CHAPTER 26

Postharvest Diseases of Pineapple and Banana

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PINEAPPLE

BOTANICAL CLASSIFICATION AND CULTIVATION OF THE CROP

The pineapple [*Ananas comosus* (L.) Merril.)] is a xerophytic, perennial plant in the Bromeliaceae family (Bartholomew and Malezieux 1994). Botanically, the pineapple fruit is a compound multiple-fruit made up of 100 to 200 berry-like fruitlets that develop from the inflorescence. The fruitlets are fused together on a central axis or core arising on a single stalk from the center of a rosette of spiky leaves. The fruit has a conical shape with larger fruitlets at the base than at the top.

Pineapple plants are propagated vegetatively from crowns, slips, or shoots (suckers). Seeds are not preferred because of their slow growth, and many cultivars are seedless. Propagation using crowns, the upper part of the fruit with attached leaves, is most common. The crown is pulled or cut off from the fruit, and the lower leaves are removed to expose the 'root primordia' before planting. Planting suckers allows faster growth of new plants in comparison to the traditional method of planting crowns. Suckers develop between leaves of fully-grown pineapple plants and are pulled off by twisting at the base. Slips are small plants attached to the peduncle at the base of pineapple fruit but are not found in all cultivars. Pineapple can be grown as an annual crop or as a ratooned crop. In ratooning, the suckers are left on the plant to produce the next crop. Disadvantages include smaller fruit due to competition of food, light, and water by the crowded suckers, and an increased incidence of some diseases and pests.

A major limitation in pineapple production is unpredictable natural flowering that results in unscheduled fruiting. The incidence of natural flowering may vary from 0% to 100% in any given year (Kuan et al. 2005). This causes serious scheduling problems for harvest and fruit processing especially in fresh market production. The tropical pineapple is highly susceptible to frost and is commonly planted within 30°N and 30°S latitudes that include tropical and subtropical zones. Natural flower induction in subtropical areas mainly occurs in cooler winter months. The duration of natural induction in a pineapple field depends on cultivar, plant size, and prevailing environmental conditions, particularly temperature (Kuan et al. 2005). Natural flowering induction in Taiwan and other locations subjected to continental-climate cold fronts is assumed to be principally mediated by ethylene bursts in the shoot apical meristem due to lower night temperatures (Wang et al. 2007). Natural flowering of selected cultivars of pineapple can be prevented with aviglycine (ReTain®),

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A major limitation in pineapple production is unpredictable natural flowering that results in unscheduled fruiting. The incidence of natural flowering may vary from 0% to 100% in any given year (Kuan et al. 2005). This causes serious scheduling problems for harvest and fruit processing especially in fresh market production. The tropical pineapple is highly susceptible to frost and is commonly planted within 30°N and 30°S latitudes that include tropical and subtropical zones. Natural flower induction in subtropical areas mainly occurs in cooler winter months. The duration of natural induction in a pineapple field depends on cultivar, plant size, and prevailing environmental conditions, particularly temperature (Kuan et al. 2005). Natural flowering induction in Taiwan and other locations subjected to continental-climate cold fronts is assumed to be principally mediated by ethylene bursts in the shoot apical meristem due to lower night temperatures (Wang et al. 2007). Natural flowering of selected cultivars of pineapple can be prevented with aviglycine (ReTain®),

an inhibitor of 1-aminocyclopropane-1-carboxylic acid synthase activity and thereby ethylene biosynthesis (Kuan et al. 2005; Wang et al. 2007). Other growth regulators have also been recommended for this purpose (Da Cunha et al. 2003). Flowering can be achieved later, typically with ethylene, ethephon, and acetylene-releasing calcium carbide (Bartholomew et al. 2003). Thus, prevention of natural flowering and subsequent artificial forcing at convenience are very important practices in pineapple cultivation. Any effective forcing agent, however, should result in uniformity of the harvest peak and fruit yield to maintain a steady supply to the cannery and the fresh-fruit market. Furthermore, some diseases such as fruitlet core rot, leather pocket, and inter-fruitlet corking are initiated at flowering and thus, treatments for managing these diseases are critically timed at flowering or at forcing (*see* specific diseases below).

There are genetically diverse groups of pineapple, and the commercial varieties are classified into 'Cayenne', 'Queen', and 'Red Spanish' groups based on morphological characters (Grazia et al. 1980; Leal and Soule 1977). 'Smooth Cayenne' is the world's most commonly grown and largest commercial group for processing and fresh fruit trade because of its cylindrical shape, shallow eyes, yellow flesh color, mild acid taste, and high yields (Grazia et al. 1980). 'Cayenne' has spineless leaves. 'Queen' has spiny leaves and produces very sweet fruit with deep eyes. The 'Red Spanish' group has spiny leaves and produces medium-sized fruit with an acidic taste. Other pineapple groups are 'Abacaxi' and 'Maipure'.

Because the crop is propagated vegetatively, genetic redundancy is a major challenge in breeding as well as conservation and management of pineapple germplasm (Zhou et al. 2015). New approaches such as single nucleotide polymorphism markers for the characterization of pineapple germplasm have been developed and have demonstrated that modern pineapple cultivars are comprised of progenies that are derived from different wild *Ananas* varieties. Parentage analysis has also revealed that both *A. comosus* var. *bracteatus* and *A. comosus* var. *ananassoides* are likely progenitors of commercial pineapple cultivars, whereas the delineation of traditional horticultural groups (e.g., 'Cayenne', 'Spanish', 'Queen') was not supported using this molecular approach (Zhou et al. 2015). Furthermore, inter-simple sequence repeat and simple sequence repeat markers have indicated that the genetic diversity among pineapple accessions from many countries is very high (Wang et al. 2017). The result is that new breeding strategies can be developed, and new cultivars can be selected to improve horticultural characteristics such as color and tolerance to heat damage.

The pineapple is a non-climacteric fruit with moderate respiration and low ethylene production. When the shell reaches half-yellow color, the fruit is regarded as ripe. Pineapples should be harvested when they are ready to eat as only minor changes such as degreening of the shell, yellowing of the flesh, and decline in acidity occur after harvest (Paull and Chen 2003). There is no dramatic respiratory change in pineapple or pronounced ethylene production peak during ripening; however, ethylene has a major role in ripening of climacteric fruits. Pineapples can be stored at 7 to 12°C for 14 to 20 days provided fruit are at color-break stage. Ripe fruit can be held at 7.2°C for about 7 to 10 days, and it is during this period that many of the postharvest diseases develop.

