Banana pests and diseases

by Eric Fouré and Luc de Lapeyre de Bellaire

**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 the soil fungus *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, which are now the fruits traded internationally. It should be noted that Gros Michel is still the benchmark 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 ground when the growing of Gros Michel is intensified (density greater than 1 000 plants per ha).

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.).

Tropical Race 4. Following on from Colombia in August 2019, in early 2020 the French island of Mayotte, situated in the Indian Ocean off Mozambique, became the 19th spot officially contaminated by TR4 (tropical race 4) fusarium wilt. It is a relatively recent form, described in 1990. It affects Cavendish group varieties, but hitherto had only been found in the wet tropical zones of Asia, especially Taiwan, Indonesia, Malaysia, South China, Australia and the Philippines. In 2011, FruiTrop published a full set of recommendations (see FruiTrop no.191, July-
August 2011, pages 20 and 21), to be followed very closely in order to apply effective preventive measures. An ad-hoc committee of scientists specialising in this disease was formed in order to investigate the origin of its introduction and analyse the risks of extension. This alarming news has reactivated the world phytosanitary monitoring networks, particularly in Latin America.

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 being performed on this disease, study of which 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 TR4 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.

SIGATOKAS

Sigatokas are banana plant foliar diseases caused by pathogenic fungi of the genus Mycosphaerella. There are three distinct types of sigatoka: yellow sigatoka, caused by M. musicola, black sigatoka (or black leaf streak disease – BLSD) caused by M. fijiensis, and a third form still little known and restricted in scope (India, Nigeria) caused by M. eumusa.

Due to its geographic distribution and aggressiveness, BLSD is the most worrying form of sigatoka, and one of the main production constraints on the export banana worldwide. Unlike yellow sigatoka, it also affects plantain plants. First detected in the early 20th Century on the Pacific islands of Fiji, BLSD is now present in all producer countries in Central and Latin America, Africa and Asia (except India), where it has gradually replaced yellow sigatoka. The Caribbean zone was long spared thanks to its insularity. Yet BLSD was detected in Saint Vincent and French Guiana in 2009, and then Saint Lucia in early 2010, in Martinique in September 2010 and finally in Dominica and Guadeloupe in early 2012. So the whole of the West Indies is now affected by the disease. Worldwide, only the Canaries are still free from BLSD, as well as Australia which has eradicated it several times.

BLSD is manifested by lesions on the leaves, which can very rapidly develop into necrotic streaks. The reduction of the foliar surface area of the banana plant before harvesting the cluster can therefore significantly reduce yields. However, the major effect of BLSD in export cultivation is early maturation of the fruits, which become unexportable unless suitable control methods are applied. It is disseminated mainly by wind, over distances ranging up to tens of kilometres, making large-scale control a must in order to be effective.
A variety of management strategies

In most export dessert banana production zones, control of the disease is based on regular applications of chemical fungicides by aerial or ground-level application. However, the management strategies vary greatly depending on the situations, and can lead to highly variable treatment levels.

In 1970s in the French West Indies, CIRAD alongside professionals from Guadeloupe and Martinique, developed a rational management strategy based on a biological forecasting method based on regular observation of the disease and analysis of climate descriptors. This strategy makes it possible to monitor the dynamic of the disease and activate the treatments only when they are necessary. Its main benefit therefore is limiting the number of treatments, while ensuring optimum control of the disease. Thus yellow sigatoka has been controlled for more than 40 years, with a limited number of treatments (five to seven on average per year). The lasting success of this method was also based on organisational aspects, since management was centralised by a technical unit responsible both for decision making (application date, product selection), but also execution of aerial treatments over homogeneous treatment zones. This method was then applied to BLSD control in the FWI, but also in other parts of the world, where it ensures rational and effective control of the disease.

This strategy is based on using systemic fungicides with a powerful curative effect used in regular rotations between various active ingredient families, and mixed with paraffin oils whose fungistatic effect reinforces the curative effect of the treatments. Three main chemical families of systemic fungicides were employed due to their powerful curative effect: triazoles, strobilurins and benzimidazoles, as well as a new family (SDHIs) which has just come onto the market. Other fungicides with a lesser curative effect (morpholines and pyrimidines) have also been employed in these strategies. Unfortunately, due to their mode of action, these fungicides are particularly sensitive to development of pathogen resistance. Repeated use of these products can therefore promote the progressive emergence of pathogen strains less sensitive to their action, eventually threatening the effectiveness of these strategies. This dynamic depends on whether these products are used rationally, but the very high adaptability of the pathogen means an inevitable eventual loss of effectiveness of these management strategies if they are based solely on fungicide use.

In Guadeloupe and Martinique, losses of sensitivity to benzimidazoles started to appear after ten years of rational use (though with no alternation) by means of a biological forecasting system. Triazoles had a longer useful life (twenty or so years for yellow sigatoka), since they were alternated with other fungicide families as they first came onto the market. However, sensitivity to these fungicides for control of yellow sigatoka rapidly deteriorated when only triazoles could be employed, with the other fungicides taken off the market. Fortunately, the strains of *M. fijiensis* that had invaded Guadeloupe and Martinique were sensitive to all groups of fungicides. Now only two triazoles and one strobilurin are approved in France. Since the risk of resistance is very high for strobilurins (1 treatment per year, 2 at most), triazoles remain the most frequently employed. Four to five years after the arrival of BLSD to the FWI, the sensitivity levels of the fungus to these various fungicide families remain good, although a slight decline in sensitivity to triazoles has been observed in Martinique. In Cameroon and Côte d’Ivoire, the forecasting methods for BLSD were able to limit the number of applications between 12 and 14 per year for a decade, until the progressive emergence of resistant strains in certain zones. Conversely, in Latin and Central America, these declines were much faster. Resistance to benzimidazoles, used on a massive scale when they came onto the market, was observed...
just two years after they were first used. The same phenomenon was then observed in these production zones when triazoles appeared, and then strobilurins; hence nowadays they are used very sparingly due to their low effectiveness.

