MARCHITAMIENTO POR *FUSARIUM* (MAL DE PANAMÁ) EN BANANOS: UNA REVISIÓN ACTUALIZADA DEL CONOCIMIENTO PRESENTE SOBRE SU AGENTE CAUSAL

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RESUMEN

El marchitamiento de plátano o enfermedad de Panamá causada por Fusarium oxysporum f. *sp.* cubense *(*Foc*) es una de las enfermedades de mayor importancia económica y dañina del género* Musa*. Ella fue la causa de destrucción de más de 50 000 ha de Gros Michel y su sustitución por el cultivar Cavendish, junto con importantes transformaciones de la industria de exportación de plátano durante la primera mitad del siglo pasado. Unos años después de la excelente revisión de R. H. Stover publicada en 1962, la enfermedad llegaba para establecerse. Desde entonces se ha ganado un importante conocimiento con respecto a la diversidad de Foc y su probable origen. Esto salió a través de la aplicación de la técnica de los grupos de compatibilidad vegetativa (VCG) a* Foc*, y más tarde por diferentes técnicas moleculares. La creencia de que los cultivares Cavendish podían ser atacados solo en los subtrópicos, les permitió a las grandes empresas de exportación de plátano hacer inversiones importantes sobre plantaciones Cavendish en Filipinas, península de Malasia e Indonesia a inicio de la década del noventa. Hubo muchos millones de pérdidas debido a la nueva y agresiva cepa Cavendish VCG 01213 («raza tropical 4»). En el presente artículo se realiza una revisión del conocimiento actual sobre la estructura de poblaciones* Foc*, la reacción de variedades contra diferentes VCG y los posibles mecanismos de defensa involucrados, así como las posibilidades de manejo a través de los procedimientos de control químico y biológico, con énfasis en la necesidad de prevenir la entrada de cepas agresivas a las Américas y el papel de los servicios de cuarentena de las organizaciones nacionales de protección de planta.*

Palabras clave: *Fusarium oxysporum, banano, mal de Panamá*

ABSTRACT

Fusarium wilt of banana or Panama disease caused by Fusarium oxysporum f. cubense *(*Foc*) is one of the most economic important and harmful diseases of* Musa*. It was the cause of destruction of more than 50 000 ha of Gros Michel and the substitution for Cavendish cultivars together with important transformations of the banana export industry during the first half of the last century. A few years after the excellent published review of R. H. Stover in 1962 the disease was coming to halt; an important knowledge has been gained on the diversity of* Foc *and its probable origin from then. This came out through the application of the Vegetative Compatibility Groups (VCG) technique to* Foc *and later by different molecular techniques. The believe that Cavendish cultivars can be attacked only in the subtropics, led to the large banana export companies to make important investments on Cavendish plantations in the Philippines, Peninsular Malaysia and Indonesia at the beginnings of the 90's. Many millions were lost due to the new Cavendish aggressive strain VCG 01213 («tropical race 4»). A review of the present knowledge on the structure of* Foc *populations is carried out in the present paper, the reaction of varieties against different VCGs and the possible defense mechanisms involved as well as the possibilities of the management through chemical and biological control procedures emphasizing the need to prevent the entry of the aggressive strains to the America's and the role of the quarantine services of the National Plant Protection Organizations.*

Key words: *Fusarium oxysporum, banana, Panama disease*

INTRODUCTION

The history on the *Fusarium* wilt (Panama) disease of banana and plantains caused by *Fusarium oxysporum f.* sp. *cubense* (*Foc*) has been comprehensively reviewed by Stover (1962b), Ploetz (1990c) and more recently by Ploetz and Pegg (2000).

The first description of *Fusarium* wilt of banana and plantains was by Bancroft (1876) in Australia, who was unaware that at that time was dealing with a disease today widely recognized as one of the most destructive in the history of the world agriculture. The disease was again reported in 1890 in Central America (Ashby, 1913). It has been estimated that between 1890 and the mid 50's more than 40,000 ha of the cultivar Gros Michel (AAA) were destroyed [Stover, 1962b]. The Cavendish cultivars were only reported affected in the subtropics. The currently arising of *Foc* tropical race 4 (GCV 01213-01216) has caused important losses in plantations of Malaysia and Indonesia: more than 8 million plants on traditional plantations and more than 5,000 ha of commercial Cavendish plantations has been affected with annual losses over 75 millions USD, with effects on family income of thousands of workers and farmers [Masdek *et al*., 2003; Nasdir, 2003]. Its potential introduction to the Cavendish plantations of America would have a great economic and social impact.

A considerable increment of the number of reports of the disease occurred at the beginnings of the last century, mostly related to export commercial plantations. The glo-

bal distribution of the disease (*Fig. 1*) has an important anthropogenic component; as the infected rhizomes are frequently free of symptoms, it is not unusual that *Foc* were introduced into new areas with conventional plantation material [Ploetz and Pegg, 2000]. Although the disease is well known because its economic impact on the industry, it should be taken into consideration that only 21% of the world banana production is exported (68 million ton was produced in 2002; FAOSTAT data, 2004) and hence, the 80% of the production is locally consumed. The impact of the disease in subsistence agriculture are not so well documented and probably could not be as high as that reported by the industry of banana export in the last century. It is however important for small growers in Brazil [with a wide consumption of Prata types (AAB) highly susceptible to the disease], South and Central America, South East Asia and Africa.

