CHAPTER 1

BANANA FOLIAGE DISEASES – AN OVERVIEW

BANANA FOLIAGE DISEASES – AN OVERVIEW

INTRODUCTION

The banana (*Musa*) plant is the world's largest herbaceous monocotyledon. It originates from Southeast Asia and is grown in more than 120 countries globally (Jones 2000). Bananas are the world's fourth most valuable food product, and serve as the primary source of food for over 400 million people in the tropics (Picq & Ammar-Khodja 2000). World trade totals US$ 5.3 billion annually (Sasson 1997), which represents only 10 % of the 90 billion tonnes of bananas produced worldwide each year (Ploetz 1999, Picq & Ammar-Khodja 2000). The remaining 90 % are sold on local markets by subsistence farmers.

Many fungi, bacteria, viruses and nematodes are known to attack banana plants and cause disease to the roots, corm, pseudostem, leaves and fruit. Banana plants can also be subjected to a number of disorders, mineral deficiencies, physical damage, chemical damage and genetic abnormalities (Jones 2000). Fungal diseases that affect banana foliage decrease the photosynthetic area of infected leaves, thereby reducing fruit yield and quality (Jones 2000). Chemical control of leaf diseases is expensive and not financially feasible for producers in developing countries. Some leaf pathogens also develop resistance/tolerance to particular fungicides, making them even more difficult to control (Jones 2000). International breeding programs have developed hybrids that are genetically resistant to specific leaf pathogens (Jones 2000), but these hybrids do not always have acceptable fruit and agronomic qualities.

The principal fungal leaf diseases on banana include Sigatoka-like leaf spots, speckle diseases and freckle. Several fungi are also considered to be minor pathogens on banana leaves. This overview addresses the distribution, symptoms, causal agents, disease cycle, epidemiology and control measures of banana foliage diseases. Also included is a brief summary of the endo- and epiphytes recorded to date on banana foliage.

THE BANANA PLANT

The banana plant migrated, with travellers, from Southeast Asia to the Indian peninsula, eastern Africa and the islands of the Pacific. There are approximately 1000 different types of banana varieties known in the world (Picq & Ammar-Khodja 2000). These can be subdivided into 50 groups, of which some can be eaten raw as a sweet dessert banana, or fried, steamed and cooked as a vegetable. Consumer demand for bananas began to increase during the nineteenth century, and today bananas are exported from countries in Central and South America, and South-East Asia, to markets in North America, Europe, Japan and countries of Eastern Europe and the former USSR (Picq & Ammar-Khodja 2000).

The banana plant is a perennial that rejuvenates itself approximately once a year (Picq & Ammar-Khodja 2000). It has a branched underground stem known as a rhizome or corm that gives rise to both roots and vegetative buds (Fig. 1) (Jones 2000, Picq & Ammar-Khodja 2000). The root system is relatively shallow and usually only penetrates about 60 cm vertically into the soil, but it has a horizontal spread of up to 5 m (Jones 2000). After the fruit set the mother plant is

cut down, and the new vegetative buds, known as "suckers", develop and form the pseudostem (Jones 2000). The pseudostem consists of tightly packed leaf bases with an apical meristem in the centre at soil level, giving rise to successive leaf primordia (Jones 2000). Each of these primordia differentiate into a leaf base, a petiole and a lamina. After about 25–50 leaves have been produced, the vegetative core stops producing leaves and produces, instead, an inflorescence. The peduncle of the inflorescence forces its way out of the top of the plant, lifting the bracts and allowing for the double layers of female nodes with compact fruit to emerge. Each individual banana fruit is referred to as a "finger" of the "hand", and the collective term for the "hands" is a "bunch" (Jones 2000). There are 10–15 fully functional leaves at flowering, which decrease to 5–10 leaves at harvest. The most efficient leaves for photosynthesis, counting down the plant, are leaves 2–5, as lower leaves tend to be too old (Robinson & Peterson 1999). After harvest the pseudostem dies back and is cut to ground level.

BANANA LEAF DISEASES

SIGATOKA DISEASES

Three Sigatoka diseases cause damage to banana leaves, namely yellow Sigatoka, black Sigatoka and eumusae/septoria leaf spot. Yellow Sigatoka was first recorded in Java in 1902 and later in the Sigatoka valley of Fiji (Meredith 1970). The first notable losses to the disease occurred in Fiji in 1912 and in 1924 in Australia (Wardlaw 1961, Stover 1972). Since 1930, yellow Sigatoka has disseminated rapidly due to uncontrolled movement of infected propagative material to the continents. By 1933 the Caribbean was experiencing serious crop yield losses,

and by 1937 yellow Sigatoka was taking its toll in all the banana growing areas of Columbia, the Guineas, Central America, Jamaica, Suriname, Demerara, British Honduras, French Antilles, Peru, Brazil, Mexico and the Caribbean islands (Wardlaw 1961, Stover 1972). In East Africa, by 1938, and West Africa, by 1941, the disease began to have an affect on the African banana industry (Wardlaw 1961, Stover 1972). In 1950, Ecuador became the last mass production banana area to be affected (Stover 1972). The disease now occurs in all banana-growing countries except Egypt, Israel and the Canary Islands (Carlier *et al.* 1994, Jones 2000).

Yellow Sigatoka can cause severe losses in banana production. Infection damages the leaves, thereby reducing the area of functional leaf surface and photosynthetic capacity within the plant's cells (Jones 2000). After shooting, when no new leaves are being produced, those damaged by yellow Sigatoka cannot be replaced, and the yield is affected (Jones 2000). If the disease reaches its peak just before harvest, the fruit ripen prematurely and unevenly, and some appear undersized and angular. The flesh has a buff pink colour and fruit storage time is greatly reduced (Agrios 1997). The greater and earlier the leaf damage the more pronounced the effect on yield (Jones 2000). Banana production can be reduced by as much as 50%, or even more in badly affected areas (Wardlaw 1961). The disease appears to have little or no effect on vegetative growth of banana plants in the tropics. Leaf emergence, rate of plant height increase and height of plants at shooting are not reduced as a result of infection. The reason for this is that the disease appears not to effect leaves 2–5, the main photosynthetic producers, as severely as the other leaves (Jones 2000).

Black Sigatoka was first noticed in the Sigatoka area of the Viti Levu Island in Fiji in February 1963 (Carlier *et al.* 2000a). Surveys conducted between 1964 and 1967 revealed that

black Sigatoka was spreading rapidly in the Pacific. It was reported to be present in Micronesia, New Caledonia, Papua New Guinea, Philippines, Western Samoa, Singapore, Solomon Islands, Tahiti, Taiwan, Tonga, Vanuatu, West Malaysia, Hawaiian Islands, Cook Islands and Niue. It is thought that black Sigatoka may have arrived in the Hawaiian Islands as early as 1958 and that it had been mistaken for yellow Sigatoka. Old herbarium specimens indicate that black Sigatoka was present in Taiwan from 1927 and in Papua New Guinea from 1957. In 1970, black Sigatoka was found in the Philippines on many different cultivars, but is considered to have already arrived on the island of Luzon in about 1964. Jones (1994) reported that black Sigatoka was first discovered in North Africa in 1973 and has since spread throughout East and West Africa. In 1980, both black and yellow Sigatoka were observed on the Hainan Island of China, and since then it has spread throughout southern China. In 1985 and 1993 black Sigatoka was recorded in Bhutan and Vietnam, respectively, however it has not yet been recorded in Burma, India or Bangladesh (Carlier *et al.* 2000a). In many tropical areas black Sigatoka has replaced yellow Sigatoka as the dominant leaf spot within a year. Yellow Sigatoka, however, still dominates at altitudes above 1200–1400 m in tropical regions (Mourichon *et al.* 1997).

Black Sigatoka is far more aggressive than yellow Sigatoka and affects a wider range of banana genotypes (Carlier *et al.* 2000a). It develops much faster and causes more severe damage to banana leaves. In many actively growing plants, the second leaf is often affected by the disease. Once bunching has taken place and no more leaves are produced, all remaining leaves are killed by the fungus, thereby forcing the plant to abort the bunch. Rapid destruction of the leaves of the banana plant leads to reduction in yield and premature ripening of the fruit.

During a survey for yellow and black Sigatoka on bananas in southern India, Sri Lanka, West Malaysia, Thailand and Vietnam between 1992 and 1995, an unknown pathogen causing symptoms similar to those of yellow and black Sigatoka was isolated (Carlier *et al.* 2000b, Carlier *et al.* 2000c). Since this pathogen produced a *Septoria*-like anamorph and *Mycosphaerella* teleomorph, the disease was tentatively named Septoria leaf spot (Carlier *et al.* 2000b, Carlier *et al.* 2000c). Upon further investigation it was renamed eumusae leaf spot (Crous & Mourichon 2002). Eumusae leaf spot now appears to be the dominant leaf spot disease of bananas in Thailand (Carlier *et al.* 2000c). This disease has also been found on banana leaves in Onne, Nigeria and in Mauritius (Carlier *et al.* 2000c)

Symptoms

Yellow Sigatoka exhibits five stages of symptom development on banana leaves (Table 1, Fig. 2). Initially it manifests as small (1 mm long) pale yellow flecks on the third or fourth leaf from the top of the plant (stage 1) (Fig. 2A). When conditions are favourable for infection, even the second leaf can show early symptoms (Simmonds 1959, Wardlaw 1961, Jones 1994, Jones 2000). These spots are indistinct and longitudinal, running parallel to the side veins of newly unfolded leaves (Wardlaw 1961, Agrios 1997, Mourichon *et al.* 1997). The flecks then begin to elongate into yellow green streaks, 1 x 3–4 mm (stage 2) (Fig. 2B) (Wardlaw 1961, Jones 1994, Jones 2000). Streaks enlarge to 1–2 cm in length, and their centres become dark brown or rusty red in colour (stage 3) (Fig. 2C) (Wardlaw 1961, Jones 1994, Agrios 1997). They develop definite margins with yellow, sometimes water-soaked, halos (stage 4) (Fig. 2D) (Jones 1994). Finally, the centres of lesions appear to sink and turn a light grey colour (stage 5) (Fig. 2E) (Meredith 1970, Agrios 1997, Robinson & Peterson 1999). Lesion margins, however, remain

dark brown to black, often retaining their yellow halo (Wardlaw 1961, Jones 1994, Robinson & Peterson 1999). Halos may turn dark brown as well, forming a clear ring around the mature spot (Robinson & Peterson 1999, Jones 2000). Individual spots on mature leaves are ellipsoidal and 2–5 x 12–15 mm in size (Fig. 2E) (Jones 1994). On young sucker shield leaves, the spots are usually oval or round (Jones 1994). When these spots begin to enlarge the damage to the banana plant becomes more severe. Tissue around the enlarging spots yellows and dies, and adjacent spots begin to coalesce, leading to larger areas of dead tissue on the leaf (Fig. 2F) (Simmonds 1959, Wardlaw 1961, Agrios 1997, Jones 2000). With high infection intensity the necrotic leaf areas turn white-grey and the brown borders of individual lesions become indistinct (Fig. 2F) (Robinson & Peterson 1999, Jones 2000). Mature spot symptoms are found on older leaves and infection severity appears to increase on older leaves (Jones 2000). In very severe cases, the affected leaves can die within a few weeks, leaving less than the minimum of nine leaves required for maturation of the fruit (Robinson 1996). On dead leaves distinct brown-black lesion margins are visible on the dead brown background (Jones 1994, Jones 2000).

Initial symptoms of black Sigatoka appear as chlorotic or red-brown streaks less than 0.25 mm in diameter on the lower surface of the leaf (Jones 1994). These initial streaks elongate to 20 x 2 mm and are parallel to the leaf veins, often so close that they overlap (Carlier *et al.* 2000a). During the second stage of symptom development, streaks turn a very dark brown-black in colour with a water-soaked halo (Fig. 3A) (Carlier *et al.* 2000a). Lesions become fusiform or elliptical with depressed centres, and they are surrounded by a yellow halo maintaining its water-soaked appearance (Jones 1994). The third stage results in the centre of the lesion turning a pale grey colour with a black margin surrounded by a yellow halo (Fig. 3B) (Carlier *et al.* 2000a). Where spots are coalescing entire sections of the leaf become necrotic and die, but lesions are still

visible due to their pale centres (Fig. 3C and D) (Carlier *et al.* 2000a). On healthy plants that are still growing, streaks are seen on the third, fourth and fifth leaves, whereas on stressed plants symptoms are often seen on the first and second leaves (Carlier *et al.* 2000a). On resistant cultivars, symptoms are usually only seen on the lower leaves, dependent on the resistance levels of the cultivar (Carlier *et al.* 2000a). Susceptible cultivars, however, may loose all their leaves and bunches will drop to the ground before maturing (Jones 1994).

Eumusae leaf spot lesions have an appearance very similar to that of yellow and black Sigatoka (Carlier *et al.* 2000c). It seems that in the past this disease has been mistaken for either of the Sigatoka diseases, as its causal organism has only recently been identified (Carlier *et al.* 2000c). Brown streaks expand into large spots that darken, the centres turn grey and develop a distinct dark brown border, and are ovoid or elliptical in shape when mature (Fig. 4A) (Carlier *et al.* 2000b, Carlier *et al.* 2000c, Crous & Mourichon 2002). Leaf spots are larger and more rounded (Fig. 4B) than the streaks seen in black and yellow Sigatoka infections, they closely resemble the lesions caused by Phaeoseptoria leaf spot (Carlier *et al.* 2000c). On growing plants, streaks are present on the third, fourth and fifth leaves and streaks as well as spots are present on the fifth and older leaves (Crous & Mourichon 2002). At high infection densities lesions coalesce and cause large areas of leaf tissue to become necrotic. Grey spots are visible in the necrotic area and a yellow halo precedes the expanding necrosis (Fig. 4C) (Carlier *et al.* 2000c). Where coalescence has occurred, streaks on the upper surface change from brown to black while those on the lower surface remain brown (Crous & Mourichon 2002).

