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Investigation of Inoculum Threshold and Latent Infection in *Colletotrichum gloeosporiodes* f.sp. *manihotis*, in Cassava Cultivars

C.N Fokunang[1], T Ikotun[1], C.N.Akem[2], A.G.O Dixon[2], E.A.Tembe[3], P. Koona[2]
[1]Department of Crop Protection and Environmental Biology, University of Ibadan, Oyo State, Nigeria
[2]International Institute of Tropical Agriculture, PMB 5320, Oyo Road, Ibadan, Nigeria
[3]Clinical Pharmacology Group, University of Southampton, Biomedical Sciences Building, Bassett Crescent East

Abstract: Studies were conducted at the Advance Pathology laboratory and glasshouse at the International Institute of Tropical Agriculture (IITA), Ibadan, Nigeria, to determine the inoculum threshold of fungal suspensions at different concentration levels, and also to investigate latent infection of cassava anthracnose disease pathogen, *(Colletotrichum gloeosporioides f. sp. manihotis)*, in cassava cultivars. This study showed that fungal suspension could initiate disease infection at very low inoculum concentration of 3.3×10^2 colony forming units (CFU)/ml of sterile distilled water. There was a general increase in percentage leaf infection and defoliation in the cassava cultivars with increase in inoculum concentration and incubation period. Symptomless cassava plant materials after incubation at 25 ± 2°C for seven days showed the presence of acervuli of anthracnose fungus in more than 80% of the cassava cultivars. This was an indication that symptomless cassava materials contain *C. gloeosporiodes* f. sp. *manihotis* that can only manifest itself under favourable environmental conditions during the course of growth and development of the host plant.

Key Words: Cassava, inoculum threshold, latent infection, *Colletotrichum gloeosporioides*

Introduction

Cassava, *Manihot esculenta* Crantz (Euphorbiaceae) is a starchy root crop which is among the most important tropical food crops (Cock, 1985). It is grown exclusively for food in 39 African countries, stretching over a wide belt from Madagascar in the Southeast to Senegal in the Northwest (Hahn and Keyser, 1985). Cassava cultivation is high where the annual rainfall exceeds 600mm over a period of 90-120 days, and the altitude ranges from sea level to 2000m. Cassava storage roots form the basic carbohydrate element of the diet and leaves are eaten as a preferred green vegetable in many parts of Africa, providing proteins, mineral and vitamins (IITA, 1990; Jalloh and Dahniya, 1994).

Cassava anthracnose disease (CAD) caused by *C. gloeosporioides f. sp. manihotis*, is an epidemic disease characterized by particular symptoms (cankers on stems, branches and fruits, leaf spots and shoot die-back) on aerial parts of the diseased plants (Muimba, 1982; Theberge, 1985; IITA, 1990). The appearance of the disease depends on the plant cultivar and the infected plant parts. In older stems CAD infection usually occurs as round and stringy lesions, which develops into deep cankers. Stem deformation occurs in some cassava cultivars, causing the stems to brittle and easy to break by wind action in the field (Ikotun and Hahn, 1992. CAD severity could lead to a significant loss in planting materials. In most cases severely infected stems and seeds result in a 20-45% decrease in germination (IITA, 1987).

Verhoef, (1974) studying the problems of latent infection by fungi defined it as the period between the landing of spores and the occurrence of infection. This phenomenon which has also been described as quiescent infection (Swinburne, 1986; George *et al.*, 1992), involves inhibition of development of the pathogen through physiological conditions imposed by the host until some stage of maturity has been accomplished. Inspite of many reports on latent infection of *C. gloeosporioides* on other crops, there is an information gap on the latent phase of *C. gloeosporioides* f.sp. *manihotis* in cassava cultivars. The existence of *Colletotrichum* infection on a large proportion of symptomless cassava plant is strongly suggested (IITA, 1987). Observations in the greenhouse of CAD infection on healthy cassava cuttings planted in a vector free environment suggest the presence of the fungus in a quiescent phase on plant. There is therefore the need to assess the latency of CAD on healthy plants and quantify inoculum that can initiate disease development. This investigation could help in developing proper sanitation measures in areas where there are possible source of inoculum.

This study was conducted to investigate the inoculum threshold and latent infection of *C. gloeosprioides* f. sp *manihotis*, contributing to cassava anthracnose disease development.

