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Diseases of Cassava

(*Manihot esculenta* Crantz)

J. C. LOZANO and R. H. BOOTH

Centro Internacional de Agricultura Tropical (CIAT)
Apartado Aéreo 67-13, Cali, Colombia, S.A.
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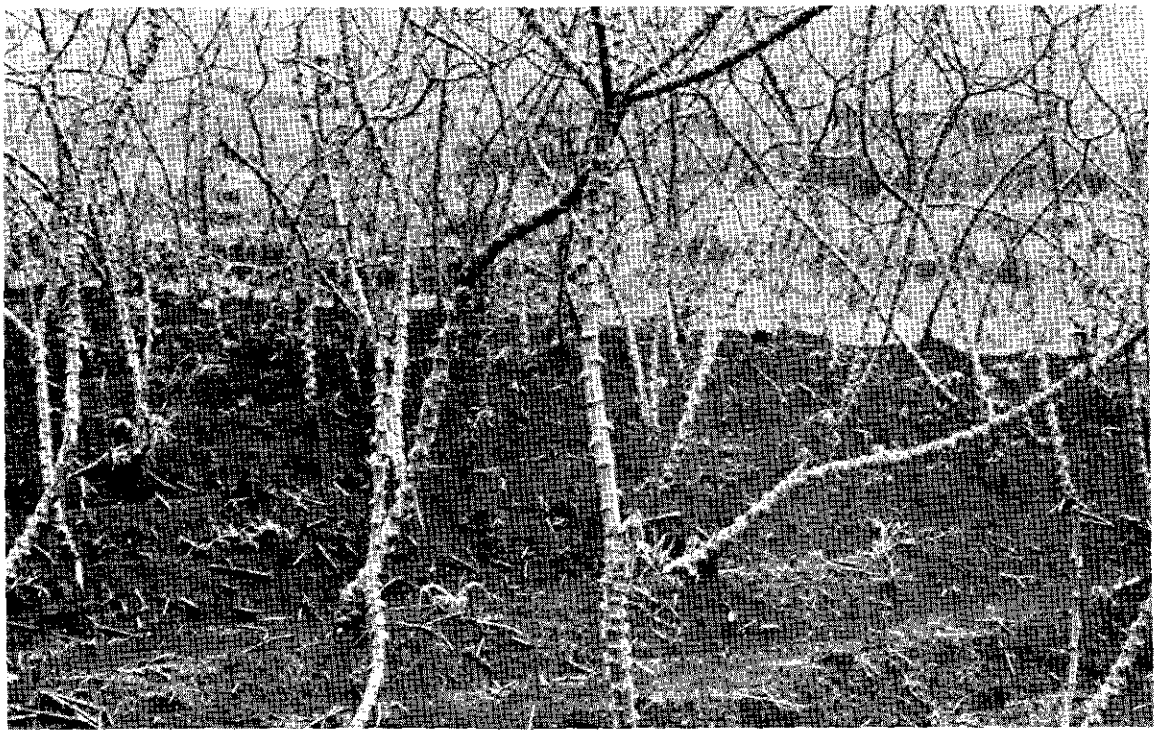


Certain cassava diseases can cause total defoliation in a plantation, with the consequent reduction in yields. **Phyllosticta (Phoma)** sp. is one of the most severe cassava pathogens during the cold and rainy periods.

Diseases of Cassava

(Manihot esculenta Crantz)

J. C. Lozano* and R. H. Booth**

Introduction

With the ever-increasing world population and the resultant decrease in food energy sources, scientific attention is turning to some of the less familiar food crops such as cassava. To increase cassava yields to a maximum, as in any other crop, it is imperative that we increase our knowledge of the many diseases that reduce yields and how they may be controlled. Limited information is currently available about cassava diseases. This paper is an attempt to gather together much of this information and present it with recent observations made by the authors.

In general, the literature implies that cassava diseases are of minor importance. Although facts relating to actual losses caused by the diseases are scarce, anyone who has observed cassava in the field will realise that these diseases are of great importance. The large number of publications which merely mention the existence of different pathogens but fail to present data on their importance, epidemiology or control, serve to illustrate our lack of knowledge.

Cassava suffers from a wide variety of diseases caused by bacteria, viruses and fungi. On a global basis cassava bacterial blight is considered one of the most devastating as it results in complete loss of yield under certain conditions. In Africa, cassava mosaic is undoubtedly one of the most important factors limiting production. Also of widespread importance are the cercospora leaf spots. Many other diseases are less widespread or are only important under certain environmental conditions. Several of the root-rots, for example, may cause severe loss in yield, particularly in poorly drained soils, and phyllosticta leaf spot may cause complete defoliation and die-back in certain cooler cassava-growing areas. Gappy

* Plant pathologist (Bacteriologist), Centro Internacional de Agricultura Tropical, CIAT, Apartado Aéreo 67-13, Cali, Colombia, S.A.

** Visiting scientist (*Cassava pathology*). Centro Internacional de Agricultura Tropical, CIAT, Apartado Aéreo 67-13, Cali, Colombia, S. A. (Permanent address: Tropical Products Institute, TPI, 56-62 Gray's Inn Road, London, WC1).

crops may result from damping-off diseases and stem-rots may cause severe losses of viability in those regions where storage of planting material is necessary. While it is not fully understood whether the cause of the rapid post-harvest deterioration of cassava roots is physiological or pathological, or a combination of the two, several micro-organisms have been shown to result in post-harvest rots and fermentations.

BACTERIAL DISEASES

Cassava bacterial blight

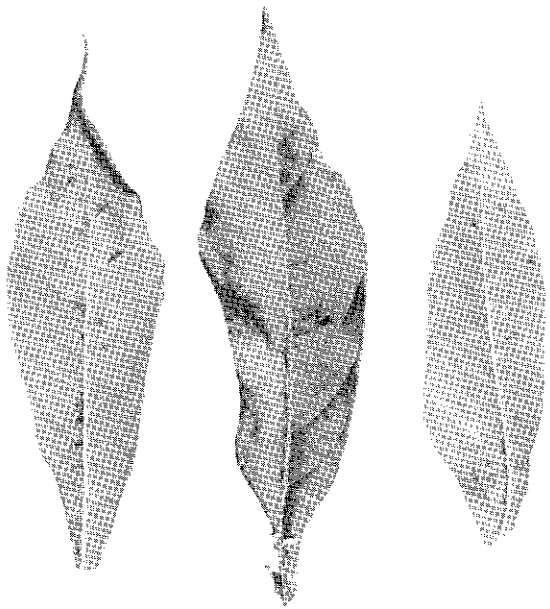
Cassava bacterial blight is the most important of several bacterial diseases reported. It was first recorded in Brazil (Bondar, 1912, Costa, 1940b) but has since been reported in Colombia and Venezuela (Lozano, 1972a; 1973; Lozano and Sequeira, 1973a, 1973b) and has been obseved in several other countries in South America and Africa. This disease is now recognised as one of the most important factors limiting production in affected areas where in wet seasons it can cause complete loss of yield (Drummond and Hipólito, 1941; Elliot, 1951; Lozano and Sequeira, 1973a).

The symptoms, epidemiology, nature of the causal organism, and control of this disease have been studied extensively by Lozano and Sequeira (1973a, 1973b). Symptoms are characterised by leaf spotting and blight, wilting, die-back, gum exudation, and vascular necrosis. Primary symptoms resulting from the planting of infected material are indicated by wilting of the young leaves followed by die-back (Fig. 1). Secondary symptoms, resulting from secondary infections, show leaf spotting followed by blight and die-back. Leaf spots are at first small, angular, and water-soaked but enlarge to cover part or all of the leaf and turn brown (Fig. 2). These necrosed leaves dry up and remain attached to the stem for a short time but later fall off. Gum is characteristically exuded from young infected stems, petioles and leaf spots. Vascular strands of infected petioles and stems necrose and appear as brown strings. This vascular discoloration may also spread into and infect roots (Lozano, 1972a). The disease has only been found in species or varieties of the genus *Manihot* (Bondar, 1915; Amaral, 1942b; Burkholder, 1942).

The causal bacterium was first named *Bacillus manihotis* Arthaud-Berthet (Bondar, 1912) but was later renamed *Phytomonas manihoti* Arthaud-Berthet and Bondar Viegas (Viegas, 1940). However, Drummond and Hipólito (1941) found that some of the characteristics of the bacterium they isolated from cassava were different from those of the species

Cassava bacterial blight. Plant showing typical leaf wilt and die-back symptoms.

originally described by Bondar (1912). Burkholder (1942) concluded that the organism should be placed in the genus *Phytomonas* and the name *Ph. manihotis* was included in Bergey's Manual (Bergey, 1948). Comparative studies of a new isolate with the strains of Burkholder and of Drummond and Hipólito were made by Amaral and Vasconcellos (1945). They concluded that all three strains belonged to *Ph. manihotis*. Later, Starr (1946) changed the name to *Xanthomonas manihotis* (Arthaud-Berthet) Starr.



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Cassava bacterial blight. Leaflets showing angular leaf spots and leaf blight.

However, as a result of studies on morphology, physiology, serology, and phage susceptibility of the bacterium isolated in Colombia and Brazil, Lozano and Sequeira (1973a) concluded that they were sufficiently different from *X. manihotis* to be considered a separate strain. They reported that the cassava blight bacterium differs from *X. manihotis* in cell size, mobility and flagellation, production of H_2S, utilisation of nitrate, hydrolysis of starch, and in several serological relationships. They also reported that a comparison with a type culture of *X. manihotis* revealed differences in pathogenicity, growth rate, serological characteristics and phage susceptibility.

