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Impact Statement

Panama Disease: A Classic and Destructive Disease of Banana

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Panama disease, also known as Fusarium wilt of banana (*Musa* spp.), is one of the most notorious of all plant diseases (9,11). Although the pathogen probably originated in Southeast Asia, the disease was first reported in Australia in 1876 (7). By 1950, few banana-producing regions remained free of the disease. Panama disease is now found in all banana-producing regions except islands in the South Pacific, the Mediterranean, Melanesia, and Somalia (Fig. 1).

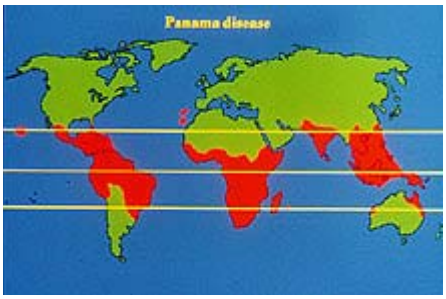

Fig. 1. Geographical distribution of Panama disease (click image for larger view).


Fig. 2. Drainage infrastructure in what was a plantation of Gros Michel in Honduras. Before 1960, widespread destruction of Gros Michel was common in export-producing areas in tropical America and western Africa (courtesy of Phillip Rowe) (click image for larger view).

Panama disease impacts the production of a wide range of banana cultivars (8). However, it is most widely known for damage it caused on a single cultivar in the early export plantations (6,11). Prior to 1960, the export trade was based almost entirely on the susceptible clultivar 'Gros Michel.' This reliance on Gros Michel and the common practice of using infected rhizomes to establish new plantations resulted in widespread and severe losses, especially in the western tropics (Fig. 2). In the Ulua Valley of Honduras alone, 30,000 hectares were lost between 1940 and 1960. Damage occurred more rapidly in areas such as Suriname, where an entire operation of 4,000 hectares was out of production within 8 years, and the Quepos area in Costa Rica, where it took 12 years for 6,000 hectares to be destroyed. Because it cost between $2,000 and $5,000 to establish a hectare of plantation at the time, direct losses during the Gros Michel era reached many millions of dollars.

By the mid-1900s, the export trade was forced to convert to resistant cultivars in the Cavendish subgroup (8). These cultivars continue to perform well in the western tropics and remain the clones on which the trades are based (Fig. 3). However, in several areas in the Eastern Hemisphere these cultivars are now damaged by Panama disease (Fig. 4). These losses are significant, and signal a serious threat to production in the Western Hemisphere because there is currently no acceptable replacement for the Cavendish cultivars. Furthermore, because

the variant of the pathogen that is responsible for these outbreaks also affects plantain, this important staple food is threatened as well.

Fig. 3. Aerial view of a plantation of 'Grand Nain,' a Cavendish clone, in the Ulua Valley, Honduras. The use of vast monocultures is a standard practice of the export trades (click image for larger view).

Fig. 4. Devastation of a Cavendish plantation in Malaysia by Panama disease in 1995 (click image for larger view).

Fig. 5. Market scene outside Jakarta, Indonesia. Note the diverse types of bananas that are offered. This is typical of local markets in the tropics (click image for larger view).

Based on its history in export production, Panama disease is undoubtedly one of the most destructive plant diseases in modern times (8,9). What is less well known is its impact on the diverse banana cultivars that are used in nonexport situations (Fig. 5). About 85% of the world's production of 89 million metric tons per year is for local consumption and marketing. Because many of the clones that are used for these markets are susceptible, losses in this sector must be significant.

Symptoms

The first internal symptoms develop in feeder roots, the initial sites of infection (1,11). They progress to the rhizome and are most prominent where the stele joins the cortex. As the pseudostem is colonized, faint brown streaks or flecks become evident on and within older leaf sheaths (Figs. 6 - 8). Eventually, large portions of the xylem turn a brick red to brown color (Fig. 9).

Fig. 6. Discoloration of the vascular system that is evident on the exterior of an old leaf sheath (click image for larger view).

Fig. 7. Discoloration of the vascular system that is evident on the exterior of an old leaf sheath (click image for larger view).

Fig. 8. Discoloration of the vascular system that is evident on the interior of an old leaf sheath (click image for larger view).

Fig. 9. Internal discoloration of the host's vascular system, a classic symptom of Panama disease (click image for larger view).

The first external symptoms of Panama disease are a yellowing of the oldest leaves (Fig. 10) or a longitudinal splitting of the lower portion of the outer leaf sheaths on the pseudostem. This is followed by a wilt and buckling of leaves at the petiole base. In some cases, these leaves remain green. As the disease progresses, younger and younger leaves collapse until the entire canopy consists of dead or dying leaves (Fig. 11).

