Fusarium wilt of banana (Panama disease) in Malawi

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Le wilt des bananiers dû au *Fusarium* (maladie de Panama) au Malawi.

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Fruits, vol.47, n°4, p. 503-508.

RESUME - Le wilt causé par *Fusarium oxysporum* f. sp. *cubense* a été observé dans le nord (districts de Karonga et de Chipita) et le sud (districts de Thyolo et de Mulanje) de la zone de production bananière du Malawi en 1989 et en 1990. Les inter-relations et les origines possibles du pathogène ont été étudiées dans quatre grands foyers de dissémination de la maladie dans le Nord (25 sites et 39 souches du pathogène) et un seul grand foyer dans le Sud (6 sites et 6 souches). La compatibilité végétative, déterminée par auxotrophie aux nitrates, a permis d'identifier 3 groupes de *F. oxysporum* f. sp. *cubense*. Trente-cinq des 45 souches testées (78%), se sont avérées compatibles avec le groupe de compatibilité végétative (VCG) 0124, trouvé dans une autre région d'Afrique de l'Est. Les souches restantes (22%) concernaient le VCG 01214, un nouveau VCG observé seulement dans les montagnes de Misuku (district de Chipita). Les souches étaient aussi polymorphes pour la production d'enzymes pectolytiques. Trois zymogrammes pectiques différents (PZGs) ont été observés parmi 42 souches testées. Deux des PZGs, identifiés par la production d'une estérase de pectine, ont été obtenus à partir d'un sous-groupe de certaines souches venant de la frontière avec la Tanzanie. Ceci suggère que les foyers de wilt causé par *Fusarium*, observés en 1989 et en 1990, étaient dûs à des populations apparentées du pathogène, et que la maladie s'était répandue du Nord vers le Sud du Malawi, probablement au cours des dernières 25 années. Dans un contexte plus large, la première manifestation du wilt dû au *Fusarium* en Malawi est considérée comme faisant partie d'un plus grand mouvement épidémique qui aurait commencé au Kenya, en Ouganda et dans le Nord de la Tanzanie il y a environ 40 ans et qui s'est aujourd'hui répandu dans toute l'Afrique de l'Est

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ABSTRACT - Fusarium wilt, caused by *Fusarium oxysporum* f. sp. *cubense*, was observed in northern (Karonga and Chitipa Districts) and southern (Thyolo and Mulanje Districts) banana-production areas in Malawi in 1989 and 1990. Relationships among and possible origins for the pathogen were assessed in four large disease foci in the north (25 fields and 39 strains of the pathogen) and one large focus in the south (6 fields and 6 strains). Vegetative compatibility, determined with nitrate auxotrophs, identified three groups of *F. oxysporum* f. sp. *cubense*. Thirty-five of the 45 strains that were tested (78%) were compatible with vegetative compatibility group (VCG) 0124, a VCG found elsewhere in East Africa. The remaining strains (22%) comprised VCG 01214, a new VCG found only in the Misuku Hills (Chitipa District). Strains were also polymorphic for the production of pectolytic enzymes. Three different pectic zymograms (PZGs) were observed among 42 strains that were tested. Two of the PZGs, distinguished by the production of a pectin esterase, were produced by a subset of strains from the Tanzanian border. It is suggested that foci of fusarium wilt that were studied in 1989 and 1990 were caused by closely related populations of the pathogen, and that the disease has spread from northern to southern Malawi, probably in the last 25 years. In a wider context, the outbreak of fusarium wilt in Malawi is considered to be part of a larger epidemic that began in Kenya, Uganda and northern Tanzania some 40 years ago and which has now spread throughout East Africa.

MOTS CLÉS : *Musa,* Malawi, *Fusarium oxysporum,* wilt, maladie de Panama, champignon pathogène, provenance, distribution de la population, identification, souche, compatibilité végétative.

KEYWORDS: *Musa,* Malawi, *Fusarium oxysporum,* wilt, Panama disease, pathogenic fungi, provenance, population distribution, identification, strain, vegetative compatibility.