Lozano and Sequeira (1973a) reported the cassava blight bacterium as a gram-negative slender rod, mobile by means of a single polar flagellum, not encapsulated, and not spore forming. It is an aerobic, fast-growing bacterium which forms no pigment on sugar-containing media. It hydrolyses starch and gelatin and reduces litmus milk. It does not induce a hypersensitive reaction on tobacco leaves or cause soft-rotting of potato tubers, or cassava roots. It produces levan, catalase, arginine dehydrolase and lipase, but does not produce H_2S, indole, urease, tyrosinase or phenylalanine deaminase. It is able to grow on media containing NaCl or tetrazolium

chloride at maximum concentrations of 2.5 and 0.2%, respectively. The bacterium utilises nitrate and ammonium as sources of nitrogen; most simple sugars can serve as sources of carbon, but acid is not produced; various amino acids and other organic acids are readily utilised. It can be separated by serological and phage-typing methods from species of *Erwinia*, *Pseudomonas* and *Xanthomonas*, including *X. manihotis*. A species of *Bdellovibrio* caused lysis specifically on this bacterium and could be used to separate it from other plant pathogenic bacteria. As a result of this Lozano and Sequeira (1973a) concluded that the cassava blight bacterium should be considered a strain of *X. manihotis* but needed further revision.

The bacterium normally penetrates the host via stomatal openings and wounds of epidermal tissues (Pereira and Zagatto, 1967; Lozano and Sequeira, 1973a). The bacterium eventually invades the vascular tissues and causes extensive breakdown of parenchymatous tissues in leaves and young shoots. Movement into the stem and petioles takes place primarily through the xylem vessels (Drummond and Hipólito, 1941; Amaral, 1942b, 1945) and possibly through the phloem (Amaral, 1942b; Pereira and Zagatto, 1967). Movement through the pith tissues has also been reported (Drummond and Hipólito, 1941). In mature, highly lignified tissues of old stems the bacterium remains restricted to the vascular tissues. In general, plants develop typical symptoms within eleven to thirteen days of infection (Amaral, 1942b; Pereira and Zagatto, 1967; Lozano, 1972a; Lozano and Sequeira, 1973b).

The possibility that the pathogen spreads from one area to another by infected cuttings or contaminated insects was suggested by Amaral (1945). Many workers (Carneiro, 1940; Goncalves, 1939, 1948, 1953; Drummond and Hipólito, 1941; Lozano, 1972a; Lozano and Sequeira, 1973b) have suggested or demonstrated that the pathogen could be spread by the movement of soil during cultural operations and by the use of infested tools. Drummond and Hipólito (1941) reported an increase in leaf spotting during the rainy season. Lozano and Sequeira (1973b) have clearly demonstrated that rain splashing is the most important means of dissemination in localised areas and that dissemination from one area to another or from one growing season to another occurs largely through infected planting material.

It has been reported (CIAT, 1971, 1972; Lozano and Sequeira, 1973b) that by pruning most of the above ground portion of infected plants spread of the disease may be delayed. However, the success of this method is dependent on the susceptibility of the cultivar and the interval between initial infection and pruning. A successful means of controlling the disease has been developed by Lozano and Wholey (1974, in press). This method involves the rooting of disease free stem tips of infected plants and can

thus be used to clean up infected cultivars or stocks and so provide certified bacteria-free cassava 'seed'. The existence of varietal resistance to this disease has also been noted (Carneiro 1940; Goncalves, 1939, 1948; Drummond, 1946; Drummond and Goncalves, 1953; Lozano and Sequeira, 1973b). Both a hypersensitive reaction and a restriction to penetration and systemic invasion have been observed (Lozano and Sequeira, 1973b). A combination of the use of resistant varieties and disease free planting material would thus appear to be a promising means of control.

Other bacterial diseases

Other bacterial diseases of cassava include a disease reported in Uganda (Hansford, 1938) characterised by leaf spotting and necrosis of the petioles with subsequent defoliation. The stems also become infected but wilting is not described. The pathogen was named *Bacterium cassavae* (Hansford) Burkholder (Bergey, 1957). This organism is a gram-negative, facultative, anaerobic rod which does not form capsules. It is mobile by means of a few peritrichous flagella; it liquifies gelatin; it forms base from milk and acid from glucose, sucrose, maltose, and glycerol, but not from lactose or reduced nitrates. On agar it forms yellow, smooth, entire and translucent colonies.

Wiehe and Dowson (1953) reported a bacterial disease of cassava in Malawi (Nyasaland). This disease is characterised by leaf spots which are at first yellow and circular. As these spots enlarge, they become angular with a brown centre and a broad yellow halo. The leaf veins radiating from the margins of these spots become dark brown but the leaves are shed before the petioles become infected thus preventing stem infection. Under humid conditions a sticky liquid containing bacteria is exuded from lower leaf surfaces and rain splashing of this exudate spreads the disease. The causal agent was named *Xanthomonas cassavae* sp. n. which is a gram-negative rod mobile by means of polar flagellum. Colonies on nutrient agar and glucose agar are pale yellow, confluent, and viscous. It forms acid from sucrose, small amounts of acid from dextrose and maltose, and no acid from lactose, salicin, glycerol, or mannitol. It produces hydrogen sulphide from peptone and nitrites from nitrates (Wiehe and Dowson, 1953; Dowson, 1957).

Pseudomonas solanacearum E.F.SM. has been reported (Burkholder, 1942; Amaral, 1945; Kelman, 1953; Orejuela, 1965) as a pathogen of cassava in Brazil. It induces wilt in young plants but not leaf spotting or gum exudation. Several other bacterial species have been reported inducing soft-rots and/or fermentations of harvested cassava roots. These are discussed below in the section on root-rots.

VIRUS AND MYCOPLASMA-LIKE DISEASES

Several virus diseases are reported and although considerable losses may result from some of these diseases, such as cassava mosaic disease in Africa, research has been scattered and spasmodic. In a recent review of the status of these diseases, Lozano (1972b) pointed out that the information available is limited and incomplete. Disease symptoms are frequently described in general terms but full details are rarely given, losses from these diseases are seldom ascertained, and frequently little information is available on such important subjects as host range and transmission. Similarly, few reports give any detailed biological, physiological, physical, or chemical characteristics of the infective agent.

Cassava mosaic disease

This disease was first reported by Walburg in 1894. It occurs in all parts of East, West, and Central Africa and adjacent islands (Storey, 1936; Storey and Nichols, 1938; Chant, 1959; Jennings, 1960a, 1970) and reported losses in yield from this disease range from 20 to 90% (Lefevre, 1935; Chant, 1959; Jennings, 1960a; Doku, 1965; Beck, 1971). This disease does not appear in other countries and is noticeably absent from the Americas, the recognised source of origin of the crop. It may thus be regarded as a new or introduced disease to this crop.

The symptoms in cassava are characteristic of a mosaic disease. Early in the development of the leaf, chlorotic areas can be observed and leaflets are frequently distorted (Fig. 3). Leaves are sometimes reduced in size, misshapen and twisted, with bright yellow areas separated by normal green tissue (Fig. 4) (Jennings, 1960a). This disease has only been found in *Manihot* spp.

Successful transmission by *Bemisia* spp. (whitefly) has been reported. For whiteflies to become viruliferous, it is necessary for them to feed for at least four hours on young diseased leaves followed by a further four-hour incubation period (Storey and Nichols, 1938; Chant, 1958; Jennings, 1960a).

Several attempts have been made to purify the infective agent without success. Gálvez and Kitajima (pers. comm.) were unable to find virus-like particles in either leaf-dip preparations or in ultra-thin sections. Their purification trials were also unsuccessful. Barbee (pers. comm.) reports finding two components in a disease extract by his purification procedure.

Gálvez (pers. comm.), however, suggests that this disease should not be classified as caused by a virus until further research clarifies the identity of the causal agent.

The only effective control of this disease is by the use of resistant varieties (Storey, 1936; Jennings, 1960a; Doku, 1965; Beck, 1971; Dubern, 1972; Hahn, 1972).

