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Comparing the regional epidemiology of the cassava mosaic and cassava brown streak virus pandemics in Africa

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ABSTRACT

The rapid geographical expansion of the cassava mosaic disease (CMD) pandemic, caused by cassava mosaic geminiviruses, has devastated cassava crops in 12 countries of East and Central Africa since the late 1980s. Region-level surveys have revealed a continuing pattern of annual spread westward and southward along a contiguous 'front'. More recently, outbreaks of cassava brown streak disease (CBSD) were reported from Uganda and other parts of East Africa that had been hitherto unaffected by the disease. Recent survey data reveal several significant contrasts between the regional epidemiology of these two pandemics: (i) severe CMD radiates out from an initial centre of origin, whilst CBSD seems to be spreading from independent 'hot-spots'; (ii) the severe CMD pandemic has arisen from recombination and synergy between virus species, whilst the CBSD pandemic seems to be a 'new encounter' situation between host and pathogen; (iii) CMD pandemic spread has been tightly linked with the appearance of super-abundant *Bemisia tabaci* whitefly vector populations, in contrast to CBSD, where outbreaks have occurred 3–12 years after whitefly population increases; (iv) the CMGs causing CMD are transmitted in a persistent manner, whilst the two cassava brown streak viruses appear to be semi-persistently transmitted; and (v) different patterns of symptom expression mean that phytosanitary measures could be implemented easily for CMD but have limited effectiveness, whereas similar measures are difficult to apply for CBSD but are potentially very effective. An important similarity between the pandemics is that the viruses occurring in pandemic-affected areas are also found elsewhere, indicating that contrary to earlier published conclusions, the viruses per se are unlikely to be the key factors driving the two pandemics. A diagrammatic representation illustrates the temporal relationship between *B. tabaci* abundance and changing incidences of both CMD and CBSD in the Great Lakes region. This emphasizes the pivotal role played by the vector in both pandemics and the urgent need to identify effective and sustainable strategies for controlling whiteflies on cassava.

1. Introduction

Cassava is one of the most important food staple crops in many countries of sub-Saharan Africa. Human population growth rates of many of the countries of this sub-region continue to be some of the highest in the world, and consequently there is an urgent need to match this growth with concomitant increases in food production. Cassava production in Africa is restricted by a diverse set of con-

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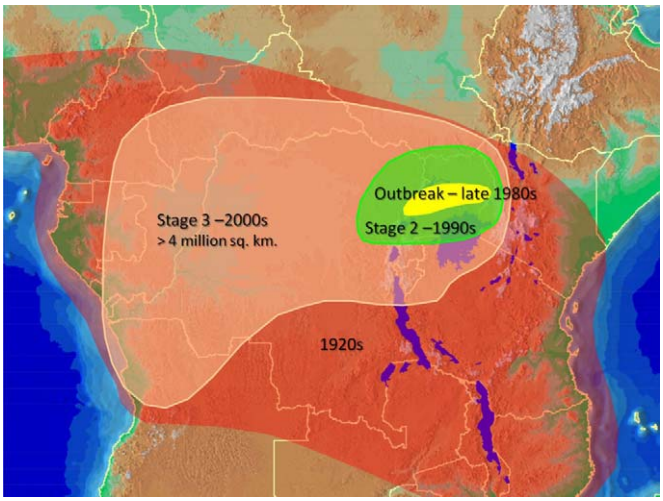

Fig. 1. Stages of expansion of the CMD pandemic in East and Central Africa.

straints. Arguably the most economically important, however, are the two virus diseases: cassava mosaic disease (CMD) and cassava brown streak disease (CBSD). Both have been recognized for many decades, but have become increasingly damaging in recent years.

CMD is caused by viruses of the family *Geminiviridae*: genus *Begomovirus* (Bock and Woods, 1983; Hong et al., 1993), referred to collectively as cassava mosaic geminiviruses (CMGs). Nine species are currently recognized (Fauquet et al., 2003; Legg, 2008), of which eight have been reported from Africa. CMD occurs wherever cassava is grown in Africa, from Senegal in the north-west to Mozambique in the south-east, as well as on the off-shore islands of Madagascar, Mauritius, Seychelles, Zanzibar and Cape Verde (Thresh and Cooter, 2005). The biology of the CMGs has been the subject of much study, although the volume and scope of this research has increased greatly since the early 1990s. Key areas of interest have included: molecular biology (Ndunguru et al., 2005; Patil and Fauquet, 2009), vector transmission (Dubern, 1994; Maruthi et al., 2002), field and regional-level epidemiology (Fargette et al., 1993, 1994; Legg, 1999; Legg et al., 2006), resistance breeding (Akano et al., 2002; Dixon et al., 2003; Jennings, 1994) and management (Thresh et al., 1994a; Thresh and Cooter, 2005).

CBSD was first reported in the early 1930s from the coastal zone of Tanzania, in East Africa (Storey, 1936), the same region in which the earliest observations of CMD had been made (Warburg, 1894). For the remainder of the 20th century, the distribution of CBSD was largely confined to lowland coastal areas of Kenya, Tanzania and Mozambique, as well as the surrounds of Lake Malawi in Tanzania and Malawi (Hillocks et al., 1999; Nichols, 1950; Legg and Raya, 1998). Unlike CMD, which only expresses symptoms in the leaves of cassava, CBSD's most economically significant symptom is a dry brown-black necrotic rot of the tuberous roots (Nichols, 1950). Other important symptoms include yellow blotchy chlorosis or feathery chlorosis in the secondary and tertiary veins, as well as brown necrotic streaks on the green portions of stems and seed capsules. Leaf symptoms are most prominent on the lower parts of the plant, which can make them hard to recognize unless plants are examined carefully and before leaves have senesced. All of these leaf, stem and root symptom types may occur in severely affected plants. More commonly, however, infected plants will have only one or two symptom types. Since the main source of yield loss to CBSD is presumed to be root rot, germplasm improvement efforts have focused mainly on selecting varieties that do not show root necrosis, or express symptoms at a late stage of growth. These varieties are typically referred to as 'tolerant'.

