# Banana diseases and pests

## Panama disease

Panama disease or Fusarium Wilt was first identified in 1874 in Australia. It is now observed in almost all tropical and subtropical banana production zones. It is caused by a soil fungus of a very common genus, *Fusarium oxysporum* sp. *cubense* (FOC).

Different races have been identified. Under certain conditions (soil type, climate, crop intensification, drainage, etc.) each can cause serious vascular damage to the different banana varietal groups, making them practically non-productive.

- **Race 1** originated in Asia and spread widely via movement of plant material in the form of suckers when the major export banana cultivation areas were established in the early twentieth century. It caused by the progressive disappearance of production of the Gros Michel variety in the Caribbean and Latin America in the 1940s and 1950s, when the variety formed the basis of international trade. Gros Michel was replaced in the industrial plantations by the resistant Cavendish varieties discovered in South-East Asia and that are now the fruits traded internationally. It should be noted that Gros Michel is still the reference for dessert banana consumption in most African and Latin American countries; production is still substantial at approximately 6 million tonnes per year. It appears that race 1 is not active in the areas in which it is cultivated extensively and combined with other varieties and other crops (hence at low density). Experiments conducted in Colombia have shown that Panama disease gains importance when the growing of Gros Michel is intensified (density greater than 1 000 plants per ha).

Gros Michel

Panama disease

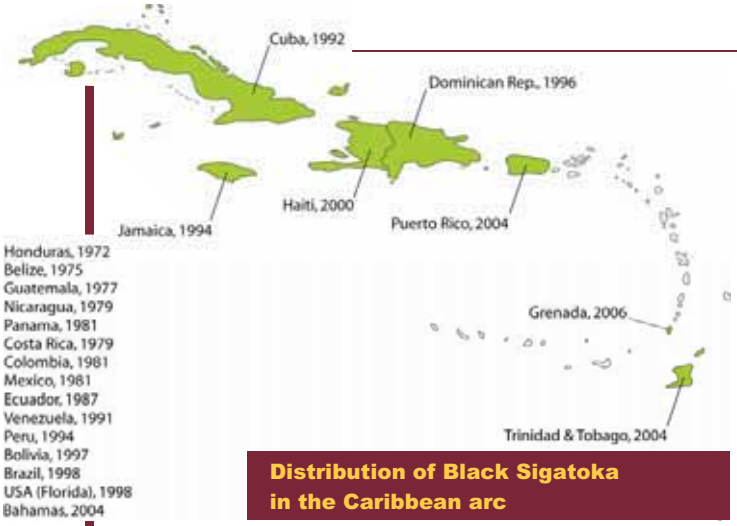
- **Race 2** affects the Bluggoe subgroup (ABB, cooking bananas).
- **Race 3** affects *Heliconia* spp. and sometimes Gros Michel.
- **Race 4**, identified in the Canary Islands in 1931, affects the Cavendish group sporadically and under certain environmental conditions but only in subtropical zones (Canary Islands, South Africa, Taiwan, Australia) where it is relatively well controlled by the appropriate cultural techniques (buffer zones, fallow, etc.).
- **Race T4** was described recently (1995) and also affects Cavendish group varieties but only in a few tropical areas—Indonesia (Sumatra and Java) and Malaysia.

All the specialists agree that the main cause of the spread of the disease is the movement of plant material (suckers and corms) from susceptible, infected plantations. Contamination via the soil from an infected area is very slow.

### Prevention and control

As for numerous soil pathogens, control methods are limited and consist essentially of keeping areas containing the outbreaks in quarantine. Not much international work is performed on this disease whose study is complicated. Control methods are not specific to bananas and are and will remain very limited. Conventional genetic improvement remains an important and as yet little-explored pathway.

International awareness of the importance of respecting rules for the movement of germplasm and the wide adoption of tissue culture plants by the banana industry should limit the present risks. The dispersion of race T4 is under surveillance. However, with strict control of germplasm movement and the surveillance and eradication of infected plants, the prospect of rapid spread of the disease is very improbable.




