant germplasm was made from Amani. Seventy initially healthy cultivars, including the new introductions, were evaluated at Serere, in what is now Soroti district of north-

eastern Uganda, in 1942. Test rows were interplanted with 'spreader' rows of a diseased local cultivar, *Nyumbazunga*. Material introduced from Amani typically remained uninfected after 8 months field exposure, or had CMD incidences $< 20\%$. In contrast, most local cultivars developed incidences of 60–100%, although it is notable that local cultivars featured in both the 1–20 and 20–40% incidence categories, and a single local cultivar, *Binti Misi*, was entirely unaffected. Further evaluations for yield and root quality were made in subsequent years and, by the late 1940s, six cultivars had been identified for multiplication in the north-eastern Teso region (now Kumi and Soroti districts), which was worst affected by CMD. These were *Aipin Valenca* (from Brazil via Amani), *Msitu* (from Zanzibar via Amani), *Binti Misi* (local from northern Uganda), *37244 E* (Amani-bred cross), *F 279* (from Java via Amani) and *Kru* (from Ghana via Amani). Key aspects of the control programme that was implemented were: the imposition of a 6-month cassava-free quarantine period at Serere to permit the establishment of a CMD-free nuclear multiplication block for improved cultivars; the subsequent replacement of all diseased local cultivars with new material along an 'advancing front'; and the enforcement of a rigorously enforced regime of removing (roguing) all diseased plants. As a result of this approach, Teso had sufficient CMD-free planting material to supply the district's needs by 1951 and no significant problems were experienced during the subsequent decade. Indeed, there were no further reports of CMD problems until the first reports in 1988 of what was to become the regional CMD pandemic, described later.

An interesting comparison can be made between data on the response to CMD of cassava cultivars grown in early experimental trials in both Uganda (Jameson, 1964) and Zanzibar (Briant and Johns, 1940) (Table 1). Methodologies differed slightly in the two sets of experiments. Most notably, only CMD-free planting material was used in the Ugandan trials while, in Zanzibar, a proportion of cuttings sprouted diseased. In both countries, however, trials were planted in locations in which CMD spread was expected to occur readily and 'spreader rows' of heavily diseased local cultivars were used. By using the multiple infection transformation of Gregory (1948), it is possible to calculate for both data sets the change in CMD attributable to spread by whiteflies during each experiment. It is apparent from these data that CMD spread much more rapidly on Zanzibar than at Serere, Uganda during this period. This suggests that, although the CMD problem in Uganda in the 1940s was considered to be acute (Jameson, 1964), this was more likely to have resulted from increases in the intensity of cultivation of sensitive local cultivars than to any change in the inherent virulence of the virus or whitefly vector.

Table 1
Comparison of CMD epidemiology data for cultivars grown on Zanzibar in 1938–1939 and at Serere, Uganda in 1942–1943[a]

Cultivar	Uganda[b]		Zanzibar[c]		
	Whitefly infection (%)	CMD change (MIU)	Cutting infection (%)	Whitefly infection (%)	CMD change (MIU)
Msitu	20	22	38	35	82
Mpezaze	8	8	17	22	31
Kru	2	2	11	42	63
Ankrah	12	13	73	13	69
F279	2	2	35	19	34

[a] Cutting infection, incidence of CMD attributed to the use of infected cuttings; Whitefly infection, incidence of plants newly diseased with CMD and assumed to have been infected by the whitefly vector during the course of the experiment; CMD change, increase in the incidence of CMD after transformation of percentage data to multiple infection units (MIU) (Gregory, 1948).

[b] Jameson (1964).

[c] Briant and Johns (1940).