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Introduction

Fusarium wilt of banana, caused by *Fusarium oxysporum* f. sp. *cubense*, has a long and destructive history. The disease destroyed an estimated 40,000 hectares in export plantations of Gros Michel before 1960, and has affected the production of locally consumed cultivars for more than 100 years (PLOETZ *et al.*, 1990; SIMMONDS, 1966). Epidemics on the latter cultivars continue to impact the livelihood of subsistence farmers in the developing world.

Historically, three different epidemics of fusarium wilt have occurred on the African continent (PLOETZ *et al.*, 1990). Two of these epidemics have been on export cultivars. One on Gros Michel in West Africa ceased to be a problem after the cultivar's widespread use in the region was discontinued. The other, on Cavendish cultivars, continues to be an important but localized problem in the Natal and Transvaal Provinces of South Africa.

A third outbreak of fusarium wilt in Africa occurs on diverse, locally consumed cultivars. The disease was first recognized on these cultivars in the early 1950s in Kenya, northern Tanzania, and Uganda, and subsequent reports indicate that fusarium wilt has now spread to many neighbouing countries (Anonymous, 1954; JAMESON, 1953; SEBASIGARI and STOVER, 1988; PLOETZ *et al.*, 1990; WALLACE, 1952). Despite the importance of the East African fusarium wilt epidemic, scant research has been conducted on the disease or causal fungus in the region.

Fusarium wilt was observed in several different areas in Malawi in 1989 and 1990 (PLOETZ *et al.*, 1990; 1991). The objectives of the present study were to document the current distribution of fusarium wilt in Malawi, and to investigate the relatedness and possible origins of the pathogen in the affected areas.

Materials and methods

Disease surveys

Disease surveys were conducted during 1989 and 1990 (table 1 and figure 1). In 1989, samples were taken from Kaporo North, Kaporo South and the Misuku Hills extension planning areas (EPAs); since collection locations were not recorded in 1989, these samples are listed as sites xx in table 2. In 1990, additional samples were taken from four large disease foci in the north (in figure 1, focus 1 = sites 11 - 14 and 19 - 20; focus 2 = sites 15 - 18; focus 3 = sites 1 - 7; and focus 4 = sites 8 - 10) and one focus in the south (focus 5 = sites 21 - 26). During 1989 and 1990, a total of 26 sites in the north and 6 sites in the south were sampled.

Pathogen isolation

In the laboratory, discolored vascular strands were cut from samples, surface-disinfested in 70% ethanol for 10-15 sec, in 10% commercial bleach for 2 min, rinsed with sterile water, blotted dry on sterile paper towels, and placed in sterile, 9-cm-dia petri dishes. Molten (45°C), 1.5% water agar (WA) amended with danitol miticide (Chevron Chemical Co.) and either 100 mg streptomycin sulfate or 10 mg rifamycin + 250 mg ampicillin L^{-1} was poured over vascular strands and allowed to solidify. Single spores were recovered from pure colonies of *F. oxysporum* f. sp. *cubense* which grew from tissue and transferred to a mineral salts medium used during vegetative compatibility tests (minimal medium) or Difco potato dextrose agar (PDA). Forty-five monoconidial strains, representing 31 of the 32 total sites that were visited, were recovered and stored on sterile filter paper at 4°C as described by Correll *et al.* (1986), or in sterile 50% glycerol at –80°C.

Figure 1. Locations in the Karonga and Blantyre Agricultural Development Divisions (ADDs) in Malawi. Numbered sites in the ADDs are locations that were sampled for Fusarium oxysporum *f. sp.* cubense*; they are described in table 1. The areas that are circled with a dotted line define the five large disease foci that were studied.*

