Chapter 12

# The Viruses and Virus Diseases of Cassava

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## Introduction

Crops that are propagated vegetatively are particularly prone to damage by viruses as infection tends to build up in successive cycles of propagation. Cassava is no exception to this generalization and at least 16 different viruses have been isolated from the crop. Moreover, other as yet undescribed viruses are likely to occur and may even be prevalent in some areas. This is because cassava has received far less attention from virologists than it merits as one of the world's most important and widely grown food crops.

A full list of the viruses that have been isolated from cassava is presented in Table 12.1 and key references appear in the bibliography. The viruses asterisked in the table have been detected somewhat fortuitously in studies undertaken for other reasons. There is only limited information on the properties, distribution, effects and importance of these viruses. They require further attention, but meanwhile they should be considered in operating quarantine controls on the movement of vegetative propagules between different cassava-growing areas. These viruses are not considered further here and the main emphasis is on those known to cause diseases of economic importance.

A feature of cassava viruses is that they are of diverse taxonomic groups (Table 12.1). Another is that their known distribution is largely or entirely restricted to only one of the continents in which cassava is grown, or to an even more localized geographic area. For this reason the viruses and virus diseases of Africa, South/Central America and the Indian subcontinent are considered separately.

## The Viruses and Virus Diseases of Cassava in South and Central America

Cassava originated in the Neotropics and was not introduced to other regions until relatively recently. This may explain why only one of the viruses of cassava occuring in South and Central America has been found elsewhere. Moreover, several of the Neotropical viruses of cassava do not cause symptoms and have no obvious deleterious effects, which may reflect a long period of co-evolution between the host and its pathogens.

Three virus diseases justify detailed attention here. Three other viruses are listed in Table 12.1 and are considered briefly, but they do not

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**Table 12.1.** The viruses of cassava.

| |
|---|
| Africa |
| *African cassava mosaic virus* (*Geminiviridae: Begomovirus*) |
| *East African cassava mosaic virus* (*Geminiviridae: Begomovirus)* |
| *South African cassava mosaic virus* (*Geminiviridae: Begomovirus*) |
| *Cassava brown streak virus* (*Potyviridae: Ipomovirus*) |
| *Cassava Ivorian bacilliform virus** (unassigned) |
| Cassava Kumi viruses* |
| Cassava 'Q' virus* |
| *Cassava common mosaic virus** (*Potexvirus*) |
| South/Central America |
| *Cassava common mosaic virus* (*Potexvirus*) |
| *Cassava virus X* (*Potexvirus*)* |
| *Cassava vein mosaic virus* (*Caulimoviridae*) |
| *Cassava Colombian symptomless virus* (*Potexvirus*)* |
| *Cassava American latent virus* (*Comoviridae: Nepovirus*)* |
| Cassava frogskin 'virus' |
| Asia/Pacific |
| *Cassava common mosaic virus** (*Potexvirus*) |
| *Indian cassava mosaic virus* (*Geminiviridae: Begomovirus*) |
| *Cassava green mottle virus** (*Comoviridae: Nepovirus*) |

Officially recognized viruses are given in italics, together with family and genus in parentheses. Viruses that are unimportant or for which little information is available are asterisked and not considered in detail in the accompanying text. (Source: Thresh *et al.*, 1994b; Thresh *et al.*, 1998c.)

cause symptoms, appear to be unimportant and are not discussed further.

## Cassava common mosaic disease

### *History*

Cassava common mosaic disease (CsCMD) was first reported in southern Brazil (Silberschmid, 1938; Costa, 1940). The disease has since been recorded in other South American countries and there is one report from Africa (Aiton *et al.*, 1988) and another from Asia (Chen *et al.*, 1981). CsCMD has no known vector and spread in the field is attributed to mechanical transmission. The disease is generally of only minor importance, although there are some areas where it is prevalent and control efforts are needed.

### *Symptoms*

Leaves of cassava plants affected by CsCMD develop mosaic and chlorotic symptoms (Plate 1a). On some of the affected leaves there are dark and light green patches that are delimited by veins. Symptoms are most severe during relatively cool periods and cassava grown in the semitropical areas of South America is most affected by the disease. In these relatively cool conditions, the affected plants are sometimes stunted and yield losses can be up to 60% (Costa and Kitajima, 1972b).

### *Distribution and prevalence*

CsCMD has been reported from many South American countries, but was not recorded in a survey of cassava-growing areas of Colombia (Nolt *et al.*, 1992). The disease is most prevalent in southern Brazil and Paraguay. In these regions the disease is important and phytosanitary control measures are recommended to reduce losses. More than 1000 cassava accessions in the EMBRAPA/CNPMF (Empresa Brasileira de Pesquisa Agropecuaria/Centro Nacional de Pesquisa en Mandioca y Fruticultura) collection at Cruz das Almas in Bahia, northeast Brazil, have been tested for the causal virus and the incidence was < 1%.

### *Aetiology*

CsCMD is caused by *Cassava common mosaic virus* (CsCMV) which can infect species belonging to

several families of dicotyledonous plants (Silva *et al.*, 1963; Kitajima *et al.*, 1965). The virus was ascribed originally to the potexvirus group that is now referred to as the genus *Potexvirus*.

The CsCMV virion is a semi-flexuous rod that is *c.* 15 × 495 nm (Kitajima *et al.*, 1965) and contains RNA (Silva *et al.*, 1963). Nuclear inclusions typical of the potexviruses can be found in cassava and the herbaceous host *Nicotiana benthamiana*. CsCMV is known to systemically infect cassava, *Euphorbia* spp., *Cnidoscolus aconitifolius* (chaya), *N. benthamiana* and species of several other dicotyledonous families (Costa and Kitajima, 1972a).

The viral particles of CsCMV contain a single coat protein having a relative molecular weight of 26,000 daltons (Nolt *et al.*, 1991). The CsCMV genome is single-stranded RNA and the complete sequence is known (Calvert *et al.*, 1996). The organizational structure, proteins and their predicted weights are similar to those of other potexviruses.

### *Epidemiology and control*

There are no known vectors of CsCMV and the primary source of inoculum is infected planting material. The virus is systemic in cassava and almost all stem cuttings are infected when obtained from an infected plant. CsCMV is very stable and can be spread by mechanical transmission on machetes and other implements used to prepare cuttings. Although this mode of transmission is inefficient, it is the only known means of plant-to-plant spread.

Eliminating (roguing) plants that express CsCMV symptoms provides adequate control. The symptoms are usually obvious on the first leaves produced by infected stem cuttings. This is the best time to identify and remove diseased plants. If the plants are not rogued early they should be marked and the stems burned later after harvesting the tuberous roots. Only healthy plants should be selected as a source of vegetative propagules. To minimize the risk of mechanical transmission, cutting tools should be disinfected at regular intervals (Lozano and Nolt, 1989). With care in selecting planting material, CsCMD can be eradicated or reduced to a level of minor economic significance.

## Cassava vein mosaic disease

### *History*

The first report of cassava vein mosaic disease (CVMD) was in 1940 (Costa, 1940). The areas where this disease is most prevalent are remote and the conditions are semiarid. The region is inhabited mostly by poor rural communities and the lack of economic resources has contributed to the incomplete knowledge about this disease. Probably because the symptoms are sporadic and generally less apparent at the end of the cassava growth cycle, this disease has received inadequate attention, especially considering the large area now known to be affected.

### *Symptoms*

The leaf symptoms of CVMD occur in flushes. After an infected stem cutting sprouts, the first four to six leaves express vein chlorosis that appears as a chevron pattern or coalesces to form ringspots (Plate 1b). Leaf deformation and epinasty are common severe symptoms. Plants then appear to 'grow out' of the infection and produce several symptomless leaves. These are followed by another series of leaves with symptoms. The expression of symptoms is influenced by the climatic conditions prevailing. Symptoms are more pronounced in the semiarid areas as compared to those expressed by the same variety grown in the wetter coastal regions of northeast Brazil. Except for the period just after sprouting, CVMD does not seem to affect plant vigour. The affected leaves senesce and fall prematurely from the plants which reduces leaf area. As infected cassava matures, it is often difficult to see any leaves with mosaic symptoms.