IMPORTANCE OF THE CROP

■ World production of pineapple reached 28 million tons (Mt) in 2017 (Anonymous 2017). About 70% of the harvest is consumed as fresh fruit, and there is a growing demand for juice as a beverage. The top producing countries are: Costa Rica (3.1 Mt), the Philippines (2.7 Mt), Brazil (2.3 Mt), China (2.1 Mt), Thailand (2.1 Mt), India (1.8 Mt), Indonesia (1.8 Mt), Nigeria (1.6 Mt), and Colombia (3.1 Mt). After removing the crown, rind, eyes, and core, the flesh is consumed. The pineapple is a popular fruit, largely because of its attractive flavor with a refreshing sugar-acid balance. The mature fruit is a good source of carbohydrates (Sinclair 1993).

The proteolytic enzyme bromelain obtained from the fruit (Yamada et al. 1976) or mature plant stem (Nakasone and Paull 1998) is used for tenderizing meat, chill-proofing beer, increasing the solubility of gelatin, stabilizing latex paints, and in the leather-tanning process. In medicine, bromelain is used as a digestive and as an anti-inflammatory after surgery (Nakasone and Paull 1998).

POSTHARVEST DISEASES

■ Harvested pineapples are affected by several economically important diseases caused by fungal and bacterial pathogens. The symptoms of post-harvest diseases of pineapple fruit may develop externally or internally. Infections of fruit leading to postharvest decays or disorders can be classified as follows: 1) pre-flower infections such as by *Penicillium funiculosum* that start at the floret before the flower opens and later cause inter-fruitlet

corking, leathery pocket, and fruitlet core rot of the mature fruit; 2) flower infections after the flower opens such as those by bacteria that later cause pink and marbling disease; 3) wound infections of fruit during harvesting and handling that are caused most frequently by *Thielaviopsis paradoxa*; and 4) physiological disorders such as internal browning or black heart caused by chilling injury (Rohrbach and Phillips 1990).

Black Rot (Thielaviopsis Fruit Rot)

Level of Importance

Black rot of pineapples, also known as Thielaviopsis fruit rot, water blister, soft rot, water rot, and stem-end rot, is the most important and widespread postharvest disease of harvested pineapple. The disease occurs in all major pineapple-producing countries, including India, the Philippines, Malaysia, Australia, Hawaii, Cuba, Puerto Rico, Nigeria, the Ivory Coast, South Africa (Snowdon 1990), and Sri Lanka (Abeygunawardena 1969; Damunupola and Adikaram 2000).

Symptoms

Initially, black rot is not externally apparent. The disease starts as a soft, watery rot of the flesh (Fig. 26.1) with an overlying glassy, water-soaked, brittle skin (Sinclair 1993). The advancing area of decay is pale, and water-soaked. Eventually, the skin, flesh, and core disintegrate, and the juice leaks through the shell. Diseased tissues darken due to the presence of the dark fungal mycelium and chlamydospores. In advanced stages, the rot leaves a fruit shell containing only a few black fibers, and the shell collapses under the slightest pressure (Pegg et al. 1995; Rohrbach and Phillips 1990). Black rot does not occur in the field unless the fruit is overripe or injured. The disease can also start at the detached stem-end (Fig. 26.2A) (Kumar 2007) and can readily infect bruised or other damaged areas of the fruit. The pathogen occasionally also infects the upper portion of the pineapple crown where it produces a black, charcoal-like rot (Fig. 26.2B). Base rot and butt rot of the pineapple plant are caused by the same fungus.

FIG. 26.2. **A,** Stem-end rot and **B,** crown bud rot caused by the black rot pathogen, *Thielaviopsis paradoxa*. (Courtesy N. K. B. Adikaram—© APS)

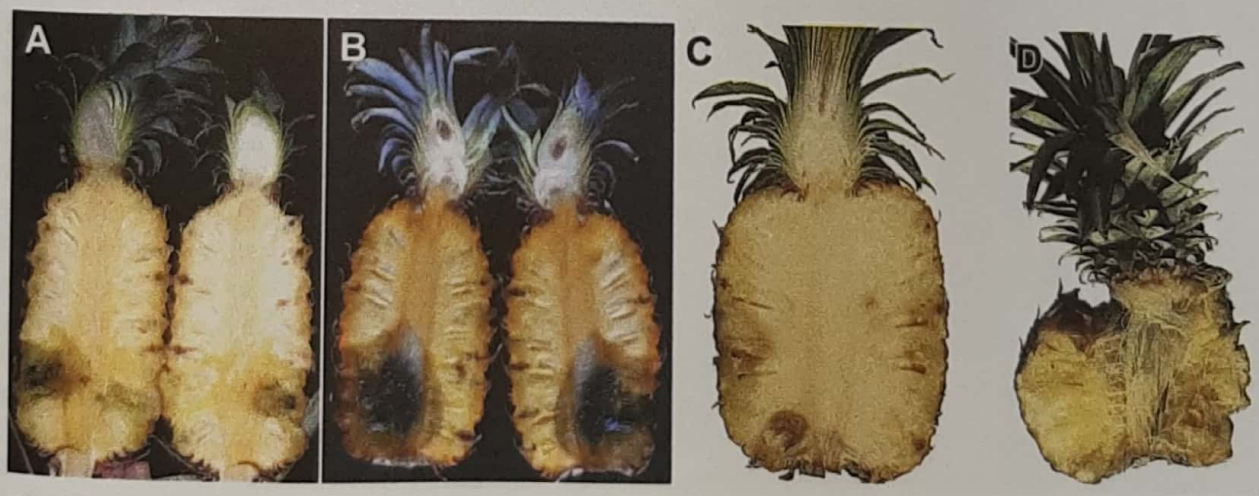

FIG. 26.1. Black rot is caused by *Thielaviopsis paradoxa*. Cut-open ripe pineapple fruit showing **A,** soft, watery black rot of the flesh. **B,** At advanced stages, the infection turns black due to fungal growth. **C,** Early water blister symptoms (initial lesion of black rot); and **D,** late symptoms of water blister (advanced lesion of black rot). (A and B, Courtesy N. K. B. Adikaram—© APS; C and D, Courtesy UCANR—Reproduced by permission)

Causal Organisms

Black rot is caused by *Thielaviopsis paradoxa* [syn. *Chalara paradoxa* (anamorph), *Ceratocystis paradoxa* (teleomorph)]. Taxonomic studies suggest that this species is far less common, has a more restricted host range than previously considered, and that isolates from pineapple should be assigned to a different species, *T. ethacetica* (Mbenoun et al. 2014). The fungus is common in pineapple fields and also causes butt (base) rot and white leaf spot of pineapple. Asexual reproduction is by hyaline endoconidia produced in phialides (i.e., endoconidiophores), and chlamydospores are formed as survival structures (Fig. 26.3).

