

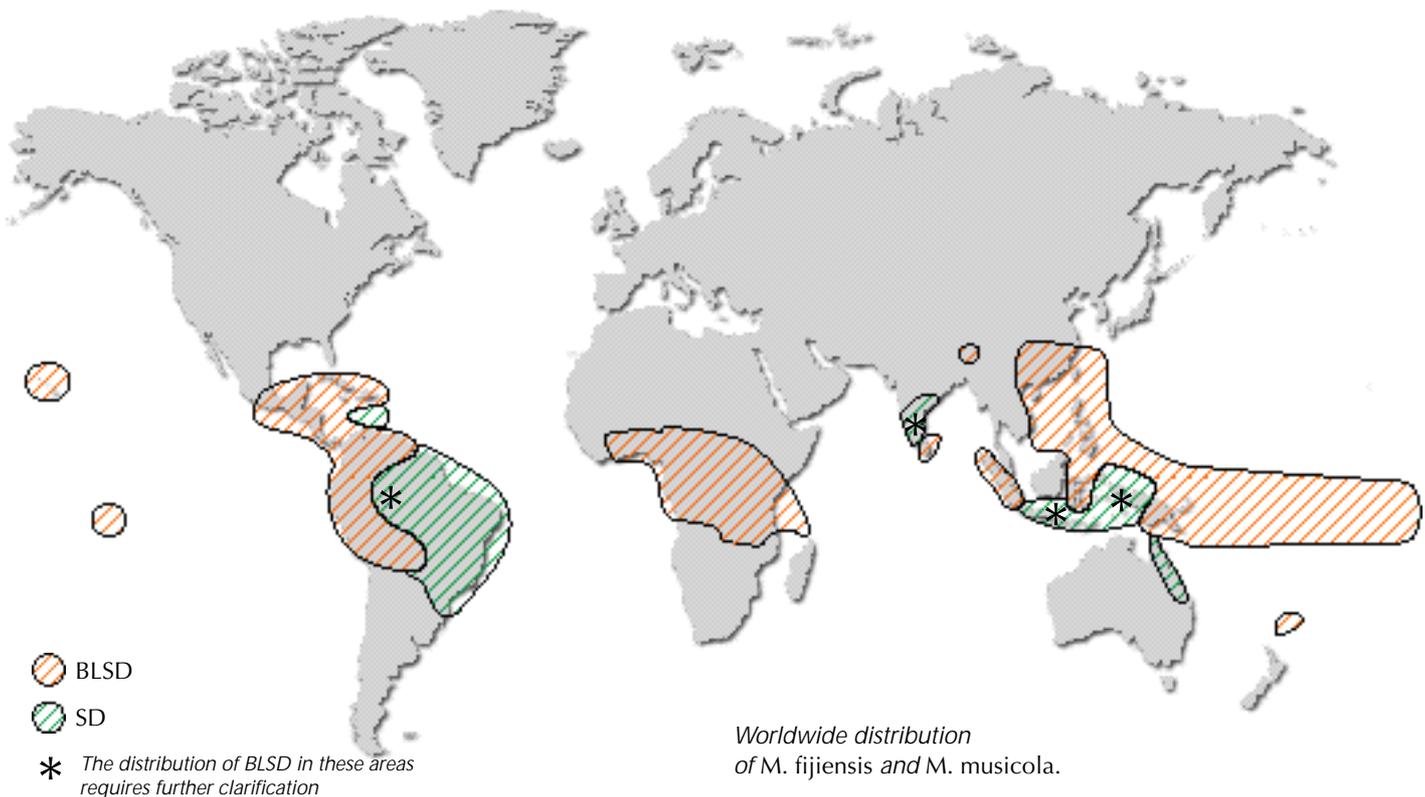
SIGATOKA LEAF SPOT DISEASES

Black leaf streak disease (black Sigatoka) *Sigatoka disease (yellow Sigatoka)*

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SD. Plantain field seriously damaged.

