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Disease and Pest Constraints to Banana Production

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Abstract

Diseases and pests of edible banana[1] probably first became problems when certain genotypes were grown as monocultures. Fusarium wilt and Sigatoka leaf spot were the first diseases to limit production of 'Gros Michel' (AAA genome), the first export banana cultivar grown in the Americas. Cultivars in the Cavendish subgroup (AAA genome), which produce the current export bananas, are very susceptible to black leaf streak. This disease has spread globally and replaced the less virulent Sigatoka leaf spot as the dominant leaf spot in most locations. Other fungal diseases important in some regions include the Cavendish-attacking strains of Fusarium wilt, eumusae leaf spot, freckle and Cladosporium speckle. The pathogen causing Moko bacterial wilt originated on native *Heliconia* in the Americas. Introduced banana became a new host. Blood bacterial wilt and Xanthomonas wilt are other bacterial diseases locally important in Indonesia and East Africa respectively. Bunchy top and bract mosaic are damaging virus diseases, which have a limited distribution at present. Virus diseases mosaic and streak are less damaging, but more widespread. Pre- and post-harvest diseases of fruit also cause losses. The burrowing nematode is the most important root parasite of banana in lowland tropical areas, and the most damaging banana insect pest is the rhizome-attacking banana weevil. The development of aseptic shoot-tip culture methods has enabled selected banana germplasm to be multiplied quickly and has reduced many of the risks associated with the international movement of germplasm. A major drawback to breeding for resistance has been the sterility of the popular Cavendish clones. However, genetic engineering and other biotechnology-based approaches to improvement are viable alternative methods. Chemical control has been employed successfully in the past to counter pests and pathogens, but resistance to fungicides and pesticides is becoming a major issue. Biological control methods are being employed more often in integrated pest management programmes.

INTRODUCTION

Edible banana cultivars are believed to have first originated in Melanesia and Southeast Asia through the selection of wild banana species for parthenocarpy and sterility (Simmonds, 1962). During this selection process, further crosses with wild species and the more fertile cultivated genotypes occurred as early cultivars were traded and spread across the Far East and southern Asia. Subspecies of *Musa acuminata* and *M. balbisiana* are the main components of modern banana cultivars in the Eumusa series, which form the most widespread and cultivated of the edible banana types (Simmonds and Shepherd, 1955). Because components of *M. acuminata* subspecies *banksii* and *errans,* which are believed to have originated in the Philippines-New Guinea region, are

[1] The term 'banana' is used throughout the text to include banana and plantain (*Musa* spp.).

found in almost all banana cultivars, these near identical subspecies may be the origin of the first cultivated landraces (Carreel et al., 2002). *Banksii* and *errans* are more susceptible to disease than other *M. acuminata* subspecies and this factor may explain why many banana cultivars are susceptible to pathogens (Jones, 2000). Different *Musa* species have been shown to be components of Fe'i banana cultivars in the Australimusa series which have upright bunches and are only grown locally in Melanesia and the Pacific (Jones, 2000). These banana types seem less prone to disease problems, but they have never been grown in large plantations.

The early cultivation of banana would have occurred in clearings in the rainforest by shifting cultivators and later in gardens mixed with other crops by more settled agriculturists. Many cultivars with differing genetic backgrounds and differing reactions to pathogens and pests would most likely have been grown together. Disease problems under these methods of cultivation would not have been severe enough to influence the selection of disease-resistant clones over susceptible ones. Horticultural considerations were probably paramount and the reason for some clones being preferentially propagated and distributed (Jones, 2000). Disease and pest problems seem to have only emerged when banana began to be planted on a large scale as a monoculture of certain favoured cultivars. This era coincided with advances in the sciences of plant pathology and agricultural entomology.

THE FIRST EPIDEMICS OF BANANA DISEASES

Symptoms of Fusarium wilt, caused by the fungus *Fusarium oxysporum* f. sp. *cubense*, were first described in Australia in 1876 on 'Silk' (AAB genome) (Ploetz and Pegg, 2000). The disease was fatal to 'Silk' and could not be effectively controlled. 'Silk', which was much favoured in South and Southeast Asia, was introduced into the West Indies before 1750 and used as a shade plant in cocoa plantations. Some introductions would most likely have carried *F. oxysporum* f. sp. *cubense*. Thus, Fusarium wilt may have been introduced into the New World. First reports of the disease on 'Gros Michel' (AAA genome) come from Costa Rica and Panama in 1890. It seems likely that the pathogen was distributed on planting material of 'Gros Michel' that was mass propagated for plantation use (Stover, 1962; Ploetz and Pegg, 2000). At this time, large consignments of suckers and rhizomes may also have been transported between countries by multinational fruit companies to supplement local stocks of commercial cultivars, thus encouraging disease dissemination. The scene was set for an epidemic in banana plantations.

A dramatic increase in new country records of Fusarium wilt occurred in the early and mid-1900s. As production on affected 'Gros Michel' plantations steadily declined, new plantations were established on virgin land. However, *F. oxysporum* f. sp. *cubense* was introduced to new areas with planting material and/or in soil attached to machinery, and the process began again. Banana production became shifting cultivation. The first colonial banana breeding programmes in the Caribbean were initiated as a response to Fusarium wilt (Jones, 2000) and were based on improving 'Gros Michel', which possessed some female fertility. However, it was only the introduction of resistant Cavendish cultivars (AAA genome) to replace susceptible 'Gros Michel' in the 1950s and 1960s that saved the industry from collapse (Stover, 1962; Ploetz and Pegg, 2000).

