9840

MICROFILMADO

CIAT
CENTRO DE DOCUMENTACION

Superelongation: a *Sphaceloma* disease of cassava*

J.P. Krausz
J.C. Lozano
H.D. Thurston**

Abstract

A new and serious disease of cassava was first reported to be causing serious epidemics in Colombia in 1972 and has since been found in Central America, Brazil and Venezuela. The causal organism was identified as a fungus of the species *Sphaceloma*, probably *S. manihoticola*. The disease results in extensive elongation of the internodes of infected plants and causes serious yield loss. Artificial inoculation of young cassava plants was achieved in a mist chamber, and free water was found to be necessary for conidial germination. Pathogen dissemination appears to be associated with wind-blown spores and, more extensively, with infected stem cuttings used for propagation. The pathogen also attacks *Manihot glaziovii*. Resistant germplasm within *M. esculenta* has been identified.

A new disease of cassava was reported by Lozano in 1972 (2) and by Lozano and Booth in 1973 (4) to be causing epidemics in various parts of Colombia. It was named the superelongation disease (superalargamiento in Spanish) of cassava because it was characterized by an extensive elongation of the internodes of infected plants. In various parts of Colombia, farmers have had to abandon cassava crops because of the disease. A fungus consistently isolated from infected plant material was proven to be the causal agent, but its identification was uncertain. The following investigation was undertaken to learn more about the etiology and epidemiology of the disease.

Materials and methods

The pathogen was isolated from sporulating cankers by macerating them in sterile water and streaking the resulting suspension onto acidified water agar. The cultures were incubated at room temperature until germinating conidia could be isolated and removed aseptically to petri dishes containing potato dextrose agar (PDA). Cultures were maintained in petri dishes at room temperature and transferred periodically at two-week intervals. Isolations were made from cassava

* Based on a portion of the doctoral thesis of the senior author conducted at CIAT, Cali, Colombia

** First and third authors are former Graduate Research Assistant and Professor, respectively, Dept. of Plant Pathology, Cornell University, Ithaca, NY 14853; second author is Plant Pathologist, Cassava Program, CIAT, Cali, Colombia.

and from *Manihot glaziovii* material from widely scattered areas of Colombia and Costa Rica.

Conidial-mycelial suspensions containing 1 x 10^6 conidia/ml were prepared by homogenizing entire fungal cultures with distilled water in a Waring blender. The suspensions were sprayed upon young cassava plants with a piston-action hand atomizer. Inoculated plants were incubated in a mist chamber for periods ranging from 8 to 24 h at a temperature range of 24 to 29°C.

To test the effect of moisture on conidial germination, drops of a conidial suspension (5.3 x 10^6 conidia/ml) were placed onto three microscope slides. Two of these slides were air dried, and then one of these was remoistened with a drop of sterile distilled water. All three slides were placed into a moist chamber at 100% RH for 19 h at 25°C. The percentage of conidial germination was recorded for three replicates of this test.

The identification of the causal agent was based upon observation of symptoms on infected cassava, colony characteristics on artificial media, and morphology of conidia and conidiophores. Infected cassava plants were observed at various stages of disease development in naturally infested fields and in artificially inoculated plants. Fifteen isolates of the pathogen obtained from different regions of Colombia and one from Costa Rica, were grown and observed on PDA containing 0.8% yeast extract. Riddell mounts (7) were prepared to observe single conidiophores and to determine the manner of conidial production.

The critical temperatures for the pathogen's growth were determined by transferring small pieces of fungal tissue (2 mm diameter) to PDA and placing the cultures in incubation chambers at 6, 9, 15, 18, 21, 24, 27, 30, 33 and 36°C (±0.5°C) for 30 days. Colony growth was determined by averaging the two largest perpendicular diameters of each colony, and two or more colonies were measured at each temperature.

To determine the mode of ingress of the pathogen into host tissue, drops of a conidial suspension were placed onto cassava leaves in petri dish moist chambers and were incubated at room temperature. After periods ranging from 12 to 60 h, inoculated leaf samples were removed and treated with the staining method reported by Latch and Hanson (3).

