CHAPTER 8

Cassava Diseases

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World production of cassava roots was estimated at 233 million tons in 2008. Africa was the largest producer with 118 million tons on almost 12 million hectares, followed by Asia with 78.7 million tons on 3.97 million ha. Cassava (*Manihot esculenta* Crantz) is a significant staple, providing a basic daily source of dietary energy for almost one billion people in 105 countries. It also has numerous agroindustrial uses. Cassava grows on marginal lands, tolerates drought, and can grow in low-fertility soils. Cassava is also the most inexpensive source of starch that exists, being used in more than 300 industrial products (FAOSTAT, 2010).

Cassava is still widely cultivated under traditional management. This suggests that large numbers of farmers may be ignorant of the crop's diseases and their integrated management. Hence, several diseases threaten the sustainability of cassava production and its profitability. The principal diseases attacking the crop are:

Cassava bacterial blight (CBB[4]; *Xanthomonas axonopodis* pv. *manihotis* or *Xam*)
Phytophthora root rots (PRR; *Phytophthora* spp.)
Superelongation disease (SED; *Sphaceloma manihoticola*)
Cassava frogskin disease (CFSD; *Candidatus* phytoplasma, Cfdp of the 16SrIII-L and rpIII-H subgroups)
Cassava mosaic disease (CMD; begomovirus complex)
Cassava brown streak disease (CBSD; an ipomovirus)
Brown leaf spot (*Cercosporidium henningsii*)
Diffuse leaf spot (*Cercospora vicosae*)
White leaf spot (*Phaeoramularia manihotis*)
Anthracnose (*Colletotrichum* spp.)

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

Diseases Caused by Fungi

Superelongation disease (*Elsinoe brasiliensis*)

Importance. Superelongation disease (SED) attacks susceptible cultivars, especially during the rainy seasons. Damage caused by SED is highly variable, depending on the level of cultivar resistance, climatic conditions, concentration of the initial inoculum, and the degree of contamination of planting materials (Álvarez and Llano 2002).

Losses can exceed 80% of total production in young crops, whereas significant losses do not occur in crops that are more than 6 months old. In Colombia, SED is found in the Eastern Plains, Atlantic Coast, and inter-Andean valleys. The disease is acute in agroecological areas with annual mean temperatures of 28 °C and annual precipitation of more than 1500 mm. In the greenhouse, 8 h of misting at temperatures of 25 to 30 °C was sufficient to cause an outbreak, indicating how easily the pathogen develops in the field (Mejía 2001).

Distribution. Superelongation disease was first observed by Bitancour and Jenkins in 1950, on *Manihot glaziovii* Muell.-Arg. in Brazil and Nicaragua and on *M. esculenta* in the Dominican Republic and Guatemala. The disease has since been reported (in order of reporting year) in Costa Rica (Larios and Moreno 1976), Colombia (Lozano and Booth 1979), Mexico (Rodríguez 1979), Cuba (Pino 1980), Venezuela (Rondón and

Aponte 1981), the Dominican Republic (Sosa 1992), Barbados, Panama (Chávez 1992; Zeigler 2000), Brazil (where it is restricted to the western regions of the country) (Álvarez et al. 2003d), and Trinidad and Tobago (Reeder et al. 2008). At the end of 2008, the disease was detected in Thailand (E Álvarez 2008, pers. comm.). The disease appears to be unknown in Africa.

Symptoms and epidemiology. The characteristic symptom of this disease is the exaggerated lengthening of stem internodes (Zeigler et al. 1980), creating thin and weak stems. Diseased plants are much taller and/or weaker and spindlier than healthy ones. In green sections of stems, and in petioles and leaves, deformations develop in associations with cankers. The lens-shaped cankers often have dark margins and are variable in size. In leaves, cankers are found on the underside, along the primary or secondary nervures. In stems, they may be more diffuse. Frequently, young leaves curl, and do not develop fully nor do the leaf blades expand completely. Leaves also develop irregular white spots (Figure 8-1). Sometimes partial or total death of leaves occurs, resulting in considerable defoliation. Dieback of the plant may also occur.

The disease spreads from one place to another through the use of infected stakes. The principal focuses of infection frequently constitute the shoots originating from residues of old plants left in the field after harvest. The disease spreads rapidly during the rainy season. This rapid dissemination is believed to occur through the formation of spores in the cankers. These spores can survive for more than 6 months in infected plants and are carried by rain and wind.

Etiology. Superelongation disease is caused by the fungus *Elsinoe brasiliensis*, which initially grows on the epidermis of the host and, after penetration, grows in the intercellular spaces in tissues of the epidermis and cortex. The fungus produces gibberellins, which promote the exaggerated growth in the plant's internodes. Gibberellins, as suggested by previous studies for other pathogens (Muromtsev and Globus 1975), play an essential role in the fungus's nutrition. The fungus, which has a low production of hydrolytic enzymes, uses this hormone to obtain sugars from the plant, promoting, at the molecular level, hydrolysis of carbohydrates with greater mass (Mejía 2001).

According to Álvarez and Molina (2000), the pathogen's genetic diversity in Colombia is broad, presenting differences among isolates within a single location and between locations. Isolates from the Atlantic Coast, Eastern Plains, and inter-Andean valleys of Colombia and from central and southern Brazil comprise two evolutionary units, with each unit relating to its respective country (Álvarez et al, 2001).

For gene 18S rRNA, obtained from two isolates of *E. brasiliensis*, the sequencing of a region involving ITS1 and ITS2 was reported to GenBank (accessions AY739018 and AY739019; CIAT 2004).

Host range. *Elsinoe brasiliensis* and *Sphaceloma* species (the asexual state), which both attack cassava, have a wide range of Euphorbiaceae hosts, including *Euphorbia brasiliensis* L., *E. hypericifolia* L., *Jatropha aconitifolia* Muell. var. *papaya* Arbelaez, *J. curcas* L., *Manihot carthaginensis* Muell., *M. esculenta*, and *M. glaziovii*. These hosts are cosmopolitan weeds and widely cultivated ornamentals.

Many regions in Africa and Asia have climatic conditions that closely resemble to those of the Eastern Plains, Atlantic Coast, and inter-Andean valleys of

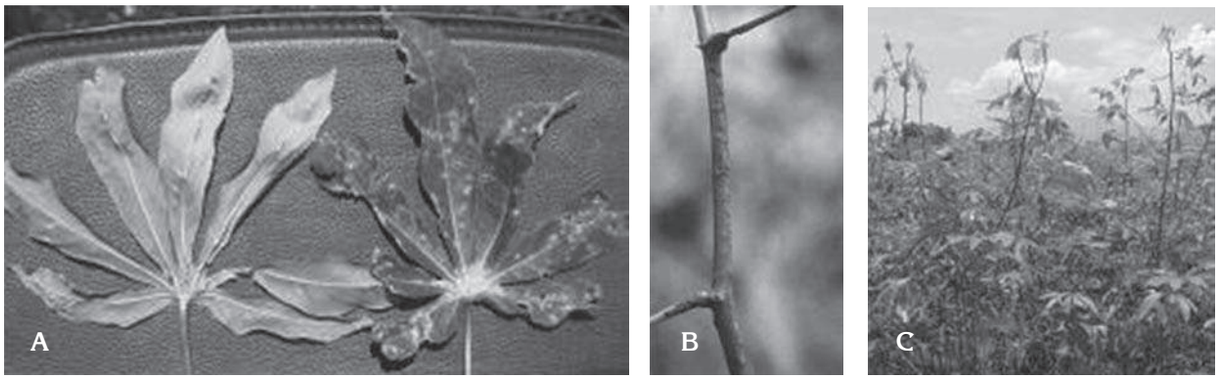

Figure 8-1. Symptoms of superelongation disease in cassava: **(A)** cankers on leaves, **(B)** cankers on petioles and stem, and **(C)** elongated stem.

Colombia, where the pathogen causes considerable losses. These African and Asian regions therefore face the danger that the pathogen will be introduced through planting materials of ornamentals such as *Jatropha* spp. L., which are not necessarily restricted by the same sanitary regulations as cassava.

Because the host range is broad, completely eradicating the pathogen is impossible and a certain amount of sufficient inoculum will be present throughout the year. In Brazil, the weed *Euphorbia heterophylla* L. was shown to be host to strains of *Elsinoe brasiliensis* that were highly pathogenic to cassava (Álvarez et al. 2003d). Furthermore, the genetically very variable hosts are also able to maintain a variable population of the pathogen (Zeigler 2000).

Integrated disease management. The use of healthy seed, obtained from disease-free plants or from plants derived from meristem culture, comprises a tool that may be sufficient to maintain disease-free crops. However, one preventive method for eradicating the pathogen is to immerse infected stakes for 10 min in captafol at 4.8 g/L of active ingredient (a.i.). When symptoms are observed in the field, foliar spraying should be carried out with difenoconazole at 0.07 cc/ha, followed by crop rotation with grasses.

In areas where the pathogen is endemic, planting should be carried out during periods with the least precipitation (CIAT 2003b). Infected plants (cassava or other Euphorbiaceae hosts) should be destroyed as soon as they are identified. The best way to eliminate this material is to pull up infected plants and burn them *in situ* (Zeigler 2000).

Varietal resistance. The selection of resistant varieties is perhaps the best alternative for controlling SED. Between 1995 and 2007, CIAT evaluated about 6400 genotypes at Villavicencio (Colombia) and found 257 with resistance to SED. On-farm evaluations at Sincelejo (Sucre, Colombia) showed the following as resistant: M Ven 25 and CM 4843-1, followed by ICA Catumare, ICA Cebucán, ICA Negrita, Vergara (CM 6438-14), and CM 4574-7 (CIAT 2001, 2002b, 2003a).

Pathogenic races of *E. brasiliensis* exist and are of high genetic variability. While they should be taken into account when improving resistance to SED (Álvarez and Molina 2000; Álvarez et al. 2003d), they are not thought to pose serious constraints to varietal improvement (Zeigler 2000).

Biological control. Spraying with suspensions of *Pseudomonas putida* considerably reduced the severity of damage caused by SED, thereby significantly increasing cassava yields (CIAT 1985).

Brown leaf spot (*Cercospora henningsii*)

Importance. Brown leaf spot has a broad geographical distribution, being found in Asia, North America, Africa, and Latin America. It attacks naturally *M. esculenta*, *M. glaziovii*, and *M. piauhynsis* Ule (Ferdinando et al. 1968; Golato and Meossi 1966; Powell 1972). In India, *Cercospora henningsii* is an important pathogen, causing severe defoliation (Edison 2002).

Symptoms and epidemiology. Symptoms in cassava leaves are characterized by leaf spots visible on both sides. On the leaves' upper surface, uniform brown spots appear, with defined and dark margins. On the leaves' undersurface, the lesions have less-defined margins and, towards the center, the brown spots have a gray-olive background because of the presence of the fungus's conidiophores and conidia. As these circular lesions grow, from 3 to 12 mm in diameter, they take up an irregular angular form, their expansion being limited by the leaves' major veins (Figure 8-2).

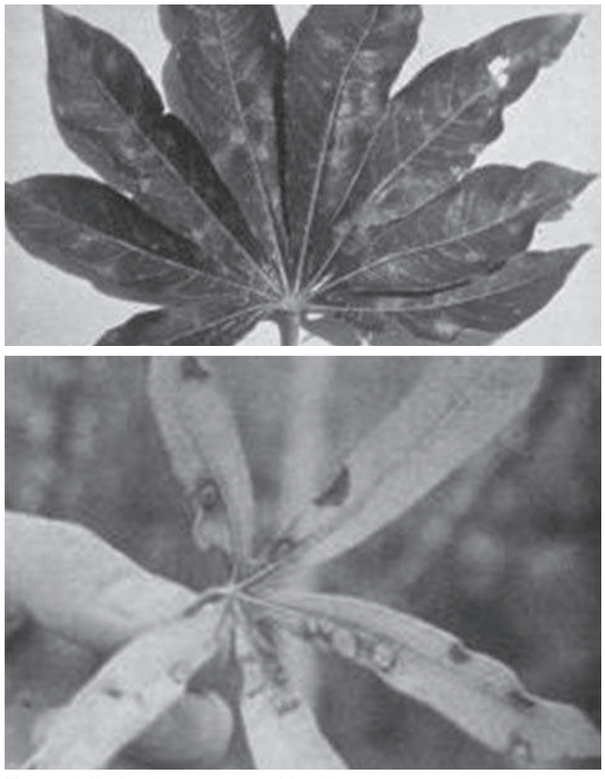

Figure 8-2. Leaf spots caused by *Cercospora henningsii*.

