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BLACK SIGATOKA AND MOKO: IMPACT AND SPREAD OF TWO DESTRUCTIVE BANANA DISEASES IN THE CARIBBEAN BASIN

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ABSTRACT. Black Sigatoka (aka black leaf streak), caused by the fungus Mycosphaerella fijiensis, and Moko disease, caused by the bacterium Ralstonia solancearum phylotype II, are among the most destructive diseases of banana. Black Sigatoka first appeared in the Western Hemisphere in Honduras in 1972, and then spread rapidly to other producing areas on the mainland. However, its movement in the Caribbean Islands has been slower and less extensive. The history of Moko disease is similar, in that it is widely spread in mainland tropical America but is still absent on most of the Caribbean Islands. Inter-island dispersal of M. fijiensis and R. solancearum is constrained by several factors. The pathogens are disseminated most effectively by man, but are thought to spread naturally up to, respectively, 200 and 90 km. Given the later possibilities and the distances that are involved, natural spread in most outbreaks of these diseases in the Caribbean cannot be ruled out. Only the arrival of black Sigatoka in Cuba, Florida and Jamaica, and that of Moko disease in Grenada and Jamaica are clearly the result of anthropogenic dissemination. The future spread and impact of these diseases in the region is discussed.

KEY WORDS: black Sigatoka, black leaf streak, Mycosphaerella fijiensis, Moko disease, Ralstonia solancearum phylotype II, Pseudocercospora fijiensis, quarantine, detection, sanitation, eradication

INTRODUCTION

Banana and plantain (a type of banana) are among the most important agricultural products in the tropics. Over 100 million metric tons of fruit are produced annually and that that enters international commerce is worth \$5 billion yr^{-1} (FAO, 2008). Locally consumed fruit are major staple foods in sub-Saharan Africa and Latin America; in Rwanda and Uganda, per capita consumption approaches 1 kg day^{-1}.

Diseases are major constraints in the production of this important crop. They affect every organ of the plant, and are caused by fungi, bacteria, viruses and nematodes. They reduce yield; affect the appearance, shelf life and marketability of harvested fruit; debilitate the host plant; and, in the case of systemic vascular wilts, kill plants.

There are ca 100 significant diseases of banana, a handful of which cause major damage (Jones, 2000; Stover, 1972; Wardlaw, 1961). Among the most notorious are black Sigatoka (aka black leaf streak) and Moko disease. Neither of these diseases is native to the Caribbean basin. This paper details their spread in the region, discusses the causal agents and the symptoms they cause on banana, outlines their epidemiology and management, and examines their current and potential impact. This review concludes with a warning of other diseases that threaten banana production in the Caribbean Basin.

OVERVIEW OF DISEASES

Black Sigatoka. In the Caribbean, black Sigatoka has been detected in Cuba (1990), Jamaica (1995), the Dominican Republic (1996), Haiti (1997), Trinidad (2003), Grand Bahama (2004) and Puerto Rico (2004) (Carlier et al., 2000; EPPO, 1998; Fortune et al., 2003; Irish et al., 2006; Ploetz, 2004).

Black Sigatoka affects diverse banana cultivars, including those that normally resist yellow Sigatoka, a related, globally distributed disease of this crop (Carlier et al., 2000). Black Sigatoka is a great threat due to its wide host range, great virulence and damage potential, and the ease with which it moves naturally and anthropogenically.

Symptoms of black Sigatoka begin as minute reddish brown flecks on the lower leaf surface (Ploetz et al., 2003). They become visible on the upper surface, elongate, darken and often develop a wet, oily appearance. Dark borders and yellow haloes surround spots longer than 1–2 cm, and they become grey and sunken as they mature. In susceptible cultivars, the entire leaf surface may be killed and no healthy leaf tissue remains by the time fruit mature. In severe cases, the bunch either does not develop fully or falls from the plant. The disease also causes fruit to ripen prematurely, which can be a serious problem when fruit are shipped long distances.

Black Sigatoka is caused by an ascomycete fungus, Mycosphaerella fijiensis (anamorph: Pseudocercospora fijiensis). Mycosphaerella fijiensis is closely related to M. eumusae, cause of eumusae leaf spot, and M. musicola, cause of yellow Sigatoka. DNA sequence analyses suggest that these major leaf pathogens of banana may have evolved from a common ancestor (Carlier et al., 2000; Crous and Mourichon, 2002).

