# CHAPTER 16

# Cassava Viral Diseases of South America

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Vegetatively propagated cassava is particularly prone to damage by viruses as infection tends to build up in successive cycles of propagation. At least 16 different viruses have been isolated so far from cassava, but there are probably others that have yet to be described (Calvert and Thresh 2002).

Because the center of origin of cassava is in the Neotropics and its introduction into other regions has been relatively recent, only one of the viruses attacking this crop in Central and South America has been found elsewhere. In addition, several Neotropical viral diseases are asymptomatic and do not damage plants, reflecting long periods of coevolution between host and pathogens.

The main cassava viruses causing diseases of economic importance that deserve special attention in plant quarantine controls are the African cassava mosaic virus (ACMV), East African cassava mosaic virus (EACMV)[4], South African cassava mosaic virus (SACMV), cassava brown streak virus (CBSV), Indian cassava mosaic virus (ICMV), cassava common mosaic virus (CsCMV), cassava vein mosaic virus (CVMV), and cassava frogskin virus (CFSV). In South and Central America, particular attention should be paid to the latter three.

## Cassava Common Mosaic Disease

### Background and distribution

Cassava common mosaic disease (CsCMD) was first reported in southern Brazil (Silberschmid 1938; Costa 1940). It has since been found in several countries of South America, and in Africa and Asia.

Usually, the disease is not important in Latin America and the Caribbean. No detailed studies exist of affected areas in 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.

The disease has no known vector and its dissemination throughout a crop is attributed to mechanical transmission.

### Description

Plants infected by CsCMD develop symptoms of mosaic and chlorosis in leaves. Sometimes, infected leaves present clear, dark green spots, bordered by veins. Symptoms are more severe during prolonged and relatively cold periods—a frequent situation in the South American subtropics. Under these conditions, infected plants are usually dwarfed and yield losses may be as high as 60% (Costa and Kitajima 1972) (Figure 16-1).

### Etiology and epidemiology

Cassava common mosaic disease is caused by the virus of the same name (CsCMV) which can infect species belonging to several families of dicotyledonous plants. This virus was originally classified in the potexvirus group, that is now referred to as the genus Potexvirus. The virions of CsCMD are elongated, semi-flexuose particles that measure 15 × 495 nanometers (Kitajima et al. 1965) and contain RNA.

In cassava, the virus presents the nuclear inclusions typical of potexviruses, as found in another

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4. For an explanation of this and other abbreviations and acronyms, see *Appendix 1: Acronyms, Abbreviations, and Technical Terminology*, this volume.

Figure 16-1. Symptoms caused by CsCMD.

host *Nicotiana benthamiana*. The virus is known to also systemically infect *Euphorbia* spp., *Cnidoscolus conitifolius*, and other species of the Euphorbiaceae family (Costa and Kitajima 1972).

The CsCMV viral particles contain a simple protein cover with a molecular weight of 26,000 Da (Nolt et al. 1991). The genome consists of single-stranded RNA, of which the complete sequence is known (Calvert et al. 1996). The organizational structure, proteins, and molecular weights are usually similar to those of other potexviruses.

The principal source of inoculum is infected plant material. Because the virus spreads systemically through the plant, stakes from diseased plants are also infected. The virus is highly stable and may spread through mechanical transmission on machetes or other implements used in agricultural tasks. Although this mode of transmission is inefficient, it is the only known means of dissemination from plant to plant.

### Management and control

Eliminating plants that express CsCMV symptoms provides adequate control. The symptoms are evident in primary leaves. If this is not done early in the cropping cycle, the plants must then be marked and the stems burned after the roots are harvested. To minimize the risk of mechanical transmission, cutting tools should be periodically disinfected (Lozano and Nolt 1989). Care in selecting healthy planting materials can eradicate CsCMV or at least mitigate, to a minimum, the economic damage it causes.