Moko disease, caused by race 2 of the bacterium *Ralstonia solanacearum*, can be confused with Panama disease because it causes many of the above symptoms (12) (e.g., Figs. 12 and 13). However, unlike Panama disease Moko causes wilt and chlorosis on plants that are younger than about 4 months old, and will also discolor internal portions of fruit.

Fig. 10. External symptoms of wilting and foliar chlorosis in a banana plant affected by Panama disease (click image

Fig. 11. Terminal phase of Panama disease in a planting of 'Apple' banana in southern Florida (click image

for larger view).

Fig. 12. External symptoms of Moko disease. Note the resemblance of these symptoms to those that are caused by Panama disease (click image for larger view).

for larger view).

Fig. 13. Internal symptoms of Moko disease. Note the resemblance of these symptoms to those that are caused by Panama disease (click image for larger view).

Causal agent

Panama disease is caused by the soilborne hyphomycete, *Fusarium oxysporum* Schlect. f. sp. *cubense*. It is one of more than 100 formae speciales (special forms) of *F. oxysporum* that cause vascular wilts of flowering plants (2,4). It contains pathogenic and saprophytic strains that cannot be distinguished morphologically. Colonies grow 4 - 7 mm/day on PDA at 24°C, with slight to significant aerial mycelium, and white to purple pigmentation. Sporodochia are tan to orange, and sclerotia are blue and submerged.

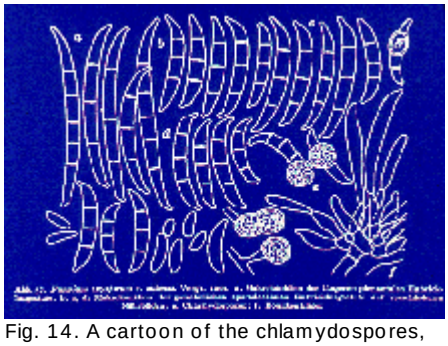

Fig. 14. A cartoon of the chlamydospores, conidiophores, and micro- and macroconidia of *Fusarium oxysporum* f. sp. *cubense* that appeared in Wollenweber and Reinking's classic monograph *Die Fusarien* (15). These features differ neither between this and other formae speciales, nor between pathogenic and nonpathogenic of *F. oxysporum* (click image for larger view).

Micro- and macroconidia are produced on branched and unbranched monophialides (Fig. 14). Microconidia are 5 - 16 x 2.4 - 3.5 µm, one- or two-celled, oval- to kidney-shaped, and are borne in false heads. Macroconidia are 27 - 55 x 3.3 - 5.5 µm, four- to eight-celled and sickle-shaped with foot-shaped basal cells. Terminal and intercalary chlamydospores are 7 - 11 µm in diameter, usually globose and are formed singly or in pairs in hyphae or conidia. Atypically for the species, chlamydospores are not produced by isolates of *F. oxysporum* f. sp. *cubense* in vegetative compatibility group (VCG) 01214 (8).

Four races of *F. oxysporum* f. sp. *cubense* have been described, only three of which affect banana (race 3 is a pathogen of heliconia) (8,11). Race 1 caused the epidemics on Gros Michel and also affects the cultivars 'Maqueño,' 'Silk,' 'Pome,' 'Pisang Awak,' and the hybrid 'I.C.2.' Race 2 affects cooking bananas, such as 'Bluggoe,' and some bred tetraploids. Race 4 is most destructive since it affects race 1 and race 2 susceptible clones as well as the Cavendish cultivars. Until recently, it had been reported only in subtropical regions where cold winter temperatures are thought to be a predisposing factor. However, within the last decade, considerable damage has occurred in Cavendish monocultures in tropical Southeast Asia (8). A distinct population of the pathogen, VCG 01213-01216, is responsible for these outbreaks. Although it is currently restricted to Asia and northern Australia, it has caused grave concern in the western trades due to their dependence on the Cavendish clones.

Vegetative or somatic compatibility has been used extensively to characterize worldwide populations of this pathogen (8). Over 20 VCGs have been reported to date, which is an indicator of the great genetic diversity that

occurs within this taxon.