**Distribution of Black Sigatoka in the Caribbean arc**

## Sigatoka leaf streak diseases

Two main types of leaf streak disease endanger the banana industry: Black Sigatoka and Yellow Sigatoka. A new species called *Mycosphaerella eumusa* is even more aggressive than Black Sigatoka and seems to be spreading in Asia and the Indian Ocean. Black Sigatoka (also called black leaf streak disease or BLS) is caused by the fungal leaf parasite *Mycosphaerella fijiensis*.

Spread is from plant to plant in continental zones. The sea is a natural obstacle. Although the risk of natural dissemination of the spores of the fungus by wind cannot be ruled out, the spread of the disease from one zone to another is generally the result of uncontrolled movement of plant material. The disease is present in all the producer countries in Latin America, Africa and Asia. The Caribbean countries were long protected by their island geography. The new feature that strongly increases the risk for the Lesser Antilles is the spread of the disease in the Greater Antilles in Cuba, Jamaica, the Dominican Republic, Haiti, Puerto Rico, Grenada and Trinidad & Tobago. The fungus destroys the foliage of banana plants. The disease appears in the form of small black streaks that soon develop into necrotic patches. The spread of lesions causes the total destruction of banana leaves before the bunch is harvested, with the fruits being at an advanced stage of ripeness making them unsaleable.

The process is exactly the same as that of Yellow Sigatoka, another fungal disease observed for about 60 years in all the continents. This is caused by the fungus *Mycosphaerella musicola* and led to rational chemical control set up by professionals in Martinique and Guadeloupe. Spraying is performed in relation to surveillance of the disease. Today, Yellow Sigatoka is controlled with a small number of sprays (five to seven per year). There are fundamental differences between the two leaf streak diseases. Unlike Yellow Sigatoka, Black Sigatoka can infect both export banana and plantain. As it spreads rapidly, it is also more difficult to control. Depending on the country and control facilities and techniques, control requires from 12 to more than 50 sprays per year.

### Two control strategies

The export banana plantations in the major Latin American producer countries form vast agro-industrial complexes in alluvial plains. Given the size of plantations (several hundred or even several thousand hectares), contamination from outside is weak. There are no nearby centres of infection. The agroclimatic homogeneity makes it possible to organise and rationalise crop spraying for large complexes. The low cost of labour facilitates essential control work (regular deleafing).

In this context, the impact of spraying as a nuisance is not always taken into account by the large companies that do not hesitate to use systematic control strategies leading to more than 50 sprays per year. Application is at regular intervals and generally consists of contact fungicides (chlorothalonil, dithiocarbamate, etc.) that by definition are of low efficacy—treatment every 10 to 15 days—requiring a large number of sprays to control the disease. Systemic fungicides are sometimes used but always as a water-based emulsion.

CIRAD has developed a rational strategy using warning methods based either on disease monitoring in the plantation or on the observation of climatic descriptors (evaporation, temperature, etc.). It has been applied in particular in Guadeloupe, Martinique, Cameroon and Côte d'Ivoire. It consists of performing spraying only at the appropriate moment. The main objectives are:

- improving control efficacy while decreasing the number of sprays per year;
- limiting the risks of the selection of fungicide-resistant races;
- reducing pollution and increasing respect for human health and the environment (urban centres, rivers, water bodies, reservoirs, etc.).

The strategy is also based on the rational alternate use of systemic fungicides (benzimidazoles, triazoles, etc.) that are effective for a long time. Mixing them with a low volume (13 to 15 litres per ha) of petroleum oil (also fungistatic) extends the efficacy of each spray and therefore helps to reduce the number of sprays per year. These two types of leaf streak control strategy have similar efficacy. However, the consequences are totally different with regard to the appearance of resistance in the fungus.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**Black Sigatoka**

The systemic fungicides available on the market have a single-site effect on the pathogen, enhancing the inducing of resistant fungal strains when these substances are used in excess. In Central America, resistance to benzimidazoles was observed only two years after their first utilisation. This led to greater use of contact products, with 15 to 40 kg active substance per hectare per year. Warning techniques and a reduced number of sprays resulted in the appearance of resistance phenomena in Guadeloupe, Martinique, Cameroon and Côte d'Ivoire only after 10 or even 15 years of use.

### New control methods are essential

Present control strategies cannot be used indefinitely. Thought should soon be focused on the adopting of an overall approach combining new hybrids resistant to the leaf streak diseases and cropping systems that make it possible to conserve this resistance.