## Cassava Vein Mosaic Disease

### Background and distribution

The first report on cassava vein mosaic disease (CVMD) was in 1940 (Costa 1940). The areas where the disease is prevalent are still inhabited by mainly rural communities where the lack of economic resources contributes to ignorance on this disease. Because symptoms are sporadic and usually not readily visible, the disease is unlikely to receive adequate attention at the end of the crop's growing cycle (Figure 16-2).

The disease is very common in the semiarid areas of Northeast Brazil. However, its presence in other regions of the country has also been reported, that is, in the States of Ceará, Pernambuco, Alagoas, Piauí, and Bahia (Calvert et al. 1995), and in some neighboring regions.

### Description

The first four or five leaves of infected stakes present chlorotic veins. The chlorosis starts forming a pattern

Figure 16-2. Symptoms caused by CVMV.

of rings that, as they join, create a circular spot. Leaves with severe symptoms commonly have deformed blades and show epinasty. Sometimes, symptoms disappear and their expression is influenced by climatic conditions. Leaves of infected plants become prematurely old and fall, reducing leaf area. Frequently, in mature plants, observing leaves with symptoms of mosaic is difficult, as these are more pronounced in the semiarid areas than in the humid coastal regions of Northeast Brazil. The disease does not seem to affect plant vigor.

### Etiology and epidemiology

Cassava vein mosaic disease is caused by a virus of the same name (CVMV), which presents isometric particles, 50 nm in diameter (Kitajima and Costa 1966). The genome consists of double-stranded DNA, which has a length of 8200 base pairs.

The CVMV virus was at first tentatively classified as a member of the caulimovirus group. The complete sequence of CVMV has been determined and the genomic organization differs from that of either the caulimoviruses or badnaviruses (Calvert et al. 1995). The virus will probably be classified as a unique genus of the plant pararetroviruses.

Very little is known about the epidemiology and control of CVMV. The only known host is cassava and the primary mode of dissemination is by infected planting materials. Commercial varieties are rarely found totally infected. Dissemination occurring within the field suggests the existence of a vector as yet unidentified. However, few studies exist on the virus's dissemination and more research is needed to establish the effectiveness of using virus-free material. The virus can remain in a latent state in plants, especially during the rainy seasons of the Brazilian coastal regions.

### Management and control

The disease can be effectively controlled by removing infected plant materials immediately the symptoms appear. Many infected plants seem to tolerate CVMV and produce stems of normal appearance that could be used as good planting material. Although the economic importance of CVMD has not been fully quantified, it can cause losses, especially if it appears at the beginning of the cropping cycle.

## In Brief: Viral Diseases in South America

- In South America, different viral diseases attack cassava. Some are asymptomatic and are not economically important to the crop.
- Common mosaic has been reported in Brazil and other South American countries. This disease develops symptoms of mosaic and chlorosis in infected plants and is transmitted mechanically.
- The vein mosaic virus is found mainly in Northeast Brazil. Infected plants present chlorosis of the veins and, when symptoms are severe, the leaves become deformed and present epinasty. These phenomena are influenced by climatic conditions. The virus can spread from plant to plant and, although its economic importance has not been fully quantified, it can cause losses.

## Cassava Frogskin Disease

### Background and distribution

Cassava frogskin disease (CFSD) was first reported in 1971, in the Department of Cauca, southern Colombia (Pineda et al. 1983). Its place of origin seems to be the Amazon Region of Brazil or Colombia, where it infects the different cassava varieties cultivated by indigenous communities. However, the farmers assumed that the disease was a physiological disorder associated with the varieties and, therefore, did not report it earlier.

In the Amazon, the disease is known as *lagarto-jacaré* because the symptoms expressed by roots resemble that lizard's skin. Along the North Coast, Colombia, in 1981, an allegedly new disease called "Caribbean mosaic" was reported as presenting symptoms of mosaic in the leaves of cassava variety Secundina (Calvert 1994). Research demonstrated that *lagarto-jacaré*, caiman-lizard disease, and Caribbean mosaic are all the one and same CFSD.

Of the cassava diseases, frogskin is considered to be one of the most damaging to the crop (Lozano and Nolt 1989), as it directly affects root production, causing yield losses of 90% or more (Figure 16-3).

