BACTERIAL BLIGHT OF CASSAVA IN CENTRAL AND SOUTH AMERICA: ETIOLOGY, EPIDEMIOLOGY AND CONTROL

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SUMMARY

Bacterial blight of cassava caused by a taxon of the genus *Xanthomonas* is a serious disease in Central and South America and has been observed in parts of Africa. Symptoms include leaf spotting, wilting, dieback, gum exudation on young shoots, and vascular discoloration in mature stems and roots of susceptible cultivars. Dispersal by rain splashing is the most important means of dissemination within localized areas. Dissemination from one area to another occurs through movement of infected planting material or by the use of contaminated tools. Delay in the spread of the disease has been obtained by pruning infected plants. The use of resistant cultivars and the production of certified bacteria free planting material, obtained from plants propagated from shoot tip cuttings, provides a satisfactory means of control.

RESUME

Le wilt bactérien du manioc provoqué par un taxon du gène *Xanthomonas* représente une maladie grave en Amérique Centrale et du Sud et a été observé en maints endroits de l'Afrique. Parmi les symptômes il y a la tache des feuilles, le flérissement, la fanaison, l'écoulement de la sève sur les jeunes pousses et la décoloration vasculaire des tiges adultes et des racines des cultivars sensibles. La diffusion par les gouttes d'eau de pluie est la voie la plus rapide de dissémination dans les zones affectées. La dissémination d'un endroit à un autre se fait par le déplacement de matériel végétal infecté ou à travers des outils déjà contaminés. Le retardement de la diffusion de cette maladie a été rendu possible en éliminant les plantes infectées. L'utilisation de cultivars résistants et la production de matériel végétal certifié indemne de bactérie obtenue à partir de plantes propagées issues de boutures d'extrémité des pousses constituent un moyen satisfaisant de lutte.

RESUMEN

El tizón bacterial de la yuca, causado por una especie del género *Xanthomonas*, es una enfermedad seria en Centro y Sud América y ha sido observado en algunas partes de Africa. Los síntomas incluyen el manchado de la hoja, marchitez, acronecrosis, exudación de goma en vástagos jóvenes y decoloración vascular de tallos maduros en los cultivares susceptibles. La dispersión por salpicaduras debidas a la lluvia es la forma principal de diseminación dentro de un área localizada. La diseminación de un área a otra ocurre por el movimiento de material infectado o por el empleo de herramientas contaminadas. Con la poda de las plantas infectadas se ha obtenido un retraso en la dispersión de la enfermedad. El uso de cultivares resistentes y la producción de material certificado de siembra libre de bacterias, obtenidos de plantas propagadas a partir de estacas terminales, provee un medio satisfactorio de control.

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INTRODUCTION

Cassava bacterial blight is the most important of several bacterial diseases of cassava reported[12,17,18,20]. This disease has caused severe losses in several Latin-American countries and Africa where epidemics have been recorded in several of the important cassava growing areas. The disease is now recognized as one of the most important factors limiting production in affected areas where, in wet seasons, it can result in complete loss of yield[12, 18, 19, 20].

The disease was first recorded in Brazil in 1912[5], but has since been reported in Colombia and Venezuela[18, 19, 20], Nigeria[17] Zaire (Cock, personal communication) and has been observed in several other countries of tropical America and Africa. It has only been recorded in species and cultivars of the genus *Manihot*.[1, 7, 8]

SYMPTOMS

Symptoms of the disease comprise angular leaf spotting and leaf blight, wilting, die-back, gum exudation, and necrosis in the vascular tissues of stems and roots. This complete syndrome is unique among diseases induced by a single plant pathogenic bacterium. Primary symptoms, following the planting of infected material, are wilting of the young germinated sprouts followed shortly by die-back. (Fig. 1) Secondary symptoms, following secondary infections, consist of angular leaf spotting followed by blight, defoliation, wilting and die-back. Leaf spots develop initially as water-soaked, angular areas, clearly visible on the abaxial surface of the leaves (Fig. 2). These spots become brown or dark-brown and sometimes, depending upon the susceptibility of the cultivar, a yellow halo surrounds the spots. Spots enlarge and coalesce, forming a large necrotic 'blighted' area. Necrosed areas spread throughout the entire leaf which, as a result, rolls and dries up. These blighted leaves remain attached to the stem for a short time before falling. (Fig. 3).

