R. H. Stover
Tropical Agriculture Research Services, La Lima, Honduras

Sigatoka Leaf Spots of Bananas and

Sigatoka leaf spots involve three very closely related pathogens: *Mycosphaerella musicola* Leach ex Mulder, the cause of Sigatoka, first identified in Java in 1902; *M. fijiensis* Morelet, the cause of black leaf streak, described in Fiji in 1964; and *M. fijiensis* var. *difformis*, the cause of black Sigatoka, discovered in Honduras in 1972. Studies done on Sigatoka and black leaf streak up to 1970 have been reported (2,15). This article concerns developments since 1970, with particular reference to black Sigatoka.

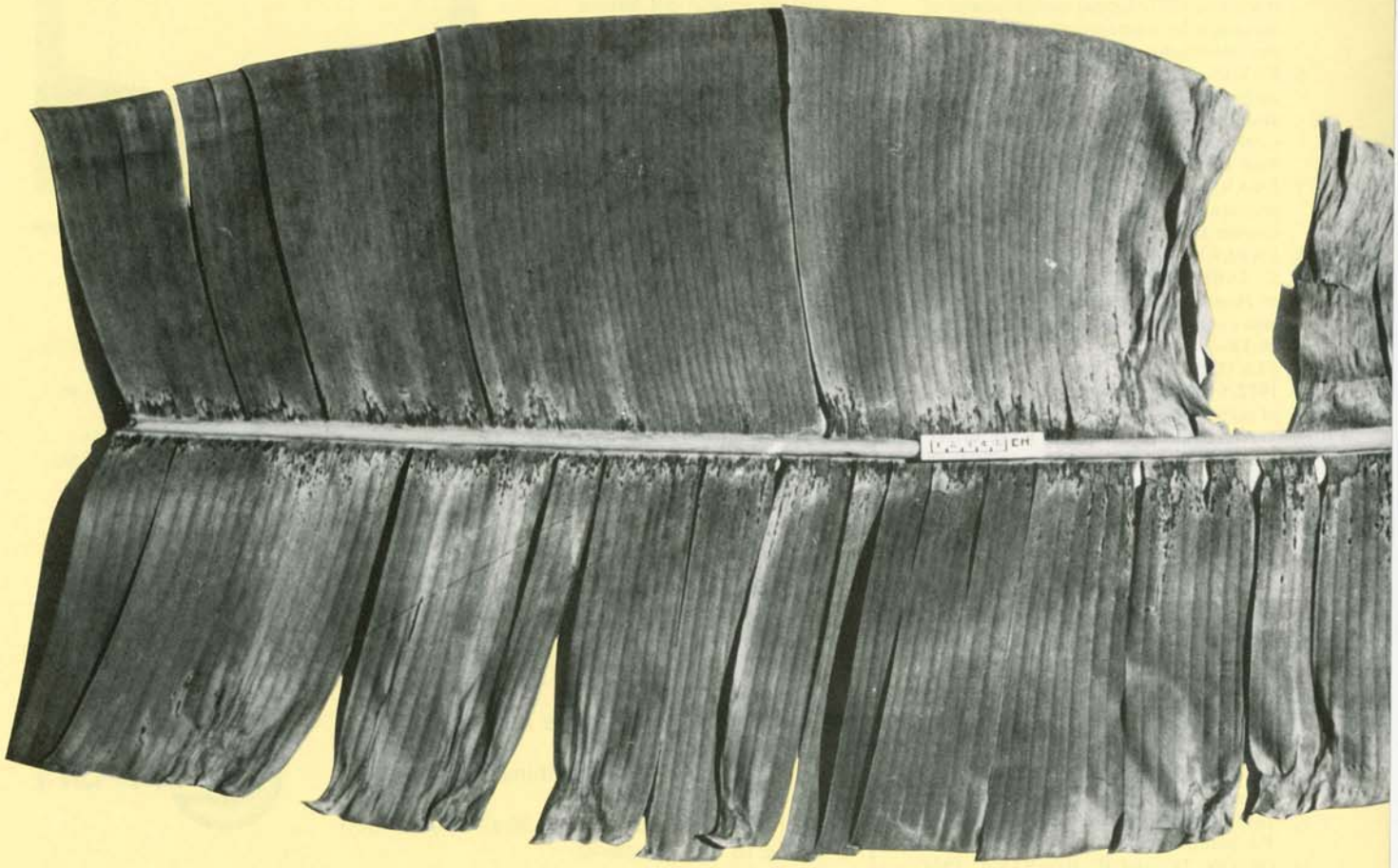
Before black Sigatoka was discovered, Sigatoka disease referred to spotting caused by *M. musicola* and black leaf streak referred to disease caused by *M. fijiensis*. Plantation personnel in Central America coined the term "black Sigatoka" for the disease caused by *M. fijiensis* var. *difformis* because the mass spotting had an overall darker appearance than that of Sigatoka; the name is now in widespread use. As a group, the three closely related conditions are best called Sigatoka leaf spots. The term "yellow Sigatoka" is often heard with reference to *M. musicola* in Central America but should be avoided.