Disease Cycle and Epidemiology

The pathogen penetrates the host through wounds and bruises that occur on the shell during harvest or through natural growth cracks (Pegg et al. 1995). The cut stalks can be a main point of entry, but any bruised region of the fruit can be invaded. Infection occurs within 8 to 12 h following wounding. There is evidence that the pathogen infects fruit in the field and remains quiescent. Infection can also occur on the harvested fruit. In both situations, symptoms of black rot develop several days after harvest during storage or marketing. Therefore, the grower seldom sees the disease. Fresh fruit are mostly marketed with the crowns intact, which eliminates a major entry point for the fungus (Pegg et al. 1995).

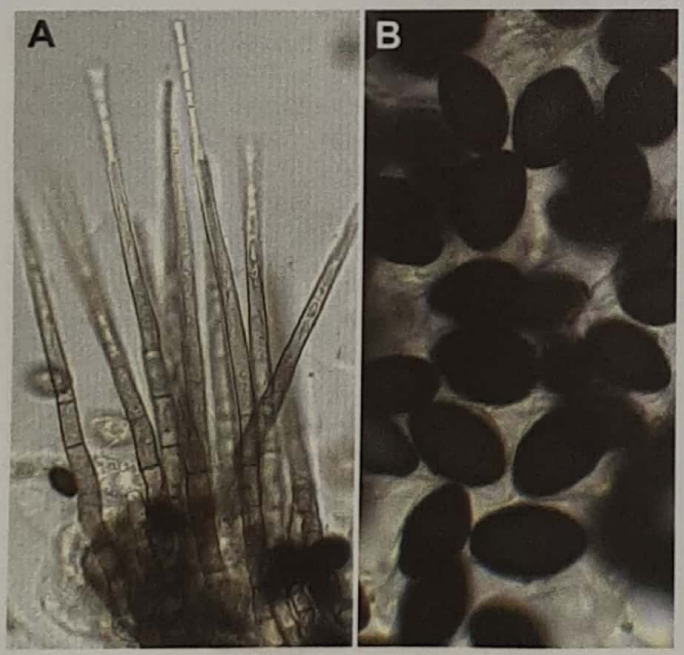

FIG. 26.3. *Thielaviopsis paradoxa*. **A,** Endoconidiophores (pigmented elongated cells) and endoconidia (hylaine rectangular cells at top of endocondiophores); and **B,** elliptical, blackish chlamydospores. (Courtesy N. K. B. Adikaram—© APS)

Critical Biological and Environmental Factors

Factors that favor disease development include the amount of bruising and wounding during harvesting and packing, the level of inoculum on the fruit, and temperature during transportation and marketing (Rohrbach and Schmitt 1994). Black rot is most common and severe during warm, wet weather (Pegg 1993) causing serious losses in the fresh fruit industry (Nakasone and Paull 1998; Snowdon 1990). This is because growth of the fungus is rapid at temperatures between 21 to 32°C (Petty et al. 2006). Rainfall prior to harvest is also very favorable for postharvest disease development.

Management

Fruit handling and sanitation practices

Fruit should be carefully handled to minimize mechanical damage during harvest and handling (Nakasone and Paull 1998). Sunburnt and damaged fruit should be rejected at the packinghouse or removed during sorting. Removing pineapple culls and rejected fruit from packing areas and the market is important to reduce inoculum (Pegg et al. 1995). Refrigeration also decreases the incidence of black rot. Storage of harvested fruit at 7°C inhibits growth of the fungus.

Chemical treatments

The most effective postharvest fungicides for controlling black rot are systemic methyl benzimidazole compounds (Fungicide Resistance Action Committee – FRAC Code 1). Other fungicides available in some countries include the demethylation inhibitor triadimefon (FRAC Code 3), the phenylpyrrole fludioxonil (FRAC Code 12), the quinone outside inhibitor azoxystrobin (FRAC Code 11), and the macrolide natamycin (FRAC Code 48) (Adaskaveg *unpublished*). Fruit should be treated with a fungicide within 6 h after harvest for the most effective control, and the application can be done by dipping, high-volume sprays, or drenches to protect injuries including the harvesting cut of the peduncle. When fruit are harvested during warm, wet weather, the cut stem end should be dipped in a fungicide within 4 to 5 h (Pegg 1993). Complete inhibition of conidial germination and mycelial growth of *T. paradoxa* has been observed in 2 to 3% acetic acid. Black rot of Mauritius (Queen) pineapple was minimal when fruit were dipped for three minutes in 4% or 5% acetic acid and stored for 7 days at 28 ± 2°C (Wijeratnam et al. 2006).

Physical

Hot water treatment at 54°C for 3 min controls the disease of fruit stored at 10°C for 21 days followed by 28 ± 2°C (ambient temperature) for 48 h or stored at 28 ± 2°C for 6 days (Wijeratnam et al. 2005). Treatment with a fruit coating after harvest has also been effective in reducing the disease (Wijeratnam et al. 2006).

Biological

The use of microbial antagonists as agents of biological control, such as *Pichia guilliermondii* or a yeast mixture, has proved to be effective. Control was comparable to the current industry practice of holding fruit at low temperature (8 to 10°C) and treatment with a fungicide (Reyes et al. 2004). Fruit susceptibility varies with cultivar; 'Red Spanish' types are more resistant than 'Smooth Cayenne' (Nakasone and Paull 1998; Rohrbach and Schmitt 1994).

Fruitlet Core Rot, Leathery Pocket, and Inter-Fruitlet Corking

Level of Importance

These diseases are widespread in all major pineapple-producing countries of the world (Snowdon 1990). However, their appearance is sporadic (Pegg et al. 1995), and they rarely occur at epidemic levels. Low-acid cultivars and rough-leaf pineapples grown commercially are more susceptible than 'Smooth Cayenne' (Pegg et al. 1995; Rohrbach and Schmitt 1994). The diseases are more common on fruit maturing during the winter or spring. Inter-fruitlet corking is limited almost exclusively to fruit developing through early autumn.