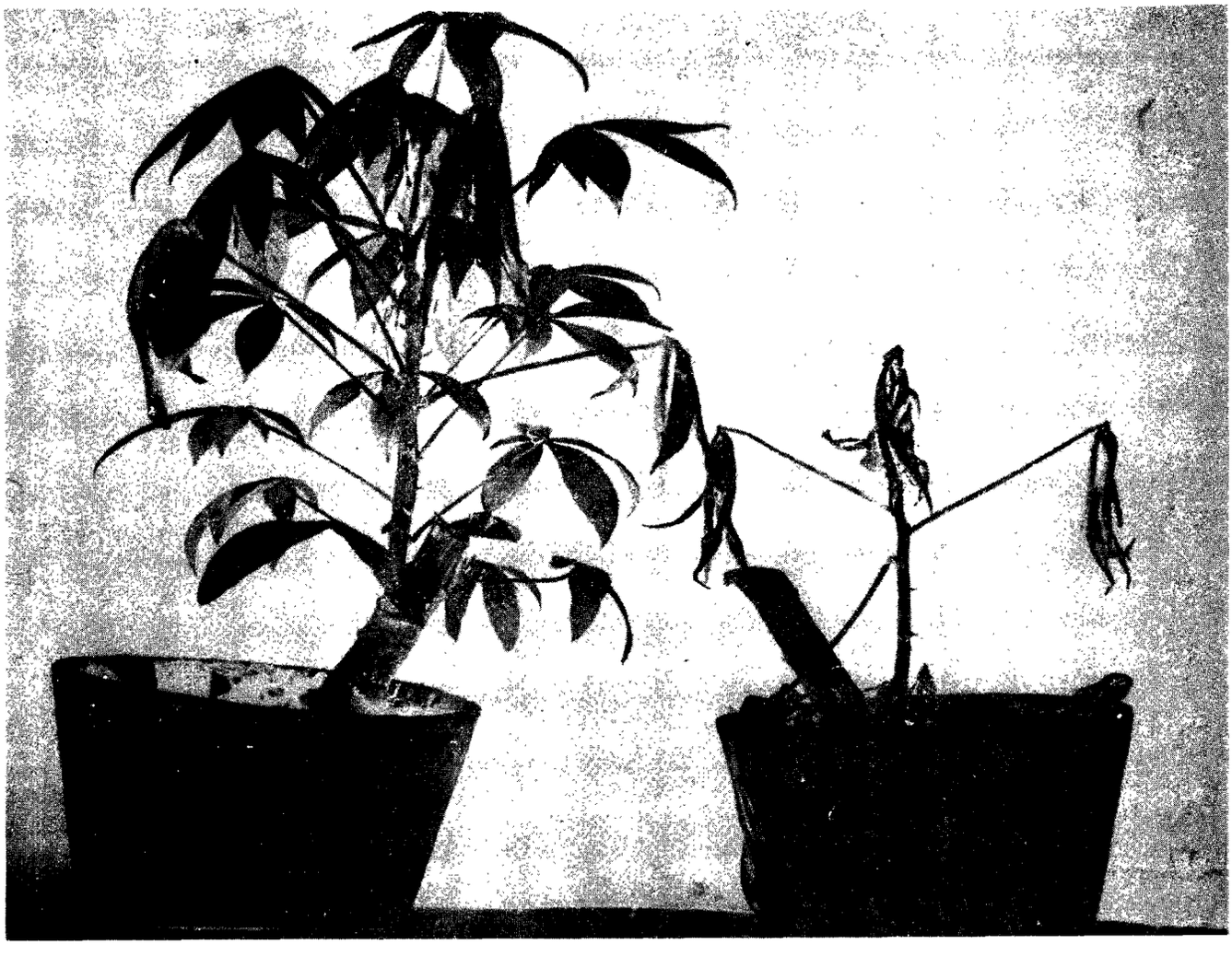


Figure 1. *Dissemination of CBB by infected vegetative "seed". Left: Healthy sprout from a healthy stem cutting. Right: Diseased sprout from an infected stem cutting (18).*

Figure 2. *Cassava leaf lobes showing typical leaf spots on abaxial side.*

Figure 3. *Infected cassava plant showing wilting, necrosed leaves still attached to stem. Note that young or emerging sprouts are not affected yet by the pathogen.*

Figure 4. *Sprouting cassava plants after the tops were heavily infected by CBB. Some of the new shoots that emerged remain apparently healthy, but others are already infected showing wilting and die-back.*

Leaf spots often exude a yellowish, sticky gum that collects in droplets, mostly on the lower leaf surface and along major and minor veins. Gum is usually also exuded from cracks which develop on young infected stems and petioles. This gum dries to form a yellowish glistening scab.