At the same time as the Fusarium wilt problem was at its height in the Americas, another serious fungal pathogen began to make its presence felt on the international scene. Sigatoka leaf spot caused by the fungus *Mycosphaerella musicola* was first recorded on Java in 1902 and was later recognised as a serious problem of dessert banana cultivated in Fiji in 1913. Reaching Sri Lanka by at least 1919, the Philippines in 1921 and Australia in 1924, the pathogen began its subsequent global spread. In the 1930s, the disease was reported throughout the Central American-Caribbean region. It was also found in China, West Malaysia and parts of South America and in East Africa at this time. First reports from Brazil, West Africa and India came in the 1940s. Most other banana-growing areas were affected in the 1950s and 1960s (Meredith, 1970). Sigatoka leaf spot did not kill plants, but affected yields because it caused leaf necrosis, which reduced photosynthetic capabilities. In addition, fruit on or from affected plants ripened prematurely, which was of concern to exporters. Breeding programmes in the Caribbean region turned their attention to developing a Sigatoka leaf spot-resistant banana (Jones, 2000). Vast pipeline systems to deliver Bordeaux mixture at high volumes were laid in export plantations in Central America to control the disease. Later, other fungicides, such as petroleum oil alone or with other chemicals, were developed for low-volume delivery. Benomyl, the first systemic fungicide, was introduced in Honduras in 1967 and was in widespread use by 1972 (Jones, 2000).

Through the use of Fusarium wilt-resistant Cavendish cultivars and fungicides, the export industries had been saved. However, problems still plagued many smallholders, who grew susceptible dessert banana cultivars for local consumption, as they could not afford fungicides to control Sigatoka leaf spot. Luckily, many cooking banana cultivars favoured by people in developing countries had resistance. However, worse was yet to come.

BLACK LEAF STREAK – A POTENT LEAF SPOT DISEASE

Black leaf streak, caused by the leaf spot fungus *Mycosphaerella fijiensis*, was first identified in Fiji in 1963. It was quickly found to be more virulent than Sigatoka leaf spot on Cavendish cultivars and to attack cultivars that were hitherto resistant to leaf spot, such as those in the plantain subgroup (AAB genome). It was also more difficult to control, and more regular applications of fungicide were needed in plantations (Carlier et al., 2000a). Surveys conducted in the Pacific region between 1964 and 1967 showed that the disease was present in many locations including parts of the Pacific Rim. An old herbarium specimen indicated that the pathogen had been present in Taiwan in 1927. Stover (1978) speculated that the pathogen had originated in the Papua New Guinea region, and this was found later to be in the region of its greatest genetic variability (Carlier et al., 2000a).

It was in 1972 that black leaf streak (or black Sigatoka, as it was called locally) arrived in Central America. After its introduction to Honduras, the pathogen quickly spread to neighbouring countries in Central America and by the early 1980s had reached Mexico and Panama. It arrived in Colombia in 1981, Ecuador in 1986, Venezuela in 1991, Bolivia in 1997 and Brazil in 1998. Although protected from aerial spore dissemination by predominant westerly winds, the Caribbean became threatened after the entry of the disease into Cuba in 1992 (Jones, 2003). Since then, the disease has been identified in Jamaica in 1995, the Dominican Republic in 1996, Florida in 1998 (Jones, 2003) and Trinidad in 2003 (Fortune et al., 2005). The Bahamas (Ploetz, 2004) and Puerto Rico (Irish et al., 2006) followed in 2004. It is now only a matter of time before black leaf streak is found in the Windward and Leeward Islands growing areas. Its establishment could threaten the fair-trade, smallholder-based banana export industries of these islands.

The first authenticated record of black leaf streak in Africa came from Gabon in 1978 (Frossard, 1980), though there is some disputed evidence for it being present in Zambia in 1973 (Raemakers, 1975; Dabek and Waller, 1990). The disease spread westwards reaching Nigeria and Ghana in 1986. It was the threat posed by the arrival of this serious leaf spot in countries where plantain was important that prompted the establishment of the International Network for the Improvement of Banana and Plantain (INIBAP) in the mid-1980s. Black leaf streak also spread westward from Gabon to the Democratic Republic of Congo and was reported in Rwanda in 1987, Uganda in 1990 and Malawi in 1992. An independent outbreak was noted on the island of Pemba in the Indian Ocean in 1987, and this resulted in spread to nearby Zanzibar, mainland Tanzania and coastal Kenya in 1988 (Jones, 2003). The disease has reached Madagascar and other islands in the Indian Ocean (Jones, 2003), but has not yet been reported from most of southern Africa. This is most likely related to climatic factors.

Black leaf streak is only being kept under control in commercial plantations in many locations in the Americas by the use of sophisticated fungicide application strategies (Carlier et al., 2000a). Success depends on continued monitoring of fungicide resistance within local populations of the fungus and adjusting the components of spray schedules accordingly. Some areas, such as Atlantic coastal plains of Costa Rica, may be becoming marginal for banana production because of a climate that favours the disease for much of the year and increased costs associated with fungicide application and labour. The future would seem to rely on a shift in production to drier areas with below-canopy irrigation. In other areas, annual or biennial planting with tissue-cultured material, as in Taiwan and parts of China, may be necessary. Bananas are produced under the organic label in the American-Caribbean region, so it may be possible to develop strategies that enables banana to be grown with less fungicide, which is harmful to the environment and plantation workers.

Black leaf streak has reduced the productivity of some traditional banana cultivars in regions where leaf spot was not previously a problem. Among those affected have been the plantains of West Africa and the Americas, and the highland banana cultivars of East Africa. Few clones other than ABB cooking banana cultivars have strong resistance. The disease has undoubtedly contributed to a general decline in yields of individual plants in many parts of the world. It is a major constraint on banana production.