3.4. Aetiology and epidemiology of cassava mosaic: Kenya 1970s/1980s

CMD featured prominently within a major UK-funded tropical plant virus research project begun in Kenya in the early 1970s and sustained for a decade. Geminate virus particles were isolated and visualised from plants with symptoms of CMD during the early stages of the programme (Bock, 1975), and early molecular studies demonstrated the presence of single-stranded circular DNA within the particles (Harrison et al., 1977). The aetiology of CMD remained unclear for some years, however, and the virus that was associated with affected cassava plants was initially referred to as cassava latent virus (CLV) (Bock et al., 1978, 1981). Following the successful identification of a common herbaceous host test plant (*N. benthamiana*) for the two recognised strains of CLV and the use of a CMD-sensitive cassava cultivar from Brazil for mechanical transmission tests, it was finally demonstrated that a cassava mosaic geminivirus causes CMD (Bock and Woods, 1983). Two distinct 'strains' of the virus were recognised during the early phase of the Kenyan programme, one from the coastal region and the other from the most important cassava production area in western Kenya (Bock et al., 1981). However, it was not until a greater diversity of CMD-affected material had been examined from a number of countries in Africa and Asia that serological and nucleotide sequencing evidence was obtained indicating the occurrence of three distinct cassava mosaic geminiviruses (Hong et al., 1993). Two occur in Africa, and the third in India and Sri Lanka. The distinct and largely non-overlapping distributions of the two African viruses, namely: *African cassava mosaic virus* (ACMV) and *East African cassava mosaic virus* (EACMV) were described subsequently by Swanson and Harrison (1994). These viruses and *Indian cassava mosaic virus* are now regarded as separate virus species of the family *Geminiviridae*, genus *Begomovirus*.

Epidemiological data were obtained from numerous experiments performed both in coastal and western Kenya (Bock et al., 1977; Bock, 1983, 1987, 1994a). The overall conclusion was that CMD infection pressure was generally low throughout Kenya, except at locations in the wetter parts of the coastal region in which there was intensive cultivation of cassava and a high incidence of CMD (Bock, 1987). In western Kenya, by contrast, there was little or no spread of CMD. Final CMD incidences, following a year's field exposure of initially disease-free local cultivars in the Busia area of Western Province, ranged from 0 to 4.3% (Bock, 1983). In similar experiments with three highly susceptible Brazilian cultivars, final incidences were 2, 3 and 10% (Bock, 1994a). These results contrasted with final CMD incidences of up to 70% in the same cultivars grown concurrently in the coastal region. The Kenyan team concluded that the use of infected cuttings as propagules was by far the most important factor influencing the incidence of CMD in Kenya. It was proposed, therefore, that the impact of the disease could be reduced effectively by adopting simple phytosanitation measures (Bock et al., 1977; Bock, 1994b). However, these were not promoted at the time or since and their effectiveness remains uncertain.

4. The current CMD pandemic in East Africa

4.1. Origins and early expansion in Uganda

Following the problems of the 1930s and 1940s, CMD was apparently maintained at an acceptable and relatively benign level in Uganda until the mid-1980s. At the end of this period, CMD was regarded as just one of several diseases affecting cassava (Otim-Nape, 1987). The situation changed when a severe outbreak of CMD was reported in Luwero district of north-central Uganda in 1988 (Otim-Nape, 1988; Otim-Nape et al., 1994, 1997), although insecurity at the time in northern and north-eastern Uganda restricted the scope for wider investigation. It was thought initially that the contrasts in CMD incidence and spread between northern and southern parts of Uganda could be explained by the differences in climate (Otim-Nape, 1993). The generally higher temperatures and drier conditions of the savannah areas of northern Uganda were considered to be a

key factor in promoting population development of the whitefly vector, *B. tabaci* (Gennadius), and therefore spread of CMD (Otim-Nape, 1993). This view was based on previous epidemiological studies in Kenya and Ivory Coast, West Africa, in which temperature was considered to be the key variable driving *B. tabaci* population increase and, thereby, CMD spread (Fargette et al., 1993a,b; Fishpool et al., 1995). However, this explanation became increasingly untenable as the area characterised by a high incidence of CMD extended southwards at a rate of 20–30 km per year during the early 1990s and into the more humid, cooler, former forest environment of south-central Uganda (Otim-Nape et al., 1997; Legg and Ogwal, 1998). Further detailed studies of the 'front' of what was now regarded as an expanding epidemic revealed both that populations of *B. tabaci* at and behind the 'front' were significantly higher than those ahead of the 'front' (Legg and Ogwal, 1998), and also that the disease associated with the epidemic elicited unusually severe symptoms (Gibson et al., 1996). The progress of the epidemic 'front' was mapped during regular monitoring surveys from 1992, during which time it expanded southwards through central, southern and southwestern Uganda (Otim-Nape et al., 1997), reaching the border with western Kenya in 1995 (Gibson, 1996) and northwestern Tanzania in 1998 (Legg and Okao-Okuja, 1999; Legg et al., 1999). Much of western Kenya and northwestern Tanzania were affected by 1999, when typically severe epidemic symptoms were recorded in southern Nyanza, south of Winam Gulf, on the east side of Lake Victoria (J.P. Legg, unpublished data).