The veins found within the necrotic area are black. Sometimes, depending on how susceptible the variety is, an undefined yellow halo or discolored area can be observed around the lesions. As the disease progresses, infected leaves become yellow and dry before falling off, possibly because of toxic substances secreted by the pathogen. Susceptible varieties may undergo severe, or even total, defoliation during the hot rainy season.

When wind or rain carry conidia that have dropped from wounds of infected tissues towards leaves of a new planting, primary infections occur. If environmental humidity is sufficiently high, the conidia will germinate, producing branched germinal tubes that frequently anastomose (Chevaugeon 1956; Viégas 1941).

When lesions mature, stromata appear from which conidiophores emerge. Secondary cycles of the disease are repeated throughout the rainy season, when wind or rain carries conidia to new susceptible tissues of the plant. The fungus survives the dry season in old lesions, frequently those of fallen leaves. It renews activity with the advent of the rainy season and growth of new leaves in the host.

Chevaugeon (1956) observed that, in a cassava plant, the lower leaves are more susceptible than the youngest leaves. However, certain susceptible species (e.g., *M. carthaginensis* Muell.) and *M. esculenta* cultivars can be severely attacked. Severe symptoms have been observed in young leaves, petioles, and even fruits of *M. carthaginensis*. Although plants "hardened" by unfavorable conditions appear more resistant, no significant differences in susceptibility were found between plants growing in fertile soils and those growing in poor soils (Chevaugeon 1956).

Etiology. *Cercospora henningsii,* causal agent of the disease, grows in the intercellular spaces of leaf tissues, producing stromata from which conidiophores are produced in dense fascicles. The conidiophores are pale olive brown, semi-transparent, with uniform width and color, and non-branching. Sometimes, black perithecia appear, disseminated in the necrotic tissue of leaf spots and on the leaves' upper surface (Powell 1972). The perfect state of *C. henningsii* is *Mycosphaerella manihotis* (Ghesquière 1932; Chevaugeon 1956).

Management and control. To reduce the severity of infection, recommended cultural practices include reducing excess humidity during planting (Golato and Meossi 1966). Fungicides based on copper oxide and copper oxychloride, suspended in mineral oil, and applied at 12 L/ha also provide good chemical control (Golato and Meossi 1966). The best control over the disease can be achieved by using resistant varieties. Significant differences in varietal resistance have been found in Africa (Chevaugeon 1956; Umanah 1970), Brazil (Viégas 1941), and the extensive collection of cassava varieties held at CIAT, Colombia (CIAT 1972).

Diffuse leaf spot (*Cercospora vicosae*)

Importance. This disease is found where brown leaf spot predominates, that is, in the hot cassava-growing areas of Brazil and Colombia (CIAT 1972; Viégas 1941). The pathogen causes severe defoliation in susceptible cultivars but, in Colombia, does not cause heavy crop losses.

Symptoms and epidemiology. This disease is characterized by the presence of large leaf spots, with undefined margins. Each spot may cover one fifth, or more, of the leaf lobe. On the leaves' upper surfaces, the spots are uniformly brown, whereas, on the lower surfaces, spots also have grayish centers caused by the presence of the fungus's conidia and conidiophores. The spots' general appearance is similar to that of the spots induced by *Phoma* sp., although lesions induced by the latter have concentric rings on the leaves' upper surfaces (Figure 8-3).

Defoliation may occur in susceptible cultivars, being more severe at the end of the rainy season and/or vegetative cycle. As the disease progresses, leaves become yellow and dry before falling off.

Symptoms of this disease can be confused with those of cassava bacterial blight (CBB; see below), except that the blight lesions are noticeably aqueous.

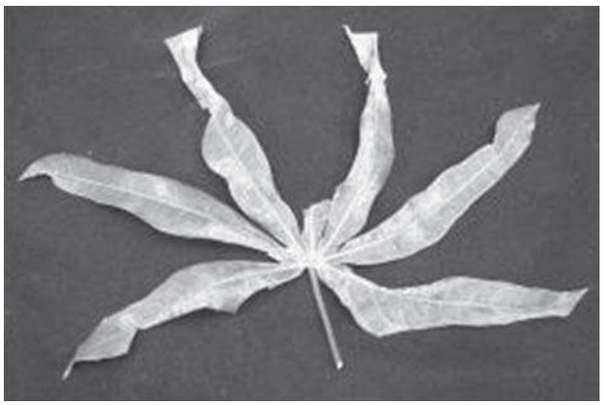

Figure 8-3. Leaf spots caused by *Cercospora vicosae* in a cassava leaf.

Etiology. The fungus does not form stromata but sporulates abundantly. The conidiophores are reddish dark brown (Chupp 1953). The fungus has been recorded as a pathogen occurring only on *Manihot* spp. Mill. As its incidence on a single plant or in a given planting is very low and apparently confined to the plant's lower leaves, its importance is relatively less.

Management and control.

- Planting with healthy and resistant cultivars
- Using cultural practices that reduce humidity during planting

White leaf spot (*Phaeoramularia manihotis*)

Importance. This fungus is commonly found in the cold humid cassava-growing regions of Asia, America, North America, tropical Africa, and Latin America (Castaño 1969; Chevaugeon 1956; CIAT 1972). In these areas, the pathogen may cause considerable defoliation in susceptible varieties of *M. esculenta*, the only known host species (Chevaugeon 1956; Viégas 1941).

Symptoms and epidemiology. Leaf spots caused by *P. manihotis* are smaller, with a different color, to those induced by *C. henningsii*. They vary from circular to angular, with diameters of usually 1 to 7 mm. They are normally white, but sometimes yellowish brown. Lesions are sunken on both sides, to half of the thickness of a healthy leaf blade. On the lower leaf surface, the white spots can be distinguished but they frequently have diffusely colored margins, which sometimes appear as brown-violet irregular lines, surrounded by brown or yellowish halos. The spots' centers have a velvety grayish aspect during the pathogen's fruiting (Figure 8-4).

The fungus penetrates the host through stomatal cavities and then invades the host's tissues through the intercellular spaces. When leaf spots reach 5 to 7 mm in diameter, a stroma is formed, which produces conidiophores. The disease's secondary cycles are repeated throughout the rainy season as conidia are dispersed by wind or rain splash. The fungus survives the dry season in old infected tissues and renews activity at the beginning of the rainy season and with the host's new growth.

Etiology. *Phaeoramularia manihotis*, the causal agent, forms thin stromata in lesions on leaves. The stromata produce conidiophores in loose fascicles that emerge through the stromata and are usually olive brown (Powell 1972).

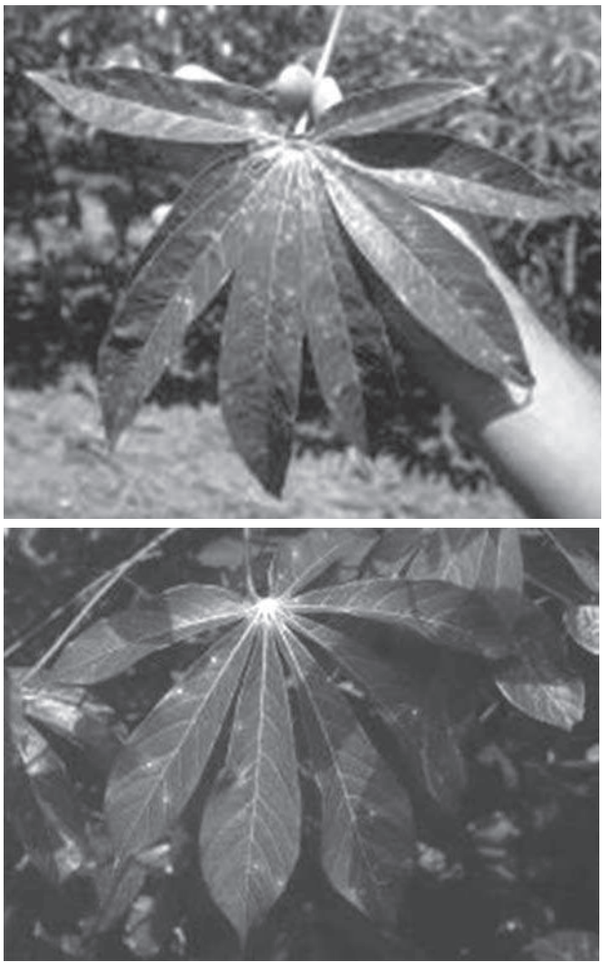

Figure 8-4. Leaf spots caused by *Phaeoramularia manihotis*.

White leaf spot is very similar to brown leaf spot. However, brown spot usually occurs in warm but not humid areas, whereas white spot appears in cold humid areas. These differences in their geographical distribution are also observed in Africa and Latin America, and are probably the result of different responses of the respective causal agents to temperatures and humidity. The optimal temperature for germinating *C. henningsii* conidia is 39 °C, with a maximum temperature of 43 °C. For *P. manihotis*, these temperatures are, respectively, 33 and 43 °C (Chevaugeon 1956).

Management and control. The control measures recommended for this disease are similar to those for brown leaf spot. Specifically resistant varieties are unknown, but field studies suggest they exist (JC Lozano 1979, unpublished data).

Concentric ring leaf spot (*Phoma* spp.)

Importance. This fungal disease, caused by *Phoma* spp., usually appears in the cold cassava-growing areas of Colombia (CIAT 1972), Brazil (Viégas 1943a), Philippines, tropical Africa, and India (Ferdinando et al. 1968). According to Edison (2002), this disease is an emerging problem in certain areas where cassava cultivation is intensive. During the rainy season and when the temperature is below 22 °C, the disease may cause severe defoliation in susceptible varieties and almost always produces stem dieback.

Symptoms and epidemiology. The disease is characterized by the presence of large dark brown leaf spots, with usually undefined margins. These lesions are commonly found at leaf points, margins of leaf lobes, or along the central vein or other secondary veins. Initially, lesions appear as concentric rings of brown pycnidia on the leaf's upper surface (Figure 8-5). These rings are not found on old injuries because the rain drags away mature pycnidia. In these cases, the spots are uniformly brown, and are very similar to those caused by *Cercospora vicosae*. On the lower leaf surfaces, very few pycnidia occur. Hence, lesions are uniformly brown.

Under conditions of high relative humidity, lesions may be covered by braid-like chains of grayish-brown hyphae. On the lower leaf surfaces, the nervures within the lesions become necrotic, forming black bands that emerge from the spots. These spots grow, causing leaf blight. The fungus invades the infected leaf and then the petiole, which becomes dark brown as it necroses. Leaves wilt and then fall, resulting in severe defoliation in susceptible cultivars. These cultivars may present dieback during epiphytotes and even total plant death. Necrotic stems become dark brown and frequently appear covered with pycnidia.

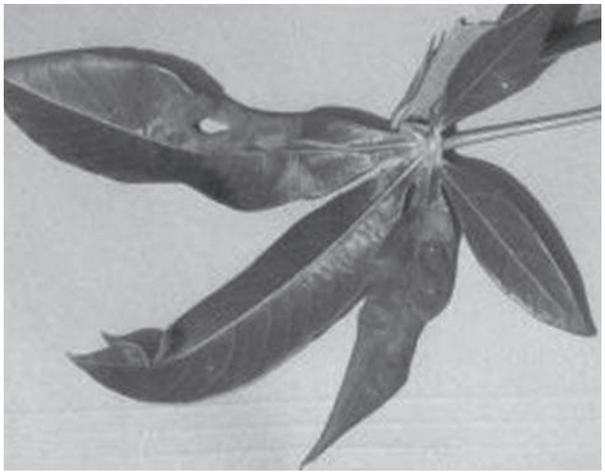

Figure 8-5. Leaf spots caused by *Phoma* sp. in cassava.