By the 1980s, the disease had appeared in most cassava-producing regions of Colombia and was

Figure 16-3. General appearance of roots infected by cassava frogskin disease.

steadily spreading. It has now been reported from Brazil, Costa Rica, Panama, Peru, and Venezuela. In Panama's case, this disease was first detected in 1999, in cassava plots planted with materials that came from Costa Rica, a country that had already reported the disease.

## Description

Symptom expression is variable according to temperature and genotype. In most varieties, infected plants do not present visible signs in their aerial parts, which sometimes appear healthy and vigorous. Stems of these plants are thicker, especially at the base of the plant, as the increased thickness of stems is related to the lack of starch accumulation in roots. However, because these stems are thick, farmers tend to select them as planting materials.

In the roots, symptoms range from very mild to severe, depending on the plant's age and climatic factors (Figure 16-4). Dry or hot conditions tend to inhibit the development of symptoms, whereas cooler conditions favor expression. Even in mildly infected plants, economic losses occur because of the lack of starch accumulation.

Symptoms consist of small longitudinal fissures located near the callus where the roots originate. They then continue appearing along the roots' length. As the young roots increase in diameter, the fissures tend to scar, giving the lesions a lip-like form. As the roots mature, the lesions increase in size and number, and join to create the appearance of a net or honeycomb. The root peel or epidermis appears cork-like and easily comes off. According to the severity of symptoms, the lesions' depth and number increase until the roots become deformed. All these symptoms may occur

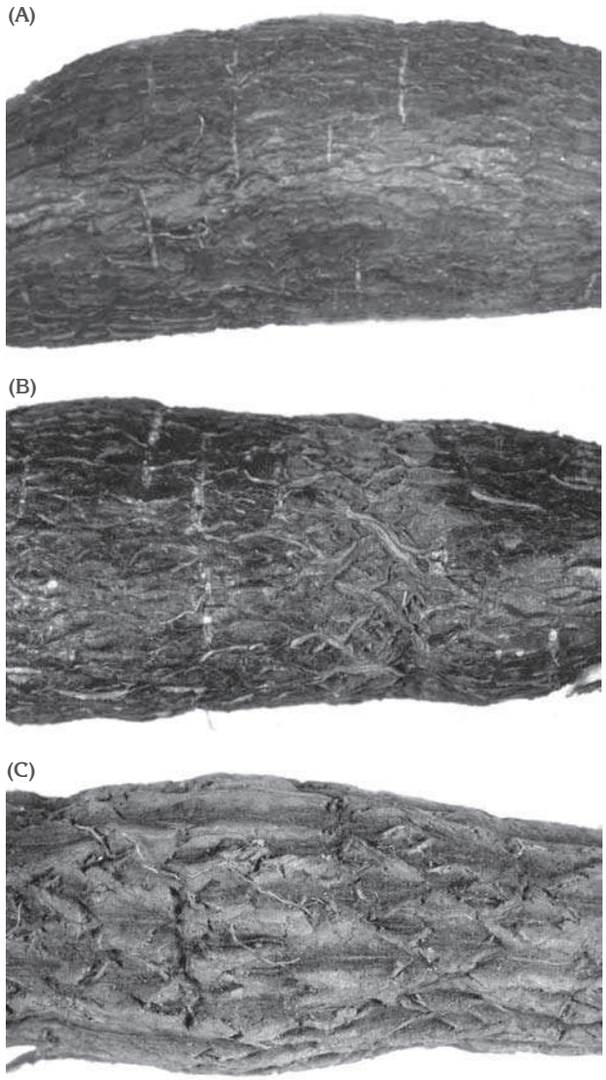


Figure 16-4. Symptoms of frogskin disease in cassava roots: **(A)** healthy root; **(B)** root with mild symptoms; and **(C)** root with severe symptoms.

throughout the root's length or may be restricted to one part, mostly towards the middle.