Mycosphaerella fijiensis produces two primary spores, ascospores and conidia. The pseudothecia in which ascospores are produced are mainly globose, 47-85 m in dia, and immersed in the leaf tissue (Carlier et al., 2000). They occur on both leaf surfaces although they are most common on the upper side. Their ostioles protrude above the leaf surface and are dark brown and conspicuous. Asci are bitunicate, obclavate and lack paraphyses, and ascospores are colorless, 12.5-16.5 m x 2.5-3.8 m, two-celled, and constricted at the septum. The teleomorph is virtually indistinguishable from that of M. eumusae and M. musicola.

Conidia are produced in streaks early in their development, mainly on the lower leaf surface (Carlier et al., 2000). Conidiophores are pale brown, single- to six-celled, straight to geniculate, occasionally branched, subcylindric, 16.5–62.5 x 4–7 μm and in predominantly hypophyllous fascicles (Crous and Mourichon, 2002). Conidiogenous cells are up to 25 μm long, 2–4 μm wide at the apex, and have 1–3 thickened scars. Conidia are subhyaline, obclavate to cylindric-obclavate, have an obclavate basal cell, usually six- to eight-celled, 10–120 x 2.5–5 μm, with hila that are slightly thickened and darkened at the rim. Although the presence of this basal scar was shown recently to be phylogenetically unimportant in cercosporoid fungi (the former anamorphic genus for this pathogen, Paracercospora, was based on this feature) (Crous and Mourichon, 2002; Stewart et al., 1997), it is a valuable diagnostic character that enables M. fijiensis to be distinguished from M. eumusae and M. musicola.

M. fijiensis spreads naturally primarily via ascospores, both within the plant canopy, and within and between plantations (Carlier et al., 2000). Since ascospores are sensitive to UV light, they do not survive long after they are exposed to sunlight. Their ephemeral nature limits the spread of this disease to probably no more than 200 km (Parnell et al., 1998). However, anthropogenic spread via infected plants, leaves and plantation residues can move M. fijiensis great distances.

Black Sigatoka can be difficult and expensive to manage (Ploetz, 2000). In export plantations where monocultures of susceptible Cavendish cultivars are grown, fungicides in oil emulsions are applied frequently via aircraft. Although these practices can be very effective, their expense restricts their use to the export trades. When susceptible cultivars are grown for local consumption, management is restricted to backpack application of fungicides or cultural practices, such as the removal of affected leaves and improved in-plantation drainage and ventilation. However, these measures are not available or economical for many local producers who, thus, experience consistent losses due to the disease. Fortunately, disease pressure is reduced in multicropped situations that are common among small-holders.

Moko disease. Moko disease has been confirmed on Trinidad (1890s), Grenada (1978), Jamaica (2004) and St Vincent and the Grenadines (2005) (IPP, 2007; Pro-MED, 2004; Thwaites et al., 2000).

Moko disease, caused by Ralstonia solanacearum, affects diverse dessert bananas, plantains and cooking bananas (Thwaites et al., 2000). 'Bluggoe' is especially susceptible, and the disease is named after a synonym of this cultivar, 'Moko'. The pathogen that causes this disease is thought to be endemic to the Central and South American mainland, occurring on native relatives of banana, Heliconia spp. (see below). In the Western Hemisphere, Moko disease is now recognized on banana in an area extending from the Amazon Basin to Guatemala and southern Mexico.

Moko disease was first recorded on banana in the Caribbean on Trinidad in the 1890s, where it eliminated 'Bluggoe' (Thwaites et al., 2000). Another outbreak in Trinidad in the 1960s devastated the island's export trade, and the disease has since spread to Grenada, Jamaica, and St Vincent and the Grenadines. The only verified establishment of Moko disease in the Eastern Hemisphere is in the Philippines, where infested planting material from Honduras is thought to be responsible (Fagan and Prior, 2006). A similar outbreak on ornamental heliconia in Australia was eradicated (Hyde et al., 1992). Reports of Moko disease in India and several countries in Africa are erroneous.

Moko is a classic "new encounter" disease (Buddenhagen, 1960; French and Sequeria, 1970). Heliconia spp. are common understory plants in tropical America that are in the same taxonomic order as banana, the Zingiberales. The Moko pathogen coevolved on these banana relatives in the New World, and made the host jump from heliconia to banana when that crop was introduced from the Eastern Hemisphere.

Externally, the oldest leaves in the canopy become chlorotic, wilt, buckle and ultimately die (Ploetz et al., 2003). Younger leaves are then affected until the entire canopy is involved.