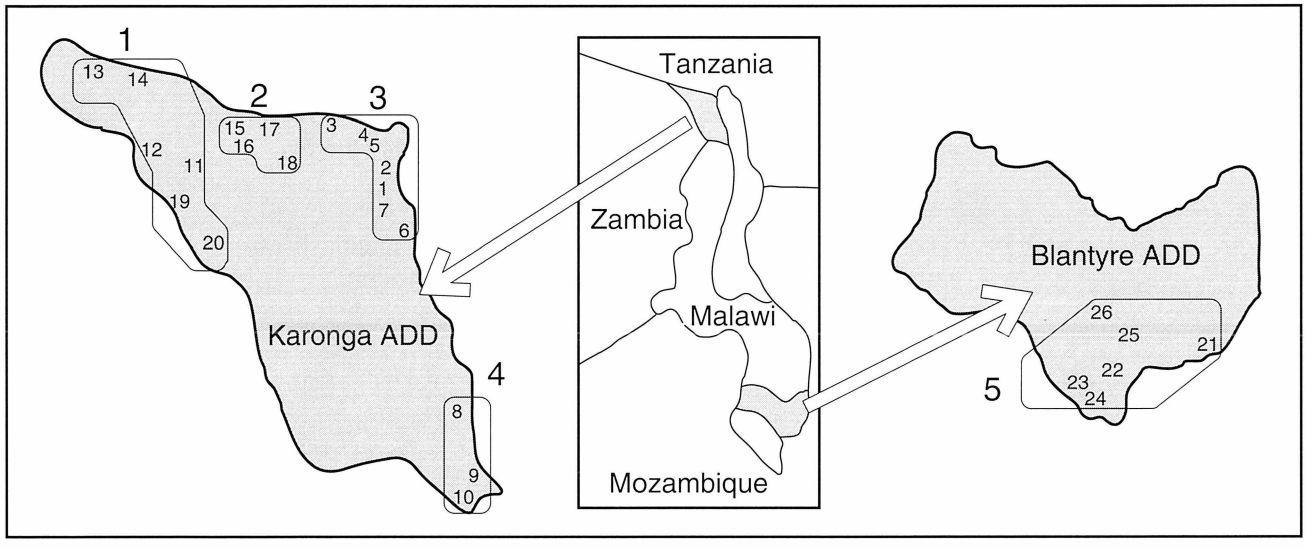

Table 1. Locations in the Karonga and Blantyre ADDs which were sampled for *Fusarium oxysporum* f. sp. *cubense* in 1990.

Site*	City/village	Symptomatic cultivars	Year fusarium wilt (FW) first recognized; comments
			Karonga ADD
Kaporo North EPA (KR/1)			
1	Kasantha	Bluggoe	1987; Bluggoe planted in mid-1960s
2	rural	Bluggoe	1985; owner may have observed FW in southern Tanzania in mid-1950s
3	Mwandambo	Bluggoe	1984; Bluggoe planted by great-grand-parents of elderly owner
4	rural**	Bluggoe, Silk, Pisang awak,	1975; FW first evident on Pisang awak in 1987
5	rural**	Pisang awak	1969; Bluggoe eliminated by FW; Pisang awak planted in 1978 and affected by FW since 1989
Kaporo South EPA (KR/2)			
7	Kafwala	Bluggoe, Silk, Pisang awak	1983; FW on Pisang awak since 1989
Karonga North EPA (KR/3)			
6	Karonga	Pisang awak, Silk	?; Bluggoe not observed
Karonga South EPA (KR/4)			
8	Chilemba	Bluggoe	1984
9	Mwakhwawa	Bluggoe	1981; planted in 1947
10	Hara Plain	Bluggoe	?; owner reported observing FW 20 km further south in Mlowe
CT/2 EPA			
11	rural	Bluggoe	?
12	Chitipa	Bluggoe, Silver Bluggoe	1986; Pisang awak not affected
CT/1 EPA			
13	Mwamkumbwa	Bluggoe, Silver Bluggoe	1988; Pisang awak not affected; owner observed FW in Zambia
14	Mwamkumbwa	Bluggoe	?
Misuku Hills EPA (CT/3)			
15	Misuku Center	Bluggoe, Silver Bluggoe	1976; Pisang awak rare in area
16	Misuku Center	Bluggoe	1979; Bluggoe planted in 1937; grower reports Tanzanian origin for FW in Misuku Hills
17	rural	Bluggoe	1978; Bluggoe planted in 1942
18	Yeyeyene	Bluggoe	?; Bluggoe planted in 1954; grower reported bringing other banana cultivars to the area from Uganda
CT/4 EPA			
19	Chesenga	Bluggoe	1988 [high incidence (35%) suggests longer presence]
20	Kasololo	Bluggoe, Silver Bluggoe	?; owner not aware of problem on either cultivar
			Blantyre ADD
Mulanje District			
21	Nesa area	Bluggoe	1989; Bluggoe planted in 1965
Thyolo District			
22	Kerengendarge	Bluggoe	? (a "long time")
23	Thekerani	Bluggoe	1989; Bluggoe planted before 1954
24	Thekerani	Bluggoe	1980?
25	Semu	Bluggoe	?
26	Kautuka (Mbandanga E.)	Bluggoe	1990; Bluggoe planted in 1975; Pisang awak not affected