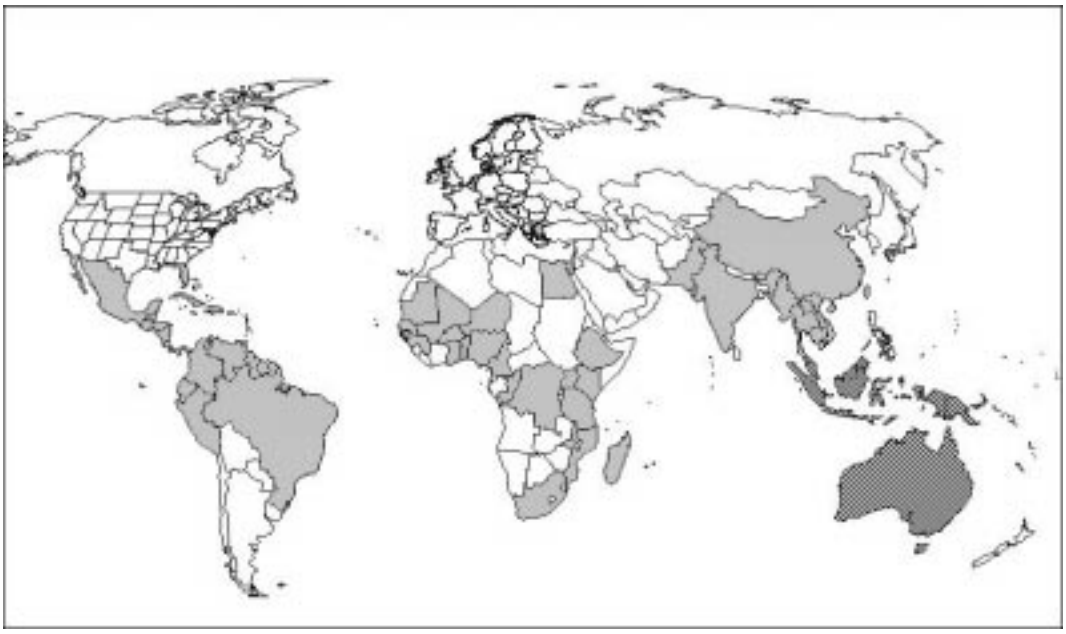

Figure 1. World distribution of *Fusarium oxysporum f.* sp. *cubense*: race 1 and 2 (countries shadowed with squares); subtropical race 4 (areas in grey South Africa, Canary Island, Taiwan and Australia); tropical race 4 [in dark oblique lines, Malaysia, Indonesia, Papua New Guinea and Australia (north Queensland)].

Disease distribution is highly related to the introduction of new cultivars to the growing areas [Stover, 1962b]. The cultivar Manzano (Silk, AAB) was introduced to the West Indies before 1750 as a shadow plant in cacao and coffee plantations and its presence indicates the establishment of *Fusarium* wilt. *Fusarium* wilt epidemics were related to the increment of Gros Michel demand by import countries and to the own disease effect. The lost of plantations productivity determine the use of new areas free of the disease. The cycle of planting and abandonment impulses the disease distribution. Based on the written records, the history of Fusarium wilt in the Western Hemisphere is unclear.

The first official report of the disease in Cuba was by Smith (1910). Johnston (1915), reported that the cultivars Gros Michel, Manzano and Burro Criollo (Bluggoe, ABB), were already severely infected in 1910, although there are antecedents of the presence of *Fusarium* wilt in Manzano since the last years of XIX Century. The distribution of the disease was highly related to the introduction of diseased material in free areas. The management practice relies exclusively in the destruction of affected areas and the replacement of the cultivar Gros Michel by Robusta (Cavendish subgroup, AAA). With the wide adoption of the Cavendish cultivars, the disease lost its economical importance lasting confined to the cultivars Manzano and Burro Criollo in small grower's plots and houses backyards. Due to the impact of Black Sigatoka in the costs of production, 12000 ha of FHIA hybrids with partial resistance to the disease has been introduced in substitution of the susceptible Cavendish banana and 63,000 ha of Burro CEMSA [Cardaba type BBB, according to Valmayor *et al.*, 2000 and Stover and Simmonds, 1987] and Pisang awak (ABB, susceptible to the disease) in substitution of the plantains [Pérez *et al.*, 2002]. In localities with conducible soils re-emergent outbreaks of *Fusarium* wilt has been reported in the cultivars Burro CEMSA, FHIA 03, FHIA 18 and Pisang awak [Pérez *et al.*, 2004a].

Fusarium wilt occurs in Africa in four areas: the Canary Island, West Africa, South Africa and East Africa. In Canary Island and South Africa Foc race 4 affect

Cavendish and all isolates belong to VCG 0120 suggesting a common origin [Stover, 1990]. In West Africa, from Zaire to Ghana, there were epidemics of *Fusarium* wilt on Gros Michel during the fifties of the last century; now occurs sporadically on the remaining plantations of Gros Michel. In East Africa, *Fusarium* wilt spread has been related to the distribution of cultivars Pisang awak and Bluggoe (AAB, highly susceptible) introduced after the Second World War [Stover, 1962b]. More recently there have been reports of the disease affecting highland banana AAA [Tushemereirwe, 1992; Ploetz *et al*., 1994].