OTHER IMPORTANT LEAF-ATTACKING FUNGAL DISEASES OF BANANA

Eumusae leaf spot caused by *Mycosphaerella eumusae* has very similar symptoms to leaf spots caused by *M. musicola* and *M. fijiensis* producing coalescing necrotic lesions on banana leaves (Carlier et al., 2000b). The causal fungus was originally considered to have a *Septoria* stage (Carlier et al., 2000b,c), but its anamorphic state has since been identified as *Pseudocercospora* (Crous and Mourichon, 2002). Although intensive surveys have not been conducted, eumusae leaf spot may be the dominant banana leaf spot disease in India, Sri Lanka, West Malaysia and Thailand. Its dominance could explain the confusing reports related to the distribution of black leaf streak, with which it may compete, in Southeast Asia. Other countries where the pathogen has been identified are Vietnam, Mauritius and Nigeria. It may have been introduced to Nigeria after the establishment of black leaf streak, which is still the dominant leaf spot. Symptoms caused by *M. eumusae* have been seen on cultivars, such as ‘Embul’ (syn. ‘Mysore’, AAB genome) in Sri Lanka, which has resistance to *M. musicola* and *M. fijiensis* (Carlier et al., 2000b). More work needs to be undertaken to determine the exact distribution of *M.*

eumusae, factors that limit its distribution and the reaction of important clones to infection, especially effects on yield and ripening.

Symptoms of freckle disease caused by *Guignardia musae* (anamorph *Phyllosticta musarum*) appear as dark brown to black spots on leaves and fruit. These spots can cluster and form streaks. Leaves that become severely affected yellow and die. Freckle, which is widespread in Asia and the Pacific on a variety of cultivars, is serious on export Cavendish cultivars grown in Taiwan and the Philippines. Freckle is controlled by the same fungicides used to control black leaf streak (Jones, 2000). Different forms of freckle seem likely to attack different hosts (Jones, 2000). In the South Pacific, it is cooking banana cultivars of the ABB genome, such as 'Bluggoe', that are predominantly attacked, whereas Cavendish clones of the AAA genome are unaffected. The situation in Taiwan and Hawaii is the reverse. In Southeast Asia, both ABB and AAA cultivars are affected. Freckle can be a problem on plantation-grown 'Pisang Berangan' (syn. 'Lakatan', AAA genome) in Malaysia. A preliminary analysis of genetic variability of isolates from Asia and Australasia has identified three distinct clades, which have distinct geographical distributions (S. Van Brunschot and J. Henderson, pers. commun.). More research needs to be conducted to confirm that more than one fungal species can produce symptoms of freckle.

Cladosporium speckle caused by *Metulocladosporiella musae* (Crous et al., 2005) is a widespread leaf disease that can affect various cultivars. Pencil-thin streaks, which coalesce and darken, are found on infected leaves. In West Africa, it has been reported in the past as significant on Cavendish types (Frossard, 1963). In East Africa, it can compound problems on East African Highland cultivars already affected by black leaf streak. The disease can be severe on smallholdings of 'Kluai Khai' (syn. 'Pisang Mas', AA genome,) in Thailand and plantations of 'Pisang Berangan' in Malaysia (Jones, 2000). Similar symptoms are caused by *M. musicola*, another species in the same genus, which has been identified only in East and South Africa (Crous et al., 2005).

Other leaf diseases caused by fungi that can be significant at certain times and in certain localities are black cross leaf spot, Cordana leaf spot, diamond leaf spot, Mycosphaerella leaf speckle and rust (Jones, 2000).

FUSARIUM WILT – A NEW DANGER TO CAVENDISH PLANTATIONS

The introduction of Cavendish cultivars into areas where plantation-grown 'Gros Michel' was succumbing to Fusarium wilt, caused by strains of race 1 of *F. oxysporum* f. sp. *cubense*, saved the banana export industry of the Americas. However, a new threat is emerging from strains of the pathogen that can attack Cavendish clones. These strains belong to *F. oxysporum* f. sp. *cubense* race 4. Although 'Dwarf Cavendish' in the Canary Islands had been affected by Fusarium wilt since 1924 (Stover, 1962), the danger first came to world attention when many Cavendish plants grown in other subtropical areas, such as South Africa, Taiwan and parts of Australia, began to succumb in great numbers. Work undertaken in Australia suggested that plant stress caused by winter conditions allowed specific strains of the fungus, now known colloquially as 'subtropical race 4', to invade (Ploetz and Pegg, 2000).

In the early 1990s, Cavendish cultivars planted on a large scale for the first time in Indonesia and Malaysia also became affected. Here, in the tropics, plants do not become stressed because of cool temperatures. The strains of pathogen involved could overcome the natural defences of Cavendish cultivars growing under optimum conditions. As the fungus spread in plantations, production decreased and cultivation became uneconomical.

These strains, which have been found to be the cause of the Fusarium wilt epidemic on Cavendish in subtropical Taiwan that began in 1967 (Su et al., 1986) and are currently threatening production in areas of mainland China, are known colloquially as 'tropical race 4'. Besides Cavendish, other hitherto resistant cultivars are also under threat from 'tropical race 4'.

