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Workshop
Fusarium Wilt: The Banana Disease that Refuses to Go Away

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Abstract

Fusarium wilt, also known as Panama disease, is the most important lethal disease of banana. This workshop began with a summary of the early history of, and research on, this important disease. Its current status and recent developments were then highlighted for different regions. The causal soilborne fungus, *Fusarium oxysporum* f. sp. *cubense* (Foc), is found in most banana-producing regions and is phylogenetically diverse. Pathotypes ("races") that affect different groups of cultivars have been useful for describing host reactions and new disease outbreaks, even though races 1 and 2 are heterogeneous (i.e. numerous clonal lineages and vegetative compatibility groups or VCGs have been identified). In contrast, populations of the pathogen that affect the Cavendish subgroup are fairly well defined and are separated into subtropical race 4 (SR4) (VCG 0120 and related VCGs) and tropical race 4 (TR4) (VCG 01213-01216). Although SR4 and TR4 affect many of the same cultivars, they interact significantly with temperature: TR4 kills plants in the tropics, but SR4 only affects plants that have been predisposed to disease by cold temperatures (in areas with pronounced winters). TR4 is decimating Cavendish monocultures in southern Asia and would affect 85% of global production were it disseminated more widely. Measures are needed to diagnose, interdict and manage TR4 and other variants of Foc worldwide. To that end, recent research to interdict, understand and manage this disease was discussed. The workshop concluded with an open forum/discussion on results that were presented during the symposium and on key issues and data gaps that should be addressed by future work.

INTRODUCTION

Fusarium wilt (Panama disease) is the most significant vascular wilt disease of banana (Ploetz, 1990; Ploetz and Pegg, 2000; Stover, 1962). Simmonds (1966) ranked it as one of the six most destructive plant diseases. This notorious reputation stems from damage it caused in the first export trades, which were based on 'Gros Michel' (AAA). Extreme susceptibility to Fusarium wilt, the use of infected suckers to establish new plantings and the practice of monoculture doomed 'Gros Michel' and led to its eventual replacement by the Cavendish clones. Interested readers are referred to Stover's (1962) monograph for a comprehensive early history of the disease, and to a recent retrospective on the 'Gros Michel' epidemics and on the political intrigue and social upheaval that surrounded the early trades (Ploetz, 2005).

Fusarium wilt was first reported in Australia. Bancroft (1876) speculated that a fungus caused the disease and recognised that suckers that looked healthy could harbour the disease. Smith (1910) was the first to isolate the pathogen, which he called *Fusarium cubense*, from banana, and Brandes (1919) was the first to complete Koch's postulates. During the ensuing decades, almost all first reports of the disease in an area were on 'Gros Michel' (Stover, 1962). Wollenweber and Reinking (1935) recognised that *F. cubense* was a variant of the common soilborne fungus, *Fusarium oxysporum*. Soon after, their *F. oxysporum* var. *cubense* was renamed *F. oxysporum* f. sp. *cubense* (Foc) by Snyder and Hansen (1940). Although Snyder and Hansen's name is still used to designate this pathogen, research over the last decade indicates that it is represented by at least three phylogenetically distinct taxa (Fourie et al., 2009; Koenig et al., 1997; O'Donnell et al., 1998; Ploetz, 2006; Taylor et al., 1999). As is discussed below, the assignment of unique names to these phylospecies is warranted and should be considered to distinguish these significantly different fungi.

As the 'Gros Michel' era ended, Stover (1962): i) contributed new insights on the taxonomy, variation and physiology of Foc; ii) helped describe Foc's interaction with banana; iii) characterised resistance and susceptibility in banana to this disease; iv) studied the influence of edaphic factors on the pathogen and introduced flood fallowing as a means for cleansing contaminated soil. In several areas, Stover's results have been superseded by newer work, often with tools that were not available during his career. However, several of his conclusions have been corroborated, including: 1) when they are available, resistant genotypes of banana are the best tools for managing this disease (Jones, 1994; Ploetz and Pegg, 2000); 2) the impact of effective soil treatments, such as flood fallow, are at best temporary (Herbert and Marx, 1990); and 3) with only one, clear exception, Foc appears to have coevolved with its banana host in Southeast Asia (Ploetz, 2006a,b; Ploetz and Pegg, 1997); Simmond's (1966) hypothesis that strains of Foc could arise (be selected) from the native mycoflora in a new site have not been supported with experimental evidence.