The currently arising of *Foc* tropical race 4 (GCV 01213) and the damages caused by the disease in highland AAA cultivars in East Africa, renewed the interest in *Fusarium* wilt and drowns the attention to the great variation that exist within and between populations of *Foc.*

Fusarium wilt symptoms

Foc causes a typical wilt syndrome on the infected plants accompanied by the necrosis and rotting of roots, rhizome and pseudostem vessels (*Fig. 2*). First external symptoms in susceptible plants are the appearance of pale green streaks on the base of the petiole and the brown-reddish discoloration of the vessels under the epidermis of the petiole two weeks in advance of the most typical symptoms. These symptoms appear between 2 and 5 months after infection of roots [Stover, 1962b]. Two syndromes can develop afterward [Stover, 1959b]: the yellow leaf syndrome (*Fig. 2A*); yellowing of the oldest leaves that progress from the bottom to the upper leaves until remain green the unfurled leaf of the plants, process that can take a couple of weeks) and the non-yellow leaf syndrome (*Fig. 2B*); when the leaves remains green except by the presence of the streaking of petiole and the falling of them because petiole collapse and folding) difficult to distinguish from the fallen of leaves by the effect of the wind or other causes. The unfurled leaf can frequently show some necrosis that is a symptom of *Fusarium* wilt if there is no presence of head rot by *F. moniliforme*. Eventually, all the leaves of the plants fold and die. Pseudostems can remain stand up by 1 or 2 months (*Fig. 2D*). In active growing plants it is possible to observe the splitting of the pseudostem just over the soil level (*Fig. 2C*). First symptoms of the disease occurred in hair roots that are the initial place of infection, and after it progresses to the rhizome being more accentuated in the limits of the cortex and central cylinder in the more vascularized area (*Fig. 2H*). The pathogen passes through the affected vessels to the new growing shoot (*Fig. 2I*). The most characteristic symptoms of the disease are the brown-reddish discoloration of the internal vessels of the pseudostem (*Figs. 2F* and *2G*). The oldest leaf sheaths can show brownish streaks (*Fig. 2E*). New emerging leaves can be shorter than normal and there are not internal fruit symptoms.

Fusarium wilt can be readily distinguished from Bacterial wilt by *Ralstonia solanacearum* and other bacterial wilts on banana by the absence in the former of: *a*) symptoms in young shoots of less than 4-5 months of age, *b*) of internal symptoms in the fruits and *c*) of bacterial exudates when the pseudostems of the affected plant are cut across.

Variability of *Foc* populations

It had been assumed that *Foc* originated in Asian region and subsequently dispersed to African and America as the host moving to new regions [Stover, 1962b; Ploetz, 1990b]. Edible bananas originated in Asia and are now growing in virtually all areas located between 30°N and 30°S latitudes. *Fusarium* wilt has been reported from all banana growing regions of the world except South Pacific Islands, Somalia and Mediterranean countries.

Important amount of research has contributed to a better understanding of the variability, phylogenetic relations and evolution of the pathogen using phenetic and genetic characters in the last fifteen years. Numerous methods has been used to characterize *Foc* including pathogenicity [Stover and Waite, 1960; Stover, 1962b; Su *et al*., 1977, 1986; Stover and Buddenhaguen, 1986]; vegetative compatibility [VCG; Ploetz and Correll, 1988; Ploetz, 1990a and b; Brake *et al*., 1990; Leslie, 1990 and 1993; Moore *et al*., 1993; Pegg *et al*., 1993; Hernández *et al*., 1993; Batlle and Pérez, 1999; Ploetz and Pegg, 2000], volatile aldehydes production over the space of rice cultures [Brandes 1919; Stover 1962a; Moore *et al*., 1991, Batlle and Pérez, 2003], electrophoretic karyotyping [Miao, 1990; Boehm *et al*., 1994], DNA restriction fragment polymorphism [RAPD-PCR, RFLP, AFLP analysis, Bentley and Bassam, 1996; Koenig *et al*., 1997; Bentley *et al*., 1998; Groenewald *et al*., 2004], nucleotide sequence data of nuclear and mitochondrial genes [O'Donnell *et al*., 1998].

Pathogenic races of *Foc* are not genetically defined and they are only groups of isolates that attack differential cultivars. Four races have been previously defined. Stover (1959), Waite (1953), Stover and Waite (1960) and Stover (1962a and b), reported two races on the base of pathogenicity in Manzano and Gros Michel (race 1) and to Bluggoe (race 2). Race 3 was isolated from *Heliconia* [Waite, 1963] which is currently questioned if it could be considered in the formae specialis *cubense* due to the last findings on *Heliconia* and *Musa* genetic dissimilarities. Race 1 almost destroyed banana export industry based on Gros Michel (AAA) and was the cause of the almost disappearance of the cultivar Manzano (subgroup Silk, AAB) in Cuba. Finally, race 4 attacks Cavendish cultivars and most of the cultivars that are also susceptible to races 1 and 2 [Su *et al*., 1977 and 1986; Stover and Simmonds, 1987; Stover, 1990]. Before the more recently outbreaks in Southeast Asia, Cavendish cultivars had only been attacked in subtropical production areas of Canary

Islands, South Africa, Taiwan and Australia, where the cold winter predispose Cavendish to damage that would not normally occurs [Waite, 1953; Stover and Malo, 1972]. More recently, a unique population of the pathogen VCG 01213-01216 is responsible for the affected Cavendish (Grand Nain, Williams and Valery) monocultures in tropical peninsular Malaysia and Indonesia (Sumatra, Java, Halmahera) where there are not factors of predisposition [Pegg *et al*., 1994; Pegg *et al*., 1996; Bentley *et al*., 1998]. Pisang mas (AA, syn. Sucrier), Pisang berangan (syn. Lakatan) and other cultivars which are resistant to *Fusarium* wilt in other locations are also affected in these areas.

There are evidences that there are more pathogenic races than those so far recognized and that if more differentials were used in pathogenicity tests under controlled conditions new pathotypes would be recognized [Stover and Buddenhaguen, 1986; Ploetz, 1994, Pérez *et al*., 2003 and 2004b]. Some isolates are pathogenic to Gros Michel as well as to Bluggoe en Florida [Ploetz and Bentley, 2001]. Pérez *et al*. (2004b), find out different responses of the reaction of the cultivars Pelipita and Pisang lilin to single conidia isolates of the *Foc* race 2 (pathogenic to Bluggoe) belonging to the VCG 0124 and 0128 (*Table 4*). Pisang awak has been reported susceptible to race 1 [Ploetz and Pegg, 1990] but has shown susceptibility to race 2 isolates in Cuba [Pérez *et al*., 2004b].