4.2. Characteristics and impact of the current pandemic in East Africa

Otim-Nape et al. (1997) described the dynamics of the CMD epidemic in Uganda by defining a series of six zones traversing the 'front' along a north–south axis. The zones were distinguished ecologically, from the pre-epidemic zone ahead of the epidemic front to the post-epidemic zone of recovery behind it. Each zone was characterised in terms of the intensity and productivity of cassava cultivation, the incidence, severity and nature of CMD infection, the abundance of whitefly vectors and the response of farmers to the disease. The zonation represents both a spatial series at any given time for locations spanning the 'front' and a temporal series to describe the sequential changes that occur with time at any given location as the 'front' advances.

A simpler yet quantitative classification system has been used to provide a monitoring/forecasting tool (Legg et al., 1999). This combines CMD incidence and infection type (cutting or current season whitefly-borne) geo-referenced data collected from field surveys to develop maps from which 'epidemic', 'epidemic expansion' and 'non-epidemic' zones can be defined. 'Threatened' zones are then identified as non-epidemic zones in which cassava cultivation is important and which are near epidemic expansion zones. These graphics provide researchers, donors and other cassava production 'stakeholders' with an important tool for raising awareness among threatened farming communities and for planning control initiatives.

The effects of the CMD epidemic on farming communities in Uganda first became apparent in the early 1990s. The impact was greatest in the north-eastern districts of Soroti and Kumi, in which there was a heavy reliance on the cultivar *Ebwanateraka* that proved to be highly sensitive to the severe form of CMD. In each of these two districts, cassava production declined by 80–90% between 1990 and 1993, and many farmers ceased cultivating the crop (Thresh et al., 1994). In 1993, the failure of maize, bean and other food crops due to drought was compounded by the virtual absence of cassava as the usual food security reserve. Consequently, widespread food shortages and famine-related deaths were reported (Thresh et al., 1994). A common response of farmers to the problem was to cultivate alternative crops, most commonly sweet potato. The impact of the epidemic in central and western parts of Uganda was less acute, largely, it is thought, because of the cultivation of a wider diversity of cultivars, some of which had a degree of tolerance to the epidemic form of CMD, although the pattern of a reduction in area of cassava cultivated and substitution with alternative crops was similar.

Various attempts have been made to quantify the losses associated with the CMD epidemic. The most widely reported estimate (Otim-Nape et al., 1997; Otim-Nape and Thresh, 1998) is based on the assumption that, during the mid-1990s, annual losses were on average equivalent to the total production of four districts. Assuming an average district production of 150 000 tonnes and a conservative crop value of USD 100/tonne, total annual losses were estimated at 600 000 tonnes worth USD 60 million per year.

4.3. Biological mechanisms influencing the progress of the pandemic

As the impact of the CMD epidemic in Uganda became evident, a biological explanation was sought for the big changes that had occurred in the hitherto relatively benign pathosystem. The finding that there had been a significant change in the severity of disease (Gibson et al., 1996) led to intensive efforts to characterise virus isolates from severely diseased cassava plants. Numerous samples from different parts of Uganda including the epidemic area had been tested before 1996, using serological techniques that distinguished between ACMV and EACMV, and all isolates were identified as ACMV (Swanson and Harrison, 1994). In 1996, however, polymerase chain reaction (PCR)-based techniques were used to clone and sequence the DNA-A of virus isolates collected from parts of Uganda affected by the epidemic (Zhou et al., 1997). A novel cassava mosaic geminivirus variant was detected and designated UgV. It was shown to be a recombinant hybrid in which most of the coat protein gene was derived from ACMV, but the 3′ and 5′ parts of this gene and the remainder of the DNA-A were derived from EACMV (Zhou et al., 1997). Consequently, the variant is regarded as a strain of EACMV by Deng et al. (1997). Subsequent studies have demonstrated that, while the symptoms elicited by the variant, referred to here as UgV/EACMV-Ug, are more severe than those of ACMV, the most severe symptoms result from co-infection by both ACMV and UgV/EACMV-Ug (Harrison et al., 1997). Cassava leaf samples collected from CMD-affected plants at and behind the epidemic front commonly contained either ACMV and UgV/EACMV-Ug or UgV/EACMV-Ug alone, whereas those collected ahead of the front contained only ACMV. It has been suggested that UgV/EACMV-Ug has arisen relatively recently (Harrison et al., 1997), although the approximate date and location of its origin remain obscure, particularly since EACMV has never been recorded from Uganda.