The root system of infected plants usually does not develop in the same way as healthy plants. The roots remain thin and woody, with a thick, cork-like peel. Their starch content is very low. Sometimes, within one plant, some roots bulk normally, although they may be more fibrous, while others are severely affected.

## Diagnosis

The disease can be detected by carefully examining the roots for the characteristic symptoms, whether these are mild or severe.

This disease is easily transmitted through grafts. Hence, grafting can be used as another diagnostic method. The test consists of grafting an indicator variety (such as 'Secundina', accession CIAT M Col 2063) that has been duly certified as virus-free onto the plants being evaluated (Figure 16-5).

To increase the germination rate of the grafts, buds are best removed from the stocks. After 3 or 4 weeks, the plants should be checked to confirm the presence of symptoms such as mosaic in the foliage of shoots, thus indicating the disease's presence. To ensure effective appearance of symptoms, grafts must be kept at an average temperature of 28 °C. Where they are grown in a greenhouse or screenhouse, they may be placed under tables.

The disease may be eliminated through thermotherapy and *in vitro* meristem culture (Mafla et al. 1984). Once treated, grafts must be made with variety Secundina to confirm the planting materials' health.

Figure 16-5. Detecting cassava frogskin disease through grafting test.

Notable progress in the characterization and detection of the virus associated with this disease has led to the development of a molecular diagnostic method, using RT-PCR (Reverse Transcriptase-PCR). Comparative studies of the two methodologies available for detecting this virus have shown that new molecular technology of detection is more effective and reliable than the symptomatology and the use of warning plants.

## Etiology and epidemiology

Identifying the causal agent of CFSD has been a challenge since the disease was first discovered. However, based on 30 years of experimental data and advances made in the development and implementation of molecular techniques, the disease has been associated with a reovirus—the CFSV (Cuervo 1990; Calvert et al., 2008).

The presence of virus-like isometric particles of about 70 nm in diameter was observed through the electron microscope in tissue sections from cassava leaves, petioles, stems, and roots.

So far, nine species of viral double-stranded (ds) RNA are associated with this disease, and complementary DNA (cDNA) clones to six genomic segments have been synthesized from purified viral dsRNAs. The putative proteins predicted from the sequence of the cassava viral cDNA clones obtained show similarities to the P1, P2, P3, P4, P5, and P10 proteins of rice ragged stunt (reo)virus (RRSV). Phylogenic analyses confirm that CFSV is a member of the family Reoviridae and that it is most closely related to RRSV (Calvert et al. 2008).

This virus has been detected in samples collected in different regions of Colombia and has never been detected in healthy plants.

To date, 30% of the reovirus's genome has been sequenced and the existence of genetic variability in this virus was verified by examining infected plants collected from different regions of Colombia. Molecular analysis of the samples revealed at least three different strains of the virus (Calvert et al. 2008; Cuervo 2006).

Field studies on transmission indicate that frogskin disease spreads from plant to plant. Although the transmission rate is relatively low, compared with many plant viruses transmitted through a vector, dissemination patterns suggest that the disease is transmitted by an aerial vector.

The whitefly *Bemisia tuberculata* has frequently been associated with the disease (Angel et al. 1990), but the insect's efficiency in transmitting it is low. Although more than 100 experiments on transmission through *B. tuberculata* were conducted, no correlations were found between the number of insects and the percentage of transmission. This indicates that the vector of this disease has not yet been clearly identified.

When the percentage of plants infected by CFSD is low, dissemination of the disease is very slow. Even so, if due precautions are not taken in each cycle, the incidence of CFSD increases. The higher the number of infected plants in the field, the faster the rate of dissemination becomes. Use of vegetative seed (stakes) from infected cassava fields therefore becomes the disease's main mode of dissemination.

Parallel research at CIAT has also associated CFSD with a phytoplasm (see Chapter 8). PCR techniques allowed the detection of a phytoplasm in leaves infected with frogskin disease. (Álvarez et al. 2009)

### Resistance

Field studies have demonstrated that different levels of resistance exist among cassava varieties. The number of lines presenting significant levels of resistance after several years of evaluation indicates that the use of resistant materials would be the most useful measure for controlling this disease. Resistant lines lose less starch and suffer fewer yield losses, compared with susceptible lines.