* Sites correspond to those on map in Figure 1.

** These sites were each within 5 km of the only bridge in Malawi which crossed the Songwe River into southern Tanzania.

Vegetative compatibility tests

Standard protocols for generating and using nitrate auxotrophic (*nit*) mutants in nutritional complementation tests were followed (CORRELL *et al.*, 1987; PUHALLA, 1985). In short, mutants with a *nit*1 phenotype were generated for all, and NitM mutants were generated for some of the studied strains. Mutants were then paired with NitM and *nit*1 testers, respectively, from previously described VCGs of *F. oxysporum* f. sp. *cubense* (PLOETZ, 1990; PLOETZ, unpublished). Strains were considered vegetatively compatible if a tuft of prototrophic (heterokaryotic) growth developed between *nit* mutants. Strains that did not complement testers were tested for self-compatiblity, and in all possible combinations with each other. All pairings were conducted at least twice.

Pectolytic enzyme analyses

Production of pectolytic enzymes by 42 of the strains was assessed using a modification of previously described protocols (CRUICKSHANK, 1983; CRUICKSHANK *et al.*, 1980). Briefly, strains were cultured in stationary, liquid medium that contained citrus pectin as the sole source of carbon. After 7 da, culture supernatant was harvested, clarified with a low speed centrifugation, and frozen at – 80°C prior to use. Enzyme activity for culture supernatants was determined with a standard cup plate assay. Pectolytic enzymes were separated electrophoretically in horizontal 0.7% polyacrylamide gels amended with citrus pectin, and wells were each loaded with 0.005 units of activity. Modifications of the previously described protocols included use of a tris-glycine buffer (pH 8.7) and use of constant amperage instead of constant voltage (25 mA for voltage ranging from 200 to 300V). In addition, band resolution was increased by increasing run times to 5 hr and staining time in ruthenium red to overnight.

Results

Disease surveys

In 1989, fusarium wilt was found in the Misuku Hills, Kaporo North and Kaporo South EPAs (BRAUNWORTH, unpublished). A more extensive survey in 1990 revealed the disease in five additional EPAs in northern Malawi and, for the first time, in the Blantyre agricultural development division (ADD), some 700 km south of affected areas in the north.

In the north and south, Bluggoe (known locally as Harare and Kholobowa, respectively) was either the first or the only cultivar affected by fusarium wilt (table 1). Only in areas in which the disease had been present for seven or more years did cultivars other than Bluggoe or the closely related Silver Bluggoe (Mbufu) also succumb to the disease. Fusarium wilt was observed on Pisang awak (Zambia) and Silk (Sukali) in only three EPAs in northeast Malawi: Kaporo North, Kaporo South and Karonga North (table 1).

Vegetative compatibility tests

Seventy-eight percent (35 of 45) of the recovered strains were compatible with testers for either VCG 0124 or both VCG 0124 and VCG 0125; no strains complemented only VCG 0125 (table 2). The 10 strains that did not complement previously described testers, were later found to complement each another. All of these strains were recovered from the Misuku Hills, located on the Tanzanian border, and together they compromised a new VCG of *F. oxysporum* f. sp. *cubense*, VCG 01214.