Although CBSD was thought to have a viral aetiology at the time of its 'discovery' (Storey, 1936), this has only recently been proven definitively with a classical demonstration of Koch's postulates (Winter et al., 2010). Field studies, however, confirmed a consistent association between foliar symptoms of CBSD and root necrosis (Hillocks et al., 1996), and a growing volume of molecular studies has confirmed the viral aetiology of CBSD with the cloning and sequencing of partial or complete viral genomes from CBSD-infected plants (Mbanzibwa et al., 2009; Monger et al., 2001, 2010; Winter et al., 2010). Two distinct but similar virus species are now recognized: *Cassava brown streak virus* (CBSV) and *Ugandan cassava brown streak virus* (UCBSV). In this article, these are referred to collectively as cassava brown streak viruses (CBSVs). Both are in the family *Potyviridae*, genus *Ipomovirus*. CBSVs, like the CMGs, have been shown to be transmitted by the whitefly vector, *Bemisia tabaci* (Gennadius) (Maruthi et al., 2005) and both are disseminated readily through cuttings taken inadvertently from infected parent material.

2. The dual pandemic of CMD and CBSD

2.1. The CMD pandemic

A rapidly spreading epidemic of an unusually severe form of CMD was first observed in north-central Uganda in the late 1980s (Otim-Nape et al., 1994). Key features of the epidemic were the very severe CMD symptoms (Gibson et al., 1996), the high incidence of current-season whitefly-borne infection (Otim-Nape et al., 1997) and up to 100-fold increases in the abundance of the whitefly vector (Legg and Ogwal, 1998). Characterization of the viruses occurring in the epidemic-affected area demonstrated a consistent association with a recombinant CMG variant, *East African cassava mosaic virus*-Uganda (EACMV-UG) (Zhou et al., 1997), as well as the common occurrence of mixed infections of EACMV-UG and the previously occurring *African cassava mosaic virus* (ACMV) (Harrison et al., 1997; Pita et al., 2001). The severity of symptoms commonly led to the almost complete elimination of the most vulnerable varieties. Cassava crops were abandoned, and widespread food shortages and some famine-related deaths were reported in Uganda during the early 1990s (Thresh et al., 1994a). Spread to the neighbouring countries of the Great Lakes region and beyond was reported subsequently (Bigirimana et al., 2004; Gibson, 1996; Legg, 1999; Neuenschwander et al., 2002), resulting in the designation of the phenomenon as a pandemic (Otim-Nape et al., 1997). By 2005, the area affected had expanded to almost 3 million sq. km across nine countries, and annual losses attributable to CMD in Africa were estimated to be greater than 13 million tonnes – slightly more than a third of total production (Legg et al., 2006). The CMD pandemic continues to expand its range to the west, south and east, and the most recent published reports of new occurrences are from Angola (Lava Kumar et al., 2009) and Cameroon (Akinbade et al., 2010).

2.2. The CBSD pandemic

For most of its known history, CBSD seems to have been confined to coastal East Africa and the shores of Lake Malawi. From 2004, however, CBSD-like symptoms began to be seen in central-southern areas of Uganda (Alicai et al., 2007). It soon became apparent that CBSD was becoming increasingly prevalent over time (Alicai et al., 2007). Subsequent reports of new occurrences in western Kenya (H. Obiero, unpublished data) and north-western Tanzania (Jeremiah and Legg, 2008) have confirmed that new outbreaks of CBSD are not confined to Uganda and justify the pandemic designation. Incidences of the disease have increased rapidly, and the dramatic impact of root losses resulting from CBSD root symp-

toms (Jeremiah and Legg, 2008) has led to widespread concern amongst those producing and utilizing cassava throughout East and Central Africa.

Although both major pandemics appear to share a number of features in common, not least the whitefly vector and the fact that both were first reported in Uganda, a closer examination of their respective characteristics reveals more contrasts than similarities. In this paper, we review some of the key differences and then use this analysis to provide recommendations for current and future management programmes. These recommendations focus primarily on CBSD, recognizing that CMD-resistant varieties are already available and being widely deployed (Legg et al., 2006; Otim-Nape et al., 1997; Thresh and Cooter, 2005).

3. Comparisons and contrasts between the CMD and CBSD pandemics

3.1. Contrast 1: spread radiating out from an initial central origin vs. isolated 'hot-spots'

The pattern of spread of the severe CMD pandemic has been thoroughly characterized through almost uninterrupted monitoring studies conducted in the Great Lakes region of East and Central Africa since 1993 (Legg et al., 2006; Otim-Nape et al., 1997; Sseruwagi et al., 2004). Most importantly, the CMD pandemic has been shown to 'advance' along a contiguous 'front' at rates of up to 100 km per year. A recent analysis of the rate of spread around the shores of Lake Victoria from Uganda to Tanzania between 1992 and 2007 demonstrated an average rate of spread of 38 km/yr along the western side and 24 km/yr around the eastern side (Legg, 2010). The consequence of this pattern of spread is that the CMD pandemic has taken a form that can be compared with a ripple effect spreading out from the point of origin. Although this has been a continuous process, a series of stages can be identified (Fig. 1). CMD was largely benign in all cassava-growing areas prior to the late 1980s, following the initial 'first-encounter' epidemics that were widely reported in Madagascar and elsewhere during the 1920s and 1930s (see reviews by Cours et al., 1997; Thresh et al., 1997). Stage 1 of the EACMV-UG associated epidemic corresponds to the period when the problem was confined to Uganda (late 1980s and early 1990s). During the late 1990s (Stage 2 – regional pandemic), the newly-designated pandemic spread beyond the borders of Uganda into neighbouring countries, including: Kenya, Tanzania, Rwanda and the eastern part of the Democratic Republic of Congo (DRC). Finally, in the first decade of the 2000s (Stage 3 – continental pandemic), the CMD pandemic spread over a much greater area to countries distant from Uganda, including: Republic of Congo, Gabon and Angola, although it should be noted that pandemic monitoring in Central Africa has been much less frequent and less intensive than in East Africa. There are several reasons for this, including: the difficulty in accessing much of this vast and largely forested area in view of the virtual absence of motorable roads; insecurity; shortages of trained local personnel; and the lack of adequate funds.