### Management and control

The following recommendations are aimed at preventing the introduction and dissemination of frogskin disease in cassava-producing areas:

1. As the disease spreads mainly through the use of contaminated stakes, the most important control measure is to obtain planting materials (stakes) from healthy plantings that have been technically managed, with excellent plant health control.

2. At harvest, the stakes selected for future planting should be placed beside their respective roots. Later evaluation will confirm the absence of symptoms.

3. As a method of integrated pest management, tools should be disinfected with detergent or chlorine solution.

4. Heavily infected cassava plantings (i.e., at more than 10%) must be burned, including both roots and aerial parts. Harvest residues, particularly stems, should also be eliminated because they can re-sprout.

5. Systems of plant health surveillance and quarantine must be strengthened to prevent the introduction of infected planting materials to national territory, or their mobilization within that territory.

### Cassava frogskin disease in brief

- Frogskin disease is a serious disease for the cassava crop because it directly affects root production and can cause yield losses of more than 90%.

- The disease has been continually spreading and already affects other regions of Colombia and other countries.

- Symptom expression varies with temperature and cassava genotype.

- The root system of infected plants usually does not develop to the same extent as healthy roots. Instead, they become thin and woody, with very low starch content.

- Although the causal agent has not yet been fully identified, research to date suggests that it is probably of viral origin. However, its association with a phytoplasm, or a combination of both types of organisms, has not been ruled out.

- The disease spreads mainly by planting contaminated vegetative seed (stakes). It also appears to be transmitted, albeit slowly, by an aerial vector.

- Different levels of resistance exist among cassava varieties. Hence, with the use of tolerant varieties, healthy planting materials, and good plant health control, CFSD is one disease that can be controlled.

## Viral diseases in Africa

In terms of economic and social importance, perhaps the most relevant cassava diseases that are propagated by infected planting materials are: cassava mosaic disease (CMD) and cassava brown streak disease (CBSD), two viral diseases only present in Africa and, in the case of CMD, India and Sri Lanka as well (Monger et al. 2001; Calvert and Thresh 2002; Thresh et al. 1994). Because these diseases are not present in the Neotropics, CIAT has not carried out much research on them. Major research achievements on these diseases have been made at the International Institute of Tropical Agriculture (IITA) based at Ibadan, Nigeria, and other collaborating institutions.

Cassava mosaic disease has attracted the attention since long time ago and considerable knowledge on the disease and its vector (the whitefly *Bemisia tabaci*) is available (Legg and Fauquet 2004; Legg and Thresh 2000; Thresh and Cooter 2005; Patil and Fauquet 2009). Symptoms in the leaves are characteristic and easy to identify (Figure 16-6) although variable from a green mosaic to a yellow mosaic, distortion of leaflets, rupturing of tissue, and premature leaf abscission. Resistance to CMD has been identified and analyzed (Fargette et al. 1996; Hahn et al. 1980; Thresh et al. 1998) or developed through genetic transformation (Zhang et al. 2005). Molecular markers associated with resistance to CMD have been identified and successfully used (Akano et al. 2002; Okogbenin et al. 2007). More recently there have been reports on the association of at least two different satellite DNAs with CMD (Ndunguru et al. 2008; Patil and Fauquet 2010).