0191-2917/80/08075007/$03.00/0

The Pathogens

Morphology and taxonomy. The three pathogens are distinguished by characteristics of the conidiophores and conidia (Table 1). Simple conidiophores emerging from stomata on the lower surface of young streaks and tapering conidia with basal scars readily separate the *M. fijiensis* pathogens from *M. musicola* (5). Although the two *M. fijiensis* pathogens have biological differences, the only morphological difference is the occurrence of sporodochia in young spots of the black Sigatoka pathogen. Characteristics of perithecia, ascospores, and spérmagonia are similar among the three pathogens.

Virulence. Black leaf streak was far more destructive than Sigatoka in Fiji (2) and more difficult to control in the Philippines (14). Inoculations in Honduras and field observations disclosed that spotting appeared 8–10 days faster with black Sigatoka than with Sigatoka. Black Sigatoka became epidemic in



Plantains

Honduras during 1973–1974 and replaced the Sigatoka pathogen within 2 years (21).

Virulence is easily measured by the rate of spotting and is indicated by the number or age of the youngest leaf spotted (13). Before the onset of *M. fijiensis* var. *difformis*, spotting appeared on leaves 4–5 of unsprayed Valery bananas in Honduras (Fig. 1). When *M. fijiensis* var. *difformis* replaced *M. musicola* in the same area, spotting appeared on leaves 3–4 and sometimes on leaves 2–3.

Behavior in culture. The *M. fijiensis* pathogens form an indistinguishable cultural group (Fig. 2). The two main types are: 1) a dark gray or gray-brown colony (DGB) with a crenate edge and 2) a pale gray or pink colony (PGP). The DGB colonies produce more conidia when first isolated but become unstable with time, yielding PGP cultures with few or no conidia. Original isolates are unstable but eventually give rise to colonies (gray or rose in *M. musicola* and PGP in the *M. fijiensis* pathogens) that are stable and nonsporulating, with little or no virulence (16). Spermagonia with spermatia are produced in many isolates, but perithecia or protoperithecia have never been found.

In areas sprayed with benomyl or thiophanate-methyl, a distinctive *Cercospora* sp. is obtained from lesions of black Sigatoka and Sigatoka (19). This fast-growing, nonvirulent *Cercospora* (Fig. 2) produces minutely verrucose conidia in culture, with scars on simple conidiophores. This *Cercospora* is not obtained from the black leaf streak pathogen in the Philippines in areas sprayed for several years with thiophanate-methyl but is found throughout Central America and Surinam. These pathogens must have common genes that produce the same *Cercospora* sp. when subjected to benzimidazole fungicides. The role this nonvirulent *Cercospora* sp. plays in pathogenesis is not known. The level of tolerance to benzimidazoles is high (up to 600 μg/ml) even when ascospores, conidia, and normal *Cercospora* thalli taken from the same leaf spots are sensitive to 0.1 μg/ml.

Distribution outside Asia. The probable center of origin and the distribution of the *M. fijiensis* pathogens in Asia have been described (17). The first appearances outside Asia and the Pacific Islands were recorded in Honduras in 1972 (21) and in Zambia in 1973 (6). In 1979, *M. fijiensis* was found in Gambia. In Central America, the black Sigatoka pathogen spread slowly during 1973–1978, but recently the rate of spread has increased (Fig. 3).

After the 1973 epidemic in Honduras (21), black Sigatoka appeared in Belize in 1975. In Honduras, spotting spread slowly westward along the coast and reached the Motagua Valley of Guatemala in early 1977. Because winds are predominantly from the east and northeast, extension eastward along the coast and southward toward the interior of the country was slower. The pathogen reached the lower Aguán Valley banana zone in February 1978. This was an isolated focus of about 2 ha in the 2,000-ha Isletas plantation and probably was derived from infected leaf trash on trucks picking up rejected bananas. In 1979 the disease spread over the entire Isletas plantation and appeared in the Coyoles area of the upper Aguán Valley and the interior Comayagua Valley. After gradually spreading over a radius of 150 km in northern Honduras and adjacent Guatemala, the disease suddenly appeared some 500 km distant in the Santa Clara area and Central Plateau of Costa Rica in October 1979. About 4,000 ha of plantains were infected; the pathogen probably was introduced the year before. In 1979, black Sigatoka spread throughout the Chinandega banana zone of Nicaragua.