The zone of the recent outbreak of CBSD (south-central Uganda) is several hundred km from the original endemic zone in coastal East Africa (Fig. 2). Shortly after the first reports of CBSD had been registered in Uganda, similar occurrences were noted in both western Kenya (H. Obiero, unpublished data) and north-western Tanzania (S. Jeremiah, unpublished data). Following the first reports of CBSD in the Lake Zone of north-western Tanzania, it quickly became apparent that the incidence and severity of the disease was greatest in Mara Region, on the eastern side of the Lake. Recent survey data obtained from six countries in the Great Lakes region confirmed that incidences of CBSD in 2009 were greatest in southern Uganda and in the Tanzania outbreak zone in Mara Region

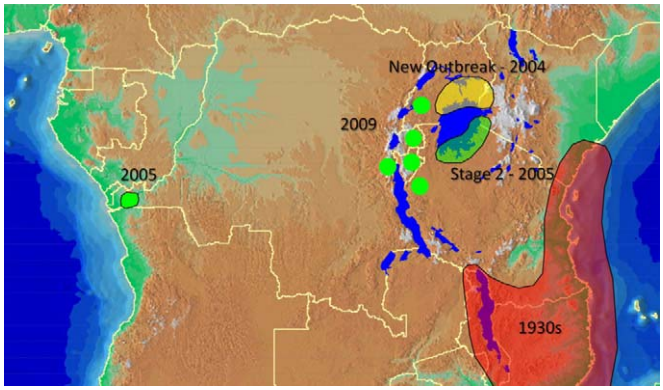

Fig. 2. Distribution of CBSD in East and Central Africa and new outbreak locations. *Red zone*: Endemic Area, affected by CBSD since the 1930s. *Yellow zone*: first 'new' CBSD outbreak reported from southern Uganda in 2004. *Green zones* (stage 2). Subsequent outbreaks along the southern shores of Lake Victoria and in parts of western Democratic Republic of Congo (DRC). DRC report, together with a series of recent ones (green circles) have yet to be confirmed with laboratory diagnostic tests.

(Anon., 2010). Unconfirmed reports of the presence of CBSD have also been made for Burundi, Rwanda and eastern DRC. Available evidence thus suggests that, unlike the CMD pandemic, CBSD is NOT spreading out from an initial infected zone along a contiguous front. By contrast, it has a regional form more like a series of isolated or partially isolated 'hot-spots' that have developed independently. The reason for this is uncertain, but it seems likely that spread has occurred from geographically dispersed 'founder' infections, which may have originated either from CBSV-infected cuttings transported from endemic regions or through natural infections by whiteflies coming from local alternative hosts. Preliminary efforts to detect CBSVs in more than 60 alternative host plants in Uganda have been unsuccessful (G. Okao-Okuja, unpublished data), but more comprehensive studies with a broader geographical coverage will be required before this mechanism can be ruled out as a likely source of new outbreaks.

The path of development of the CMD pandemic from the northern side of Lake Victoria (north-central Uganda) to the south-eastern side in Mara Region, Tanzania, can be traced around both sides of the Lake (Legg, 2010) and took approximately 15 years. This time difference is much greater than that noted between outbreaks of CBSD on the two sides of the Lake, which were separated by only 1–2 years, and provides one of the biggest differences in the epidemiological characters of these two pandemics.

3.2. Similarity 1: pandemic-associated viruses occur outside the pandemic-affected zone

A clear association has been demonstrated repeatedly between the CMD pandemic and the occurrence of EACMV-UG, throughout the pandemic-affected zone (Lava Kumar et al., 2009; Legg et al., 2001; Neuenschwander et al., 2002; Zhou et al., 1997). However, EACMV-UG has also been reported from locations in countries unaffected by the pandemic, including: South Africa (Berry and Rey, 2001), Zimbabwe (Berry and Rey, 2001) and Burkina Faso (Tiendrébéogo et al., 2009). Early reports that discussed the role of EACMV-UG in the CMD pandemic proposed it as a driving factor and a key determinant for the occurrence and spread of the pandemic (Harrison et al., 1997; Pita et al., 2001). The non-pandemic occurrences of EACMV-UG suggest that it would be more appropriate to describe it as a contributory rather than a determining factor.

The genetic diversity of CBSVs is much less well characterized than that of the CMGs. Whilst there are 178 complete DNA-A or DNA-B sequences for CMGs currently held on the Genbank