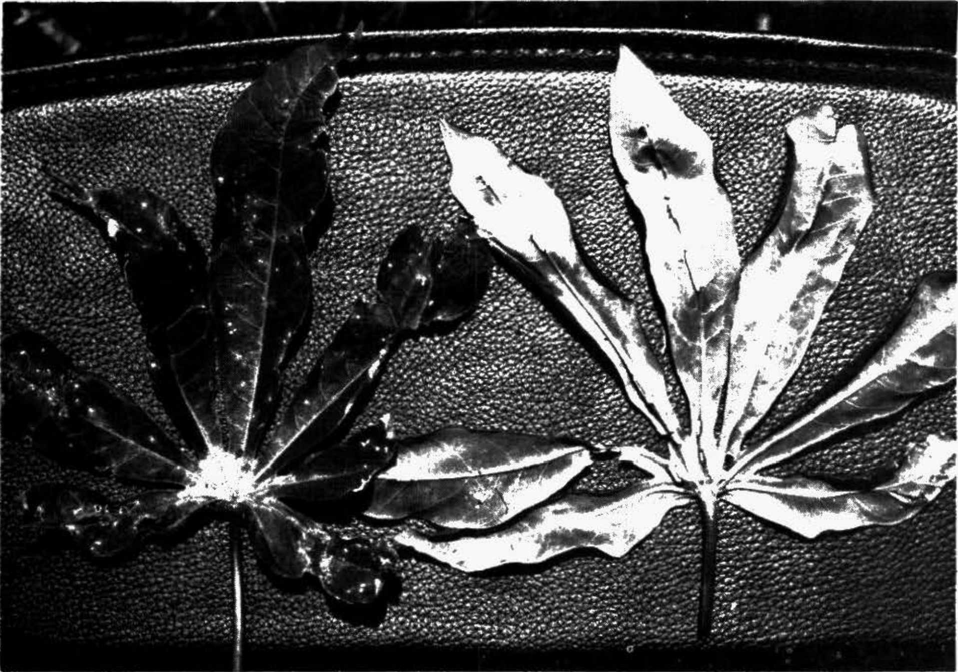
Observations on the dissemination of the pathogen were made at various stages during a naturally occurring epiphytotic. Sources of primary inoculum were identified and the pattern of disease spread from these sources was observed. Observations were also made on the spread of the disease from an artificially inoculated plot of cassava to an adjacent uninoculated plot 30 m distant.

Results

The disease occurs primarily on the younger portions of the plant. It is characterized by an exaggerated elongation of the internodes of young stems, by distortion and leaf curl of young leaves, and by canker formation on infected stems, petioles, and leaf midribs and main veins (Fig. 1a-b). The initial symptoms appear as small, puckered, slightly chlorotic spots on the laminae of young, fully expanded leaves. These spots soon become necrotic and take on a khaki brown to ash white color. Often a narrow, slightly chlorotic halo surrounds the older spots. As the spots age, they expand slightly (1-3 mm in diameter) and sometimes form a distinct, narrow dark brown margin. With fully advanced spots, the centers become thin and lighter in color; they often disintegrate, giving the leaf a shot-hole appearance (Fig. 1c). During severe infection, the leaves may become completely deformed and necrotic, resulting in premature leaf drop (Fig. 1b).

The inoculation method using a mist chamber proved satisfactory for obtaining uniform infection. Very little infection occurred after 8 h of incubation in the chamber, and severity of infection increased progressively up to 24 h of mist. Disease symptoms appeared 5 to 6 days after inoculation under these conditions. The pathogen obtained ingress by direct penetration as observed on the stained leaf sections.

Free water was necessary for conidial germination. Virtually no germination occurred at 100% RH unless free water was present. The optimum temperature for rapidity and percentage of conidial germination over a 24-h period was 28.5°C.



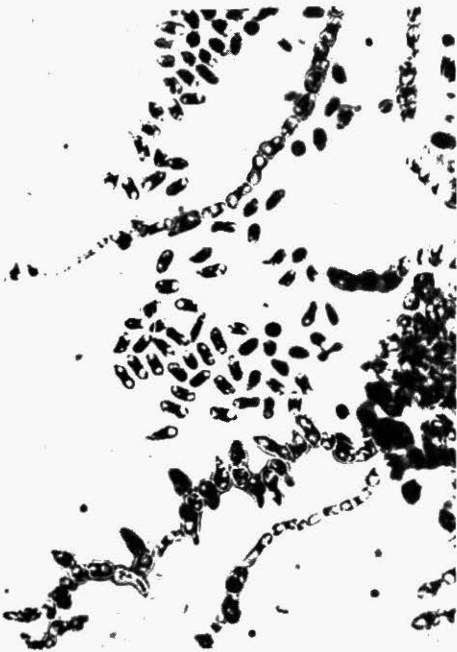


Figure 1. **(a) Leaf symptoms of the superelongation disease on cassava showing leaf spots, cankers on the midribs and main veins, and ragged appearance of entire leaf. (b) Terminal portion of infected plant showing characteristic elongation of internodes, leaf deformation and premature leaf drop. (c) Conidia and hyphae of the superelongation pathogen (x 1400).**

Colonies grown from spores streaked onto agar media consist of small, slow-growing, raised, yeastlike colonies with deeply convoluted surfaces. The colony consists of pseudoparenchyma, often with a gummy or tenacious consistency. On PDA the colonies are usually various shades of light orange, but the color can range from a light orange › a light brown.