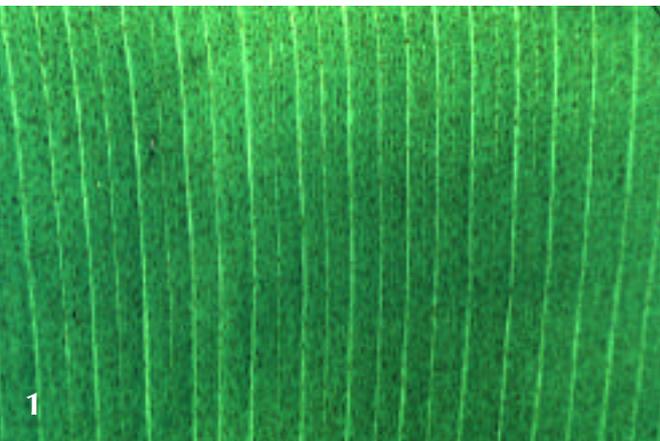


Sigatoka leaf spot diseases of bananas involve two related ascomycetous fungi: *Mycosphaerella fijiensis* Morelet, which causes black leaf streak disease (BLS), and *M. musicola* Leach ex Mulder, which causes Sigatoka disease (SD). The two pathogens can be distinguished morphologically essentially on conidia and conidiophore characteristics. Thickened cell walls, which are noticeable at the base of conidia of *Paracercospora fijiensis* (Morelet) Deighton (anamorph of *M. fijiensis*), are absent from conidia of *Pseudocercospora musae* (Zimm.) Deighton (anamorph of *M. musicola*). Conidia of *P. fijiensis* are also on average longer and more flexuous than those of *P. musae*. Conidiophores of *P. musae* are usually short and bottle shaped while those of *P. fijiensis* are elongated, often bent and have conspicuous conidial scars. These differences between anamorphs are easily observed directly on leaf samples and in sporulating cultures after pathogen isolation. Molecular methods have also been developed for determination of the species.

Symptoms

Symptoms of BLS and SD are sometimes difficult to differentiate. In general, the first symptom is the appearance on the upper leaf surface of pale yellow streaks (SD) or dark brown streaks on the lower leaf surface (BLS), both 1-2 mm long which enlarge to form necrotic lesions with yellow haloes and light grey centres. Lesions can coalesce and destroy large areas of leaf tissue which results in reduced yields and premature ripening of fruit.

BLS is more serious than SD because symptoms appear on younger leaves, which is generally due to a greater amount of inoculum, and hence more damage is caused to photosynthetic tissue. BLS also affects many cultivars that have resistance to SD, such as those in the plantain subgroup (AAB). Yield losses of up to 50 % have been reported in some cases.



BLSD. Symptoms on the lower leaf surface

1. Stage 2
2. Stage 3
3. Stages 4 et 5



Disease distribution

M. musicola was first identified in Java in 1902. Since 1962, it has been reported in most of the world's banana growing areas, making SD one of the most important epidemic plant diseases. BLSD was first recognised on the South-eastern coast of Viti Levu in Fiji in 1963. Subsequently, the disease has been reported throughout the Pacific (Torres Strait & Cape York Peninsula region of Australia, Papua New Guinea, Solomon Islands, Vanuatu, New Caledonia, Norfolk Island, Federated States of Micronesia, Tonga, Western Samoa, Niue, Cook Islands, Tahiti and Hawaii). BLSD has also been found in Asia (Bhutan, Taiwan, southern China including Hainan Island, Vietnam, Philippines, West Malaysia and Sumatra in Indonesia). However, the distribution of the disease in Southeast Asia requires further clarification, especially in the Indonesian archipelago. BLSD was identified in Latin America for the first time in 1972 in Honduras. It spread northwards to Guatemala, Belize and southern Mexico and southwards to El Salvador, Nicaragua, Costa Rica, Panama, Colombia, Ecuador, Peru and Bolivia. BLSD has recently been reported in Venezuela, Cuba, Jamaica and Dominican Republic from where it threatens the rest of the Caribbean. In Africa, the first records were in Zambia in 1973 and in Gabon in 1978. BLSD spread along the West coast to Cameroon, Nigeria, Benin, Togo, Ghana and Côte d'Ivoire. The disease occurs in the Congo and its eastward spread most likely occurred across Democratic Republic of Congo (ex Zaire) to Burundi, Rwanda, Western Tanzania, Uganda, Kenya, and Central African Republic. An introduction occurred on the island of Pemba around 1987 and BLSD spread from there to Zanzibar and coastal areas of Kenya

and Tanzania. BLSD is also found in Malawi and the Comoro Islands. The present distribution is undoubtedly underestimated.

BLSD and SD are disseminated locally by ascospores and conidia. Long distance spread is believed to be by the movement of germplasm (infected suckers, diseased leaves) and wind-borne ascospores. In the Pacific and in lowland areas of Latin America and Africa, symptoms of SD are now rarely observed and this disease has been supplanted by BLSD. In plantations in Honduras, BLSD was reported to have replaced SD in less than 3 years. However, SD could still be found in lowland areas of the Philippines 26 years after the introduction of BLSD. SD is more adapted to cooler temperatures and is often dominant at altitudes over 1200-1400 m where BLSD is rarely seen.

Control measures

Systemic fungicides provide effective control of BLSD in commercial plantations but their effects on the environment are cause for concern. Although frequency of spray applications can be appreciably reduced when used in conjunction with disease forecasting systems, resistance to some of these fungicides have developed both in *M. fijiensis* and *M. musicola* strains in the Caribbean and Central America. Genetic resistance to BLSD and SD is clearly the best long-term goal for disease control especially for small-holders who cannot afford to purchase chemicals.