### *Distribution and prevalence*

CVMD is very common in the semiarid zone of northeastern Brazil, although there are also reports from other regions of the country. The disease is common in the Brazilian States of Ceará, Pernambuco, Alagoas, Piaui and Bahia (Calvert *et al.*, 1995) and the distribution extends into some of the neighbouring states.

### *Aetiology*

CVMD is caused by *Cassava vein mosaic virus* (CVMV). This has isometric particles, *c.* 50 nm in diameter (Kitajima and Costa, 1966) and the genome consists of ds-DNA, *c.* 8200 bases long. Initially CVMV was regarded as a tentative member of the caulimovirus group. This attribution was based on the ds-DNA genome and the particle structure (Lin and Kitajima, 1980). The complete sequence of CVMV has been determined and the genomic organization differs from that of caulimoviruses and badnaviruses (Calvert *et al.*, 1995). The virus will probably be classified as a unique genus of the plant pararetroviruses.

### *Epidemiology and control*

Little is known about the epidemiology or control of CVMV. The only known host is cassava and the primary mode of dissemination is in infected propagules. It is not uncommon to find a farmer's variety that is totally infected. Spread occurs within fields which suggests that there is a vector, but none has yet been identified. There have been few studies on virus spread, but CVMV-infected cassava is common throughout a large area of the northeastern semiarid region of Brazil. Consequently, a vector of the virus is suspected. Until more is known about the rate of spread, the effectiveness of using 'clean' (virus-free) planting material will not be known. The virus can be latent in plants, especially during the cool, rainy seasons of the coastal regions of Brazil. 'Roguing' of planting material may be an effective control practice if diseased plants are identified and removed soon after sprouting. Most infected cassava plants appear to tolerate CVMV and produce stems of normal appearance that make good planting material. Little is known about disease loss. In the few studies that have been done, the yields of diseased plants were slightly less than from uninfected controls, but the differences were not significant statistically (Santos *et al.*, 1995). Although the full economic importance of CVMD is not well quantified, it appears that it could cause losses, especially if a drought occurs during the beginning of the growth cycle.

## Cassava frogskin disease

### *History*

Cassava frogskin disease (CFSD) is a virus-like disease that affects cassava and was first reported from southern Colombia (Pineda *et al.*, 1983). A similar disorder in northern Colombia was called Caribbean mosaic disease because of the mosaic leaf symptoms expressed by the cassava landrace 'Secundina'. In the Amazon regions of Brazil and Colombia, CFSD is called *jacare* (cayman), because of the distinctive ridges on the affected roots. Tests under uniform conditions have shown that these three disorders are manifestations of the same disease and cause the same symptoms in standard indicator varieties of cassava.

The origin of CFSD is most probably the Amazon region of Colombia, Peru or Brazil. The disease can be found in cassava grown in very isolated indigenous Amazonian Indian communities. They regard it as a physiological disorder rather than a disease and associate it with particular varieties. The native name for one variety collected from the Amazonian region of Colombia is *jacare*. Because of the geographic isolation, or the belief that the root symptoms are caused by a physiological disorder, this disease was not 'discovered' in the lowland tropics. In 1971, an apparently new disease that caused severe losses occurred in the mid-altitude Andean mountains of southern Colombia. The disorder was then recognized by scientists and named cassava frogskin disease (Hernández *et al.*, 1975).

The distribution of frogskin disease is continuing to expand. By the 1980s, it was prevalent throughout most cassava growing regions of Colombia. It has also spread to Venezuela and Costa Rica. Recently, CFSD was reported in Panama and it was established that the affected plants were grown from stem cuttings imported from Costa Rica. In Brazil, the movement of vegetative material of cassava is disseminating the disease from the Amazon region into the more semiarid areas of northeast of Brazil. CFSD also occurs in the Amazon region of Peru.

### *Symptoms*

The expression of CFSD symptoms is influenced by temperature and host genotype. A few

cassava genotypes develop mosaic symptoms on the leaves and these clones can be severely stunted. In most other genotypes, the leaves of infected plants are symptomless and appear normal. The stems of these plants may be slightly enlarged, especially near the ground. The thickening of the affected stems is associated with a lack of starch accumulation in the roots. Because of their apparent vigour, these stems are selected preferentially by farmers as they seem to provide very desirable planting material. The root symptoms range from very mild to very severe. The severity of the symptoms depends on the age of the roots and climatic factors. Hot dry conditions tend to inhibit symptom development, whereas cooler temperatures enhance symptoms. In the lowland tropics, years with above average rainfall tend to be cooler than usual and the symptoms are more severe. In hot dry years, CFSD-infected plants have few if any symptoms. The characteristic root symptoms of surface ridges develop when the root periderm and corky layers enlarge to form raised, lip-shaped fissures (Plate 1c). Severely affected roots do not accumulate starch and often show zones of constrictions. Root symptoms are most severe in plants raised from CFSD-affected stem cuttings. Newly infected plants usually have mild or no symptoms unless infected at an early stage of growth.

### *Aetiology*

The causal agent of FSD has not been proven definitively, although isometric virus-like particles 70–80 nm in diameter can be found in thin sections of the leaves, petioles, stems and roots of affected plants, whatever the source. Viroplasm-like bodies are also found in leaves of infected plants.

At least nine species of ds-RNA are associated consistently with infected plants (Cuervo, 1989). The symptoms of hyperplasia in the root cortex are similar to the tumours caused by other plant reoviruses. The particle morphology, ds-RNA pattern and root symptoms are consistent with the causal agent being a reovirus.

CFSD is readily transmitted through grafts. To detect the disease and certify the status of a plant with regard to CFSD, a stem cutting of the plant to be tested is grafted to a plant of the indicator variety Secundina (CIAT accession M Col 2063; Calvert, 1994). The test plant is used as the rootstock and the buds of the rootstock stem should be removed to increase the likelihood of successful grafts. Plants should be grown in an area where temperatures are normally below 30°C to ensure optimum symptom expression. After 3 or 4 weeks, plants are checked, and any mosaic symptoms on the leaves of the scion indicate that the plant is affected by CFSD. The disease can be eliminated from infected plants by thermotherapy and meristem culture *in vitro* (Maffla *et al.*, 1984).

### *Epidemiology and control*

Several studies indicate that the whitefly *Bemisia tuberculata* is the vector of CFSD (Angel *et al.*, 1990; Velasquez, 1991), although the efficiency of transmission seems to be low. In the field, the disease spreads very slowly, but progressively. In one trial to assess the rate of spread, the incidence of infected plants eventually exceeded 10%, but only after three crop cycles. The amount of spread increased as the incidence of infected plants increased.

The initial dissemination of CFSD is through the use of infected stem cuttings and spread within plantings is attributed to *B. tuberculata*. Most cassava varieties infected with CFSD express no leaf or stem symptoms and when harvesting the crop, farmers usually remove the stems before harvesting the roots. Since the stems of the diseased plants are often thicker than those of healthy ones diseased plants are often selected to provide propagules.

CFSD can be controlled by rigorous selection. Roots should be inspected for symptoms at harvest and only cuttings from apparently healthy plants that bear normal roots should be selected. This is usually adequate to maintain the disease at low levels that cause little economic loss. When the incidence of CFSD has become substantial, it is advisable to collect propagation material from a less affected source. In areas where cassava is harvested mechanically and it is not possible to inspect the roots, the use of stem cuttings from plants that are inspected and certified as being free of CFSD is very effective in controlling the disease.