Materials and Methods

Experimental Site: The *in vitro* study on latent infection was conducted at the Advance Pathology laboratory of the Tuber and Root Crop Improvement Program (TRIP), while the inoculum threshold studies conducted at the greenhouse at the International Institute of Tropical Agriculture (IITA), Ibadan, Nigeria.

Determination of inoculum threshold of *C. gloeosporioides* f. sp. manihotis in cassava cultivars: Stem cuttings (20cm long) of three cassava cultivars (TMS 30572, TME1 and TMS 30211) were planted in plastic pots (10.5cm diameter) filled with steam-sterilized soils. Spore suspension were prepared from 7 day-old axenic culture of *C. gloeosporioides* f. sp. *manihotis*, and adjusted with the aid of a haemocytometer to concentrations of: 3.2×10^2; 2.5×10^3; 4.0×10^4; 1.5×10^5; 2.4×10^6 spores/ml of sterile distilled water. In each spore suspension solution, a drop of tween-80 was added as a surface-wetting agent. The potted cassava plants at one-month-old were sprayed till run-off with the different concentrations of the spore suspensions. The plants after spraying with fungal suspensions were covered with moist plastic bags to increase relative humidity favourable for the pathogen invasion. Temperature and relative humidity were monitored with a recording hygrothermograph. The plants were maintained for 72h in a controlled environment chamber at temperature of 26-30°C and 80-98% relative humidity. The experimental design was a randomized complete block design, in split plot arrangement. The cassava cultivars represented the main plots and the inoculum concentration treatments as the subplots. The trial was replicated four replication times, with the control treatment sprayed with sterile distilled water. Data was collected for leaf defoliation (fallen infected leaves), leaf infection (leaves showing disease symptoms after spray) and wilting or death of infected plants at 5 days interval after inoculation, for thirty days.

Investigation of latent infection by *C. gloeosporioides* f. sp. *manihotis*, on symptomless cassava materials: Tender symptomless cassava stems cuttings and leaves of twenty-five cassava cultivars from IITA germplasm collection were harvested in the field at three months after planting. The materials were washed in running tapwater, surfaced-sterilized for 3-5 minutes in 10% sodium hypochlorite, rinsed in five successive changes of sterile distilled water. Five stem segments or leaves per clone was incubated at 24-30°C and 68-85% relative humidity, in large sterilized plastic Petri dishes (15cm diameter), containing two layers of moist sterilized Whatman No 2 filter papers. A randomized complete block design arrangement was used and replicated four times. Observation for fresh tissue collapse was recorded daily, and the tissues were considered collapsed when

the initial natural colours were lost (change from green to brown). Collapsed tissues were observed with the aid of a stereo-binocular microscope for the presence of acervuli of *C. gloeosporioides* f. sp. *manihotis*. The acervuli number (pinkish black or brown conidia mass), was recorded, and the log transformation of the data was used for statistical analysis.

Statistical analysis: The data for defoliation, wilt or death of infected plant, leaf infection and the log transformation of acervuli number were subjected to analysis of variance (SAS Institute. 1989). Fischer protected LSD test of mean separation was done in cases where the ANOVA showed significance.

Results

Effect of inoculum concentration of *C. gloeosporioides* f. sp. *manihotis* on cassava cultivars: There was a general increase in percentage defoliation with increase in inoculum concentration (Fig 1). There was a maximum percentage defoliation of above 80%

Table 1: Cassava cultivar reaction to *Colletotrichum gloeosporioides* f. sp. *manihotis* inoculum.

Cassava	CAD symptoms parameters at indicated days of inoculation			
Cultivar	Defoliation		Leaf infection	
	20 DAI	25 DAI	20 DAI	25 DAI
TME1	47.00a	56.58a	54.54a	64.21a
88/02549	44.88a	54.38a	49.13	55.75b
30572	33.08b	44.54b	43.92c	49.21c
Mean	41.65	51.83	49.19	56.39
CV	19.59	19.10	17.56	16.98

CAD = Cassava anthracnose disease; DAI = Days after inoculation. Means followed by the same letter in the same column are not significantly different (p 0.05) by Duncan Multiple Range Test.

Table 2: Overall effect of inoculum concentration of *C. Gloeosporiodes* f. sp. *manihotis* in cassava cultivar 30572, 88/02549 and TME1.