In 1980, black Sigatoka spread to the banana plantations on the Atlantic coast of Costa Rica. By 1982 black Sigatoka will have spread throughout Central America from the Tapachula area of southern Mexico to the banana zones on the Atlantic and Pacific coasts of Panama.

How Disease Develops

Infection and spotting patterns. Spores of all three pathogens germinate on the moist lower leaf surface within 2 hours, but stomata usually are not penetrated until after 48–72 hours of humidity at or near saturation and temperatures above 20 C. Once infection is established, hyphae of the *M. fijiensis* pathogens emerge from the stomata and either develop into conidiophores or grow across the surface to infect adjacent stomata. The black Sigatoka pathogen apparently moves from one stoma to another much more often than *M. musicola* and causes lesions over entire leaves. The first small lesions (2–3 mm long) appear around the stoma about 10–14 days after infection.

Infection by *M. musicola* often results in distinct spotting patterns (2,15), depending on whether inocula were derived from conidia (line spotting) or ascospores (tip spotting). With black Sigatoka, most infection is derived from ascospores, and spore patterns usually cannot be distinguished. Infection is evident first by streaks along the left edge of the first leaf to unfurl, then by streaks on the leaf tip and down the right side of the apex. In sprayed areas, infection along the edges of the leaf base is frequent. As the leaf ages, streaks develop over most of the leaf if inoculum is abundant and the weather is favorable.

In areas where plants are sprayed with oil and a dithiocarbamate or benzimidazole fungicide for black Sigatoka, a distinct spotting pattern along either side of the vein develops (Fig. 4), particularly in mature or fruited plants. This vein spotting becomes evident 50–70 days after streaks appear along the left edge of the leaf tip. Although streaks along the edge usually do not develop further, those along the vein evolve into mass spotting. In the Philippines, where the same oil-based sprays are used against black leaf streak, spotting occurs mostly over the apical third of the leaf and seldom along the vein. Why *M. fijiensis* should react differently to the same sprays is not known. Vein spotting is probably related

to leaf thickness, oil penetration, and spray action on lesion development. The lamina is 530–780 μm thick near the vein and 220–300 μm thick near the leaf edge.

Spore production and dispersal. The contribution of conidia and ascospores to total inoculum and infection patterns in *M. musicola* is well understood (2,11,15). Although much less is known about the *M. fijiensis* pathogens, observations of spores infecting leaves, numbers of conidia produced on streaks, spore discharge from mass-spotted leaves, and Hirst spore trap counts indicate that ascospores are the major source of inoculum. Conidia contribute much less inoculum than do those of the Sigatoka pathogen (11).

Conidiophores and conidia were counted on unsprayed plants with young black Sigatoka streaks not exceeding 20 mm^2. About seven stomata per square millimeter had an average of 2.5 conidiophores per stoma, producing an average of 8.5 conidia or about 60 conidia per square millimeter or 1,200 per lesion on the lower leaf surface only. In contrast, in plants infected with *M. musicola*, 75–125 sporodochia are produced per spot on the upper leaf surface. Each sporodochium can produce an average of 5.3 crops of 50 conidia each (11). If 50% of this quantity is added for spores from the lower leaf surface (where sporodochia are smaller), a Sigatoka spot can produce more than 30,000 conidia.

Conidia of the *M. fijiensis* pathogens are dislodged by wind, whereas *M. musicola* conidia are freed from the conidiophore only when the leaf has a film of water. The attachment mechanism resulting in scars in the *M. fijiensis* pathogens must permit easy wind removal.

Conidiophore production of the *M. fijiensis* pathogens is of short duration. Stomatal chambers are used for rapid production of perithecia and spermagonia. Under optimal conditions of high temperatures, heavy rainfall, and mass infection, ascospores are mature 4 weeks after the streak stage with *M. musicola* and 2 weeks with *M. fijiensis* var. *difformis*.

When leaves with rapidly developing young mass spots of black Sigatoka were left lying on the ground to decay, discharge of ascospores was noted within 5 days and continued as long as 23 days. Ascospores of *M. musicola* survived as long as 8 weeks in the shade on leaf tissue above the ground (12). Ascospore survival time on leaves lying on the ground in the shade depends on the rate of decomposition. Leaves on the ground subjected to intermittent rainfall, dew, and partial drying in the daytime continue to discharge ascospores in declining amounts for up to 4 weeks. Only one crop of ascospores is produced per perithecium, indicating that perithecia continue to mature on the ground after leaves are cut down.