Causal agents

Yellow Sigatoka is caused by *Mycosphaerella musicola* Leach ex J.L. Mulder & R.H. Stover (anamorph *Pseudocercospora musae* (Zimm.) Deighton), a heterothallic ascomycete in the order *Dothideales*, family *Mycosphaerellaceae*. It reproduces by means of ascospores in pseudothecia (Fig. 5A) and conidia in sporodochia (Fig. 5B) (Agrios 1997). Pseudothecia form after fertilisation of receptive hyphae by compatible spermatia (Agrios 1997), and contain 10–27 asci each (Stover 1972). They are more abundant in mature spots on the upper than on the lower leaf surface (Jones 2000). Sporodochia appear during the brown spot stage (stage 4) and last throughout the mature spot stage (stage 5) of yellow Sigatoka symptom development (Agrios 1997). Sporodochia are produced on both sides of the leaf, but are more abundant on the upper surface where wind and/or rain dispersal occurs (Agrios 1997). They develop in the sub-stomatal air chamber and emerge through the stomatal pore (Jones 2000). Up to 100 conidiophores can form in a sporodochium at one time, each producing conidia (Jones 1994). Infection by either ascospores or conidia results in the same type of spot and disease development (Agrios 1997).

Mycosphaerella fijiensis Morelet is the causal agent of black Sigatoka on banana foliage. Its anamorph was initially placed in *Cercospora*, but is currently known as *Pseudocercospora fijiensis* (M. Morelet) Deighton (Carlier *et al.* 2000a). *Mycosphaerella fijiensis* also reproduces by means of conidia formed in a stroma (Fig. 6A) and ascospores formed in ascostroma (Fig. 6B). In the case of *M. fijiensis,* ascospores are the dominant means of reproduction. Ascostroma are found on both leaf surfaces but more abundantly on the upper surface (Carlier *et al.* 2000a). Ascospores are two celled, slightly constricted at the septum. Conidia are formed singly at the conidiophore apex only later becoming lateral with the development of the conidiophore (Carlier

et al. 2000a). Four mature conidia may be attached to a conidiophore at any time (Carlier *et al.* 2000a).

Mycosphaerella eumusae Crous & Mourichon (anamorph *Pseudocercospora eumusae* Crous & Mourichon) causes eumusae leaf spot lesions (Crous & Mourichon 2002). It produces two distinct types of fruiting structures on the upper surface of leaf lesions (Carlier *et al.* 2000b, Carlier *et al.* 2000c). The asexual stage comprises pycnidia that are amphigenous, hypophyllous, immersed, erumpent, flask-shaped and ostiolate when young (Carlier *et al.* 2000b, Carlier *et al.* 2000c, Crous & Mourichon 2002). Contained within the pycnidia are conidia that are pale brown to sub-hyaline, fusiform and 3–5 septate (Carlier *et al.* 2000b, Carlier *et al.* 2000c). The sexual stage manifests as globose perithecia each with a with short protruding ostiole, dark brown in colour (Carlier *et al.* 2000b, Carlier *et al.* 2000c). Asci are aparaphysate, fasciculate, bitunicate, subsessile, obovoid and straight or slightly curved with eight spores. Ascospores are tri- to multiseriate overlapping, hyaline, guttulate, thick-walled, straight, obovoid with obtuse apices (Crous & Mourichon 2002). They are uniseptate and widest in the middle of the apical cell, however the basal cell may be longer (Carlier *et al.* 2000b, Carlier *et al.* 2000c, Crous & Mourichon 2002).

Mycosphaerella fijiensis, *M. musicola* and *M. eumusae* are morphologically very similar, and can be distinguished on conidial and conidiophore characteristics only (Table 3) (Mourichon *et al.* 1997, Carlier *et al.* 2000a, Carlier *et al.* 2000b). *Mycosphaerella fijiensis*, unlike *M. musicola* and *M. eumusae*, does not form sporodochia and produces few conidiophores on the lower leaf surface mainly (Meredith 1970). *Mycosphaerella musicola* and *M. fijiensis* have been shown, using restriction fragment length polymorphism (RFLP) and internal transcribed spacer

(ITS1) region sequence analyses, to be two distinct species (Carlier *et al.* 1994). It has also been proven that *Mycosphaerella* species can be easily distinguished by random amplification of polymorphic DNA (RAPD) using 10-mer or more primers (Johanson *et al.* 1994). Johanson *et al.* (1994) indicated that *M. musicola* and *M. fijiensis* differ significantly at DNA level. Johanson and Jeger (1993) developed a set of species-specific primers for molecular detection of *M. fijiensis* and *M. musicola* from infected leaf material. Johanson *et al.* (1994), used the PCR-based technique of RAPD analysis to differentiate between *M. fijiensis* and *M. musicola*. Certain sequences present in one fungus but not the other show up during DNA amplification using a sequence specific primer for each (Johanson *et al* 1994, Molina *et al.* 2001). Etienne *et al.* (1997), developed an enzyme linked immunosorbent assay (ELISA), based on polyclonal antibodies, to detect the *Mycosphaerella* species causing both yellow and black Sigatoka on bananas.

Disease cycle and epidemiology

Spread of Sigatoka leaf diseases over long distances is thought to occur via the movement of infected banana germplasm, suckers and leaves, and by means of wind-borne ascospores (Mourichon *et al.* 1997). Once introduced into a plantation, the Sigatoka pathogens are disseminated within the plantation by means of conidia and ascospores (Fig. 7) (Jones 1994, Mourichon *et al.* 1997). Conidia are the primary agents of infection in *M. musicola,* and ascospores in *M. fijiensis* (Jones 2000). Conidia are usually dislodged and disseminated by raindrops or wind (Jones 2000), and infect the unfurling heart-leaves of the same and nearby plants (Jones 2000). Older leaves are less susceptible to infection than younger leaves (Jones 1994). Perithecia are abundant in lesions of recently dead leaf tissue. Ascospores are forcibly

ejected from perithecia when necrotic tissue was saturated for approximately 48 hours during periods of high relative humidity after rain or dew (Jones 1994). They are disseminated within a plantation or over long distances (up to 50 km) by wind currents, and result in the characteristic apical spotting of infected leaves.

Relative humidity and temperature play an important role in the life cycle of Sigatoka pathogens. Under moist conditions, ascospore germination occurs on the lower leaf surface within 2–3 hours of initial attachment, whereas conidia may take a little longer (Jones 2000). Optimal temperature for germination of conidia is 25–29 °C and 25–26 °C for ascospores in *M. musicola* (Jones 2000). In *M. fijiensis*, this temperature is about 1–2 °C higher (Carlier *et al.* 2000a). After germination, the fungus grows epiphyllically for up to 6 days before producing an appressorium and penetrating the leaf (Jones 1994). Conditions required for stomatal penetration include humidity near saturation point and temperatures above 20 °C. Hyphae, therefore, may pass over some stomata before appressorium formation is initiated (Stover 1972). Germ-tubes collapse in hot dry weather and will only elongate under moist conditions. Free water on the leaves enables an infection, most often on the lower leaf surface (Jones 1994). Factors contributing to symptom development are cultivar resistance, infection intensity and environmental conditions (Jones 1994). Heavy infection of a susceptible cultivar, under favourable conditions, results in streaks progressing to spots within 10–15 days, with extensive leaf death following soon afterwards (Jones 1994). If conditions are unfavourable, disease symptoms can take as long as 105 days to appear (Jones 2000). Yellow Sigatoka follows a seasonal pattern that begins when night temperatures rise above 18°C and relative humidity increases to >92 % (Jones 2000). Temperatures required for black Sigatoka infection to take

place are 2–3 °C higher (Carlier *et al.* 2000a). Besides climate, other factors can affect disease development and intensity, such as the physiological state of the plant and light intensity. Healthy plants are in a better position to resist disease, lesions/symptoms can be repressed by shade (Jones 2000).

The epidemiology of *M. eumusae* has not yet been studied and no research has been conducted on the disease cycle in the field. However, its *Mycosphaerella* teleomorph and similar morphology and symptoms indicate that its epidemiology and disease cycle may correspond with those of *M. fijiensis* and *M. musicola*.

Control

Sigatoka leaf diseases can be managed using integrated disease control measures. These measures are based primarily on cultural, chemical and genetic control. Sigatoka diseases in commercial plantations are predominantly controlled by the use of fungicides, while control on subsistence farms involves mainly cultural practices.

Cultural control

The primary objective of cultural control is the reduction of fungal inoculum levels in banana plantations. Regular removal and destruction of leaves showing Sigatoka symptoms is, therefore, recommended (Meredith 1970). In the tropics, this practise should take place throughout the year. In the sub-tropics, deleafing should be carried out 4–6 weeks before the wet season. Since ascospores can survive in dead leaves hanging from banana plants for several weeks, deleafing reduces the ascospore production by about 85 % (Jones 2000). Banana leaves

can be buried or piled on top of each other to reduce ascospore spread even further (Meredith 1970, Jones 2000). Planting density affects the ventilation and relative humidity within a plantation. When bananas are planted too closely, the humidity is increased which favours infection by the pathogen (Meredith 1970). Irrigation should be by means of drip and not overhead sprinkle systems, as the latter increases dissemination of the pathogen within the plantation.

Resistant cultivars

Resistant cultivars offer practical control for small-scale subsistence farmers who cannot afford chemical control measures (Mourichon *et al.* 1997). International breeding programmes have made substantial progress in developing resistant cultivars of both dessert and cooking bananas (Jones 1994). Many cooking banana cultivars are resistant to Sigatoka diseases and can be used to replace susceptible plantain species (Carlier *et al.* 2000a). However, consumer taste discernment plays a role in the acceptability of new cultivars on the market.

Biological control

Biological control is, as yet, not a viable option for control of Sigatoka. However, epiphyllic fungi on the leaves of banana have been reported to inhibit the germination of *M. musicola* spores (Meredith 1970).

Chemical control

The first fungicide used for the control of Sigatoka diseases was Bordeaux mixture (Jones 2000). This was effective in preventing infection by conidia but not infection of the unfurling cigar leaf by ascospores. Zineb, copper oxychloride suspended in mineral oil, or mineral oil on

its own, give good control of yellow Sigatoka (Agrios 1997, Jones 2000). The contact dithiocarbamate fungicides, maneb and mancozeb, give reasonable control of yellow Sigatoka when applied in oil-water emulsions (Jones 2000). Mancozeb and mineral oil is currently the only fungicide registered for the control of Sigatoka in South Africa (Nel *et al.* 1999). Recently, highly effective ergosterol-inhibiting fungicides have been developed that have revolutionised disease control (Jones 1994). Fungicides of this group most commonly used for yellow Sigatoka control are the triazoles (e.g. propiconazole, systemic, and flusilazole, non-systemic). The strobilurine fungicide group, especially azoxystrobin, showed exceptional control of yellow Sigatoka and a reasonable control of black Sigatoka when used in integrated control programmes (Knight *et al.* 2002).

Fungicides can be applied to banana leaves as a fine mist by mist-blowers on the ground or aerially by helicopters/fixed-wing planes. It is important to alternate schedules between contact and systemic fungicides. Continuous use of systemic fungicides can lead to resistance, and it is recommended that less that six sprays of the same systemic fungicide should be applied in one season. Because of the higher day and night temperatures throughout the year in the tropics, more applications are necessary for the control of Sigatoka diseases. More sprays are necessary for the control of black than yellow Sigatoka. In subtropical countries, the number of sprays are reduced significantly because of the cooler night time temperatures and dryer conditions during winter. A disease monitoring and forecasting system is often used to predict climatic conditions favourable for new infections to occur. By using such systems, fungicide applications can be reduced from 25–40 down to about 10–12 per year (Jones 1994). Fungicidal sprays in the subtropics and tropics are often combined with additional control strategies in an integrated disease management programme.

SPECKLE DISEASES

Several diseases cause speckle symptoms on banana foliage, namely Mycosphaerella speckle, Cladosporium speckle, leaf speckle and tropical speckle (Jones 2000). These are considered to be of minor economic importance, as they usually do not cause extensive damage. However, under favourable climatic conditions they may become severe. Mycosphaerella speckle and Cladosporium speckle have been reported to cause leaf death in the subtropical regions of Australia (Jones 2000) and West Africa (Frossard 1963), respectively. Since 1981, leaf speckle has been considered to be a major disease of Cavendish bananas in Taiwan (Jones 2000). Tropical speckle is not regarded as a significant disease in any of the areas in which it is found. Of these diseases, Mycosphaerella speckle has predominantly been found in sub-tropical regions, while the other three are restricted to tropical areas.

Mycosphaerella speckle

Mycosphaerella speckle symptoms comprise light brown to tan coloured irregular blotches on the lower leaf surface, and smoky patches on the upper surface (Fig. 8) (Stover 1972, Jones 2000). Lower surface symptoms darken eventually to purple-black, irregularly shaped, speckled areas visible on both leaf surfaces. These blotches coalesce to form large necrotic areas that dry out and bleach with time to grey and yellow on the lower and upper leaf surfaces, respectively. Symptoms are seldom present above the fifth leaf on a banana plant (Stover 1972), and extensive leaf tissue death is usually not seen above leaf eight on actively growing plants. Nevertheless, when bunch emergence begins and leaf production ceases, severe defoliation can occur before harvest.

Mycosphaerella speckle is caused by *Mycosphaerella musae* (Speg.) Syd. & P. Syd.. The asexual stage of the fungus has not been observed on banana leaves, though *Cercospora* type conidia are produced in culture. Ascospores land on the leaf surface and produce germ tubes that grow five times faster than those of *M. musicola* (Stover 1972). Perithecia develop in large quantities on the lower leaf surface as the necrotic leaf tissue dries out (Jones 2000). At this stage, perithecia, similar to those of *M. musicola*, become visible in dead tissue and asci are forcibly released when perithecia are wet (Stover 1972, Jones 2000). Mycosphaerella speckle development is favoured by high humidity and symptoms can appear within 45 days in a saturated atmosphere (Jones 2000). In an unsaturated environment, symptoms only occur after 80–120 days. *Mycosphaerella musae* is always more prevalent in sheltered areas like hollows or moist areas in plantations, and the disease develops more quickly on senescing or injured leaves. Infection does not take place on younger leaves, and advanced symptoms of the disease are seen mainly on older leaves (Jones 2000). Temperatures below 20 °C retard disease development, and plant growth, resulting in speckle symptoms being more pronounced in winter in subtropical areas (Jones 2000).