The vascular strands of infected petioles and stems appear as brown strings. Leaves fed by these necrosed vascular strands wilt, and young stem tissues rot, particularly in those parts of the shoot where the primary infection first occurred. Rotting is faster in young (green) than in mature (green-brown) stems, and old stem tissues remain apparently healthy. Rotting of young stem tissues results in the characteristic die-back symptom, which is therefore restricted to the immature stem portions of the plant. (Fig. 4)

Generally, roots of infected plants remain healthy. However, in some susceptible cultivars, swollen roots (tubers) may show dry-rotted spots around the necrosed vascular strands. This rotting is usually restricted to the vascular tissues; other tissues of the root remain apparently healthy.

When infections occur on young immature plants, the aerial portions may be completely destroyed. When this occurs the plants usually produce new shoots either from above or below ground portions of the stem. These young shoots are extremely susceptible and during rainy seasons rapidly become infected and so prolong the epidemic.

ETIOLOGY

The causal agent was first named *Bacillus manihotis* Arthaud-Berthet[6], but later renamed *Phytomonas manihotis* (Arthaud-Berthet and Bondar) Viegas[22]. Drummond and Hipolito[13], however, found that some of the characteristics of the bacterium they isolated from cassava were different from those originally described by Bondar[6]. The species was included under the name *Phytomonas manihotis* Burkh. in Bergey's Manual[4]. Comparative studies of a new isolate with the strains of Burkholder and of Drummond and Hipolito were made by Amaral and Vasconcellos[3]. They concluded that all three strains belonged to *Phytomonas manihotis*. Later, Starr[21] transferred the name to *Xanthomonas manihotis* (Arthaud-Berthet) Starr[5]. Resulting from studies on morphology, physiology, serology, and phage susceptibility of the bacterium as isolated in Colombia, Brazil and Venezuela, Lozano and Sequeira[10, 11, 18, 19] concluded that these were sufficiently different from *x. manihotis* to be considered as a distinctive strain. They reported[19] that the

cassava blight bacterium differed from typical *x. manihotis* in cell size, mitility and flagellation, production of H_2S, utilization of nitrate, hydrolysis of starch, and in several serological relationships. They also reported[19] that a comparison with a type culture of *x. manihotis* revealed differences in pathogenicity, growth rate, serological characteristics, and phage susceptibility.

Recently, comparative studies among different American and African isolates from blighted cassava have revealed that they possibly all belong to the same bacterial species although there are some differences in virulence and in a few physiological characteristics (Sequeira, personal communication; Ikotun, personal information).

Lozano and Sequeira[19] reported that the cassava blight bacterium (CBB) is as a Gram-negative slender rod, mobile by means of a single polar flagellum, not encapsulated, and non spore-forming. It is an aerobic, fast-growing bacterium which forms no pigment on sugar-containing media. It hydrolyzed starch and gelatin, and shows acid formation with litmus milk. It does not induce a hypersensitive reaction on tobacco leaves or cause soft-rotting of potato tubers or cassava roots. It produces catalase, arginine dehydrolase, and lipase, but does not produce H_2S, indole, urease, tyrosinase or phenylalanine deaminase. It is able to grow in ordinary media plus NaCl or tetrazolium chloride at maximum concentrations of 2.5 and 0.2 percent respectively. The bacterium uses nitrate and ammonium as sources of nitrogen; most simple sugars can serve as sources of carbon; various amino acids and other organic acids are readily utilized. It can be separated by serological and phage-typing methods from species of *Erwinia*, *Pseudomonas*, and *Xanthomonas*, including the type strain of *x. manihotis*. *Bdellovibrio* sp. causes lysis specifically of this bacterium and can be used to separate it from other plant pathogenic bacteria. As a result of this, Lozano and Sequeira[19] concluded that although the cassava blight bacterium could be considered as a strain of *x. manihotis* its taxonomy needed further revision.