Inoculum	Percentage Defoliation		Percentage leaf infection	
concentration	20 DAI	25 DAI	20 DAI	25 DAI
2.4×10^6	57.17a	68.08a	67.67a	73.67a
1.3×10^5	56.50a	71.67a	66.17a	76.08a
4.0×10^4	49.50c	59.83b	58.83b	64.83b
2.5×10^3	40.50c	50.17c	50.08c	57.92b
3.2×10^2	33.69d	40.33d	42.92d	48.75c
Control	10.27e	20.92e	5.27e	17.08d
Mean	51.83	51.83	48.49	56.39
CV	19.59	19.10	17.56	6.98

DAI = Days after inoculation.
Means in the same column followed by the same letter is not significantly different (P 0.05) by Duncan Multiple Range Test.

in cassava cultivar TME1, and 88/02549 at 25 days inoculation period. At five days inoculation period there was no significant defoliation at all levels of inoculum concentration. Defoliation of 20-25% was recorded with inoculum concentration of 2.4×10^6 spores/ml of distilled water in cultivar TME 1.

There was a general increase in percentage leaf infection in all the cassava cultivars with increase in inoculum concentration, and increased days of inoculation (Fig 2). At 25 days after inoculation (DAI) there was also a significant wilt in all the cassava cultivars. Inoculum concentration of 2.4×10^6 spores/ ml of sterile distilled water recorded leaf infection of about 85% in cassava cultivar TME1, followed by cultivar 88/02549. At 5-10 days after inoculation leaf infection was least recorded in cultivar 30572.

Cultivar reaction and inoculum concentration interaction to anthracnose disease symptoms expression: Cassava cultivars showed differential reaction to CAD symptoms expression at different inoculation periods. Cultivar TME 1 showed high level of sensitivity to defoliation and leaf infection than the other cultivars (Table 1). High defoliation of 56.58% was recorded in cultivar

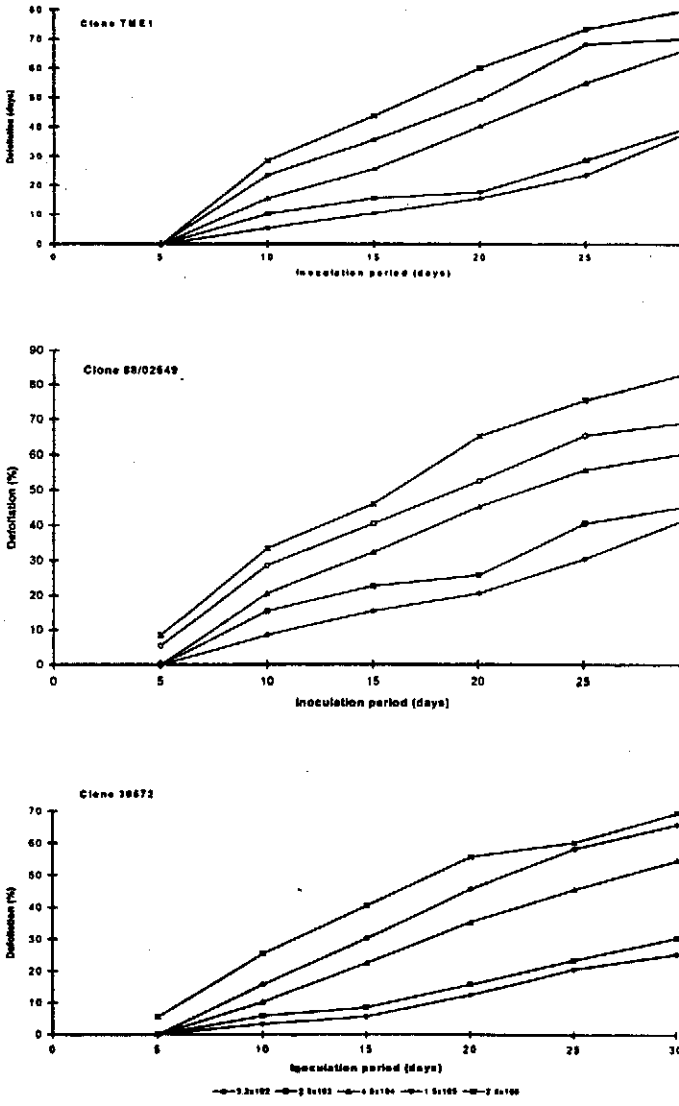

Fig. 1: Effect of inoculum concentration of *Colletotrichum gloeosporioides* f.sp *manihotis* on defoliation of cassava cultivars