Some indication of the number of ascospores produced in heavily diseased areas was obtained in Hawaii (4). From 8,000 to 33,000 ascospores of *M. fijiensis* per cubic meter of air per 24 hours were obtained in a Hirst spore trap. Trapping in Honduras and Panama yielded a maximum of 4,000/m^3 of *M. musicola* (*unpublished data*). Mature ascospores of the three pathogens are released quickly when leaves are wetted; a diurnal release pattern is noted, depending on rain and dewfall (4,15).

Abundant ascospore production is seasonal with *M. musicola*, usually paralleling rainfall (2,15). Data are not available for the *M. fijiensis* pathogens, but spotting curves parallel rainfall, suggesting the same for ascospore production.

Effect of weather. The effects of rainfall, dew, and minimum temperatures on *M. musicola* have been reviewed (2,15). Although the *M. fijiensis* pathogens have not been studied in this regard, field observations indicate similar responses to moisture and temperature. Dry weather and night temperatures below 20 C slow disease development. In Honduras, however, the *M. fijiensis* pathogens appear to be less sensitive than *M. musicola* to low temperatures when rainfall is abundant. Spotting starts to increase between June and July, peaks in October and November, and remains at high levels through December and January. Spotting levels begin to decline in February or early March, reaching the lowest levels in April, May, and early June. March through May are the driest months, but heavy dews are common (11).

Measures to Control Disease

Major advances in control during the past decade include improved low-volume application techniques with aircraft and better spray formulas (15). Volumes applied usually do not exceed 18.8 L/ha (2 gal/A); in most areas, only 9.4 L/ha (1 gal/A) is used for Sigatoka. With the exception of chlorothalonil, fungicides are applied in oil-in-water emulsions or are mixed directly into oil without water, using high-speed blenders. Aircraft are equipped with three finely tuned rotary atomizers on each wing, fly at an altitude of 15–18 m (50–60 ft), and

Table 1. Morphological features of the three Sigatoka pathogens

Pathogen	Conidiophores				Conidia
	First appearance	Form	Distribution on lesions	Morphology	
Mycosphaerella musicola	Spot stage	Dense fascicles on dark stroma (sporodochium)	Abundant on both surfaces	Straight, hyaline, mostly without septation, geniculation, or branching; no spore scars	Mostly cylindric, occasionally obclavate, 1–5 septate; same thickness throughout length; no distinct basal hilum
M. fijiensis	Early streak stage	Emerge singly or in small groups (2–5 stalks); no sporodochia	Mainly on lower surface	Straight or bent geniculate, pale to light brown, 0–3 septate, occasionally branched, slightly thickened spore scars	Obclavate to cylindroclavate 1–6 septate; tapering from hilum end to apex; distinct basal hilum (scar)
M. fijiensis var. *difformis*	Early streak stage for simple conidiophores; young spots for conidiophores on sporodochia	Emerge singly or in small groups or on dark stroma (sporodochium); occasionally produced in groups of 4–20 on spermagonial stroma	Simple conidiophores largely on lower surface; sporodochia on both surfaces	Simple conidiophores same as *M. fijiensis*; conidiophores on sporodochia same as *M. musicola* except occasionally septate; sporodochia smaller, usually about one-half size of *M. musicola*	Same as *M. fijiensis*, tapering from hilum end to apex and with scars

spray swaths 24–30 m (80–100 ft) wide. With good landing field distribution and contiguous planting, up to 162 ha an hour can be sprayed. Spray intervals vary from 8 to 16 days for the *M. fijiensis* pathogens to 21 to 40 days for *M. musicola*, depending on weather and spotting.

Fungicides. In the early 1970s, the introduction of benomyl, used along with the dithiocarbamate fungicides in oil formulations, greatly enhanced disease control. Other benzimidazoles, such as thiophanate-methyl, were also effective. Benzimidazoles in oil are unevenly translocated from the upper surface to parts of the lower surface, permitting infection in areas where translocation does not occur (Fig. 5). Little or no lateral movement occurs.

The benzimidazoles are used alone with oil in the West Indies and West Africa and in localized areas elsewhere for control of *M. musicola*. After more than 5 years, pathogen tolerance has not been recorded except in Surinam. Lack of tolerance in other areas has been attributed to the action of oil on the pathogen. Nevertheless, oil spray formulas did not prevent tolerance from developing in Surinam in *M. musicola* or in Central America and the Philippines in the *M. fijiensis* pathogens. After 2–3 years of benomyl or thiophanate-methyl use in Honduras and the Philippines, tolerant strains appeared and spread (20).