Symptoms

These three diseases vary slightly from each other; they are all internal fruit decays and are incited by the same pathogens. Fruitlet core rot, black spot, fruitlet brown rot, and eye rot are terms that have been used to describe the brown to black diseased center of individual pineapple fruitlets. Leathery pocket and inter-fruitlet corking are additional symptoms that appear as fruitlet core rot continues to develop (Fig. 26.4).

Fruitlet core rot (FCR)

'Smooth Cayenne' fruit do not show any external symptoms, and the disease is undetectable unless the interior is examined. Severely affected fruitlets may become brown and sunken as the fruit undergoes ripening. Internal symptoms consist of browning of the center of the fruitlets starting below the floral cavity and sometimes extending to the core (Fig. 26.4). The browning remains quite firm and ranges from a small speck to complete discoloration of one or more fruitlets

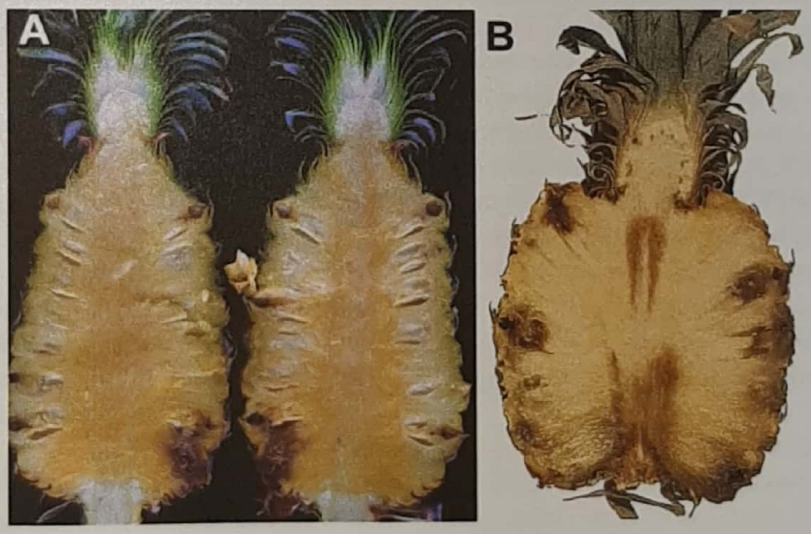

FIG. 26.4. Interior of a ripe pineapple fruit showing symptoms of leathery pocket and inter-fruitlet corking caused by *Penicillium funiculosum*, *Fusarium guttiforme*, or a mixture of both fungi. **A,** A single fruitlet affected; and **B,** several fruitlets affected. (A, Courtesy N. K. B. Adikaram—© APS; B, Courtesy UCANR—Reproduced by permission)

(Cooke et al. 2009). Fruitlets of the 'Queen' pineapple group may fail to color, a condition often referred to as 'green eye'.

Inter-fruitlet corking (IFC)

Early during development, fruit often show shiny patches on the shell where the trichomes were removed by mite feeding. Externally, corky tissue develops on the skin between the fruitlets, but usually only patches of eyes are affected. Diseased fruitlets do not enlarge as rapidly as healthy ones, resulting in distortion (Hepton and Anderson 1968). In moderate to severe cases, corkiness surrounding fruitlets prevents their development, and one side of the fruit will be malformed (Cooke et al. 2009). Fine transverse cracks may also develop on the sepals and bracts.

Leathery pocket (LP)

Fruit do not show any external symptoms. Internally, the formation of corky tissue on the fruitlet walls makes them leathery and brown.

Causal Organisms

The fungi *Penicillium funiculosum*, *Fusarium guttiforme*, and *F. subglutinans;* the yeast *Blastodendrion arztii* (syn. *Candida guilliermondii*); the pineapple fruit mite *Steneotarsoneumus ananas* (Tryon); and the pineapple red mite *Dolichotetranychus floridanus* (Banks) are associated with FCR (O'Donnell et al. 1998; Rohrbach and Schmitt 1994). *P. funiculosum* and the *Fusarium* spp. are common soil-borne fungi and cause flower infections, but the involvement of the yeast *B. arztii* is not clear. The importance of *P. funiculosum*, *F. guttiforme*, and *F. subglutinans* varies among production areas. In Brazil, *F. guttiforme* is the predominant cause of FCR, whereas *P. funiculosum* is the most common cause of FCR and LP in South Africa (Rohrbach 1980; Rohrbach and Taniguchi 1984). In Hawaii, both *P. funiculosum* and *F. guttiforme* cause FCR symptoms, while only *P. funiculosum* causes IFC and LP (Rohrbach and Schmitt 2003). *P. funiculosum* was consistently isolated from pineapple fruitlets with black spot symptoms.

The pineapple fruit mite *S. ananas* appears to enhance the virulence of *P. funiculosum* but does not act as a vector (Rohrbach and Apt 1986). *S. ananas* is light brown, and the adult male is oval with an average length of 0.2 mm and width of 0.1 mm (Petty 1975, 1978). *D. floridanus* is a large phytophagous mite found on pineapple. It is conspicuous because of its bright orange to red color. The adult mite is 0.3 to 0.4 mm long and 0.1 mm wide (Petty 1975).

P. funiculosum builds up on mite-damaged trichomes on the basal parts of heart leaves during the weeks before anthesis (Pegg et al. 1995). On closed flowers, *P. funiculosum* initially causes necrosis of the anthers and pistil, as well as cork formation on the locules. Blue-green sporulation is present on ovules and locule walls. As the disease progresses, septa between locules become dark to medium brown, and the discoloration may extend into adjacent non-capillary tissues (Rohrbach and Schmitt 2003). Further corking of locules during fruit maturing (Fig. 26.4) results in 'leathery pocket' (Rohrbach and Schmitt 1994). LP disease was formerly attributed to mite damage, but it has been established that *P. funiculosum* is the primary cause (Lim and Rohrbach 1980). With IFC, *F. guttiforme* causes a light to dark brown discoloration of septa that may extend down the entire fruitlet core. White to pinkish mycelium and sporulation of the pathogen occur in locules.