There is a danger that 'tropical race 4' strains found now in only certain parts of Asia could spread further in Asia and also to the Americas, the Caribbean and Africa. This should not happen if strict quarantine is maintained to prevent entry. However, once introduced, the cause of the first outbreaks may not be identified early enough to prevent movement in soil attached to vehicles and footwear and in running water. Spread could be slowed by the exclusive use of tissue-cultured planting material in plantations, but not contained, especially if banana plants in surrounding gardens became infected and local soil became a reservoir of inoculum. The stage would be set for another major banana industry crisis.

BACTERIAL DISEASES - A RENEWED THREAT TO BANANA CULTIVATION

Three serious diseases caused by systemic vascular bacteria threaten banana cultivation. All result in the wilt and death of a wide range of banana cultivars. Dissemination has been linked to insect vectors that carry the bacteria to the inflorescence where they may infect the plant via fresh bract scars, though the male flower cushion has more recently been implicated as the prime site of infection (I. Buddenhagen, pers. commun., 2007). All can also be transmitted on pruning knives and through infected suckers or rhizome pieces. Fruit on infected plants usually ripens early with an accompanying pulp necrosis. Most infected plants wilt and die. These bacterial diseases, which are relatively localised at the moment, threaten to become major global constraints on production if spread continues.

Moko bacterial wilt is caused by *Ralstonia solanacearum* biovar 1 race 2. It is thought to have evolved on American species of *Heliconia*, which are close relatives to *Musa* spp. Evidence suggests that Moko bacterial wilt may have been present in Guyana in South America as early as the 1840s (Buddenhagen, 1961). Later, it was responsible for the abandonment of 'Moko' (syn. 'Bluggoe', ABB genome) as a shade crop in cocoa plantations in Trinidad in the 1890s (Rorer, 1911). In the Americas, Moko bacterial wilt is present in much of Central America and parts of South America, as well as Trinidad, Grenada, Jamaica and St Vincent in the Caribbean (Thwaites et al., 2000; Anon., 2004; 2007). There are various strains of the Moko bacterium that affect banana differently. Some are spread more by insects and infect mainly through the inflorescence, while others are transmitted predominantly on pruning tools. There are also common weed hosts in the Americas that harbour the pathogen. Although not completely systemic on 'Bluggoe', the bacterium invades all parts of other cultivars including Cavendish types. Only by costly strict hygiene measures and the immediate removal and destruction of infected plants and weeds is this disease kept under control in export plantations. Smallholdings of local banana cultivars prove more vulnerable, and significant losses occur (Thwaites et al., 2000).

Moko bacterial wilt also occurs in the Philippines, where initially it was primarily a fruit disease on the local cooking cultivars 'Saba' and 'Cardaba' (ABB/BBB genome) (Thwaites et al., 2000). The timing of the pathogen's arrival in the Philippines is still unknown, but bugtok was first described there in the mid-1960s (Roperos, 1965), and so the original introduction of the pathogen was probably not associated with the importation of material from the Americas in 1968 to create new export plantations as has been suggested (Buddenhagen, 1986). However, when export plantations were established in Mindanao, systemic bacterial wilt symptoms in Cavendish cultivars became apparent. Recently, it has been confirmed that the bacteria causing symptoms of bugtok and Moko bacterial wilt in the Philippines are the same pathogen (Fegan and Prior, 2006; Thwaites et al., 1999).

The Moko bacterium gained entry to Australia on imported *Heliconia* plants from Hawaii, a location where it had not been reported, but was quickly eradicated (Akiew and Hyde, 1992). This incident demonstrates the importance of taking strict quarantine precautions with *Heliconia,* which is a widely cultivated and traded ornamental.

Blood bacterial wilt of banana was first described on dessert banana plantations newly established by the Dutch on the Saleiran Islands off Sulawesi in Indonesia in 1906. Investigations in the early 1920s showed that the disease was widespread in southern Sulawesi on wild banana species and local banana cultivars. Quarantine restrictions were imposed to help prevent spread. The pathogen was confined to Sulawesi and surrounding islands until the late 1980s when it appeared on Java. Since then, it has spread to Sumatra, Kalimantan, the Moluccan Islands and Irian Jaya (Thwaites et al., 2000). The exact means of inter-island dissemination is not known, but it is obviously linked to the movement of planting material or fruit. The disease has not been reported outside Indonesia. The pathogen has been known as *Pseudomonas celebensis*, but is now believed to be a strain within the *R. solanacearum* species complex (Taghavi et al., 1996). It is related to other strains of *R. solanaceraum* in Indonesia in Phylotype IV, but not to the various strains causing Moko bacterial wilt, which are in Phylotype II (Fegan, 2005; Fegan and Prior, 2006). This indicates a completely different origin of the two pathogens. The blood bacterial wilt bacterium appears to have evolved independently in Indonesia.

Xanthomonas wilt was first described on enset (*Ensete ventricosum*) in Ethiopia in the late 1960s (Yirgou and Bradbury, 1968), although it is likely to have been present in the late 1930s (Castellani, 1939). Banana was later recorded as another natural host in Ethiopia (Yirgou and Bradbury, 1974). Young et al. (1978) proposed the name *Xanthomonas campestris* pv. *musacaerum* for the pathogen, but recent taxonomic studies have shown it to be a pathovar of *X. vasicola* (Aritua et al., 2008). It may have evolved in Ethiopia from *X. vasicola* strains affecting other crops. The name *X. vasicola* pv. *musacearum* would seem appropriate, but this nomenclature has yet to be officially proposed. In inoculation studies, maize (Aritua et al., 2008) and the ornamental *Canna orchoides* (Ashagari, 1985) have been reported to be susceptible.

