ROOTS

CASSAVA MOSAIC AND CASSAVA BROWN STREAK VIRUS DISEASES IN AFRICA: A comparative guide to symptoms and aetiologies*

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Introduction

Vegetatively propagated crops are prone to virus infection and cassava (*Manihot esculenta* Crantz) is no exception to this generalisation. At least seventeen different viruses of cassava have been described, of which eight are known to occur in Africa (Thresh *et al*., 1994). The main attention in Africa has been on the viruses causing cassava mosaic and cassava brown streak diseases which are the subject of this publication. Relatively little attention has been given to the other viruses of cassava or to the diseases they may cause. There is limited information on their distribution and none on their effects on growth or yield. These are serious deficiencies and emphasise the inadequate attention given to the viruses of what is arguably, the most important African food crop.

Cassava mosaic disease (CMD)

Distribution

CMD was first described in 1894 in what is now Tanzania. The disease was later reported in many other countries of East, West and Central Africa. It is now known to occur in all the cassava-growing countries of Africa and the adjacent islands and also, in India and Sri Lanka. A report of the disease in Indonesia in 1931, has not been confirmed and the mosaic disease of cassava in South America is caused by a different virus.

There are great differences between regions in the overall prevalence of CMD and in the severity of the losses caused. The available information from surveys and yield loss assessments is summarised by Thresh *et al*. (1997), who on plausible assumptions, estimate the losses in Africa to be 15–24%. This is equivalent to 15–28 million tonnes, compared with the FAO production estimates for 1997 of 84 million tonnes.

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Causal Agent(s)

When CMD was first described, the causal agent was assumed to be a virus, in the absence of any visible pathogen. This view was consistent with the results of early studies showing that the disease was transmitted by a whitefly vector, now known to be *Bemisia tabaci*. However, proof of the viral aetiology was not obtained until the 1970s and 1980s, when sap inoculations to herbaceous hosts were successful and virus isolates obtained in this way were purified and characterised. After initial uncertainty, the isolates were shown to cause CMD, Koch's postulates were fulfilled and the various isolates from Africa and India were regarded as strains of a single virus of the geminivirus group and designated African cassava mosaic virus. Subsequent studies have led to the recognition of several distinct but similar viruses:

- African cassava mosaic virus (ACMV)
- East African cassava mosaic virus (EACMV)
- Indian cassava mosaic virus (ICMV)
- South African cassava mosaic virus (SACMV)

What appears to be a hybrid recombinant between ACMV and EACMV has been reported in Uganda, Kenya, Tanzania, Sudan and Democratic Republic of Congo and designated UgV. The different viruses have very similar properties and they are all members of the newly created family: *Geminiviridae*; Genus: *Begomovirus* (type member, Bean golden mosaic virus). Each of the cassava mosaic geminiviruses (CMGs) can cause CMD and there is evidence that virus combinations are more damaging than single infections.

These results have been obtained only relatively recently at the Scottish Crop Research Institute, Dundee and elsewhere (Harrison *et al*., 1995; Thresh *et al*., 1998a; Rey and Thomson, 1998). The full implications are as yet unclear and additional information is required on the distribution of the different viruses and on the interactions between them. Meanwhile, it is appropriate to refer to CMD in Africa or Asia caused by CMGs. There is no justification for referring to separate Indian, East African and South African diseases which would create needless confusion, given the limited facilities available in many parts of Africa for detecting and characterising the virus or viruses present in mosaic-affected plants.

Symptoms

The symptoms of CMD occur as characteristic leaf mosaic patterns that affect discrete areas and they are determined at an early stage of leaf development The chlorotic areas fail to expand fully so that stresses set up by unequal expansion of the lamina cause malformation and distortion. Severely affected leaves are reduced in size, misshapen and twisted, with yellow areas separated by areas of normal green colour. The plants are stunted and the young leaves absciss (Storey and Nichols, 1938; 1951).