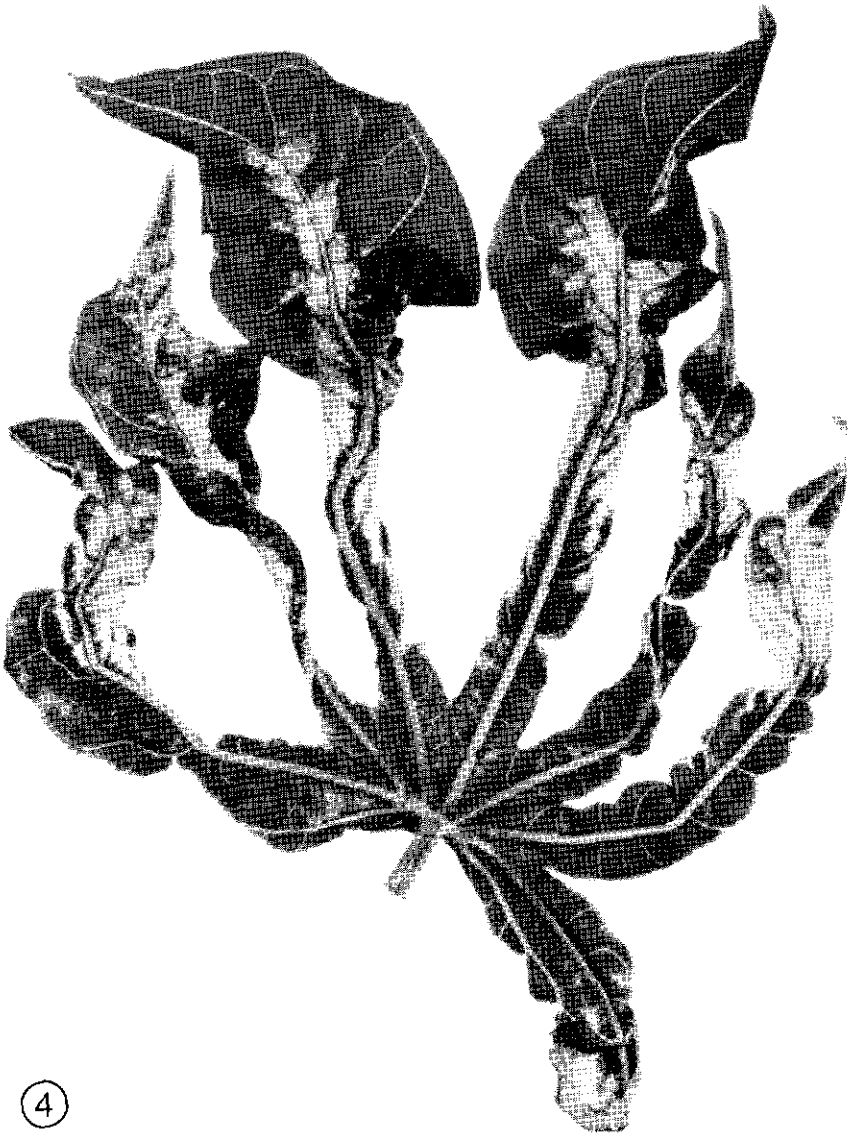
A similar mosaic disease has recently been reported in Kerala, India (Menon and Raychaudhuri, 1970). The symptoms of this disease are similar to those reported above and it is also transmitted by whitefly. As well as being found in *Manihot* spp. it has been recorded in cucumber (Menon and Raychaudhuri, 1970). Whether this is the same disease as that reported above in Africa is not known.

Cassava common mosaic disease

Common mosaic disease occurs in various parts of Brazil (Costa, 1940a; Costa *et al.*, 1970) and has also been reported in Colombia

Cassava mosaic disease (African mosaic). Leaf showing typical chlorosis and deformation (Photograph courtesy of Dr. R. Williams, IITA, Ibadan, Nigeria.)

(Kitajima and Lozano, pers. comm.). Losses in yield range from 10 to 20% but because of its ease of control the disease is considered comparatively unimportant (Costa *et al.*, 1970).



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Cassava mosaic disease (African mosaic). Chlorosis, reduction of leaf lamina and distortion of severely infected leaf. (Photograph courtesy of Dr. R. Williams, IITA, Ibadan, Nigeria.)

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Cassava common mosaic disease (Brazilian mosaic). Leaflets showing mild and severe symptoms.

The symptoms in cassava are characteristic of a mosaic disease and consist mainly of chlorosis of the leaf blade. These chlorotic areas are not usually as well demarcated as those of the cassava mosaic in Africa, but otherwise the general symptoms are similar (Fig. 5). The host range of this virus is relatively wide and it is able to attack *Manihot* spp., *Euphorbia prunofolia*, *Chenopodium amaranticolor*, *C. guinoa*, *Malva parviflora*, and *Gossypium hirsutum* (Costa *et al.*, 1970).

The disease has been transmitted mechanically and by grafting, but no natural vector has been recorded (Costa *et al.*, 1970). Virus infectivity is destroyed when infected sap is heat treated at 65–70°C for 10 minutes, but at 20°C infected sap remains infective for at least 24 hours (Kitajima and Costa, 1966a; Costa *et al.*, 1970).

The virus particles are elongated flexuous rods measuring 15 mμ diam. with a normal length of about 500 mμ (Kitajima *et al.*, 1965; Kitajima and Costa, 1966a; Costa *et al.*, 1970) with good antigenic properties (Silva, 1962; Costa and Kitajima, 1972a).

Effective control has easily been achieved by the use of clean vegetative planting material and by roguing diseased plants from plantations (Costa and Normanha, 1939; Costa *et al.*, 1970).

Cassava brown streak disease

Brown streak disease was first recorded and described in 1936 (Nichols, 1950), and is reported only to occur along the east coast of Africa and at altitudes below 3,500 ft (Nichols, 1950; Jennings, 1960b). Recent information suggests that this disease is very uncommon. Losses are difficult to estimate because the plants are usually infected simultaneously with mosaic disease. Because diseased roots are unfit for human consumption, losses are considerable (Jennings, 1972; Lozano, 1972b).

Infected plants show chlorosis of the leaves, necrosis of the root storage tissues and leaf scars remain longer than expected after normal leaf drop. Brown lesions sometimes occur on the young green stems (Nichols, 1950; Jennings, 1960b). This virus is able to infect *Manihot* spp., *Petunia hybrida, Datura stramonium, Nicotiana tabacum* and *N. glutinosa* (Lister, 1959; Jennings, 1960b; Kitajima and Costa, 1964).

Transmission by both mechanical means and by grafting has been reported (Storey, 1936; Nichols, 1950; Lister, 1959) and whereas transmission by vectors is suspected, it has not been demonstrated (Lister, 1959; Nichols, 1950). Virus infectivity is destroyed when infected sap is heat-treated to 50°C for 10 minutes and at 20°C sap is reported to lose infectiveness in less than 24 hours (Kitajima and Costa, 1964). Kitajima and Costa reported the dilution end point of the virus to be 1:1000. They found rod-shaped particles about 60 mμ long during electron microscope examinations of dried material.

Effective control has been obtained by using disease-free planting material. Resistant varieties have been reported (Jennings, 1960b; Nichols, 1950; Storey, 1936).

Cassava vein-mosaic disease

This disease is reported to occur in scattered localities in Brazil, but little reliable information is available (Costa, 1940a; Kitajima and Costa, 1966b; Costa *et al.*, 1970). Symptoms of this disease are characterised by vein-clearing and leaf curling, and it is reported to be transmitted mechanically and by grafting. *Manihot* spp. and *Datura stramonium* are the only known hosts. Electron microscope examinations of diseased material have revealed spheroidal particles of about 50–60 mμ *in vivo* (Costa, 1940a; Kitajima and Costa, 1966b; Costa *et al.*, 1970).

Witches broom disease

This has been reported in Brazil, Venezuela (Goncalves *et al.*, 1942; Normanha *et al.*, 1946; Costa *et al.*, 1970; Kitajima and Costa, 1971) and Mexico (Kitajima, Costa and Normanha, 1972; Costa and Kitajima, 1972a, 1972b). Reduction in yield can be relatively high, sometimes in excess of 80% (Goncalves *et al.*, 1942; Silberschmidt and Campos, 1944; Normanha *et al.*, 1946). Diseased plants can be recognised by their stunted appearance, shortening of internodes, and excessive proliferation of branches. However, care must be taken in diagnosing this disease as similar symptoms have been shown to result from heavy thrip infestations (Lozano and Schoonhoven, pers. comm.). In fact, little is known about this disease, but Costa *et al.* (1970), Kitajima and Costa (1971) and Costa and Kitajima (1972b) have concluded that the disease is associated with a mycoplasma-like organism.

Latent virus

A latent virus has been reported by Costa *et al.*, (1970). This virus has no symptoms in cassava but is thought to be widespread. It is a rabdovirus of 280--300 mμ (Costa *et al.*, 1970).

FUNGAL DISEASES

Many fungal diseases of cassava, varying considerably in their distribution and importance, have been reported. Those diseases considered to be most widespread or important in particular situations are described here as leaf diseases, stem-rots and root-rots.

A. Leaf diseases

Several *Cercospora* spp. have been reported to induce leaf spots on cassava. Considering severity and geographical distribution *C. henningsii* Allescher and *C. caribaea* Chupp and Ciferri appear to be the most important (Cardin, 1910; Ghesquiere and Henrard, 1924; Viegas, 1941; Golato, 1963; Golato and Meossi, 1966; Castaño, 1969). Although the economic importance of these pathogens on cassava is undetermined, several reports (Sydow, 1901; Deslandes, 1941; Chevaugeon, 1956; Normanha and Pereira, 1964; Castaño, 1969; Jennings, 1970; Golato and Meossi, 1971) suggest that they are important in certain geographic areas during the rainy seasons.

Brown leaf-spot *(C. henningsii)* is probably the most important of all the cassava leaf diseases. This disease is widely distributed and can be found in Asia and North America in addition to Africa and Latin America. The pathogen appears to have one of the widest host ranges of the *Cercospora* spp. attacking *Manihot glaziovii* (cera rubber), *M. piauhynsis,* and by artificial inoculation, *Ipomea batatas* (sweet potato), in addition to *M. esculenta* (Viegas, 1941; Golato, 1963; Ferdinando *et al.,* 1968; Powell, 1968, 1972; Golato and Meossi, 1971).