Disease Cycle and Epidemiology

Beckman and his colleagues studied the internal responses of resistant and susceptible banana cultivars to infection by *F. oxysporum* f. sp. *cubense* (1). They observed that a race 1 strain of the pathogen formed abundant microconidia in xylem vessels of Gros Michel. These propagules moved acropetally in vessels via the plant's transpirational flux, and were trapped at the scalariform ends of vessels. As the fungus continued to grow it colonized the vessel end and, within 2 - 3 days, produced microconidia on its adaxial side, thus enabling the pathogen to move through another vessel. This process continued unabated in Gros Michel, but ceased in a race-1-resistant Cavendish cultivar shortly after was it inoculated. In the later case, gels formed in infected vessels in 24 - 48 hours, followed by the growth of vascular parenchyma into vessels after 48 - 96 hours. These pathogen-induced activities in the host trapped spores of the pathogen and denied it further colonization of the host. Ultimately, the host released phenolic compounds that infused and lignified the occluding structures. Thus, in a resistant cultivar, there is a clear and rapid orchestration of host defenses to ensure that systemic colonization of the xylem does not occur.

Fig. 15. A stack of traditional vegetative seedpieces, or "suckers," of 'Kluai Namwa' in Thailand. Since infected suckers rarely exhibit symptoms of Panama disease, they are a frequent and effective means by which *Fusarium oxysporum* f. sp. *cubense* is disseminated (click image for larger view).

Rhizomes ("suckers") are used traditionally as vegetative seedpieces for banana (Fig. 15). Because they are usually free of symptoms when they are infected by *F. oxysporum* f. sp. *cubense*, infected rhizomes are a common means by which this pathogen is disseminated (11). The pathogen can also spread in soil and running water, and on farm implements and machinery. Work in the early export plantations indicated that susceptible clones could not be successfully replanted in an infested site for up to 30 years due to the long-term survival of *F. oxysporum* f. sp. *cubense* in soil and as a parasite of non-host weed species (11,14).

Root tips are the natural, initial sites of infection; wounded rhizome surfaces are apparently minor infection courts (1). In most cases, root-tip infections are stopped shortly after the pathogen reaches the xylem, due to the formation of gels and tyloses and vascular collapse. However, some of these infections are not recognized early enough in susceptible cultivars, and the colonization of the xylem and associated parenchymal tissues continues. Macroconidia and chlamydospores usually form only on dead or dying plants. The latter propagules are the most significant survival structures of the pathogen.

Management

Fig. 16. A site in South Africa that is infested

Few effective options exist for managing this lethal disease (8). Chemical measures are of limited use. In work conducted in South Africa, methyl bromide significantly reduced disease incidence, but was effective for only 3 years due to recolonization of the fumigated areas by the pathogen (3) (Fig. 16). Plant injections of carbendazim and potassium phosphonate have been reported to

with *Fusarium oxysporum* f. sp. *cubense* and is undergoing tarp fumigation with a mixture of methyl bromide and chloropicrin. Although production was successfully re-established at this site for 3 years after fumigation, recolonization of the site by the pathogen eventually negated this positive response (click image for larger view).

provide some control, but results have been erratic or unrepeatable. Heat treatment of soil was used recently to control the spread of the pathogen in the Philippines, but this method will likely suffer the fate described for methyl bromide-treated soil.

Disease-suppressive soils are found in several different locations (13). In general, these soils are recognized by the length of time that high levels of production can be maintained in the presence of the pathogen. Although disease suppression has been associated with chemical and physical edaphic factors, reasons for the phenomenon differ in various locations. For example, a close relationship between suppression and clay (montmorillonoid type) soils was found in tropical America, but in the Canary Islands suppression was associated with host mineral nutrition. Unfortunately, no reports have been made on the transfer of suppression to a disease-conducive soil.

Studies on the biological and cultural control of this disease have begun only recently. Arbuscular mycorrhizal fungi have been shown to reduce disease severity in short-term green house studies, but results from long-term field studies have not been reported. Soil amendments, endophytic fungi and rhizosphere bacteria are currently being examined in Australia and South Africa. Achieving success with these or other approaches is a daunting task due to the high susceptibility of the cultivars for which protection is desired and the perennial nature of the pathosystem.

Susceptible clones can be grown if pathogen-free propagation material is used in noninfested soil (8). Tissue-cultured plantlets are free of bacterial, fungal and nematode pathogens and should be used to establish new plantings whenever possible. It should be noted, however, that plants grown from plantlets are more susceptible to Panama disease than those that are grown from rhizomes (10). Furthermore, the expense of plantlets may make their use in subsistence agriculture impractical. In the latter cases, plantlets could be used to initiate nurseries for producing pathogen-free, conventional seedpieces.

Genetic resistance offers the greatest opportunity for managing this disease in infested soils (5). To date, pre-existing cultivars have been identified that perform well in different regions and against different populations of the pathogen. Resistant hybrids have also been produced in the breeding programs, but these generally lack the flavor or post-harvest attributes that are found in the cultivars. However, as experience increases with this recalcitrant crop, acceptable, resistant hybrids will surely be produced.

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