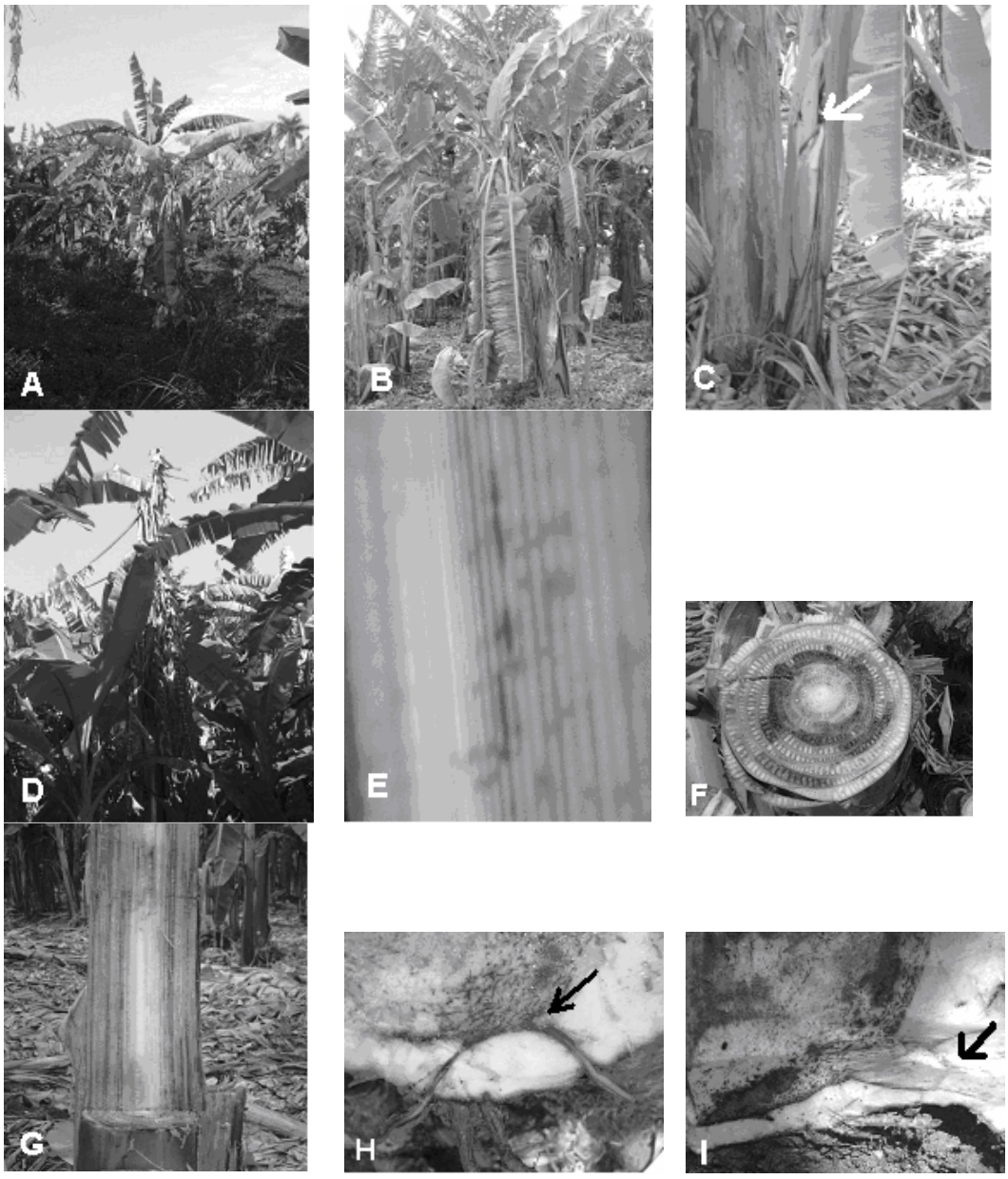

Figure 2. Fusarium wilt symptoms: A) yellow syndrome; B) non yellow syndrome; C) pseudostem splitting; D) standing died plant; E) reddish brown streaks in pseudostem sheet; F and G) decoloured vessels in pseudostem; H) decoloured vessels in secondary root and rhizome; I) decoloured vascular elements that connect mother plant and follower.

Volatile aldehyde production has been used to differentiate between isolates of *Foc* [Brandes 1919, Stover 1962b; Moore *et al*., 1991]. Brandes (1919) reports that *Foc* isolates can be grouped by the production or not of volatile aldehyde in the head space above the rice cultures [which Stover (1962a) classified as odoratum or inodoratum respectively]. Moore *et al*. (1991), found a direct relation between pathogenicity, VCG and volatile production; cultures belonging to VCG 0124, 0125 and 0128 of pathogenic races 1 and 2 do not produce volatile whereas those belonging to race 4 VCGs 0120, 129 and 01211 gave identical chromatograms peaks and speculate that the genes conferring race 4 virulence are linked to those governing volatile production. However, odoratum isolates of VCG 0126 has not been recovered from Cavendish cultivars and Cavendish cultivars has not succumbed to VCG 0126 in affected fields [Jones, 1995]. Batlle and Pérez (2003) found that Cuban *Foc* isolates of race 1 and 2 can indistinctly produce or not volatiles (*Table 1*) conforming previous reports of Brandes (1919) and Stover (1962a).