The conidia from PDA cultures are unicellular and small, 3.3. to 6.8 x 1.8 to 4.9 µ, with average dimensions of 5.3 x 2.7 µ. They are generally ovoid to oblong-elliptical, hyaline and refringent under the light microscope. When stained, the conidia often show two polar guttules (Fig. 1c). Conidia germinate by budding or by germ tubes and usually become greatly swollen before germination.

From the Riddell mounts of the fungus, it was observed that the conidiophores are short, usually unicellular, more or less cylindrical structures, tapering slightly at the apex. Numerous conidiophores are borne upon well-developed hyaline to yellowish pseudoparenchyma-like stromata. The conidiophores are phialides, producing phialospores (conidia) acrogenously.

Optimum temperature for fungal growth in vitro was 24°C, with minimum and maximum temperatures at 6 and 36°C, respectively.

The disease appears to be disseminated over long distances by means of infected cassava stem cuttings. In the very early stages of an epiphytotic, four or five infected stem cuttings were determined to be the sources of inoculum. The spread of the disease from these sources of inoculum did not show a plant-to-adjacent-plant pattern but showed initial stages of infection occurring throughout the field. This pattern of dissemination was observed throughout the course of two epiphytotics in widely separated areas of Colombia. These observations suggest the probable involvement of wind-blown spores in the dissemination of the pathogen over relatively short distances.

Discussion

The first definite report of superelongation disease was made by Lozano in 1972 (2). Since then, the disease has been recognized as causing serious epiphytotics in numerous areas of Colombia, Central America, Venezuela and Brazil. Preliminary yield trials have demonstrated losses up to 80 percent due to the disease (6), and in a number of cases farmers have completely abandoned their cassava crops due to total loss.

The pathogen is of the genus *Sphaceloma*. In 1950 Bitancourt and Jenkins (1) named a new species of fungus *Sphaceloma manihoticola*, found attacking *M. esculenta* in the Dominican Republic and Guatemala and *M. glaziovii* in Brazil and Nicaragua. Their description of the fungus and its symptoms on its hosts is superficial, but there appear to be many similarities in symptomology with the superelongation disease. It seems reasonable that the superelongation pathogen is actually the same fungus reported by Jenkins and Bitancourt as *S. manihoticola*. Confirmation of this hypothesis, however, is almost impossible because Bitancourt and Jenkins failed to find and describe spores for taxonomic purposes and made no mention of the elongation of internodes in their discussion of symptoms. Nevertheless, since they primarily used dried plant material sent to them by others to describe the new species, it is not unreasonable to suspect that they never had an opportunity to observe the possible elongated appearance of the infected cassava plants. Also, the inability to find spores on dried plant material supports experiences encountered in the present study.

Considering the above, it is recommended that the same *Sphaceloma manihoticola* be used for the superelongation pathogen until further evidence be found that would indicate clearly that, in fact, more than one species of *Sphaceloma* infects *M. esculenta* and *M. glaziovii* naturally.

A number of good sources of resistance to the disease have been found, some of which are agronomically acceptable cultivars. Only uninfected cassava cuttings should be used in planting. If the disease does become established, burning of infected plant debris is recommended.

Literature cited

1. BITANCOURT, A.A. and JENKINS, A.E. 1960. Estudos sobre as Miriangiales. II. Vinte novas especies de Elsinoaceas neotropicais. Arquivos do Instituto Biológico, São Paulo 20:1-28.

2. CENTRO INTERNACIONAL DE AGRICULTURA TROPICAL. 1973. Annual Report 1972. Cali, Colombia. 192p.

3. LATCH, G.C.M. and HANSON, R.W. 1962. Comparisons of three stem diseases of *Melilotus* and their causal agents. Phytopathology 52:303-315.

4. LOZANO, J.C. and BOOTH, R.H. 1973. The superelongation disease of cassava. CIAT, Cali, Colombia. 29p.

5. ——— and BOOTH, R.H. 1974. Diseases of cassava (*Manihot esculenta* Crantz). PANS 20:30-54.

6. KRAUSZ, J.P. 1976. The superelongation disease of cassava. Ph.D. Thesis. Cornell University, Ithaca, N.Y. 81p.

7. RIDDELL, R.W. 1950. Permanent stained mycological preparations obtained by slide culture. Mycologia 42:256-270.