TME 1 at 25 DAI and 64.21% for leaf infection at 25 DAI. Cultivar 30572 was less sensitive to disease symptoms with minimum mean value of 44.54% defoliation and 49.21% leaf infection at 25 DAI. There was also variation in inoculum concentration reaction with wilt and defoliation (Table 2). Inoculum concentration of 2.4×10^6, and 1.3×10^5 spores/ml of distilled water showed maximum defoliation and wilt symptoms at 20 and 25 DAI. The least defoliation and wilt symptoms were recorded with inoculum concentration of 3.2×10^2 spores/ml of sterile distilled water at different inoculation intervals.

Latent infection of *C. gloeosporioides* f. sp. *manihotis* on symptomless cassava materials and survival period of fresh plant parts: There was variation in acervuli number among the test cassava clones (Table 3). The log transformation of acervuli number was maximum in cultivars TMS 91/00072, 91/00695, 88/02116 with mean values of 5.35, 5.53 and 5.49 respectively. Very few stem cuttings showed absence of acervuli such as cultivar TMS 88/02549, 91/00458, 91/00040 and 91/00424. The survival period of fresh plant parts before death was maximum for cultivars 91/00684 and 91/00454, with mean survival period of 10.5 days. The lowest survival period was recorded in clone

Table 3: Latent infection of *C. gloeosporioides* f.sp *manihotis* in fresh cassava plant parts.

Cassava cultivar	Log transformation acervuli population[a]		Plant partssurviva l period/days[b]	
	Stem section	Leaf section	Stem	Leaf
91/00684	2.80 ± 0.07	0.0 ± 0.0	10.50	5.67
91/00454	2.98 ± 0.16	1.65 ± 0.58	10.50	4.00
88/01990	0.0 ± 0.0	0.0 ± 0.0	10.25	6.00
87/00613	1.95 ± 0.19	1.80 ± 0.33	10.00	5.67
91/00040	0.0 ± 0.0	1.75 ± 0.58	9.50	5.00
91/00424	0.0 ± 0.0	0.0 ± 0.0	9.50	5.00
89/00031	3.30 ± 0.05	3.75 ± 0.55	9.25	6.00
91/00495	4.78 ± 0.63	3.62 ± 0.16	9.25	3.67
91/00688	3.88 ± 0.07	2.84 ± 0.05	9.00	4.33
91/00016	1.51 ± 0.17	3.50 ± 0.63	8.75	3.33
91/00435	3.71 ± 0.04	2.76 ± 0.04	8.75	4.00
89/0008	0.0 ± 0.0	3.38 ± 0.17	8.50	4.63
91/00344	3.31 ± 0.07	1.75 ± 0.03	8.25	5.55
91/00451	5.54 ± 0.01	3.69 ± 0.08	8.25	5.65
30001	3.46 ± 0.81	0.0 ± 0.0	8.25	4.33
88/01084	2.59 ± 0.13	3.17 ± 0.51	8.00	5.33
88/00210	3.26 ± 0.09	0.0 ± 0.0	8.00	5.00
89/00073	1.48 ± 0.51	3.75 ± 0.05	8.00	4.33
91/00072	5.35 ± 0.09	3.68 ± 0.20	8.00	3.50
91/00246	0.0 ± 0.0	0.0 ± 0.0	8.00	3.33
89/00250	4.24 ± 0.16	3.07 ± 0.13	7.75	5.30
91/00313	2.72 ± 0.12	2.40 ± 0.07	7.75	4.00
91/00415	4.46 ± 0.69	2.27 ± 0.03	7.75	5.33
91934	4.37 ± 0.03	0.0 ± 0.0	7.25	4.67
91/00686	4.41 ± 0.32	2.43 ± 0.19	6.75	3.67
88/01983	3.10 ± 0.17	2.75 ± 0.04	6.75	4.33
91/00517	4.42 ± 0.25	3.10 ± 0.67	6.50	4.00
88/02555	3.80 ± 0.04	3.36 ± 0.06	6.50	3.33
91/00184	4.54 ± 0.50	3.17 ± 0.15	6.50	5.33
91/00333	4.30 ± 0.04	2.44 ± 0.05	6.50	4.00
91/01137	4.05 ± 0.08	3.10 ± 0.25	6.25	4.30
91/00458	0.0 ± 0.0	1.84 ± 0.04	6.25	4.67
88/01043	4.36 ± 0.06	2.67 ± 0.20	6.25	5.67
88/02116	5.49 ± 0.15	3.50 ± 0.55	6.25	3.33
88/00695	5.53 ± 0.22	3.16 ± 0.08	6.25	4.00
89/00011	4.84 ± 0.05	3.63 ± 0.65	6.00	3.67
88/02549	0.0 ± 0.0	0.0 ± 0.0	6.00	6.00
91/01163	2.19 ± 0.19	0.0 ± 0.0	6.00	5.33
91/00066	4.66 ± 0.33	3.70 ± 0.05	5.75	3.33
91/00430	5.29 ± 0.02	3.39 ± 0.30	5.75	4.33
91/00421	1.97 ± 0.06	1.25 ± 0.02	5.50	3.50
88/02486	4.25 ± 0.66	3.78 ± 0.15	5.25	4.60
88/00929	4.89 ± 0.20	3.82 ± 0.40	5.25	4.00
30572	3.34 ± 0.02	1.78 ± 0.03	5.25	3.30
91/00300	5.52 ± 0.70	3.92 ± 0.09	5.25	4.33
CV	19.10	15.37 11.13	17.49	
LSD (0.05)	0.49	1.20 1.16	1.22	