Evidence for the occurrence and characteristics of UgV/EACMV-Ug seem to provide a convincing explanation for the change in the virulence and economic impact of CMD in Uganda. However, an important unexplained factor in the local and regional epidemiology of the severe form of CMD is the increase in whitefly abundance observed on cassava in affected areas (Otim-Nape et al., 1996; Legg and Ogwal, 1998). It was suspected initially that a novel *B. tabaci* biotype was associated with the epidemic. Accordingly, preliminary isozyme analyses using esterases were used to compare *B. tabaci* populations from pre- and post-epidemic locations, but the high degree of variability in banding patterns made it impossible to establish differences between populations (Legg et al., 1994). More recently, increasingly sophisticated molecular marker techniques have been used to compare populations. Sequences of cloned fragments of the mitochondrial cytochrome oxidase 1 gene have been determined and comparisons made for *B. tabaci* populations from cassava in Uganda (Fauquet et al., 1998b). The results have been equivocal and more comprehensive analyses are required. However, biological assays have shown that there is no reproductive barrier between pre- and post-epidemic *B. tabaci* populations (Maruthi et al., 1999) and no difference in their virus transmission capability. Currently, the most plausible reason for increased *B. tabaci* abundance in epidemic areas arises from the demonstration of a synergistic interaction between *B. tabaci* and severely CMD-affected cassava plants. In experiments in which *B. tabaci* adults were confined on either healthy or infected cassava plants, whitefly fecundity was significantly greater on the infected plants (Colvin et al., 1999a,b), thereby providing an explanation for the increased populations observed in the field.

Additionally, the big reduction in leaf area of severely affected cassava plants, together with the preference of *B. tabaci* for green symptomless portions of the leaves, leads to increased crowding of *B. tabaci* nymphs and adults, providing a probable cue for dispersal to other plants, a factor critical to the 'auto-propagation' characteristic of the epidemic (Colvin et al., 1999a,b). Key aspects of the postulated mechanisms underlying the origin and progress of the epidemic can be summarised as follows.

1. Novel more virulent cassava mosaic geminivirus arises through hybridisation between EACMV and ACMV (Zhou et al., 1997).
2. Greater concentration of novel virus particles in cassava facilitates vector transmission (Harrison et al., 1997).
3. Vector fecundity is enhanced by synergistic interaction with infected host plants (Colvin et al., 1999a,b).
4. Drastic reduction in leaf area in cassava infected with the novel virus and preference of *B. tabaci* for symptomless portions leads to crowding of whitefly populations (Colvin et al., 1999a,b).
5. Crowding of whiteflies on severely diseased host plants promotes emigration to other plants within the same field and also to other fields, further enhancing virus spread (hypothesised).
6. The distinctive biotype of *B. tabaci* that occurs on cassava is partially restricted to the crop (Legg et al., 1994; Legg, 1996), encouraging movement of emigrants to other cassava fields (hypothesised).
7. The effects of the severe form of CMD on growth and yield are so great that cultivation of sensitive varieties is abandoned in epidemic-affected areas, leading to a decline in the intensity of cassava cultivation (Thresh et al., 1994).
8. Reduction in the intensity of cassava cultivation associated with the epidemic (Otim-Nape et al., 1997) increased duration of whitefly dispersal over longer distances than formerly between cassava crops (hypothesised).
9. Greater abundance of whiteflies emigrating from severely diseased cassava in epidemic-affected areas leads to larger numbers of immigrants reaching and colonising healthy plants in neighbouring pre-epidemic areas (hypothesised).
10. Increased immigrant whitefly populations enhances rates of CMD spread to the largely healthy plantings in pre-epidemic areas (Colvin et al., 1999a,b)

Convincing evidence has been obtained from studies in Uganda and elsewhere to explain the unique dynamics of what is now a major epidemic of CMD, although a number of outstanding research questions remain, most of which are now being studied. The largest gaps in understanding relate to whitefly movement, mainly because of the practical difficulties associated with monitoring the movement of such small insects over what may be considerable distances. Investigation of within-field movements can be addressed using currently available experimental techniques, but novel methodological approaches may be required for the study of the movement between fields that is so important to the epidemiology of CMD.