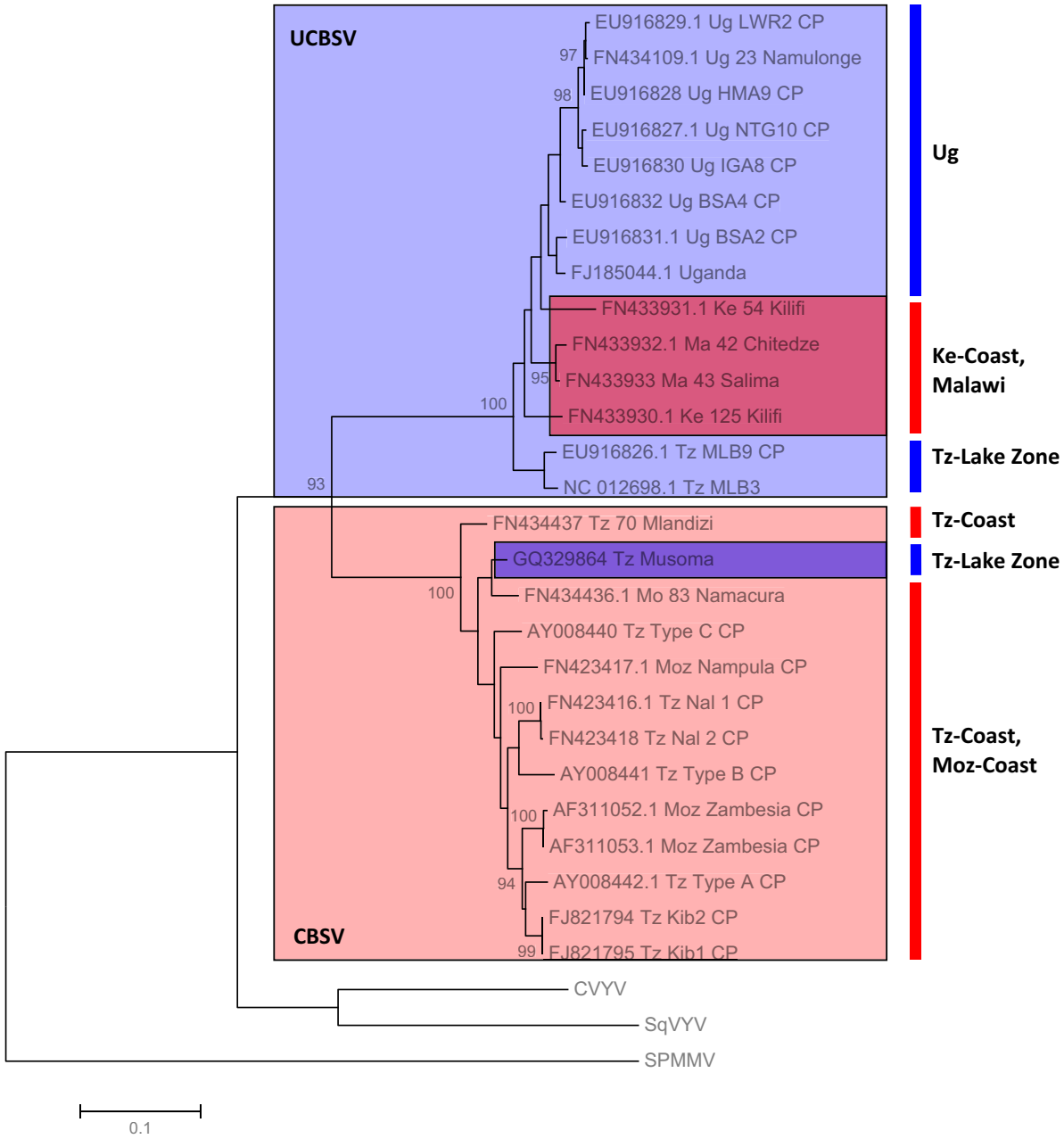

Fig. 3. CBSV coat protein (CP) and full genome sequences available in Genbank. Maximum likelihood analysis with 500 bootstraps. Ke (Kenya), Ug (Uganda) and Moz (Mozambique). Other ipomoviruses (CVYV, SqVYV and SPMMV) included as outgroups.

database, there are only 14 full CBSV sequences (NCBI, 2011). There are, however, a larger number of partial CBSV sequences, and an examination of both full and partial genome datasets reveals that all isolates are grouped into one of two major clades, which comprise the two species groupings: CBSV and UCBSV (Mbanzibwa et al., 2009). If coastal Tanzania, coastal Kenya, Mozambique and Malawi are considered as part of the original epidemic zone of CBSD, and the Great Lakes region as the recent epidemic zone, it is apparent from the distribution of sequences within the two species groups that both CBSV and UCBSV occur in both zones (Fig. 3). Further sequencing will be required to provide a more definitive picture, but there is currently no evidence to support the association of a single species group with the new outbreaks in the Great Lakes region. To the contrary, recent sequence data confirm: the co-occurrence of CBSV and UCBSV in both the Lake Zone of Tanzania and south-central Uganda, the frequent presence of mixed infections, and strain differences between Tanzanian and Ugandan UCBSV isolates (Mbanzibwa et al., 2010). As for the CMGs, it therefore appears that recent outbreaks of CBSD are not uniquely associated with a particular virus species or strain. The corollary of this is that there is some other as yet unidentified factor that is the key driver of the CBSD pandemic.

3.3. *Contrast 2: synergy versus new encounter*

There was a fundamental difference in the health status of cassava encountered by pandemic-associated CMGs and CBSVs during the early stages of pandemic development in East/Central Africa. CMD had been present in Uganda throughout cassava-producing areas ever since the earliest first encounter epidemics of the 1920s (Hall, 1928; Jameson, 1964). These early epidemics represented the first contact between pathogen and host, as CMGs spread into areas where they had never previously occurred. The first spe-

Fig. 4. CBSD symptom expression in leaves and roots: locations in East Africa. (1a/b) Coastal Tanzania; (2a/b) Lake Zone of Tanzania; (3a/b) Coastal Kenya; (4a/b) south-central Uganda.

cific diagnostic tests performed on CMD-infected material from Uganda, using ELISA-based diagnostics, showed the virus present to be ACMV and not EACMV, which was at that time thought to be largely restricted to coastal East Africa (Swanson and Harrison, 1994). It was shown subsequently, however, that the ELISA method did not distinguish between ACMV and EACMV-UG (Harrison et al., 1997), since EACMV-UG was a recombinant with an ACMV-like coat protein. Following the confirmation of the association of the pandemic with EACMV-UG, it was demonstrated by nucleic acid studies that the background pre-pandemic condition was a moderate to low incidence of relatively benign ACMV (Harrison et al., 1997; Legg et al., 2006). The severity of the CMD pandemic condition, however, was not attributable simply to the spread of EACMV-UG, but rather to the fact that EACMV-UG was interacting synergistically with ACMV. Several studies have characterized this phenomenon (Harrison et al., 1997; Pita et al., 2001), and similar synergy has been demonstrated between ACMV and other species of EACMVs (Fondong et al., 2000). In each of these cases, the synergism has been shown to be a result of the complementary function of proteins of ACMV and EACMVs in suppressing the host plant's post transcriptional gene silencing defence response (Vanitharani et al., 2004). The development of the CMD pandemic was facilitated by enhanced CMG concentrations resulting from synergistic interactions, which in turn greatly increased the probability of successful vector transmission.