### Viruses that infect cassava but are not known to cause disease

*Cassava virus X* (CsVX) and *Cassava Colombian symptomless virus* (CCSpV) are other potexviruses that infect cassava (Lennon *et al.*, 1986b). They have only been detected in Colombia, but little effort has been made to determine if they occur elsewhere. CsVX was not detected in tests on over 1000 entries in the cassava germplasm collection of CNPMF/EMBRAPA (Cruz das Almas, Bahia, Brazil). There is only one report of *Cassava American latent virus* and little is known of its distribution (Fargette *et al.*, 1991). Since these three viruses do not cause symptoms it is difficult to determine their distribution or to evaluate their importance. The FAO/IPGRI guidelines for the safe movement of cassava germplasm (Frison and Feliu, 1991) provide additional information on these viruses.

## The Viruses and Virus Diseases of Cassava in Africa

Nine viruses have been isolated from cassava in Africa (Table 12.1), but of these only *Cassava common mosaic virus* (CCMV) has been detected elsewhere. This is consistent with the view that the viruses of cassava in Africa are mainly indigenous ones that infect the crop as a consequence of spread from other hosts some time after cassava was introduced from the Neotropics in the 16th and 18th centuries.

CCMV has been detected only once in Africa (Aiton *et al.*, 1988), in material assumed to have been introduced from South America, where the virus is prevalent (see previous section). There is only very incomplete information on the occurrence and effects of four of the other viruses reported in Africa and only those causing cassava mosaic and cassava brown streak diseases are considered in detail here.

### Cassava mosaic disease

#### *History*

The symptoms of what is now known as cassava mosaic disease (CMD) were first reported more than 100 years ago in what is now Tanzania (Warburg, 1894). The disease was later identified in many other countries of sub-Saharan Africa during the early decades of the 20th century. It was particularly prevalent in Gold Coast (now Ghana), Nigeria, Cameroon, Madagascar and several of the former French Colonial territories of West and Central Africa. This led to studies on the means of spread and control. It also became apparent that some varieties of cassava were less affected by CMD than others and resistance breeding programmes began in the 1930s or 1940s in Madagascar, Tanzania and elsewhere.

In recent decades there have been major projects on the aetiology, epidemiology and control of CMD in Nigeria, Kenya, Ivory Coast and most recently in Uganda. The project in Uganda followed the onset of a particularly damaging epidemic in the late 1980s that is now affecting parts of Kenya, Tanzania and Rwanda and threatens other countries of the region (Otim-Nape *et al.*, 2000). The epidemic is the latest and most fully documented of those to have affected cassava in Africa at different times and places during the 20th century. This explains why CMD has featured so prominently and for so long in the literature on cassava in Africa. Indeed, CMD has received more attention than any other disease of an African food crop (Thresh, 1991).

#### *Symptoms*

CMD causes characteristic leaf symptoms that can usually be recognized without difficulty. The symptoms are very variable in type and severity and are of two main types that are sometimes distinguished as 'green mosaic' and 'yellow mosaic'. Leaves affected by 'green mosaic' have contrasting sectors of normal green and light green tissue. These symptoms are apparent only when the plants are examined closely and are not usually associated with an obvious decrease in leaf area, leaf number or plant size, or yield.

Leaves affected by 'yellow mosaic' are much more obvious, as they have contrasting areas of normal green and yellow tissue. Moreover, the chlorotic areas may expand less than other parts of the leaf lamina which can lead to distortion of the leaflets (Plate 1d) and rupturing of the tissues. Severe chlorosis is often associated with premature leaf abscission, a characteristic

S-shaped curvature of the petiole and an obvious decrease in growth and yield.

There are big differences between cassava varieties in the type, extent and severity of the symptoms caused by CMD and resistant varieties express much less severe symptoms than susceptible ones, especially during the late stage of crop growth when resistant varieties may become symptomless and are then said to recover. Symptom expression is also influenced by environmental factors and leaves produced during hot weather tend to be affected less than those produced at other times. Moreover, virulent strains cause more severe symptoms than avirulent ones and have greater effects on growth and yield.

There is no evidence of any consistent differences between the symptoms caused by the different cassava mosaic geminiviruses (CMGs), each of which can occur as virulent or less virulent strains. However, dual infection with two different CMGs causes more severe symptoms than either virus alone, as reported in studies in Uganda and Cameroon (Harrison *et al.*, 1997; Fondong *et al.*, 2000).

The main difficulties that arise in recording the symptoms of CMD occur when the plants being examined have been affected by pests or nutrient deficiency. The cassava green mite (*Mononychellus tanajoa*) and zinc deficiency cause particular problems. However, the damage they cause is usually similar on the different leaflets of each affected leaf, whereas CMD has less consistent effects and the two halves of a leaflet on either side of the midrib are often affected differently. This is an important distinguishing feature of CMD that should be stressed in training staff and farmers in disease recognition. However, severely damaged plants cannot be examined effectively for virus symptoms and whenever possible inspection for CMD should be made at times when the plants are growing vigorously and unaffected by drought, pests or nutrient deficiency.

In recording experiments and in screening for resistance to CMD, much use has been made of simple numerical scoring systems based on the extent and severity of the symptoms expressed. Scales of 0–4 or 1–5 have been widely used to quantify differences due to variety, season and virus strains and to assess the relationship between symptom severity and yield loss.

### *Distribution and prevalence*

CMD occurs in all the cassava-growing areas of Africa and on the adjacent islands including Cape Verde, Zanzibar, Seychelles, Mauritius and Madagascar. There are big differences between countries in the date of the first reports (Fauquet and Fargette, 1990), which is in part related to the status of cassava in the different parts of Africa and to the amount of attention given to the crop by plant pathologists.

In many African countries there is general agreement that CMD is the most important disease of cassava (Geddes, 1990), although in some areas it is regarded as less important than cassava bacterial blight (see chapter 13). Until recently there were few data to support these assumptions. The situation changed in the 1990s when the incidence and severity of CMD were assessed in representative plantings in 13 important cassava-growing countries of Africa (Table 12.2). Surveys of this type are expensive and time-consuming and inevitably the number of plantings assessed has been small in relation to the total amount of cassava being grown. Nevertheless, surveys were undertaken in Uganda following the onset of the recent pandemic and in several other countries as part of more comprehensive assessments of pest and disease problems. The results summarized in Table 12.2 indicate the prevalence of CMD and the sometimes big differences that occur between and within particular countries.

From the results obtained, three contrasting situations have been distinguished and referred to as *epidemic*, *endemic* and *benign* (Thresh *et al.*, 1997). In the *epidemic* situation CMD is being spread very rapidly by the whitefly vector (*Bemisia tabaci*) and the symptoms are prevalent and severe. Farmers experience such serious losses that food security is threatened and it may be necessary to switch to sweet potato or other alternative food crops. Control measures are essential if production is to be restored and there is an urgent need for CMD-resistant varieties of cassava, as developed and supplied through official programmes or selected by farmers from those already available. The epidemic situation, as encountered in the 1990s in much of Uganda, has now spread to adjacent areas of western Kenya, Tanzania and Rwanda and it seems inevitable that it will soon spread to Burundi