EPIDEMIOLOGY

The bacterium normally penetrates the host via stomata or through epidermal wounds[18, 19]. After penetration, the organism first invades and destroys the spongy mesophyll and then enters the vascular tissues. Once inside the vascular system, the bacterial cells are able to move systematically throughout the plant[18, 19]. Movement into the stem and petioles is thought to take place primarily through the xylem vessels[1, 13] and possibly through the phloem[1, 20]. Movement through the pith tissues also has been reported.

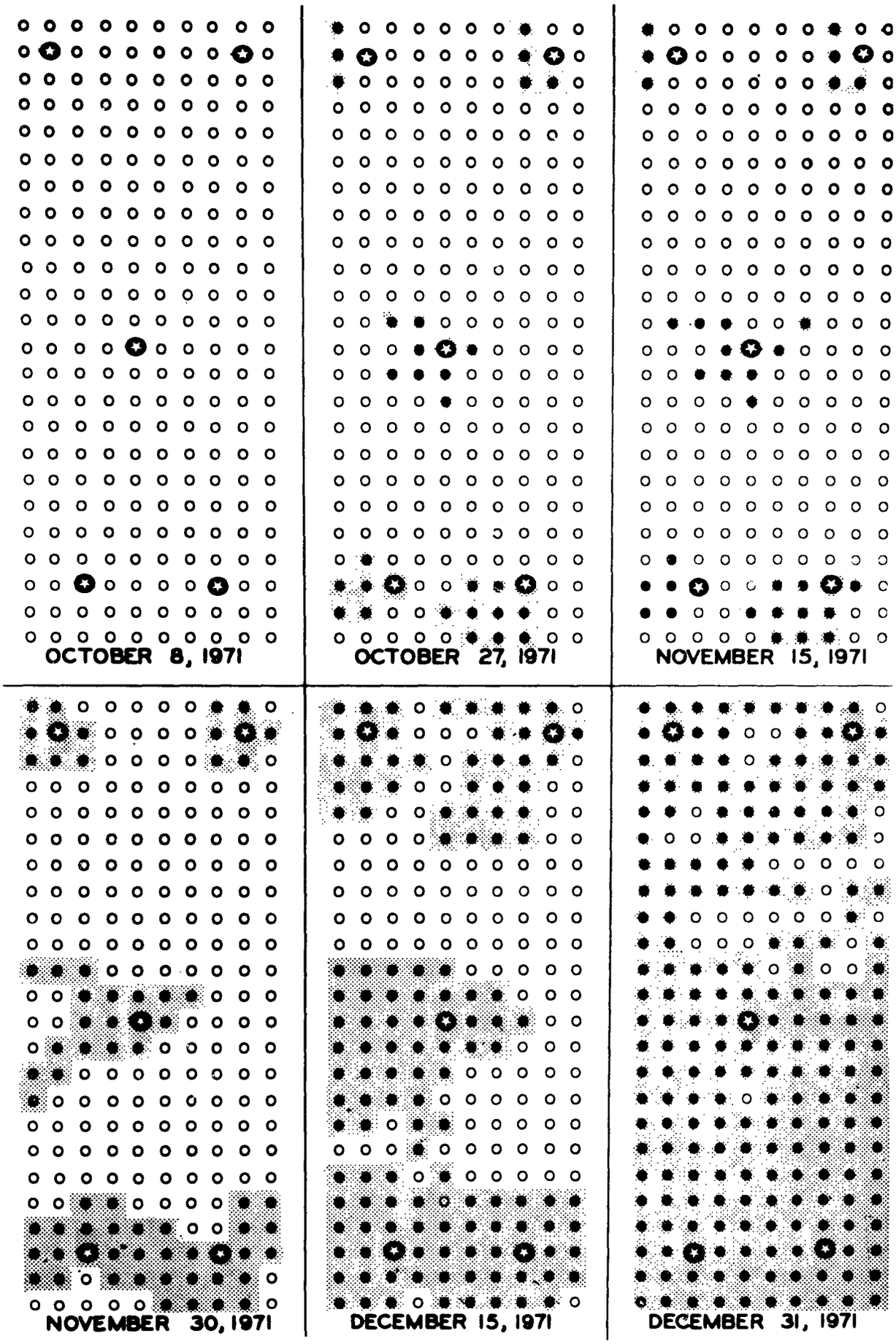
Infection by this organism is more common in, and is frequently limited to, the young tissues of the plant where it causes extensive breakdown of parenchymatous tissues of susceptible cultivars. In general, symptoms develop within eleven to thirteen days of infection[18, 19]. In highly lignified stems the bacteria remain restricted to the vascular tissues where they can survive for relatively long periods (Lozano, unpublished). It is through the lignified secondary wall, and possibly also the middle lamellae of mature vessels, forming a barrier, that the enzymes of this bacterium cannot penetrate[18, 19].