Table 1. Frequency of volatile production in 62 Cuban isolates belonging to *Foc* races 1 and 2 [Batlle and Pérez, 2003]

Race	Presence of volatile	Frequency (%)
1	+	12.8
1	–	5.2
2	+	38.5
2	–	43.5

Heterokaryon formation is a way by which normally haploid fungi can benefit from a functional diploidy as the complementation and heterosis are, and it is the first step of a parasexual cycle for transmission of characters [Leslie, 1993]. The fungi capable of developing such heterokarions are named vegetative compatible. Vegetative compatibility has resulted a useful technique to study the relationship between asexually reproducing fungi such *Foc*. The technique was developed by Cove (1976) and refined by Puhalla (1985) and Correll *et al.* (1987). Since vegetative compatibility requires that alleles at least 10 vegetative incompatibility loci (*vic loci*) be identical [Puhalla and Spieth, 1985], members of a vegetative compatibility group are usually clonally derived and close genetically related (vegetative compatibility between strains of different formae specialis has never been observed) and its unlikely that individuals that possess the same complement of VIC alleles not to be related by clonal descent. This then also indicates that two vegetative compatible individuals should also be identical for many other genes, including those that are responsible for pathogenicity, ecological adaptation and other traits that affect their roles as banana pathogens. Due to this association between vegetative compatibility and other genetically controlled traits, VCGs are strong indicators of pathogenic behaviour and are powerful tools in the study of the fungal population biology and genetics.

There have been classified at least twenty VCGs or VCG complexes (*Table 2*) in *Foc* [Ploetz y Correll, 1988; Ploetz, 1990a, b and c; Brake *et al*., 1990; Moore *et al*., 1993; Pegg *et al*., 1993]. Bentley *et al*. (1998) reported 14 new genotypes that not grouped in any of the previously known VCG.

Vegetative compatibility is useful for grouping genetically similar isolates but do not provide information on the genetic relatedness between incompatible isolates of different VCG and formae specialis. A mutation in a simple *vic* locus could result in closely related isolates being vegetative incompatible and thus, clonally related isolates may occur in different VCG. Bentley *et al*. (1998), find a few or no variation among the isolates in each VCG in independence of the host or geographic origin in an analysis of the polymorphism of the restriction fragments (RFLP) of 208 isolates of a *Foc* world collection. As the restriction patterns results almost specific for each VCG, they can predict the VCG of each isolate previous to the vegetative compatibility analysis, determining 33 genotypes and grouping the populations (with a similarity between 80 and 100%) in nine main lineages (DNA finger printing groups or DFG) concluding that the genotypes arise by simple mutation in each lineage. The world distribution of the lineages according DNA fingerprinting groups is shown in *Fig. 3*.

Foc isolates can be differentiated in two broad groups with different lineages (*Table 3*) on the base of chromosome number and amount of DNA [Boehm *et al*., 1994], volatile production [Moore *et al*., 1991]; RAPD [Koenig *et al*., 1997]; sequence analysis of mitochondrial genes [O'Donnell *et al*., 1998], DNA fingerprinting [Bentley *et al*., 1998] and AFLP analysis [Groenewald *et al*., 2004]. Most of the methods employed have a broad coincidence when grouping the isolates and minor differences arise related to specific populations of the pathogen. Different lineages have been identified which are as genetically different among them as are to other formae specialis of *Fusarium oxysporum* [Bentley *et al*., 1998]. The major

number of VCG and lineages have been found in Malaysia-Indonesia area. Based on the diversity concept (Vavilov's theory) it seems that the Malaysian-Indonesian area is the major centre of origin of *Foc.* All studies coincide to establish that *Foc* is polyphyletic. The results indicate that the races 1 and 2 have evolved together whereas races 3 and 4 are both of separate origin; while most of the *Foc* lineages have probably coevolved on Southeast Asia; several lineages have probably arisen independently.

Table 2. VCGs in *Foc* and world distribution

VCG (1)	VCG complexes	Distribution by countries (2)
0120	0120-01215	Australia, Brazil, Costa Rica, Canary Islands, Guadeloupe, Honduras, Indonesia, Jamaica, Malaysia, South Africa, Taiwan
0121	None	Indonesia, Malaysia, Taiwan
0122	None	Philippines
0123	0123 a-0123 b	Philippines, Indonesia, Malaysia, Thailand, Taiwan, Viet-Nam
0124	0124-0125-0128-01220	Australia, Burundi, Brazil, Cuba, EUA, Honduras, India, Jamaica, Kenya, Malaysia, Malawi, Nicaragua, Philippines, Thailand, Uganda, Tanzania, Viet Nam
0125	0124-0125-0128-01220	Australia, Brazil, Jamaica, Honduras, India, Kenya, Malaysia, Philippines,Thailand, Uganda, Zaire
0124/	0124-0125-0128-01220	Australia, Brazil, Cuba, EUA, Honduras, India, Indonesia, Jamaica
0125		Kenya, Malaysia, Malawi, Nicaragua, Philippines, Thailand, Uganda, Viet Nam
0126	None	Honduras, Indonesia (Papua New Guinea?), Philippines
0128	0124-0125-0128-1220	Australia, Commodes Islands, Cuba, Kenya, India, Thailand
0129	None	Australia
01210	None	Cuba, USA (Florida)
01211	None	Australia
01212	None	Kenya, Tanzania, Uganda
01213	01213-01216	Australia, Indonesia, Malaysia
01214	None	Malawi
01215	0120-01215	Costa Rica, Indonesia, Malaysia
01216	01213-01216	Australia, Malaysia, Indonesia
01217	None	Malaysia
01218	None	Indonesia, Malaysia, Philippines, Thailand
01219	None	Indonesia
01220	0124-0125-0128-1220	Australia, India, Kenya, Thailand
01221		Thailand
01222	None	India, Kenya, Uganda
1		Australia (*Heliconia chartacea*)
2		Indonesia
3		Indonesia
4		Malaysia
5		Indonesia, Malaysia, Thailand
6		Mexico
7		Philippines
8		Philippines, Thailand
9		Philippines
10		Philippines
11		Philippines
12		Thailand, Viet Nam
13		Viet Nam
14		Viet Nam
15		Indonesia
16		Indonesia, Malaysia

(1) The VCG with the numbers from 1-16 do not match with any of the precedent VCG and can be considered new VCG according Bentley *et al*. (1998).