During the 1940s, CBSD was reported from experimental stations in Uganda in cassava material introduced from the East African Agriculture and Forestry Research Organization's cassava breeding programme, based at Amani, Tanzania (Jameson, 1964). The disease was assumed to have been eradicated, however, and was not seen subsequently until 1994 (Thresh et al., 1994b), albeit at only a single location near Entebbe. Researchers familiar with the symptoms of CBSD were active in much of Uganda from 1994 to 2004, so it must be assumed that CBSVs were either absent, or present at a very low incidence, during this time. The conspicuous outbreak recorded from 2004 onwards should therefore be considered as a 'first encounter' epidemic. In contrast to CMD, new spread of CBSVs in the Great Lakes region appears to be most frequently as single species infections, although more data are required to confirm this. Single species infections of UCBSV predominate in Uganda (Alicai et al., 2007; Mbanzibwa et al., 2009, 2010). In the Lake Zone of Tanzania, mixed infections appear to be more frequent than in Uganda, but they are still less common than UCBSV alone (Mbanzibwa et al., 2010). There are currently no data on interactions between the two species of CBSVs. Although some form of mutualistic interaction may occur, the limited evidence available so far suggests that this is not a key factor in the regional development of the CBSD pandemic.

Differences in symptom expression associated with species and strains of CBSVs have been demonstrated using the herbaceous test plant, *Nicotiana benthamiana*, but such contrasts are less apparent for infections in cassava (Winter et al., 2010). The broad range of symptoms described for CBSD (Nichols, 1950) occurs in both endemic and epidemic zones (Fig. 4). Assessments of varietal response to the two CBSVs have revealed some variability. In the grafting study of Winter et al. (2010), all ten varieties tested were susceptible to infection by CBSV, whereas varieties Albert, Kibaha and TMS 30572 were not infected by UCBSV. There is some evidence for synergy between CBSVs and CMGs in *N. benthamiana* (Ogwok et al., 2010), but field survey data do not support a complementary function of the two virus groups (Anon., 2010) and it is notable that CBSD is most apparent in CMD-resistant varieties (Alicai et al., 2007; Winter et al., 2010), in which CMD is either absent or at very low incidences.

All currently available results therefore suggest that the CBSD pandemic in the Great Lakes region is a 'first encounter' situation and that the outbreaks are the consequence of neither an unusually severe virus variant, nor of synergistic interactions – either between CBSV and UCBSV or between CBSVs and CMGs.

3.4. Contrast 3: 'Tight' versus 'loose' associations between disease and vector

From some of the earliest descriptions of the CMD pandemic, a key feature has been the close association between the 'front' of severe CMD, the presence of EACMV-UG and unusually large populations of the whitefly vector, *B. tabaci* (Legg and Ogwal, 1998; Otim-Nape et al., 1996). The cause of *B. tabaci* super-abundance

Table 1
Approximate years of first reports of a severe form of CMD, EACMV-UG, CBSD and 'abundant' and peak populations of *B. tabaci* for districts/provinces in the Great Lakes region of East/Central Africa.

	Mukono, Uganda	Busia, Kenya	Bukoba, Tanzania	Tarime, Tanzania	Bujumbura, Burundi
Severe CMD	1995	1996	1998	2005	2005
EACMV-UG	1996	1999	1999	2005	2005
CBSD	2004	2009	2008	2007	Not present
Abundant *B. tabaci* year[a]	1995	1997	1998	2004	2005
Peak *B. tabaci* population year[b]	2004	2009	2007	2007	2005
Peak *B. tabaci* population[c]	39(30)	12(15)	35(10)	100(10)	58(12)

Data compiled from published and unpublished survey data, including: Gibson (1996), Otim-Nape et al. (1997), Harrison et al. (1997), Legg (1999), Legg and Okao-Okuja (1999), Legg and Thresh (2000), and Alicai et al. (2007). Unpublished survey data: S. Bigirimana (Burundi: 2003–2007, 2009): H. Obiero (Kenya: 1996–2006, 2009): I. Ndyetabula (Tanzania: 2000–2009).

[a] First year of 'abundant' *B. tabaci*. *B. tabaci* are considered to be abundant where there are >5 adults per top five leaves on the sampled shoot of cassava plants in the District/Province.

[b] Values represent the year in which populations were observed to be highest.

[c] Values represent the average abundance of adult *B. tabaci* (first value) recorded from the number of sites sampled in the district/province (in parentheses).

remains unclear, although possible hypotheses include a genetic change in *B. tabaci* populations resulting in increased fitness (Legg et al., 2002), a mutualistic interaction with CMD-infected cassava plants (Colvin et al., 2006), or a combination of the two. Significantly, the occurrence of synergy between whiteflies and CMD would greatly reinforce the spatial association between the two. The tight link between severe CMD, EACMV-UG and super-abundant *B. tabaci* was sustained as the pandemic spread beyond Uganda to other parts of East and Central Africa. The outbreak of CBSD in the Great Lakes region, by contrast, has mainly developed several years after the spread of the CMD pandemic. It therefore appears that there is a much weaker association between CBSD spread and changes in whitefly abundance than with the CMD pandemic. By summarizing data available from several unpublished sources, it is possible to compare the virus disease/vector association for both severe CMD and CBSD in several districts in cassava virus pandemic-affected parts of the Great Lakes region (Table 1). In all cases, a very strong temporal association is apparent between the first reports of severe CMD, increases in whitefly abundance and confirmations of the presence of EACMV-UG. First reports of CBSD, however, were made from 3 to 12 years after the first occurrences of abundant whiteflies. In many areas where unusual whitefly abundances were observed at the onset of the CMD pandemic, populations have continued to increase subsequently. If the year of super-abundance is defined as the one in which highest populations occurred, a much closer match becomes apparent between whitefly abundance and the emergence of CBSD.