In inoculation experiments it has been found that at least 12 hours at 100 percent relative humidity is required for consistent bacterial establishment[18]. The influence of other environmental conditions on infection and disease development have not been reported.

The possibility that the pathogen spreads from one area to another by the use of infected cuttings was suggested by Amaral[2] and demonstrated by Lozano[18] and Lozano and Sequeira[19]. (Fig. 1). Lozano[18] and Lozano and Sequeira[19] also have clearly demonstrated that the use of infected cuttings is largely responsible for the dissemination of the disease from one growing season to another and that rain splashing is the most important means of dissemination in localized areas (Figs. 5,6). This accounts for the increased incidence of the disease in the rainy seasons reported by Drummond and Hipolito[13].

Some workers have suggested that the pathogen could be readily spread by movement of soil during cultural operations or by the use of contaminated tools during pruning[9,13,14,15,16]. Although it is considered possible that bacteria may be able to penetrate roots when plants are grown in heavily infested soils[3,13,19], this means of disease spread is considered to be of minor importance because of the short survival of this pathogen in the soil (Lozano, unpublished). Similarly, contaminated irrigation water is at present regarded as being of minor importance. In contrast to this, however, the use of contaminated tools is considered to be an important means of bacterial dissemination[18,19], especially considering the extensive amount of cutting that is required during harvesting and preparing planting material.

As the bacteria enter plants through wounds, the movement of man, animal, and insects through a crop is also likely to spread the disease although little evidence is available to demonstrate this. Insects have been suggested as possible agents for dissemination[2] and their possible role in disseminating bacteria has recently been demonstrated at CIAT. Controlled experiments, using insecticides, have shown that dissemination because of insects could account for as much as 10 percent of the total dispersion from primary foci within a plot (Ikotun, personal information).[1] However, studies on dissemination from an inoculum source to plants located at different distances from it indicated that spread attributable to insects only occurred over short distances[11,18,19].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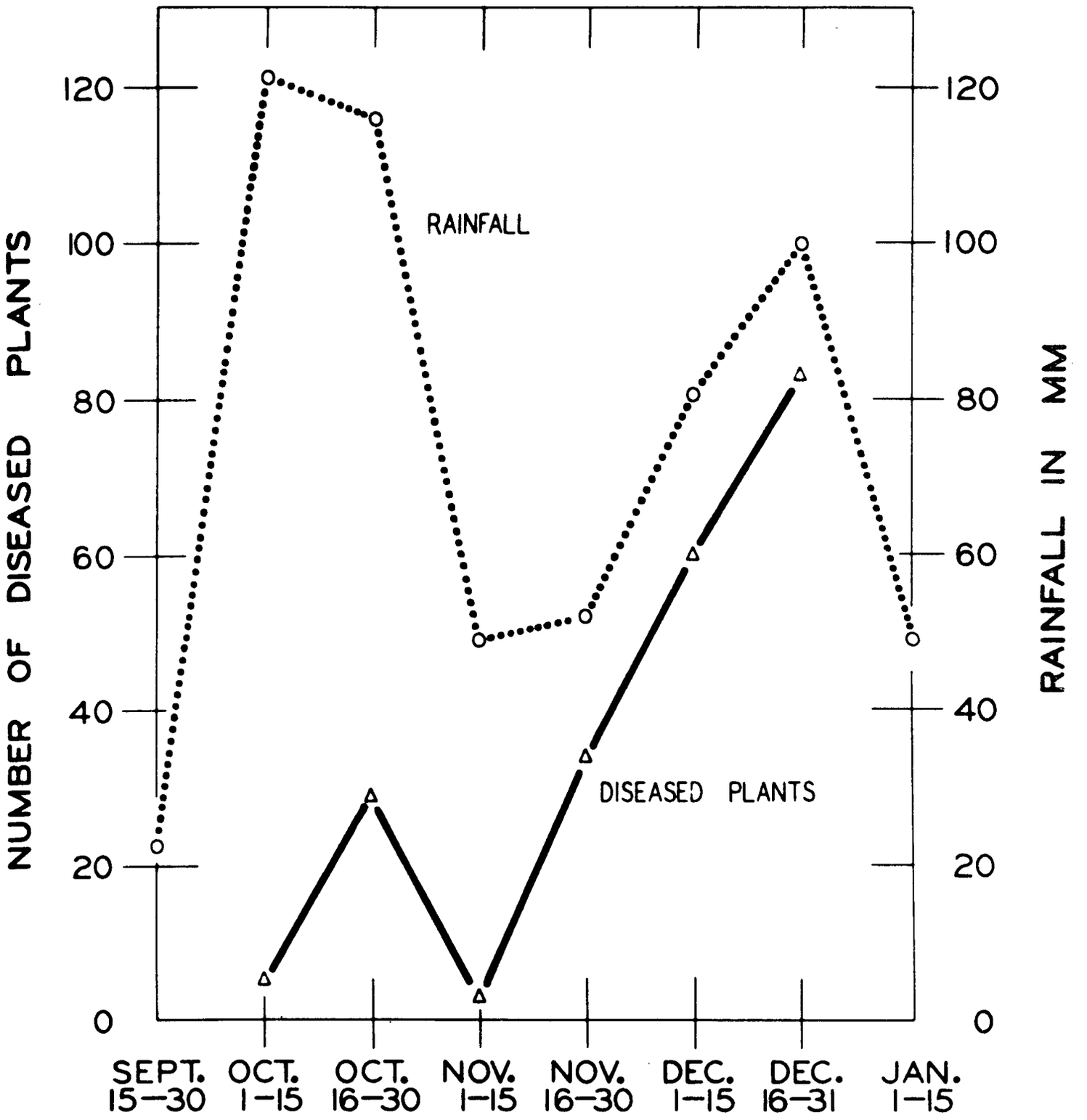