Leaves remain attached to the pseudostem which eventually collapses. Suckers can also be affected, and if suckers are cut with infested machetes they become blackened and stunted in 2-4 wk. When insect-transmitted strains of the pathogen infect cushions on the peduncle, the male bud whithers and darkens, and the causal bacterium often oozes from the bud.

Fruit may turn yellow and their peel split. Internally, pulp of affected fruit is firm, brown and later turning gray. On 'Bluggoe', the color is more reddish-brown and a red-brown liquid may occur at the fruit center. The vascular system in the rhizome, pseudostem and peduncle is also discolored light to dark brown. Severed vascular strands exude a milky discharge of the pathogen when placed in water.

Internal and external symptoms of Moko and Panama disease, caused by the fungus Fusarium oxysporum f. sp. cubense, are quite similar. However, only Moko affects fruit and plants younger than 4 months old.

Ralstonia solanacearum is an aerobic, Gram-negative, non-fluorescent, rod-shaped bacterium (Schaad et al., 2001). It is a widespread and diverse pathogen that has been divided into five biovars based on carbohydrate utilization, and five races that are determined by host range. Race 1 causes a vascular wilt on some Musa taxa, but not on the edible cultivars or heliconia (Thwaites et al., 2000). In contrast, strains that cause Moko disease are in biovar 1 and race 2, and are quite variable. They have restricted geographical distributions, occurring in a single country or region, and display varying levels of virulence on different banana cultivars and heliconia. They also display distinct colony phenotypes on Kelman's medium, and have disparate abilities to persist in soil and be vectored by insects, an important epidemiological trait. Virulent isolates of the bacterium produce extracellular polysaccharides and are not motile. More recently, R. solanacearum was subdivided into four phylotypes corresponding to groups identified via sequence analysis of the 16S-23S rRNA gene intergenic spacer region and the endoglucanase gene. Phylotype II contains strains primarily from the Americas and those that cause Moko disease (Fagan and Prior, 2006).

Root to root infection is possible, and moving water also disseminates the bacterium. However, spread by insects and man is most dangerous (Thwaites et al., 2000). Trigona bees, wasps and other flying insects have been reported to vector certain strains of this pathogen (especially the SFR, and to a lesser extent B, strains) 90 km. Insect-driven epidemics can move rapidly due to the strength and range of the insects that are involved and the rate at which plants become infectious. Within 15 days of flower infection, SFR strains begin to ooze from bracts and peduncles to initiate another cycle of infection. Contaminated farm machinery, machetes that are used for pruning, and infected fruit and rhizomes are all effective vehicles of dissemination.

Regular inspection and eradication programs are essential for the management of Moko disease. These include:

1) early recognition of the disease;
2) removal of the male bud;
3) rigorous disinfestation of farm implements, especially machetes that are used for bud removal and mat maintenance; and

4) destruction of affected and neighboring plants with herbicides. These sites can be replanted 6-12 months after host residues have decayed.

'Bluggoe' and other ABB cooking bananas with dehiscent bracts are most susceptible, and are also sources of inoculum for commercial bananas (Ploetz et al., 2003). In these situations, 'Pelipita' ABB, which has persistent bracts, or clones with aborting male buds can be used to replace susceptible cultivars. Although alternative weed hosts have been reported, their importance in the Moko disease cycle is debated (Thwaites et al., 2000).

FUTURE SPREAD AND IMPACT

Given the destructive nature of black Sigatoka and Moko disease, every effort needs to be made to reduce their movement and impact. Excluding the pathogens from the remaining pathogen-free islands would be most desirable, as disease management is always more expensive and difficult after a pathogen has arrived (Ploetz, 2007). Enhanced awareness of these diseases, how they are spread, and enforcement of quarantine measures that would inhibit their importation to new areas should receive high priorities.

Parnell et al. (1998) determined that ascospores of M. fijiensis survive only 6 hours of exposure to UV light and, on this basis, suggested that the maximum distance that black Sigatoka could move naturally was ca 200km. Given this distance, at least four outbreaks of black Sigatoka in the Caribbean could have resulted from natural movement: 1) over the Straights of Florida from Florida to Grand Bahama (100km), 2) over the Windward Passage from Cuba to Haiti (85km), 3) over the Mona Passage from the Dominican Republic to Puerto Rico (110km), and 4) over the Serpent's Mouth from Venezuela to Trinidad (35km) (Fig. 1). In contrast, distances that would have been involved in the outbreaks in Cuba, Florida and Jamaica exceed the 200km limit; they apparently resulted from man's intervention. The later conclusion agrees with activities that are thought to have occurred in Cuba (movement of infected banana suckers from Nicaragua), Florida (importation of infected banana suckers by a hobbyist) and Jamaica (infected banana trash in a disabled banana boat that was waylaid in Kingston).