The first report of Xanthomonas wilt outside Ethiopia came from banana in central Uganda in 2001 (Tushemereirwe et al., 2004). Since then, it has been found in the Democratic Republic of Congo (Ndungo et al., 2006), Rwanda (Aritua et al., 2008), Tanzania (Mgenzi et al., 2006) and Kenya (Aritua et al., 2008). The disease, which is already seriously affecting production in parts of Uganda, is now posed to do the same over a much larger area in East and Central Africa where banana is an important component of the diet.

All banana cultivars in infected areas seem susceptible to infection through pruning knives, but cultivar phenology and cultural practices affect the amount of disease in the field. The most seriously affected cultivar is 'Kayinja' (syn. 'Pisang Awak', ABB genome), and this is thought to be because of its dehiscent bracts and low-intensity management practices. The least affected are those with persistent bracts, such as 'Dwarf Cavendish', which deny insect vectors access to exposed banana tissue. There are early indications that the disease can be controlled if certain management guidelines, such as the early removal of the male flower bud, are followed. However, large-scale extension programmes in East and Central Africa are required to disseminate this information. The prognosis is not good for banana production if Xanthomonas wilt keeps spreading and cultural control practices are not adopted.

Bacterial rhizome rot caused by an *Erwinia* spp., most likely *E. chrysanthemi*, is a fairly widespread disease that can cause problems in young plants.

VIRUS DISEASES – AN EVER-PRESENT DANGER

Bunchy top disease, caused by *Banana bunchy top virus* (BBTV) in the *Babuvirus* genus, was first recorded in Fiji in 1889, though it was likely present a decade earlier (Thomas and Iskra-Caruana, 2000). The virus almost certainly did not evolve in Fiji and may have been introduced from the Indian subcontinent by sugarcane labourers. BBTV is now found in a number of Pacific and Asian counties and also in parts of northern, central and eastern Africa. Affected plants have a rosetted appearance and produce no fruit if infected early in the growth cycle. BBTV is spread from plant to plant by the black banana aphid (*Pentalonia nigronervosa*). As BBTV is systemic, suckers and rhizome pieces from infected plants disseminated as planting material can spread the disease. BBTV can also be transmitted in micro-propagated plants (Thomas and Iskra-Caruana, 2000). BBTV has never been eradicated from countries where it occurs, although it has been eradicated from certain areas in Australia through the strict enforcement of control measures, such as restrictions on plant movements, regular plant inspections, the prompt destruction of infected plants and the establishment of quarantine zones.

Typical symptoms associated with *Banana bract mosaic virus* (BBrMV) in the *Potyvirus* genus have been reported in India, Sri Lanka and the Philippines. Confused with mosaic disease of banana for many years, it was first identified as a new disease in the Philippines, where it is common on many cultivars, in 1988 (Frison and Putter, 1989). Symptoms included a distinctive mosaic on bracts and mosaics, stripes and spindle-shaped streaks on pseudostem bases. Fruit from infected plants can also be misshapen. Yield losses of 40% were noted in some cultivars (Thomas and Magnaye, 1996). In India, kokkan disease of 'Nendran' (AAB genome, plantain subgroup) was found to be caused by BBrMV. Symptoms on 'Nendran' included suppressed and distorted suckers, increased pseudostem pigmentation and a failure of fingers to fill. Other cultivars in India and Sri Lanka are also affected (Thomas et al., 2000). BBrMV is spread locally by certain aphids, such as *Aphis gossypii* and *Pentalonia nigronervosa*. Long-distance spread occurs as a result of the movement of infected planting material or tissue cultures. No natural resistance has been identified (Thomas and Magnaye, 2000).

Mosaic disease of banana caused by *Cucumber mosaic virus* (CMV) in the *Cucumovirus* genus is a widespread, but relatively minor problem in banana. However, it can be serious when young banana plants derived from tissue culture are planted near weeds, such as *Commelina*, *Stellaria*, *Bryonia* and *Solanum* spp., which can be reservoirs of the virus. In this situation, problems arise when aphid vectors, such as *A. gossypii* and *Myzus persicae*, carrying CMV leave their weed hosts to feed on the succulent tissues of the young banana plant. Symptoms induced by severe strains of CMV include a prominent mosaic on leaves, leaf deformation, a necrosis of unfurling leaves and the internal tissues of the pseudostem, and a mosaic on the peel of fruit. Mild-strain symptoms are less noticeable, especially in the summer months (Lockhart and Jones, 2000a). Long-distance spread is by means of movement of infected planting material including tissue cultures, though weeds are a much more important source of inoculum. The incidence of mosaic can be reduced by eliminating weed hosts of CMV from around plantations and by the prompt removal of any infected plants (Lockhart and Jones, 2000a).

Streak disease caused by banana streak viruses (BSV) in the *Badnavirus* genus is also widespread (Lockhart and Jones, 2000b). Highly variable, three distinct BSV virus species, namely *Banana streak GF virus*, *Banana streak Mysore virus* and *Banana streak OL virus*, have been officially recognised (Fauquet et al., 2005), but many other species will undoubtedly be confirmed in the future. BSV has been mistaken for mosaic disease in the past (Yot-Dauthy and Bové, 1966). Symptoms can vary from an inconspicuous chlorotic flecking to lethal necrosis, but the commonest symptoms are narrow, discontinuous and sometimes continuous chlorotic or yellow streaks, which run from the leaf midrib to the margins. Streaks later darken to orange and black. Yellow leaf blotches can also be a symptom. Leaves showing no symptoms may succeed leaves showing symptoms. If all leaves with symptoms are shed, the plant may appear normal. The periodicity of symptoms has been associated with temperature fluctuations. Aberrant bunch emergence, reduced bunch size and distortion of fingers may also occur. Peel splitting and necrotic streak and spot symptoms have been observed on fingers of infected 'Grand Nain' (AAA genome, Cavendish subgroup). Pseudostem sheath splitting in Cavendish cultivars can lead to death following bacterial wound infections (Lockhart and Jones, 2000b).