Based on this information a schematic representation is proposed for the spatial and temporal association between *B. tabaci* populations and the cassava viruses they transmit (Fig. 5). For a location in the path of the CMD pandemic, but not yet affected, the starting situation is a low incidence of mild to moderate CMD, caused by ACMV alone, and low populations of *B. tabaci*. Whiteflies reaching the location from neighbouring pandemic-affected areas introduce EACMV-UG and give rise immediately to increased whitefly abundance and high incidences of severe CMD. The inci-

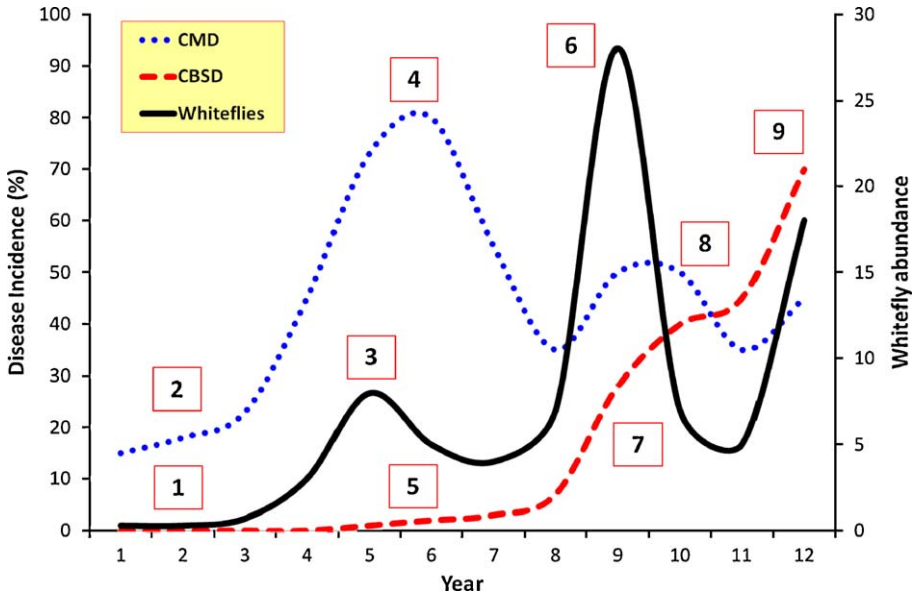

Fig. 5. Diagrammatic representation of temporal association between *B. tabaci* whitefly abundance and incidences of CMD and CBSD at a single hypothetical location in the Great Lakes region. (1) 'Background' level of local *B. tabaci* population. (2) Pre-pandemic 'Background' level of CMD, typically caused by ACMV alone. (3) Increased population of 'abundant' *B. tabaci* resulting from influx of invasive populations and/or synergistic interaction with CMD-infected plants. (4) Raised incidence of severe CMD more-or-less concurrent with increase in whitefly abundance and spread of EACMV-UG and mixed CMG infections. (5) Gradual and initially unnoticed increase in CBSD incidence from very low levels following initial peak in whitefly populations. (6) Favourable environmental conditions allow *B. tabaci* populations to become super-abundant. (7) Rapid increase in CBSD incidence associated with super-abundant *B. tabaci* populations. (8) CMD incidence peaks follow whitefly population peaks, but there is a general decline in incidence resulting from the increased cultivation of CMD-resistant varieties and selection by farmers of moderately CMD-resistant local cultivars. (9) CBSD incidence continues to increase in response to further whitefly population increase. The lack of CBSD-resistant varieties means that CBSD incidence continues to increase, in contrast to CMD.

dence then fluctuates over subsequent years, being enhanced by subsequent whitefly population increases but lowered by disease control interventions, notably the switch to CMD-resistant varieties. CBSD incidence only becomes apparent several years later. This delay is most likely to be a consequence of the combined effect of the negligible baseline incidence of CBSD as well as the probable absence of long distance dissemination of CBSVs by vectors. Furthermore, it is possible that CBSD may not appear at all in situations where there is no local inoculum source. Such a condition might explain the absence of CBSD at Bujumbura in Burundi (Table 1), despite the current presence of super-abundant whitefly populations.

3.5. Contrast 4: vector transmission mechanisms

Vector transmission of CMGs and CBSVs by *B. tabaci* has been demonstrated by several researchers (Chant, 1958; Dubern, 1994; Maruthi et al., 2005; Mware et al., 2009). The characteristics of CMG transmission were described in detail by Dubern (1994). Transmission was shown to be persistent and relatively efficient, as optimal rates of transmission could be achieved using as few as ten whitefly adults per test plant. Moreover, up to 13% of all individual *B. tabaci* adults emerging from infected plants may be infective. A key feature of the persistent transmission mechanism of CMGs is that these viruses are retained for at least 9 days, and may be retained by adult whiteflies throughout their lives (Dubern, 1994). This has important epidemiological implications, as it appears to provide the mechanism by which CMGs are carried long distances by dispersing *B. tabaci*. Long-term virus retention coupled with long range dispersal has been proposed as the basis for the spread of the CMD pandemic up to 100 km per year along the western side of Lake Victoria (Legg et al., 2006; Legg, 2010).