Pectolytic enzyme analyses

Three distinct pectic zymograms were observed (figure 2 and table 2). Pectic zymogram A (PZG-A) consisted of seven bands of polygalacturonase (PGl - PG7) activity, each visualized as clear zones in either the cathodal or anodal sectors of the gel. All VCG 0124 and VCG 0124/0125 strains from southern Malawi and some from northern Malawi had the PZG-A phenotype. In contrast, PZG- B and PZG-C were both characterized by five of the same PG bands observed in PZG-A (PG3 - PG7), a slower migrating PG2, and a band of pectin esterase activity (PEl) not found in PZG-A; it was visualized as a dark red band. PZG-B and PZG-C were distinguished from each other by the presence and absence, respectively, of PG1. All strains of VCG 01214 that were tested, as well as most of those in VCG 0124/0125 from northern Malawi, possessed either the PZG-B or PZG-C phenotype. No strains from southern Malawi or in VCG 0124 had the PZG-B or PZG-C phenotype.

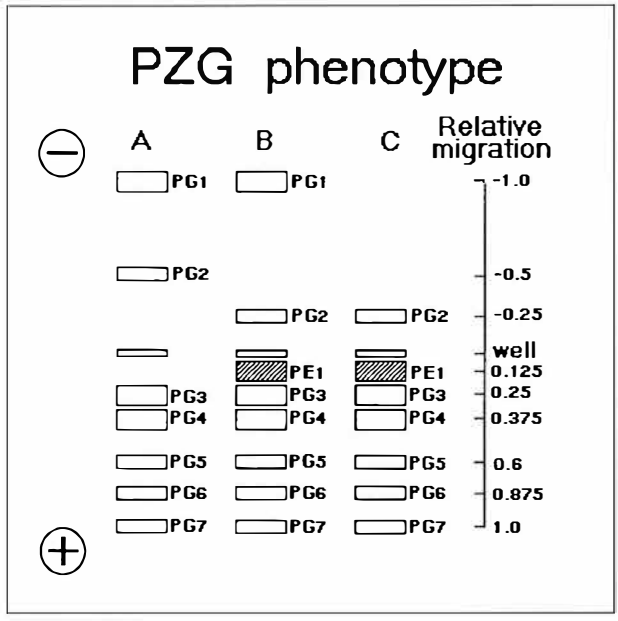

Figure 2. Pectic zymograms for strains of Fusarium oxysporum *f. sp.* cubense *from Malawi. Band migration is relative to the lowest band common to all phenotypes (PG7) and the position of the well. + and – correspond to the anodal and cathodal sectors of the gel, respectively.*

Discussion

Within the last five years, vegetative compatibility has become a commonly utilized character during studies on genetic diversity and population biology of plant pathogenic fungi (LESLIE, 1990). Since strains of a given species in

Table 2. **Attributes of strains of *Fusarium oxysporum* f. sp. *cubense* recovered in the Karonga and Blantyre Agricultural Development Divisions (ADDs) during 1989 and 1990.**