4.4. Regional expansion of the pandemic

The epidemic of severe CMD expanded rapidly into western Kenya to affect virtually the entire area of cassava cultivation in Western province and the northern part of north Nyanza province between 1995 and 1998 (Legg et al., 1999). This represents approximately one-third of the total area of cassava in Kenya. Losses to cassava production associated with this expansion have been estimated at 140 000 tonnes annually, which is equivalent to USD 14 million (Legg et al., 1999). Given the increasingly regional nature of the CMD problem, it became appropriate to refer to it as a pandemic (Otim-Nape et al., 1997). Reports were made of the occurrence of UgV/EACMV-Ug from near Juba, in southern Sudan, in 1997 (Harrison et al., 1997) and from Kisangani in the Democratic Republic of Congo (DRC), in 1998 (S. Winter, personal communication). Since the distribution of UgV/EACMV-Ug has been shown to match closely that of the pandemic-affected zone (Harrison et al., 1997), it is assumed that the range of the pandemic there-

fore extends into southern Sudan and eastern DRC. Insecurity in both regions, however, has precluded the implementation of the surveys that are needed in both areas to determine the current status of CMD and the viruses present. More recently, reports have been received from Congo Republic of unusually severe symptoms of CMD (P. Neuenschwander, personal communication), and samples are currently being analysed at IITA, Ibadan to determine the virus present. Current information on the distribution of UgV/EACMV-Ug suggests an east–west coverage exceeding 1000 km. If the severe symptoms in Congo Republic are shown to be caused by UgV/EACMV-Ug, this would more than double the documented east–west occurrence of this virus variant and would suggest that the scale of the CMD pandemic is even greater than has been assumed.

The establishment in Uganda in 1997 of PCR techniques for research on CMD has facilitated the detection and diagnosis of cassava mosaic geminiviruses from DNA samples collected throughout the East African region. Specific oligonucleotide primers, developed at the Scottish Crop Research Institute, UK (Harrison et al., 1997; Zhou et al., 1997), detect ACMV, EAMCV and UgV/EACMV-Ug singly and in mixtures. Virus diagnoses from hundreds of samples have been used to develop a virus distribution map for the region (Fig. 1) (Legg and Okao-Okuja, 1999), although, due to the dynamic nature of the CMD pandemic, this represents only a limited 'snapshot' of the continually changing situation. In addition to the progressive expansion of the distribution of UgV/EACMV-Ug associated with the pandemic, there is also a suggestion that in post-epidemic areas ACMV has been largely replaced by the more virulent UgV/EACMV-Ug and, in some areas, there appears to have been what can be regarded as competitive exclusion of ACMV by virulent strains of UgV/EACMV-Ug. A likely explanation for this is that plants containing both UgV/EACMV-Ug and ACMV are so severely diseased that they provide little material for propagation and farmers exclude them when collecting cuttings to establish new crops.

There is also evidence for the occurrence of both mild and severe strains of UgV/EACMV-Ug in post-epidemic areas (Pita et al., 1998), which is reminiscent of some of the early observations of Storey (1936). It is likely that interactions between such strains are important in the dynamics of the CMD pandemic since, while virulent strains of UgV/EACMV-UgV predominate throughout the pandemic-affected zone (Harrison et al., 1997; Legg and Okao-Okuja, 1999), there has been a clear reduction in symptom severity in post-epidemic areas, suggesting the emergence of relatively mild strains. Such changes in the virus balance in post-epidemic areas are the subject of current investigation. However, in order to clarify the situation, it will be necessary to develop a PCR-based diagnostic or other means of distinguishing between the mild and severe strains.

5. Regional comparisons of CMD epidemiology in East Africa and future perspectives

Experimental data from the early 1990s for the Javan cultivar, *F279*, which was designated *Bukalasa (B) 11* in Uganda, provide a means of comparing the epidemiology of CMD during the current pandemic and the relatively benign situation in the pre-epidemic years and earlier (Table 2). Rates of CMD spread, measured in terms of the changes in incidence attributable to whiteflies, were higher at all sites that by 1989–1990 had been affected by the CMD pandemic in Uganda than at the 1989–1990 pre-epidemic site at Namulonge, near Kampala in southern Uganda, or earlier at the other sites in Uganda and Zanzibar. Clearly, the 1989–1991 epidemic situation in Uganda contrasted markedly with that in 1942–1943, emphasising the exceptionally rapid rates of spread that are such a feature of the current pandemic.