(2) Data of Ploetz (1990a and c), Bentley *et al*. (1998); Battle and Pérez, (1999); Magnaye, (1999); Singburaudom, (1999); Rutherford, (1999); Kangire and Tushemereirwe (2003), Viljoen *et al*. (2003), Thangavelu *et al*. (2003), Masdek *et al*. (2003).

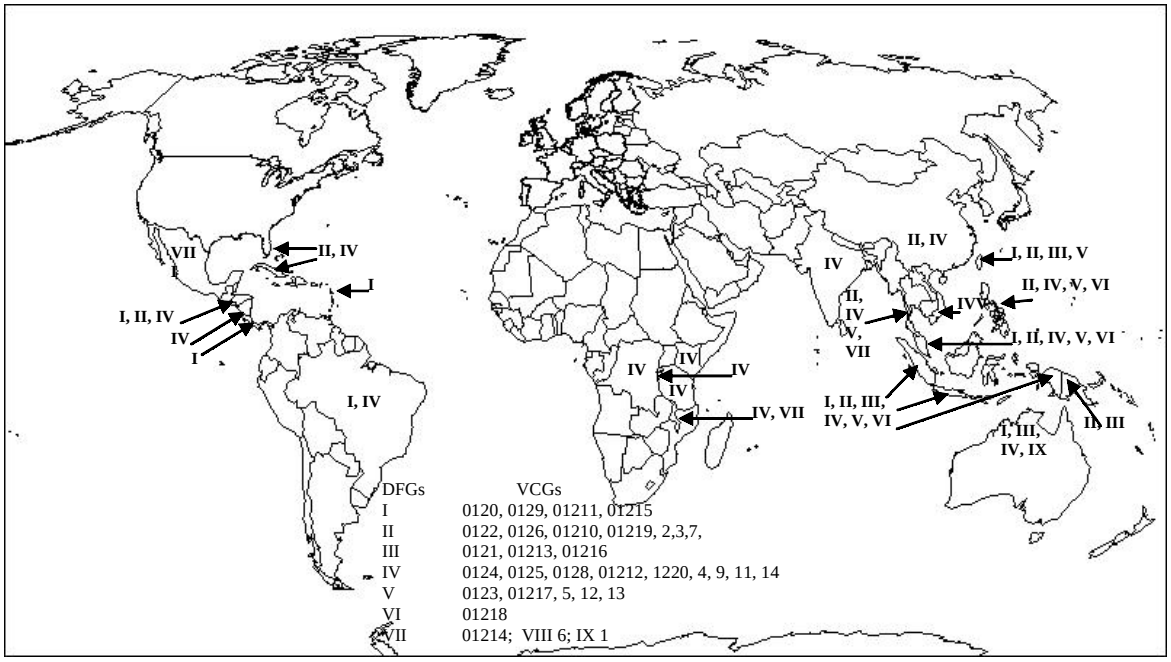

Figure 3. World distribution of *Foc* lineages according DNA fingerprinting groups (DFGs, modified from Bentley *et al*., 1998; the list of VCG and countries is in *Table 2*).

Table 3. Broad grouping of *Fusarium oxysporum f.* sp. *cubense* isolates according various methods of characterization

Method	Groupings	
	I	II
Current race classification [Stover, 1962b; Su *et al*., 1977, 1986]	Race 4	Races 1 and 2
Cultural characteristic in K2 media [Sun *et al*., 1978]	Lacinia formation	Not lacinia formation
Vegetative compatibility [Ploetz, 1990a y b; Brake *et al*., 1990; Pegg *et al*., 1993; Moore *et al*., 1993]	0120, 0121, 0122, 0126, 0129, 01211, 01213, 01215, 01216, 01219	0123, 0124, 0125, 0128, 01210*, 01212, 01214, 01217, 01218, 01220**.
Volatile production (Moore, 1994).	Odoratum	Inodoratum (1)
RAPD-PCR [Sorensen, *et al*., 1993; Sorensen *et al*., 1994]	RAPD-PCR group 1	RAPD-PCR group 2
Electrophoretic karyotyping [Boehm *et al*., 1994]	Ek I (high chromosome number; large genome size)	Ek II (fewer chromosome number; small genome size)
Pectic enzyme analysis [Pegg *et al*., 1994]	Slow moving Pectic zymogram group	Fast moving Pectic zymogram group
RFLP [Koenig *et al*., 1997]	Clonal lineages II, III, IV, V, VI, VII, IX, X (VCGs, 0120, 0122, 0123, 0126, 0129, 01211, 01214***, 01215)	Clonal lineages I and VIII (VCGs 0124, 0124/0125, 0125, 0128, 01212)
DNA finger printing genotypes [Bentley *et al*., 1998]	Clonal lineages I, II, III, VIII and IX	Clonal lineages IV, V, VI, VII
AFLP analysis [Groenewald *et al*., 2004]	Group 1 (VCGs 0120, 0120/15, 0126, 0129, 01213, 01213/16, 01216, 01219	Group 2 (VCGs 0123, 0124, 0125, 01217, 1218)

* VCG 01210 is present only in Florida and Cuba attacking Manzano and Gros Michel and is probably a VCG which evolved independently.

** VCG 1220 is a population from Western Australia attacking Cavendish but more closely genetically related to race 1.

***VCG 01214 found only in Malawi not present in Asia, is genetically distantly from other lineages and probably has evolved independently in Africa or by funding effect [Koenig *et al*., 1997].