**Table 12.2.** Surveys of the incidence of cassava mosaic disease (CMD) in India and 13 African countries.

| Country | Organization (reference) | Year | Cassava area (million ha) | CMD % iIncidence |
|---|---|---|---|---|
| Uganda | NARO (Otim-Nape *et al*., 1998b) | 1990–1992 | 0.36 | 57 |
| Uganda | NARO (Otim-Nape *et al*., 2001) | 1994 | 0.38 | 65 |
| Uganda | NARO/ESARC (Legg *et al*., 1999) | 1997 | 0.34 | 68 |
| Chad | US AID (Johnson, 1992) | 1992 | 0.07 | 40 |
| Malawi | NARS (Nyirenda *et al*., 1993) | 1992 | 0.07 | 21 |
| Tanzania | NARS/NRI (Legg and Raya, 1998) | 1993 | 0.69 | 26 |
| Ghana | ESCaPP (Yaninek *et al*., 1994; Wydra and Msikita, 1998) | 1993/94 | 0.61 | 72 |
| Benin | ESCaPP (Yaninek *et al*., 1994; Wydra and Msikita, 1998) | 1994 | 0.14 | 53 |
| Cameroon | ESCaPP (Yaninek *et al*., 1994; Wydra and Msikita, 1998) | 1994 | 0.08 | 67 |
| Nigeria | IITA (L.C. Dempster, unpublished) | 1994 | 2.00 | 55 |
| Nigeria | ESCaPP (Yaninek *et al*., 1994; Wydra and Msikita, 1998) | 1994 | 2.00 | 82 |
| Zambia | NARS/SARRNET (Muimba-Kankolongo *et al*. 1997) | 1995/96 | 0.11 | 41 |
| Zanzibar | NARS/NRI (Thresh and Mbwana, 1998) | 1998 | NA | 71 |
| South Africa | NARS (Jericho *et al*., 1999) | 1998 | < 0.01 | 31 |
| Mozambique | NARS/NRI (R.J. Hillocks and J.M. Thresh, unpublished) | 1999/00 | 0.99 | 20 |
| Kenya (Western) | KARI/NRI (Legg *et al*., 1999) | 1993 | < 0.01 | 20 |
| Kenya (Western) | KARI/NRI (Legg *et al*., 1999) | 1996 | < 0.01 | 56 |
| Kenya (Western) | KARI/ESARC (Legg *et al*., 1999) | 1998 | < 0.01 | 84 |
| Kenya (Coastal) | NARS/NRI (T. Munga and J.M. Thresh, unpublished) | 2000 | < 0.01 | 58 |
| India | UAS Bangalore (Mathew, 1989) | 1988 | 0.24 | 19 |
| A. Pradesh | UAS Bangalore (Mathew, 1989) | 1988 | NA | < 1 |
| Karnataka | UAS Bangalore (Mathew, 1989) | 1988 | NA | 5 |
| Kerala | UAS Bangalore (Mathew, 1989) | 1988 | NA | 23 |
| Tamil N. | UAS Bangalore (Mathew, 1989) | 1988 | NA | 30 |

and other parts of the region (Otim-Nape *et al*., 2000) and beyond. Similarly unstable epidemic situations were encountered previously in the 1930s in Madagascar (Cours *et al*., 1997) and more recently in the Cape Verde Islands and Akwa Ibom State of Nigeria (Anon., 1993).

In *endemic* areas there is a high incidence of CMD, but the symptoms are not usually very severe. The overall situation is stable and changes little from one year to the next. There is much use of infected cuttings as planting material and yields are undoubtedly impaired. Nevertheless, the losses have seldom been quantified and they are largely ignored by farmers or considered acceptable. Control measures are not regarded as essential, although they would undoubtedly bring substantial benefits. This is the situation in much of Ivory Coast, Ghana, Nigeria and the lowland areas of Cameroon and may extend into the Democratic Republic of Congo and other areas of Central Africa.

In *benign* areas the incidence of CMD is generally low and seldom exceeds 20%. Infection is due mainly to the use of infected planting material and there is little or no evidence of spread by whiteflies. Symptoms are usually inconspicuous and not associated with obvious deleterious effects on growth or root yield. Losses are not substantial and control measures are not considered necessary and would bring little benefit. This was formerly the situation in much of Uganda and western Kenya and is encountered currently in

large areas of Tanzania and Mozambique and in the mid-altitude agroecologies of Burundi, Malawi, South Africa and parts of Zambia.

There is an urgent need for information on the incidence and severity of CMD in other important cassava-growing areas of sub-Saharan Africa, including Sierra Leone, Liberia, Angola and Democratic Republic of Congo. It will then be possible to identify the areas that should receive priority in any attempts at intervention. Meanwhile, it should be appreciated that the situation can change dramatically and on a time-scale of only a few years. This is apparent from early experience in Madagascar and elsewhere (Cours *et al.*, 1997) and more recently in Uganda. There the situation changed rapidly from benign to epidemic and it is now changing to endemic as the original equilibrium between host and pathogen is being restored (Otim-Nape *et al.*, 2000).

### *Aetiology*

For many years CMD was assumed to be caused by a virus because the disease was transmissible by grafts and by the whitefly now known as *B. tabaci*, and yet no visible pathogen was detected. The situation changed in the 1970s when a virus was transmitted mechanically from CMD-affected cassava to the herbaceous test plant *Nicotiana clevelandii*. The status of the virus isolated was at first unclear because it could not be isolated from all the CMD-affected plants tested. Hence, the virus was initially referred to as cassava latent virus and this name continues to appear occasionally in the literature. However, the name became inappropriate when an additional test plant (*N. benthamiana*) was introduced and used to isolate and differentiate between virus isolates that all caused typical symptoms of CMD when transmitted back to cassava (Bock and Woods, 1983). The different isolates were initially referred to as strains of *African cassava mosaic virus* (ACMV) and three groups or 'clusters' of strains were distinguished. These were later regarded as separate viruses (Hong *et al.*, 1993) and they are now ascribed to the genus *Begomovirus*; family *Geminiviridae*. ACMV and *East African cassava mosaic virus* (EACMV) have not been found outside Africa, whereas *Indian cassava mosaic virus* (ICMV) seems restricted to the Indian subcontinent. A fourth virus of this type (*South African cassava mosaic virus*) has been distinguished recently in South Africa (Berrie *et al.*, 1998) and hybrid recombinant viruses have been distinguished in Uganda and Cameroon that have some of the genome properties of both ACMV and EACMV (Deng *et al.*, 1997; Zhou *et al.*, 1997).

The biological significance of the great diversity in biochemical properties of the different cassava mosaic geminiviruses has not been determined and requires investigation. Nevertheless, there is already evidence that dual infection with the hybrid recombinant virus and ACMV or with EACMV and ACMV is more damaging than any of these viruses occurring alone (Harrison *et al.*, 1997; Fondong *et al.*, 2000). The occurrence of different viruses or virus combinations in different regions could also complicate and may even undermine the effectiveness of resistance breeding programmes and quarantine controls on the movement of material between different parts of Africa. Until these issues are resolved it is important to avoid moving infected cassava between different countries or regions and especially from areas seriously affected by CMD. It is particularly important to avoid the transfer of cassava mosaic geminiviruses from Africa to the Indian subcontinent or vice versa, or from these regions to the Neotropics.

### *Effects on growth and yield*

There is an extensive literature on the effects of CMD on the growth and yield of cassava. Data have been collected at different times and places on a wide range of cultivars using two main approaches (Thresh *et al.*, 1994a). Firstly, comparisons have been made in formal experiments established with cuttings collected from healthy and CMD-affected plants. Secondly, naturally infected and healthy plants have been identified and assessed within larger plantings at experimental stations or in farmers' fields.

Some of the main findings are:

- Varieties differ greatly in their response to infection. Some are severely stunted and produce little or no yield of foliage, stem cuttings or tuberous roots, whereas others are relatively unaffected and sustain little damage.
- There is a general relationship between symptom severity and the decrease in

growth and tuberous root yield caused by CMD.