a, b = Values are means ± Standard error of 5 plant parts/petri dish of five replications

TMS 91/00300 and TMS 30572 with mean survival period of 5.25 days. Some symptomless cassava leaves after incubation at 25 ± 2 °C showed the presence of acervuli (Table 3). The log acervuli number was maximum in the leaves of cultivar one 91/00300 with mean value of 3.92. The mean survival period for fresh cassava leaves ranged from a maximum of 5.67 days in cultivar 87/00613 to a minimum of 3 days in cultivar 91/01163. There were few cassava cultivar leaves with no acervuli such as cultivar 91/00246 and 91/01163.

Discussion

The effect of inoculum concentration on CAD development showed that there was a general increase in percentage leaf defoliation with increase in inoculum concentration. There was however maximum percentages leaf defoliation of above 80% in cassava cultivar TME 1 and 88/02549 at 25 DAI. At five DAI

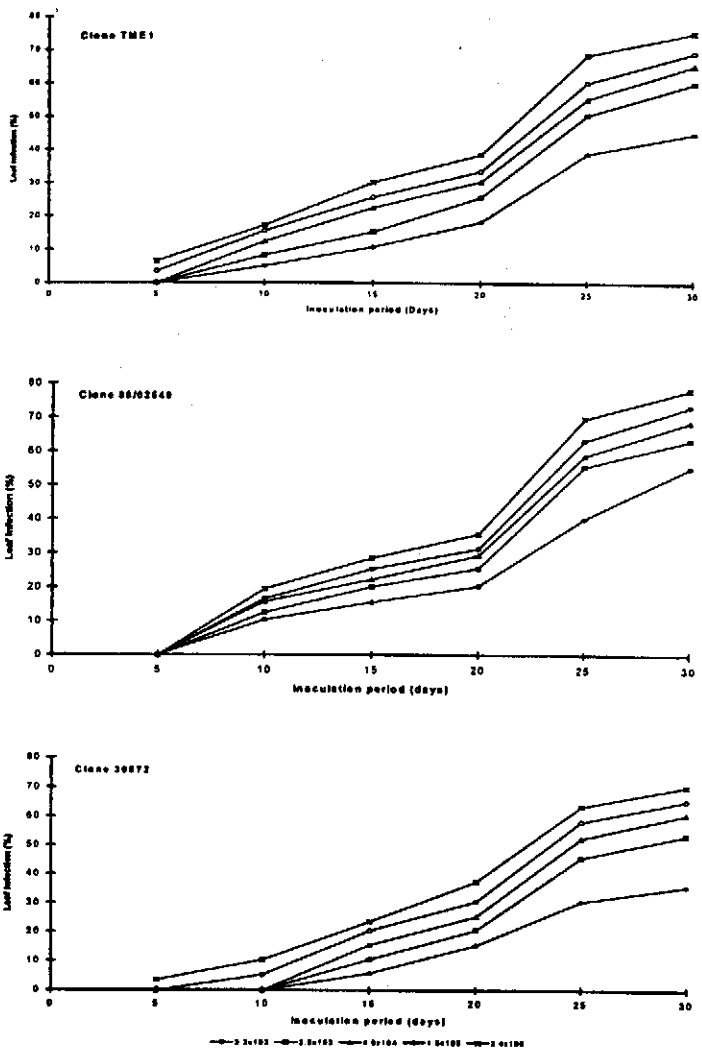

Fig. 2: Effect of inoculum concentration of *Collectotrichum gloeosporioides* f.sp manihotis on leaf infection of cassava cultivars.