C. henningsii grows in the intercellular spaces of the leaves and produces stromata from two to six cells in depth and from 20–45 μ in diameter. From these stromata conidiophores are produced in dense fascicles. The conidiophores are pale olivaceous brown (medium-dark in mass), uniform in colour and width, unbranched, 0–2 midly geniculate, rounded at the tip with a small to medium spore scar, straight or nearly so and measuring 3–5 x 10–50 μ, rarely as long as 100 μ with the longest ones sparingly septate. The amphigenous conidia, produced singly at the apex of each conidiophore, are cylindric, straight or slightly curved, with both ends bluntly rounded or with a short abconic base, plainly 2–8 septate, pale olivaceous, and measuring 4–6 (7) x 30–60 (85) μ (Chupp, 1953; Powell, 1968, 1972). Black perithecia, 100 μ diam, occasionally appear scattered in the necrotic tissue of the foliar spots on the upper surface of the leaf. The asci are elongate-clavate, eight-spored, sub-sessile, 55–72 x 10–13 μ. The ascospores are ovoid, uniseptate, constricted at the septum, 17–22 x 5.2–6.8 μ. The upper cell of these spores is of greater diameter than the lower and is drawn out as a candle flame (Chupp, 1953; Powell, 1972).

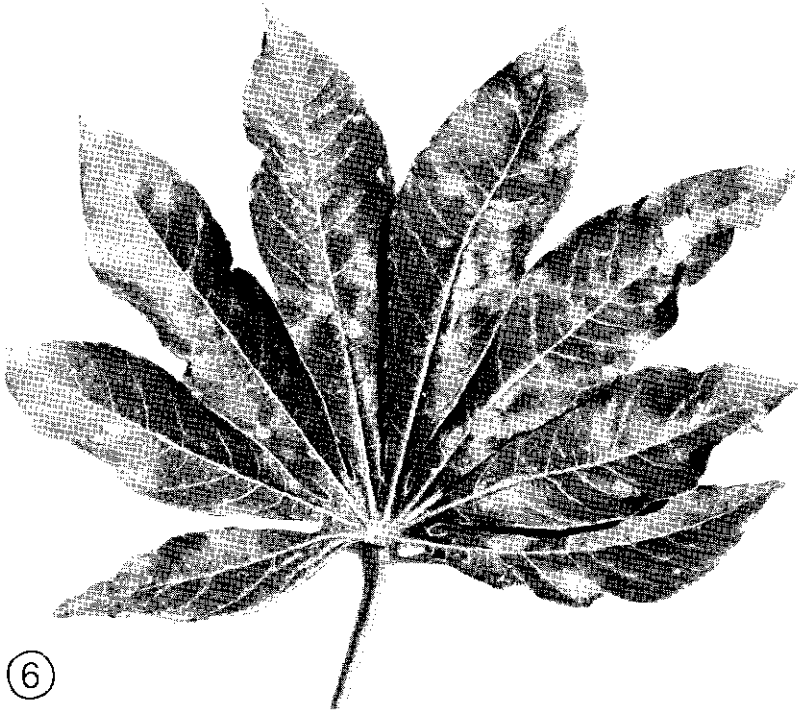
The perfect state of *C. henningsii* was reported as *Mycosphaerella manihotis* Ghesquiere Henrard non Sydow (Ghesquiere and Henrard, 1924; Ghesquiere, 1932) and later corroborated by Chevaugeon (1956). However, the genetic relationship between the stages has not been proven. Powell (1972) suggested a new nomination needs to be provided for the sexual state as the one in use is a later homonym of the name given by Sydow (1901).

C. cassavae Ell. & Ev.; *C. manihotis* P. Henn., *C. cearae* Petch, *C. manihoticola* Stev. Ined., C. *manihotis* P. Henn., *Helminthosporuim manihotis* Rangel; *H. hispaniolae* Cif., and *Septogloerum manihotis* Zinn, are all considered to be synonymous with *C. henningsii* (Ciferri, 1933, Chupp, 1953; Powell, 1972).

Symptoms on cassava are characterised by leaf spots on both sides of the leaves. On the upper surface the spots appear uniformly brown with a

distinct darker border (Fig. 6). On the lower surface the lesions have less distinct margins and in the centre the brown spots assume a greyish cast because of the presence of conidiophores and conidia of the fungus. As these flat circular lesions, 3–12 mm diam, grow they become somewhat irregular and angular in shape as they are limited by the leaf margin or major veins. Small veins within the lesions appear black. Sometimes, depending on the susceptibility of the variety, an indefinite halo or blighted area is present around the lesions. As the disease progresses, infected leaves turn yellow and dry, and eventually drop. Susceptible varieties can thus be severely defoliated during warm rainy seasons.

Primary infections are initiated in new plantings when wind or rain carry conidia from lesions on old fallen infected tissues to infection courts on leaf surfaces. If sufficient moisture is present, the conidia germinate, producing branched germ tubes which frequently anastomose. Penetration



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Brown leaf-spot **(Cercospora henningsii)**. Large brown lesions with distinct borders.

occurs through stomatal cavities and invasion of the tissues through intercellular spaces. In warm, humid conditions infection usually occurs within twelve hours (Wallace, 1931; Ciferri, 1933; Viegas 1941, 1943a, 1943b; Chevaugeon, 1956).

When these lesions mature, conidiphores are produced from the stomata. Secondary disease cycles are repeated throughout the rainy season whenever conidia are carried to new sites of infection by wind or rain. The fungus survives the dry season in old lesions, often on fallen leaves, and renews its activity with the coming of the rainy season and the renewed growth of the host.

Chevaugeon (1956) demonstrated that on a given plant the older, lower leaves are more susceptible than the younger, upper leaves. This is corroborated by other authors. However, it has been observed that some susceptible species *(M. carthagenensis)* and cultivars of *M. esculenta* may be severely and evenly attacked. Leaflets, young leaves, petioles, and even fruits of *M. carthagenensis* have been observed with severe disease symptoms. It is reported that plants that have been"hardened"by unfavorable growing conditions become more resistant (Viennot-Bourgin and Grimaldi, 1950) but no differences in susceptibility between plants grown on rich or poor soil were found (Chevaugeon, 1956).

Cultural practices, such as wider spacing, directed towards reducing excess humidity in the crop stand are recommended to reduce infection (Springensguth, 1940; Golato, 1963, Golato and Meossi, 1966). The use of copper oxides and copper oxychlorides suspended in mineral oil applied at a rate of 12 l/ha have been reported to give good control (Golato, 1963; Golato and Meossi, 1971). However, the best control of this disease is obtained by planting resistant varieties. Significant differences in varietal resistance have been reported in Africa (Chevaugeon, 1956; Umanah, 1970), Brazil (Viegas, 1941, 1943a, 1943b) and in the extensive collection of cassava varieties at CIAT, Colombia (CIAT, 1972).

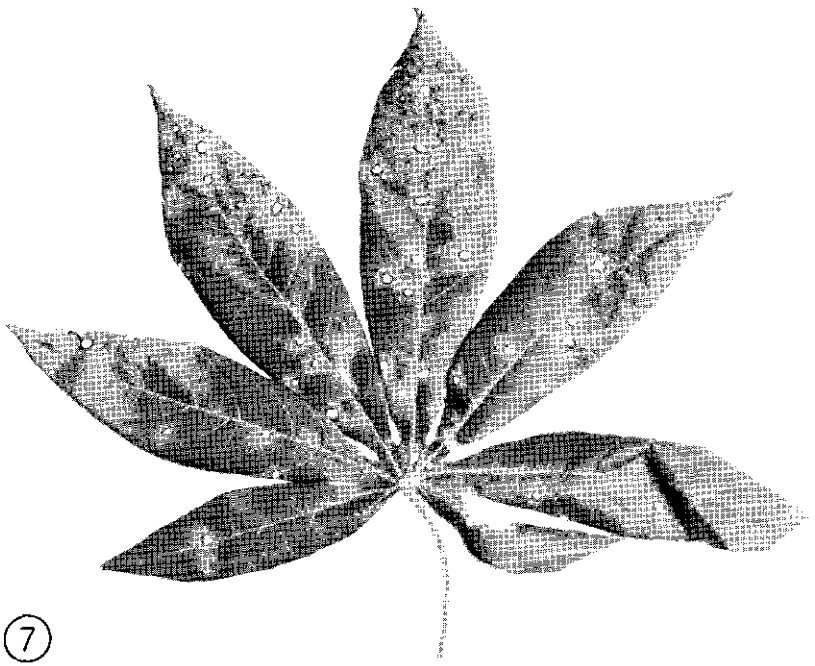
White leaf-spot *(C. caribaea)* is commonly found in the humid but cooler cassava-growing regions and has been reported in certain areas of Asia, North America, tropical Africa, and Latin America (Viegas, 1941; Viennot-Bourgin and Grimaldi, 1950; Chevaugeon, 1956; Castaño, 1969; CIAT, 1972). In these areas this pathogen may cause considerable defoliation of susceptible varieties of *M. esculenta,* the only reported host species (Viegas, 1941; Chevaugeon, 1956).

C. caribaea forms slight stromata in infected leaves from which conidiophores are produced in loose fascicles. The conidiophores which emerge through stomata are usually olivaceous brown, uniform in colour

and width; rarely branched, 1–15 geniculate, sub-truncate at the tip with a fairly large spore scar and measure 3–5 x 50–200 μ. The hypophyllous conidia are hyaline to subhyaline, obclavate-cylindric, with bluntly rounded ends, 1–6 septate, straight or nearly so, and measure 4–8 x 20–90 μ (Chupp, 1953; Powell 1968, 1972).