- Plants grown from infected cuttings are more severely affected than those of the same variety infected at an early stage of growth by whiteflies; plants infected late sustain little or no damage.
- Competition and compensation effects can occur within crop stands and both healthy and diseased plants grow better alongside diseased neighbours than alongside healthy ones. Consequently, differences between the growth and yield of healthy and diseased plants are less when comparisons are made between healthy and diseased plants each having neighbours of similar health status than between plants each having neighbours of dissimilar health status.
- Some virus strains or strain combinations cause more severe symptoms and decrease growth and yield much more than others.
- CMD influences the performance and sustainability of varieties by influencing the number, viability and growth of the stem cuttings available for propagation.

### *Overall crop loss*

The results of yield comparisons have been used to estimate the overall losses caused by CMD in whole localities, regions or countries. However, definitive estimates are only possible if detailed information is also available on the incidence and severity of the disease in different areas and on the prevalence, type, productivity and sensitivity to infection of the main varieties being grown. Such details are seldom available and the published estimates of yield loss provide only an indication of the magnitude of the damage sustained.

Watts Padwick (1956) used information from regional plant pathologists to estimate the losses caused by CMD in the former British Colonial territories of Africa. Fargette *et al*. (1988) later estimated the annual losses in Ivory Coast to be 500,000 t of roots compared to actual production at the time of 800,000 t. They assumed that all the plants being grown were affected and sustained losses in tuberous root yield of 38%, as recorded in their experiments on one of the main Ivorian varieties being grown. On similar assumptions losses in Africa were estimated to be 30 million t compared with actual production at the time of 51 million t (FAO, 1985).

These assumptions were inappropriate because the incidence of CMD is now known to be moderate or low in some important cassava-growing areas of Africa (Table 12.2). Moreover, some widely grown varieties are much less severely affected than the variety assessed in Ivory Coast. These considerations led Thresh *et al*. (1997) to estimate total losses in Africa as 12–23 million t. This estimate was based on the assumption of an overall CMD incidence of 50–60% and a loss of 30–40% in the yield of diseased plants.

Others have estimated the losses in particular areas, as in Uganda at the height of the recent pandemic (Otim-Nape *et al*., 2000). It was assumed that each year an area equivalent to four whole districts was rendered totally unproductive. This was equivalent to a loss of 60,000 ha, which could have been expected to produce 600,000 t of roots worth US$60 million at a conservative valuation of US$100 $t^{-1}$. Similarly, the losses due to the epidemic in western Kenya were estimated to exceed US$10 million in 1998 alone (Legg, 1999). The losses in Kenya have since become much greater as additional areas have been severely affected.

### *The transmission of cassava mosaic viruses by the whitefly* B. tabaci

The putative virus assumed to cause CMD in Africa was one of the first pathogens to be transmitted experimentally by whiteflies, and studies began in the 1920s when it became evident that the virus was spreading naturally and that whiteflies were the only sap-feeding insects on cassava likely to be vectors. The first transmissions were reported from Congo using adults of a species referred to as *Bemisia mosaicivecta* (Ghesquière, 1932), which was later stated to be a misprint for *B. mosaicivectura* (Storey and Nichols, 1938). The species was also referred to as *Bemisia gossypiperda* Misra & Lamba var. *mosaicivectura* (Mayné and Ghesquière, 1934). The same or a closely related species referred to as *Bemisia nigeriensis* Corbett was used in successful transmission experiments in Nigeria (Golding, 1936) and Tanzania (Storey and Nichols, 1938), where infection was achieved

by transferring infective whiteflies to the youngest leaves and shoots, but not to older ones.

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

### *Epidemiology*

The whitefly-borne viruses that cause CMD have not been reported in the Neotropics and they are assumed to have spread to cassava from indigenous African plant species. Several indigenous hosts have been identified, including *Jatropha* spp., but it is uncertain whether they are the original host(s) from which spread occurred. They certainly seem to be of little or no current importance as initial sources from which virus is spread to cassava. All the spread that occurs can be attributed to viruliferous whiteflies moving between or within cassava plantings, having acquired virus from cassava plants grown from infected cuttings or infected by whiteflies at a later stage of growth. This is consistent with the findings of epidemiological studies in Ivory Coast, Kenya and Uganda that spread into and within experimental plantings is related to the number of adult whiteflies recorded and also to the incidence of CMD in the area, as indicated by surveys of farmers' fields in the district or locality (Legg *et al.*, 1997; Otim-Nape *et al.*, 1998a), or from assessments of the health status of the propagules being used (Legg and Ogwal, 1998). New plantings are soon colonized by immigrant whiteflies moving from older stands of cassava in the area. The immigrants then reproduce to reach peak populations within a few months of planting before dispersing to other, younger, cassava (Fishpool and Burban, 1994).

The distribution of immigrant whiteflies and of plants newly affected by CMD is influenced by the direction of the prevailing wind and by the effects of wind turbulence around and within stands. The incidence of whiteflies and CMD tend to be greatest at the crop margins, especially along the windward and leeward edges and environmental gradients have been observed where whitefly populations and virus incidence decrease with increasing distance from the field boundaries (Fargette *et al.*, 1985; Colvin *et al.*, 1998). Incidence is also increased by breaks or discontinuities in the crop canopy which facilitate the alighting and establishment of viruliferous vectors (Fargette *et al.*, 1985).

### *Control measures*

There are obvious benefits to be gained by decreasing the losses caused by CMD and this can be achieved by a reduction in the incidence and/or severity of the disease. Various approaches to control are possible, as discussed in detail elsewhere (Thresh and Otim-Nape, 1994). However, the main attention has been given to the use of resistant varieties (Fargette *et al.*, 1996; Thresh *et al.*, 1998a) and phytosanitation, involving the use of CMD-free planting material and the removal (roguing) of any additional diseased plants that occur (Thresh *et al.*, 1998b).

Farmers occasionally use insecticides in attempts to restrict the spread of CMD by controlling the whitefly vector. However, the use of insecticides on cassava or other tropical root crops has received little attention from researchers in Africa and this approach is unlikely to be effective. It is also inappropriate because of the costs involved and the risks to farmers, consumers and the environment.

CROPPING PRACTICES. There are opportunities of adjusting cropping practices to decrease the losses caused by CMD. This can be done by adopting planting dates that avoid exposing young vulnerable plants to infection at times when there are likely to be the largest populations of viruliferous whiteflies (Adipala *et al.*, 1998). There are also advantages in planting away from and upwind of existing sources of infection and also in large compact blocks to minimize edge effects (Thresh and Otim-Nape, 1994). Other possibilities are to adopt close spacings or intercrops, or to interplant susceptible with resistant varieties. The benefits to be gained by adopting such practices have been established in experiments, but little or no attempt has been made to demonstrate the feasibility of these approaches. Moreover, they may be difficult for farmers to adopt within their existing cropping systems. This emphasizes the need for additional studies before attempts are made to change current farming practices.

RESISTANT VARIETIES. A feature of cassava in Africa is that many varieties are grown and there is great diversity for many different traits including susceptibility and response to CMD. Consequently, farmers who experience disease problems can usually respond by abandoning the most vulnerable varieties and adopting those that are somewhat resistant or tolerant and grow satisfactorily, even when infected. The ability of farmers to adjust to CMD in this way has long been recognized, but in the 1930s and 1940s attempts were made to breed varieties with greater levels of resistance by intercrossing cassava varieties with *Manihot glaziovii* and other species of *Manihot* (Jennings, 1994). Interspecies hybrids were backcrossed to cassava and led to the highly resistant varieties that have been developed and used in Madagascar and East Africa. Seeds of this type were also sent from East Africa to Nigeria, where selections that had been made there in the 1960s were used in the early 1970s as parents in the initial cassava improvement programme at the International Institute of Tropical Agriculture (IITA), Ibadan. This programme has been very influential and IITA clones and seeds have been widely distributed or used in National Breeding Programmes in many African countries and also by the IITA Regional Centre in Uganda (Mahungo *et al.*, 1994).