The data also suggest that, for a cultivar such as *F279*, the pre-epidemic situation recorded at Namulonge during 1989–1990 was comparable with that recorded during the CMD 'crisis' of the 1940s (Jameson, 1964). The change in the performance of *F279* as the CMD pandemic has progressed provides a good example of the shifts in balance in the relationship between a pathogen and its host that can occur during co-evolution. It

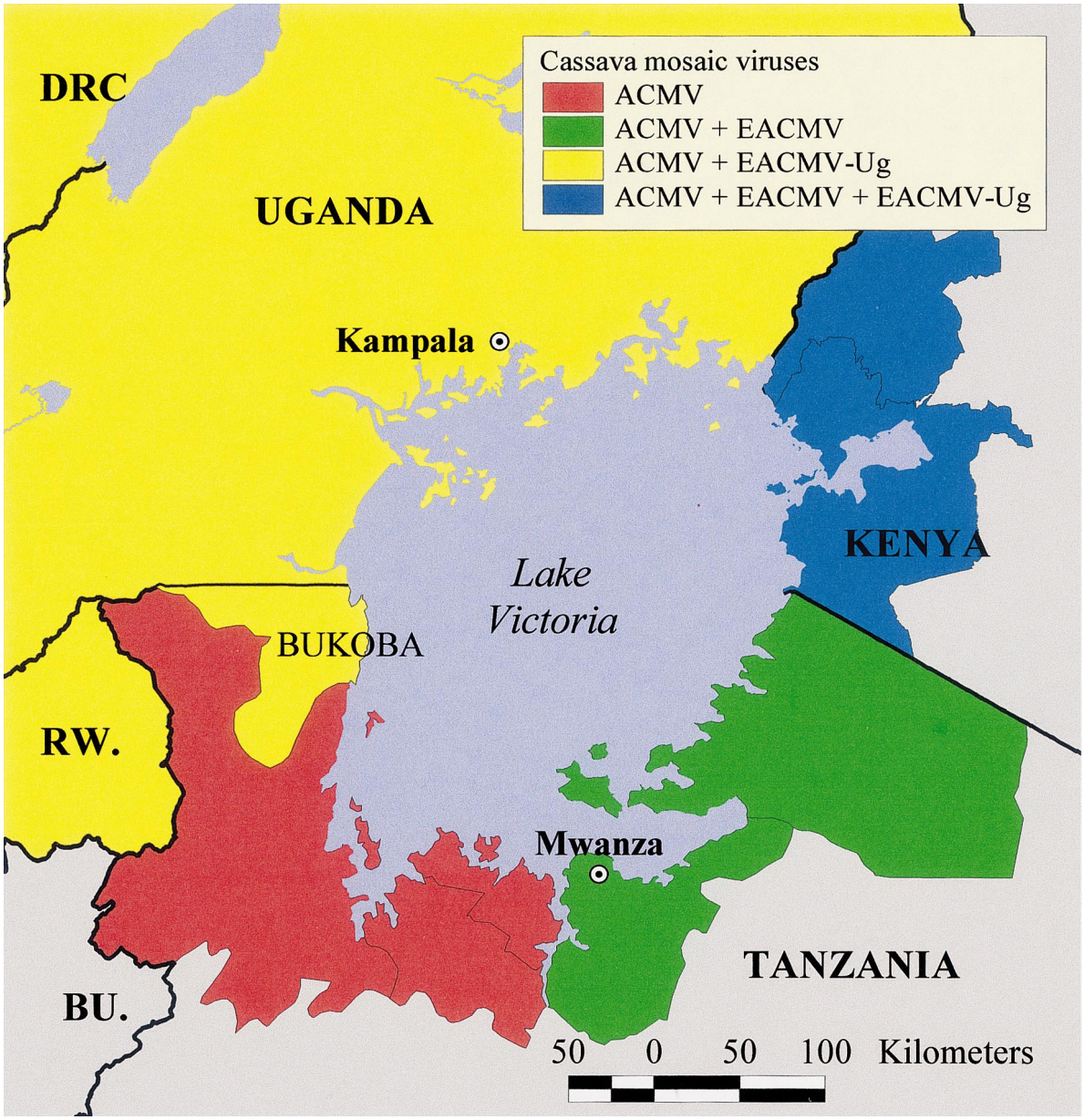

Fig. 1. Occurrence of different cassava mosaic geminiviruses in the Lake Victoria basin of East Africa. DRC (Democratic Republic of Congo, Kisangani), S. Winter, personal communication; Kenya, Ogbe et al. (1996), Legg and Okao-Okuja (1999); Rw. (Rwanda), Legg and Okao-Okuja (1999); Tanzania, Ogbe et al. (1997), Harrison et al. (1997), Legg and Okao-Okuja (1999); and Uganda, Ogbe et al. (1996), Harrison et al. (1997), Fauquet et al. (1998b), Legg and Okao-Okuja (1999).