Critical Biological and Environmental Factors That Favor Disease

A series of factors may modulate symptom development. These include time of infection, level of inoculum present, cultivar, and environmental conditions (Rohrbach and Schmitt 1994). A virulent strain of *P. funiculosum* must be present one week before forcing the plant to flower in order to colonize the mite-injured trichomes and produce sufficient inoculum to invade the closed flower. The optimum temperature for infection is 16 to 21°C during the 6 weeks following forcing. Moisture does not appear to be critical during this period. The average ascorbic acid content in fruit at harvest is negatively linked to the incidence of fruit affected by *P. funiculosum*. The risk of disease caused by this fungus is higher when flowers are initiated, and fruit mature under warmer conditions (21 to 27°C).

Fruitlet core rot

The nutritional status of the plants, especially low levels of calcium and magnesium and/or high levels of nitrogen, as well as high-humidity conditions before harvest, are critical factors that favor the development of the disease (Marie et al. 2000).

Infection by *P. funiculosum* is optimal between 16 and 20°C and is inhibited at higher than 20°C. Rainfall is needed for the fungus to build up on damaged leaf trichomes, but not for infection (Rohrbach and Taniguchi 1984). *F. guttiforme* enters the fruit through open flowers or injuries. The risk of fruitlet core rot due to *F. guttiforme* is higher when flowers are initiated and fruit mature under warmer conditions (21 to 27°C) (Pegg et al. 1995).

Management

Fungicides have not been effective for FCR, LP, and IFC induced by *P. funiculosum*, except when applied directly into the opening of the terminal leaves that is created by the emerging inflorescence. No control measures for FCR caused by *F. guttiforme* have been developed (Rohrbach and Schmitt 2003). Acaricide (e.g., endosulfan) sprays help to reduce the disease. Integrated experimental strategies, however, that included control of the pink pineapple mealy bug *D. brevipes*, the pineapple fruit mite *S. ananas*, as well as fungicide (e.g., benomyl and mancozeb) applications from one week before to eleven weeks after flower induction, reduced the disease and allowed storage of fruit for fourteen days at ambient temperature (Petty et al. 2006). The sporadic nature of this disease makes chemical control impractical and uneconomical in Queensland, Australia (Pegg et al. 1995).

Green Fruit Rot

Level of Importance

This fruit disease is not very common and is considered of minor importance. Still, Phytophthora heart (top) rot, crown rot, and root rot, which are caused by the same pathogens, are potentially major diseases of pineapple that cause significant losses. If these latter disease phases are not managed, green fruit rot can become a problem.

Symptoms

Initially, a water-soaked rot develops behind affected fruitlets with no external symptoms present (Cooke et al. 2009). There is a risk that infected fruit with no visible symptoms are harvested and sent to the market where breakdown will become evident. Therefore, when conditions favor development of the disease, samples of fresh market fruit should be cut open and examined carefully for internal rotting. As the disease progresses, a water-soaked rot with a distinct brown margin develops internally behind affected fruitlets (Joy and Sindhu 2016; Pegg et al. 1995).

Causal Organisms and Epidemiology

Green fruit rot is caused by *Phytophthora cinnamomi*. The pathogen is soil-borne and requires water for zoospore production and infection. Rain splash carrying zoospores or sporangia of the pathogen contaminates fruit. Serious losses from green fruit rot generally follow heavy rains when Phytophthora root rot has caused plants to lodge (Pegg et al. 1995). Additionally, the incidence of green fruit rot is higher on fruit close to or touching the ground especially of a ratoon crop that generally has higher inoculum levels because the pathogen increases from the previous crop. Heart rot can be caused by *P. nicotianae* (Joy and Sindhu 2016) and by *P. cinnamomi*.

Management

Horticultural practices

Plants should not be planted too deep in the soil, and soil should not be allowed to enter the hearts (tops) during planting to reduce Phytophthora crown rot. Well-drained soils are essential for minimizing the risk of Phytophthora infection of crowns, roots, and fruit. Integrated practices such as careful field selection, planting on raised beds at least 20 cm high, constructing drains to intercept run-off before it reaches the plantation, constructing drains within the field so that water is removed rapidly without causing erosion, and installing underground drains all contribute to reduce the incidence of disease (Joy and Sindhu 2016).

P. cinnamomi becomes more active as soil pH levels increase above 4.0, and liming amendments that increase pH should be used cautiously. Sulfur may be used to reduce pH in soils with a pH above 5.5, but this is not a replacement for other management practices (Joy and Sindhu 2016).

Chemical

Green fruit rot can be effectively managed by application of fungicides including phosphonates and mefenoxam. These fungicides are also used to control root and heart rot of pineapple. Pineapple plant material should be treated before planting, and the soil should be drenched or sprayed with fungicides after planting to reduce the incidence of Phytophthora crown and root rot that increase inoculum and subsequently the incidence of green fruit rot.

Marbling

Level of Importance

The disease occurs in all pineapple-growing countries, but it is serious only in lowland tropical production areas where temperatures remain above 21°C (Rohrbach and Schmitt 1994). In Thailand, 5 to 20% of the slices in canneries are marbled, and a high incidence of disease in October and November can even stop canning operations. In Hawaii, the highest levels of marbling occur in April and May (Rohrbach and Schmitt 1994).

Symptoms

The most common symptom is a yellowish to reddish brown to very dark, dull brown discoloration of internal fruit tissue (Fig. 26.5). Infected tissues generally become hardened, granular, and brittle with a woody consistency, and are speckled with discolorations. Affected fruit do not show external symptoms, however, severely infected fruit may be identified by a 'woody' sound when tapped (Rohrbach and Schmitt 1994). The disease may affect multiple fruitlets or the entire fruit, but occasionally only single fruitlets are involved. Frequently, the speckled appearance will occur in vascular tissue to the core of the fruit. Symptoms develop during the last month of fruit maturation (Rohrbach and Schmitt 1994).

Causal Organisms

Strains of the bacteria *Acetobacter pasteurianus* (syn. *Acetobacter peroxydans*) (Gosselé et al. 1983) and other *Acetobacter* species, as well as *Pantoea ananatis* (syn. *P. ananas*, *Erwinia herbicola* var. *ananas*) (Mergaert et al. 1993) are the causative organisms of this disease (Pegg et al. 1995).