Fungicides of the fixed copper and dithiocarbamate groups are used as oil-in-water emulsions for the control of *M. musae* (Stover 1972). Copper oxychloride, maneb and zineb have given good control when applied to the underside of leaves 4–6 (Stover 1972, Jones 2000). Control has also been successful when protectant fungicides were sprayed onto the lower surfaces of leaves 4–6. Fungicides such as mancozeb and propiconazole, those currently used in Sigatoka control, also provide acceptable control of Mycosphaerella speckle (Jones 2000). Leaves killed

by the fungus should be removed to reduce the level of inoculum and prevent inoculum build-up (Jones 2000).

Cladosporium speckle

Cladosporium speckle is caused by *Cladosporium musae* E.W. Mason, a weak pathogen affecting mainly mature leaves in humid climates (Stover 1972, Jones 2000). Although the disease is considered to be a minor problem, it can cause damage to susceptible cultivars under favourable environmental conditions (Jones 2000). Badly diseased leaves dry out and drop prematurely, lowering the photosynthetic capacity of the plant (Jones 2000). Cladosporium speckle has been reported in Australasia-Oceania, Asia, Africa and the Latin American-Caribbean region (Jones 2000).

Cladosporium speckle manifests as a diffuse grey-brown blotching on the upper surface of older leaves (Jones 1994). These lesions become yellow-orange and then necrotic with age (Fig. 9A) and are more commonly found along leaf margins (Jones 1994). Conidiophores that arise from a profuse epiphyllic mycelium on the adaxial surface can easily be observed with a hand-lens (Wardlaw 1961). Initial symptoms in West Africa appear as pale brown pencil-mark-like spots, approximately 0.3 x 1.5 mm and can be seen 3–4 weeks after unfurling of the youngest leaf (Stover 1972, Siboe 1994, Jones 1994, Jones 2000). Spots elongate into lesions of 15 x 30 mm that coalesce turning yellow, then violet-black (Fig. 9B) (Stover 1972, Jones 1994, Jones 2000). In Uganda, Malaysia and Thailand symptoms are similar to those described above. Initial symptoms are small spots that elongate into grey and later brown streaks, and can be easily mistaken for black Sigatoka (Jones 2000). At high levels of infection the grey lesions coalesce

forming a larger orange region surrounded by a yellow halo (Jones 2000). These regions later turn a violet-black colour before the leaf tissue dies (Jones 2000). Lesions have been observed on leaf midribs and entire leaves can become necrotic (Jones 2000). Symptoms in Central America are initially a diffuse grey-brown blotching on the upper surfaces of the oldest leaves. This later turns yellow-orange and then becomes brown and necrotic (Jones 2000).

Cladosporium speckle development is favoured by high humidity (Jones 2000). Conidia of *C. musae* are carried on air currents and germinate in moisture on the leaf surface (Jones 1994, Jones 2000). Older leaves are the first to be infected, transferring inoculum to younger ones (Jones 2000). In Kenya, severe disease was reported to be present from the sixth leaf of naturally infected plants that have not yet flowered. Complete leaf damage, however, was observed on flowered plants at harvest (Siboe 1994). Infection severity is recorded as being varied between cultivars, with dessert bananas being the most susceptible and cooking bananas the most resistant (Siboe 1994).

Fungicides used in the control of yellow and black Sigatoka are effective against Cladosporium speckle of banana (Jones 2000). In Thailand the disease is controlled by fortnightly applications of benomyl during the rainy season (Jones 2000). Early removal of diseased leaves is recommended in Ethiopia to limit inoculum buildup(Jones 2000). On the Ivory Coast, 500 g of metallic copper per hectare is applied every 15 days in the Sigatoka oil spray (Stover 1972). Maneb sprays are preferred to copper for leaf diseases, as copper sprays may cause injury (Stover 1972). Excessive plant populations favour infection due to increased humidity. Reduced planting density, therefore, reduces speckling (Stover 1972).

Leaf speckle

Leaf speckle is a minor disease on banana foliage occurring in South-East Asia. However, in 1981 it was described as a threat to commercially grown "Cavendish" bananas in Taiwan (Jones 2000). Leaf speckle symptoms have also been recorded in Australia, Malaysia, Thailand and Vietnam. They appear as small dark brown specks densely aggregated on the lower leaf surface (Jones 2000). These develop into streaks that run parallel to the leaf veins up to 4 x 0.3 mm in size. Tan coloured blotches on the upper leaf surface are concentrations of these streaks. The affected areas eventually yellow and become necrotic (Fig. 10).

Acrodontium simplex (F. Mangenot) de Hoog (*Hyphomycetes*) causes leaf speckle on banana leaves (Jones 2000). Conidia usually cover the terminal portion of the conidiophore (Ellis 1967). Disease development of the leaf speckle pathogen is affected by climatic conditions (Jones 2000). Incubation is up to 35 days in hot, wet summer months and 60 days in cool, dry winter months (Ellis 1967, Jones 2000). Inoculum builds up on unsprayed Cavendish cultivars after shooting. In southern Taiwan climatic conditions influence disease development. Fungicides used in the control of yellow and black Sigatoka, containing dithiocarbamates in oil, are effective against leaf speckle (Jones 2000).

Tropical speckle

Tropical speckle has been observed on banana leaves in hot humid environments world-wide, but it is not perceived as a threat as it has no effect on either plant growth or yield (Jones 2000). Tropical speckle is found in the Asian-Pacific, Latin-America, the Caribbean and in Africa.

Tropical speckle manifests as two types of symptoms occurring on the same leaf. The first is initially a circular, chlorotic blotch, up to 4 cm in diameter, which progresses to form tan blotches abaxially and dark brown-black pinprick specks heavily distributed adaxially (Fig. 11A). Conidiophores are clearly visible within lesions and form a bristle-like, dense mass on the adaxial surface. The second is a dark, irregular blotch on lower surface consisting of black specks with extensive discoloured areas less distinct on the upper surface (Fig. 11B). Visible conidiophores are velvety within lesions and along the midrib and peduncle.

Two fungi are thought to be the cause of tropical speckle, namely *Veronaea musae* M.B. Ellis and *Periconiella musae* Stahel ex M.B. Ellis (*Hyphomycetes*). There are several differences between the two organisms. Conidiophores of *V. musae* are unbranched and shorter than those of *P. musae* that are branched and longer. Conidia of *V. musae* are oval and have tiny papillae at the point of attachment to the conidiophore, while those of *P. musae* are more elliptical in shape. *Periconiella musae* grows slower than *V. musae* on PDA although they have similar colony characteristics. Tropical speckle inoculum germinates within 24 hours of landing on the host and produces hyphae on the lower leaf surfaces (Jones 2000). It forms a loose epiphyllic network of branched hyphae, 1–2 µm thick (Wardlaw 1961). Club-shaped stomatopodia, that are darker than

the hyphae, form and infection tubes enter the leaf via stomata. Infection hyphae grow through the stomatal air space into palisade tissue, and side branches enter cells. The fungus does not spread further than the tissue surrounding the colonised stoma. Necrosis, therefore, is confined to the area of infection, resulting in a speckled appearance (Ellis 1967). Tropical speckle agents are considered as weak parasites. In Australia and Papua New Guinea symptoms only appear in unsprayed plantations in high rainfall and shady moist areas, and in Central America on lower leaves during high rainfall seasons. Fungicides used in the control of yellow and black Sigatoka are effective against tropical speckle of banana (Jones 2000).

FRECKLE

Freckle disease attacks leaves and fruit of banana in Africa, Australasia-Oceania, Caribbean, the Philippines (abaca), South and East Asia, Taiwan and West Malaysia (Jones 2000). In some regions of Taiwan and the Philippines, freckle is regarded as being more serious than black Sigatoka. On local markets, freckle blemishes on fruit are not a problem. Heavy leaf infections, however, can cause complete defoliation and reduction in yield.

Freckle disease of bananas is caused by *Guignardia musae* F. Stevens (*Ascomycetes*) (anamorph *Phyllosticta musarum* (Cooke) Aa (*Coelomycetes*)). The pycnidial stage is usually present on the host and about five pycnidia are contained within small spots (Meredith 1968). Symptoms manifest as two types of leaf spotting on the upper surfaces of older leaves (Meredith 1968). The first is a very small, dark brown-black spotting less than 1 mm in diameter, giving the leaf a sooty appearance and rough texture. This symptom is formed by spots clustering in

lines and running diagonally or horizontally across a leaf or fruit surface (Figs. 12A, B). The second symptom is a 4-mm diameter distinct brown-black spot with a grey-fawn centre. These spots coalesce into large black areas or streaks on which pycnidia are prominent under a raised epidermis (Fig. 12C). Diseased leaves and fruit feel rough to the touch. In severe cases leaves turn yellow, wither and die prematurely (Jones 1994). Symptoms are seen on petioles, midribs and transition leaves.

During the wet season, ascospores of *G. musae* are forcibly released from perithecia and conidia of *P. musarum* exuded as white gelatinous tendrils from pycnidia. They are carried in water droplets across leaves and fruit where infection occurs. Germination occurs within 2–3 hours of initial contact with the host at a temperature of about 24 °C. Twelve hours later a lateral swelling differentiates into an appressorium in grooves on the leaf surface between adjacent host cells. At 24–72 hours, a single epidermal cell is invaded, many of these invasions cause a scattered pattern that begins to discolour due to penetration by infecting appressoria. Pycnidia develop in lesions 3 weeks after inoculation and thick intercellular hyphae accumulate 2–3 cell layers below the epidermis. They differentiate, enlarge and break through the epidermis causing secondary leaf infections to intensify symptoms and resulting in streaks. In Taiwan the incubation period varies from 20 days in warm wet weather, to 60 days in cool dry weather, and conidia present in water drop from leaves to fruit (Jones 2000).

Propiconazole has been shown to be the most effective means of controlling freckle (Jones 2000). Mancozeb is effective as well, with benomyl giving minimal control and mineral oil none. In the Philippines, propiconazole and flusilazole, used in the control of black Sigatoka,

also control freckle. In Taiwan, removal of lower diseased leaves reduces inoculum levels and bagging of bunches reduces fruit infection (Jones 2000).

MINOR LEAF DISEASES

Some diseases of banana leaves are considered to be of minor economic importance. They are usually caused by opportunistic pathogens, but the symptoms they cause can cover entire leaves. However, these diseases seldom result in a reduced yield as with Sigatoka and speckle diseases. Some minor diseases of banana leaves include Cordana leaf spot, Phaeoseptoria leaf spot, black cross leaf spot, Deightoniella leaf spot, Drechslera leaf spot, Malayan leaf spot, Pestalotiopsis leaf spot, Pyricularia leaf spot and rust.

Cordana leaf spot

Cordana leaf spot is caused by *Cordana musae* (Zimm.) Höhn. and *Cordana johnstonii* M.B. Ellis, two species that can be morphologically differentiated on the grounds of conidial size and shape, with *C. johnstonii* having longer and wider conidia than *C. musae* (Jones 2000). It has a world-wide distribution and has been reported as a serious problem on plantains in Central America during and after wet seasons (Wardlaw 1961, Stover 1972, Jones 2000). Epidemics have occurred on the "Williams" cultivar in Southern New South Wales, Australia (Jones 2000). Cordana specimens collected in Australia since the 1930's showed that *C. johnstonii* was limited to New South Wales, Lord Howe Island and Norfolk Island, and *C. musae* to Queensland and the Northern Territory, suggesting that *C. johnstonii* is better adapted to cooler environments than *C.*

musae (Jones 2000). Characteristic Cordana leaf spot lesions are large, oval to fusiform, pale brown and necrotic (Fig. 13) (Jones 2000). Leaf spots caused by *C. johnstonii* are smaller, more regular in outline and more tapered than the larger oval spots of *C. musae* (Jones 2000). Cordana leaf spot often infects leaf margins where the lamina is more likely to tear due to senescence or nutritional deficiency (Jones 2000). Quite frequently, *C. musae* also infects around lesions caused by pathogens such as, *M. musicola*, *M. fijiensis* and *M. eumusae* (Stover 1972, Jones 1994, Jones 2000).

Cordana johnstonii sporulates profusely on the adaxial surface and *C. musae* on the abaxial surface of leaf spots on green leaves and leaves in leaf litter during cool misty weather (Jones 1994, Jones 2000). Conidia are forcibly released under conditions of decreasing vapour pressure at dawn when humidity drops (Stover 1972, Jones 2000). The conidia germinate and produce germ tubes and appressoria that penetrate the leaf surface (Jones 2000). Germination and infection time is 6–48 hours, depending on temperature, and symptoms are produced within 4–10 days (Jones 2000). Under laboratory conditions the optimum temperature for conidium production on living leaves is 22 °C and on detached leaves 22–25 °C (Jones 2000). *Cordana musae* is usually not economically important and therefore does not warrant control (Jones 2000). However, when outbreaks arise, the fungicides used in the control of yellow and black Sigatoka provide adequate control of this fungus (Stover 1972, Jones 2000).

Phaeoseptoria leaf spot

Phaeoseptoria musae Punithalingham is the organism responsible for Phaeoseptoria leaf spot of banana (Jones 2000). It is thought that *Mycosphaerella* is possibly the perfect stage. Conidia are dispersed by rain splashes and ascospores are forcibly ejected during wet weather. Phaeseptoria leaf spot affects only older leaves, causing ellipsoidal to ovoid spots with pale centres, a dark brown border and a yellow halo (Punithalingham 1983). At high densities lesions coalesce, forming large irregularly shaped necrotic areas with off-white to yellow centres. Phaeoseptoria leaf spot causes severe blight of banana leaves in Australia, Cameroon Colombia, East Malaysia Ghana, Guyana, Honduras, Kenya, India, Tanzania, Trinidad and Uganda (Jones 2000). There is no information available on control measures for the disease.