is notable, however, that the frequency of such shifts appears to be relatively low. It has been argued that, far from being isolated events, evolutionary changes in geminiviruses such as the recombination between ACMV and EACMV to produce UgV/EACMV-Ug, may be relatively common (Fauquet et al., 1998a). However, even if such changes are common, the frequency with

which they have an important epidemiological and therefore economic effect seems relatively low. Indeed, it could be argued that the recombination event that has seemingly triggered the CMD pandemic in East Africa was highly improbable, in view of the absence of any evidence for the occurrence of EACMV in Uganda and the apparently independent co-existence of EACMV and ACMV in mixed infections in parts of western Kenya and northwestern Tanzania (Harrison et al., 1997; Legg and Okao-Okuja, 1999).

From the perspective of CMD management, it is encouraging that CMD-resistant germplasm developed at IITA, Ibadan, Nigeria, in the 1970s and developed from progenies derived from Amani in Tanzania has shown good levels of resistance under the pandemic conditions in Uganda (Otim-Nape et al., 1998). Furthermore, more recent germplasm selections based on crosses between West African landraces and CMD-resistant lines derived from the early Amani programme appear to be almost immune to infection. Conventional breeding, therefore, seems to have provided cassava with the ability to co-exist with cassava mosaic geminiviruses in what can be regarded as an evolutionary 'battle' for survival (Thresh, 1990). With the prospect of genetic engineering techniques being used to strengthen the breeders 'arsenal', the scope for management of CMD in the 21st century seems promising. Arguably, a more critical constraint to achieving this in sub-Saharan Africa is the slow pace with which newly developed varieties and CMD management practices are being diffused into and within farming communities. While progress continues to be made in understanding cassava mosaic geminiviruses and the disease they cause, and in developing and improving control techniques, farmers can benefit greatly from an increased emphasis on the dissemination of currently available technologies. This is an important issue to be considered by those responsible for strategic planning and the disbursement of resources for agricultural improvement both nationally and internationally. In East Africa during the 1990s, significant progress has been made in elucidating the dynamic complexity of the CMD pandemic. Associated with this, however, has been a vigorous programme of dissemination of control technologies, based on the deployment of resistant varieties (Thresh and Otim-Nape, 1994; Thresh et al., 1994, 1998; Otim-Nape et al., 1994, 1997; Anonymous, 1999; Legg et al., 1999) that has necessitated strong regional and international collaboration. The integration of research and its effective application can be seen as a model for

Table 2
Comparison of rates of CMD spread into cv. *F279* between locations and dates[a]

Country (location)	Year/epidemiological status	Whitefly infection (%)	CMD increment (MIU)
Uganda (Serere)[b]	1942–1943	2	2
Zanzibar (Kizimbani)[c]	1938–1939	19	34
Uganda[d] (Namulonge)	1989–1990/pre-epidemic	0	0
	1990–1991/pre-epidemic	0	0
Uganda (Bulindi)	1989–1990/epidemic	42	54
	1990–1991/epidemic	40	51
Uganda (Migyera)	1989–1990/epidemic	72	127
	1990–1991/epidemic	75	139
Uganda (Kagando)	1990–1991/epidemic	92	253
Uganda (Mubuku)	1990–1991/epidemic	37	46

[a] Cutting infection, incidence of CMD attributed to the use of infected cuttings; Whitefly infection, incidence of plants newly diseased with CMD and assumed to have been infected by the whitefly vector during the course of the experiment; CMD change, increase in the incidence of CMD after transformation of percentage data to multiple infection units (MIU) (Gregory, 1948).

[b] Data presented in Table 1 from Jameson (1964).

[c] Data presented in Table 1 from Briant and Johns (1940).

[d] Uganda data from Otim-Nape et al. (1998), in which *F279* is referred to by the local name of *Bukalasa (B) II*.

similar such situations as they arise elsewhere, and provides grounds for optimism into the 21st century for the improved management of what remains the most important constraint to cassava production in Africa.

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