Tolerant strains appeared even when dithiocarbamate fungicides were used with benzimidazoles. In Honduras, tolerant strains spread rapidly into areas where a mixture of mancozeb (Dithane M-45) and benomyl (Benlate) in oil was applied. When benomyl was removed, further spread could not be detected; 1 year later, resistance fell from 300 μg/ml or more to 10 μg/ml (20). Resistance levels of 10–50 μg/ml persist in scattered pockets among less than 10% of the ascospores.

After the appearance of benomyl tolerance in Honduras and subsequent deterioration in control, clearance to use chlorothalonil was obtained (22). Chlorothalonil retards or stops development of young lesions and proved highly effective. Chlorothalonil is phytotoxic with oil and was the first aircraft-applied banana fungicide not used in an oil formulation.

Quarantine, exclusion, and eradication. Distribution of the Sigatoka diseases and methods of preventing spread into new areas separated by natural barriers have been described (18). The diseases are spread by 1) trucks carrying bananas and plantains that use infected leaves to protect the fruit from bruising and sunburn, 2) moving rhizomes and suckers for planting purposes from infected plants without appropriate treatment (18), and 3) wind-transported ascospores. Wind dispersal from small areas of infection to new areas is probably slight when distances exceed 50 km.

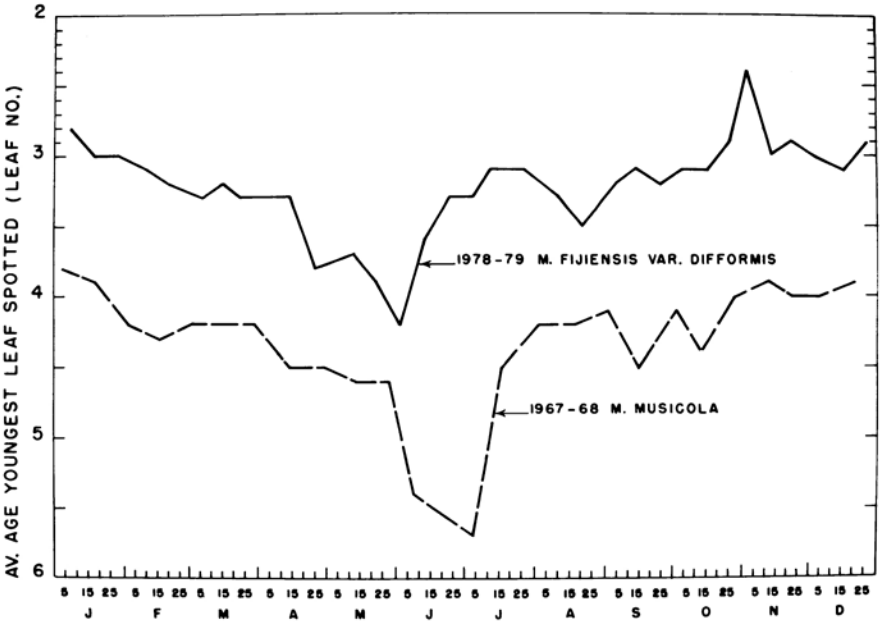


Fig. 1. Average age of youngest leaf of unsprayed Valery bananas spotted by *M. musicola* during 1967–1968 and by *M. fijiensis* var. *difformis* during 1978–1979 in the same area of Honduras. Because a new leaf emerges every 8–10 days, the difference in rate of disease development is indicated by the leaf on which spotting first appears. Leaves 4–5 were affected first with *M. musicola* and leaves 3–4 and sometimes 2–3 with *M. fijiensis* var. *difformis*.

Fig. 2. Cultures of *M. fijiensis* (upper left, dark gray colony; lower left, pale gray colony), *M. musicola* (upper right), and a fast-growing, nonvirulent, benomyl-tolerant *Cercospora* sp. (lower right) after 18 days on Mycophyl agar. Cultures of *M. fijiensis* are indistinguishable from those of *M. fijiensis* var. *difformis*.

Overland spread of black Sigatoka into Colombia from Central America can be greatly delayed by the natural barrier of forested and uninhabited portions of the Darien Peninsula in Panama (Fig. 3). As small farms, with their inevitable patches of plantains and bananas, spread into the area, however, a chain of scattered *Musa* plantings will eventually reach into Colombia and provide a dispersal route by wind-borne and man-transported means.

Large plantings of bananas and plantains in the Caribbean islands are separated from Central America by the Caribbean Sea. Also, the prevailing easterly winds blow toward the mainland. The *M. fijiensis* pathogens are unlikely to breach this natural water barrier unless carried by man.