FIGURE 6. SPREAD OF CBB IN THE FIELD FROM INITIAL SOURCES OF INFECTION. RELATION OF TOTAL RAINFALL (MM) AND NUMBER OF DISEASED PLANTS IN EACH 15-DAY PERIOD (20).

During dry periods when disease development is slow little bacteria-containing gum is exuded, and hence the spread of the disease is halted. The bacteria, however, remain viable in the plant and become active during subsequent rainy periods.

CONTROL

Delaying the spread of the disease by pruning most of the above ground portions of infected plants has been reported[11,18,19]. However, the success of this method depends on the susceptibility of the cultivar and the interval between initial infection and pruning. This method is most successful with mildly infected resistant and moderately resistant cultivars, and has little effect with severely infected susceptible cultivars as the new shoots rapidly become re-infected and so require regular and extensive pruning. Such extensive pruning must affect the productivity of the plant and quality as well as yield of roots. Although in certain circumstances this method may be useful to slow or break the spread of an epidemic, it can never give complete control and is incompatible with normal production of the crop.

For the complete avoidance of this disease where it does not yet occur, exclusion of the pathogen by the use of clean propagating material has been suggested[9,13,15,16]. A successful means of producing bacteria-free cuttings has been developed by Wholey and myself at CIAT. We are able to root bacteria-free stem tips even from infected plants and can thus obtain clean stocks from infected cultivars and have provided certified bacteria-free cassava propagating material. (Fig. 7) Physical treatments, such as exposure to hot air and water, microwaves and ultraviolet light to inactivate bacteria in infected planting material have so far been unsuccessful. (Prada, Zarate, and Lozano unpublished).

Crop rotation has also been suggested as a means of control[9]. At CIAT we have found that if all infected plant debris is removed and destroyed by burning, an interval of six months between successive cassava crops is sufficient to prevent carry-over of the disease in the soil.