BSV is spread locally by some mealybugs (*Planococcus* spp.), which can be farmed and moved from plant to plant by ants. Long-distance spread is by the movement of infected planting material. Stresses associated with shoot-tip culturing clones containing a B genome component derived from *M. balbisiana* may also result in infected plants. Under stress conditions, genomic sequences of BSV integrated into the DNA of the B genome can give rise to episomal infections. This has become a major problem when tissue cultures, especially those of bred hybrids with a B genome component, are moved internationally (Lockhart and Jones, 2000b). However, research has resulted in treatments being devised to eliminate episomal BSV and other viruses from banana tissue (Panis et al., 2005).

FRUIT DISEASES – PROBLEMS FOR GROWERS, TRADERS AND CONSUMERS

Banana fruit suffers from both pre- and post-harvest problems, which can cause spoiling and waste, especially where markets are discerning. Cigar-end and tip-end rots affect fruit before harvest. The former disease, which has an ashy gray appearance, is caused by *Verticillium theobromae* and *Trachysphaera fructigena* while the latter is the result of infection by *Deightoniella torulosa*, *Fusarium moniliforme, V. theobromae* or other fungi. All cause a rot beginning at the flower end of the fruit, which usually progresses no more than a third of the way down the finger (Jones and Stover, 2000).

Diamond spot is a pre-harvest disease caused by *F. solani*, *F. pallidoroseum* and possibly other fungi invading fruit peel lesions initiated by *Cercospora hayi*. It was very common in certain places in Central America in the 1960s, but is no longer important (Jones and Stover, 2000).

Freckle is an important pre-harvest disease of banana fruit caused by the fungus *G. musae*. The fungus makes banana peel unsightly which is a problem if fruit is produced for markets where external appearance is important. The disease, which also affects leaves, must be controlled in the field, as infection occurs prior to harvest (Jones and Stover, 2000).

Bugtok, caused by the strain of *R. solanacearum* that also causes Moko bacterial wilt, is important as a pre-harvest disease only in the Philippines. Infection occurs through the male bud. Moving up the peduncle in the vascular tissues, the bacterium invades the bunch. This results in a discoloration and hardening of the pulp of some or all of the fruit. Affected fruit is inedible. The local cooking banana cultivars 'Saba' and 'Cardaba' show typical symptoms because they seem to be resistant enough to prevent the bacterium becoming completely systemic, but not enough to prevent it moving into the fruit. Bagging the bunch and peduncle completely to prevent the entry of insect vectors controls bugtok (Thwaites et al., 2000).

Another pre-harvest problem is bacterial finger-tip rot or 'gumming'. The pulp of the fruit first appears gelatinous and later can become rusty red. However, only a few fingers of the youngest hand usually become infected. The bacterium is thought to gain entry through the flower end of the finger (Jones, 2000). In Taiwan, *Burkholderia cenocepacia* has been implicated as the causal agent (Lee and Chan, 2006).

Anthracnose caused by the fungus *Colletotrichum musae* can affect fruit before harvest though it is primarily a post-harvest disease. The fungus is an inhabitant of banana plantations being commonly found in senescing banana tissue. Spores of the fungus reach the fruit on the plant in wind, rain splash or possible on insects visiting flowers. Spores in re-circulated washing water attach to fruit in the packing shed. On germinating, fungal appressoria form on the green peel and remain dormant until ripening or wounding. Lesions then increase in size and coalesce. Anthracnose is controlled by removing dead and dying banana material from plantations to reduce inoculum levels, handling fruit with care and/or a post-harvest fungicide treatment (Jones and Stover, 2000; Muirhead and Jones, 2000).

The post-harvest disease crown rot is caused by *C. musae* and/or various *Fusarium* spp., such as *F. pallidoroseum*. Many other fungi have also been implicated in the disease (Muirhead and Jones, 2000). The crown is the pad of tissue from which the fruit pedicels arise. It remains on the hand after it is cut from the peduncle and the exposed tissue is vulnerable to infection. Invading fungi can partially or completely rot the crown and also spread down the pedicels causing the fingers to fall off on handling. Sometimes, the fingers themselves can be invaded. Crown rot is controlled by ensuring that cuts to remove hands and to divide them into clusters are straight edged and by treating crowns with fungicide. Banana debris, which is an inoculum source, should be cleared from plantations (Muirhead and Jones, 2000).

NEMATODES – A SERIOUS AND INSIDIOUS PROBLEM

Radopholus similis, the burrowing nematode, was first described in Fiji in 1893 and is one of the most important root parasites of banana. It is especially prevalent on Cavendish cultivars grown in commercial plantations in tropical lowlands. It is also common on plantain and other cooking banana types in the lowlands of Central and East Africa, but not western and southern Africa. It is not found at the highest altitudes of banana cultivation nor at the highest latitudes, such as the Canary Islands and Taiwan. Its distribution seems to reflect historical movements of infested Cavendish planting material (Sarah, 2000). The burrowing nematode destroys root and rhizome tissue, reducing water and mineral uptake that can lead to a significant reduction in yield and increase the time period between bunches. In addition, plants have a tendency to uproot or topple.