Disease Cycle and Epidemiology

Infection usually occurs through open flowers but may also occur through growth cracks in the fruit surface during the later stages of fruit development. Bacteria may be vectored to the flowers by insects. The bacteria remain quiescent in the flower and developing fruit until approximately one month before fruit maturity. Low fruit acid and sugar contents are associated with high levels of the disease (Rohrbach and Schmitt 1994).

FIG. 26.5. Typical granular, darkening symptoms of marbling disease of pineapple caused by *Acetobacter* species and *Pantoea ananatis*. (Courtesy K. G. Rohrbach—© APS. Reproduced, by permission, from Ploetz et al., 1994.)

Critical Biological and Environmental Factors That Favor Disease

Marbling disease is similar to pink disease, but infected fruit tissues have a brown granular appearance without heating during the canning process. In contrast to pink disease, marbling occurs when fruit are developing at warmer conditions (>21 to 27°C). Moisture does not appear to be critical for infection, but the disease is enhanced by rainfall during flowering and when fruit mature during dry, hot conditions and rainfall occurs during the last 6 to 8 weeks before harvest (Rohrbach 1989). Application of surfactants prior to and during flowering significantly increases the disease in Hawaii, indicating that the bacteria are ubiquitous on the plant surface.

Management

There are no control measures known for marbling. Affected fruit with clearly visible symptoms should be eliminated during processing, reducing processing costs (Pegg et al. 1995). Infected fruit can be detected by examining the external appearance and by testing fruit firmness. Differences in cultivar susceptibility have been noted; 'Smooth Cayenne' is moderately resistant (Rohrbach and Schmitt 1994).

Pink Disease

Level of Importance

Pink disease is primarily a problem in canned pineapple products and is of little importance in fresh fruit. The disease occurs only sporadically when fruit develop under cool, wet conditions. It therefore occurs mainly in the springtime in certain production areas. The bacterial pathogens are killed at high temperatures.

The disease has been described from Hawaii, the Philippines, Australia, and Mexico (Snowdon 1990). Outbreaks in Australia are very infrequent and local, often affecting only one or two flushes of fruit in a few fields. Occasional economically significant epidemics have been reported in Hawaii and Taiwan in the months of February, March, and April and in the Philippines from August to September (Hine 1976; Rohrbach and Schmitt 1994).

Symptoms

Pink disease refers to the pinkish discoloration of affected fruit flesh (Fig. 26.6). Most often, affected fruit do not show any external symptoms even when fully ripe. Internally, the flesh may be water-soaked or light pink and have an aromatic odor reminiscent of cantaloupe melons, but none of these symptoms may be

immediately obvious. In some fruit, only one or a few fruitlets may be infected (Fig. 26.6). In highly translucent, low-sugar fruit, the entire inner cylinder of the fruit used for fruit canning may be invaded (Fig. 26.6) (Pegg et al. 1995). The production of 2,5-diketogluconate by *Pantoea citrea* (*see below*) appears to be responsible for the dark color that is characteristic of pink disease (Pujol and Kado 2000). Some bacterial strains do not cause symptoms on fresh fruit, and brownish-pink discoloration occurs only when fruit are heated during canning (Hine 1975; Rohrbach and Pfeiffer 1976). The disease is of considerable importance for the processing industry, and great care is needed to ensure that infected fruit tissue does not enter the canned product.

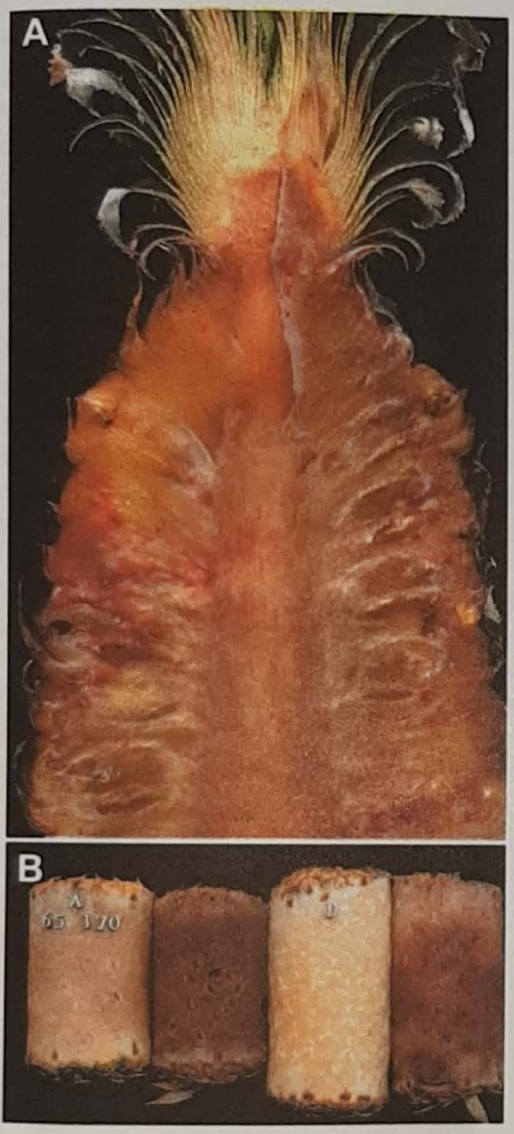

FIG. 26.6. Pink disease of pineapple. **A,** Symptoms within a ripe pineapple; **B,** Symptoms on hybrid pineapple cultivar 65-370 caused by ("A") *Glucanobacter oxydans*, showing pinkish discoloration of uncooked fruit cylinder (left) and chocolate brown discoloration of cooked fruit cylinder (right). Symptomless fruit cylinder ("D") infected with *Pantoea ananas* and, on the right, an infected, discolored cooked cylinder. (A, Courtesy N. K. B. Adikaram—© APS; B, Courtesy K. G. Rohrbach—© APS. Reproduced, by permission, from Ploetz et al., 1994.)