Site (a)	Strain (b)	Cultivar	VCG	Pectic zymogram phenotype
		Karonga ADD		
Kaporo North EPA (KR/1)				
1	MW 55	Bluggoe	0124	not tested
2	MW 64	Bluggoe	0124	A
	MW 73	Bluggoe	0124	A
3	MW 58	Bluggoe	0124	A
4	MW 53	Silk	0124 / 0125	C
	MW 60	Pisang awak	0124 / 0125	C
5	MW 56	Pisang awak	0124 / 0125	B
xx	MW 11	Bluggoe	0124 / 0125	B
Kaporo South EPA (KR/2)				
7	MW 52	Silk	0124	A
	MW 59	Pisang awak	0124 / 0125	B
	MW 65	Bluggoe	0124	A
xx	MW 5	Pisang awak	0124 / 0125	B
xx	MW 9	Pisang awak	0124 / 0125	B
Karonga North EPA (KR/3)				
6	MW 57	Pisang awak	0124	A
Karonga South EPA (KR/4)				
8	MW 61	Bluggoe	0124 / 0125	A
	MW 78	Bluggoe	0124	A
9	MW 54	Bluggoe	0124	A
	MW 63	Bluggoe	0124 / 0125	A
10	MW 62	Bluggoe	0124	A
CT/2 EPA				
11	MW 43	Bluggoe	0124	A
	MW 49	Bluggoe	0124	A
12	MW 50	Bluggoe	0124	not tested
CTA/2 EPA				
13	MW 38	Bluggoe	0124	A
14	MW 39	Bluggoe	0124 / 0125	B
Misuku Hills EPA (CT/3)				
15	MW 40	Bluggoe	01214	B
	MW 41	Silver Bluggoe	01214	C
	MW 42	Bluggoe	01214	B
	MW 44	Bluggoe	01214	B
	MW 51	Bluggoe	01214	B
	MW 89	Bluggoe	01214	C
16	MW 46	Bluggoe	01214	C
17	MW 48	Bluggoe	01214	B
	MW 87	Bluggoe	0124	B
xx	MW 2	Bluggoe	01214	C
xx	MW 7	Bluggoe	01214	C
xx	MW 15	Bluggoe	0124 / 0125	B
CT/4 EPA				
19	MW 47	Bluggoe	0124	A
20	MW 45	Bluggoe	0124	A
	MW 86	Bluggoe	0124 / 0125	A

Table 2. Attributes of strains of *Fusarium oxysporum* f. sp. *cubense* recovered in the Karonga and Blantyre Agricultural Development Divisions (ADDs) during 1989 and 1990 (cont'd.).

Site (a)	Strain (b)	Cultivar	VCG	Pectic zymogram phenotype
		Blantyre ADD		
Mulanje District				
21	MW 71	Bluggoe	0124	A
Thyolo District				
22	MW 68	Bluggoe	0124 / 0125	A
23	MW 67	Bluggoe	0124	not tested
24	MW 70	Bluggoe	0124 / 0125	A
25	MW 69	Bluggoe	0124	A
26	MW 66	Bluggoe	0124 / 0125	A

(a) Sites correspond to those on map in figure 1.
(b) When more than one strain is reported for a given site, each was recovered from a different plant.

different VCGs have a reduced ability to exchange genetic information, vegetative compatibility focuses evolutionary processes and defines genetically isolated subpopulations within that species. In asexual taxa, such as *F. oxysporum* f. sp. *cubense*, VCGs generally define clonally related populations.

During the current study, vegetative compatibility helped distinguish strains of *F. oxysporum* f. sp. *cubense* from Malawi. Many of the strains that were examined during this work bridged both VCGs 0124 and 0125. Bridging is a fairly common phenomenon in VCGs 0124 and 0125 (PLOETZ, 1990). Although bridging is not well understood, it is thought that VCG 0124 and VCG 0125 represent closely related, but diverging populations of this pathogen. Even though bridging was not detected between VCG 0124/0125 and VCG 01214, the 0124/0125 complex and VCG 01214 might also be related since some of the strains from each group attacked Bluggoe and possessed the PZG-B pectic zymogram phenotype. Thus, strains of *F. oxysporum* f. sp. *cubense* from Malawi appear to be closely related.

Previously, vegetative compatibility was assessed among strains of *F. oxysporum* f. sp. *cubense* from Burundi, northern Tanzania, Uganda, Zaire, and the islands of Pemba and Zanzibar (SEBASIGARI and STOVER, 1988). Although only eight strains were tested, the results from this work parallel those in the current study. Four of the strains (50%) from Burundi, Tanzania, Uganda and Zaire were in the 0124/0125 complex and four, all from Tanzania, comprised a new VCG, VCG 01212. Interestingly, the VCG 0124/0125 strains possess PZG-B phenotypes, and those of VCG 01212 possess PZG-A phenotypes (unpublished data). When one considers the vast areas of land devoted to banana production and the wide distribution of fusarium wilt in East Africa, it is probable that significant, unrecognized variability in *F. oxysporum* f. sp. *cubense* still exists in this region.