Symptoms appear as a reddish-brown discoloration on the surface of washed roots or rhizomes. Banana clones vary in their susceptibility, with plantains being exceptionally susceptible. Good resistance has been found in 'Silk' and 'Mysore' (Sarah, 2000). Control has traditionally been achieved by paring rhizomes to remove infested tissue followed by a hot-water treatment or a dip in a nematocide before planting. Allowing land to lie fallow or to be sown with non-host plants reduces populations. The use of nematode-free plants, such as those derived from tissue culture, as planting material in virgin soil avoids a problem for many years. Nematocide application to soil is the most common control method in commercial plantations, but nematocides endanger human health and some are being withdrawn. Biological control measures are being developed as an alternative (Sarah, 2000).

Pratylenchus coffeae and *P. goodeyi*, the root-lesion nematodes, are also found on banana in the tropics. The former is important in the Pacific and Southeast Asia. It is significant on 'Kluai Namwa' (syn. 'Pisang Awak', ABB genome) in Thailand. The latter occurs in the highlands of East, Central and West Africa and subtropical areas and seems more adapted to cooler climates. Both are often found in association with plantain. Although Cavendish cultivars are susceptible, root-lesion nematodes are not so common in plantations where *R. similis* is dominant. Symptoms are identical to those caused by the burrowing nematode. Damage leads to a reduction in the size of the root system and plant toppling. Control is currently achieved by using methods used to control *R. similis* (Gowen, 2000).

Other nematodes that can cause problems include the spiral nematode (*Helicotylenchus multicintus*) and root-knot nematodes (*Meloidogyne incognita, M. javanica*) (Jones, 2000).

INSECT PESTS – POTENTIAL FOR SERIOUS DAMAGE

The most important insect pest of banana is the banana weevil (*Cosmopolites sordidus*), which has a widespread distribution. The insect burrows into the rhizome, and its galleries weaken the plant, which can topple (Stover and Simmonds, 1987). In East Africa, the banana weevil is believed to contribute significantly to the general decline in productivity of the East African highland banana cultivars. It has traditionally been controlled by planting weevil-free rhizomes and with chemicals, but there are now human health considerations and resistance has developed. In recent years, efforts have concentrated on attempts to trap weevils by the use of pheromones and in developing other biological control methods.

Stalk borers, such as *Castniomera humboldti* in Central and South America and *Odoiporus longicollis* in Asia, occasionally tunnel into the banana pseudostem and can cause yield losses. There is no control, but ants are known to be predatory on *C. humboldti*.

Fruit and flower pests are important, especially for export markets where consumers demand blemish-free fruit. Red rust thrips (*Chaetanaphothrips orchidii* and *C. sigipennis*) cause a reddish stain between the fingers. Infestations of *Thrips florum* result in corky scabbing of the peel in India, Australia and the Philippines, especially during hot and dry weather. Peel scarring beetles (*Colapsis* spp., especially *C. ostmarkii*) can also be a problem in Central and South America. The banana scab moth (*Nacoleia octasema*) is a serious pest in the Pacific where the larvae feed on young fingers, which produces severe scarring and cracking on older fruit (Stover and Simmonds, 1987). Control has been obtained by insecticide-impregnated or dusted bags, insecticide strips hanging in bags, insecticide bunch and flower injection methods, male bud removal and the use of predatory insects.

Foliage feeders can be destructive at times. Caterpillars, such as species of *Antichoris*, *Caligo*, *Osiphanes* and *Sibene* can partially defoliate banana plants in Central and South America. Bagworm (*Oiketicus kirbyi*) can be a problem in Central America, but natural parasites usually control outbreaks (Stover and Simmonds, 1987). The banana skipper (*Erionota thrax*) can be serious in Asia and Papua New Guinea, but a parasitic wasp (*Cotesia erionotae*) is an effective biocontrol agent (Anon., 2005). Insecticides are usually used if control of other leaf feeders is needed.

Sucking insects, such as aphids, mealybugs, scales and lace bugs, are occasionally important. However, it is the role of some as virus vectors that is far more significant. *Pentalonia nigronervosa*, the aphid that transmits BBTV, is probably the most infamous. The excretions of sucking insects can also result in the growth of sooty moulds on fruit, which is undesirable. Spider mites, which suck, but are not insects, also occasionally cause damage.

APPROACHES FOR CONTROLLING BANANA PATHOGENS AND PESTS

There are three main problems facing banana cultivation today. The first is the vulnerability of long-established, popular cultivars, such as those in the Cavendish, Pome (AAB), Silk, plantain and East African highland subgroups of banana, to attack by pathogens/pests. The second is the spread of important pests/pathogens from their centre of origin to new banana-growing regions. This is exemplified by the recent spread of Xanthomonas wilt across East Africa and Cavendish-attacking Fusarium wilt across China. The third problem is the increasing resistance of pathogens/pests to chemicals used in control. An example is the struggle to maintain control of black leaf streak in parts of the Americas. Banana has never been under as much pressure as today. What approaches are being used to counter the threats to banana cultivation?

History has shown that quarantine precautions can delay the arrival of serious pests and diseases, but may not prevent them from arriving in the long term. However, even a delay gives time for more research into a possible solution to the problem. Banana germplasm should only be moved between countries and banana-growing regions within larger countries as virus-tested tissue cultures. Despite a strong message on safe movement being disseminated over a number of years, the long-distance, unsafe movement of vegetative planting material still occurs. The movement of banana material other than as in-vitro cultures should be discouraged. This is especially important to prevent the entry of exotic pests and pathogens into new continents. Once introduced to a new continent, it is almost impossible to stop spread across the international boundaries of countries where banana cultivation is contiguous, such as in East Africa and many parts of Asia and Central America.