Field studies suggest that the more mature lower leaves may be more resistant than the young upper leaves. However, total defoliation, accompanied by partial or total dieback, has been observed in susceptible cultivars.

Favorable conditions for the germination of fungal spores occur at temperatures between 20 and 25 °C. With artificial inoculation, infection is only achieved when inoculated plants are kept for 48 h at less than 24 °C and with 100% relative humidity (JC Lozano 1979, unpublished data). Under field conditions, disease always occurs during the rainy season and in areas where the temperature is less than 22 °C.

The fungus's survival mechanism during dry hot periods is unknown. Viégas (1943b) suggested that the fungus may produce its sexual state on infected stems and leaf residues. However, this has not yet been observed or recorded.

Etiology. The causal agent produces numerous, spherical, dark brown pycnidia, either individually or in small clusters, on surfaces of leaves or stems. Pycnidia measure 100–170 μm in diameter, their walls are formed by polyhedral cells; and their ostiole measures 15–20 μm. Conidiophores are short and hyaline, producing small conidia (15–20 μm) that are unicellular and ovoid or elongated (Ferdinando et al. 1968; Viégas 1943a). On Lima-bean agar, the fungus forms pycnidia in profuse quantities, appearing in concentric rings.

Management and control. To date, no measures of control exist for the disease, even though it causes heavy losses in areas where environmental conditions are propitious for its development. Although no reports exist on varietal resistance, in the field in Colombia, resistance has been observed in naturally infected plantings. Chemical treatments such as carbendazim (3 g/L a.i.) and benomyl (0.6 g/L a.i.) during the rainy season may be equally effective in those areas where the disease is endemic.

Cassava ash (*Oidium manihotis*)

Importance. This disease was first recorded in Africa in 1913 (Saccardo 1913) and has since appeared in Latin America (CIAT 1972; Viégas 1943a) and Asia (Park 1934). The disease is characterized by the presence of yellowish undefined spots on *M. esculenta*

leaves. Although it is widely disseminated and frequently occurs during the dry season, the disease is considered to be of minor importance as it usually attacks only the lower leaves, in which it induces some necrosis.

Symptoms and epidemiology. The first symptoms of disease are characterized by the appearance of a white mycelium that grows on the leaf surface (Figure 8-6). The fungus penetrates the host cells, using haustoria. The infected cells become chlorotic and form undefined yellowish lesions. Within these yellowish areas, pale brown necrotic areas frequently appear. These are angular in shape and of different sizes. In some cassava varieties, the disease stops in the state of yellowish undefined lesions, which then may become confused with those induced by insects and mites.

Fully developed mature leaves seem to be most susceptible to pathogenic attack, although the young leaves of some varieties may also present symptoms. The disease commonly appears during the dry season and in warm areas.

Etiology. The sexual state of the causal agent, *Oidium manihotis*, is *Erysiphe manihotis* (Ferdinando et al. 1968). The fungus's mycelium is white, producing numerous haustoria on the host's epidermis. Conidiophores rest in an erect position. They are simple, with the upper parts both longer and wider, as they form the conidia. Conidia are oval or cylindrical, unicellular, hyaline, and measure 12–20 × 20–40 μm. They are produced in basipetal chains (Ferdinando et al. 1968; Saccardo 1913; Viégas 1943b).

Management and control. Although specific control of the disease is considered unnecessary, observations suggest that resistant varieties exist (CIAT 1972). Ferdinando et al. (1968) suggest that spraying with sulfur-based compounds can control the disease.

Figure 8-6. Cassava ash symptoms, caused by *Oidium* sp.

Cassava anthracnose (*Glomerella manihotis*)

Although cassava anthracnose has been known for a long time, it has been considered of minor importance. It is characterized by the presence of sunken leaf spots, 10 mm in diameter, that are similar to those caused by *C. henningsii*. The latter, however, appear towards the base of leaves, thus causing their total death.

The pathogen also causes young stems to wilt and induces cankers on mature stems (Irvine 1969) (Figure 8-7). New leaves, produced at the beginning of the rainy season, are the most susceptible. The disease tends to disappear when the dry season begins (Irvine 1969). This finding agrees with results obtained from artificial inoculations with an aqueous suspension of spores from the pathogen. Inoculation is successful if incubation is at 100% relative humidity for 60 h. The fungus will stop invading plant tissue when relative humidity drops to 70% (CIAT 1972). The insect *Pseudotheraptus devastans* Distant is associated with the disease (Fokunang et al. 2000), contributing to the pathogen's dissemination and increasing the severity of symptoms.

The organism causing this disease has been variously called *Glomerella manihotis*, *Colletotrichum manihotis* (Vanderweyen 1962), *Gloeosporium manihotis* (Bouriquet 1946), and *Glomerella cingulata* (Irvine 1969). All these names possibly refer to one species, but this hypothesis is yet to be confirmed.

Stem anthracnose caused by a *Colletotrichum* sp. was recorded in Nigeria (IITA 1972). Green portions of

Figure 8-7. Leaves and stem show cankers caused by *Glomerella manihotis*.

the stems presented shallow oval depressions that were pale brown, but with a point of normal green tissue in the center. In the ligneous portions of the stems, lesions were round, swollen, and in bands, forming deep cankers on the epidermis and cortex, and sometimes deforming the stem. Its importance is unknown but its prevalence, occurrence, and dissemination are considerable. In Asia stem anthracnose was recorded in Thailand (E Álvarez 2009, pers. comm.) (Figure 8-8).

Figure 8-8. Disease symptoms observed on cassava stems.

Cassava rust (*Uromyces* spp.)

Importance. Although recorded in Brazil and Colombia, this disease is considered to be of minor importance. It appears at the end of dry periods, sometimes causing a type of shoot proliferation in stem apices (Normanha 1970).

Symptoms and epidemiology. Infection is characterized by pustule formation on leaf veins, petioles, or green branches (Figure 8-9). Pustules are light to dark brown, depending on their age or class of fungal fructification. Mature pustules are readily parasitized by the fungus *Darluca filum*. They are sometimes surrounded by chlorotic halos and, usually, induce deformation of affected parts. Wind is the principal dissemination agent.

Etiology. In cassava, several species of rust pathogens have been recorded in different parts of the world. However, its incidence and severity are low. Some species of rust appear to occur only where temperatures are moderate and rainfall is high. Other species predominate during hot dry seasons.

Stem rots

In many cassava-growing areas, continuous cassava planting is not possible and stakes must be stored for later propagation. Stored stakes are attacked by three diseases that induce necrosis (CIAT 1972). These diseases considerably reduce stake viability, directly and indirectly, by increasing dehydration and causing necrosis.

Although the three different causal agents have been recognized, the diseases these induce are not

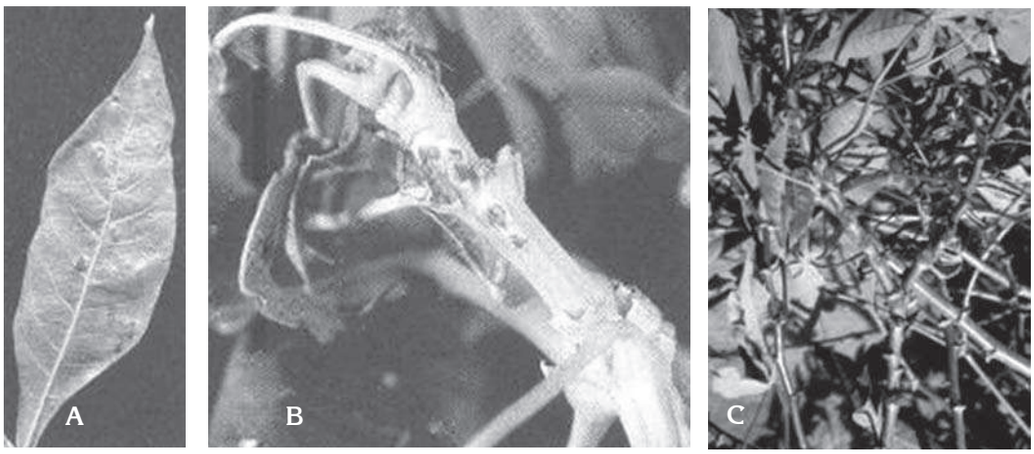

Figure 8-9. Symptoms of cassava rust characterized by pustule formation on **(A)** leaf, and **(B)** and **(C)** stems.

clearly differentiated in most cases. Macroscopically, the diseases look similar, particularly during their first developmental stages. Furthermore, more than one causal agent may be present, creating a syndrome.

The three diseases causing stem rots are stem necrosis caused by *Glomerella cingulata*, dry stem and root rot caused by *Diplodia* sp., and necrosis caused by an unidentified Basidiomycete (Lozano and Booth 1979).

Stem necrosis (*Glomerella cingulata*)

Importance. This disease is the most common of the three that induce rots or necrosis in stored cassava stakes. It also attacks residues of old stems left in cassava plantings.

Symptoms. Necrosis of stored stakes appears first at the ends and then progresses slowly towards the middle, before disseminating to all stakes (Figure 8-10). The disease occurs as a black discoloration of vascular bundles. It then develops surface blisters that later break, exposing groups of black perithecia in well-developed stromata.

Etiology. The causal organism appears to be *Glomerella cingulata* (Commonwealth Mycological Institute 1979, pers. comm.). Ascospores are hyaline, unicellular, and slightly curved. Infection probably occurs through wounds and is favored by high environmental relative humidity.

The relationship between this fungus and *Colletotrichum* sp., which causes anthracnose in cassava, has not still been determined. For example, the appearance of two types of symptoms may be due to two different states of the same agent rather than of two agents.

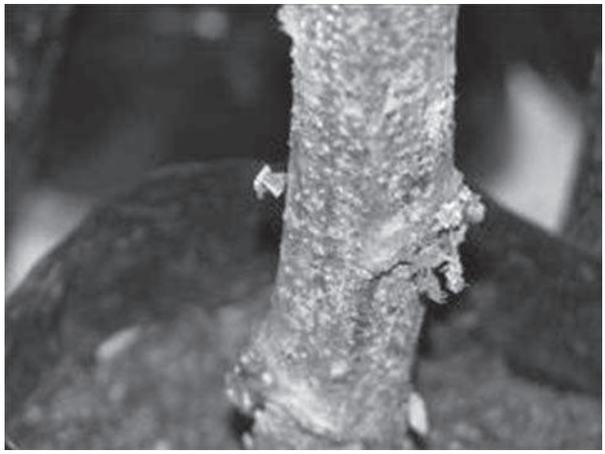

Figure 8-10. Necrosis caused by *Glomerella cingulata* in cassava stakes.

Dry rot of stem and root (*Diplodia* sp.)

Importance. This disease attacks stored cassava planting materials and residue stems left in the field. Its occurrence is not as common as necrosis caused by *Glomerella* spp.

Symptoms and epidemiology. The disease has two phases. The first is when root rot starts when soils are infested or when stakes from diseased plants are used. Symptoms, similar to those induced by root pathogens, consist in sudden plant death caused by root deterioration.

The second phase includes stem rot caused by systemic invasion of the fungus from the roots or by penetration through wounds. The disease is characterized by black discoloration and necrosis of the vascular bundles, which extend from the infection sites, that is, wounds in the stem. In the epidermis, they appear as blisters under which the stem's internal tissues are discolored black or dark brown. The blisters break, showing confluent masses of black pycnidia (Figure 8-11). Gum may be excreted, and partial or total wilting occurs. Dieback may also occur.

The pathogen disseminates across great distances through stakes from infected plantings. Within the same crop, dissemination is by wind and rain during fungal fructifications, use of infested tools and irrigation water, and land preparation for later plantings.