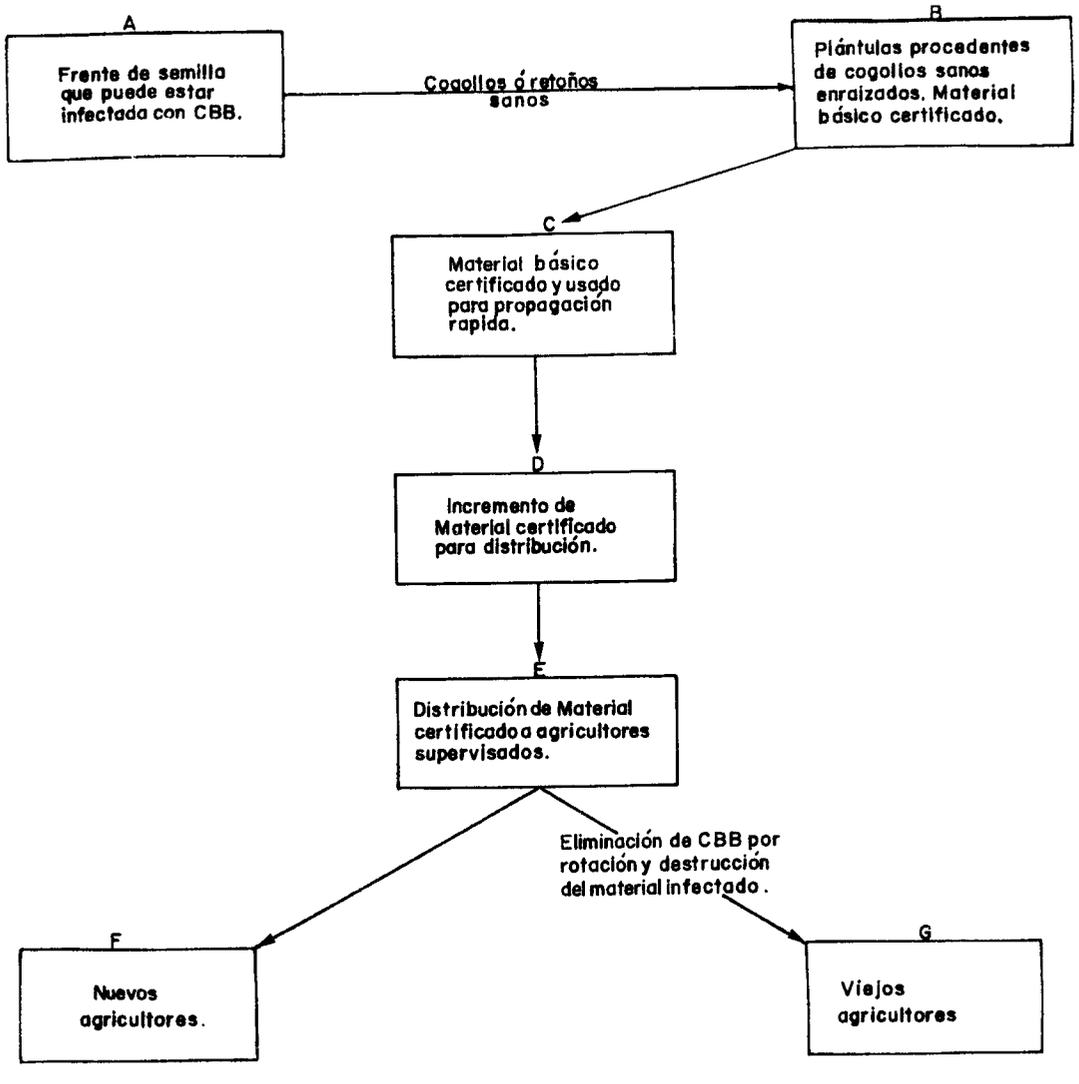

FIGURE 7. SUGGESTED SCHEME FOR A CBB-FREE "SEED" CERTIFICATION PROGRAM (LOZANA AND WHOLEY, UNPUBLISHED).

Control by the use of cultivars resistant to bacteria was first suggested by Goncalves[15]. Numerous field resistant cultivars have since been reported[9,14,20]. These field observations have been confirmed in greenhouse studies conducted by Lozano and Sequeira[19]. Their studies also revealed that three possible types of resistance exist in different cultivars; one type apparently limits penetration, another type limits systematic invasion and establishment, and the third type is apparently based on a hypersensitive response of the host[19].

A combination of the use of rotation, resistant cultivars and the use of bacteria-free planting material appears to be a most promising means of controlling this important disease.

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