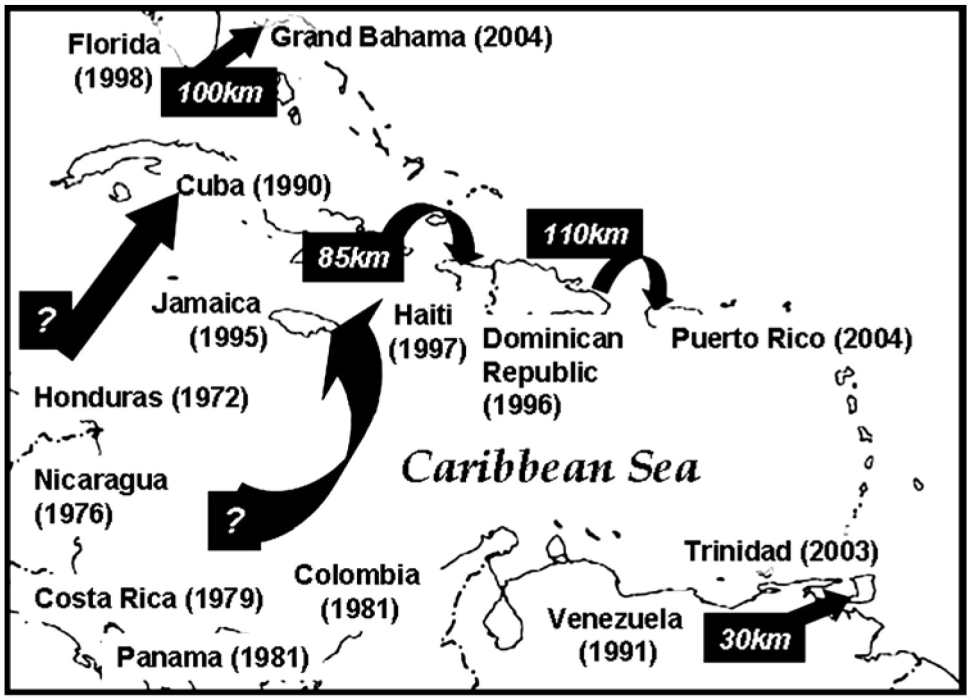

Figure 1. Countries in the Caribbean Basin in which black Sigatoka has been reported and the (year) in which detection first occurred. Natural movement was possible from Florida to Grand Bahama (100km), Cuba to Haiti (85km), the Dominican Republic to Puerto Rico (110km), and Venezuela to Trinidad (35km). In contrast, the outbreaks in Florida, Cuba and Jamaica were greater than 200km from the nearest infested landmass and were probably not natural (resulted from the unsafe importation of germplasm or movement of infested plantation trash).

Insect-vectored strains of the Moko pathogen are capable of moving up to 90km, and the SFR strains are moved most efficiently. Since SFR strains are present in Venezuela, Trinidad and Grenada (Thwaites et al., 2000), natural dissemination was possible over the 30 km distance from Venezuela (presumed to be infested prior to the 1890s) to Trinidad, and from Grenada to Carriacou, the southernmost island in the Grenadines (Fig. 2). Outbreaks of Moko disease in Jamaica and Grenada exceed the indicated 90km range and, thus, probably did not occur naturally.

Unfortunately, these pathogens will continue to move in the Caribbean, and cyclonic winds would enable natural movement even against the prevailing east-to-west tradewinds. Note is made of the generally eastern movement of black Sigatoka in the four outbreaks that are discussed above (Fig. 1), and the movement of Moko disease from Grenada to Carriacou (Fig. 2), which occurred soon after Hurricane Ivan passed through the area in 2004, the first serious hurricane in the area since 1955. The uncommon occurrence of hurricanes at that latitude may have helped isolate Moko disease on Granada for the 27 years that elapsed before it was found on Carriacou in 2005.