Black cross leaf spot

Black cross leaf spot, caused by *Phyllachora musicola* C. Booth & D.E. Shaw, has been reported from American Samoa, Australia, Fiji, Indonesia, New Caledonia, Niue, Papua New Guinea, Philippines, Tonga, Vanuatu and Western Samoa (Jones 2000). An asexual state has not been observed, but it is thought to be *Scolecobasidium* (Hyde 1992). Black cross leaf spot is found on the lower surface of older leaves as large, black, four-pointed crosses, stars or diamonds (Fig. 14A). Upper surface symptoms are usually black dots, but in Western Samoa they are yellow diamond-shaped spots interspersed with dark brown lines (Fig. 14B). The typical black crosses on abaxial surface are the mature stroma of *P. musicola*. During humid conditions, white masses of spore exudate collect along the arms of the stromatic black cross (Jones 2000).

Ascospores are carried in water drops and spread the disease between leaves. Ascospores become airborne when forcibly ejected from perithecia, dispersing disease over wider areas (Wardlaw 1961). Commercial AAA cultivars (Cavendish) are fairly resistant to the disease so no control measures are practised. In Western Samoa planting in sunny places rather than in shade is recommended.

Deightoniella leaf spot

Deightoniella leaf spot is caused by *Deightoniella torulosa* (Syd.) M.B. Ellis. The disease has been recorded on senescing or injured leaves in Bermuda, Brazil, Central America, Ceylon, Ethiopia, Ghana, India, Jamaica, Peru, Philippines, Sierra Leone, Suriname and Trinidad (Jones *et al.* 2000a). Lesions are most prevalent along the leaf blade and on older lower leaves. The disease is mainly reported from Cavendish cultivars in plantations, though various abacá and enset varieties have been reported to be susceptible (Jones *et al.* 2000a). Initial symptoms are tiny, black, necrotic spots (1–2 mm) that become oval with a black border (may be confused with Cordana leaf spot at this stage) (Fig. 15) forming a smoky colouring over a tan background. Mature spots reach up to 25 mm in diameter, coalesce and form bands of necrotic tissue along leaf margins (Stover 1972). Stem lesions are similar to speckle symptoms having a defined black margin, 1–2 mm wide, and a yellow halo that encompasses each lesion. Mature lesions brown and dry out, leaving the original black spots visible. This results in a speckle-like spotting on petioles and pseudostem. *Deightoniella torulosa* is found in living and dead leaf tissue. Inoculum is produced during rain and dew periods when spores are forcibly discharged and become airborne when the humidity drops (Ellis 1957). They germinate in surface water and symptoms

develop within days. Spores are not transmitted long distances and viability is lost within 4 days at humidities less than 95 % (Wardlaw 1961). No control measures are required on banana, however dead leaf litter should be removed to prevent inoculum build-up. Only resistant abaca and enset cultivars should be planted in areas where the disease is known to occur (Jones *et al.* 2000a). Additional control measures encompass reduction of planting density, harvesting before rotting becomes severe as well as cutting down and burning badly diseased plants (Jones *et al.* 2000a).

Drechslera leaf spot

Drechslera gigantea (Heald & F.A. Wolf) S. Ito is the causal agent of Drechslera leaf spot. The disease is found in Central America, Ethiopia and Jamaica (Jones *et al.* 2000b). Symptoms manifest as tiny, sunken, red spots with pale green or yellow borders (Meredith 1968). These spots become oval toward the leaf veins, centres turn dark brown and dry to a white/grey surrounded by a thin defined dark brown margin with a pale yellow-green halo (Fig. 16A). Spots measure 16 x 8 mm on leaves, midribs and petioles. Symptoms on enset may coalesce and form large necrotic areas of severe blight symptoms on the unfurling, first and second leaves (Fig. 16B). The fungus also causes eyespot on banana leaves of suckers less than 2 m tall, during wet weather and heavy dewfall. Conidia are forcibly expelled under decreased humidity and surface moisture favours germination and infection (Stover 1972). Early removal of diseased leaves is recommended to reduce inoculum levels in plantations (Jones *et al.* 2000b).

Malayan leaf spot

Malayan leaf spot, caused by *Haplobasidion musae* M.B. Ellis, affects banana leaves in Fiji, Papua New Guinea, Tonga, West Malaysia and Western Samoa (Jones 2000). Infection occurs on young leaves soon after they emerge (Jones 2000). Adaxial symptoms are diamond shaped, grey-white spots, with the longer axis parallel to leaf veins. Abaxial symptoms display a velvety mass of mycelium (Ellis 1957). In West Malaysia spots are ellipsoidal or round, with spotting in white, grey or brown. Lesions are paler on the adaxial surface and have dark purple borders. In Papua New Guinea there is no typical diamond shape, lesions vary in size, are round and ellipsoidal with well-defined dark borders and grey centres on the leaf blade and midrib (Fig. 17). At high infection intensities, tissue surrounding lesions yellows and large necrotic areas develop with the dark borders and grey centres of the original lesions still visible (Jones 2000). Shade and cool temperatures favour development of symptoms (Jones 2000). Short daylight hours, high humidity and high rainfall render leaves more susceptible when the plant is near flowering at cooler temperatures. The disease occurs in West Malaysia at altitudes between 1372 m and 1525 m, and in Papua New Guinea well above 1000 m. In western Samoa disease incidence increases with altitude, and in Tonga the cooler climate at sea level is conducive to the disease (Jones 2000). Leaves sprayed with oil predispose banana plants to infection by *H. musae*. Maneb applied in water is recommended to delay symptom appearance in areas were disease is severe (Jones 2000).

Pestalotiopsis leaf spot

Pestalotiopsis leaf spot, that occurs in Central America and Jamaica, is caused by *Pestalotiopsis palmarum* (Cooke) Steyaert (Jones 2000). Symptoms manifest as yellow-brown circular spots between leaf veins. Spots develop until they reach vascular tissue, then spread and linearly extend lesions (Meredith 1968). Grey-fawn spots can develop around tears or abrasions in leaf margins, with necrosis progressing outwards and possible extension to the midrib. Concentric zones are visible within spots, beginning at the centre, with a narrow dark brown band surrounded by a bright yellow-orange halo (Stover 1972). No control strategies are currently available.

Pyricularia leaf spot (enset)

Pyricularia leaf spot occurs in Sidamo and North Omo in Ethiopia (Tessera & Quimio 2000). It forms circular, oblong or spindle-shaped lesions with dark borders. Leaves, midribs, petioles and leaf sheaths are affected (Figs. 18A, B) and lesions coalesce and form large necrotic areas (Tessera & Quimio 2000). A *Pyricularia* sp. has been isolated and found to be pathogenic to enset and not to the AAA (Cavendish) banana cultivars. Most enset cultivars are susceptible to the fungus, but older plants are not affected. Pyricularia leaf spot can become severe on young enset seedlings and lead to leaf death. Older plants are not badly affected, and removal of infected leaves is recommended to prevent disease spread (Tessera & Quimio 2000).

Rust

Two different fungi cause rust on banana leaves, *Uromyces musae* Henn and *Uredo musae* Cummins (Jones 2000). Uredosori form on the lower surfaces of older leaves as lesions elongate, broaden and coalesce, causing the leaf to yellow. At high infection densities necrosis sets in and powdery, light brown masses of uredospores cover lesions (Fig. 19). Uredosori act as points of entry for secondary infections (Wardlaw 1961). Wind blown uredospores are thought to transfer the fungus between plants (Jones 2000). They germinate in surface water and invade the leaf tissue. Cavendish cultivars are susceptible to rust when sprayed with mineral oil. Rust symptoms are found on banana leaves in Australia, Congo, Fiji, Malaysia, Nigeria, Papua New Guinea, Philippines, Wallis Islands and Western Samoa (Jones 2000). No control is warranted for rust. Severe outbreaks that have occurred on Cavendish plantations can be related to the use of oil or benomyl to control leaf spot diseases (Jones 2000).

ENDO- AND EPIPHYTES OF BANANAS

Numerous fungi are associated with the foliage of banana plants. Some of these fungal taxa have been reported as endo- or epiphytes on *Musa* species, particularly on wild banana (*Musa acuminata* Colla) (Table 4). Fungi isolated most commonly include *Colletotrichum gloeosporioides* (Penz.) Penz. & Sacc., *Curvularia* spp., *Fusarium* spp., *Nigrospora* spp., *Pestalotiopsis* spp., *Phomopsis* spp., xylariaceous taxa and unknown sterile species (Brown *et al.* 1998, Photita *et al.* 2001, Photita *et al.* 2002). Some of these, e.g. *C. gloeosporioides*, can cause disease in banana fruit, whereas others, such as *Phoma* and *Phomopsis* species, may become

problematic under environmental stress (Brown *et al.* 1998). However, many asymptomatic endophytic colonisers exist mutualistically with their hosts, possibly benefiting the latter by protecting them from attack by pathogens (Petrini 1993, Dorworth & Callan 1996).

References

Agrios GN. 1997. Foliar diseases caused by ascomycetes and imperfect fungi. Pp. 307, 309 In: Plant Pathology. San Deigo, USA. Academic Press.

Brown KB, Hyde KD, Guest DI. 1998. Preliminary studies on endophytic fungal communities of *Musa acuminata* species complex in Hong Kong and Australia. *Fungal Diversity* **1**: 27–51.

Carlier J, Mourichon X, González-de-Léon D, Zapter MF, Lebrun MH. 1994. DNA Restriction fragment length polymorphisms in *Mycosphaerella* species that cause banana leaf spot diseases. *Phytopathology* **84**: 751–756.

Carlier J, Fouré E, Gauhl F, Jones DR, Lepoivre P, Mourichon X, Pasberg-Gauhl C, Romero RA. 2000a. Black leaf streak. Pp 37–79. In: Disease of banana, abaca and enset. (Ed. Jones DR). Wallingford, UK. CABI Publishing.

Carlier J, Mourichon X, Jones DR. 2000b. Septoria leaf spot. Pp 93–96. In: Disease of banana, abaca and enset. (Ed. Jones DR). Wallingford, UK. CABI Publishing.

Carlier J, Zapater MF, Lapeyre F, Jones D.R, Mourichon X. 2000c. Septoria leaf spot of banana: a newly discovered disease caused by *Mycosphaerella eumusae* (Anamorph *Septoria eumusae*). *Phytopathology* **90**: 884–890.

Crous PW, Mourichon X. 2002. *Mycosphaerella eumusae* and its anamorph *Pseudocercospora eumusae* spp. nov.: causal agent of eumusae leaf spot disease of banana. *Sydowia* **54**: 35–43.

Dorworth CE, Callan BE. 1996. Manipulation of endophytic fungi to promote their utility as vegetation biocontrol agents. Pp 209–218. In: Endophytic fungi in grasses and woody plants. (Eds. Redlin SC, Carris LM). St. Paul, Minnesota, USA. American Phytopathological Society Press.

Ellis MB. 1957. Some species of *Deightoniella*. *Mycological Papers* **66**: 1–12.

Ellis MB. 1967. Dematiaceous Hyphomycetes. Kew, Surrey, UK. Commonwealth Mycological Institute.

Etienne L, Steden C, Suter J. 1997. A new diagnostic tool for *Mycosphaerella* spp. in banana leaves. Pp 377–383. In: Diagnosis and identification of plant pathogens. (Ed. Dehne HW). Netherlands, Kluwer Academic Publishers.

Frossard P. 1963. Une cladosporiose du bananier en Côte d'Ivoire. *Fruits* **18**: 443–453.

Hyde KD. 1992. *Phyllachlora musicola*. IMI descriptions of fungi and bacteria no. 1127. *Mycopathologia* **119**: 57–58.

Johanson A, Crowhurst RN, Rikkerink EHA, Fullerton RA, Templeton MD. 1994. The use of species-specific DNA probes for the identification of *Mycosphaerella fijiensis* and *M. musicola*, the causal agents of Sigatoka disease of banana. *Plant Pathology* **43**: 701–707.

Johanson A, Jeger MJ. 1993. Use of PCR for detection of *Mycosphaerella fijiensis* and *M. musicola*, the causal agents of Sigatoka leaf spots in banana and plantain. *Mycological Research* **96**: 670–674.

Jones DR. 1994. Part 1: Banana. Pp 2–3. In: Compendium of Tropical Fruit Diseases. (Eds. Ploetz RC, Zentmyer GA, Nishijima WT, Rohrbach KG, Ohr HD) St. Paul, Minnesota, USA. The American Phytopathological Society.

Jones DR. 2000. Fungal diseases of the foliage. Pp 37–141. In: Disease of banana, abaca and enset. (Ed. Jones DR). Wallingford, UK. CABI Publishing.

Jones DR, Lomerio EO, Tessera M, Quimio AJ. 2000a. Deightoniella leaf spot. Pp 102–104. In: Disease of banana, abaca and enset. (Ed. Jones DR). Wallingford, UK. CABI Publishing.

Jones DR, Tessera M, Quimio AJ. 2000b. Drechslera leaf spot. P 105. In: Disease of banana, abaca and enset. (Ed. Jones DR). Wallingford, UK. CABI Publishing.

Knight S, Wirz M, Amil A, Hall A. 2002. The role of fungicide resistance management in maintaining the effectiveness of integrated strategies for control of black Sigatoka. 2nd International workshop on *Mycosphaerella* leaf spot diseases of bananas. San José, Costa Rica.

Meredith DS. 1968. Freckle disease of banana in Hawaii caused by *Phyllostictina musarum*. *Annals of Applied Biology* **62:** 328–340.

Meredith DS. 1970. Banana leaf spot disease (Sigatoka) caused by *Mycosphaerella musicola* Leach. Kew, Surrey, UK. Commonwealth Mycological Institute.

Molina C, Kaemmer D, Aponte S, Weising K, Kahl G. 2001. Microsatellite markers for the fungal banana pathogen *Mycosphaerella musicola*. *Molecular Ecology Notes* 1: 137–139.