Etiology. The causal agent of dry rot of stem and root is *Diplodia manihotis*. In both the host and laboratory cultures, this organism produces pycnidia

Figure 8-11. Stem rot in a stake infected by *Diplodia* sp.

that erupt through the stem or root surface, becoming confluent, stromal, and ostiolate. The conidiophores are short and simple, producing dark two-cell conidia that are slightly elongated on reaching maturity. Infection is believed to occur through wounds, and is favored by high environmental relative humidity.

Management and control. To control the disease, the cassava crop should be rotated with nonsusceptible crops such as maize or sorghum, particularly when incidence is more than 3%. Planting stakes from healthy crops should be used and tools disinfected. Planting materials should be selected and handled carefully both before and after storage. Only viable cuttings or buds should be planted. One recommendation is to immerse cuttings in a solution of captan (3 g/L) and benomyl (3 g/L) for 5 min. Captan may be replaced by copper oxychloride.

Root rots

Root rots in cassava are important where soils are poorly drained or where excessively rainy seasons occur. In early growth, many microorganisms are capable of inducing not only root rots in young cassava plants, but also in the storage roots of mature plants. Although several root diseases have been reported, little information exists about them. Not even the symptoms are well described.

Usually, infection kills young plants at germination or shortly afterwards. Infection in plants older than 4 months may result in partial or total wilt, depending on whether the root rot is soft or dry. Once invaded by one or more primary pathogens, infected roots may be invaded by a wide spectrum of other microorganisms. These are usually the otherwise weak saprophytic parasites, which become capable of degrading root tissues and masking the identity of the primary causal agent. The resulting root rots therefore appear to have the same syndrome of symptoms.

Pathogens causing root rots include *Phytophthora* spp., *Fusarium* sp., *Scytalidium lignicola, Rosellinia* spp., *Sclerotium* sp., and *Fomes lignosus* (Ferdinando et al. 1968; Jennings 1970; Pereira 1998; Viégas 1955).

Some of these diseases often develop when cassava is planted immediately after woody crops such as coffee. Soils of such crops are infested with pathogens that attack ligneous plants such as cassava. These pathogens may be fungi or bacteria that cause root deterioration, either as the crop grows or after harvest when roots are stored.

Control measures for these diseases are similar, the best comprising cultural practices such as good drainage, selection of loose-textured soils, crop rotation, early harvest, and avoiding soils prone to flooding. Treatments with fungicides may help establish the crop, preventing root rots from attacking during the crop's first months. Ridomil® (2.5 kg/ha), applied to the soil, and foliar applications of Alliette® (0.4 kg/ha) have shown good results. Fungicides based on plant extracts, oils, and cytokinins help control soil fungi, while offering a nonpolluting organic alternative. Resistant varieties have also been reported (Castaño 1953; CIAT 1998; Drummond and Gonçalves 1957; Fassi 1957; Müller and De Carneiro 1970; Sánchez 1998).

Root rot or "black rot" (*Rosellinia* spp.)

Importance. This disease has been reported in many cassava-growing regions with heavy, poorly drained soils that have a high content of organic matter. It is also found in cassava crops planted after forest crops or ligneous perennial species (Castaño 1953; Viégas 1955). The disease has also been called "black rot" because of the characteristic black color of infected tissues and root cankers.

In Colombia, dry rots are found in the Coffee Belt and in crops planted where coffee, cacao, or *guamo* (a shade tree used in coffee plantations) had previously been grown.

Symptoms and epidemiology. Initially, the root epidermis is covered with white rhizomorphs that later become black (Figure 8-12). Internally, infected tissues of bulked roots are slightly discolored and exude liquid on pressure. The black mycelial bundles penetrate the tissues, where they grow, forming small cavities that contain mycelium of an off-white color. The infected roots have a characteristic odor of decaying wood.

Etiology. *Rosellinia necatrix*, the perithecial state of *Dematophora necatrix*, is the causal agent of this disease (Castaño 1953; Viégas 1955). This fungus induces root rot in other ligneous and herbaceous plants (Castaño 1953; Viégas 1955). However, very little information is available on the epidemiology of

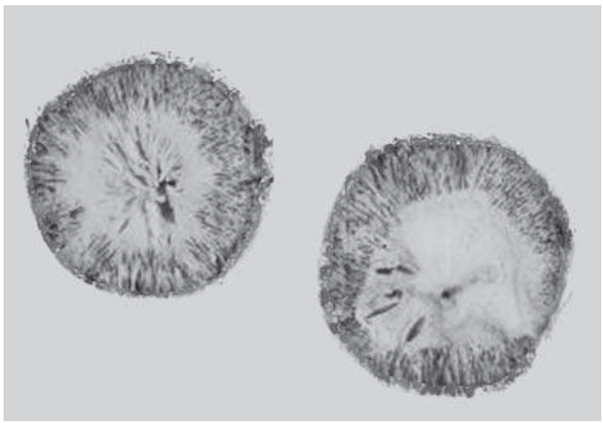

Figure 8-12. Rot caused by *Rosellinia necatrix* in cassava roots.

the fungus in cassava. Its sexual state is generally believed to occur only very rarely (Castaño 1953). Other *Rosellinia* species also attack cassava.

Management and control. Although the disease has not been reported in young plants, the recommendation is still to avoid selecting planting materials from infected crops.

- Rotate with grasses whenever the incidence of plant death or root rot reaches 3%.
- Eliminate infected cassava residues and/or litter from perennial trees (e.g., trunks and decaying branches).
- Plant in loose-textured soils.
- Improve soil drainage.
- Treat by solarization, exposing the soil to the sun for 3 months.
- Chemical control with Topsin (thiophanate-methyl) at 2 g/L of commercial product and applied to the soil before planting.
- Applications of Sincocin (plant extract) to the soil at 1 L/ha are recommended. Stakes may also be immersed in a solution of the product at 1%.

Root rot (*Sclerotium rolfsii*)

This disease commonly occurs in young stakes and mature roots, covering affected parts with a cottony mat. It has been reported only in Latin America (CIAT 1972; Ferdinando et al. 1968). The white mycelium, which is found in infected roots or towards the base of stems, is also disseminated through the soil. This mycelium can, sometimes, penetrate roots through wounds, causing subsequent rot. Although it is rarely lethal to young plants, this fungus may cause a high incidence of root necrosis in a plant.

The disease is caused by *Sclerotium rolfsii*, a common soil organism but a weak pathogen. It has a white mycelium of cottony appearance. It also produces numerous round sclerotia, which characteristically form in the host or laboratory cultures.

Cottony cassava rot (*Fomes lignosus*)

Although this disease is known in Latin America, it is currently of minor importance. The disease is identified by the presence of a mass of white mycelium under the cortex of bulked roots and by the presence of white mycelial threads that look like cotton fibers covering part or all the epidermis of infected roots to the base of stems. Internally, the infected tissues look dehydrated and have a characteristic odor of decaying wood. Young plants may become infected and sometimes suffer sudden wilting, defoliation, and root necrosis.

The organism causing the disease is *Fomes lignosus* (IITA 1972; Jennings 1970).

Diseases Caused by Pseudo-fungi

Root rots (*Phytophthora* spp.)

Importance. Root rots are a very common problem in cassava production, causing yield losses that may be as high as 80% of total production.

Distribution. Root rot caused by *Phytophthora* spp. affect cassava in different agroecological areas in Africa (Fassi 1957), tropical America (Müller and De Carneiro 1970), and India (Johnson and Palaniswami 1999). In Nigeria, Cameroon, and Benin, the pathogens causing root diseases of economic importance include *Sclerotium rolfsii*, *Botryodiplodia theobromae*, *Fomes lignosus*, *Rosellinia necatrix, Rhizoctonia solani, Phytophthora* spp., and *Fusarium* spp. (Hillocks and Wydra 2002).

Recent reports mention that cassava rots may cause losses between 5% and sometimes 100% in Latin America, Asia, and Africa, specifically, Colombia, Brazil (W Fukuda and C Fukuda 1996, EMBRAPA, Brazil; F Takatsu 1996, University of Brasília, Brazil, pers. comm.), Cuba (M Folgueras 2002, INIVIT, pers.

comm.), Mexico (LF Cadavid 2005, CLAYUCA, pers. comm.), India (J George 2004, CTCRI, pers. comm.), Uganda (W Serubombwe 2003, NARO, pers. comm.), Nigeria, Kenya, Indonesia, Ghana, Ecuador and probably in many other countries.

In Asia, root rots have recently been described in the Nondindang District, Buriram Province, Khonburi District, Nakhon Ratchasima Province (Figure 8-13)—areas characterized by loam sandy soil. The genotypes showing symptoms are Rayong 5, Kasetsart 50, and Huay Bong 60 (E Álvarez 2009, pers. comm.). The disease was also observed at the Rayong Field Crop Research Center, affecting genotype Huay Bong 80 (Figure 8-14). Cassava root rots have also been observed in Vietnam.

In India, *Phytophthora palmivora* is emerging as a serious threat to cassava in several industrial areas of Tamil Nadu, where it is endemic. Crop losses are as high as 50%. Differential reaction of cassava varieties to infection by *Phytophthora* was observed (Edison 2002).

Symptoms and epidemiology. *Phytophthora drechsleri* macerates the root parenchyma, causing a

Figure 8-14. Cassava root rot symptoms observed in Rayong and at the Thai Tapioca Development Institute (TTDI) in Huay Bong, Nakhon Ratchasima Province.

Figure 8-13. Cassava plants showing symptoms of root rot and wilting in **(A)** Buriram Province and **(B)** Nakhon Ratchasima Province, Thailand.

penetrating odor and changing root color to cream (Figure 8-15A). *P. tropicalis* has been isolated from crops in Colombia (Figure 8-15B). In the State of Sergipe (Brazil), in 1976–1979, *P. drechsleri* was found to cause rot in the neck and roots, irreversible wilting of aerial parts, and defoliation (Souza Filho and Tupinamba 1979) (Figure 8-15C). In contrast, *P. nicotianae* var. *nicotianae* shows little pepstatin activity. The odor is mild, with brown discoloration (Soto et al. 1988). Root attack by *P. drechsleri* leads to leaves falling and branch tips drying up before the plant dies (Figuereido and Albuquerque 1970). *Phytophthora nicotianae* also causes a similar leaf blight in cassava (Erwin and Ribeiro 1996; Lima et al. 1993).

Etiology. Farmers widely believe that root rots are caused by excess water in the soil. However, a study conducted in different edaphoclimatic areas of Colombia showed that different *Phytophthora* spp. are the major cause of cassava root rots (Sánchez 1998). Other pathogens also causing root rots include:

Fomes lignosus
Sclerotium rolfsii
Armillariella mellea
Fusarium spp.
Rhizoctonia sp.
Rhizopus sp.
Rosellinia necatrix (Lozano and Booth 1979)
Pythium chamaehyphon (GenBank accession AY745748; CIAT 2004)

Eleven species of *Phytophthora* have been reported as causing root rot. These are:

P. arecae (Coleman) Pethybridge (Álvarez et al. 1997c)
P. capsici Leonian (Lima et al. 1993)
P. citricola (CIAT 1999, 2000)
P. cryptogea Pethybr. & Lafferty
P. drechsleri Tucker (Figueiredo and Albuquerque 1970; Muller and De Carneiro 1970)
P. erythroseptica Pethybridge (Fassi 1957)
P. meadii (Barragán and Álvarez 1998)
P. melonis (GenBank accession AY 739021; CIAT 2000, 2004)
P. nicotianae Breda de Haan var. *nicotinae* (Dastur) (Soto et al. 1988)
P. palmivora (Johnson and Palaniswami 1999; (Álvarez and Llano 2002)
P. tropicalis (GenBank accession AY 739022; CIAT 2000, 2004).