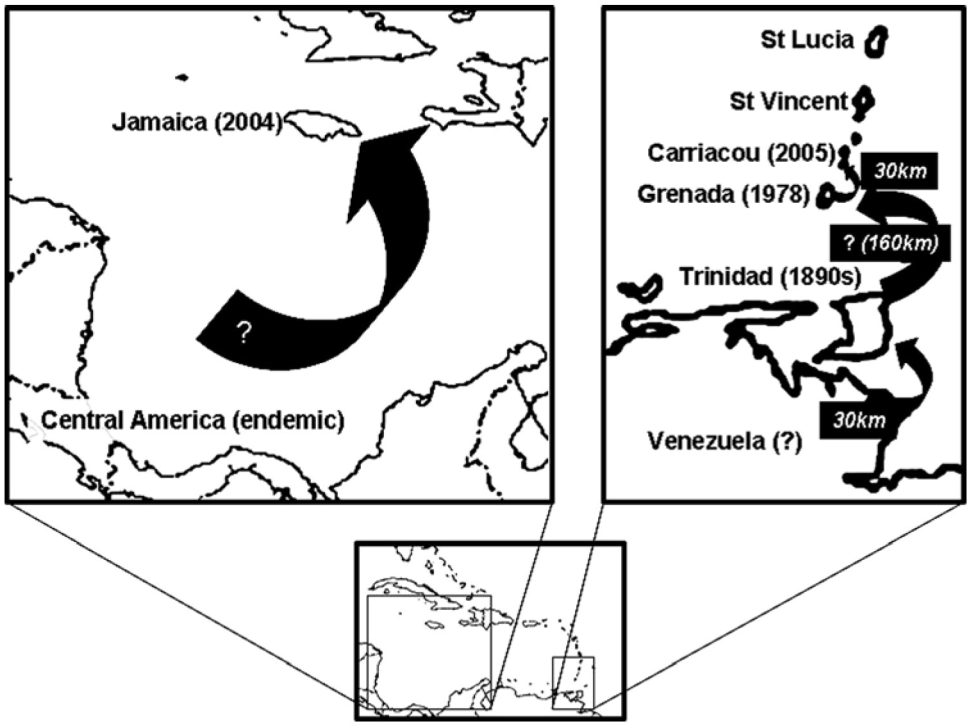

Figure 2. Caribbean islands in which Moko disease has been detected are listed with (year) of detection. The movement from Venezuela to Trinidad (30km) and Grenada to Carriacou (30km) may have resulted from the natural spread of insect-vectored SFR strains of the Moko pathogen, whereas the outbreaks in Jamaica and Grenada were more than 90 km from the nearest infested landmass (=supposed maximum range for the pathogen) and probably resulted from anthropogenic activities.

SUMMARY

In summary, natural spread cannot be ruled out in at least four outbreaks of black Sigatoka and two outbreaks of Moko disease in the Caribbean. However, it is probable that outbreaks that preceded these occurrences relied on human assistance. Had black Sigatoka not been moved by man to Cuba and Florida, subsequent outbreaks of the disease in Haiti, the Dominican Republic, Puerto Rico, and Grand Bahama, may not have occurred. Likewise, if Moko disease had not been moved to Grenada, it is possible that it would not have moved to Carriacou.

Thus, it appears that man has been a significant factor in the establishment and subsequent movement of black Sigatoka and Moko disease in the Caribbean. Their histories should serve as precautionary warnings. Under no circumstances should traditional seedpieces of banana (suckers) be moved among islands in the region. Such activity risks further movement of black Sigatoka and Moko disease, as well as equally serious diseases from outside the region (Ploetz, 2008). Only virus-indexed, tissue culture plantlets should be used when moving new banana germplasm across international borders (Diekmann and Putter, 1996).

Once these diseases are established in an area, their management will take different approaches. Black Sigatoka-tolerant cultivars are widely used in Cuba, and may assume greater importance on other Caribbean islands as the disease spreads, yields are reduced, and other control measures

are shown to be noneconomic. Markets, consumer preferences, environmental conditions (which ultimately determine disease pressure), and the types of producers who grow these fruit will determine the extent to which cultural and chemical control measures are used. Effective sanitary measures exist for Moko disease (see above), and production can be maintained when they are used in infested areas. Cultivars with dehiscent bracts or small, nondeveloping male buds, could also be used to replace susceptible clones where insect-vectored strains of the pathogen exist.

LITERATURE CITED

Buddenhagen, I.W. 1960. Strains of Pseudomonas solanacearum in indigenous hosts in banana plantations in Costa Rica, and their relationship to bacterial wilt of banana. Phytopathology 50:660-664.

Carlier, J., Four , E., Gauhl, F. Jones, D.R., Lepoivre, P., Mourichon, X., Pasberg-Gauhl, C., and Romero, R.A. 2000. Black leaf streak. Pp. 37-79 in: D.R. Jones (ed.) Diseases of Banana, Abac and Enset. CABI Publishing Wallingfrd, Oxon UK.