Plant with stunted growth due to CMD

The leaf chlorosis may be pale yellow or nearly white, or just discernibly paler than normal. The chlorotic areas are usually clearly demarcated and vary in size from the whole leaflet to small flecks or spots. Leaflets may show a uniform mosaic pattern or the pattern is localised to a few areas which are often at the bases of the leaflets.

Distortion, reduction in leaflet size and general growth retardation, appear to be secondary effects associated with symptom severity.

Symptoms vary from leaf to leaf, shoot to shoot and plant to plant, even for the same variety and virus strain in the same locality. Variation in symptoms may be due to differences in virus strain, the sensitivity of the host, plant age and environmental factors such as soil fertility, soil moisture availability, radiation and temperature. Cool temperatures usually enhance symptom expression, while warm temperatures restrict it.

Leaves affected by cassava mosaic disease

Some leaves situated between affected ones may seem normal and give the appearance of recovery. This behaviour depends on the ambient temperature and host-plant resistance. However, symptoms may recur on recovered plants when environmental conditions favour symptom expression. The first few leaves produced by an infected cutting are sometimes symptomless and are followed by severely affected leaves. There is a tendency for symptom severity to diminish as plants age, especially in resistant varieties. Symptoms tend to reappear on the axillary growth after the shoot tips are removed and this procedure is sometimes adopted to enhance symptom expression in screening for resistance.

Cassava green mite (*Mononychellus tanajoa* Bondar) is found in most cassava-growing areas of Africa and symptoms may be confused with or mask those of both CMD and CBSD (see Fig.1d.). A feature that facilitates a distinction between mite and virus damage is that the symptoms caused by mites are similar on each leaflet of the same leaf and on each side of the midrib. The symptoms of mosaic usually differ on the different leaflets and either side of the midrib.

Transmission and Spread

CMGs are disseminated in the stem cuttings used routinely for vegetative propagation. They are also transmitted by the whitefly, *Bemisia tabaci* Gennadius. Two other species of whitefly (*Bemisia afer* Priesner & Hosny and *Aleurodicus dispersus* Russell) also infest cassava in Africa and India but they have not been tested adequately as possible vectors. Dissemination in stem cuttings can lead to the introduction of CMD to new areas and accounts for the occurrence of the disease in areas where there is little or no spread by the whitefly vector. Spread between plants is by the whitefly and can be rapid in some areas, as shown by experience in Côte d'Ivoire, Nigeria, Uganda and more recently in west Kenya and north-west Tanzania.

Management

The basic approach to controlling CMD should be to select stem cuttings for propagation from symptomless mother plants. This is seldom done and inadvertently, much use is made of infected planting material. However, there is considerable evidence of the advantages to be gained from a more discriminating approach to the selection of planting material (Thresh *et al*., 1998b). Selection is easy and can be very effective if the parent plants are growing vigorously and express conspicuous

symptoms when infected. Difficulties arise if the plants are resistant and express inconspicuous symptoms, or if the leaves absciss or are damaged following drought or pest attack. Problems can also arise if leaves are discoloured and distorted, due to the effects of zinc or other mineral deficiency.

It has long been recognised that some varieties are resistant to CMD and sustain little or no damage when infected. Such varieties have been widely used as a means of control. However, they are not always available or may not have all the other favourable attributes required by farmers. This explains why susceptible varieties are still widely grown, especially in areas where CMD is not a prevalent or serious problem and there are no compelling reasons for adopting virus-resistant varieties.

Little use is made of insecticides to control the whitefly vector and such measures are inappropriate for a widely grown subsistence crop. Only limited attention has been given to other possible control measures such as the use of intercrops, crop disposition or the manipulation of planting date to decrease the risk of infection (Thresh and Otim-Nape, 1994). Such measures merit consideration in the current search for integrated means of control that seek to make the most effective use of phytosanitation and resistant varieties (Hillocks, 1997).