# Bacterial diseases

Bacterial diseases are an increasing concern for growers because of the way in which they spread and the lack of resistant varieties.

Moko disease

## Moko disease

caused by *Ralstonia solanacearum* (biovar 1 race 2) formerly *Pseudomonas solanecearum*

Two types of symptoms are observed depending on whether the bacterium is spread via the soil or by a machete or by insects that visit male flowers or their scars after abscission. Upward bacterial colonisation results first in chlorosis and the wilting of the three youngest leaves and then the death of the plant. A cross section of the pseudostem (or corm) reveals reddish-brown colouring of the vascular vessels. The presence of abundant bacterial exudate is a further sign of bacterial infection. If the contaminated plant bears a fruit bunch, the bacterium colonises all the vascular bundles of the fruits via the rachis. Accumulation of ethylene may cause the premature yellowing of the fruits and cross sections display serious browning. When the bacterium is spread by a machete after the cutting of the pseudostem, the contaminated suckers blacken and become stunted in 2 to 4 weeks. The disease was described for the first time in Trinidad in 1910 and is still absent from the Lesser Antilles, except in Trinidad and Grenada. In contrast, it spread rapidly in the Amazon basin in Brazil and in eastern Peru, going as far as northern Guatemala and southern Mexico. It covers a large geographic area. Moko disease spread to the Philippines in 1968 via plant material. There are no resistant varieties or chemical control methods. Only eradication and quarantine give results.

## Bacterial wilt

Banana Xanthomonas Wilt (BXW), Banana Bacterial Wilt Disease (BBW), caused by *Xanthomonas campestris* pv. *musacearum*

The symptoms are observed above all on the emergence of spear leaves, especially at flowering. Flower bracts become discoloured and the male bud blackens and shrivels. The leaves yellow, wilt, blacken, dry and crumble (including the pseudostem). Yellow or brown vascular streaks are observed throughout the plant together with pale bacterial secretion on a section at the base of the pseudostem or at the corm. This causes bunches to wilt, with premature maturation and a reddish brown colour inside the fruit. The plant dies within a month of the appearance of any of these symptoms (one month after infection). The disease is spread by foraging insects, infected plant material (suckers, bunches and leaves), tools and man, and also by animals, run-off, rainwater splashes and wind. There are no resistant varieties. Control is by a six-month quarantine period and the destruction of infected plants and those nearby. Free movement of animals is forbidden. This wilt was observed and described in enset in Ethiopia in about 1968 (this affected the staple foodstuff of 12 million people), and then in Uganda where it has spread since 2001 (75 km per year). Uganda is the second largest banana producer with 10.5 million tonnes (250 to 450 kg per person) and this had decreased by nearly 40% in 2006. Spread has been rapid, with the disease reaching the Congo in 2004, Rwanda in 2005 and Burundi, Tanzania and Kenya in 2006.

# Virus diseases

Virus diseases of banana (dessert and cooking fruits) have spread increasingly in recent years as a result mainly of the ease of plant movement and demand for diversification. They consist of banana bunchy top disease and mosaic diseases including banana mosaic, banana streak disease and bract mosaic. The economic damage varies, affecting all cultivated bananas and both large estates and village plantations. Banana bunchy top disease (caused by the banana bunchy top babuvirus, BBTV) can cause losses of 90 or even 100 percent of production. Banana streak disease (caused by the banana streak badnavirus, BSV) causes losses of 40 to 60 percent, and banana bract mosaic (caused by the banana bract mosaic potyvirus, BBrMV) results in losses of more than 40%.

Spread is either by vector from outbreaks or by the use of infected germplasm—suckers or tissue culture plants—or, in the special case of BSV, from so-called 'silent' bananas with a virus sequence incorporated in the genome of the species *Musa balbisiana* and capable of producing viral particles as a result of stress.

## Banana bunchy top disease (BBTV)

The plants are markedly stunted and rosetted at the top. The narrow, erect, brittle leaves display strongly chlorotic borders. The characteristic symptom is the appearance of discontinuous dark green streaks along the pseudostem, the main leaf vein and the secondary veins. When the mother plant is infected, so are all the suckers. The most effective vector is the banana aphid *Pentalonia nigronervosa*.