Crous, P.W., and Mourichon, X. 2002. Mycosphaerella eumusae and its anamorph Pseudocercospora eumusae spp. nov.: causal agent of eumusae leaf spot disease of banana. Sydowia 54:35-43.

Diekmann, M. and Putter, C.A.J. (eds). 1996. FAO/IPGRI Technical Guidelines for the Safe Movement of Germplasm. No. 15, Musa 2nd edn. Food and Agriculture Organization of the United Nations / International Plant Genetic Resources Institute, Rome, 28 pp.

EPPO. 1998. 128. Black Sigatoka disease of banana and plantain is spreading in the Americas. EPPO Reporting Service 1998, No. 798.

Fagan, M., and Prior, P. 2006. Diverse members of the Ralstonia solanacearum species complex cause bacterial wilts of banana. Australasian Plant Pathology 35: 93–101.

FAO. 2008. FAOSTAT online database at: <http://www.fao.org/default.htm>

Fortune, M.P., Gosine, S., Chow, S., Dilbar, A., St, Hill, A., Gibbs, H., and Rambaran, N. 2005. First report of black sigatoka disease (causal agent Mycosphaerella fijiensis) from Trinidad. Plant Pathology 54:246.

French, E. R. and Sequeira, L. 1970. Strains of Pseudomonas solanacearum from Central and South America: A comparative study. Phytopathology 60:506-512.

Hyde, K.D., McCulloch, B., Akiew, E., Peterson, R.A., and Diatloff, A. 1992. Strategies used to eradicate bacterial wilt of Heliconia (race 2) in Cairns, Australia, following introduction of the disease from Hawaii. Australasian Plant Pathology 21:29-31.

IPP. 2007. Moko disease declaration. International Phytosanitary Portal Report number VC-1/1. 13 Jun 07.

Irish, B. M., Goenaga, R., and Ploetz, R. C. 2006. Confirmation via the Polymerase Chain Reaction of Mycosphaerella fijiensis, causal agent of black Sigatoka of Musa spp., in Puerto Rico. Plant Disease 90:684.

Parnell, M., Burt, P.J.A., and Wilson, K. 1998. The influence of exposure to ultraviolet radiation in simulated sunlight on ascospores causing black Sigatoka disease of banana and plantain. International Journal of Biometerology 42:22-27.

Ploetz, R.C., Thomas, J.E., and Slabaugh, W. 2003. Diseases of Banana and Plantain. pp. 73-134. In: R.C. Ploetz (ed.) Diseases of Tropical Fruit Crops. CABI Publishing. Wallingford, UK. 544 pp.

Ploetz, R.C. 2000. Management of the most important disease of banana and plantain, black Sigatoka. Pesticide Outlook 11:19-23.

Ploetz, R.C. 2004. First report of black Sigatoka of banana caused by Mycosphaerella fijiensis on Grand Bahama Island. Plant Disease 88:772.

Ploetz, R.C. 2007. Diseases of tropical perennial crops: Challenging problems in diverse environments. Plant Disease 91:644-663.

Ploetz, R.C. 2008. Tropical race 4 of Panama disease: A dangerous threat to sustainable production of banana and plantain. Poster. Caribbean Food Crops Society. Miami.

ProMED. Moko Disease, Banana – Jamaica (St. James). Jamaica Observer [edited]. St James banana farmers bracing for impact of Moko disease. 16 Mar 2004.

Schaad, N.W., Jones, J.B., and Chun, W. (eds.) 2001. Laboratory Guide for the Identification of Plant Pathogenic Bacteria. APS Press, St. Paul.

Stewart, E.L., Liu, Z., Crous, P.W., and Szabo, L.J. 1999. Phylogenetic relationships among some cercosporoid anamorphs of Mycosphaerella based on rDNA sequence analysis. Mycological Research 103:1491-1499.

Stover, R.H. 1972. Banana, Plantain and Abaca Diseases. Commonwealth Mycological Institute, Kew, Surrey, UK, 316 pp.

Thwaites, R., Eden-Green, S.J., and Black, R. 2000. Diseases caused by bacteria. pp. 213-239 in: Jones, D.R. (ed.) 2000. Diseases of Banana, Abac and Enset. CABI Publishing. Wallingford, UK. 544 pp.

Wardlaw, C.W. 1961. Banana Diseases including Plantains and Abaca. Longmans, Green and Co Ltd., London, UK. 648 pp.