Mourichon X, Carlier J, Fouré E. 1997. Sigatoka Leaf Spot Diseases. PROMUSA Sigatoka working group. Montpellier.

Nel A, Krause M, Ramautar N, Van Zyl K. 1999. A guide for the control of plant diseases. Pretoria, South Africa. Government printer.

Petrini O. 1993. Endophytes of *Pteridium* spp.: some considerations for biological control. *Sydowia* **45**: 330–338.

Photita W, Lumyong S, Lumyong P, Hyde KD. 2001. Endophytic fungi of wild banana (*Musa acuminata*) at Doi Suthep Pui National Park, Thailand. *Mycological Research* **105**: 1508–1513.

Photita W, Lumyong S, Lumyong P, Hyde KD, McKenzie EHC. 2002. Index of fungi described from the *Musaceae*. *Mycotaxon* **81**: 491–503.

Picq C, Ammar-Khodja P. 2000. Bananas. International Plant Genetic Resources Institute.

Ploetz R. 1999. The most important disease of a most important fruit. APSnet (Plant Pathology Online).

Punithalingam E. 1983. *Phaeoseptoria musae*. Description of pathogenic fungi and bacteria no. 772. Kew, Surrey, UK. Commonwealth Mycological Institute.

Robinson JC. 1996. Bananas and Plantains. Crop production science in horticulture 5. Wallingford, UK. CABI Publishing.

Robinson JC, Peterson RA. 1999. Banana leaf diseases in South Africa with an emphasis on the yellow Sigatoka problem. *Banana Growers Association of South Africa Yearbook* **3**: 83–90.

Sasson A. 1997. Importance of tropical and subtropical horticulture, future prospects of biotechnology in the tropical and subtropical horticulture species. International Society for Horticultural Science (ISHS). *Leiden/Acta Horticulturae* **460**: 12–26.

Siboe GM. 1994. Taxonomy of the fungus causing speckling disease of bananas (*Musa* spp.) in Kenya. *The African Journal of Mycology and Biotechnology* **2**: 1–6.

Simmonds NW. 1959. Diseases. Pp 366–408. In: Bananas. London, UK. Longmans.

Stover RH. 1972. Fungus diseases of the foliage. Pp 37–111. In: Banana, plantain and abaca diseases. Kew, Surrey, UK. Commonwealth Mycological Institute.

Tessera M, Quimio AJ. 2000. Pyricularia leaf spot of enset. Pp 108. In: Disease of banana, abaca and enset. (Ed. Jones DR). Wallingford, UK. CABI Publishing.

Wardlaw CW. 1961. Leaf-Spot (Sigatoka disease), Sigatoka Disease: Control Measures, Other Leaf Diseases. Pp 314–407. In: Banana Diseases. Edinburgh, Longmans.

Table 1: Stages of development of yellow Sigatoka lesions on banana leaves (Stover 1972, Jones 1994, Agrios 1997, Robinson & Peterson 1999, Jones 2000).

Stage	Symptom
1 (speck/fleck)	Yellow-green flecks shorter than 1 mm.
2a (Initial streak)	Flecks enlarge to 3–4 mm x 1 mm streaks, the lesion becomes yellow.
2b (Final streak)	Streaks become a dark brown colour.
3 (Initial spot)	Streaks enlarge and become spots, definite margins that may become water-soaked darken to brown.
4 (Brown spot)	Spots have a definite dark brown margin, sometimes with a yellow halo, the lesion centre begins to sink in. Conidia are produced on the surface of the lesion.
5 (Mature spot)	Sunken lesion centres turn grey but retain their brown border often with a halo. Ascospores are produced in the grey central area of the mature spot.

Table 2: Differences between conidial and ascospore infection of banana leaves by *Mycosphaerella musicola* (adapted from Simmonds 1959, Jones 2000).

Characteristic	Conidia	Ascospores
Development	Night temperature ≥ 18°C	Night temperature ≥ 21°C
Leaf stage	Stage 4 and 5	Stage 5
Fruiting structures	On both sides of leaves	Mainly on upper surface
Dispersal	Rain or dew	Rain
Spread	Water-splashes (airborne)	Carried on wind-currents
	Short distance spread	Long distance spread
Infection Moisture	High humidity	Film of water on leaf
Germination	24–48 hr	2–3 hr
Penetration	4–6 d	48–72 hr
Survival		47–48 weeks
Production time	Wet season, continuous	Middle-late wet season
Production area	Leaf surface	Internal to leaf
Transmission	Water	Ejected, airborne
Infection	Heart leaf	Tips of 2nd and 3rd leaves
Pattern	"Line spotting"	"Tip spotting"

Table 3: Morphological differences between the anamorph structures of *M. musicola, M. fijiensis* and *M. eumusae* (Crous & Mourichon 2002).

Species	Sporodochia	Conidiogenous cells	Conidiophores	Conidia
M. fijiensis	Sporodochia absent, produces few conidiophores on the lower leaf surface	Up to 25 x 2–4 µm at apex, 1–3 thickened scars.	Hypophyllous fascicles, pale brown, 0–5 septate, straight to geniculate, occasionally branched, subcylindrical, 16.5–62.5 x 4–7 µm. One or more scars are present near tip	Sub-hyaline, obclavate to cylindric obclavate, 1–10 septate, slightly thickened and darkened hila, 10–120 x 2.5–5 µm.
M. musicola	Amphigenous, dark brown substomatal stromata.		Pale brown, aseptate, unbranched, straight to bottle-shaped, 5–25 µm long, lack any visible scarring.	Smooth, pale olivaceous, cylindrical to cylindric obclavate, straight to curved, 0–8 septate, subtruncate to obclavate ends, no significant scarring, 10–80 x 2–6 µm.
M. eumusae	Epiphyllous, dark brown substomatal stromata.	Truncate ends	Sub-hyaline to pale olivaceous, pale brown at the base, sub-cylindrical, 0–3 septate, 10–25 x 3–5 µm, longer and more septate than *M. musicola*.	Sub-hyaline to pale olivaceous, sub-cylindrical, 3–8 septate, subtruncate ends, no visible scarring, 18–65 x 2–3 µm.

Table 4: Summary of fungi reported from banana leaves (Brown *et al.* 1998, Photita *et al.* 2001, Photita *et al.* 2002).

Phylum	Species	Authority
Ascomycota	*Alternaria alternata*	(Fr.) Keissl.
	Antennularia tenuis	Earle
	Anthostomella moelleriana	G. Winter
	Botryosphaeria musae	
	Cladosporium cladosporioides	(Fresen.) G.A. de Vries
	Cladosporium liukiuensis	
	Colletotrichum gloeosporioides	(Penz.) Penz. & Sacc.
	Colletotrichum musae	(Berk. & M.A. Curtis) Arx
	Cryptosporella musarum	J.N. Kapoor
	Diaporthe musae	Speg.
	Dothidea musae	Klotzsch
	Dothidella musae	Höhn.
	Epicoccum nigrum	Link
	Fusarium lateritium	Nees
	Fusarium solani	(Mart.) Sacc.
	Glomerella musarum	Petch
	Guignardia cocoicola	Punithalingham
	Guignardia musae	F. Stevens
	Lasiodiplodia theobromae	(Pat.) Griffiths & Maubl.
	Leptosphaeria musae	
	Leptosphaeria musigena	
	Leptosphaeria taichungensis	
	Metasphaeria taiwanensis	
	Micronectriella stoveri	C. Booth
	Microsphaeriopsis sp.	
	Mycosphaerella formosana	
	Mycosphaerella liukiuensis	
	Nectria foliicola	Berk. & M.A. Curtis
	Nectria nymaniana	Henn.
	Phacidium musae	Lév.
	Phyllachora musicola	C. Booth & D.E. Shaw
	Physalospora fallaciosa	Sacc.
	Plicaria musicola	Henn.
	Pyriculariopsis parasitica	(Sacc. & Berl.) M.B. Ellis
	Sphaerulina musae	
	Sphaerulina musicola	
	Sphaerulina pulii	J.M. Yen
	Stachybotrys sp.	
	Venturia musae	

	Verticillium sp.	
Basidiomycota	*Cyphella musicola*	Berk. & M.A. Curtis
	Helotium musicola	Speg.
	Psathyra musicola	Henn.
	Uredo musae	Cummins
	Uredo musicola	
	Uromyces musae	Henn.
Mitosporic	*Alternaria musae*	Bouriquet & Bataille
	Cercospora fengshanensis	T.Y. Lin & J.M. Yen
	Cercospora hayi	Calp.
	Cercospora musae	Massee
	Cercospora musae sapienti	A.K. Kar & M. Mandal
	Cercospora musaecola	Sawada
	Cercospora pingtungensis	T.Y. Lin & J.M. Yen
	Chaetophoma musae	Cooke
	Fusidium musae	
	Gliomastix elata	C.H. Dickinson
	Hainesia tellingsii	Koord.
	Haplobasidion musae	M.B. Ellis
	Leptothyrium musae	Cif. & Gonz. Frag.
	Nigrospora musae	McLennan & Hoëtte
	Nigrospora oryzae	(Berk. & Broome) Petch
	Parapyricularia musae	M.B. Ellis & Peregrine
	Pellionella musae	
	Penicillium atrobrunneum	Cooke
	Pestalotiopsis chethallensis	Sohi & O. Prakash
	Pestalotiopsis palmarum	(Cooke) Steyaert
	Phomatospora musae	
	Phyllosticta gastonis	Roum.
	Phyllosticta musae	F. Stevens & E. Young
	Phyllosticta musicola	F. Stevens & E. Young
	Pyricularia angulata	Hashioka
	Sphaeropsis paradisiaca	Mont.
	Veronea musae	

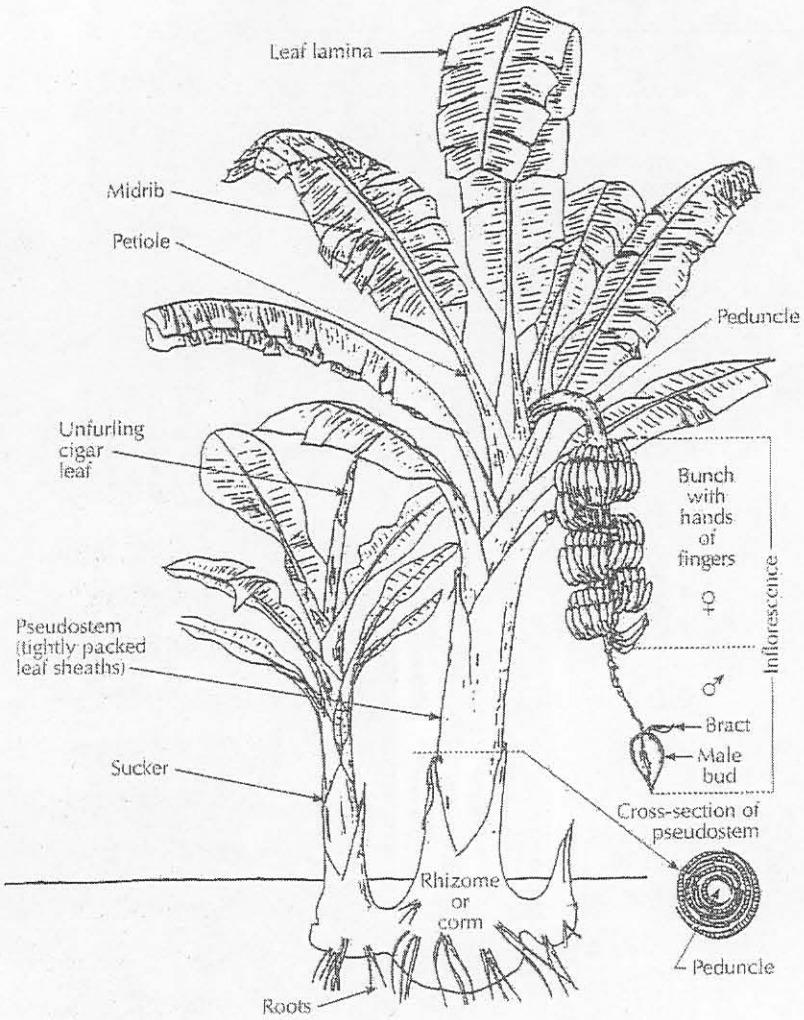

Figure 1: Anatomy of the banana plant (Jones 2000).