## Mosaic diseases

**Banana mosaic**
caused by cucumber mosaic cucumovirus (CMV)

Infected plants display leaf chlorosis and mottling of the main vein and the pseudostem. Secondary infections may appear in the form of bacterial rots in the sheaths forming the pseudostem. The virus can be spread by a broad range of aphids. The disease can also be spread by pruning tools.

**Banana streak disease (BSV)**

The leaf lamina displays discontinuous yellow streaks that rapidly become necrotic. The main vein is unaffected. In severe forms of the disease, the cigar tip becomes necrotic and the plant dies. If the mother-plant is infected so are all the suckers.


© Thierry Lescot
Banana streak disease

The disease is transmitted by mealybugs—*Planococcus citri*, *Saccharicoccus sacchari* and *Dysmicoccus brevipes*. In recent years, BSV infections unrelated to external contamination have been described in various parts of the world. There are two different causes: tissue culture plants derived from micropropagated healthy interspecific hybrid varieties of banana and the hybrid progeny of crosses between healthy *Musa acuminata* (genome A) and *Musa balbisiana* (genome B) parents. Various abiotic stresses cause the appearance of the disease in these hybrids, correlated with the presence in the genome of the *M. balbisiana* parent of endogenous viral sequences of BSV containing all the information required to synthesise the infectious virus.

**Banana bract mosaic (BBrMV)**

The first stages of infection consist of greenish yellow streaks turning into brownish red necrosis on the leaf lamina and veins. Yellow mottling or whitish streaks are seen on the pseudostem according to the variety infected. Bract mosaic is the final symptom. The disease is transmitted to all the suckers by aphids (*Ropalosiphum madiis*, *Myzus persicae*).

## Prevention and control

The only control method available today to fight these banana virus diseases is control of the vector and the use of healthy plant material. Indeed, there are no bananas with natural resistance to these diseases and no cure other than eradication after a virus attack.

The procedure to be followed is based mainly on the use of disease-free germplasm—suckers or tissue culture material screened for viruses—and the cutting back of weed growth where aphids multiply.

# Banana borers

Originating in South-East Asia, the banana borer has spread to all subtropical and tropical banana and plantain production regions. The insect (*Cosmopolites sordidus*) is 9 to 16 mm long and 4 mm wide. It moves freely in the soil at the feet of banana plants or in plant debris. It is nocturnal and very sensitive to drying. The pest is spread mainly via infested plant material. The adults do no damage. The females lay eggs in the banana rhizome and the larvae feed on this, driving tunnels. These tunnels disturb water and mineral supply of plants, lengthen the production cycle, cause serious decreases in yield and weaken the anchorage of the plants, making them more sensitive to wind. Strong attacks can lead to the death of the plant. In addition to classic chemical treatment, the use of healthy planting material (tissue culture plants) used in clean soil (after fallows) is a method of borer control. New borer trapping methods using pheromones are available. A control system combining entomophagous nematodes and sordidin traps is being developed.

However, the banana borer remains a major pest constraint for banana crops—whether on industrial plantations or smallholdings. It seems fairly unlikely that improved varieties can be bred rapidly. Control at the farm scale based on the use of traps and the maintaining of low levels of inoculum are being studied and may in time form an alternative to chemical control.


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Banana borer on a corm

# Nematodes

Nematode

Numerous nematode species parasitise banana roots and corms. Root knot nematodes (*Meloidogyne* spp.) and spiral nematodes (*Helicotylenchus* spp.) are found all over the world in all kinds of crop. However, the most damage is caused by the migrating nematodes *Pratylenchus* spp. and *Radopholus similis*. The latter species is found everywhere in the hottest banana growing zones and especially in intensive plantations where it arrived via germplasm movements during the spread of the crop during the past two centuries. *Pratylenchus coffeae* is also present in the hottest zones but is generally indigenous and found mainly on plantain crops. *Pratylenchus goodeyi* prefers cooler areas and originated on the Africa plateaux. It is observed in certain subtropical zones such as the Canary Islands, for example.