Cassava brown streak disease (CBSD)

Distribution

CBSD was first described by Storey (1936) who recorded it in the foothills of the Usumbara Mountains of Tanganyika (now Tanzania). Nichols (1950) later reported that the disease was endemic in all East African coastal cassava growing areas, from the north-east border of Kenya to Mozambique and was widespread at lower altitudes in Nyasaland (now Malawi). More recent surveys have confirmed this distribution in Tanzania and Malawi (Hillocks *et al*., 1996, 1998; Legg and Raya, 1998; Sweetmore, 1994). Surveys conducted in 1999 revealed that the disease was widespread in Mozambique in the two Provinces of Zambesia and Nampula that were assessed (R. Hillocks, J.M. Thresh, J. Tomas and R. Xavier, unpublished report). In southern Tanzania, CBSD is common at altitudes below 300 m, less common between 300 m and 700 m and rare at altitudes above 700 m, where natural spread does not seem to occur.

Causal agent

Since CBSD was first described, the causal agent was assumed to be a virus, in the absence of a visible pathogen. This seemed to be confirmed when the disease was sap-transmitted to a range of herbaceous indicator hosts by Lister (1959) and in later experiments of Bock and Guthrie (1976). Virus particles were then detected by electron microscopy in leaf samples showing typical CBSD symptoms that were sent to the UK. The particles were elongate, flexuous filaments 650–690 nm long (Lennon *et al*., 1986) that contained ‘pin-wheel’ inclusions, typical of potyviruses (Harrison *et al*., 1995). The exact aetiology of the disease remained a matter of speculation until recent work at Bristol University in the UK, where the coat protein gene of CBSV was cloned and sequenced. The virus has now been shown to be an Ipomovirus, a whitefly-transmitted potyvirus (G. Foster, unpublished report). The provisional taxonomy of CBSV would be as follows: Phylum: RNA virus; Class: 1 (Picornia-like viruses); Order: 2; Family: *Potyviridae*: Genus: *Ipomovirus* (Type member: sweet potato mild mottle virus); species: CBSV.

Symptoms

All parts of the cassava plant may show symptoms of CBSV infection but the aspects of the syndrome that are manifest and to what degree, depend on environmental conditions, the growth stage of the crop relative to the time of infection and the sensitivity of the cultivar. Cassava mosaic and CBSD both cause chlorotic leaf mottle, although the symptoms of the two diseases are quite distinct when they occur separately. Leaf symptoms of CBSD may be absent on infected plants under certain undefined environmental conditions, especially on new growth sprouting after drought-induced defoliation. Nichols (1950) distinguished two types of foliar symptoms associated with CBSD:

o *Leaf symptoms* (Type 1) – chlorosis appears first along the margins of the secondary veins later affect tertiary veins and may develop into chlorotic blotches.

Leaf infected with CBSD showing chlorosis around the veins

CBSD foliar symptoms

o *Leaf symptoms* (Type 2) – chlorosis not clearly associated with the veins but in roughly circular patches between the main veins. In advanced stages of disease, much of the lamina may be affected. Diseased leaves remain attached to the plant for several weeks. During very hot weather, symptoms do not appear on newly formed foliage.

The presence of stem symptoms also seems to be variable and may differ with cultivar. They are usually present in the advanced stages of the disease and often indicate the presence of root symptoms.

o *Stem symptoms* – are not consistently associated with CBSD, except in the more sensitive varieties. On young green stem tissues, purple/brown lesions may be observed on the exterior surface which are seen to have penetrated into the cortex on stripping off the outer bark. Necrotic lesions in the leaf scars appear after leaves have shed due to normal senescence. In severe infections, these lesions develop to kill the dormant axillary buds. Once axillary buds are killed, a general shrinkage of the node occurs and death of the internodal tissue follows, so that the branch dies from the tip downwards, to cause 'die back'.