In these situations, the implementation of rational strategies is no longer possible, and a strategy of systematic management has been progressively applied, based on use of contact products with a preventive effect. The main ones include chlorothalonil or mancozeb. They must be applied very regularly to ensure their effectiveness, often in mixture with systemic fungicides, with sometimes more than 50 applications per year. The doses of active substance used have also increased massively, from 2 to 4 kg of active substance per year with forecasting methods, to 30 or even 70 kg in systematic strategies. So use of these strategies represents an economic, logistical and above all environmental cost incompatible with many production zones, especially in the FWI.

The FWI also has to cope with a particularly limiting context in terms of implementing chemical management, even if rational. Current European legislation strictly limits the range of products usable, with only three fungicides used today: two triazoles and one strobilurin. While two new products are set to be approved, the implementation of optimum rational management remains highly constrained. On the other hand, the prohibition of any aerial treatment in Guadeloupe and then Martinique in 2013 has also transformed the organisation of disease management in the FWI, previously centralised around an independent technical unit responsible for generalised treatment. Individualisation of disease management and the lack of ground-based treatment equipment are currently threatening the effectiveness of disease control in these areas: the risk of development of resistant strains is becoming higher, with disease management no longer a collective strategy.

**Essential new means of disease management**

The sustainability of chemical disease management, whether rational or systematic, is therefore compromised in the long term. We need to be able to develop new management tools and methods to offer viable alternatives for BLSD control in the various production contexts worldwide. These alternatives will be based on the development of integrated approaches, relying on a combination of various tools enabling the disease and/or its damage to be controlled (losses in terms of fruit yield and quality).

The creation of new hybrid banana varieties with long-lasting resistance to the disease is an avenue long contemplated as an alternative to the Cavendish variety, widely used but particularly sensitive to the disease. However the processes of creating and selecting new varieties take a long time, and the list of agronomic and organoleptic criteria that these varieties have to meet makes the task particularly difficult. The variety CIRAD 925 recently developed in the FWI shows promising potential, currently under study and assessment by the researchers and the French export industry.

Whether or not they are BLSD-tolerant, the varieties cultivated must be incorporated in innovative and sustainable cultivation systems, employing optimised cropping management methods (plant management, inoculum management and control of fruit conservation by defoliation, etc.), potentially making use of biocontrol products, or introducing spatial and temporal modifications to the cropping system, thereby enabling a gradual reduction in use of chemical management.
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**

*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 the planting tools used (machetes, etc.) 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 for example 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. It is controlled by a quarantine period lasting for several months and the destruction of infected plants and those nearby. Free movement of animals is for-
bitten. 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 was 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. The spread has been rapid, with the disease reaching the Democratic Republic of Congo in 2004, Rwanda in 2005 and Burundi, Tanzania and Kenya in 2006.

**VIRAL DISEASES**

Viral diseases of the 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 production losses of 90 or even 100 percent. 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%. It spreads 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 in particular as a result of stress (abiotic phenomena, weather conditions, intensive in vitro or in vivo propagation of plant material, etc.).

**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 the 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.

The disease is transmitted by various mealybug species—*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: 1) tissue culture plants derived from micropropagated healthy interspecific hybrid varieties of banana and 2) 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 (e-BSV) containing all the information required to synthesise the infectious virus.

**Banana bract mosaic (BBrmMV)**

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 maidis*, *Myzus persicae*).

The only control method available today to fight these banana virus diseases is control of the vector and the use of healthy plant stock. 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 stock. The adults do no damage. The females lay eggs in the banana rhizome and the larvae feed on this, digging tunnels. These tunnels disturb water and mineral supply of plants, lengthening the production cycle, causing serious decreases in yield and weakening plant anchorage, making them more sensitive to wind. Severe attacks can lead to the death of the plant. In addition to classic chemical treatment, the use of healthy planting stock (tissue culture plants) used in clean soil (after falling) is a method of borer control. New borer trapping methods using pheromones (sordidin) 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 (plantains are highly susceptible to the banana borer). It seems fairly unlikely that improved varieties can be bred rapidly. Plantation level control based on the use of traps and maintaining low levels of infestation are being studied, and may in time form an alternative to chemical control.
Nematodes

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 over 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 may be aggravated by other pathogens (fungi and bacteria). In particular, fungi of the genus Cylindrocladium are pathogenic and can cause lesions similar to those made by nematodes. The combination of the two pests may cause very serious damage under certain conditions. 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 severely reduced bunch weight and lengthen the period between harvests. Furthermore, destruction of the roots weakens plant anchorage in the ground and increases the risk of toppling, especially during hurricane periods, with a major economic impact.

Prevention and control

Control methods involving the application of chemicals (mainly organophosphorus compounds and carbamates) that carry substantial sanitary and environmental risks are still used in intensive plantations. 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 soil 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 (see Phytoma No. 584, July-August 2005). These methods are widely used by producers in Martinique and Guadeloupe, where they have contributed to a reduction in pesticide use of more than 50 % over the past decade.

In the more or less near future, biological antagonists, root symbionts (mycorrhiza) and above all genetic resistance (by hybridisation or clonal selection) will be employed in setting up increasingly effective integrated protection strategies. However, the great complexity of nematode populations makes it tricky to develop these more targeted techniques. To be effective, they will need to be able to take into account the diversity of the cultivation 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.

There are two distinct 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.

**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.

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.

Chemical control of these diseases does not always yield satisfactory results. Indeed, it is sometimes ineffective according to the production zone and time of 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 fungicide discharges near packing stations.
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