The genetic diversity of these pathogens is broad and was determined through studies in Colombia with 80 isolates obtained from roots, young stems, and soils from 19 municipalities. These studies included the pathogen's pathogenicity, virulence, morphology, and molecular analysis of the internal transcribed spacer (ITS) region of the pathogen's ribosomal DNA. Eleven genetic groups were identified through PCR-RFLP (Álvarez et al. 1997a, 1997c, 2000; Sánchez 1998). *Phytophthora tropicalis* was identified through sequencing of the ITS region of ribosomal DNA and isoenzymes, showing it to be genetically similar to *P. capsici* (CIAT 2000). The isolate was obtained from cassava roots in Barcelona, Quindío; *P. palmivora* was isolated from cassava roots at CIAT, Valle del Cauca.

Integrated disease management. The integrated management of root rots includes the use of varietal resistance and/or cultural practices.

Figure 8-15. Root rots **(A** and **B)** and plant wilt **(C)** caused by *Phytophthora* spp.

Varietal resistance. A principal tool for managing root rots caused by various *Phytophthora* species is the use of varietal resistance. Various examples exist of the successful adoption of cassava clones resistant to *Phytophthora* spp. In 1990, the Brazilian Agricultural Research Corporation (Embrapa) and the Agricultural Research Center for the Humid Tropics (CPATU) released two cassava clones resistant to root rots: cvs. Mae Joana (IM-175) and Zolhudinha (IM-158). Both clones came from the State of Amazonas and are planted in the várzea ecosystem (a type of floodplains) of northern Brazil. The adoption of these clones, together with the application of appropriate cultural practices, increased root yields by more than 80% in this region (Lozano 1991b).

High yields and resistance to root rot caused by *P. drechsleri* were obtained in clones MD-33 and Pao (Mendonça et al. 2003). Pereira (1998) reported resistance to *P. drechsleri* in seven cultivars from a group of 31 evaluated. Barragán and Álvarez (1998) reported 15 resistant genotypes from a group of 60 elite genotypes evaluated. In 2003, Llano et al. reported six individuals from a family of 126 individuals, with high resistance to *P. tropicalis, P. palmivora*, and *P. melonis.* Although harvesting roots 14 months after planting resulted in increased yield, it also demonstrated a higher incidence of root rots, thus showing that root rot incidence varies according to clones and harvest time.

In a participatory research study, indigenous communities of the Colombian Amazon adopted cassava clones resistant to *Phytophthora* spp. (Llano and Álvarez 2008; Llano et al. 2001). These clones were selected in the laboratory (harvested roots) and greenhouse (stems) from 700 genotypes provided by Embrapa and CIAT.

To obtain reliable information on the genetics of such a complex disease, Takatsu and Fukuda (1990) concluded that standardized methods were needed for inoculating and evaluating resistance to each cassava root rot pathogen. CIAT and the National University of Colombia–Palmira identified cassava clones resistant to *P. nicotianae* var. *nicotianae* by first inoculating bulked roots of plants that were 10 to 12 months old. They then added a suspension of the fungus to a nutritive solution in which 45-day-old seedlings were growing. The roots of seedlings were colonized by the pathogen. The inoculated roots were evaluated in terms of the percentage of the pathogen's colonization of cortical and parenchymatous tissues.

Inoculated bulked roots demonstrated variation in the severity of symptoms, depending on whether they came from resistant or susceptible clones. The inoculation method was easier to carry out, less expensive, and with faster results than the seedling method. No correlation was found between the two inoculation methods (López and Lozano 1992).

Cassava seedlings planted in soil were also evaluated. The soil had previously been inoculated with a suspension of each of zoospores, oospores, or chlamydospores applied separately (Lima et al. 1993). Each inoculum type caused wilt and seedling death.

In 1995, Lima and Takatsu (1995) published the reactions of 13 cassava clones that had been stem-inoculated with three isolates of *P. drechsleri* in the greenhouse. The isolate with the most virulence was inoculated into roots in the field. To inoculate roots without harvesting them, inoculum was deposited in a small wound. The correlation between inoculated plants in the screenhouse and roots inoculated in the field was +0.24.

In other studies (Loke 2004), several biochemical and morphological markers, and leaf resistance were identified for preselecting clones for resistance to *P. tropicalis* in cassava populations, based on (1) reduced area of the parenchyma with the presence of scopoletin in roots after harvest; (2) a high relationship between iron and manganese; and (3) resistance in leaves 72 h after inoculation. Scopoletin is a coumarin that is found in very low concentrations in fresh roots but which increases considerably after harvest. This substance is easy to quantify in roots, using ultraviolet light, and is related to the cassava root's susceptibility to postharvest physiological deterioration.

Loke (2004) also demonstrated the benefits of using an index of resistance to *P. tropicalis* that includes molecular markers. The objective of this index is to select genotypes with durable resistance, based on a large diversity of resistance or defense mechanisms.

Several studies to identify the genetic base of resistance to *Phytophthora* have been conducted. For 25 cassava clones, a correlation of +0.31 was observed between resistance during penetration (in the peel, both epidermis and subepidermis) and after penetration (in the parenchyma). This finding indicated that these forms of resistance are moderately associated (Corredor 2005; Loke 2004). Alvarez et al. (2003c),

Llano et al. (2004), and Loke (2004) evaluated the cassava K family (150 F_1 individuals from the cross TMS 30572 × CM 2177-2), inoculating root fragments. Nineteen QTLs were identified as associated with resistance to different species of *Phytophthora* and *Pythium*, three of which explained between 8.3% and 11% of phenotypic variance.

Those QTLs that were expressed were also found to vary from one cropping cycle to another, depending on prevailing environmental conditions. Minor genes were demonstrated as controlling resistance to *P. tropicalis, P. melonis*, and *P. palmivora*, with a high genotype × environment interaction existing. Although the population showed differences within its genetic base for resistance to *Phytophthora*, levels of resistance were not sufficiently high for use in improvement programs. Hence, identifying contrasting parents for the disease would be useful, as well as developing new populations for determining QTLs (Llano et al. 2004; Loke et al. 2004).

To identify genomic sequences in cassava that are homologous with genes of resistance to diseases of different plant species, two cassava families were evaluated for their resistance to *P. tropicalis* (GenBank accession AY 739022), *P. melonis* (GenBank accession AY 739021), and *P. palmivora*, all causal agents of root rot. Two strategies were used to search for genes for resistance: (1) hybridization with probes for maize and rice, using RFLP; and (2) amplifying conserved regions of DNA, using the degenerate primers NBS and Pto kinase. Three cassava clones resistant to *P. tropicalis* and *P. palmivora* were used, obtaining clones that were sequenced and homologized with known genes of resistance.

With hybridization, cassava demonstrated very low homology with the monocotyledon genes tested. Twenty-eight NBS and 2 Pto kinase clones were obtained, of which 14 showed homologous sequence with resistance gene analogs (RGAs) and NBS-LRR (GenBank accessions: AY730038, AY730040, AY730041, AY737490, AY745762, AY745763, AY745764, AY745765, AY745766, AY745767, AY745768, AY745769, AY745770, and AY745771). Four of these showed an open reading framework (ORF) with conserved motifs in the nucleotide-binding site (NBS) region, which means they were considered to be RGAs. Altogether, three classes of RGAs were identified, none of which showed association with resistance to *Phytophthora* (Llano et al. 2004).

Cultural practices. The best cultural practices for the integrated management of root rots are summarized below:

- Selecting an appropriate, well-drained, and moderately deep soil. If the land is flat and soils are clayey, planting should be done on ridges.
- To catalyze resistance, fertilizers should be applied in drench form, using potassium sources, and/or as foliar sprays, using potassium phosphites.
- If rot incidence reaches 3%, the cassava crop should be rotated with grasses, at least once a year.
- Eradicating diseased plants by removing infected roots from the field and burning them.
- Selecting healthy plants to obtain clean seed. Where the farming area is infested, then stakes should be treated with metalaxyl at 0.3 g/L a.i.
- Treating stakes in hot water at 49 °C for 49 min is an alternative to chemical treatment (Álvarez et al. 2003b).

Immersing stakes in a suspension of *Trichoderma harzianum* and *T. viride* at 2.5×10^8 spores/L, and later applying the same suspension in drench form (CIAT 2006, 2007). Biological control of rots with isolates of *T. harzianum* and *T. viride* is promising (Bedoya et al. 2000; CIAT 2006, 2007; Edison 2002). Field trials in different agroecological zones of Colombia have shown that soil inoculated with strains of these types of *Trichoderma* will increase cassava yield (CIAT 2001, 2006, 2007). Isolates of *Trichoderma* spp. were selected on the basis of *in vitro* antagonism, production of secondary metabolites that inhibit *Phytophthora* spp., and bioassays in screenhouses.

To identify practices of disease management that are feasible for indigenous communities in the northwestern region of the Amazon (Colombia), participatory research trials were established, with the women farmers making the evaluations. Soil amendments were incorporated. These were ash, organic matter (dry leaves), and a 1:1 mixture of both materials. Dosage was 200 g/plant. Cassava was also associated with cowpea (*Vigna unguiculata*), and stakes selected from the middle part of healthy plants.

In these trials, cassava yield increased by 446% with applications of the ash and organic matter mixture. Where only ash was used, yield increased by 272%. Stake selection increased yield by 366%. Compared with traditional management, these practices reduced root rots by 100% (incorporation of the ash and organic matter mixture), 99% (association with cowpea), 94.2% (ash only), and 89.7% (stake selection) (Llano and Álvarez 2008).

Other Causal Agents of Cassava Rots

Other fungal root rots

Other fungal species can induce root rots in cassava plants at different growth stages, but little information is available on these diseases and their importance. These root rots are caused by:

Armillariella mellea, which attacks both the stem base and roots of mature plants (Arraudeau 1967; CIAT 1972)
Phaeolus manihotis (Heim 1931)
Lasiodiplodia theobromae (Vanderweyen 1962)
Pythium sp. (CIAT 1972)
Fusarium sp. (CIAT 1972)
Clitocybe tabescens (Arraudeau 1967)
Sphaceloma manihoticola (Bitancourt and Jenkins 1950)
Rhizopus spp. (Majunder et al. 1956)
Rhizoctonia sp. (Gonçalves and Franco 1941)
Aspergillus spp. (Clerck and Caurie 1968)
Nattrassia mangiferae (*Scytalidium* sp.); *Verticillium* sp.; and *Rigidoporus* sp.

Bacterial root rots

Some bacterial species belonging to the *Bacillus*, *Erwinia*, and *Corynebacterium* genera are also believed to cause soft rots and/or fermentation in bulked cassava roots (Akinrele 1964; Averre 1967). Symptoms of these soft rots are similar and are frequently accompanied by fermentation. These agents probably penetrate roots through wounds produced by farmers during cultivation or by animals, insects, or fungi. They are frequently accompanied by other saprophytic microorganisms that help advance deterioration.

The causal agent of cassava bacterial blight (see below) can also induce necrosis, discoloration, and dry rot in the vascular tissues of infected roots (Lozano 1973; Lozano and Sequeira 1974).

Cassava heart rot

This physiological disorder damages bulked roots (Averre 1967). It occurs in moist and poorly drained soils in which roots present a dry internal necrosis that extends irregularly from the center to cortical tissues. This disorder is observed in only 10%–20% of the roots of an infected plant. The larger and thicker roots are believed to be the most susceptible.

Postharvest physiological deterioration (PPD)

The cause of cassava roots' rapid deterioration after harvest is unknown, whether it results from physiological or pathological effects, or a combination of the two. Numerous microorganisms have nevertheless been isolated from deteriorated roots, with several being known to cause discoloration and rot.

Bacterial Diseases

Cassava bacterial blight (*Xanthomonas axonopodis* pv. *manihotis*)

Importance. Cassava bacterial blight (CBB) is regarded as one of the most limiting diseases of cassava production, as it can cause total crop loss in affected areas.

During the 1960s and 1970s, this disease caused major damage to the cassava crop. However, the application of integrated management programs, introduction of quarantine measures in some countries, and identification and planting of resistant varieties have led to its satisfactory control (Hillocks and Wydra 2002; Lozano 1986).