Causal Organisms

Species in at least three genera of bacteria have historically been implicated as causal agents of pink disease: *Pantoea* (*Erwinia*), *Gluconobacter*, and *Acetobacter* (Kontaxis and Hayward 1978; Rohrbach 1976). Initially, the disease was reproduced using a species of *Acetomonas* (Hine 1976); but Rohrbach and Pfeiffer (1976) completed Koch's postulates by heating fresh fruit inoculated with a bacterial isolate from fruit with pink disease and identified the pathogen as *P. agglomerans* (syn. *E. herbicola*, *Enterobacter agglomerans*). Subsequently, the pathogen was identified as *P. citrea* using molecular analyses (Cha et al. 1997). Additional taxonomic studies indicated that the latter species should be correctly classified as *Tatumella morbirosei* (Brady et al. 2008). Furthermore, isolates from pineapple with pink disease in Mexico were identified as *T. ptyseos* (Marín-Cevada et al. 2010). The two pathogens in the Enterobacteriaceae, *T. morbirosei* and *T. ptyseos* (Marín-Cevada and Fuentes-Ramírez 2016), as well as members of the Acetobacteraceae, *G. oxydans* and *A. aceti* (Rohrbach and Johnson 2003), are currently considered the causal pathogens of the disease. Possibly, *T. citrea* may also be involved since it also produces 2-ketogluconate dehydrogenase that oxidizes 2-ketogluconate to 2,5-diketo-d-gluconic acid that is responsible for the discoloration of fruit tissue typical of pink disease (Brady et al. 2010). Depending on the species and strains involved and the severity of infection, browning symptoms may appear in the fruit flesh before heating (Rohrbach and Pfeiffer 1976), or a pinkish discoloration and wilted appearance may be detectable in fruit in the field before harvest (Rohrbach 1989).

Disease Cycle and Epidemiology

It is believed that during cold weather the pathogens infect through the numerous florets that grow out from the developing fruit. The consistent correlation between spraying insecticides during the flowering season and the concomitant reduction of pink disease suggests that insects have a role in disseminating the pathogens among flowers during nectar feeding (Kado 2003; Pegg et al. 1995). Nectar is probably an energy source for the bacteria. Once inside the flower, they remain quiescent in the nectary gland or stylar canal and locule until the

fruit matures, sugar concentrations increase, and translucence occurs (Rohrbach and Schmitt 1994).

Once the pathogen has entered the fruit, it colonizes the intercellular spaces of the tissues. Over time, infected tissues will show moderate water-soaking, but no soft-rotting symptoms. Because pineapple plants are propagated year-round, pink disease is sustained by transmission of the pathogen from field to field.

Critical Biological and Environmental Factors That Favor Disease

Drought before flowering followed by rainfall during flowering increases disease incidence. The pathogens are thought to be among the epiphytic organisms that are carried from infected rotting fruit by insects and mites to healthy open flowers. The disease appears to be limited to production areas where pineapple fruit develop under cooler conditions because the disease rarely occurs in the lowland tropics. Pink disease bacteria cannot survive temperatures above 38°C (Pegg et al. 1995). Thus, pink disease only occurs when flowering occurs during cool weather or a rainy season (around 18°C) and fruit mature during periods when temperatures do not exceed 29°C (Rohrbach 1989).

Management

Cultural practices and natural host resistance

Susceptibility to the disease can be reduced by the use of potash fertilizer. Pineapple cultivars and hybrids vary from highly resistant to very susceptible to pink disease. Cultivation of relatively resistant groups such as 'Smooth Cayenne', provides control of the disease (Rohrbach and Schmitt 1994).

Chemical treatments

Controlling the insect vectors with insecticides is the primary means of managing pink disease. The disease has been managed in the Philippines by applying insecticides during flowering (Kontaxis 1978). Applications starting at the red-bud stage and followed by three additional applications at 5-day intervals throughout flowering have resulted in the highest level of control.

Yeasty Rot

Level of Importance

Yeasts are among the most common organisms found in nature. In damaged and overripe fruit and in fruit with inter-fruitlet cracking, already present yeasts start growing or newly disseminated yeasts invade (Paull 1997). At warm temperatures, they infect and cause fermentation where sugars are converted to alcohol, and carbon dioxide gas is released.

Symptoms

Early symptoms of yeasty rot include the release of gas bubbles (Fig. 26.7A) and juice through cracks and points of injury where the infection occurred (Pegg et al. 1995). The skin turns brown and leathery (Paull 1997). With leakage of juice, the fruit becomes spongy. Internally, the decaying flesh is bright yellow and has large gas cavities (Fig. 26.7B). At late stages of disease, the shell surrounds a mass of spongy, fibrous tissue (Pegg et al. 1995). Some yeasts do not produce gas but cause a glassy spoilage with a distinctive aroma (Snowdon 1990).

Causal Organisms and Disease Cycle

Several species of the genus *Saccharomyces* cause yeasty rot. The disease is widespread and associated with ripe, overripe, and damaged fruit, mainly during the spring season (Pegg et al. 1995). In the spring, rapid changes in fruit growth result from the shift from cold, dry to warm, wet weather that can cause basal cracking between fruitlets. Fruit affected by even minor frost

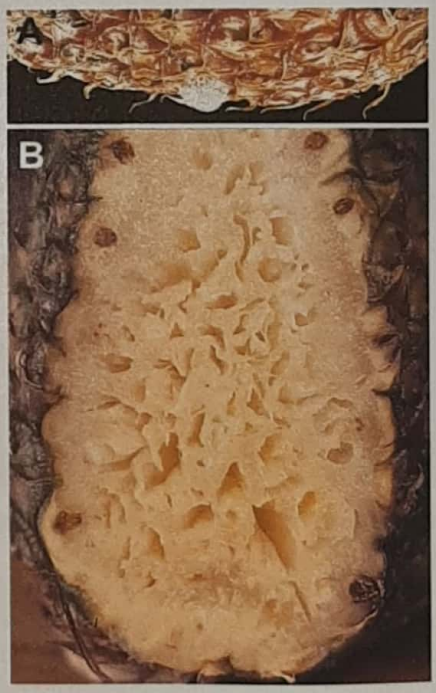

FIG. 26.7. Yeast fermentation of pineapple. **A,** Gas bubbles on fruit surface due to internal yeast decay; and **B,** advanced stage of decay shown in a fruit longitudinal section. (Courtesy UCANR—Reproduced by permission)

damage are prone to cracking as they ripen in spring. Juice exuding from wounds is immediately colonized by yeasts, and these fruits are severely damaged or decayed as they ripen. The disease may occur in the plantation or as a post-harvest problem (Pegg et al. 1995).