While the name *C. caribaea* Chupp and Ciferri is widely accepted for this fungus, Powell (1972) states that the name is not at present valid and will only be validated by the publication of a full latin description. This species can easily be distinguished from other *Cercospora* spp. on *M. esculenta* by the leaf symptoms and by the hyaline conidia produced (Chupp, 1953; Powell, 1968).

Lesions caused by *C. caribaea* are smaller and different in colour to those induced by *C. henningsii.* They are circular to angular, usually 1–7 mm diameter, and white, or rarely yellowish brown (Fig. 7). The lesions are sunken from both sides to about one-half the thickness of the healthy



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White leaf-spot *(Cercospora caribaea).* Small white lesions with distinct violet-brown border and diffuse yellow halos.

leaf blade. While the white spots remain distinct, the lesions frequently have a diffuse coloured border on the lower leaf surface. The border sometimes appears as an irregular violet-brown line surrounded by a yellow or brownish halo. The centre of the spots are given a greyish velvety aspect during the fructification of the pathogen which occurs predominantly on the underside of the leaf.

Penetration occurs through stomatal cavities and invasion of the tissues through intercellular spaces. When the leaf spots thus produced reach about 5–7 mm a stroma is formed from which the conidiophores are later produced. Secondary disease cycles are repeated throughout the rainy season when the conidia are dispersed by rain splash. The fungus survives the dry season in old, infected tissues and renews its activity with the coming of the rainy season and the renewed growth of the host.

Recommended control measures for this disease are similar to those for brown leaf spot. Specific resistant varieties have not been reported, but field observations suggest such resistance exists.

The development of the two diseases, brown and white leaf spots, is similar but generally brown leaf spot is more common in hot, dry regions and white leaf spot in humid, cooler cassava-growing areas. These distribution differences reported in Africa (Chevaugeon, 1956) and Latin America (CIAT, 1972) are probably the result of differences in temperature and moisture responses of the two causal fungi. The optimum temperatures for conidial germination of *C. henningsii* and *C. cariabea are* 39 and 33°C, respectively, and the maximum temperatures to allow germination are 43 and 33°C, respectively. Conidia of *C. henningsii* will germinate at 50% R.H. with optimum germination at 90% while conidia of *C. caribaea* need to be immersed in water for normal germination. Nutritional studies have also revealed differences between the two fungi; *C. henningsii* is able to utilise acetate, citrate, and various amino acids but not pentoses. *C. caribaea* however utilises pentoses as energy and carbon sources but does not generally utilise trioses (Chevaugeon, 1956; Powell, 1968).

C. viscosae Muller and Chupp is the causal agent of a diffuse leaf spot in the warm cassava-growing areas of Brazil and Colombia (Viegas, 1941; CIAT, 1972). Leaf spots are large and brown without definite borders. Each spot frequently covers one-fifth or more of the leaf lobe. The upper surface of the spot is uniformly brown but on the under surface the centres of the brown lesions assume a greyish cast because of the presence of conidia and conidiophores of the fungus. The general appearance of the lesions is similar to those induced by *Phyllosticta* sp. but can be distinguished from the latter which usually have concentric rings around the lesions on the upper leaf surface.

The fungus does not form a stromata but sporulates profusely. The conidiophores produced in coremoid fascicles are dark reddish-brown, measuring 4–6 x 50–150 μ. The conidia produced are cylindro-obclavate and 4–6 x 25–100 μ (Chupp, 1953).

C. viscosae has only been found infecting *Manihot* spp. The disease occurs during the rainy season in warm cassava growing areas where brown leaf-spot is also usually prevalent. The disease is not usually serious and is confined to the older leaves where some defoliation may occur.

C. manihobae Viegas has been reported to induce distinct leaf spots on *M. esculenta* in Brazil (Viegas, 1941, 1943b; Chupp, 1953). Leaf spots are reported (Viegas, 1941, 1943b) to be characteristically snow-white in appearance, but a full description of the disease is not available.

The fungus produces medium dark coloured conidiophores measuring 3–5 x 50–200 μ. The conidia are hyaline to subhyaline, obclavate-cylindric, and 4–8 x 20–90 μ (Chupp, 1953).

Phyllosticta *leaf-spot*

This disease is commonly found in the cooler cassava-growing areas of Colombia (CIAT, 1972) and Brazil (Viegas, 1943a) and has also been reported in the Philippines (Sydow, 1913), Tropical Africa (Vincens, 1915), and India (Ferdinando *et al.*, 1968). During rainy seasons and when the temperature is below 22°C, this disease may cause severe defoliation of susceptible varieties, finally resulting in die-back of the plants. The disease has also been reported to occur on *Manihot heptaphylla, M. dichotoma* (Reinking, 1919, Viegas, 1943a) and *M. aipi* (Spegazzini, 1913; Viegas, 1943a) in addition to *M. esculenta* (Viegas, 1943a).

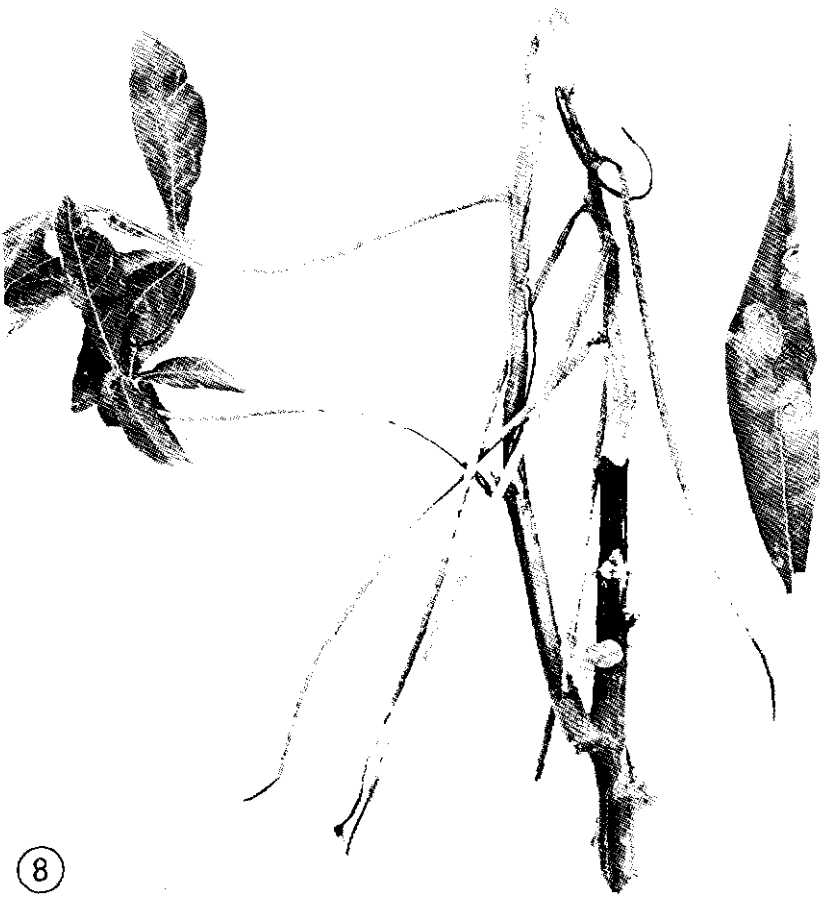
The causal agent of this disease has not been clearly defined, and several *Phyllosticta* spp. have been reported (Sydow, 1913; Vincens, 1915; Reinking, 1919; Viegas, 1943a; CIAT, 1972) as inducing the same disease syndrome. Vincens (1915) first described the causal agent as *Haplographium manihoticola* Vincens, but the pathogenicity of this fungus was later questioned by Viegas (1943a). *Phyllosticta manihoticola* Sydow (Sydow, 1913), *P. manihot* Sacc. (Saccardo, 1931), and *P. manihobae* Viegas (Viegas, 1943a) have all since been reported as pathogenic on cassava. As the full definition and taxonomic validity of these species have not been fully determined, the possibility remains that they could be synonymous and that there is only a single cassava pathogenic

species. Recent studies and observations indicate that this fungus should be classified as a *Phoma* sp. (Powell, personal communication). A full taxonomic study of a wide range of pathogenic isolates is urgently needed to clarify this point.

The causal fungus produces numerous epidermal pycnidia which are dark-brown, globose, and borne singly or in small clusters on infected leaves and stems. The pycnidia are 100–170 μ in diameter with walls formed of polyhydrical cells and have an ostiole measuring 15–20 μ. The conidiophores are short and hyaline and produce small (15–20 μ), one-celled, ovoid to elongate conidia (Viegas, 1943a; Ferdinando *et al.*, 1968). The fungus isolated in Colombia forms profuse pycnidia in concentric rings on lima bean agar.

The disease on cassava is characterised by the presence of large brown leaf spots, usually with indefinite margins. These lesions are commonly found at the tips or edges of the leaf lobes or along the midrib or main veins. The upper surface of the lesions initially consists of concentric rings formed by brown pycnidia (Fig. 8). These rings are frequently absent from old lesions as mature pycnidia are washed off by rain drops. In these cases the uniformly brown lesions may resemble those caused by *C. viscosae*. On the lower surface few pycnidia are produced so the lesions are uniformly brown. Under conditions of high relative humidity, the lesions may be covered with a greyish-brown hyphal weft. On the underside of the leaves the veins and veinlets around the lesions become necrosed thus forming black strings radiating out from the lesions. These lesions grow, causing a leaf blight, and finally the whole leaf and petiole become dark brown and are necrosed. At this stage the leaves wilt and then drop, in some cases causing extensive defoliation. In severe infections the fungus also attacks the young shoots causing a die-back (Fig. 8). Diseased stems turn brown and are frequently covered with pycnidia.