## Underground enemies!

*Pratylenchus* spp. and *Radopholus similis* are migratory endoparasites whose full biological cycle lasts for 20 - 25 days in root and corm tissues. Juvenile forms and females are always mobile and can leave the roots when conditions are no longer favourable. These migratory forms can then colonise other roots. As they move within and between cells, these nematodes feed on parenchyma cell cortical cytoplasm, destroying cell walls and creating tunnels that become necrotic and can extend to the whole of the cortex. Root and corm necrosis is accentuated by other pathogens (fungi and bacteria). In particular, fungi of the genus *Cylindrocladium* are strongly pathogenic and can cause lesions similar to those made by nematodes. The combination of the two pests causes very serious damage. The destruction of underground tissue leads to a decrease in water and mineral nutrition resulting in slowed plant growth and development. This can lead to severe decrease in bunch weight and lengthen the period between harvests. Furthermore, destruction of the roots weakens the anchorage of the plants in the ground and increases the risk of toppling, especially during hurricane periods, with a strong economic impact.

## Prevention and control

Control methods in intensive plantations are still largely dominated by application of chemicals (mainly organophosphorus compounds and carbamates) that carry substantial sanitary and environmental risks. For this reason, in spite of their efficacy and very easy application, their use will be increasingly limited in favour of alternative control measures. These include cultural practices improving fertility (tillage, irrigation, organic ameliorators, etc.) that indirectly improve plant tolerance to pest pressure. More direct methods such as the use of fallow and the planting of micropropagated bananas are now in common use and lead to a strong decrease in nematode populations (cf. Phytoma No. 584, July-August 2005).

These methods are widely used by growers in Martinique and Guadeloupe, where they have contributed to a 50-percent reduction in pesticide spraying in the past ten years.

Operations involving biological antagonists, root symbiots (mycorrhizal fungi) and especially genetic resistance may allow the setting up of increasingly effective integrated control strategies in the fairly near future. However, it is necessary to be aware that the great complexity of nematode populations makes delicate the development of these more closely targeted techniques. To be effective, they must be able to handle the diversity of cultural and ecological situations.

# Post harvest diseases

Storage diseases (wound anthracnose, ripe-fruit (quiescent) anthracnose and crown rots) strongly limit the sale of exported bananas. *Colletotrichum musae* causes both forms of anthracnose, while crown rots result from a larger parasite complex consisting of *C. musae* but also other organisms: *Fusarium*, *Verticillium*, *Botryodiplodia*, etc.


Ripe-fruit anthracnose

Distinction is made between two forms of anthracnose:

- **ripe-fruit (quiescent) anthracnose**: brown lesions develop on fruits after ripening and subsequently in the sales channel. This disease rarely has serious commercial consequences.

- **wound (non-quiescent) anthracnose**: broad brown lesions occur on fingers wounded during harvesting or packing. The symptoms are observed when fruits are unpacked after sea transport and have serious commercial consequences.

Wound anthracnose

**Crown rots** are fungi that spread from cut surfaces when fruits are prepared at the packing stage. This damage is also visible after sea transport and has serious commercial consequences.

**The fungi** that cause post-harvest diseases are widespread in banana plantations and hence on bunches if these are not protected. In other words, control of infection begins when the inflorescence shoots at the top of the leaf cluster. Anthracnose results mainly from contamination by *Colletotrichum musae* in the field. It is not possible to detect infected fruit with the naked eye at harvesting but a test can be performed more than three weeks before cutting. Fruits are infected mainly during the first month of flowering. Spores are spread by water and develop on the organs when they start to decompose (old leaves, bracts and above all flowers). Control of the disease must begin in the field and then continue in the packing shed.

Treatment by swabbing

Hands can be contaminated by crown rot at various stages in the chain. This greatly complicates the implementation of control measures, but hand contamination by washing water is probably the main cause.

Treatment by spraying

Chemical control of these diseases does not always give satisfactory results. Indeed, it is sometimes ineffective according to the production zone and the time of the year and resistance to fungicide has developed in the various fungal species involved. Finally, interest in developing methods other than chemical control is increasing. Indeed, these post-harvest treatments raise two crucial problems—the risks of residues in fruits and the processing of the fungicide preparations discharge near packing stations.

Crown rots

Healthy crown