Distribution. Cassava bacterial blight has been known in Latin America since 1912, when it was reported in Brazil (Kemp 2000). It spread to the cassava-growing regions of Africa and Asia in the 1970s (Boher and Verdier 1994; Bradbury 1986). In Latin America, the disease has been reported from most of the cassava-growing regions of Bolivia, Brazil, Colombia, Cuba, the Dominican Republic, Mexico, Panama, Trinidad and Tobago, and Venezuela (Cajar 1981; Fukuda 1992; Languidey 1981; Lozano and Sequeira 1974; Rajnauth and Pegus 1988; Rodríguez 1979; Rodríguez 1992; Sosa 1992; Trujillo et al. 1982).

In Asia, CBB has been observed during the rainy season in Thailand (Figure 8-16) as well as in many other countries but it is seldom very severe (E Álvarez

Figure 8-16. Cassava bacterial blight (CBB) symptoms observed on cassava leaves of cv. Rayong 5 in Thailand.

2009, pers. comm.). The disease was first observed in Taiwan before 1945 (Leu 1976), and has since been reported from Malaysia, Indonesia, Thailand (Booth and Lozano 1978; E Álvarez and AC Bellotti 2009, pers. comm.), Vietnam (E Álvarez and AC Bellotti 2009, pers. comm.) and India (Cherian and Mathew 1981). In Africa, the disease causes severe epidemics (Hillocks and Wydra 2002), and appears in the following countries (in order of reporting year): Nigeria (Williams et al. 1973), Zaire (Maraite and Meyer 1975), Ghana (Doku and Lamptey 1977), Benin (Korang-Amoakoh and Oduro 1979, cited by Hillocks and Wydra 2002), the Democratic Republic of the Congo (Daniel et al. 1980), Côte d'Ivoire (Notteghem et al. 1980), Republic of South Africa (Manicom et al. 1981), Rwanda (Onyango and Mukunya 1982), Sudan (Kwaje 1984), Togo (Boher and Agboli 1992), Cameroon, Central African Republic, Tanzania, Kenya, and Burundi (Hillocks and Wydra 2002).

Symptoms and epidemiology. Symptoms characteristic of CBB are small, angular, aqueous-looking leaf spots found on the lower surface of the leaf blade. Or symptoms may be leaf blight or brown leaf burn, wilt, dieback, and a gummy exudation in infected young stems, petioles, and leaf spots (Figure 8-17). The vascular bundles of infected petioles and stems are also necrotic, appearing as bands of brown or black color. Symptoms occur 11 to 13 days after infection (Lozano and Booth 1979). Some susceptible varieties present dry and putrid spots around necrotic vascular bundles (Verdier 2002).

The bacterium disseminates widely through stakes from infected plants, from one cropping cycle to another, and from one area to another. Within the crop, the principal means of dispersal are water splash from rain and contaminated tools. The movement of people and animals within the crop, especially during or after rain, may also help disperse the pathogen (Lozano 1973).

Although the pathogen survives poorly in soil, this can be source of inoculum if it is contaminated, as well as irrigation water, although in reduced proportions. The bacterium can survive epiphytically on many weeds, which serve as sources of inoculum if control is

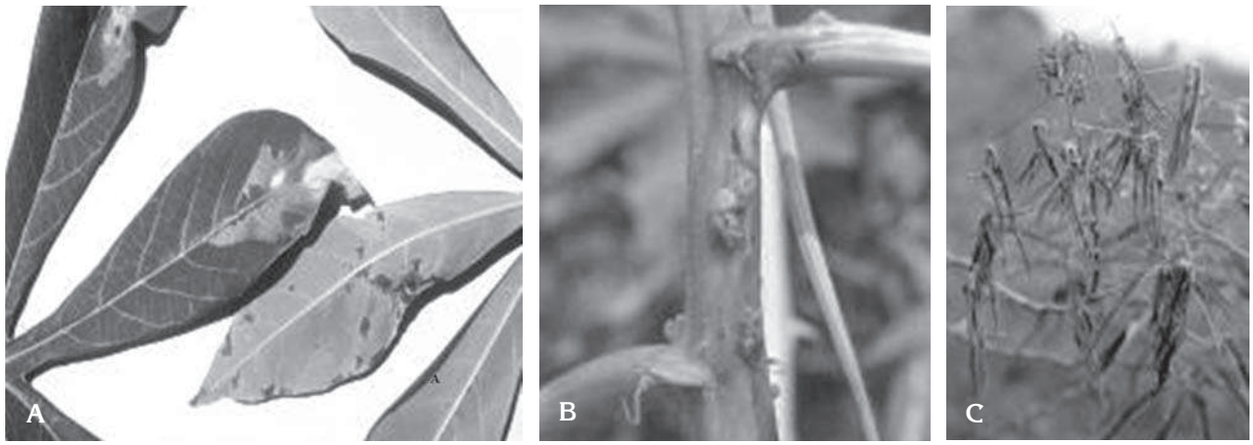

Figure 8-17. Symptoms of cassava bacterial blight, induced by the bacterium *Xanthomonas axonopodis* pv. *manihotis*: **(A)** angular leaf spots and leaf blight, **(B)** exudate on stem, and **(C)** plant wilt.

inadequate. Insects spread the disease over short distances.

The severity of CBB becomes greater when temperatures fluctuate widely between day and night. Hence, the disease is not important in areas of stable temperatures such as the Amazon Region, where the cloud cover does not permit marked fluctuations in temperatures.

Etiology. The causal organism, *Xanthomonas axonopodis* pv. *manihotis* (*Xam*), is a Gram-negative bacterium that is shaped like a slim cane. It is mobile by means of a polar flagellum. Its cells are not encapsulated, and the bacterium does not form spores.

The organism penetrates the host through stomas and wounds in the plant's epidermis. Infection is systemic, moving through the stems and petioles in xylem vessels and possibly also the phloem.

Xam can be detected, using the polymerase chain reaction (PCR), which amplifies a DNA fragment of 898 bp. This methodology permits detection to as low as 300 cfu/mL in leaves and stems infected by CBB (Verdier et al. 1998). When Verdier and Mosquera (1999) used the specific probe P898, they detected the bacterium in raw extracts of infected leaves and stems, and in cassava fruits and sexual seed. According to Verdier et al. (1993), pathogen diversity is narrow in Africa but broad in South America, cassava's center of origin.

Restrepo et al. (1996) reported that the diversity of the Colombian strains is very broad, at both pathogenic and genetic levels. Diversity is also high in Brazil (Restrepo et al. 1999) and Venezuela (Verdier et al. 1998).

Previous studies also revealed geographical differentiation among pathogen populations, according to ecozone. Evidence also exists of pathotypes moving within and between regions, probably because of movements of infected planting materials. In Colombia, analysis of pathogenic characteristics of *Xam* strains collected in three ecozones led to the definition of different pathotypes specific to each ecozone (Restrepo 1999).

An analysis, using the AFLP technique, of the genetic variability of 85 *Xam* isolates from Brazil, Colombia, Cuba, and Venezuela distinguished three groups: (1) a cluster at a similarity level of 0.6 and formed of isolates from different localities in Colombia; (2) a cluster at 0.7 and comprising 81% of the Venezuelan isolates included in this study, and 4 Brazilian isolates; and (3) a cluster at 0.4 and formed by most of the Brazilian isolates, 3 isolates from Venezuela, 1 from Cuba, and 3 from Colombia. In this last group, clustering below the 0.4 similarity level also occurred, indicating great genetic variability within the Brazilian sites, possibly related to the also high level of genetic diversity observed for the host plant (Sánchez et al. 1999). When new pathogen strains are introduced into a given area, the genetic diversity already found within the pathogen population is increased, thereby favoring the development of new pathotypes (Restrepo 1999).

Integrated disease management. To control the disease, integrated management should be carried out, involving varietal resistance, cultural practices, and biological control.

Varietal resistance. The genetic control of CBB is the most efficient and economic method for the farmer, but the cassava cropping cycle is long, with a very low production of planting materials. Hence, the time involved in producing resistant varieties is very long. At CIAT, resistant varieties are identified through evaluations in the Eastern Plains and the Atlantic Coast, where the disease is acute and endemic. They are also evaluated in the greenhouse, under controlled conditions, with temperatures at 30 °C and relative humidity at 95%.

In several greenhouse studies, plants of different cassava varieties were inoculated with 39 isolates from different regions of Colombia, Venezuela, and Brazil. Fifteen genotypes were identified as having high to intermediate resistance to CBB, scoring between 1.0 and 2.5 on a scale of severity from 1.0 to 5.0. These varieties included M Esc Fla 039, M Esc Fla 021, M Bra 383, M Col 2066, CM 3311-4, CM 7772-13, and SM 1779-8 (CIAT 1999, 2000, 2001, 2002b, 2003a).

Between 1995 and 2007, about 6400 cassava genotypes were evaluated in Villavicencio (Colombia) for their field resistance to CBB. Of these, 117 were identified as having partial resistance (CIAT 2001, 2002b, 2003a, 2006, 2007).

In a 10 × 10 diallelic study, carried out in Villavicencio, with 45 families and 30 plants per family, the cassava genotype CM 4574-7 was identified as having high general combination ability. Its progenies showed increased resistance to CBB and SED (Calle et al. 2005).

Tolerant varieties also exist such as M Bra 685, M Bra 886, ICA Catumare, ICA Cebucán, ICA Negrita, Vergara (CM 6438-14), CM 4574-7, and Chiroza. However, the disease has increased in severity in ICA Catumare, for which adequate selection of clean seed was not performed (Álvarez and Llano 2002). Several genotypes have also been identified as having resistance to several pathotypes of the bacterium (Álvarez et al. 1999).

Zinsou et al. (2004) recommended the cassava genotype TMS 30572 for farmers in Benin, because of its high yield and relatively stable resistance to CBB across different environments. Kpémoua (1995) showed that resistance to *Xam* is associated with the production of phenolic compounds and the reinforcement of cell walls in the vascular system during early infection.

To determine the genetic control of resistance, 150 F_1 individuals of the cross TMS 30572 × CM 2177-2 were inoculated with the pathogen and evaluated for resistance under controlled conditions in the greenhouse. Five different *Xam* strains from the world's major cassava-growing areas were used in the study. Genetic analysis identified six genomic regions that control resistance to all *Xam* strains. One region controlled >60% of resistance to each of the strains CIO-1 and CIO-136. Two regions accounted for >70% of resistance to strain CIO-84. Another 80% of resistance to strains CIO-136 and ORST X-27 could be explained by 3 loci for each strain (Jorge et al. 2000).

In three instances, the same genomic regions controlled resistance to two strains. A marker was obtained by Southern hybridization of a PCR amplification product from cassava, using heterologous primers designed from conserved regions of the *Xanthomonas* resistance gene in rice (*Xa21*). The region it marked accounted for 60% of phenotypic variance for resistance to strain CIO-136. A backcross population, derived from crossing members of the mapping population, has been developed and will provide more recombinations for fine mapping towards cloning resistance genes, and for studying intra-locus and inter-loci genetic interactions (Jorge et al. 2000).

A molecular genetic map of cassava was recently constructed from an F_1 cross of noninbred parents. RFLP, AFLP, EST, SSR markers were used to map resistance to CBB. The F_1 cross was evaluated with *Xam* strains under both field and greenhouse conditions. Nine quantitative trait loci (QTLs), located on linkage groups B, D, L, N, and X, explained the phenotypic variance of the crop's response to *Xam* in the greenhouse.

Jorge et al. (2001) reported eight QTLs associated with resistance to CBB, and found changes in the expression of QTLs from one cropping cycle to another in the field, which could be related to changes in the pathogen's population structure. A QTL, located in linkage group D, was conserved over two cropping cycles and in resistance evaluations in the greenhouse. In a previous study, Jorge et al. (2000) showed that 12 different QTLs control resistance to five *Xam* strains.