Field observations suggest that the older lower leaves may be more resistant than younger upper leaves. However, young leaves, fully expanded mature leaves, and green stem parts have all been seen with severe disease symptoms. It has also been observed that disease occurrence is correlated with conditions permitting spore germination. Maximum spore germination has been observed between 20 and 25°C and artificial inoculations succeeded only at temperatures below 25°C. Similarly, under field conditions the disease is always found at higher altitudes or in lowland areas during the rainy season. Survival of the fungus during dry periods or from one season to another is not understood. It is suggested (Viegas, 1943b) that the fungus may produce a sexual stage in infected stem and leaf debris, but this has not yet been confirmed.



(8)

Phyllosticta leaf-spot *(Phyllosticta* spp.). Leaflet showing large brown lesions with concentric rings. Young stem showing die-back and presence of pycnidia.

No control measures have been reported for the disease, which can cause serious losses in certain areas under specific environmental conditions. Although no reports of varietal resistance are available, field resistant cultivars have been observed in naturally infected plantations in Colombia. Chemical treatment during rainy seasons could also be beneficial in those areas where the disease is known to be endemic.

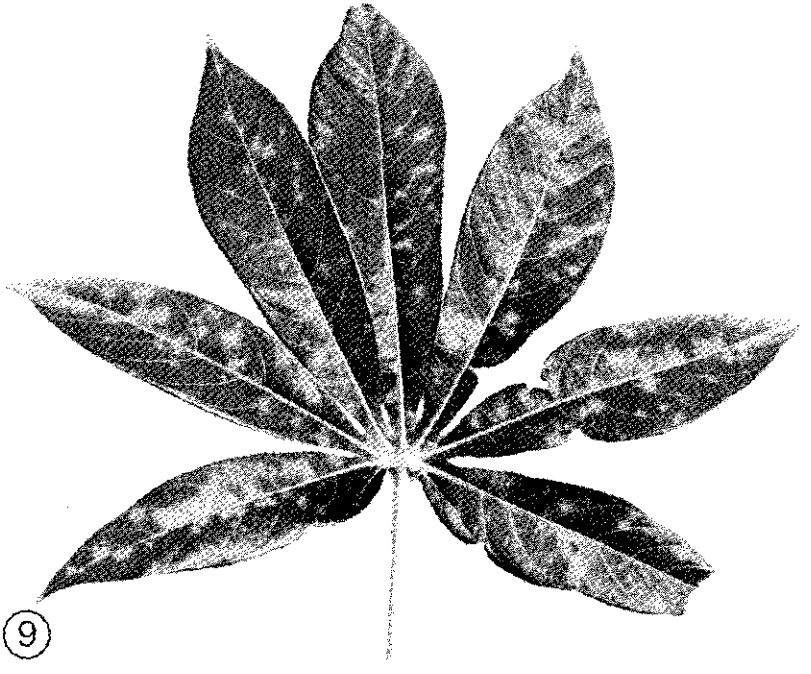
Cassava ash disease

The disease was first reported in Africa (Saccardo, 1913) but has since been reported in Latin America (Viegas, 1943a; CIAT, 1972) and Asia

(Park, 1934) and observed in several other countries. The disease is known only to cause yellowish undefined leaf spots on *M. esculenta.* Although widely distributed and of common occurrence, this disease is considered to be of relatively minor importance.

The causal agent has been named as *Oidium manihotis* P. Henn. the sexual stage of which has been described as *Erysiphe manihotis* (Ferdinando *et al.*, 1968). The fungal mycelium is white, producing numerous haustoria on the host epidermis. Conidiophores are upright and simple with the upper portion increasing in both length and width as conidia are formed. The conidia are oval or cylindrical, one-celled, hyaline, measuring 12–20 x 20–40 μ, and produced in basipetal chains (Saccardo, 1913; Viegas, 1943b; Ferdinando *et al.*, 1968).

The first symptom of the disease is the appearance of white mycelium growing over the leaf surface. The fungus penetrates the cells by means of haustoria, infected cells becoming chlorotic and thus forming yellowish undefined lesions (Fig. 9). Within these yellowish areas pale-brown angular



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Cassava ash disease *(Oidium manihotis).* Typical undefined yellowish leaf lesions.

water-soaked spots of different sizes frequently develop and necrose. In certain varieties, the disease never progresses beyond the yellowish undefined lesion stage. These symptoms are sometimes confused with those induced by insects and spiders.

Mature, fully expanded leaves appear to be the most susceptible, but young leaves of certain varieties are also frequently infected. The disease is found commonly during dry seasons in the warmer cassava-growing areas.

Although specific control measures against this disease are not generally considered necessary, resistant varieties have been observed (CIAT, 1972). It has also been suggested (Ferdinando *et al.*, 1968) that spray applications of sulphur compounds control the disease.

Superelongation disease

Superelongation disease has recently been found inducing epiphytotics in several areas of Colombia (CIAT, 1972; Lozano, 1972; Lozano and Booth, 1973). The disease has been found during rainy seasons, and at the onset of dry periods, infection and disease spread decline. The yield of heavily infected plants is severely reduced.

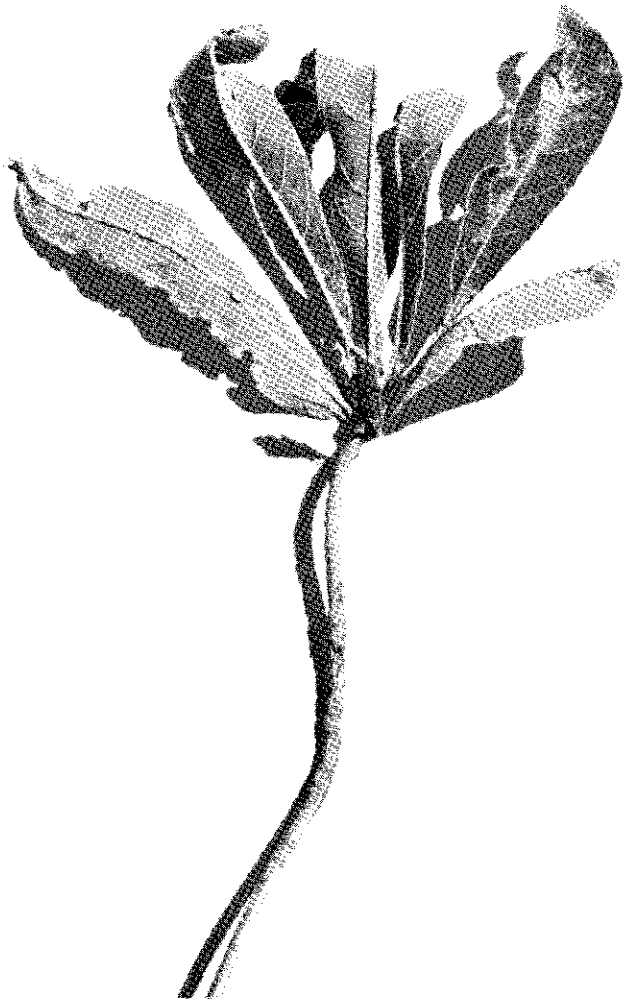
A fungus, according to CMI* it is possibly a species of *Taphrina* or *Sphaceloma*, has been found to be the causal agent. This organism grows well in any artifical media containing peptone and sugar forming yeast-like colonies. Each colony is circular, corrugated, slightly sunken and of a hard consistency. Initially the colonies are yellowish but after fifteen days incubation they turn dark brown. These colonies are formed of a promycelial type of structure. A delicate septate mycelium composed of binucleate, elongate or vasicular cells is formed around each colony. On artificial media tiny binucleate spores are produced in ten days. Preliminary observations suggest that these are blastospores capable of multiplying by budding (Lozano and Booth, 1973).

Histological studies have shown that the fungus initially grows over the epidermis and that following penetration it grows in the intercellular spaces of the epidermis and cortex. Infection of vascular or medullary tissues has not been observed. Following infection, mycelial aggregates which are formed in the cortex push up and rupture surrounding epidermal cells to form a canker. Most cells around such cankers are abnormally large (Lozano and Booth, 1973).

* Commonwealth Mycological Institute

In the field the disease is recognised by the exaggerated elongation of the internodes of young stems which appear thin and weak (Fig. 10). Infected plants are considerably taller than healthy ones. Young shoots, petioles and leaves frequently show a distortion which is usually associated with lens-shaped cankers formed along the midribs or veins of leaves and on the petioles and young stems. Often leaves are not completely developed and leaf lamina not fully expanded. White irregular spots are frequently present on young leaves (Fig. 11). Partial or total necrosis of young leaf laminae sometimes occurs and results in considerable defoliation. The cankers vary in size and are normally lens-shaped but may be more diffuse on the stems and resemble damage by thrips.

The superelongation disease (*Taphrina* sp. or *Sphaceloma* sp.). General symptoms showing young stem and petiole elongation, leaf deformation and distortion, and cankers.



(11)

The superelongation disease (*Taphrina* sp. or *Sphaceloma* sp.). Leaf showing leaf curl symptoms, irregular white spots on the leaf lamina, and cankers on the midribs, veins and petiole.