Based on the present work and information gathered during the 1990 survey, it is possible to speculate on the origin of *F. oxysporum* f. sp. *cubense* in Malawi. By 1990, producers had recognized fusarium wilt in the Misuku Hills and Kaporo North EPAs, both of which border southern Tanzania, for 15 and 22 years, respectively. At that time, one of the producers in the Kaporo North EPA, an immigrant from Tanzania, suggested that the disease had come from southern Tanzania. Since the diversity of banana cultivars that were affected and VCG and PZG diversity in the pathogen was greater here than elsewhere in Malawi, a southern Tanzanian origin for F. oxysporum f. sp. cubense in Malawi is probable. Obviously, precautions should be taken against future, inadvertent introductions of banana pathogens, such as banana bunchy top virus and race 4 of *F. oxysporum* f. sp. *cubense*, which are not currently found in Malawi.

Acknowledgements

This research was supported, in part, under Grant No. COM-5600-G-00-0023-0, Program in Science and Technology Cooperation, Office of the Science Advisor, U.S. Agency for International Development (USAID). We also acknowledge support provided during the survey for R.C. Ploetz and W.S. Braunworth, Jr. by USAID, Malawi Research and Extension Project Project (MARE), support provided for A.G. Channer by the Crop Protection Research Unit, Department of Agriculture, Reading University, and funding for C.T. Chizala, D.L.N. Banda and D.W. Makina provided by the Chief Agricultural Research Officer of Malawi.

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Fruits, vol.47, n°4, p. 503-508.

RESUMEN - El wilt causado por *Fusarium oxysporum* f. sp. *cubense*, ha sido observado en las zonas bananeras de Malawi en el Norte (distritos de Karonga y Chipita) y en el Sur (distritos de Thyolo y Mulanje) en 1989 y 1990. Las relaciones y las posibles origenes de los patógenos han sido evaluadas en cuatro grandes focos (o zonas de extensión de la enfermedad en el Norte (25 campos y 39 cepas des patógeno) y una grande zona de extensión de la enfermedad en el Sur (6 campos y 6 cepas). La compatibilidad vegetativa, determinada con auxotrofia al nitrato, permite la identificación de 3 grupos de *Fusarium oxysporum* f. sp. *cubense*. 35 de las 45 cepas probadas (78%) han sido compatible con el grupo 0124 de compatibilidad vegetativa (VCG), un VCG encontrado en otro sitio de Africa del Este. Las otras cepas (22%) constituyen CCG 01214, un nuevo VCG encontrado exclusivamente en los montes Misuku (distrito de Chitipa). Las cepas han sido polimorfas para la producción de enzimas pectolíticas. Tres diferentes zimogramos pecticos (PZGs) han sido observados entre las 42 cepas probadas. Entre ellas, dos, distinguidas por la producción de una pectinesterasa, han sido producidas por un subgrupo de cepas de la frontera de Tanzania. Esto sugiere que las zonas de extensión de la enfermedad del wilt estudiadas en 1989 y 1990, han sido causadas por poblaciónes del patógeno muy proximas y que la enfermedad se extendió desde el Norte hacia el Sur del Malawi probablemente durante los últimos 25 años. En un contexto más amplio, la rápida extensión del wilt debido a *Fusarium* en el Malawi es considerada como parte de una epidemia más importante iniciada en Kenya, Uganda y Norte de la Tanzania algunos 40 años antes y que se extendió ahora en todo el Africa del Este.

PALABRAS CLAVES : *Musa,* Malawi, *Fusarium oxysporum,* wilt, enfermedad de Panama, hongos patógenos, procedencia, distribución de la población, identificación, cepa, compatibilidad vegetativa.