Hurtado et al. (2005) detected the molecular marker, microsatellite SSRY 65, that could select resistant genotypes in a cassava family corresponding to the cross CM 9208-13 × M Nga 19. Furthermore, the authors identified two RGAs of the NBS class through amplification with PCR, using two primers designed by Llano (2003). These RGAs could identify plant individuals that were resistant to the bacterium.

One approach to assessing cassava genetic diversity involves the structural analysis of genotypes resistant to CBB. Multiple correspondence analysis of AFLP data, using two primer combinations for cassava genotypes resistant and susceptible to two strains of *Xam,* elucidated the genetic structure of cassava germplasm resistant to CBB (Sánchez et al. 1999). Results revealed a random distribution of resistance or susceptibility, suggesting that resistance to CBB has arisen independently many times in cassava germplasm.

Phenolic compounds have been implicated in the resistance of cassava (*Manihot esculenta*) to xanthomonads. Cassava cultivars M Col 22 and CM 523-7 were inoculated with *Xam* and *X. cassavae*. CM 523-7 was susceptible to both pathogens, whereas M Col 22 was susceptible to *Xam* and resistant to *X. cassavae*. In the resistant interaction, no disease symptoms were observed in leaves. Bacterial growth was greatly reduced, and cell wall-bound peroxidase activity increased twofold, probably related to lignin deposition (Pereira et al. 2000).

Preformed putative defenses include copious latex production, which contains protease, ß-1,3 glucanase, and lysozyme activities. ESTs from a latex

cDNA library revealed a constitutive expression of many defense-related genes including chitinase, glucanase, and PAL. A cDNA-AFLP analysis of cassava leaves suffering a hypersensitive response to *Pseudomonas syringae* pv. *tomato* revealed that 78 genes, new to cassava, had expressed differentially. Homologs of a metalloprotease, glucanase, peroxidase, and ACC oxidase were all found to be upregulated. Pathogenicity determinants of *Xam* are being studied in the disruption of the *gum* biosynthesis gene (its EPS is produced copiously in plants) and the *pel* gene (pectate lyase appears as a single isoform) (Kemp et al. 2001).

RGAs were amplified as a means of elucidating the putative genes involved in cassava's defense response. For the cDNA-AFLP technique, of about 3600 cDNA fragments screened, 353 fragments were specific to a resistant variety. Sequence analyses showed significant homology with resistance genes, NPK-1 related proteins, senescence-related proteins, and other known proteins involved in disease resistance reactions.

Using degenerate primers, 12 classes of RGAs were identified in cassava. Screening a cassava cDNA library (root and leaf) with class-specific RGA probes also led to the identification of 16 expressed gene clones. Sequence analysis of clone L16 confirmed the constitutive expression of a protein that shares characteristics with previously reported resistance genes (Restrepo et al. 2001).

López et al. (2004a) identified 6046 unigenes and characterized a group of genes putatively involved in cassava's defense response to *Xam* infection. López et al. (2004b) identified the *RXam1* gene, homolog of *Xa21* from rice, in a 3600-bp DNA fragment. The gene is induced in the resistant variety (M Bra 685), 72 h after infection by *Xam*.

Cultural practices. The following practices are recommended:

- Use of healthy planting materials obtained from disease-free crops, plants derived from meristem culture, and by rooting buds and/or shoots

- Treating stakes by immersing them for 10 min in a solution of cupric fungicides such as copper oxychloride or Orthocide® (captan) at 3 to 6 g/L

- Immersion in extract of citrus fruit seeds (Lonlife®)

- Heat treatment of stakes (Álvarez et al. 2008; CIAT 2007), using hot water at 49 °C for 49 min. Incidence of CBB in untreated stakes was 37%, but dropped to 7% when treated with hot water. It dropped further to 0% when stakes were pretreated at 49 °C for 10 min 24 h before being treated with hot water for 5 h. Treatment with hot water did not, in practical terms, affect stake germination, reducing it by only 18% in the most prolonged treatment (Ramírez et al. 2000). The induction of enzymes that activate under stress conditions is probably responsible for conserving high stake germination, even after prolonged treatment in hot water.

Lozano (1986) also mentions the following practices for managing the disease:

- Planting at the end of rainy periods
- Crop rotation with grasses
- Planting barriers of maize to prevent dissemination by wind
- Improving soil drainage
- Weed control
- Fertilizers adapted mainly with sources of potassium
- Eradicating diseased plants
- Preventing the movement of people, machines, and animals from infected lots to healthy lots
- Eliminating infected materials after harvest by burning branches and stems
- Incorporating harvest residues into the soil

In field studies conducted in Benin and Togo by Wydra et al. (2001), locally and regionally well-adapted control measures for CBB were identified such as:

- Using locally preferred resistant varieties
- Intercropping with locally used crops
- Amending soils with local materials
- Fertilizer applications and recommendations on phytosanitary measures carried out to reduce disease

Complementary studies elucidated some mechanisms of resistance at the biochemical and genetic levels and molecular host-pathogen interactions.

New methods for detecting *Xanthomonas campestris* pv. *manihotis* (*Xcm*), using immunological and genetic techniques, were developed. Research results were partly verified under African conditions such as testing the cassava genome mapping population for reaction towards African strains to identify genetic markers and/or resistance related genes.

Biological control. Spraying with suspensions of *Pseudomonas putida* reduced the severity of damage caused by CBB, while cassava yields increased significantly (CIAT 1985). However, this practice has not been adapted for farming conditions.

Bacterial stem rot (*Erwinia carotovora* pv. *carotovora*)

Importance. This disease is important for the damage it does to the quality and germinability of planting stakes.

Symptoms. The disease is characterized by an aqueous and smelly stem rot or by medullary necrosis of the plant's ligneous parts (Figure 8-18). Infected plants show bud wilt. The stem's surfaces typically show perforations made by insects of the genus *Anastrepha* Schiner, which act as vectors for the bacterium. These orifices are easy to distinguish by the presence of dry latex, discharged as the stem is perforated. Diseased stakes used for planting will not germinate or they produce weak spindly plants, with a limited number of bulked roots (CIAT 1972).

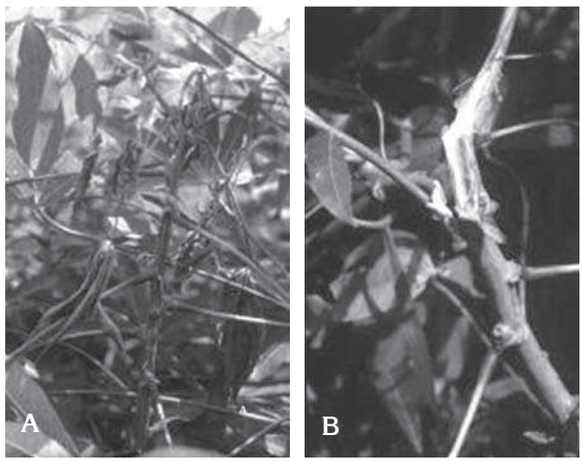

Figure 8-18. Symptoms caused by *Erwinia carotovora*: **(A)** wilt, and **(B)** damage to the medulla.

Management and control.
- Using healthy seed
- Planting with varieties resistant to the insect vector
- Burning infected stems

Bacterial stem gall (*Agrobacterium tumefaciens*)

Symptoms and epidemiology. This disease generally appears on the lower parts of stems in plants older than 6 months. Characteristic symptoms, found on stem nodes, are galls that often become very large, presenting a proliferation of buds on the epidermis (Figure 8-19). Infected plants may become weak and spindly, and in the early days of infection, suffer dieback to as far as major galls. A single plant could have several galls on a stem and even along lower branches (Lozano et al 1981).

The disease is usually initiated by infested soil being rain-splashed onto wounds caused by natural defoliation in stems of the plant's lower parts.

Management and control. Control is achieved through rotation with another crop when more than 3% of the planting is infected; disinfecting machetes with 2% sodium hypochlorite; always using planting stakes from healthy crops; and burning diseased materials within the crop (Lozano et al 1981).

Another bacterial disease is caused by *Erwinia herbicola*.

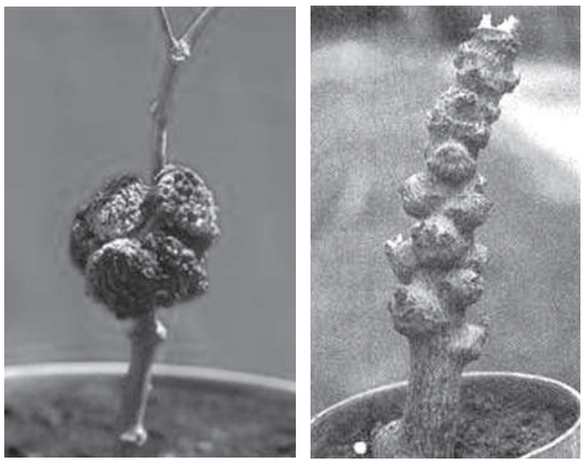

Figure 8-19. Galls on stem caused by *Agrobacterium tumefaciens*.

Diseases caused by '*Candidatus* Phytoplasmas'

(previously known as mycoplasma-like organisms or MLOs)

Cassava frogskin disease (*Ca.* phytoplasma, subgroup 16SrIII-L and rpIII-H)

Importance. Cassava frogskin disease (CFSD) is an economically important disease affecting cassava roots. It was reported for the first time in 1971, in the Department of Cauca, southern Colombia. Its origin appears to be the Amazon region of Brazil or Colombia (Pineda et al. 1983).

Frogskin disease directly affects root production, causing losses of 90% or more. Symptoms consist of small, longitudinal fissures distributed throughout the root. As roots increase in diameter, the fissures tend to heal, giving the injuries a lip form. The root cortex or epidermis appears cork-like and peels off easily. Depending on the severity of symptoms, the depth and number of lesions increase until the root becomes deformed (Álvarez et al. 2003a; Pineda et al. 1983).

Distribution. In the 1980s, the disease occurred in most cassava-growing regions of Colombia and has continually spread. It has now been reported in Brazil, Costa Rica, Panama, Peru, and Venezuela (Calvert and Cuervo 2002), as well as in Nicaragua and Honduras. In Venezuela, it was reported for the first time in the States of Barinas and Aragua, with incidences between 11.4% and 14.3%, in cassava stakes grafted with 'Secundina', a variety used to diagnose the disease (Chaparro and Trujillo 2001).

Symptoms and epidemiology. Frogskin mostly attacks cassava roots, reducing their diameter, but some varieties may also show symptoms in leaves such as mosaic, chlorosis, curling, and/or curvature in leaf margins (Figure 8-20A). However, these symptoms are difficult to distinguish under field conditions, and may be confused with damage from mites, thrips, viruses, and micronutrient deficiencies, or they can be masked when temperatures are >30 °C.

Characteristic CFSD symptoms in the roots include a woody aspect and the thick, cork-like peel, which is also fragile and opaque. The peel also presents lip-like slits that may join to create a net-like or honeycomb pattern (Figures 8-20B and 8-20C). When roots do not bulk adequately (Figure 20D), the stems tend to be thicker than normal. In contrast, the roots of healthy plants are well developed, with thin, brilliant, and flexible peel.