During the rainy seasons the spread of the disease is extremely rapid. Dissemination is thought to occur by wind and/or rain-borne spores. High relative humidity appears to be necessary for spore germination and infection. Symptoms in the form of yellow leaf markings appear within six to eight days of inoculation and cankers are rapidly formed.

Field observations of more than two hundred cultivars of *M. esculenta* have indicated possible sources of resistance. Preliminary studies to control this disease using chemical sprays also show promise. A similar disease causing stem elongation and leaf spotting and characterised by pustules on stem, petioles and midribs has been reported in Mexico (Normanha, pers. comm.).

Anthracnose (wither-tip)

This disease has been reported as a disease of cassava in many countries (Bouriquet, 1946; Vanderweyen, 1962; Affran, 1968; Doku, 1969; CIAT, 1972) but it is generally considered to be of minor importance. Sunken leaf-spots about 10 mm diam. and similar to those caused by *C. henningsii* are produced at the base of leaves, which may subsequently die. Stems may also be attacked causing a wilt of very young stems and producing cankers on older ones (Vanderweyen, 1962; Irvine, 1969). New leaves produced at the beginning of rainy seasons are reported to be the most susceptible, and the disease tends to disappear at the approach of dry seasons (Doku, 1969; Irvine, 1969). Similarly, it has been found that artificial inoculations using spore suspensions are only successful when the plants are kept for 60 h at 100% R.H. (CIAT, 1972).

The causal organism has been variously reported as *Glomerella manihotis* Chev., *Colletotrichum manihotis* Henn. (Vanderweyen, 1962), *Gloesporium manihotis* (Bouriquet, 1946), and *Glomerella cingulata* (Irvine, 1969). It is possible that all these refer to the same fungus.

A stem anthracnose caused by a *Colletotrichum* sp., has recently been reported in Nigeria (IITA, 1972). On young green stems oval, pale brown, shallow depressions bearing a spot of normal green tissue in the centre are formed. On the bark of woody stems it produces raised, round, stringy lesions which develop into deep cankers and may distort the stem. The importance is not known.

Leaf and stem rust spot

This has been reported in Brazil (Amaral, 1942a; Normanha, 1970) and appears at the end of dry periods causing a kind of witches broom at the apex of the stems (Normanha, 1970). In Colombia leaf, petiole and stem pustules have been observed on cassava growing in cool upland regions, but Normanha (1970) states that the disease is rarely serious except occasionally in the north-east of Brazil during the hot, dry seasons.

B. Stem-rot disease

Three stem-rot diseases have been observed on stems stored for planting (CIAT, 1972). (The storage of planting material is necessary in those areas which do not have a continuous growing season.) At CIAT these diseases greatly reduce viability, directly and also indirectly through increased desiccation of the cuttings. About 18% of apparently disease-free planting material was discarded because of disease after fifty days storage at ambient conditions in the laboratory. To reduce loss of viability because of desiccation stem cuttings were dipped in paraffin wax which, however, considerably increased disease incidence.

While three distinct diseases have been recognised, it is not always possible to distinguish among them. Macroscopically, these diseases may appear similar, particularly during their early stages of development. Furthermore, more than one of the rot producing organisms may be present.

Glomerella stem-rot

This disease is the most common stem-rot of stored cassava cuttings. The same fungus also infects old stem debris left in cassava plantations. The rot first appears at the cut ends and gradually spreads throughout the cuttings. A black discolouration of the vascular strands precedes the development of surface blisters which later rupture the epidermis exposing black groups of perithecia in a well developed stroma (Fig. 12).

According to CMI the causal organism appears to fall within the general broad concept of *Glomerella cingulata* (Stonem.) Spauld. Schrenk. Ascospores are hyaline, one-celled, and slightly curved. Infection is thought to occur through wounds and to be favoured by high relative humidities. The relationship between this fungus and the *Colletotrichum* sp. that causes anthracnose of cassava has not been determined. However, the possibility exists that these may be two different stages of the same fungus.

Botryodiplodia *stem-rot*

This disease has been found infecting stored stem cuttings and old stem debris in the field but is much less common than glomerella stem-rot. The disease characteristically shows black discolouration and necrosis of the vascular strands spreading outwards from wounded parts of the stem.

(12)

Glomerella stem-rot *(Glomerella cingulata)*. Pieces of stem showing eruptive blisters and groups of black perithecia.

Blisters are produced on the epidermis beneath which the internal infected tissues appear dark brown or black. These blisters rupture to reveal masses of black confluent pycnidia.

According to CMI the causal agent of the disease is *Botryodiplodia theobromae* Pat. In both host and artificial culture this organism produces black mycelium and pycnidia which are erumpent, confluent, stromatic, and ostiolate. The conidiophores are short and simple and produce dark conidia that are two-celled at maturity and slightly elongate. Infection is thought to occur through wounds and to be favoured by high relative humidites.

An unidentified stem-rot

A third stem-rot is caused by an unidentified basidiomycete. This disease, although relatively uncommon has been observed on old, mature and young stem pieces both in the field and in storage. Infected stem pieces are necrosed showing slight brown discolouration and at times a white mycelium can be observed growing beneath the epidermis. Under certain humid conditions small white cup-shaped basidiocarps arise from the epidermis of heavily infected cuttings (Fig. 13). The identification of this basidiomycete and the importance of all three stem-rots need to be investigated.

Other woody pathogens reported in the section on root-rots infect the stem bases of cassava plants and may also be involved in losses of stored stems.

The occurrence of these stem-rots is favoured by high relative humidity; infection probably occurs through wounds. Stem material intended for planting purposes should be handled with extreme care and carefully selected so that only cuttings with viable buds are used. The use of fungicides and surface sterilants to reduce the incidence of these diseases is being investigated.

C. Root-rot diseases

Root-rot diseases of cassava are important in areas with badly drained soils or during periods of excessive rainfall. Many of these pathogens induce damping-off during the early stages of plant growth and rot of the thickened roots during later stages. Although several root-rot diseases have been reported, few details are available and the symptoms described for each disease are similar. Generally, infection of young plants causes damping-off while infection of older tissues results in a partial or complete wilting and a soft or dry-rot of the thickened roots. Frequently, following infection by one or several pathogens, a broad spectrum of weak pathogens and/or saprophytes invade the diseased roots, masking the identity of the

(13)

Stem-rot (unidentified basidiomycete). Piece of stem showing typical basidiocarps, together with an uninfected control.

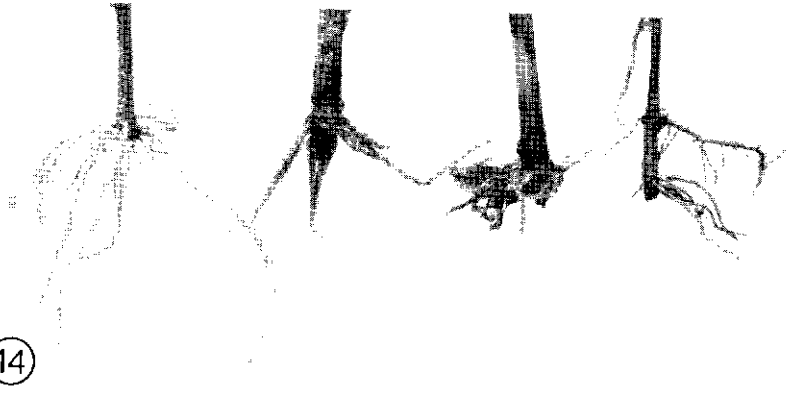
initial causal agent and causing all root-rots to appear similar. Several of these diseases caused by woody pathogens are more commonly found when cassava is planted following a woody crop, such as coffee, or immediately after forest clearance. Root-rots of the growing crop are caused by both fungi and bacteria. Several organisms are reported to cause post-harvest deterioration of cassava roots.

The control of these diseases is similar and is best achieved through such cultural practices as good drainage, selection of lighter soils and the avoidance of waterlogged areas, crop rotation, and early harvest. For damping-off diseases fungicides may aid establishment. In a few cases, resistant varieties have been reported (Drummond and Goncalves, 1946, 1957; Castaño, 1953; Fassi, 1957; Muller and Carneiro, 1970).

Phytophthora *root-rot*

This disease has been reported infecting cassava plantations in both Africa (Fassi, 1957) and tropical America (Vanderweyen, 1962; Muller and Carneiro, 1970) where it has caused yield losses of up to 80%. The pathogen attacks mature or young plants, frequently near drainage ditches, causing sudden wilting and a severe soft rot of the swollen roots. Initially infected young roots show spreading water-soaked patches which later turn brown (Fig. 14). Infected swollen roots frequently exude a pungent watery liquid and eventually decompose completely in the soil (Fig. 15).

Three *Phytophthora* spp. have been reported as inducing disease in cassava roots: *P. drechsleri* in Brazil (Muller and Carneiro, 1970) and Colombia (CIAT, 1972), and *P. erythroseptica* and *P. cryptogea* Path, in tropical Africa (Fassi, 1957; Vanderweyen, 1962). These fungi which also cause root-rots of several other plant species are well known.