The transition by the trades from 'Gros Michel' to the Cavendish cultivars coincided with a dramatic reduction in the amount and types of research that were done on Fusarium wilt (Ploetz, 1990). Directly and indirectly, the disease changed the ways in which this multinational business produced and marketed its product. In addition to the massive replanting effort that this entailed, Cavendish fruit required different, far gentler measures for harvesting, handling and shipping than were used for 'Gros Michel'.

The above focus on export bananas drew attention away from the impact of Fusarium wilt outside the trades (Ploetz, 1994). Diverse clones are attacked, many of which are as susceptible as 'Gros Michel' (Ploetz and Pegg, 2000). Although these cultivars are still produced in diversified, multicropped situations, their intensive, monoculture production usually collapses shortly after it begins (Ploetz, 2006b). For example, only temporary production of 'Silk' (AAB) is possible in Brazil, Colombia, Venezuela and other areas in tropical America, and continued production of this cultivar relies on new, virgin sites, as was required by the 'Gros Michel'-based trades.

Since the Cavendish cultivars had been grown successfully for decades in the same soils that were used to produce 'Gros Michel', it was presumed that future problems with Fusarium wilt would be restricted to the above cultivars, at least in the Americas (Buddenhagen, 1990). The illusion that the Cavendish clones were generally immune to Fusarium wilt in the tropics was shattered in the early 1990s. Serious outbreaks of Fusarium wilt developed in Sumatra and peninsular Malaysia in Cavendish plantations

that had been planted for Middle Eastern and Japanese markets (Ploetz, 2006b). In these and now several other locations in Southeast Asia, Cavendish has proven to be as susceptible as was 'Gros Michel' decades before in the American epidemics.

A new variant of Foc, tropical race 4 (TR4), is responsible for the Southeast Asian outbreaks (Ploetz, 2006a,b, 2009). Unlike subtropical outbreaks that affect cold-stressed Cavendish in Australia, the Canary Islands and South Africa, TR4 affects Cavendish in the absence of predisposing factors. Although it is found only in Southeast Asia, TR4 continues to spread in that region. It represents the worst threat to sustainable banana production worldwide and, due to its wide host range, would impact 85% of the world's banana production if it were widely spread (Ploetz, 2009). The great fear is that TR4 would devastate export bananas and bananas grown by smallholders if it spread to the Americas and Africa. TR4 poses a serious threat to a multibillion dollar industry and the food stability and income of millions of poor farmers.

THE PATHOGEN

Fusarium oxysporum is a species complex of morphologically similar filamentous fungi (O'Donnell et al., 2009). It is comprised of mainly saprophytic strains, but also contains plant pathogens that cause vascular wilts, rots and damping off of hundreds of host species (Baayen et al., 2000; Domsch et al., 1980; Nelson et al., 1983). Agriculturally and economically, it is the most important taxon in *Fusarium*. Over 150 special forms, formae speciales, of *F. oxysporum* are known (Baayen et al., 2000; O'Donnell et al., 2009). Each forma specialis has a unique host range of one or a usually closely related set of species.

Foc affects primarily banana and plantain, but other banana relatives are also susceptible (Ploetz, 2006b). Abaca, *Musa textilis*, source of an important cordage fibre, suffered considerable commercial losses before production ceased in Central America in 1956 (Waite, 1954). *Musa schizocarpa* is a parent of rare bananas in Papua New Guinea, whereas enset, *Ensete ventricosum*, an important food crop in Ethiopia, is affected experimentally by an unusual member of Foc. Waite (1963) reported that several species of *Heliconia* were affected in Central and South America in the mid-1900s; he named the causal strains race 3 of Foc. Unfortunately, Waite did not deposit strains of race 3 in culture collections and wilted heliconias have not been found in the Americas since his original report.

Diverse phenetic and genetic characters have been used to study variation in Foc (Boehm et al., 1994; Groenwald et al., 2006; Moore et al., 1993; Stover, 1962). Significant progress to understand the population biology of Foc resulted after the development of a straightforward method for determining vegetative compatibility groups (VCGs) in the late 1980s (Ploetz and Correll, 1988; Puhalla, 1985), and over 20 VCGs of Foc have been reported to date (Ploetz and Pegg, 2000).