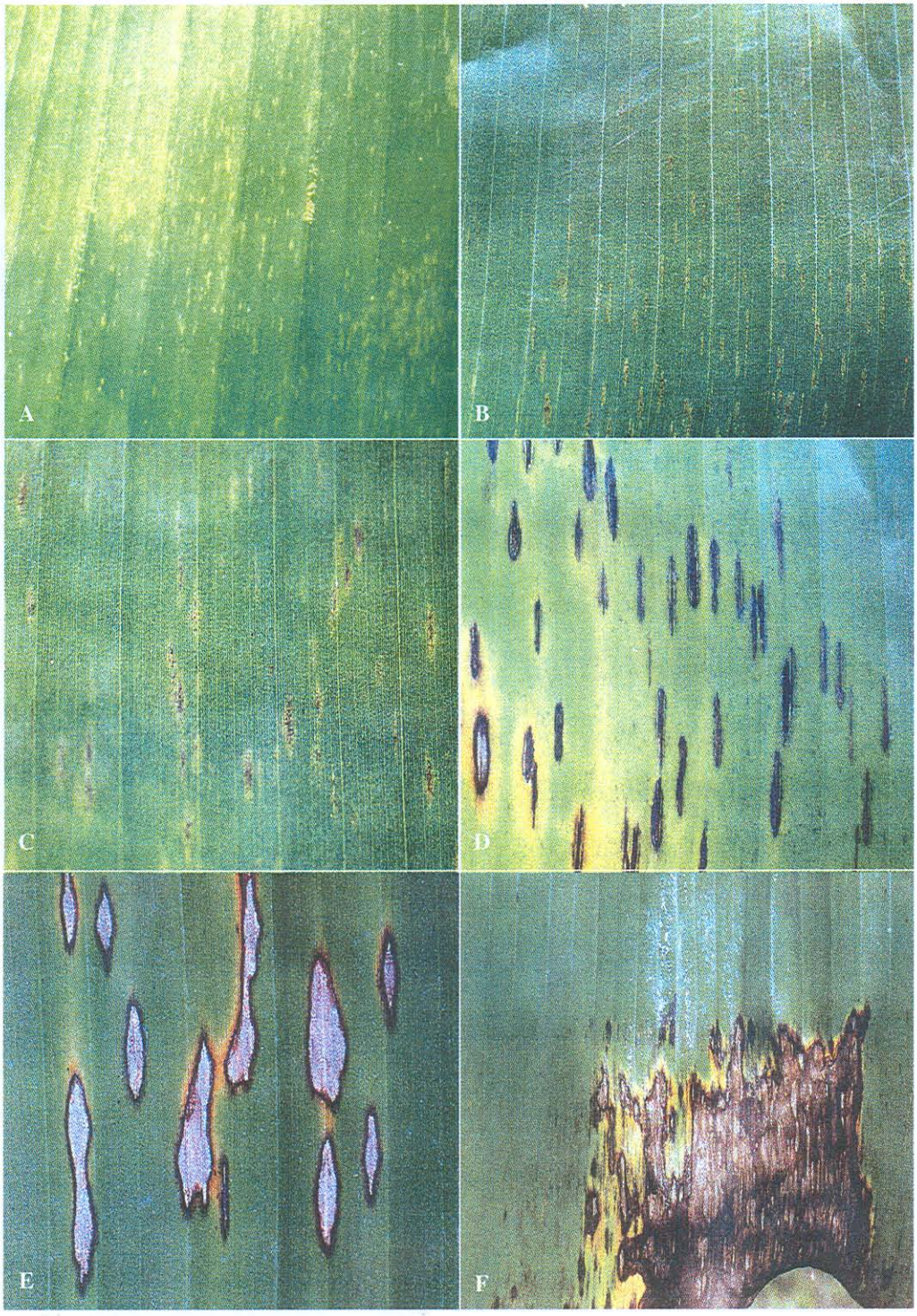

Figure 2: Damage caused by *Mycosphaerella musicola* to the banana plant. A. Stage 1. B. Stage 2. C. Stage 3. D. Stage 4. E. Stage 5. F. Severe leaf infection (photos courtesy of R.A. Peterson).

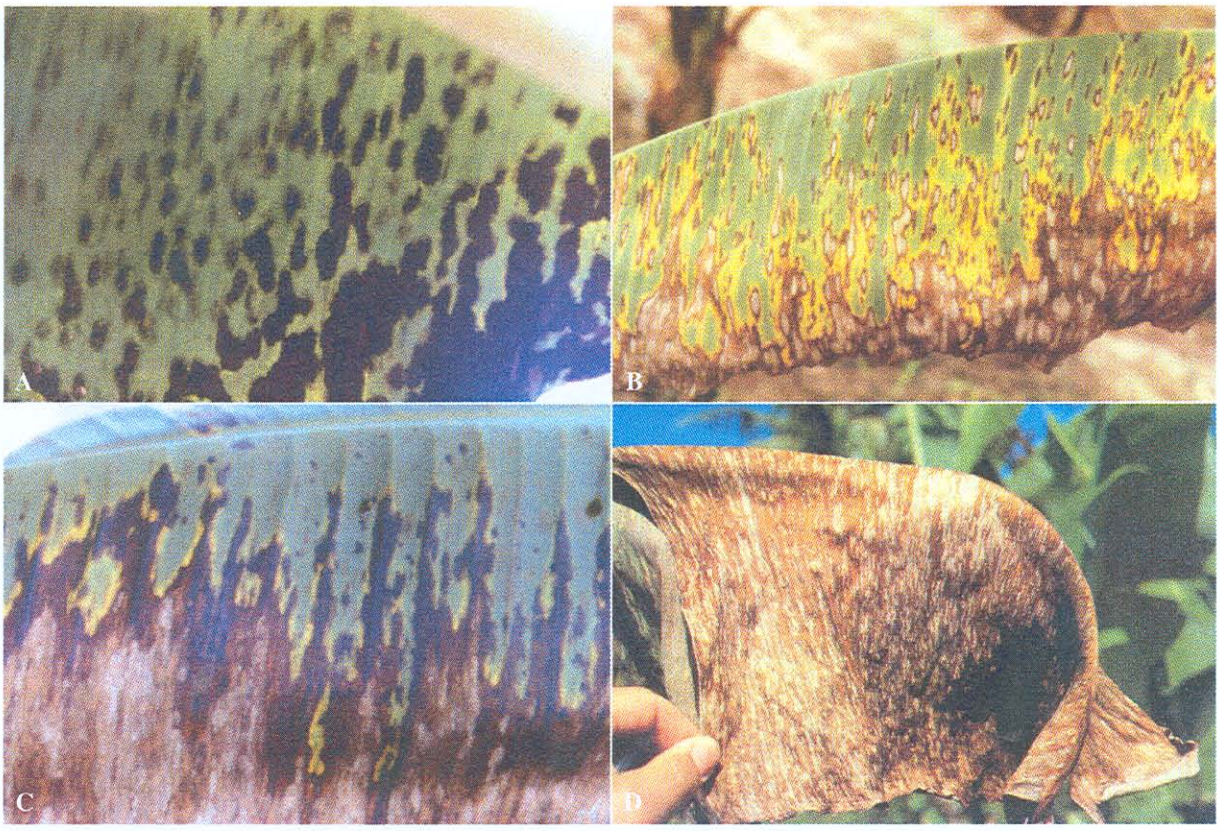

Figure 3: Black Sigatoka symptoms on banana leaves of the Cavendish subgroup. A. Lower surface symptoms between stages 4 and 5. B. Stage 6 symptoms on the upper leaf surface (plantain subgroup). C. and D. Stage 6 symptoms on the upper leaf surface. (Jones 2000).

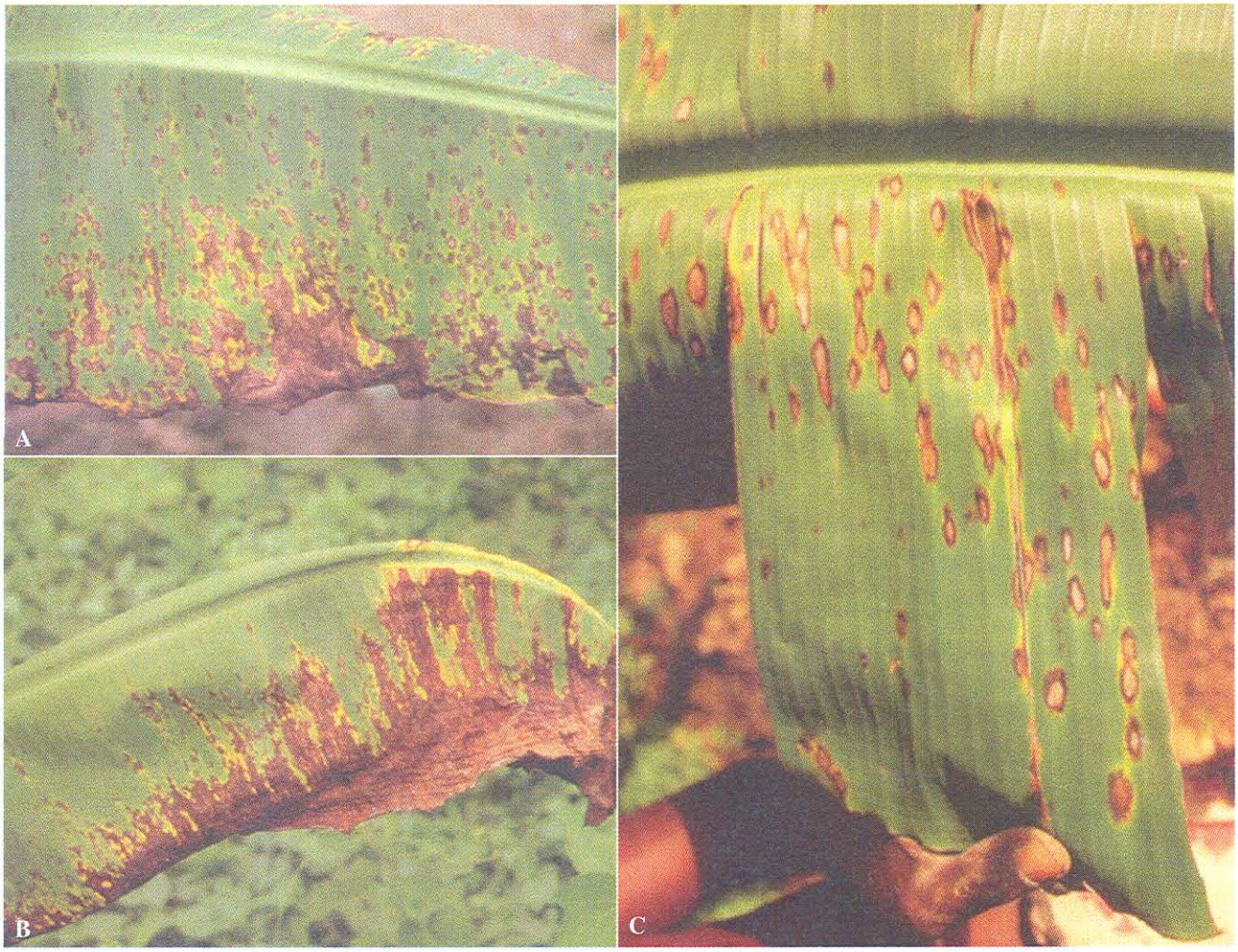

Figure 4: Mature lesions of eumusae leaf spot on A. Grande Naine (Thailand) B. Embul (Sri Lanka) and C. Anamala (Sri Lanka) cultivars (Jones 2000).

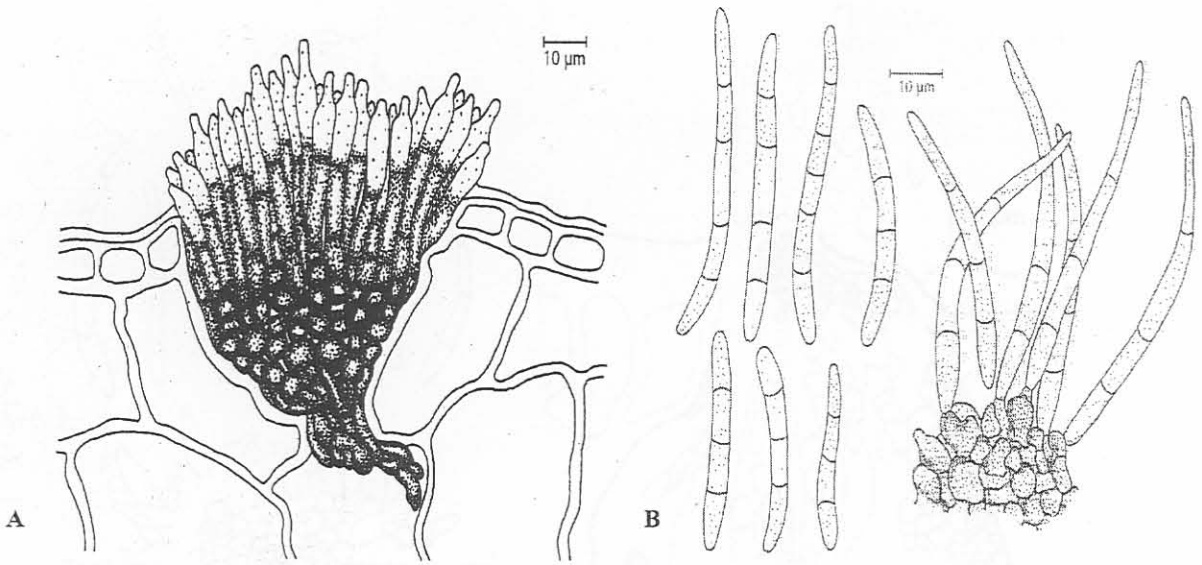

Figure 5: A. Sporodochium of *Mycosphaerella musicola* showing bottle-shaped conidiophores borne terminally on stromatal hyphae. B. Conidia and conidiophores of *M. musicola*. (Jones 2000)

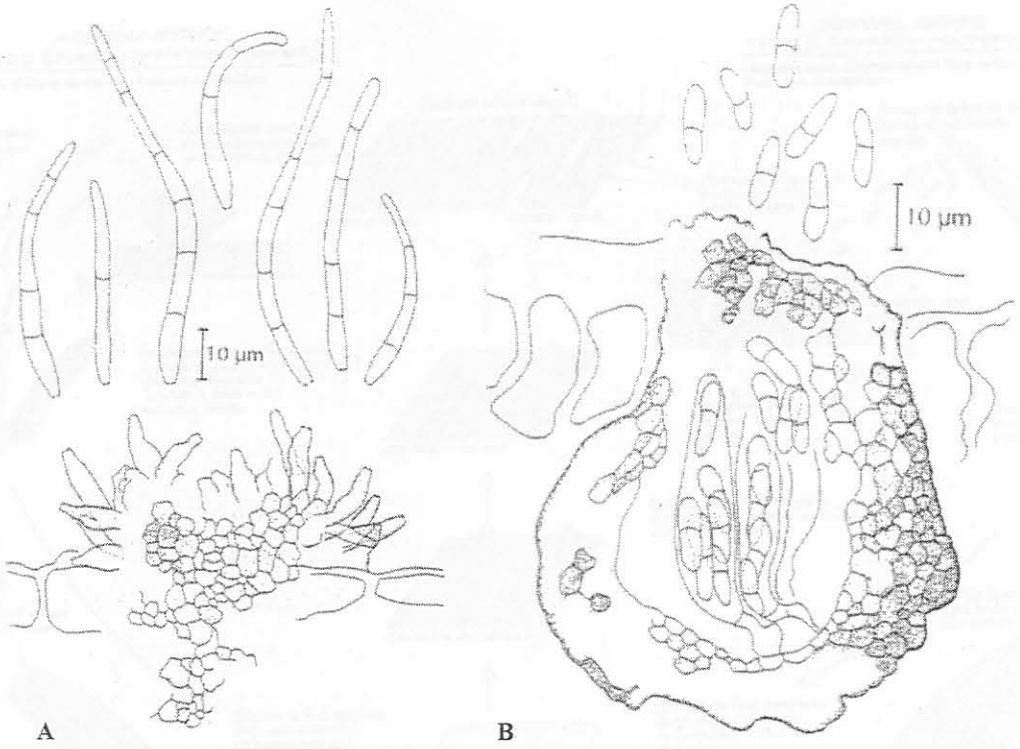

Figure 6: The asexual and sexual stages of *Mycosphaerella fijiensis* on banana leaves. A. Stroma with conidiogenous cells and conidia. B. Ascostroma with asci and ascospores (Jones 2000).