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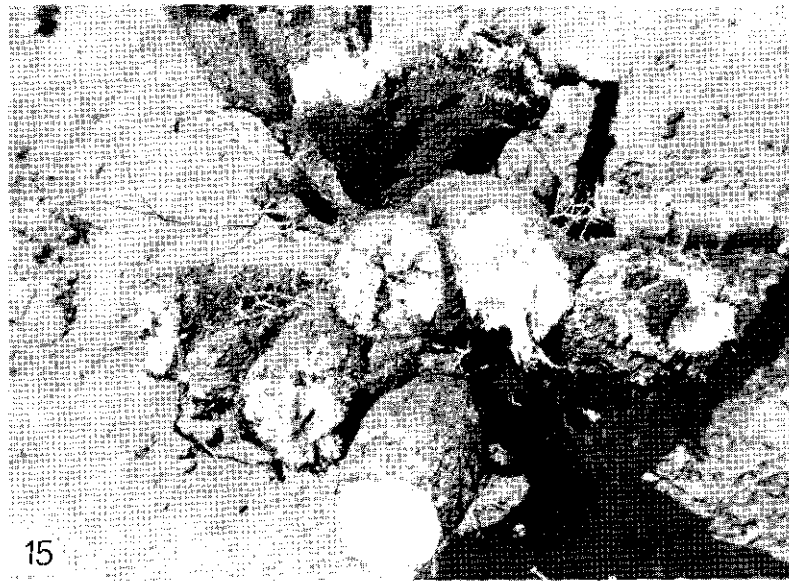
Phytophthora root-rot *(Phytophthora drechsleri).* Root-rot of young plants together with and uninoculated control.



Phytophthora root-rot *(Phytophthora drechsleri).* Typical root-rot of swollen roots.

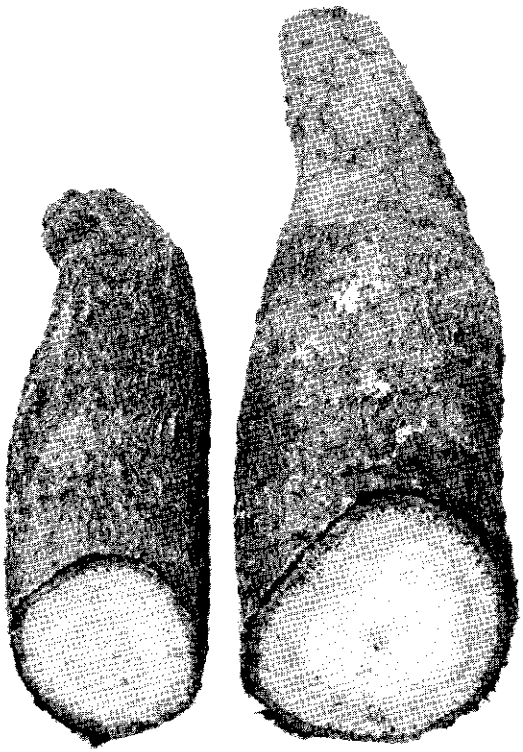
White thread disease

This is the most widespread and serious root disease of cassava in Africa where its appearance on swollen roots is sometimes taken as an indication of the maturity of the crop. Although this disease is known in Latin America it is not of major importance there at present. The disease is recognised by a white mycelial mat under the bark of swollen roots and by the presence of white cotton-like mycelial threads coating part or all of the exterior of infected roots up to the stem base. Internal infected tissues of swollen roots appear dry and have a characteristic rotting wood odour. Occasionally young plants are infected resulting in a sudden wilt and defoliation, all the roots being necrosed. The causal organism of the disease is *Fomes lignosus* (Klot.) Bres. (Vanderweyen, 1962; Affran, 1968; Doku, 1969; Jennings, 1970; IITA, 1972), a basidiomycete belonging to the *Polyporaceae.*

Rosellinia *root-rot*

This disease has been reported from many cassava regions with wet soils which are high in organic matter, and most frequently where cassava

is grown following a woody or forest crop (Drummond and Goncalves, 1946; Castaño, 1953; Viegas, 1955). The disease has also been named "black rot" on account of the characteristic black discolouration and cankers on portions of infected plants below the ground. In the early stage of infection white rhizomorphs that eventually turn black cover root surfaces. Internally, the infected tissues of swollen roots become slightly discoloured and exude a watery liquid when squeezed. Black mycelial strands penetrate into and grow throughout the infected tissues and small cavities containing whitish mycelium may be formed (Fig. 16). Infected swollen roots have a characteristic rotting wood odour. There are no reports that young plants are infected by this disease, but care should be taken to select planting material that does not come from infected plants.



(16)

Rosellinia root-rot (*Rosellinia necatrix*). Root-rot of swollen roots.

Rosellinia necatrix (Hartig). Berl. the perithecial stage of *Dematophora necatriz* is the causal agent (Castaño, 1953; Viegas, 1955). This fungus induces root-rots in other woody and herbaceous plants (Castaño, 1953; Viegas, 1955; Alexopoulus, 1962) and is adequately described in the literature. Little information is available, however, about the epidemiology of this fungus on cassava, the sexual stage is thought to occur rarely (Castaño, 1953; Alexopoulus, 1962).

Sclerotium *root-rot*

This root-rot is commonly observed on young cuttings, on more mature roots, and as a coating on swollen roots of cassava in Latin America (Viegas, 1943a, 1943b; Ferdinando *et al.*, 1968; Martin, 1970; CIAT, 1972). White mycelium radiates into the soil from infected roots or stem bases. This mycelium may on occasions penetrate the roots through wounds and cause rotting. While young plants are rarely killed by this disease considerable root necrosis may occur.

The disease is caused by *Sclerotium rolfsii* Sacc., a common but weak soil pathogen which has cottony-white mycelium and characteristically forms numerous rounded sclerotia, both on the host and in artificial culture.

Other root-rot diseases

Several other fungi may induce damping-off and root-rots of cassava, but little or no information is available regarding their occurrence or importance. *Armillariella mellea* Vahl. is reported associated with a stem-base and root-rot of mature plants (Vanderweyen, 1962; Arraudeau, 1967; CIAT, 1972). *Pheolus manihotis* (Heim, 1931), *Lasiodiplodia theobromae* Griff. et Mubl. (Vanderweyen, 1962), *Pythium* sp., *Fusarium* sp. (CIAT, 1972), *Clitocybe tabescens* (Arraudeau, 1967), *Sphaceloma manihoticola* B. et Jenkins (Bitancourt and Jenkins, 1950), *Rhizopues* spp. (Majumder *et al.*, 1956), *Rhizoctonia* sp. (Goncalves and Franco, 1941), and *Aspergillus* spp. (Clerk and Caurie, 1968) are all reported as causing rotting of cassava roots.

Species of *Bacillus, Erwinia,* and *Corynebacterium* have been reported as inducing soft-rots and/or fermentations in swollen roots (Collard, 1963, Akinrele, 1964; Averre, 1967). The symptoms of these soft-rots are similar and are frequently accompanied by fermentations. The bacteria are

thought to enter swollen roots through wounds induced by man during cultural operations, by animals or insects, or by fungi, and are frequently accompanied by many other saprophytic micro-organisms.

Pathogenic species of the genus *Bacillus* form spores in most media containing sugar. *Erwinia* spp. can be isolated and distinguished using the Kado and Heskett medium (1970); their pectinase activity as detected on sodium-polypectate medium, and their peritrichous flagella. *Corynebacterium* spp. can also be isolated and distinguished by the use of selective media (Kado and Heskett, 1970), pleomorphism of their cells, and their gram-positive reaction.

Cassava blight bacteria may also induce necrosis, discolouration, and dry-rot of the vascular tissues of swollen roots (Lozano, 1972a; Lozand and Sequeira, 1973b).

Core root-rot is a physiological disorder that causes damage to swollen roots in tropical Africa (Barat *et al.*, 1959; Averre, 1967). It also occurs in wet, badly drained soils where it takes the form of a dry internal necrosis, irregularly spreading out from the centre into the cortical tissues. This disorder is observed in only 10–20% of the roots of an infected plant, and only the larger thicker roots are thought to be susceptible.

While it is not fully understood whether the rapid deterioration of cassava roots that occurs after harvest is the result of physiological or pathological causes, or a combination of both, numerous micro-organisms have been isolated from deteriorated roots. Several of these are known to cause discolouration and rotting. The literature relevant to the post-harvest deterioration of cassava roots has been reviewed by Ingram and Humphries (1972). The important role of mechanical damage in deterioration and its possible control by wound healing and curing has been described by Booth (1972, 1973a, 1973b).

CONCLUSIONS

It can be seen that many diseases which in general are poorly understood, attack and reduce cassava yields. It is also well known that extensive losses occur following harvest of the roots. If full use is to be made of this important food crop it is necessary to increase our understanding and to intensify research into all aspects of cassava production and utilisation. The importance of reducing losses from fungal, bacterial, and viral pathogens cannot be over-emphasised. However, with the possible exception of cassava bacterial blight, little or no information is available on the means,

of controlling many cassava diseases. In several instances the existence of resistant cultivars has been noted but these have rarely been fully described or confirmed in controlled experiments. In some cases disease resistance may be found in agronomically acceptable cultivars, but in the other cases breeding will be required to transfer resistance into proven agronomic types. Thus, extensive research is required to evaluate host/pathogen reactions and determine the sources of resistance to the many diseases so that this information may be used by breeders and agronomists in selecting and breeding for improved cultivars. For those diseases to which sources of resistance cannot be found, other means of disease control, need to be sought.

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Resistance to the majority of the diseases common to cassava has been found, with great economic advantages. Resistance to cassava bacterial blight has been identified at CIAT, after evaluating 1,500 cultivars.