The phylogenies of these populations have been investigated with cDNA and DNA sequence work (Fourie et al., 2009; Koenig et al. 1997; O'Donnell et al. 1998; Ploetz 2006; Taylor et al. 1999). These studies: i) identified several clonal lineages within the taxon; ii) delineated relationships within Foc and among the Foc VCGs; iii) demonstrated that some of the clonal lineages of Foc were more closely related to other formae speciales of *F. oxysporum* than to other lineages of Foc; iv) suggested that one of the lineages, defined by VCG 01214, may have evolved in Africa (outside the Southeast Asian homeland of banana); and v) indicated that recombination between VCGs

0124/0125 and 01212 probably occurred (it is unclear whether this is historic or ongoing, and whether sexual or parasexual mechanisms have been involved).

The significant genetic differences that are evident among the VCG 01214, VCG 0120 and VCG 0124 clades indicate that they are phylospecies, genetically related groups of individuals that are related by descent. Thus, it is phylogenetically misleading to refer to these individuals with the same name (Ploetz, 2006a). Assigning unique names to each of these monophyletic groups is warranted.

Foc Races

"Race" has been used to classify strains of Foc since the mid 1900s (Stover, 1962). Pathotypes ("races") that affect different groups of banana cultivars are not nearly as well defined as are races of some other plant pathogens (pathosystems in which a gene-for-gene interaction are known provide the best examples). Races 1 and 2 of Foc are heterogeneous, and numerous clonal lineages and VCGs have been identified in each (Ploetz and Pegg, 2000). Furthermore, the extent to which the variable responses of different banana genotypes to Fusarium wilt has been due to differences in the pathogen and differences in the screening environment are unknown. Stover and Buddenhagen (1986) discussed ambiguity in Foc races and the poorly understood or unknown nature of environmental and genetic factors that impact the disease response.

Although they are imperfect measures of pathogenic diversity in Foc, race designations are, nonetheless, useful when describing host reactions and new disease outbreaks. Three races of Foc are conventionally recognised on banana: 1, 2 and 4 [race 3 was described for isolates of Foc that affected *Heliconia* spp. (Waite, 1963)]. Below are listed suscepts that have been reported for each race.

Race 1. Race 1 was responsible for the epidemics on 'Gros Michel' and also affects 'I.C.2' (AAAA), 'Silk', 'Pome' (AAB), 'Pisang Awak' (ABB) and 'Maqueno' (AAB). 'I.C.2' was a hybrid produced by the first banana-breeding programme, located at the International College of Tropical Agriculture in Trinidad (Shepherd, 1974). Named 'Golden Beauty' when it was released in 1928, 'I.C.2' was developed as a Fusarium wilt-resistant replacement for 'Gros Michel'. Its subsequent susceptibility in Honduras is recognised as a failure to account for pathogenic variation in Foc during the screening process (Stover and Buddenhagen, 1986). Due to its exceptional flavour, 'Silk' may be the most esteemed of all dessert bananas. Its extreme susceptibility to race 1 has eliminated it in much of the Western Hemisphere. Significant commercial production was recently decimated in Venezuela and the cultivar is now uncommon in Brazil, one of the world's leading banana producers. 'Pome' is less susceptible than 'Silk' to race 1, but is affected nonetheless. 'Pisang Awak' is one of the hardiest and most widely distributed bananas worldwide. Over 70% of the bananas that are produced in Thailand are of this clone, and it has gained wide acceptance in East Africa due to its use as a beer banana and for its drought tolerance. In these and other areas, the clone is very susceptible to race 1. 'Maqueno' is a member of the Maia Maoli-Popo'ulu subgroup, which is most prevalent in the Pacific. It has been used as a female parent in the *Fundación Hondurena de Investigación Agrícola* (FHIA) breeding programme, but is very susceptible to race 1. Recently, Fusarium wilt began to move into the Pacific, a region that had been generally free of the disease (G. Wall, Univ. Guam, pers. commun.). As Fusarium wilt spreads in the Pacific, it threatens 'Maqueno' and other cultivars in the Maia Maoli-Popo'ulu subgroup.

Race 2. Race 2 affects cooking bananas, especially those in the Bluggoe subgroup (ABB). The most important cultivar in this group, 'Bluggoe', was widely planted in Latin America before the spread of race 2 and Moko disease, caused by *Ralstonia solanacearum*. It is also important in East Africa. 'Bodles Altafort' (AAAA) is a race 1-resistant hybrid between 'Gros Michel' and 'Pisang Lilin' (AA) that was developed in Jamaica. It succumbed to race 2 when it was deployed to India (Shepherd, 1974).