(1) Not valid for Cuban populations of race 1 and 2 in which indistinctly can be found odoratum and inodoratum isolates [Batlle and Pérez, 2003].

In conclusion, most genotypes of *Foc* have probably co-evolved with banana in Asia and have been spread from its centre of origin by the movement of banana plants. Several genotypes however, have also probably arisen independently in different regions of the world (as is the case of VCGs 01210 in Cuba, 01212 in Tanzania, 01214 in Malawi and 01211 in Australia).

Infection, epidemiology and disease cycle

The new introductions of *Foc* to disease free areas has been due to the movement of rhizomes and infected plants product basically to the human activity.

The pathogen can remain immobile in soil in diseased tissues as chlamydospores which are stimulated to germinate by host or non-host root exudates or by the contact with susceptible healthy tissue [Stover, 1972]. Mycelia and conidia are produced after 6-8 hours of chlamydospore germination and new chlamydospores after 2-3 days. Infection takes place through secondary or tertiary feeder roots but not through main root, unless there is exposition of the central core [Trujillo, 1963]. Most of the infections are blocked but some of them become systemic passing through rhizome and pseudostem. Even in presence of high amount of inoculum the plant can not be invaded through rhizome or pseudostem. The pathogen passes to the vascular zone of rhizome in the places of insertion of the diseased roots. The pathogen move out of the vascular system to the adjacent parenchyma in the advanced stages of the disease forming conidia and chlamydospores which are released to the soil when the plant die, lasting dormant by several years [Stover, 1962c].

Foc presents a minor competitive ability than other common fungal species in the soil as are *F. solani*, *F. pallidoroseum, Rhizoctonia* sp. y *Pythium* sp. [Stover and Waite, 1954; Stover, 1962c; Trujillo and Snyder, 1963] and do not spread by itself in soil by vegetative growing [Trujillo and Snyder, 1963]. However, in spite of being a poorer competitor than other fungal species of soil, due to the more random distribution of chlamydospores in those places where the disease has been present, it is able to colonize substrates of roots and leaves increasing its saprophytic growth and lasting in the soil by many years. The disease cycle is repeated when chlamydospores germinates and growth saprophytically in plant debris or by host invasion. There are no forms of dispersal outside the plants. The disease is then transmitted by infected rhizomes and mechanical movement of soil in machinery residues, superficial running water and periodic inundations due to rivers outflows. An interesting point for a future research is the study of the status of the populations of *Foc* in Central America and Caribbean soils of farms planted of cv. Gros Michel and destroyed by *Fusarium* wilt after 40 years of Cavendish cultivation.

Foc is essentially confined to the xylem elements. The parenchyma that surround the vascular tissue usually dies previously to fungal invasion and are invaded by the hyphen that last densely packed in the lumen of these cells [Trujillo, 1963]. The pathogen multiplies abundantly (by budding as yeasts) in the xylem of affected plants. Some of the conidia are small enough to pass through the plaques of xylem. When a vessel is colonized conidia are produced in the next 2-3 days at the outside of the vessel allowing moving to a new vessel. Host react to infection trying to develop a gel wall over and added to plaque that impedes the fungal advance [Beckman, 1964, 1990; Beckman and Halmos, 1962; Beckman *et al*., 1962]. In Gros Michel the colonization process occurs without limitation meanwhile in Cavendish cultivars the colonization is stopped by gel accumulation in the first 24-48 hours followed by development of a vascular parenchyma that impedes any further colonization. De Ascensao and Dubery (2000), reported that cortical tissue of the resistant cultivar Goldfinger (FHIA 01), react to elicitation by *Foc* race 4, with a strong lignin deposition whereas in the susceptible cultivar Williams this process is weak.

Alternative hosts

Foc can invade the weed roots in banana plantations as saprophyte or as a weak parasite of the tissues of senescent roots in decomposition remaining in soil by long periods. There are reports of the isolations of *Foc* from roots of the weeds *Euphorbia heterophylla* L. (*Euphorbiaceae*), *Tridax procumbens* L. (*Poaceae), Chloris inflata* (Link.) and *Cyanthillium cinereum* L. (Asteraceae) [Waite y Dunlap, 1953]; *Cyperus iria* L., *Cyperus rotundus* L., *Gnaphalium purpureum* L., *Fimbristylis koidzuminana* Ohwi [Su *et al*., 1986] and from decolorated roots without wilting of the species *Paspalum fasciculatum* Sw., *Panicum purpurascens* (Roddi.), *Ixophorus unisetus* Schl., and *Commelina* spp. [Podovan, 2003].

Reaction of cultivars

Much of the current knowledge on the reaction of banana cultivars to *Foc* infection has been achieved by the results of *Fusarium* trials of the *Musa* Testing Program organized by INIBAP and has been in a general way summarized by Ploetz and Pegg (2000). The results of artificial inoculations of the *Foc* Cuban populations belonging to VCG 01210 (race 1), 0124 and 0128 (race 2) are shown in *Table 4* [Pérez *et al*., 2003, 2004b].

The reports of the reaction ratings for different cultivars in different countries are variable in dependence of the population of *Foc*, the pressure of infection, soil temperature [Trujillo, 1963] and the nature of the material used in the test with artificial inoculations (tissue culture plants are more susceptible than conventional propagation materials [Smith *et al*., 1998]. These factors should be taken into account when comparing results of trials.

Hwang *et al*. (1992) and Ho *et al*. (2001), obtain somaclonal resistant plants from Giant Cavendish to *Foc* race 4 in Taiwan and Malaysia in tissue culture. Herrera

et al. (1999) and Bermúdez *et al*. (2002) obtained resistant mutants from Gros Michel tissue culture plants by induction of mutation with gamma particles bombardment and screening by repetitive inoculations with *Foc* in laboratory and field.