4. SD. Conidia of *Pseudocercospora musae*.

5. SD. Stage 5 symptoms. Isolated necrosis on the upper surface leaf.

6. SD. Plantain necrosed leaves in Highlands.



The observed behaviour of banana towards BLS D may be classified into two types of interactions :

✓ **Compatible interaction**

Compatible interactions are characterized by a full development of the disease with necrosis and sporulations. A gradient can be observed within this phenotype. Some bananas show a susceptibility reaction to BLS D comparable to that of cv. Grande Naine (AAA, Cavendish).

The development of the disease from stage 1 to necrosis is rapid. The sporulation rates can reach a high level if the climatic conditions are favourable for a rapid development of the disease. By the time the bunches are harvested, the plant generally has few, if any, functional leaves. Other bananas shown partial resistance from a moderate level (Pisang Berlin, Pisang Mas) to a very pronounced level comparable, in this case, to that of cv. Pisang Ceylan (AAB, sub-group Mysore) or of cv. Fougamou (ABB, sub-group Pisang Awak). In situations of pronounced partial resistance, the development of the disease from stage 1 to necrosis is slow and there is low sporulation. This results in a large number of functional leaves at the time of harvest. A more precise characterization of partially resistant cultivars is now being conducted under controlled conditions in order to make estimations of the various infection cycle parameters of the pathogen.

✓ **Incompatible interaction**

Bananas classified in this category show very pronounced resistance behaviour comparable to that of Yangambi km5 (AAA, sub-group Ibota). In this phenotype, the development of symptoms is blocked and there is no sexual and asexual sporulation. A microscopic study showed that the defence reactions brought into play by the host begin just after the penetration of the fungus in the stomata. The behaviour of bananas classified in this phenotype seems very similar to the hypersensitivity reactions observed in other host-pathogen systems. The variable reactions of several highly resistant banana varieties artificially inoculated with *M. fijiensis* isolates, suggests the existence of specific interactions. The breakdown of the banana variety Paka has already been observed in a Pacific Island (Rarotonga), which may indicate that this type of resistance can be more easily overcome by the pathogen and consequently is not durable.

The inheritance of the BLS D resistance from the wild diploid Calcutta 4 has been studied using crosses between this diploid and susceptible triploid plantains. A genetic model including one major recessive allele and two independent alleles with additive effects is suggested (IITA). Progeny of crosses made between wild diploids (AA) are now being studied in order to better understand the inheritance of BLS D resistance and to localize genes or quantitative trait loci (QTLs) implicated in this resistance using genetic maps.

Breeding for resistance

The introduction of BLS D resistance to bananas and plantains by conventional breeding programmes is based on the



BLS D. Stage 6 symptoms:

7. Isolated necrosis on the upper surface leaf. Climatic conditions are not very favourable for the development of the disease. Reduced symptom density.

8. Coalescing necrosis on the upper surface leaf. Climatic conditions are favourable for a rapid development of the disease.

9. BLS D. Complete necrosis of the leaf system of a Cavendish (AAA) banana before harvesting stage.

10. BLS D. Conidiophore of *P. fijiensis* with two conidia.

use of the resistance existing in wild *Musa* species, especially *M. acuminata* ssp. *burmannica*, ssp. *malaccensis* and ssp. *siamea*, and in diploid cultivars such as “Paka” (AA) and “Pisang lilin” (AA). Hybrids from breeding programmes are being tested in the field at selected sites around the world in the International *Musa* Testing Programme (IMTP) organized by INIBAP. Although a BLS D resistant “Cavendish” type dessert banana has still to be developed, much progress is being made in the improvement of *Musa* types for local consumption. In the Phase I of IMTP, three tetraploids from the *Fundación Hondureña de Investigación Agrícola* (FHIA) programme were identified as having resistance to BLS D and potential for cultivation in many countries. FHIA-01 and

FHIA-02 are dessert bananas with an apple or acidic flavour. FHIA-03 is a robust cooking banana which may also be eaten as a dessert banana when fully ripe. Virus tested, *in vitro* plantlets of these genotypes are available from INIBAP on request.