The conventional breeding of banana for improved resistance to pathogens has been attempted since the 1920s (Jones, 2000). Tetraploid hybrids have been produced by the *Fundación Hondureña de Investigación Agrícola* (FHIA) that have desirable qualities and resistances (Rowe and Rosales, 2000). West African-based breeding programmes at the International Institute of Tropical Agriculture (IITA) and the *Centre Africain de Recherches sur Bananiers et Plantains* (CARBAP, previously CRBP) have concentrated on plantains with the development and dissemination of a number of tetraploid hybrids with potential (Jones, 2000). A programme run by the *Empresa Brasiliera de Pesquisa Agropecuária* (EMBRAPA) has specialised in the improvement of cultivars in the Silk and Pome subgroups important to Brazil (Jones, 2000). The *Centre de Coopération Internationale en Recherche Agronomique pour le Développement* (CIRAD) in Guadeloupe has been concentrating on improving export types of banana. This has been attempted by developing auto-tetraploids by doubling the chromosome number of homogenous synthetic diploids and producing hybrid triploids in crosses with other bred diploids (Jones, 2000). In addition, there are also a number of banana breeding programmes in India coordinated by the National Research Centre for Banana (NRCB). These aim to improve important local cultivars. A new breeding programme aimed at improving East African highland cultivars has recently been initiated in Uganda by IITA.

Unfortunately, conventional breeding programmes have not been very successful considering the time and effort that has been expended over the years. The new bananas have usually fallen down on taste, cooking quality or the ability to travel well if developed for export markets. In general, despite the susceptibilities of the existing cultivars to pest and pathogens, bred hybrids have still not been economically viable as replacements for export and local banana industries. Other than the success of the FHIA hybrids in Cuba, small niche markets for bred hybrid fruit in certain areas, such as with 'FHIA-01' in subtropical parts of Australia, is the best that has been achieved. Nevertheless, some valuable diploids that are useful as parents in crosses have been produced, and new approaches, such as triploid breeding, are being tried. Much is possible, but the development of a new clone that has the potential to be planted widely because of improved resistances and desirable agronomic characteristics is elusive. However, conventional breeding as a means of incorporating genes for resistance into germplasm to create a useful banana should be supported and needs to continue. A major breakthrough will hopefully occur in the future.

The multinational banana export companies would like to grow a black leaf streak-resistant banana with the same productivity and taste as Cavendish cultivars. However, Cavendish cultivars are almost completely sterile and unsuitable for breeding conventionally. This has led to moves to introduce DNA inserts that may act as fungal resistant genes into the Cavendish genome using genetic engineering methods. This approach has been made possible by the development of techniques that allow banana plants to be regenerated from single protoplasts, cell suspensions and somatic embryos (Jones, 2000). Nevertheless, the science is complex, and a transformed Cavendish with useful black leaf streak-resistance traits has not yet been developed and commercially adopted. Genetic engineering techniques have also been developed to transform locally important banana clones, such as plantain and East African highland types, grown by smallholders in developing countries.

A banana resistant to black leaf streak is not the only target of genetic engineering. Field resistance to other fungal diseases, such as Fusarium wilt, and virus, bacterial, nematode and weevil problems is also being sought (Atkinson et al., 2003). A programme to develop transformed banana cultivars with resistance to Xanthomonas wilt is currently in progress in Uganda (Tripathi et al., 2004). Transformed material is available for testing, but there have been problems in obtaining permission from governments to undertake outdoor trials with genetically modified organisms (GMOs) because of biosecurity considerations. Another underlying problem is the reluctance of consumers to buy food derived from transformed crops.

Other technological innovations used in attempts to improve *Musa* germplasm have been the use of radiation as a mutagen and also the exploitation of somaclonal variation in tissue-cultured material in the hope of producing banana mutants that have desirable qualities. The former technique has not resulted in any material that has been adopted commercially, but the latter has resulted in various somaclonal variants of Cavendish being grown in Taiwan because of their increased field resistance to Fusarium wilt (Hwang and Tang, 2000).

Musa germplasm has been and is presently being screened for reaction to various pathogens and pests in attempts to identify sources of resistance in various clones. Progress is also being made on sequencing the *Musa* genome. The ultimate aim is to identify genes for pest and disease resistance, drought tolerance and other important attributes, and to document their allelic diversity. This will aid future banana breeding, whether conventional or technological (Anon., 2006a,b). Coupled with the banana work is another sequencing project to reveal the genome of *M. fijiensis*. The results will help understand the processes involved in pathogenicity and resistance. The goal is to reduce fungicide use (Anon., 2006c).

As chemicals fail, research into the control of banana pests and pathogens is more and more aimed at developing biological and cultural techniques to reduce their impact. This trend towards more sustainable means of control is being encouraged by funding bodies keen to acquire 'green' credentials. The stimulation of healthy roots is an important mechanism for optimum growth and to prevent a decline in fertility (Turner and Rosales, 2005). The search for predators and competitors of problem organisms is increasing. Combining biological, cultural and chemical methods to optimise control is seen as effective and desirable. Integrated pest management is viewed as the way forward (Blomme et al., 2005; Frison et al., 1998). The growing of banana in unfavourable climates for disease is being investigated in the Americas and the Caribbean. Natural products for use in organic farming systems are also being investigated (Holderness et al., 2000; Rosales et al., 1998). Banana plants grown from tissue cultures for one cycle or a limited number of ratoon cycles would also reduce the chances of the build-up of many pests and diseases. The cultivation of banana in plantings with other crops, such as citrus, coffee and cacao, as occurs now in parts of the Americas, may be another option for the future.

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