there was no significant defoliation at all levels of inoculum concentration of the fungal isolates. Inoculum concentration of 2.4×10^6 spores/ ml of sterile distilled water caused leaf infection of about 85% in cassava clone TME 1.

Due to the complexity of the factors known to influence the development of CAD severity, Irvine (1969) and Lozano and Booth, (1974), Makambila (1987) reported that an understanding of the influence of inoculum concentration of disease infection is important in order to estimate quantitatively the host-pathogen and environmental interaction. In this study increased inoculum concentration of *C. gloeosporioides* f.sp *manihotis* had a significant effect on defoliation and leaf infection, with increased in incubation period

There was however cultivar sensitivity to fungal inoculum, and cultivar TME 1 was more sensitive to the pathogen inoculum, recording a high leaf infection and defoliation at high inoculum concentration of 2.4×10^6 spores/ml of sterile distilled water. The higher the inoculum concentration, the more severe the disease symptom expression. This agrees with the findings of Muimba (1982) that *C. manihotis* inoculum containing as few as 4.5×10^5 spores/ml were significantly more effective in causing defoliation than other concentration dosages. This relationship was

interpreted by Van der Plank (1978), as self-inhibition of spore germination on susceptible sites of host when inoculum is abundant. A few fungal cells may bring about the infection process provided it is placed in a right infection court under optimal environmental conditions. However this is not always true as the morphogenic stimulus of the spore may be too weak, and spores in mass help one another to infect even better up to a point. Frantz (1991) reported that the rate of increase of infection could attain a maximum then diminish with the addition of more inoculum. High levels of the inoculum in most cases decrease the time the symptoms will appear but rarely too high inoculum dose may decrease infection (Manners, 1993). This study could enable the setting up of a standard test in which environment and inoculum of the pathogen are controlled to the point at which screening test could be reasonably comparable.

Studies on latent infection and the survival period of cassava materials showed that more than 80% of the cassava cultivar showed the presence of acervuli of *C. gloeosporioides* f. sp. *manihotis*. This is an indication that a symptomless cassava material could contain CAD propagules that may only manifest later in the course of their growth and development. Many fungal diseases of plants can have a symptomless phase that may be extremely brief, particularly if the pathogen elicit a hypersensitive response, but the incubation period and latent period are often markedly prolonged. Report by Jeffries *et al.* (1990) showed that latent infection involves inhibition of development of the pathogen through physiological conditions imposed by the host, until some stage of maturation has been accomplished. (Bailey *et al.*, 1992; Prusky and Keen, 1993 and Williamson, (1994), showed that spores of *Colletotrichum gloeosporioides* of fruits tropical and subtropical regions, germinate on the surface of the fruit and form melanized appressoria which do not begin to produce infection peg until the fruits mature. This appressoria are essential for infection of the disease to occur.

There are postulations that latent infection in *C. gloeosporioides* f. sp. *manihotis* is due to resistance and pre-formed fungitoxic compounds. Phenolics and peroxidases as resistant components in cassava has been reported by Van der Bruggen and Maraite, (1987). The tendency of CAD pathogens to enter a latent or quiescent phase in their life cycles confers a substantial ecological advantage on the pathogen. This could pose a problem in breeding for disease resistance or in the design of control strategies. The results obtain in this study have shown that the causal agent of CAD may reside on apparently healthy shoots or plant parts of cassava cultivars and then develop only when conditions become favourable in the field. This passive form of the fungus may be potentially capable of causing severe infection in susceptible cultivar materials. The short survival period of the cassava leaves and stem pieces is an indication of a close association between early stem and leaf browning and the development of acervuli. Muimba, (1982) suggested that early death of cassava tissue could create a suitable environment for the development of *C. gloeosporioides* f. sp. *manihotis*. This observation was earlier reported by Pappelis (1965), that factors which influence weakening and cell death increase susceptibility of the host to pathogen infection.

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