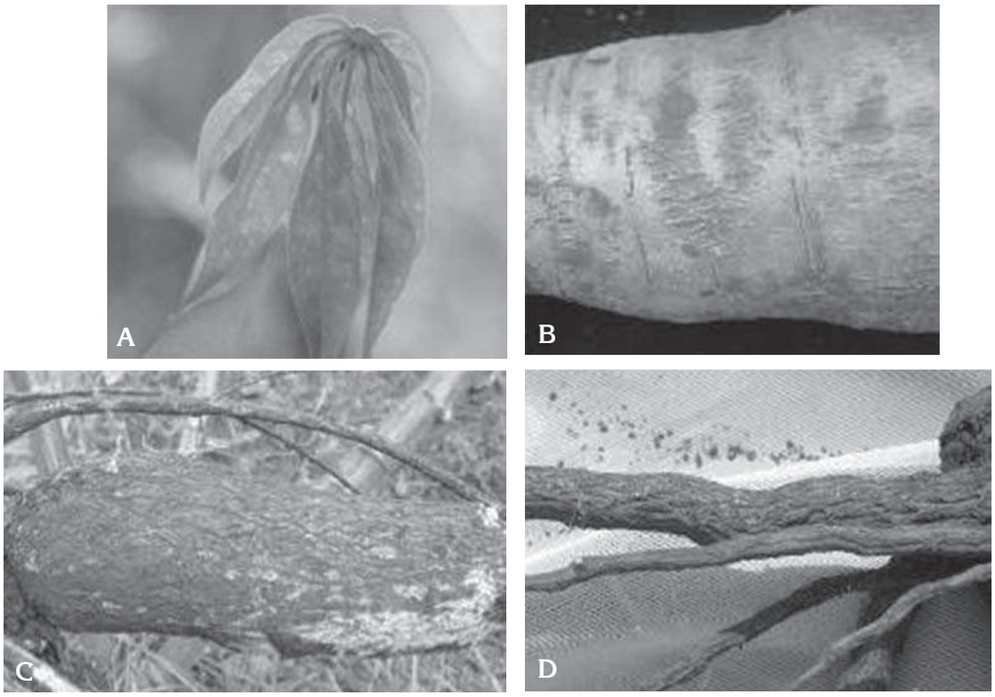

Figure 8-20. Symptoms of cassava frogskin disease in **(A)** leaves, **(B)** and **(C)** presence of lips in root, and (D) reduced root bulking.

Molecular tests, carried out on plants of cassava and pink vinca (*Catharanthus roseus* (L.) G. Don) after transmission trials with dodder (*Cuscuta* sp. L.), detected the presence of phytoplasmas associated with the 16SrIII group. Graft transmission could transfer phytoplasmas from infected to healthy plants (CIAT 2005).

Insects were collected to identify the vector or vectors of the phytoplasma causing the disease. A homology of 90% was found among sequenced fragments from tissue of the insect *Scaphytopius marginelineatus* Stål (Hemiptera: Auchenorrhyncha: Cicadellidae) and from tissues of two cassava varieties.

Etiology. The CFSD-associated phytoplasmas were identified as group 16SrIII strains by restriction fragment length polymorphism (RFLP) and sequence analyses of amplified rDNA products, and results were corroborated by PCRs employing group 16SrIII-specific rRNA gene or ribosomal protein (rp) gene primers. Collectively, RFLP analyses indicated that CFSD strains differed from all phytoplasmas described previously in group 16SrIII and, on this basis, the strains were tentatively assigned to new ribosomal and ribosomal protein subgroups 16SrIII-L and rpIII-H, respectively. This is the first molecular identification of a phytoplasma associated with CFSD in cassava in Colombia (Álvarez et al. 2009).

The phytoplasma was not detected in healthy plants from the same varieties harvested in disease-free fields. These results point towards the possible role played by phytoplasmas in this disease (Álvarez et al. 2003a; CIAT 2002a). The importance of the CFSD in cassava production systems has motivated other scientific groups at CIAT, such as the Virology group, to undertake efforts to understand the characteristics of the disease, its symptoms and its management practices.

Cuttings from CFSD-infected plants in the greenhouse were taken, and rooted in deionized water with different doses of chlortetracycline. Inhibition of leaf symptoms caused by CFSD was successful in two experiments when 50 ppm chlortetracycline were used, thus indicating that CFSD is not caused by a virus. Nested PCR also showed that phytoplasmas were present in leaves of infected plants when treated with 0 ppm tetracycline (CIAT 2003b).

Although the disease spreads mostly through infected stakes, the disease is believed to have insect vectors. Numerous homopteran species (e.g., planthoppers, tree hoppers, and froghoppers) were collected from cassava fields in 9 departments and 17 sites in Colombia. Three genera—*Scaphytopius fuliginosus* Osborn, *Empoasca* sp. Walsh, and *Stirellus bicolor* Van Duzee (Hemiptera: Cicadellidae)—were the most frequently collected. These three species are known vectors of viruses and phytoplasmas for other crops. Based on the evidence of high homology (80%) between insect and phytoplasma detected in cassava, *Sc. fuliginosus* appears to be a potential candidate as the vector for CFSD (CIAT 2003b). However, tests for transmission have not yet effectively confirmed this hypothesis. The whitefly (*Bemisia tuberculata*) is still associated with the disease transmission.

Integrated disease management. To date, the disease is managed principally by using stakes from healthy plants. Heat treatment, followed by meristem culture, has been used to obtain plants free of CFSD. Grafting with the susceptible variety Secundina is useful for monitoring the effectiveness of the heat treatment (Flor et al. 2001). Treating stakes at temperatures of more than 55 °C appears promising but needs adjusting to reduce losses by the consequent low germination of stakes.

Plantings with more than 10% of incidence (foliage, stakes, and roots) should be burned. Plant health surveillance and quarantine systems need to be strengthened to prevent the entry or mobilization of planting materials from areas with the disease.

Field and greenhouse studies carried out at CIAT have reported 30 genotypes with different levels of resistance. These findings were confirmed through the expression of leaf symptoms in grafts with variety Secundina (CIAT 2003b; Cuervo 2006). The use of tolerant varieties will be a useful tool in controlling this disease.

Witches' broom

Importance. This disease, known as *superbrotamiento* in Spanish, has been reported in Brazil, Venezuela, Mexico, and Peru (Figure 8-21). Although its incidence is not significant, the percentage of witches' broom in affected plantings is much higher than that of other diseases caused by American phytoplasmas. Crop losses can reach 80% (Lozano et al. 1981). In Asia a new cassava disease was observed at Quang Ngai, Vietnam (Figure 8-22). Typical

Figure 8-21. Symptoms of "*superbrotamiento*" in cassava. (Photo: B Pineda.)

symptoms similar to witches' broom are widespread in southern Vietnam, in Plangyao, Chacheoengsao, Thailand, and also in the Philippines (Figure 8-23). The disease may seriously affect yields and the availability of clean planting material.

Symptoms. Several symptomatologies exist:

1. Plants exhibit dwarfism and an exaggerated proliferation of buds. Sprouts have short internodes and small leaves, but do not show deformation or chlorosis.
2. Proliferation of weak spindly sprouts on the stakes.

Figure 8-23. Disease symptoms observed on cassava plants in the Philippines.

3. Stakes produce only a few dwarf and weak spindly sprouts that never reach normal size.
4. When the affected cassava is uprooted, the roots are thinner and smaller, with rough-textured skins, and drastically reduced starch content.

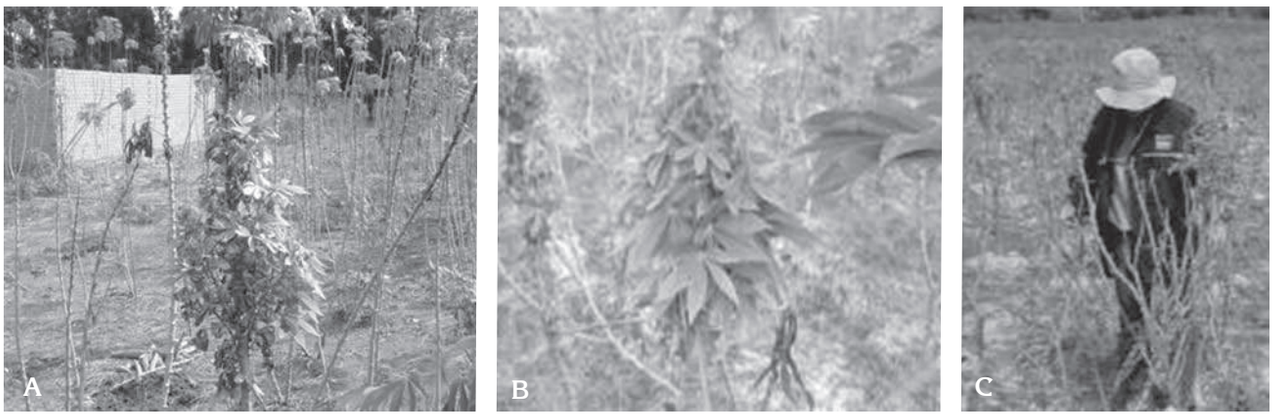

Figure 8-22. Cassava plants in Vietnam with exaggerated bud proliferation; **(A) (B)** shoot proliferation and/or usually **(C)** rachitic branches growing from a single stake; and shoots with short internodes and small leaves that show no deformation or chlorosis. (Photos: JF Mejía.)

The disease is transmitted mechanically and by the use of stakes from diseased plants (Lozano et al. 1981).

Etiology. The transmission of cassava phytoplasmas by *Cuscuta* sp. into pink vinca was 100% positive. Symptoms appeared 3 weeks after implanting the host parasite into pink vinca in growth chambers at 18–20 °C. No transmission was achieved with the insect *Scaphytopius fuliginosus*, even 3 months after exposure to feeding, whether cassava to cassava, cassava to vinca, or vinca to vinca (Valencia et al. 1993).

In Vietnam, disease recognition was carried out in the country's central and southern regions (Quang Ngai and Dong Nai provinces). Samples for diagnosing phytoplasmas were collected in southern Vietnam at Hung Loc Agricultural Research Center and from a farmer's plot in Dong Nai province, both sites about 60 km from Ho Chi Minh City. Phytoplasmas were detected in the samples collected in Thailand and Vietnam. Diagnosis results confirm the association of symptoms (high bud proliferation shoots with short internodes, and small leaves) with phytoplasmas.

Phytoplasmas were detected in roots, small leaves, and leaf veins showing symptoms. No phytoplasmas of the 16SrIII group (reported in America) were found in the samples from Thailand and Vietnam. However, only samples from eastern Thailand and southern Vietnam have been evaluated. These results need to be confirmed. Molecular tests based on the 16Sr gene indicated that differences exist between the phytoplasmas detected in eastern Thailand and southern Vietnam (E Álvarez, JF Mejía, and A Bertaccini 2009, pers. comm.).

Management. The use of healthy planting materials and the elimination of diseased plants in the field are recommended to prevent the disease (Lozano et al. 1981). The disease is reduced by selecting stakes from healthy plants and by restricting the movement of cassava planting stakes, especially from infected areas, and that of related species such as *Jatropha*. Varietal resistance also exists.

Antholysis

Importance. Antholysis in cassava was observed in crops in southwestern Colombia in 1981 by Jayasinghe et al. (1983), severely in some experimental clones. However, this disease does not have economic importance and is only sometimes observed.

Symptoms. The disease appears in the inflorescence, with a characteristic virescence in the petals, which, instead of being their normal pink, become green. Hypertrophy of the petals is later observed and they become structures similar to leaves (phyllody). The floral racemes lose their normal appearance and resemble sprouts, giving this syndrome its name "antholysis" (*antho* – flower; *lysis* – dissolve, loosen) (Figure 8-24).

Figure 8-24. Symptoms of antholysis in cassava: **(A)** healthy flower, **(B)** virescence, and **(C)** phyllody. (Photos: B Pineda.)

Infected flowers commonly exhibit a very swollen gynophore and develop internodes in the floral receptacle, a phenomenon known as apostasis. Furthermore, elongation of the receptacle occurs above the insertion of the pistil, with development of sprouts. Flower fertility is lost, resulting in nonfunctional flowers that abort prematurely. Affected plants do not present symptoms in other organs and, moreover, germination did not differ between infected and healthy stakes (Jayasinghe et al. 1983).

Etiology. By using an electron microscope, Jayasinghe et al. (1983) observed oval or spherical pleomorphic structures only in phloem tissues. Transmission is 100% by stakes. Under greenhouse conditions, symptoms of antholysis appear within 1 month of planting, contrasting with healthy plants, which take 5 months to flower.

Treatment with penicillin (500 to 1000 ppm) did not reduce symptoms, whereas tetracycline reduced antholysis by 90%. This sensitivity and detection by Dienes' stain confirmed that the causal agent is a phytoplasma (Jayasinghe et al. 1983).

Management. The disease is reduced by selecting stakes from healthy plants. Varietal resistance also exists.

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