Some of the varieties produced in this way are so highly resistant to CMD that they sustain little or no damage, even under epidemic conditions. They are not readily infected and when infected usually develop inconspicuous symptoms that become even less conspicuous as growth proceeds and infected plants may eventually become symptomless. Moreover, virus is not fully systemic in highly resistant varieties and a substantial proportion of the cuttings collected from infected plants are free of virus and grow into healthy plants. This 'self-cleansing', 'reversion' phenomenon is important in restricting the progressive build-up of disease that would otherwise occur during successive cycles of vegetative propagation (Fargette *et al.*, 1994; Thresh *et al.*, 1998a).

Although highly resistant varieties of this type are available they are seldom widely grown and in many countries farmers continue to grow local varieties that have little or no resistance to CMD. This explains why the disease is so prevalent in many areas and why such serious losses have occurred during the current pandemic in East Africa. The reasons for this unsatisfactory situation and the factors influencing farmers' choices of variety are complex and not fully understood (Nweke *et al.*, 1994). In some areas little or no attempt has been made to introduce resistant varieties or to promote their use. This can be because of a lack of resources or incentive, or because CMD is not regarded as such a damaging disease that the use of resistant varieties is essential. Moreover, the resistant varieties may not be entirely satisfactory in other respects and do not always meet the exacting requirements of growers and consumers. Recent experience in Uganda is that any such defects may be overlooked or regarded as unimportant in epidemic conditions when CMD is causing serious losses and undermines food security, but not when production has been restored. Such factors as the taste, palatability and other quality characteristics of cassava varieties then become paramount (Otim-Nape *et al.*, 2000).

Undoubtedly, a greater use of CMD-resistant varieties would bring substantial benefits by decreasing the losses caused by the disease and facilitate control by other means. However, such benefits will be difficult to achieve until a full range of resistant varieties is available that meet all the requirements of producers and

consumers. Until then CMD will continue to cause problems. It seems inevitable that susceptible varieties will be retained in at least some areas and that CMD will continue to cause substantial, albeit generally acceptable, losses. This emphasizes the need for management procedures that will improve the health status of susceptible varieties and enable them to be grown successfully and more productively.

PHYTOSANITATION. The use of virus-free propagules is a basic approach to the control of many virus diseases and can bring obvious advantages (Thresh and Otim-Nape, 1994; Thresh *et al.*, 1998b). Crop establishment and initial growth are improved and there is a reduction in the number of primary sources of infection from which subsequent virus spread can occur. The yield benefits are particularly great with cassava because plants grown from infected cuttings sustain the greatest damage and much of the spread of CMD occurs during the early stages of crop growth. Moreover, whiteflies reproduce more rapidly on CMD-infected than on healthy plants and so infected plants contribute a disproportionately large proportion of the total vector population within a crop stand (Colvin *et al.*, 1999).

Clearly, there are powerful arguments for using CMD-free planting material and this approach has been advocated repeatedly. However, it has not been widely adopted, even in official cassava improvement programmes. The reasons for this are many and complex. In some areas CMD is so prevalent that it is regarded as a normal feature of cassava, CMD-free stocks are not available and farmers simply propagate from whatever plants are available and deemed suitable to provide cuttings. Even where CMD is less prevalent and there is an opportunity to select cuttings from uninfected plants, farmers seldom do so. They may be unaware of the benefits to be gained and of the basic features of CMD and its dissemination in infected cuttings and subsequent spread by whiteflies. Moreover, even if farmers are made aware it may be difficult or even impossible for them to distinguish uninfected plants at the time cuttings are required because the plants are leafless following drought or pest attack.

These difficulties are not easily overcome and there are obvious problems in contacting and changing the practices of the millions of cassava growers in Africa, many of whom are not readily accessible and poorly educated. Nevertheless, this was done widely in Uganda during the recent pandemic (Otim-Nape *et al.*, 2000). The effects of CMD were then so severe that farmers were very receptive to any measures that would alleviate the problem and emergency funds became available from donors for mass training programmes for farmers, extensionists and opinion leaders. Selection was shown to be feasible and was adopted widely by farmers in some of the worst affected areas who were anxious to improve the health status of their plantings as a means of restoring production. The problem in Uganda now is to ensure that farmers will continue to select 'clean' planting material as the CMD situation returns to normal. There is also a need to achieve similar results elsewhere in areas where there is no serious CMD problem and so less incentive to adopt basic control measures, or to provide special funding for training farmers. Until this is done it seems inevitable that CMD will remain prevalent in many areas and yields will be impaired because of the widespread use of infected propagules.

## Cassava brown streak disease

Cassava brown streak disease (CBSD) has been recognized since early studies in the 1930s, in what is now Tanzania. It was then established that the symptoms of the disease were distinct from those of CMD and that CBSD was more important than mosaic in some coastal areas of Tanzania (Storey, 1936). There has since been research on the aetiology, transmission and other features of CBSD in Tanzania, Kenya and elsewhere in eastern and southern Africa and at laboratories in the UK. However, research has been sporadic and the aetiology of the disease has been established only recently. Many uncertainties remain, especially relating to the effects of CBSD on crop yield and the natural means of spread.

### *Symptoms*

The symptoms of CBSD are unusual in that they can affect a wide range of organs including leaves, stems, tuberous roots and fruits. Moreover, the symptoms are very variable in type and

severity and some varieties are affected much less than others and frequently express symptoms only during the early stages of growth.

The name 'brown streak' was given to CBSD because of the brown elongate necrotic lesions that develop on the young green stem tissue of affected plants. This name is not altogether appropriate because only some varieties of cassava are so affected and the symptoms may be confused with the superficial circular necrotic spots of unknown cause that develop on the stems of some varieties (Nichols, 1950). Unlike the symptoms of CBSD the affected tissue does not extend into the cortex and the condition is not graft-transmissible.

The stem symptoms of CBSD are very variable in extent and severity and may be restricted to only one or a few shoots of each affected plant. In contrast, highly sensitive varieties develop very conspicuous stem symptoms on many branches, the leaves become necrotic and absciss and the shoots die back. The most severely affected plants eventually die but others recover, especially during periods of high temperature.

The leaf symptoms of CBSD are also variable and they are quite distinct from those of CMD in type and in affecting only the mature leaves. The most easily recognizable leaf symptoms occur as a characteristic 'feathery' chlorosis closely orientated along the secondary and tertiary veins and affecting many of the leaves or leaflets (Plate 2a). The symptoms are recognized less readily if they are relatively inconspicuous and restricted to only parts of some leaflets on affected plants. Other leaf symptoms occur as yellow blotches that are not closely associated with the leaf veins (Plate 2b). These symptoms affect different proportions of the leaf and they may or may not be conspicuous. They are particularly difficult to recognize when they develop only in the oldest leaves as they begin to discolour and senesce naturally. Such leaves soon absciss and the plants may then appear to be unaffected, especially at hot times of year when younger leaves develop inconspicuous symptoms or grow normally.

CBSD causes necrosis of the tuberous roots (Plate 2c) which also develop characteristic constrictions (Plate 2d). However, some varieties do not express root necrosis or do so only at a late stage of crop growth. These varieties are damaged much less severely than those that develop extensive symptoms at an early stage.

### *Distribution and prevalence*

In early studies on CBSD it was established that the disease occurred in coastal areas of Kenya and Tanzania and it was assumed to be present in adjacent areas of coastal Mozambique (Nichols, 1950). The disease was also reported at the time in Uganda and Malawi, especially at lower altitudes in southern Malawi towards the Mozambique border. However, there appear to have been no detailed surveys of the incidence or severity of CBSD and the overall prevalence and importance of the disease was unclear.