Race 4. Race 4 affects race 1- and race 2-suscepts, cultivars in the Cavendish subgroup and diverse additional cultivars, such as 'Pisang Mas' (AA) (Ploetz and Pegg, 2000). The Cavendish cultivars are affected mainly in the eastern subtropics. For decades, losses have occurred in subtropical Australia (New South Wales and Queensland), the Canary and Madeira Islands and South Africa (Natal and Transvaal) (Ploetz, 1990). In these areas, race 4 is comprised of isolates in VCG 0120-01215, and to a lesser extent in Australia, VCGs 0129 and 01211 (Ploetz and Pegg, 2000). Cavendish is also affected in Taiwan (Hwang and Ko, 2004). Previous reports indicated that this was another subtropical example of race 4, even though production areas in Taiwan have relatively warm winters and are technically in the tropics (Kaohsiung, 22ºS latitude, is south of the Tropic of Cancer) (Ploetz, 2006b). In hindsight, damage in Taiwan may be due to TR4. Cold winter temperatures in the subtropics are thought to predispose Cavendish to race 4. For example, photosynthesis (carbon assimilation, A, as μmol CO_2 $m^{-2}s^{-1}$) of a Cavendish cultivar was reduced 75% during the winter in Queensland, Australia (Moore et al., 1993). Although damage occurs on Cavendish in the tropics, it is uncommon and also associated with predisposing factors (Ploetz, 1994). Wilt pockets occurred in Guadeloupe (17ºS) where ash from a volcanic eruption lowered soil pH, and in Jamaica (18ºS) in low-lying and poorly drained soils above 700 m in elevation. In summary, until recently, Cavendish succumbed in exceptional situations but performed well in good soils in the lowland tropics. Since these cultivars had resisted Fusarium wilt for decades in the same soils in which 'Gros Michel' was devastated, it appeared that the 'Gros Michel' populations of Foc were incapable of mutating to virulence on Cavendish (Buddenhagen, 1990).

The Special Threat Posed By Tropical Race 4 (TR4). The recent outbreaks of TR4 have been surprising and disconcerting. Isolates of TR4 are in VCG 01213-01216, a unique population that was originally identified in Taiwan and is now known to define TR4 (Ploetz, 2006a,b). TR4 has also been reported in Australia (Northern Territory), China (Hainan, Guangdong, Guangxi), Indonesia (Halmahera, Irian Jaya, Java, Sulawesi and Sumatra), Malaysia (Peninsular and Sarawak) and the Philippines (Molina, 2009). The role that 01213-01216 plays in the Taiwanese epidemics should be examined. Although the distribution and prevalence of different Foc VCGs in Taiwan has not been determined [three other VCGs exist there, 0120-01215, 0121 and 0123 (Ploetz and Pegg, 2000)], the climate and the importance of Fusarium wilt on Cavendish on the island suggest that 01213-01216 may be most significant. A closer examination of the situation could shed light on resistance that has been developed in Taiwan among somaclonal variants of 'Giant Cavendish', the so-called 'Giant Cavendish tissue-culture variants' (GCTCVs) (Hwang and Ko, 2004). To date, it is not known which VCGs predominate on Cavendish in Taiwan or what VCGs impact the GCTCVs.

TR4 is distinguished from subtropical race 4 because it is genetically distinct and damages Cavendish in the tropics (Ploetz, 2006b). Research is needed on the host ranges of the two pathotypes. Although they are similar, some clones, such as 'Pisang Lilin', are affected only by TR4, and the reaction of other clones that are affected in the tropics, such

as ‘Pisang Berangan’ (AAA), is not known in the subtropics since they are not produced there. The tropical response of other clones that are affected in the subtropics is also not known [e.g. ‘Yangambi Km 5’ (AAA), ‘FHIA-03’ (AABB) and ‘FHIA-23’ (AAAA)].

Given the importance of TR4, the desire to keep the pathogen out of noninfested production areas and the need to detect early outbreaks, recent research has focused on rapid and reliable diagnostic procedures (Groenwald et al., 2006; Lin et al., 2009). This research, and a new multiplex PCR diagnostic specific for TR4, were reported at this meeting (Waalwijk et al., 2009) and discussed during the workshop. Details of the TR4-specific diagnostic methodology have since been published (Dita et al., 2010). Obviously, keeping TR4 out of noninfested areas is a high priority. Action plans, similar to the one that has been developed for tropical America (Pocasangre et al., 2009), should be developed for Africa and areas in Asia that remain free of TR4. Where TR4 is established, it will be necessary to institute new, holistic measures and to accept some losses due to this disease. In addition, using more tolerant cultivars (which are, admittedly, limited in number), single production cycles (treating the crop as an annual rather than perennial), intercropping, fallowing with nonhost crops, flooding and the use of silicon or other soil amendments may be needed. Research is needed to identify which measures are most effective.