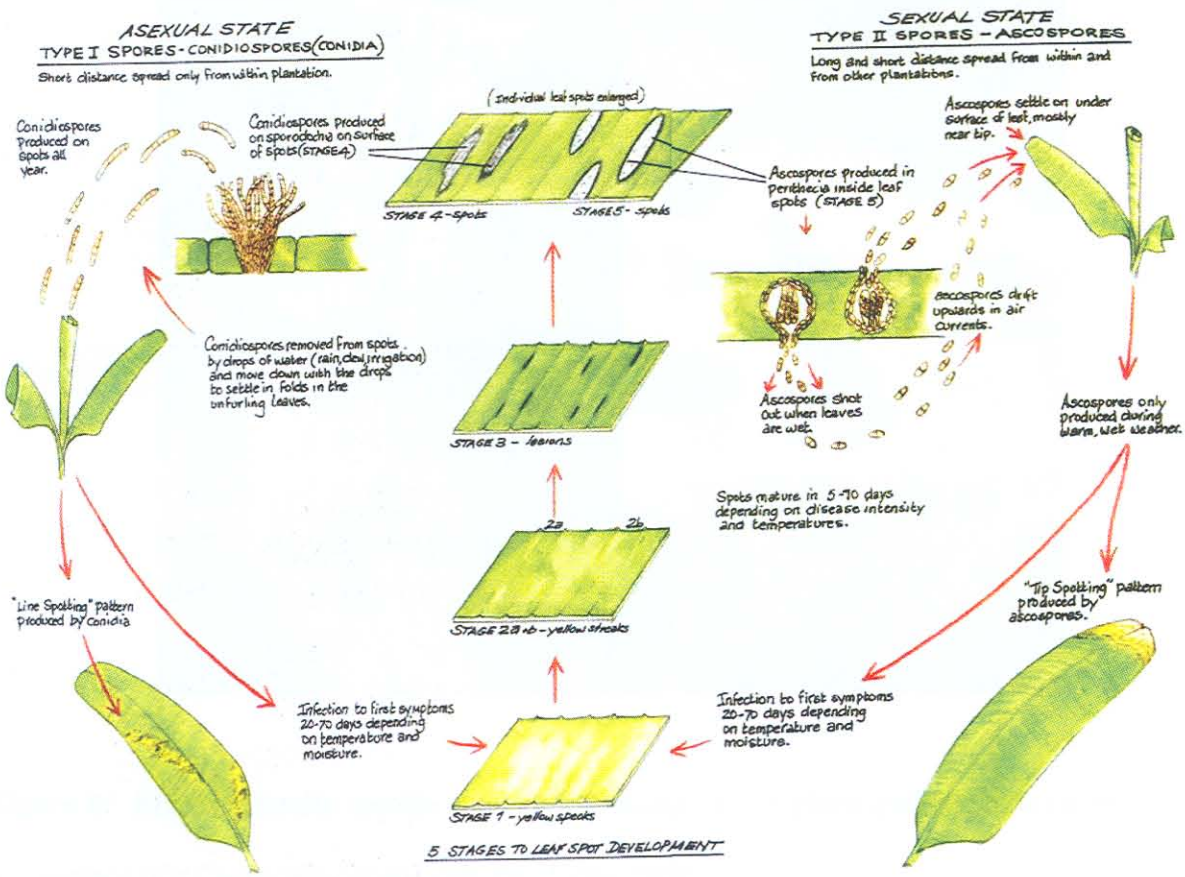

Figure 7: Life cycle of yellow Sigatoka (courtesy of R.A. Peterson)

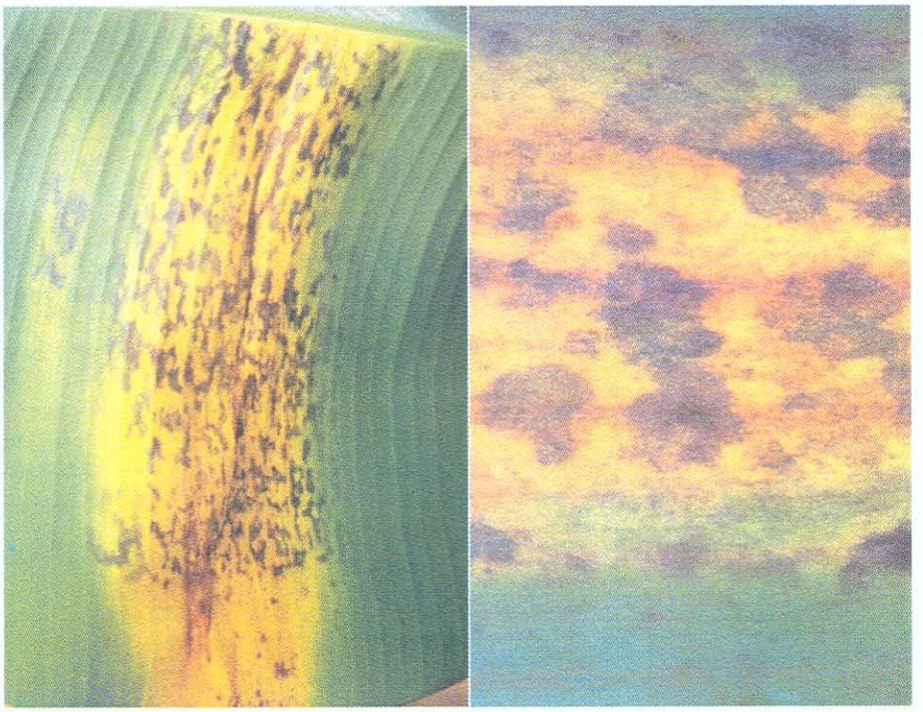

Figure 8: Mycosphaerella speckle causing increasing loss of photosynthetic area on the upper leaf surface of a Cavendish banana cultivar (Jones 2000).

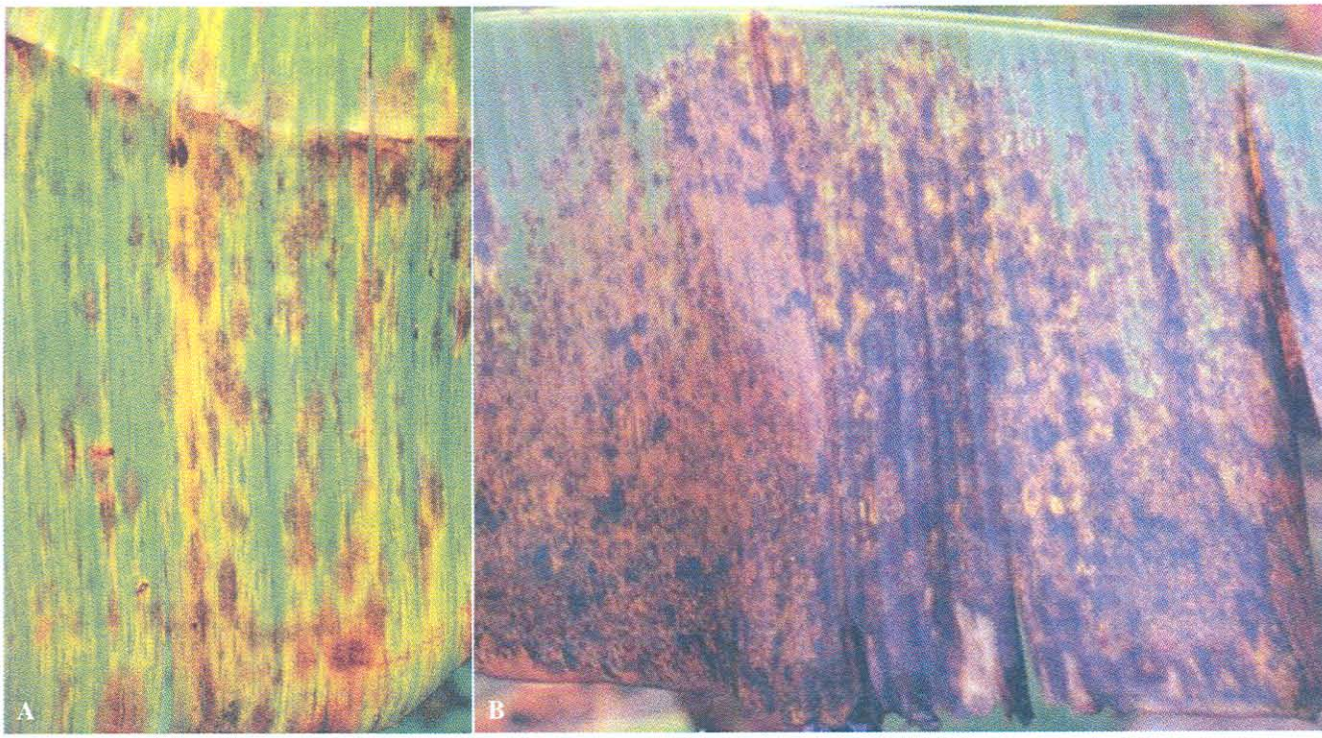

Figure 9: Symptoms of Cladosporium leaf speckle. A. Symptoms on a dessert banana cultivar. B. Leaf necrosis (Jones 2000).

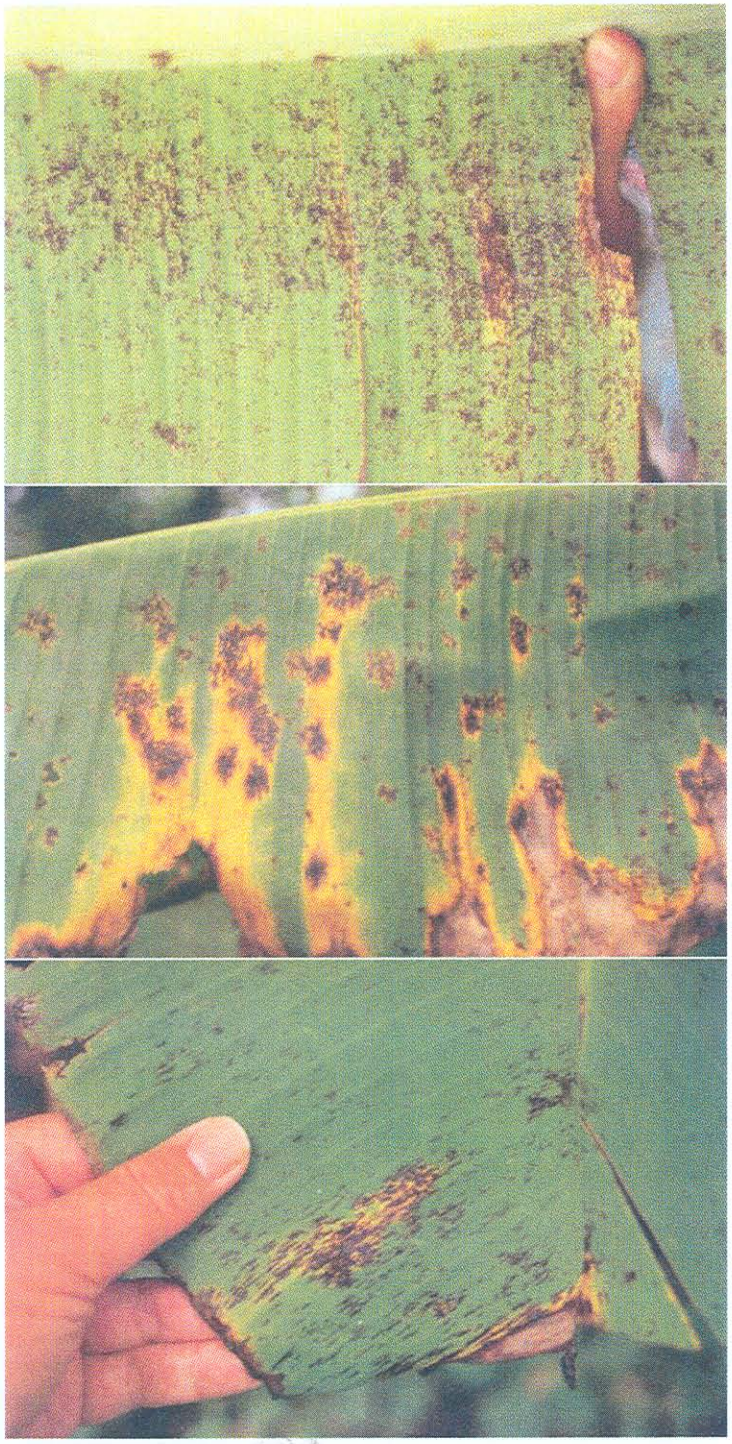

Figure 10: Various symptoms of leaf speckle (Jones 2000).

Figure 11: Symptoms of tropical speckle on the abaxial leaf surface A. Tan blotch speckle. B. Grey to black patch speckle (Jones 2000).