For many years, cassava researchers were unable to identify the vector of CBSVs, although it had been assumed that the whitefly species, *B. tabaci* or *Bemisia afer* (Priesner and Hosny), were the most likely candidates. The question was finally resolved through a combination of insectary and field studies (Maruthi et al., 2005) through which transmission was confirmed for *B. tabaci* but not for *B. after*. This placed CBSV in line with its closest ipomovirus relatives, *Cucumber vein yellowing virus* (CVYV) (Mansour and Al-Musa, 1993), and *Squash vein yellowing virus* (SqVYV) (Adkins et al., 2008), which are both transmitted by *B. tabaci*. Additionally, both CVYV (Harpaz and Cohen, 1965) and SqVYV (Webb et al., 2008) have been shown to be transmitted semi-persistently. The first published confirmation of the transmission of CBSVs by *B. tabaci* (Maruthi et al., 2005) indicated that infection efficiency was 22% when using up to 120 whitefly adults per plant. Preliminary results from more recent studies, however, have given slightly higher efficiency rates (S. Jeremiah, unpublished data), although more than 50 adults per target plant were still required to obtain maximum transmission efficiencies. In addition, preliminary tests of retention periods indicate that CBSVs may not be retained by *B. tabaci* for much longer than 24 h once away from infected host plants (N. M. Maruthi, unpublished data). The overall picture, therefore, suggests that *B. tabaci* whiteflies are less efficient transmitters of CBSVs than CMGs, and that they retain CMGs for much longer periods than CBSVs. Further investigation is required to clarify the precise characteristics of the transmission of CBSVs, but even the current incomplete picture is consistent with the regional epidemiological characteristics of the CMD and CBSD pandemics.

3.6. Contrast 5: different patterns of symptom expression lead to different roles of farmers in spread

As with most viruses of vegetatively propagated crops, CMGs and CBSVs persist from one cropping cycle to the next through the use of infected propagation material when selecting cuttings to plant the next season's crop. Under normal circumstances, farmers do not distinguish between infected and healthy sources of planting material. Similarly, most farmers are also very unlikely to remove infected plants (rogue) from a cassava crop during the course of the growing season. The combined effect of these two missed opportunities for intervention is that incidences of CMD and CBSD are usually maintained from season to season, even where there is little whitefly vectoring activity. This situation may be changed, however, either where symptoms become more severe and limit the availability or suitability of cuttings, as has happened with the CMD pandemic, or where farmers are provided with training both on the symptoms of virus diseases and on the potential benefits of phytosanitary measures, such as selection of disease-free stems for planting material or roguing out infected plants in the early stages of crop development. Consequently, significant levels of roguing have been recorded from countries affected by the CMD pandemic, such as Kenya, where socio-economic surveys of cassava farmers revealed that 38% practise roguing to control CMD (Kamau et al., 2005).

Roguing and selection of healthy stems to reduce CMD incidence can be achieved readily by trained farmers, since only above-ground symptoms are expressed and symptom expression is a reliable indicator of infection (Fargette et al., 1987). However, since external infection sources play the primary role in the infection of initially disease-free crops (Fargette et al., 1985) and whiteflies can carry CMGs over relatively long distances, roguing is often only partially successful, and may be detrimental by decreasing plant density. Severe CMD may be to some extent self-limiting, however, as the most severely affected plants are discarded by farmers and their stems rejected for use as planting material to establish a new crop. With CBSD, phytosanitary measures are much harder to implement, both since symptoms of infected plants can be hard to recognize, even for trained agricultural workers, and also since there is a weaker association between infection and symptom expression than for CMD/CMGs. Paradoxically, however, the benefits of phytosanitary measures for CBSD are likely to be significantly greater than for CMD. Since CBSVs are carried by whiteflies for much shorter distances than CMGs, as a result of the semi-persistent transmission mode, the threat of infection from distant inoculum sources is much less than it is for CMD. The phytosanitary measures of roguing and healthy planting material selection are therefore more likely to be of direct benefit, as is the planting of new crops away from neighbouring cassava fields.

Since whiteflies appear to play a lesser role in the regional epidemiology of CBSD than they do for CMD, the corollary is that farmers play a greater role in aiding the spread of CBSD than they do for CMD. Cryptic symptoms mean inevitably that CBSD is more likely than CMD to be disseminated in infected cassava stems and less likely to be recognized when cuttings sprout at the new location. This fact highlights the need for special attention to be given to the development and implementation of strict measures of phytosanitation, including quarantine to control movements of cassava planting material. This is particularly critical for countries in which some areas are affected by CBSD whilst others are not, as well as for countries neighbouring CBSD-affected countries.

4. Pandemic management

Although CMGs and CBSVs both depend on cultivated cassava as their main host, in most other respects, the epidemiology of these two groups of viruses contrasts greatly. As begomoviruses, the CMGs are part of one of the most successful and diverse groups of plant viruses, and CMGs are distributed throughout Africa and parts of South Asia (Legg and Fauquet, 2004; Patil and Fauquet,

2009). They have evolved an intimate relationship with their whitefly vector that has facilitated the colonization of virtually all cassava plantings on the African continent. Furthermore, recombination and inter-species synergism has allowed CMGs to exploit their interactions with whiteflies to an even greater degree, and has been an important contributory factor to the CMD pandemic that has affected large areas of East and Central Africa in recent decades. By contrast, CBSVs as ipomoviruses are members of a diverse but very small group of viruses and have been confined for much of their history to a relatively small area of coastal East Africa and around the shores of Lake Malawi (Hillocks and Jennings, 2003; Nichols, 1950). During the early years of research into CBSD, infected cassava cuttings were intentionally carried to mid-altitude locations in order to assess symptom expression and determine the reasons for the lack of CBSD spread at such elevations (Nichols, 1950). Whilst plants growing at these locations expressed clear symptoms of CBSD, no plant-to-plant infection was recorded and it was concluded that conditions at these altitudes were 'inimical' to CBSD spread. The rapid spread of CBSD through parts of the Great Lakes region of East and Central Africa from 2004 onwards (Alicai et al., 2007) has clearly demonstrated that there was nothing inherent in these mid-altitude environments that precluded CBSD spread. Rather, abundant whitefly populations, which are a critical determining factor for spread and which had been absent previously, were now present. Numbers of *B. tabaci* have increased more than 100-fold in recent years in many parts of East and Central Africa (Legg et al., 2002). Not only has this whitefly been conclusively proven as the vector of CBSVs (Maruthi et al., 2005; Mware et al., 2009), but new data on the mechanism of transmission suggest that CBSVs, like other ipomoviruses, are transmitted semi-persistently. This mode of transmission is consistent with empirical data from the field which indicate that CBSVs may spread rapidly from local infection sources, but are not carried over long distances as the CMGs.