By the time an outbreak is discovered in a new area, the pathogen has usually been there for at least 1 year and often for 2 years or longer (21). Spread during this initial period is not always detectable by

outbreaks of spotting and usually is too extensive to eradicate. Where natural mountain, water, forest, or other nonhost barriers separate the infested area from other banana and plantain zones, quarantines around the infested area can impede movement by man. Quarantine restrictions are difficult to enforce, however, even along main roads, and traffic along trails and rivers in remote areas is unrestricted. Bananas and plantains are staples in the tropical diet, and any severe restriction on movement results in evasive measures that effectively bypass the quarantine.

Cultivar resistance. Resistance to *M. musicola* in common edible clones, wild diploids, and bred tetraploids has been recorded (8–10,23). All the AAA edible clones are highly susceptible, whereas AAB plantains have some resistance. The ABB plantains have the highest level of resistance of edible cultivars. The parthenocarpic and seeded diploids used as male parents in crossing with the cultivar Gros Michel to produce tetraploids are resistant. As a result, most tetraploids have varying levels of resistance (9,10). The highest levels of resistance in wild diploids are found in *Musa acuminata* subsp. *burmannica* and subsp. *malaccensis*.

Observations on resistance of mostly edible clones to the *M. fijiensis* pathogens were made in Hawaii (3) and Fiji (1). The ranking of resistance was similar to that for *M. musicola*, except that AAB plantains were considered very susceptible in Hawaii and moderately so in Fiji. As with *M. musicola*, the ABB plantains had more resistance than the AAB. In all cases, however, spotting levels were higher with the *M. fijiensis* pathogens, reflecting their greater virulence.

In Honduras, the Philippines, and Taiwan, the diploid and some tetraploid clones highly resistant to *M. musicola* also have some resistance to the *M. fijiensis* pathogens. Bodles Altafort, a Sigatoka-resistant tetraploid bred in Jamaica, had some resistance to *M. fijiensis* in the Philippines.

In Honduras, diploid clones highly resistant to *M. musicola* (23) are also resistant to *M. fijiensis* var. *difformis*. As resistance levels of *M. musicola* fall, attacks by *M. fijiensis* var. *difformis* are more severe. Thus, clones with moderate to low levels of resistance to *M. musicola* are susceptible to black Sigatoka, although less so than the highly susceptible Cavendish cultivars (AAA). For example, the commercially important horn plantain (AAB) has some resistance to *M. musicola* and does not require spraying, whereas with black Sigatoka, spraying is necessary during the rainy season to prevent heavy defoliation and loss of fruit.

The high level of resistance in *M. acuminata* subsp. *burmannica* and subsp. *malaccensis* is readily incorporated into diploids for use in breeding new banana cultivars (8). Thus far, no races have appeared that can attack highly resistant diploids and tetraploids. These diploids can be used to breed resistant AAB plantains (7), but breeding is a slow process and new cultivars resistant to leaf spot will not be available for another decade.

Fig. 3. Major banana-growing zones (black areas) in southern Mexico (Tapachula), Central America, and Colombia (Uraba). Black Sigatoka, now in Honduras, Guatemala, Belize, Nicaragua, and Costa Rica, will spread into Mexico and throughout Central America by 1982. The Darien forest barrier should delay spread into Colombia.

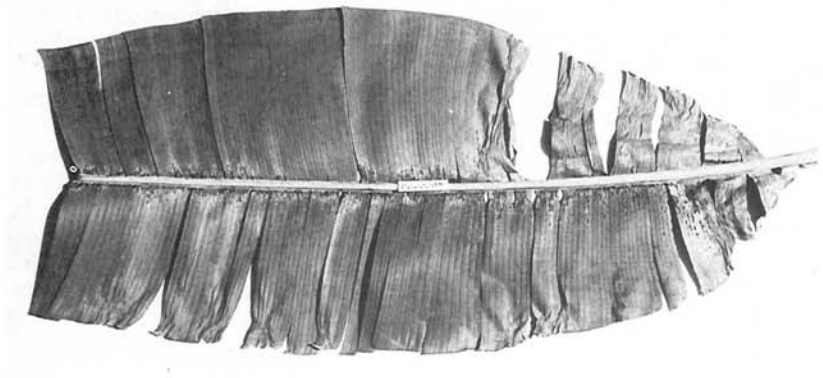


Fig. 4. Vein spotting typical of black Sigatoka in areas where plants are sprayed with oil formulations of dithiocarbamate or benzimidazole fungicides. Benlate in an oil-in-water emulsion has been applied five times to this leaf.