Information on the current incidence of CBSD has been obtained in recent surveys in Uganda, Tanzania, Mozambique and coastal Kenya. The disease was found in only one planting in Uganda (G.W. Otim-Nape and J.M. Thresh, unpublished observation) and in 62 (19%) of the 325 plantings examined in Tanzania, although the overall incidence in the country as a whole was only 6% (Legg and Raya, 1998). The incidence was much higher in the lowland coastal areas of Kenya and Tanzania and on Oguja Island of Zanzibar, as confirmed in additional detailed surveys (Thresh and Mbwana, 1998; Hillocks *et al.*, 1999; J.M. Thresh and T. Munga, unpublished).

Surveys conducted in 1999 confirmed the occurrence of CBSD in Nampula and Zambezia provinces of Mozambique, which are the two most important cassava-growing areas of the country. The overall incidence based on assessments of leaf and stem symptoms was 49% in Zambezia and 28% in Nampula, but the incidence was much higher in some districts, varieties and plantings, especially in lowland coastal areas (R. Hillocks and J.M. Thresh, unpublished). Moreover, the leaf symptoms were sometimes inconspicuous and not readily distinguished, which suggested that the results underestimate the true incidence of infection.

Symptoms also tended to be inconspicuous in reconnaissance surveys carried out in Malawi during the early 1990s (J.M. Thresh and A. Sweetmore, unpublished). CBSD was then present in many areas and was most prevalent at mid-altitudes along the northwestern shore of Lake Malawi. These areas had been used to supply planting material to many other parts of Malawi, following the severe effects of the 1990–1991 drought and thus contributed to the

widespread occurrence of CBSD. There may also have been movement of planting material across the border into Zimbabwe and Zambia, where CBSD is known to occur. The disease has not been reported in South Africa or Angola, or in any of the countries of West and Central Africa.

### *Aetiology*

From the outset CBSD was assumed to be caused by a virus because it was graft-transmissible and no visible pathogen was detected. The first evidence of a virus was obtained by sap inoculation from cassava to herbaceous hosts and back to cassava (Lister, 1959) and also by electron microscopy (Kitajima and Costa, 1964). Virus isolates in herbaceous hosts were later shown to have elongate particles 650–690 nm long (Lennon *et al.*, 1986a). They resembled those of viruses now ascribed to the genus *Carlavirus*, but no serological relationship was demonstrated at the time with any definitive virus of this type.

There was later evidence that two different elongate viruses occur in CBSD-affected plants (Lennon *et al.*, 1986a; Brunt, 1990) and isolates in herbaceous hosts were shown to induce 'pin-wheel' inclusions of the type produced by viruses now attributed to the family *Potyviridae*. This is consistent with the recent conclusion that CBSD is caused by a virus of the genus *Ipomovirus*, which is one of the four genera comprising the *Potyviridae* (Monger *et al.*, 2001).

### *Effects on growth and yield*

There is only limited information on the effects of CBSD on growth and yield. In studies on a local Kenyan variety the main effect was on the quality of the roots produced and not on root weight or number (Bock, 1994). However, yields of marketable roots were decreased in a more recent study with other varieties in Tanzania (R. Hillocks and M.D. Raya, unpublished). Apart from any such loss of yield, necrosis decreases the value of the roots produced which become unusable and unsaleable if the damage is extensive. This may necessitate farmers having to harvest prematurely before much deterioration of the roots has occurred, but this incurs a yield penalty. Additional studies are required with a wide range of varieties harvested after different periods to establish the full significance of these effects.

### *Epidemiology and control*

There is little information on the epidemiology and control of CBSD and there are many uncertainties which impede the development of effective management strategies. One of the problems has been the lack of assured virus-free stocks of planting material for epidemiology experiments and for use by farmers. Another has been the failure to identify the natural means of spread between plants. These issues are now being addressed in projects in Tanzania and Mozambique. Moreover, in these countries and also in Kenya and Malawi breeding lines are being assessed for resistance to CBSD, as in earlier studies in Tanzania between 1937 and 1957 (Jennings, 1957).

From experience in several countries it is apparent that much use is being made of CBSD-infected planting material which is an effective means of perpetuating and disseminating the disease. However, there is evidence of natural spread between plants as clones introduced from West Africa or other areas that are free from CBSD have become infected when grown at sites in Mozambique, Malawi, Kenya and Tanzania where infection is rife. Plants raised from seed introduced from West Africa have also become infected at these sites.

There is little evidence on temporal or spatial patterns of spread, but this is known to have been slow in an experiment at a site in coastal Kenya (Bock, 1994) and rapid in recent trials at sites in coastal Tanzania (M. Raya, K. Mtunda and R.J. Hillocks, unpublished information) and Mozambique (R. Macia and J.M. Thresh, unpublished information). This emphasizes the need for additional studies to determine the circumstances under which spread occurs and the scope for utilizing the benefits of virus-free planting material to replace the contaminated stocks now being used widely. Virus-free stocks can be produced by rigorous selection (Mtunda *et al.*, 1999) and in future this may be facilitated by using the sensitive methods of virus-detection now being developed. It is also possible to use meristem-tip and/or heat therapy to eliminate CBSV from clones that seem to be totally infected (Kaiser and Teemba, 1979).

Natural spread of CBSD between plants is attributed to an arthropod vector or vectors as yet unidentified. However, only few transmission experiments have been done, mainly involving the aphid *Myzus persicae* and the whitefly species *B. tabaci* and *Bemisia afer* (= *Bemisia hancockii*). The two whitefly species have been considered because they are two of the few sap-feeding insects to have had a long association with cassava in Africa. Moreover, CBSV is now attributed to the same genus of the *Potyviridae* as *Sweet potato mild mottle virus* which is transmitted by *B. tabaci*. It is also notable that *B. afer* seems to be particularly common in coastal areas of eastern and southern Africa where CBSD is most prevalent. This emphasizes the need for additional studies with *B. afer* and also of insect species that visit but do not colonize and breed on cassava. At least some of the spread may be from hosts other than cassava, as CBSV has been detected only in eastern and southern Africa and it is assumed to have indigenous hosts from which it spread to cassava after the crop was introduced. The identification of a vector will help to explain the current limited geographic distribution of CBSD, which occurs mainly in the lowland coastal areas of eastern and southern Africa. Such knowledge would facilitate the development of specific control measures. Meanwhile, the emphasis has been on the use of varieties that do not develop severe root necrosis, or do so only at a late stage of crop growth. This attitude of 'living with' the disease is similar to that adopted in many areas to cassava mosaic disease and provides a means of avoiding serious losses. However, any yield penalty incurred through the widespread use of tolerant varieties has not been quantified and could be substantial. This suggests that there could be benefits in developing and exploiting virus-resistant varieties and effective methods of phytosanitation.

## The Viruses and Virus Diseases of Cassava in Asia and the Pacific Regions

Cassava is grown in many countries of South-East Asia and the Pacific and these areas account for an estimated 27% of total world production. Cassava mosaic disease (CMD) is the only virus disease known to be important in the region and it seems to be restricted to India and Sri Lanka. An early report of CMD in Indonesia (Muller, 1931) has not been confirmed and the symptoms were later attributed to a mineral deficiency (Bolhuis, 1949). *Cassava green mottle virus* has been detected in cassava originating from the Pacific region (Table 12.1; Lennon *et al.*, 1987), but its prevalence and importance is not known and it is not considered further here.

### Cassava mosaic disease (CMD)

CMD was not reported in India until 1966 (Alagianagalingam and Ramakrishnan, 1966), although it is known to have been present earlier (Abraham, 1956) and it has since been recorded in Sri Lanka (Austin, 1986). The disease has received much less attention in Asia than in Africa. Nevertheless, it is clear that many of the research findings from Africa as summarized in an earlier section (pp. 242–249) also apply to India and Sri Lanka.