Knowledge of the extent and distribution of variability within the two pathogens is necessary to make the genetic improvement of banana more efficient through the introduction of durable resistance. Important genetic variability among populations of *M. fijiensis* from different geographical regions (Southeast Asia including the Philippines and Papua New Guinea, Pacific Islands, Africa and Latin America) has been identified using DNA restriction fragment length polymorphism (RFLP) markers. The highest levels of allelic diversity were found in the two Southeast Asian populations. Most of the alleles (> 88 %) detected in Africa, Latin America and Pacific Islands populations were also detected in Southeast Asian populations. These results were consistent with the hypothesis that *M. fijiensis* originated in Southeast Asia and spread recently to other parts of the world. The level of allelic diversity in *M. fijiensis* populations from regions other than Southeast Asia was found to be considerably less, indicating founder effects. However, the level of diversity detected in Latin America, Pacific Islands and Africa remains large and might be sufficient for rapid adaptations to newly introduced resistance. Gametic disequilibrium analysis among RFLP loci have shown that genetic recombination plays an important role in *M. fijiensis* population structure. Thus, combining specific resistance genes within individual cultivars (pyramiding) may not be durable and the use of variety mixtures or partial resistance might be more appropriate. Finally, founder effects have also led to marked genetic differentiation between geographic populations, suggesting occasional *M. fijiensis* migration events between continents. With appropriate quarantine measures, Southeast Asia, Africa, Latin America and Pacific Islands could be considered as separate epidemiological units requiring independent disease

management. Studies of the population structure of *M. musicola* revealed genetic diversity in a sample set of Australian and overseas isolates with many alleles present at each RFLP locus.

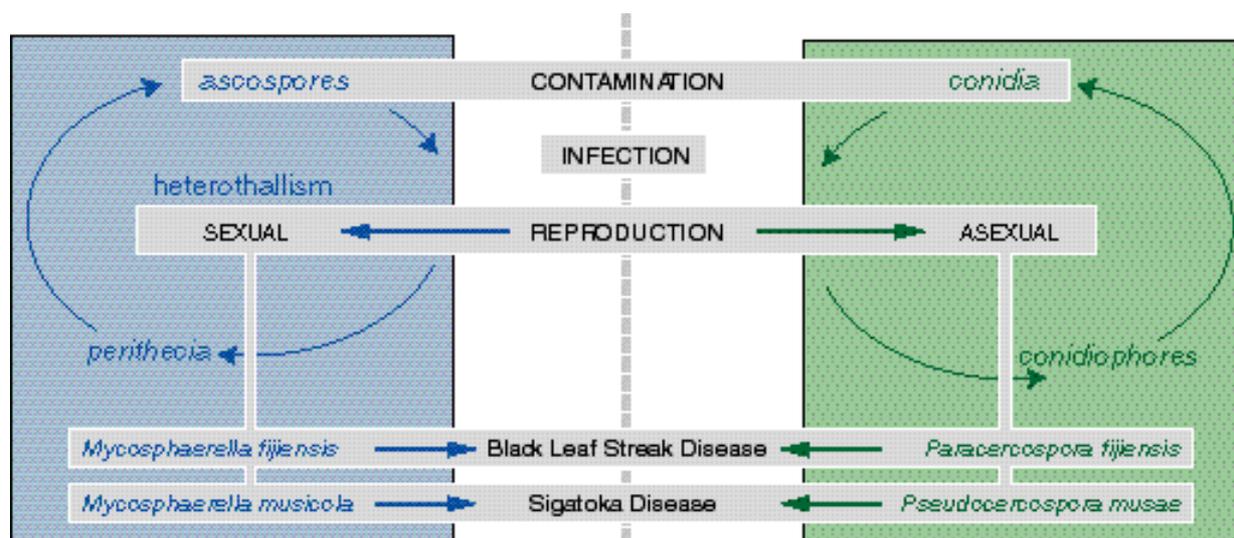
Research needs

Population studies of the two pathogens are required at the continental level to determine if the different banana-producing regions correspond to only one or several epidemiological units. These studies should be conducted using molecular markers and pathogenic tests. Techniques also need to be developed through which host-pathogen interactions can be quickly and reliably ascertained in controlled conditions. Pathogenic variability among genetically differentiated populations of the two pathogens could then be evaluated by using a standard set of *Musa* cultivars. Population studies should help to define, for the different regions, a set of isolates that represent the pathogenic variability in terms of virulence and aggressiveness for screening material for resistance.

In addition to pathogen populations studies and genetic analysis of BLS and Sigatoka resistance, epidemiological studies are also required for resistance breeding efforts. It is particularly important to know which specific components of the partial resistance greatly reduce the rate of the disease development in the field. Finally, evolution of the pathogen population in response to the selective pressure exerted by resistant cultivars should be evaluated to adjust control strategies for durable resistance.

You are invited to contribute to the on-going research by sending necrosed leaf samples. To obtain more accurate information on the on-going research and on the way to send samples, please contact Jean Carlier at CIRAD-AMIS, Laboratoire de pathologie végétale (Programme BBPI), BP 5035, 34032 Montpellier Cedex 1, France (e-mail: carlier.j@cirad.fr).

Infectious cycle of *M. fijiensis* and *M. musicola*



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