Outlook and Needs

The imminent spread of black Sigatoka throughout Central America will double the costs of control because more frequent spraying will be necessary. About 85% of the costs involve spray ingredients and the remainder, aircraft application. Spraying will have to be started on plantains if yield and quality are to be maintained. The increased costs can be met only by maximizing production per hectare on first-class soils. Marginal levels of production will no longer be economic.

Good control can be obtained with combined dithiocarbamate and benzimidazole fungicides. If tolerance to the benzimidazole develops, however, control with present dithiocarbamate formulations is not adequate when rainfall is heavy, and chlorothalonil must be used. This dependence on one fungicide is undesirable, and the control arsenal must be broadened.

As costs continue to rise, the need for resistant cultivars becomes imperative. Fortunately, good sources of resistance have been identified and incorporated

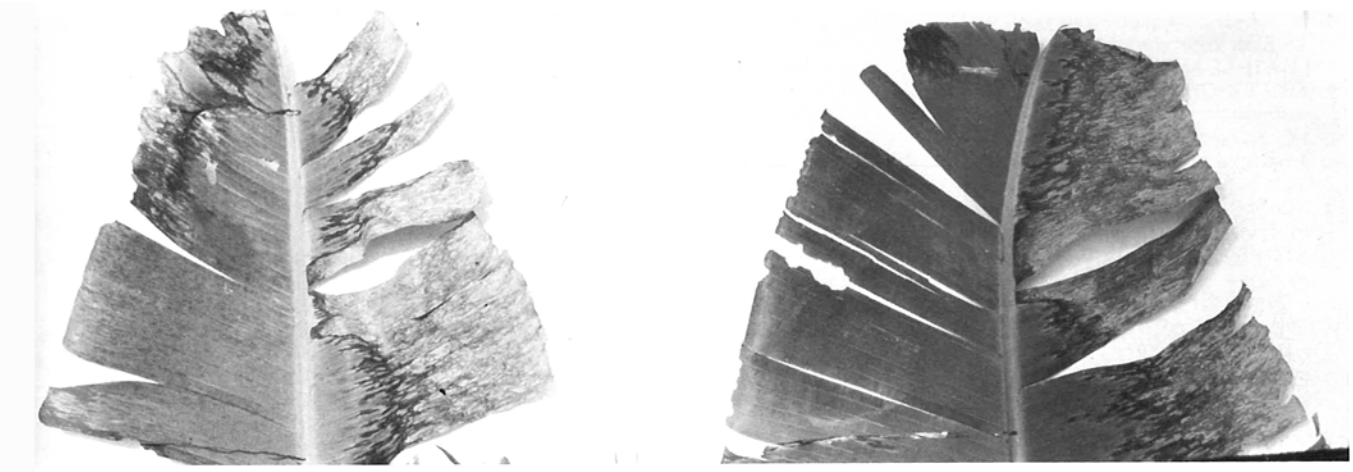


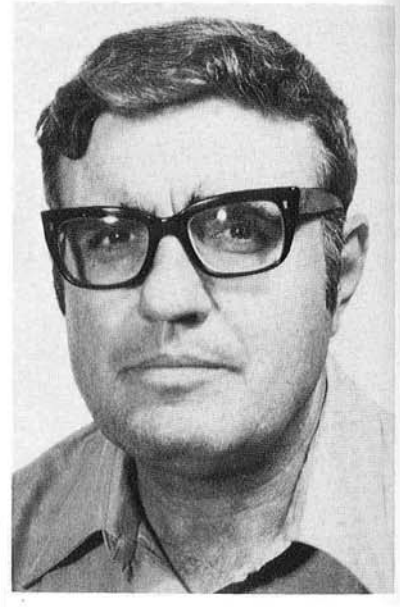
Fig. 5. The left side of each leaf was sprayed three times at 10-day intervals with benzimidazole fungicides in oil; spotting on the right side was caused by natural inoculum in an area where plants are not sprayed. (Left) Application to the upper left side gave good control of black Sigatoka, but translaminar movement was not complete and some infection occurred on the lower surface. (Right) Application to the lower surface almost completely controlled infection.

into lines useful in schemes for developing new cultivars of bananas. Nothing, however, has been done about breeding resistant plantains. Banana and plantain breeding is slow and expensive, and more support is needed to speed up development of resistant cultivars. In the meantime, cheaper chemical methods of control must be found.