Are There Additional Races Of Foc? Pathogenic diversity is not adequately defined with the current races of Foc (race 1 affects ‘Gros Michel’, race 2 ‘Bluggoe’ and race 4 ‘Gros Michel’, ‘Bluggoe’ and Cavendish) (Ploetz and Pegg, 2000). For example, isolates from East Africa and Florida affect ‘Gros Michel’ and ‘Bluggoe’, but not Cavendish (Ploetz, unpublished; Stover, pers. commun.). New races might also be added as new susceptible cultivars are identified. For example, ‘Hua Moa’ (AAB), a cultivar in the Maia Maoli-Popo’ulu subgroup, succumbed in recent field trials in Florida (Ploetz et al., 1999).

ADDITIONAL RESEARCH NEEDS AND ACTION PLANS

Research is needed on the basic biology, population biology and epidemiology of this pathosystem. For example, the influence of edaphic and other environmental conditions on the outbreak and development of this disease is incompletely understood (Stover and Buddenhagen, 1986). In addition, the epidemiology of the TR4 outbreaks is confusing. The Cavendish epidemics have developed in plantations that were established with tissue-culture plantlets (supposedly pathogen free) and in areas without a recent history of banana cultivation. Relevant questions are: 1) How long can the pathogen survive in the absence of a banana host?; 2) What alternative, non-banana hosts are present in these production areas, and what is their distribution and impact?; and 3) If the tissue-culture plantlets were pathogen free, how was the pathogen moved to these production areas? What is the potential role of insect vectors (if any) and aerial dissemination?

The interactions between host, pathogen and environment need to be better understood. Two research groups announced ongoing efforts towards transcriptome and genome sequencing of multiple races of Fusarium wilt pathogens. This work will be instrumental in facilitating opportunities to increase our knowledge of Fusarium-banana interactions. Host-pathogen screening should be expanded, including testing of closely related resistant and susceptible germplasm. There is also a need to prospect for new, potentially resistant germplasm, and to characterise the pathogenic and genetic variability of Fusarium wilt races, clonal lineages and VCGs.

The development of a standardised small-plant (tissue-culture) screening bioassay that can be adapted to local conditions as needed was reported (Dita et al., 2009), and details of the methodology were recently published (Dita et al., 2010). This is considered an important starting point to accurately and reliably assess host response and pathogen virulence (useful for wild-type and mutant strains) and facilitate comparisons across different geographical locations. Validation of such small-plant bioassays conducted under controlled conditions is needed to demonstrate that disease reactions are representative of field trials. Although most experiments and reports on this objective focus on the types of inoculum that are used, other factors in the screening assay are more important (Ploetz, 1989). These include: 1) high light intensity, 2) an inert potting medium (e.g., silica sand or Perlite), 3) uniform, healthy plantlets with actively growing root systems, 4) temperature control (maintenance within an optimal range of 20-30ºC is necessary), 5) inoculum from virulent, wild-type isolates, and 6) to gauge disease that develops, the inclusion of a virulent isolate of Foc or of internal resistant and susceptible genotypes of banana (since small plants develop more severe disease, it is necessary to standardise host response relative to these controls). Observation of these criteria would enable useful and comparable disease experiments in different locations.

Continental action plans to limit the movement of Fusarium wilt pathogens and prevent the entry of TR4 into Africa and the Americas need to be developed or further refined, and supported by effective public awareness campaigns, reliable diagnostic methods, and strict quarantine policies and procedures. The availability of a new TR4 diagnostic tool will facilitate surveillance in regions where the pathogen has not yet been found and lead to more efficient identification and faster containment of uncharacterized Fusarium wilt outbreaks. Field evaluation of important cultivars from Latin America and Africa (e.g. plantain and highland bananas) in countries where TR4 is already present (e.g. Asia) should contribute to increased preparedness in TR4-free countries.

More research is needed to identify reliable and efficacious options for inoculum reduction in soil, e.g. by using rotation crops, fumigation, the potential value of soil amendments (e.g. silicon), solarisation and biological control. Areawide comprehensive IPM management programmes need to be better integrated, and more communication is needed between local companies and farmers. The community also needs to look into cost reductions for disease-free planting material, taking into account that tissue culture is not always an option. Workshop participants also discussed the establishment of an international collection of Fusarium pathogens associated with *Musa* and the continued recognition of using strict biosafety protocols for the movement of pathogens.

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