#### *Distribution and prevalence*

There is little current information on the incidence of CMD in India and the only available data were obtained during a reconnaissance survey in 1988 (Mathew, 1989). Twenty fields were assessed in each of 18 districts, including 11 districts of Kerala State. The overall incidence of CMD was higher in the two main cassava-growing states of Kerala (23%) and Tamil Nadu (30%) than in Andhra Pradesh (< 1%) and Karnataka (5%), which are outside the main cassava-growing areas. However, the number of fields examined was limited, especially when considered in relation to the large area of cassava being grown (Table 12.2). There is a need for additional more comprehensive surveys, especially as CMD seems to have become more prevalent in recent years. This was evident on a 1996 tour of the main cassava-growing areas of Kerala and around Salem in Tamil Nadu. Many of the fields visited in the lowland areas were almost totally affected and in some localities the symptoms were unusually severe and associated with poor yields. The incidence was much less in the upland areas and in a lowland planting established with cuttings

obtained from the hills (M. Thankappen and J.M. Thresh, unpublished observations).

### *Aetiology*

The symptoms of mosaic disease on cassava in India are similar to those reported in Africa and the name cassava mosaic disease (CMD) has been adopted in some publications and Indian cassava mosaic disease (ICMD) in others. Malathi and Sreenivasan (1983) first isolated a geminivirus from CMD-affected plants in India, as in the earlier studies in Africa. Four Indian isolates were included in serological tests with isolates from coastal and western Kenya using polyclonal antisera prepared against African and Indian isolates (Malathi *et al.*, 1985, 1987). Three of the Indian isolates reacted positively with African antisera but they were distinguishable serologically from African isolates and so were regarded as being of a separate strain of ACMV. In subsequent tests using a panel of monoclonal antibodies, Indian and Sri Lankan isolates were distinguished from those from East and West Africa and later referred to as *Indian cassava mosaic virus* as described previously (p. 245).

### *Effects on growth and yield*

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

### *Transmission by the whitefly* B. tabaci

CMD spreads naturally in India and following earlier experience in Africa (pp. 246–247), the main attention has been on *B. tabaci* in the search for an insect vector. Successful transmissions have been reported using whiteflies transferred from infected to healthy cassava, from infected cassava to herbaceous hosts and between herbaceous hosts. High rates of transmission were achieved in some experiments, as between cassava (19%) and from cassava to *Nicotiana tabacum* cv. Jayasri (100%), *N. rosulata* (67%) and 11 other *Nicotiana* spp. (20–25%) using 50 whiteflies per test plant (Mathew and Muniyappa, 1993). However, such high rates of transmission seem to be exceptional and not readily reproducible. Much lower rates of transmission were reported in other studies (e.g. Nair, 1975), some of which were completely unsuccessful (Malathi *et al.*, 1985; Palaniswami *et al.*, 1996). Another inconsistency is that transmissions from cassava to cucumber were achieved in some trials (Menon and Raychaudhuri, 1970), but not in others (Mathew and Muniyappa, 1993). The reasons for this and the apparent difficulty experienced in transmitting Indian isolates by whiteflies compared with those in Africa, have not been determined. One possibility is that the whiteflies on cassava in India are less well adapted to their host than those in at least some parts of Africa where a cassava biotype of *B. tabaci* has been distinguished (Burban *et al.*, 1992). It certainly seems particularly difficult to transmit Indian isolates to cassava and similar difficulties have been recorded with other isolates in studies in glasshouses in temperate conditions (B.D. Harrison and P.J. Markham, personal communication). Despite these difficulties there is no reason to doubt that *B. tabaci* is the vector of ICMV and studies on epidemiology, control and whitefly population dynamics have proceeded on this assumption (e.g. Mahto and Sinha, 1978).

### *Epidemiology*

The area of cassava grown in India is considerably less than in Africa. Nevertheless, the crop is grown in diverse environments including the

lowland humid forest areas of coastal Kerala, the upland foothills of the Western Ghats and the irrigated areas of Tamil Nadu where there is a prolonged dry season.

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

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

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

### *Control*

Cassava in India is grown under very different conditions from those in Africa. The relatively high productivity of cassava achieved in India is associated with the limited use of intercropping and with generally good husbandry practices. These include effective weed control, the establishment of uniform stands, the routine application of fertilizers and in some areas the use of irrigation. Moreover, the Indian crop is unaffected by either the cassava green mite or the cassava mealybug which have had such damaging effects in many parts of Africa (see Chapter 11).

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

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

Virus-free stocks have been obtained by rigorous selection and through the use of meristem-tip therapy. They have been used in experiments and shown to remain largely free of

CMD in areas where there is limited spread by whiteflies. Substantial increases in yield have been achieved in this way (Nair, 1990; Nair and Thankappen, 1990), but only limited attempts have been made to encourage the widespread adoption of such material.

## General Discussion

From the foregoing account it is clear that the viruses and virus diseases of cassava have received considerable attention, especially those occurring in Africa. Nevertheless, the available information is very incomplete and many uncertainties remain. For example, the status, distribution and effects of several of the viruses listed in Table 12.1 have not been determined and further research may show them to be more widespread and damaging than present evidence suggests. There is also uncertainty concerning the epidemiology and mode of spread of cassava brown streak, cassava frogskin and other diseases and an urgent need to confirm the role of the whitefly or other vectors involved.

These deficiencies can be remedied by an allocation of expertise and resources commensurate with the importance of cassava as the basic staple food crop of large and populous areas of the tropics. However, a problem is likely to be encountered in achieving this because increasingly donors and grant agencies are allocating funds in response to the perceived needs of farmers, who may be totally unaware that virus problems exist. This is evident from recent experience with cassava brown streak disease in Mozambique and Tanzania and with frogskin disease in South America. In these areas farmers have created or exacerbated the problem by making extensive use of virus-infected cuttings as planting material and losses due to disease are regarded as inevitable and a normal feature of cassava in the localities affected.

These and other experiences elsewhere indicate the difficulty of achieving sustained improvements in the overall health status of cassava by adopting virus-free cuttings and by deploying resistant varieties and other research findings. It is necessary to change the attitudes and practices of millions of farmers, many of whom are remote, poorly educated and lack resources and access to extension personnel and technical advice. There is a tendency to ignore or underestimate the importance of virus diseases unless the losses sustained are so great that rural livelihoods and food security are undermined. Relief or emergency measures are then necessary and farmers also respond by exploiting the genetic diversity available and switching to less vulnerable varieties. Once production has been restored virus diseases again receive relatively little attention even though they impair productivity and the yield penalty may be substantial.

Clearly, these difficulties will not be overcome quickly or easily and losses due to viruses are likely to continue in the foreseeable future. Indeed, they may even increase if damaging viruses, strains or strain combinations reach new areas by natural spread or through the movement of infected propagules. This emphasizes the importance of stringent quarantine controls on the movement of cassava material to maintain the present limited distribution of cassava viruses. Several of these are restricted to particular continents or regions and are likely to cause considerable damage if they are spread elsewhere. The need to prevent New World viruses reaching Africa or Asia, and Old World viruses being introduced to the Americas has long been apparent and appropriate quarantine measures have been devised and enforced (Frison and Feliu, 1991). These measures should be revised now that additional viruses of cassava have been characterized and new methods of virus detection have been developed. There is also a need to consider the implications of recent findings on the diversity and variability of cassava mosaic geminiviruses and the occurrence of particularly damaging strains or strain combinations.

Experience with cassava mosaic disease in Africa over many years and more recently with frogskin disease in South America is that the situation is labile and can change rapidly. This is also apparent from recent experience with the whitefly *B. tabaci* which seems to be adapting to cassava in different countries of South America where previously cassava was not infested. Moreover, the damaging 'B' biotype of *B. tabaci* has spread recently to parts of northern and southern Africa and could lead to increased problems. These developments emphasize the importance of continued research on the viruses and virus vectors of cassava to monitor and combat

new problems as they arise and to deal more effectively with those already known.

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