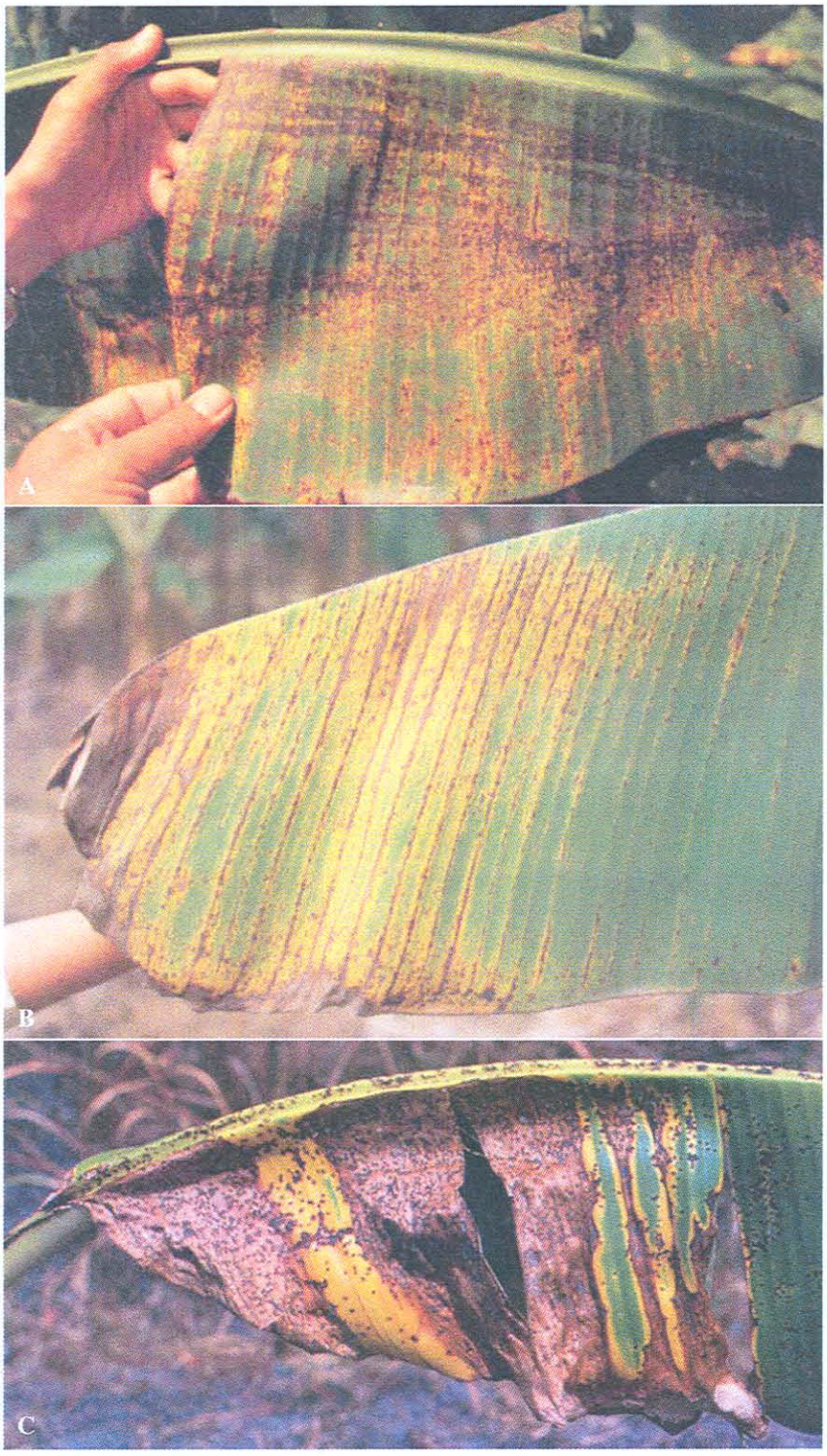

Figure 12: Symptoms of freckle A. Water droplet distribution of symptoms. B. Vein line association of symptoms. C. Larger spots also present on midrib of leaf (Jones 2000).

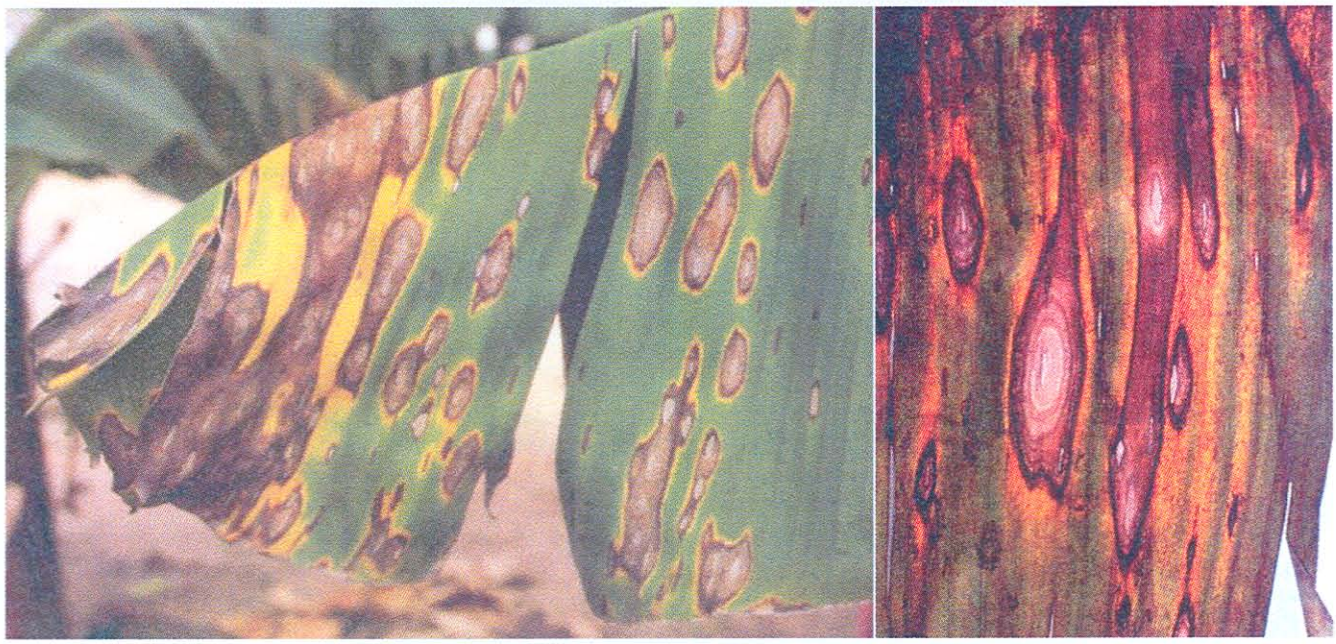

Figure 13: Symptoms of Cordana leaf spot on Cavendish cultivars (Jones 2000).

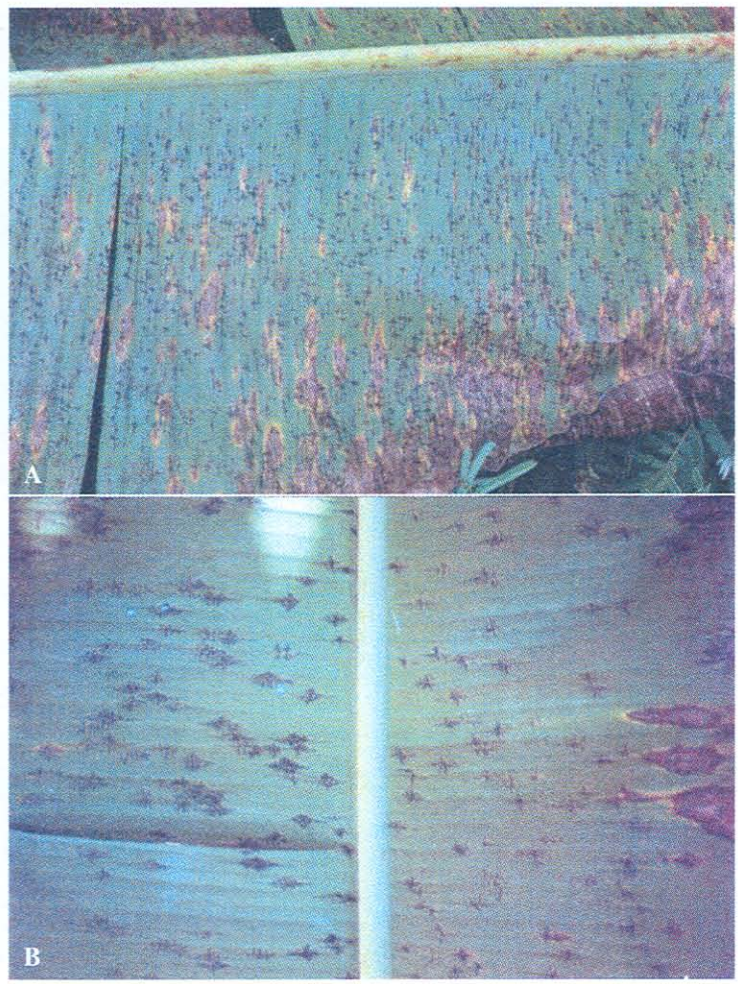

Figure 14: Symptoms of black cross leaf spot on A. Abaxial surface B. Adaxial surface of a banana leaf (Jones 2000).

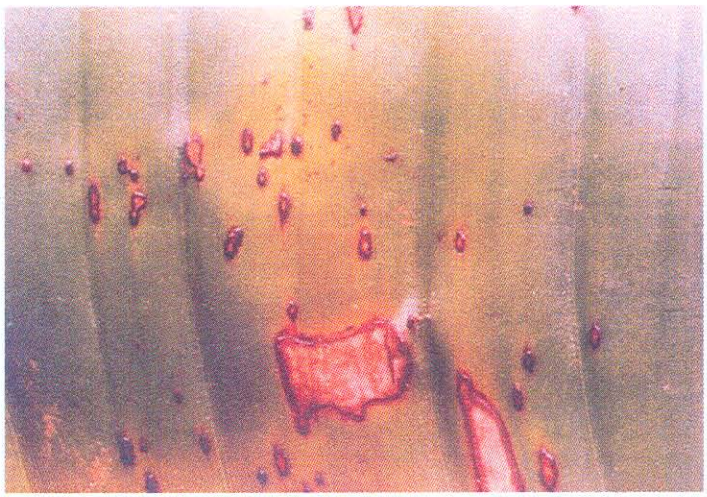

Figure 15: Symptoms of Deightoniella leaf spot on enset (Jones 2000).

Figure 16: Symptoms of Drechslera leaf spot on A. Banana B. Enset (Jones 2000).

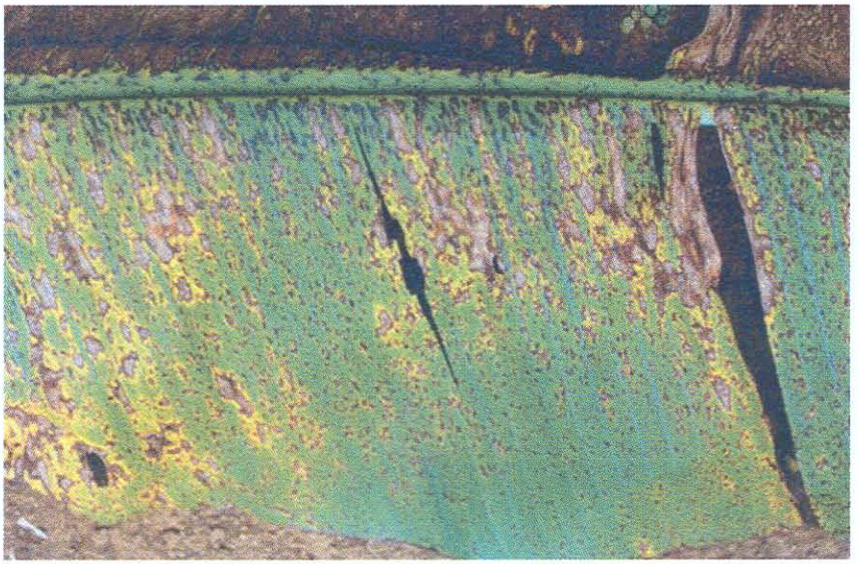

Figure 17: Symptoms of Malayan leaf spot on leaf and midrib (Jones 2000).

Figure 18: Symptoms of Pyricularia leaf spot A. Leaf midrib and lamina of enset. B. Petioles and leaf sheaths of enset (Jones 2000).

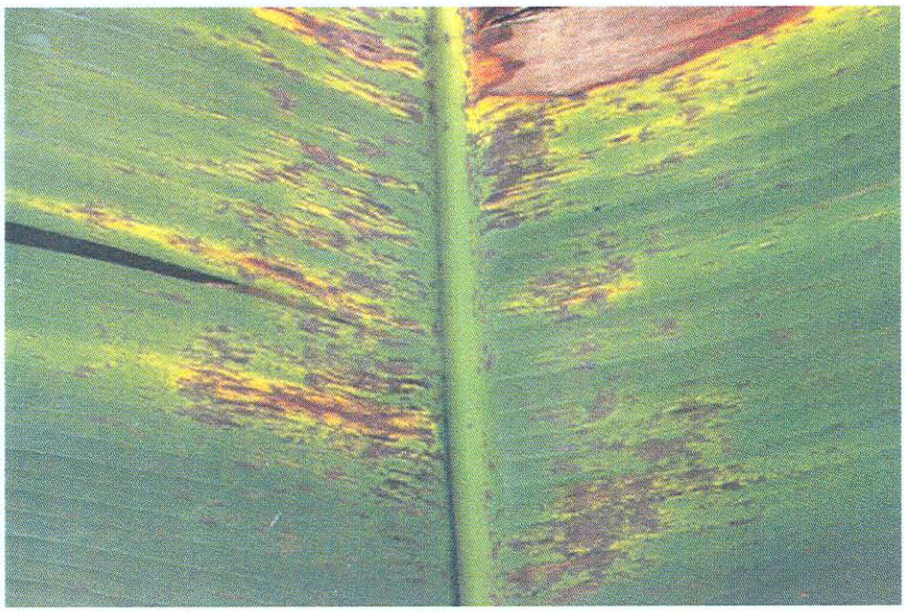

Figure 19: Symptoms of rust on a Williams banana cultivar (Jones 2000).