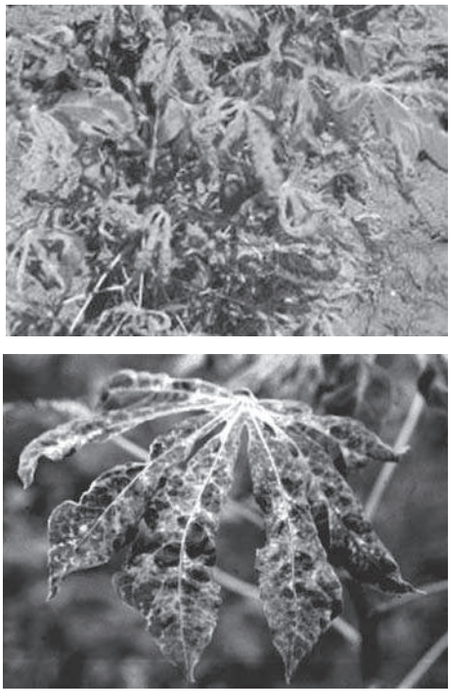

Figure 16-6. Cassava mosaic disease (CMD) in cassava. (Photos: Legg, Owor, and Okao-Okuja; G. Mkamilo.)

Cassava brown streak disease, on the other hand, remained a minor disease problem restricted to the coastal areas of East Africa. Recently, however, it started to spread westbound and is now a major concern in many regions of Africa (Hillocks et al. 2002; Hillocks and Jennings 2003). The disease is also transmitted by *B. tabaci* (Maruthi et al. 2005) and has been characterized from the molecular point of view (Mbanzibwa et al. 2009a, 2009b; Monger et al. 2001; Monger et al. 2010). CBSD is named after the brown elongated necrotic lesions that often develop on young stem tissue as well as in roots (Figure 16-7). Necrosis

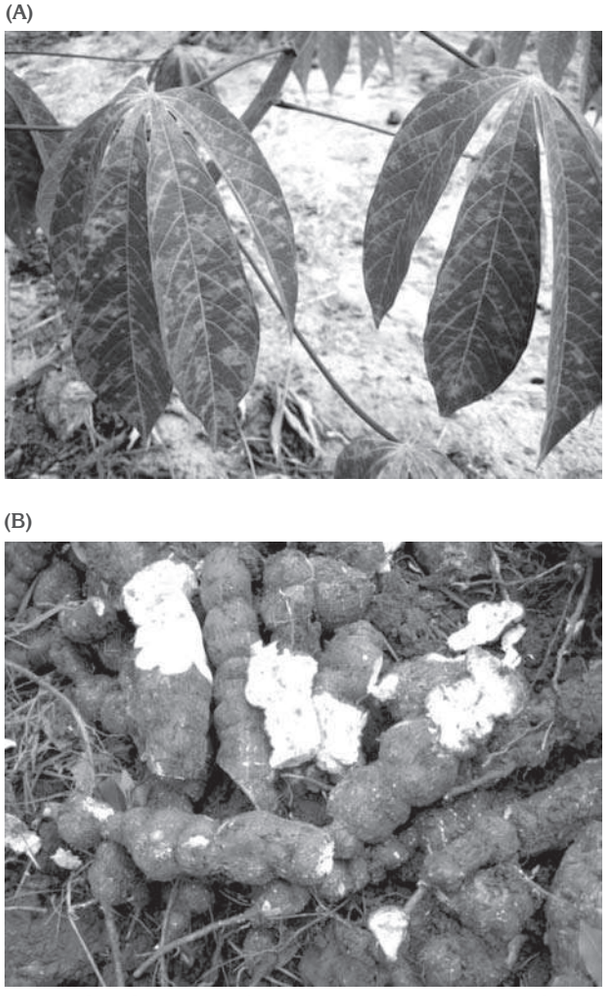


Figure 16-7. Symptoms of cassava brown streak disease (CBSD) on **(A)** leaves in Tanzania and **(B)** roots in Uganda. (Photos: R. Howeler.)

in the roots greatly reduces their economic value. The degree of root necrosis and the characteristic constrictions associated is variable with some varieties only expressing these symptoms late in crop growth (Calvert and Thresh 2002). Symptoms can only be observed in the leaves but are highly variable ("featherly" chlorosis to yellow blotches associated to leaf veins) and often inconspicuous.

In response to the expanding relevance of CBSD, efforts to develop tolerant/resistance cultivars have increased in recent years. New sources of resistance seem to have been found in a backcross population involving *M. esculenta* subsp. *flabellifolia* (M Fregene 2012, pers. comm.).

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