The contrasting patterns of spread exhibited by viruses of these two groups, and the two pandemics that they are causing, mean that very different management strategies are required. These can be summarized as follows:

4.1. Host plant resistance

CMD. High levels of resistance to all CMGs have been developed through conventional breeding programmes using two different sources of resistance (Dixon et al., 2003; Hahn et al., 1980) and resistant varieties have been widely deployed in CMD management programmes, both for early 'first-encounter' epidemics (Cours, 1951; Cours et al., 1997), as well as more recently for CMD pandemic mitigation (Legg et al., 1999; Otim-Nape et al., 1997). Conventional host plant resistance is the most widely practised control measure for CMD. Although transgenic strategies have been developed (Chellappan et al., 2004; Zhang et al., 2005), they have yet to be deployed, and their current value may be limited in view of the high levels of resistance already available.

CMSD. All cassava varieties tested so far have been shown to be susceptible to one or both CBSVs (Winter et al., 2010). Field evidence indicates that some varieties show 'tolerance' through reduced and/or delayed incidence of root symptoms (Hillocks et al., 2001; Hillocks and Jennings, 2003). Although several recent studies have been conducted to investigate resistance to CBSD (Mtunda, 2009; Munga, 2008), none has been able to identify the high levels of virus resistance that have been produced for CMGs. As potyviruses, CBSVs may be more suitable than CMGs for the development of transgene-based control techniques, and testing of target strategies has been initiated (Patil et al., 2010). Future studies should focus on incorporating transgenes conferring robust CBSD-resistance into conventionally bred CMD-resistant lines. In addition to having the desired agronomic traits, these lines should also preferably have resistance to *Bemisia* whiteflies. Although effective sources of resistance have been identified for non-*Bemisia* whitefly species in Latin America (Bellotti and Arias, 2001), preliminary results suggest that these are less effective against African *B. tabaci* (J. Colvin, unpublished data) and further research into alternative sources of resistance to *B. tabaci* is required.

4.2. Phytosanitation

CMD. Roguing and selection of disease-free stems for planting are feasible and have been recommended for CMD (Bock, 1994; Jameson, 1964; Thresh et al., 1998). However, such phytosanitation measures are seldom adopted, in part because of the ease with which initially CMD-free plantings may become infected from external inoculum sources. Simplicity of symptom recognition, however, makes the application of phytosanitation measures relatively straightforward at official institutional sites producing propagation material.

CBSD. Selection of disease-free stems and roguing are difficult to implement for CBSD in view of the difficulty of accurate symptom recognition, although this problem can be partly addressed through staff training. Since CBSD spreads over shorter distances than CMD, local phytosanitation has the potential to provide much more effective control than is achievable for CMD. Isolation of propagation sites, from other potentially CBSD-infected neighbouring cassava fields is likely to be of great benefit. Since symptoms are often cryptic or unclear it is vital to ensure that starting material for cutting multiplication schemes is virus-free. In order to achieve this, systematic virus-testing programmes are recommended for primary multiplication sites (Abarshi et al., 2010). In view of the great risk that CBSVs pose to cassava-growing countries currently unaffected by CBSD, virus indexing should be an essential quarantine requirement for cassava germplasm exchange within and between regions and countries, and such transfers should only be made in indexed tissue culture form (Frison, 1994).

4.3. Monitoring and forecasting

CMD. Monitoring surveys have provided a detailed picture of the progress of the CMD pandemic through East Africa (Legg et al., 2006; Sseruwagi et al., 2004). Based on the relative predictability of the pattern of CMD pandemic expansion, it has also been possible to forecast future patterns of disease development and assess levels of risk to regions near to pandemic-affected regions (Legg et al., 2006). This work has greatly aided the targeting of control interventions and will continue to be valuable for this purpose.

CBSD. Limited data are available on the distribution and importance of CBSD as relatively few surveys have considered this disease. Recent data obtained, however, reveal a pattern of epidemic development that is much less structured and predictable than that of the CMD pandemic. Consequently, it is not yet possible to make predictions of the likely future pattern of CBSD spread. It is anticipated that this situation may change, however, as more detailed knowledge becomes available on the local and regional epidemiology of CBSD. Generating this knowledge should be an important future research target.

5. Conclusion

The management of the current pandemic of CMD covering more than 4 million sq. km in 12 countries has presented a major challenge for two decades to all concerned with cassava in Africa. Accordingly, concerted efforts have been made to mitigate this situation by a broad range of stakeholders, including both national and international research institutes, as well as development partners.

The subsequent emergence of the CBSD pandemic, and the lack of effective resistance to this disease in cultivated cassava, has exacerbated an already critical threat to Africa's most important food security crop. In view of the scale of existing damage and future threats posed by these pandemics, it is essential to identify and then implement effective management strategies for both diseases. Recognizing the important contrasts between them will greatly aid the development of effective strategies. A critical difference in management tactics for the two diseases is the high level of robust resistance available for CMD, contrasting with the virtual absence of effective resistance for CBSD. This highlights the importance of investing in research to develop host plant resistance to CBSD that is of comparable effectiveness to that currently available for CMD. The chances of achieving this will be greatest if both conventional and transgenic approaches are explored. Equally important, is the recognition of the vital common factor that underpins the 'success' of both the CMD and CBSD pandemics, namely the whitefly vector. Long-term sustainable control of the two diseases will depend on the development and dissemination of effective control methods for this highly adaptable insect. Consequently, it is suggested that a much higher priority should be accorded to vector management by all stakeholders during the development of future plans to mitigate the dual cassava virus pandemics. Improved whitefly management will not only provide a solution to both current pandemics, but in addition, will significantly reduce the likelihood for the emergence of new epidemics caused by variant strains or novel virus species.

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