Early screening has been a main objective in the selection of resistant plants against different formae specialis of *F. oxysporum* in conventional and biotechnological breeding. Crude extracts and fusaric acid from Foc cultures has been used in the selection of resistant plants [Bacon *et al.*, 1996; Matsumoto *et al*., 1995 and 1999; Campanioni *et al*., 2003], etc.

Disease management

Use of resistant cultivars. The use of resistant genotypes has proved to be the main measure of control.

Cultural and quarantine measures. In disease free areas preventative and quarantine procedures should be implemented to avoid the entry of the pathogen. New genotypes should be introduced in tissue culture from certified healthy sources.

The equipments employed in infected areas should not be used in free areas unless it has been carefully washed and disinfected.

Sanitation and destruction of diseased plants and fields should be carried out taking into consideration that the cutting and spreading of plant residues could contribute to the dispersal of the pathogen chlamydospores in soil and to the dispersal of the disease.

Chemical control. There are not effective measures of chemical control. Fumigated soils with methyl bromide can be re-infected in two or three years in fields with susceptible cultivars.

Use of antagonists. Mid and long term perspectives. There are not published papers on the practical use of antagonist in scientific bibliography. Most of the reports concern to greenhouse and laboratory results not confirmed or repetitive at the field. Disease suppressive soils are associated to high populations of actinomycetes and bacteria whereas the conducible ones present higher populations of filamentous fungi and yeasts [Peng *et al*. 1999]. The suppressiveness of soils are based on microbial interactions being fluorescent *Pseudomonas* and non-pathogenic *F. oxysporum* the main species involved in the competence for nutrients (specially carbon and iron), competence for colonization at the site of infection and the induction of resistance in the plants [Lemanceau *et al*., 1988].

Most of the ongoing research on the biocontrol of *Fusarium* wilt on banana has been directed to the reduction of the inoculum in soil and the use of endophyte antagonists.

Mitov and Oliva (1975) reported the efficacy of the antagonist *T. lignorum* (now *T. harzianum*) and the reduction of infection by *Foc* in Bluggoe plants treated with the antagonist previous to inoculation with *Foc.* Similar results were reported by Perez *et al*. (2003 and 2004a). Treatments with *T. harzianum* A34, allowed keeping plantations of Burro CEMSA during five years in production in conducible soils of fields previously destroyed by the disease.

Table 4. Reaction of cultivars to artificial inoculation with different races and VCG of Cuban populations of *Foc* [Pérez *et al*., 2003]

Cultivars	Frequency of diseased plants (%)			Mean severity (*)		
	01210 (1)**	0124 (2)	0128 (2)	01210 (1)	0124 (2)	0128 (2)
Manzano	60,0	0,0	0,0	4,6	1,0	1,0
Gros Michel	100,0	0,0	0,0	5,0	1,0	1,0
Burro criollo	0,0	60,0	40,0	1,0	4,0	3,8
Pelipita	0,0	0,0	100	1,0	1,0	5,0
Pisang awak	25,0	0,0	33,3	2,4	4,3	4,5
Musa acuminata 2	66,6	33,3	33,3	1,8	1,3	4,3
Pisang Lilin	25,0	0,0	25,0	4,7	2,6	4,3
Calcutta 4	0,0	33,3	25,0	4,5	2,3	2,2
Pisang jari buaya	20,0	40,0	20,0	1,6	3,6	3,2
Paka	66,6	0,0	0,0	4,3	5,0	5,0
Yangambi Km 5	100	33,3	33,3	5,0	3,3	4,0
FHIA 02	0,0	0,0	0,0	0,0	0,0	0,0
FHIA 03	20,0	60,0	0,0	1,8	2,4	3,6
FHIA 04	0,0	0,0	0,0	0,0	0,0	0,0
FHIA 18	20,0	0,0	0,0	1,8	0,0	1,6
FHIA 21	0	20,0	0,0	0,0	2,6	1,0

* Scale from 0 (healthy to 5 dead). ** Races in parenthesis.

Some research needs

A great advance has been achieved in the last 15 years on *Fusarium* wilt and its causal agent *Fusarium oxysporum f.* sp. *cubense*. However there still some black holes and research needs:

1. A considerable amount of information has been obtained from the diversity of strains of *Foc* in Southeast Asia (particularly from Thailand, Malaysia and Indonesia) Australia, some countries of Central America (Honduras and Costa Rica) and more recently from Cuba. However there is a relatively little information on this issue from China, Viet Nam, and Northern India most of the countries from the Caribbean basin (where a relative few isolates has been studied) and there is almost a complete lack of data from South America (Indians went to Malaya, Fiji, Caribbean Island and Guiana to work in sugarcane and as traders and later to East Africa from Western India; with them went Bluggoe, Pisang awak Silk and Ney Poovan. Portuguese's Spaniels and Dutch from Asia-Pacific and Africa to Central, South America and the Caribbean).

2.Even when there is a more comprehensive understanding of the variability of *Foc* populations since its grouping in VCGs and lineages, the classification based on pathogenicity need further research and must be considered in the future for practical purposes of disease management and breeding.

3. There is still the need of developing inoculation and testing procedures.

4. There is a lack of published scientific research work on biocontrol, particularly with practical field results. There is also a lack of negative results published. *Foc* inoculum remains as chlamydospore in the soil and there is an opportunity for soil antagonist as *Trichoderma harzianum* to colonize and reduce the population of Foc low by antibiosis or direct parasitism. At the same time there is a lack of information on the ecology of the antagonists in the rhizoplane of *Musa* plants as well as the resistance response to potentially pathogenic *Foc* population of plants previously challenged with non pathogenic *Fusarium oxysporum*.

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