Literature Cited

1. FIRMAN, I. D. 1972. Susceptibility of banana cultivars to fungus leaf disease in Fiji. Trop. Agric. (Trinidad) 49:189-196.
2. MEREDITH, D. S. 1970. Banana leaf spot disease (Sigatoka) caused by *Mycosphaerella musicola* Leach. No. 11. Commonw. Mycol. Inst., Kew, Surrey, England. 147 pp.
3. MEREDITH, D. S., and J. S. LAURENCE. 1970. Black leaf streak disease of bananas (*Mycosphaerella fijiensis*): Susceptibility of cultivars. Trop. Agric. (Trinidad) 47:275-287.
4. MEREDITH, D. S., J. S. LAURENCE, and I. D. FIRMAN. 1973. Ascospore release and dispersal in black leaf streak disease of bananas (*Mycosphaerella fijiensis*). Trans. Br. Mycol. Soc. 60:547-554.
5. MULDER, J. L., and R. H. STOVER. 1976. *Mycosphaerella* species causing banana leaf spot. Trans. Br. Mycol. Soc. 67:77-82.
6. RAEMAEKERS, R. 1975. Black leaf streak like disease in Zambia. PANS 21:396-400.
7. ROWE, P. R. 1976. Possibilités d'amelioration génétique des rendements des plantain. Fruits 31:531-536.
8. ROWE, P. R., and D. L. RICHARDSON. 1975. Breeding bananas for disease

resistance, fruit quality and yield. Bull. 2. Tropical Agricultural Research Services (SIATSA), La Lima, Honduras.
9. SHILLINGFORD, C. A. 1975. Varietal susceptibility of bananas to infection by *Mycosphaerella musicola* in sprayed and unsprayed plots. Trop. Agric. (Trinidad) 57:157-165.
10. SIMMONDS, N. W. 1966. Bananas. 2nd ed. Longmans, Green, London. 512 pp.
11. STOVER, R. H. 1970. Leaf spot of bananas caused by *Mycosphaerella musicola*: Role of conidia in epidemiology. Phytopathology 60:856-860.
12. STOVER, R. H. 1971. Ascospore survival in *Mycosphaerella musicola*. Phytopathology 61:139-141.
13. STOVER, R. H. 1971. A proposed international scale for estimating intensity of banana leaf spot. Trop. Agric. (Trinidad) 48:185-196.
14. STOVER, R. H. 1971. Banana leaf spot caused by *Mycosphaerella musicola*: Contrasting features of Sigatoka and black leaf streak control. Plant Dis. Rep. 55:437-439.
15. STOVER, R. H. 1972. Banana, plantain, and abaca diseases. Common. Mycol. Inst., Kew, Surrey, England. 316 pp.
16. STOVER, R. H. 1976. Distribution and cultural characteristics of the pathogens causing banana leaf spot. Trop. Agric. (Trinidad) 53:111-114.
17. STOVER, R. H. 1977. Distribution and probable origin of *Mycosphaerella fijiensis* in Southeast Asia. Trop. Agric. (Trinidad) 55:65-68.
18. STOVER, R. H. 1977. Bananas (*Musa* spp.). Pages 71-79 in: W. B. Hewitt and L. Chiarappa. Plant Health and Quarantine in International Transfer of Genetic Resources. CRC Press, Cleveland.
19. STOVER, R. H. 1977. A non-virulent benomyl tolerant *Cercospora* from leaf spots caused by *Mycosphaerella fijiensis* var. *difformis* and *M. musicola*. Trans. Br. Mycol. Soc. 69:500-502.
20. STOVER, R. H. 1979. Field observations on benomyl tolerance in ascospores of *Mycosphaerella fijiensis* var. *difformis*. Trans. Br. Mycol. Soc. 72:518-519.
21. STOVER, R. H., and J. D. DICKSON. 1976. Banana leaf spot caused by *Mycosphaerella musicola* and *M. fijiensis* var. *difformis*: A comparison of the first Central American epidemics. FAO Plant Prot. Bull. 24:36-42.
22. STOVER, R. H., W. R. SLABAUGH, and M. D. GROVE. 1978. Effect of chlorothalonil on a severe outbreak of banana leafspot caused by benomyl tolerant *Mycosphaerella fijiensis* var. *difformis*. (Abstr.) Phytopathol. News 12:268.
23. VAKILI, N. G. 1968. Responses of *Musa acuminata* species and edible cultivars to infection of *Mycosphaerella musicola*. Trop. Agric. (Trinidad) 45:13-22.



R. H. Stover

Dr. Stover studied bacteriology at the University of Guelph, Canada, and plant pathology and mycology at the University of Toronto. He began studies on tropical plant diseases in 1951 with the United Fruit Company in Honduras and later became head of the company's Plant Pathology Department and director of tropical research. At present, he is director of Tropical Agriculture Research Services (SIATSA), a United Brands subsidiary that provides technical assistance and consulting in tropical agriculture worldwide.