Stem necrosis on stems of plants infected with CBSD

o *Root symptoms* – are characteristic of CBSD in some cultivars and are the most destructive

Cassava tuber infected with CBSD

component of the syndrome. Root symptoms usually develop after foliar symptoms and the period between infection and onset of root necrosis seems to be cultivar-dependent. Some cultivars have been identified in which root necrosis does not develop until more than 8 months after planting an infected cutting, despite the earlier presence of clear foliar symptoms (Hillocks *et al*., 1996). In the most sensitive cultivars where planting material has been derived from infected stock, root necrosis can become apparent from 5 months after planting (R. Hillocks and M. Raya, unpublished).

Root symptoms are variable on the outside of the root and may appear as radial constrictions and/or pits and fissures in the surface bark. Tissue surrounding the pits is stained brown or black. Below the pits, the cortex is necrotic. The internal symptoms consist of a yellow/brown, corky necrosis of the starch-bearing tissue, sometimes with blue/black streaks. The lesions seem to remain discrete, although in sensitive varieties, almost the whole of the starch storage tissue may be affected. Decay and soft rot ensue only in the advanced stages of infection and when secondary organisms invade. Sometimes, the roots appear healthy on the outside with no obvious constrictions or size reduction, but when cut open, they are found to be necrotic.

Transmission and spread

Storey (1936) demonstrated that the causal agent of CBSD was graft-transmissible, and that cuttings from affected plants gave rise to plants showing characteristic foliar symptoms of the disease. Thus the disease is readily introduced into newly planted

Scoring system used to assess the severity of root symptoms of CBSD

areas through the use of infected planting material. In the most sensitive varieties, under lowland conditions, severe symptoms result when the disease is established at this early stage. Storey (1939) believed that the disease was caused by an insect-borne virus and that the most likely vector was a whitefly (*Bemisia* spp.). Observations in field trials conducted in Tanzania indicate that considerable spread takes place between plants but transmission experiments with mixed populations of *B. tabacii* and *B. afer* have so far been unsuccessful. In Kenya, Bock (1994) was also unable to transmit CBSD with *B. tabaci* (which is known to transmit CMGs), or with six species of aphid. Lennon *et al.* (1986) also reported failure to transmit CBSV with the aphid *Myzus persicae* Sulz. A second whitefly species, *B. afer* occurs in East Africa, together with B. *tabaci*, reaching highest population densities in some of the areas where the incidence of CBSV is greatest (Robertson, 1987). *B. afer* was considered generally the less abundant whitefly species in the cassava growing areas of East Africa. However, surveys in Malawi showed that *B. afer* was the predominant species on cassava in most parts of the country and may be the main vector of CMGs there (Munthali, 1992). CBSD has also been recorded from the shores around Lake Malawi (Legg and Raya, 1997). Bock (1994) suggested that *B. afer* is the putative vector and recent progress on

classification of the causal agent as an ipomovirus, again points to a whitefly vector. Transmission experiments are continuing both in Tanzania and at NRI in the UK.

Management

As with CMGs, the basic approach to control for CBSD is to select planting material from symptomless mother plants. The health of the stock needs to be maintained by continued selection and roguing of any infected individuals which appear at sprouting. The success of this approach depends on the amount of disease in surrounding cassava and the rate of spread. The mechanism of spread is unknown for CBSV and the value of virus-free planting material cannot yet be predicted. However, this may be worthwhile for areas of low disease pressure with little or no disease spread. For areas of high disease pressure on the coast of Tanzania and Mozambique for instance, release of virus-free planting material needs to be combined with deployment of cultivars which exhibit some form of resistance. Local cultivars such as 'Nanchinyaya' in southern Tanzania which seem to be tolerant of infection and are slow to develop root necrosis, could be used. Surveys conducted in Tanzania have indicated that there are other cultivars with varying degrees of resistance to infection with, or tolerance to, CBSV.

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