Management

Young, developing fruit that will ripen in the spring in frost-prone areas must be protected against damage by covering with paper bags (Pegg et al. 1995). Fruit should also be protected from sunburn and mechanical damage. Fruit showing even minor inter-fruitlet cracking should not be consigned to the market. Any fruit showing fractures between fruitlets should be picked at the earliest stages of fruit maturity to minimize losses from yeasty rot (Pegg et al. 1995).

Internal Browning

Internal browning (IB), also known as black heart or endogenous brown spot (EBS), is a physiological disorder that develops when pineapple fruit are exposed to low temperatures (8 to 15°C) after harvest or in the field. Field-induced IB is most prevalent in Australia (Swete-Kelly and Bagshaw 1993). Symptoms include translucent, water-soaked spots initially at the base of the fruitlets (Abdullah et al. 1985). These spots later become brown and may enlarge and coalesce to form a darker tissue mass along the core (Fig. 26.8). The pattern and the extent of symptom development may vary with the cultivar. The browning is due to oxidation of phenolic substances by polyphenol oxidase (PPO) (Stewart et al. 2001). The disorder has been reported on commercial cultivars in many pineapple-growing countries. Because the affected fruit show no external symptoms, the disorder is often not detected until fruit are sliced, resulting in considerable customer dissatisfaction (Stewart et al. 2002). Harvesting fruit at an early maturity stage tends to limit IB development. Postharvest hot water dips at 38°C for 60 min induced tolerance in 'Mauritius' pineapple to cold injury and reduced IB (Weerahewa and Adikaram 2005). Control of IB was also attempted by inhibiting the expression of PPO in genetically engineered pineapple plants (Graham et al. 1998; Ko et al. 2006).

FIG. 26.8. Development of internal browning symptoms in 'Kew' pineapple ('Smooth Cayenne') during (from left to right) 7, 14, 16, 18, or 21 days of cold storage. (Courtesy N. K. B. Adikaram—© APS)

OTHER DISEASES

A white, gray, green, pink, black, or other-colored mold, known as **surface mold,** is often found on the cut stem (peduncle) (Fig. 26.9A) and sometimes the fruit surface (Fig. 26.9B) or crown of fruit during marketing.

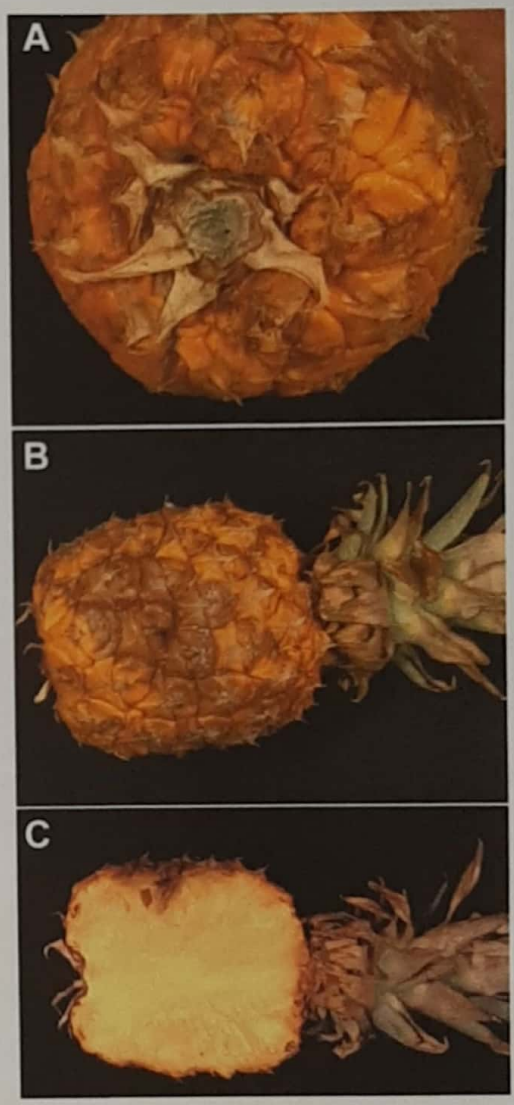

FIG. 26.9. Surface mold. **A,** Cut fruit stem end (peduncle) colonized by a *Penicillium* species with greenish sporulation; **B,** colonization and discoloration of the fruit surface; and **C,** Penicillium side rot that is occasionally found on mature ripe fruit during marketing. (Courtesy J. E. Adaskaveg and H. Förster—© APS)

The surface mold reduces the appearance of the fruit and may occasionally cause a shallow decay (Fig. 26.9C). Mold growth on the cut stem end is common because the moisture and nutrients provide a favorable environment for growth of many filamentous fungi such as *Penicillium* spp. and yeasts. Surface mold growth can be effectively managed by treating fruit after harvest (where permitted) with a broad-spectrum fungicide such as triademefon, fludioxonil, fludioxonil/azoxystrobin, or natamycin.

BANANA

BOTANICAL CLASSIFICATION AND GEOGRAPHICAL ORIGIN

■ Bananas (genus *Musa*, family Musaceae) are perennial plants, and most edible forms are derived from the wild species *Musa acuminata* Colla (AA) and *M. balbisiana* Colla (BB) or a combination of both. Cultivars are diploid, triploid, or tetraploid and are described by their name and genetic makeup. For example, the name 'Lady Finger AAB' indicates a triploid hybrid of two genomes, A and B, with more genetic inheritance from *M. acuminata* than *M. balbisiana*. Banana hybrids are also known as *Musa* × *paradisiaca* L. Plantains (mostly ABB, *M. acuminata* × *M. balbisiana*) are another hybrid. Plantains have a high starch content, even when fully ripe, in contrast to the sweet dessert bananas, and are used for cooking. Both types of bananas are affected by similar postharvest diseases.

The banana plant has an underground stem or rhizome. The above-ground portion of the plant consists of leaves and fused petiole bases that form a pseudostem. The plant produces a terminal inflorescence, which emerges through the pseudostem and bends downwards after extrusion. The inflorescence develops into several flower clusters. In cultivated species, fruit arise without fertilization (parthenocarpy). Fruit take 12 to 15 weeks to develop from anthesis to harvest maturity.

The edible fruit develops from an inferior ovary. Fruit are seedless, fleshy berries that grow in clusters at each node. A cluster is commercially referred to as a 'hand'. A bunch of bananas can have several hands, each hand consisting of a cluster of 10 to 20 fruit. Individual fruits in a hand are called 'fingers'. The disc through which the fingers in a hand are attached to the bunch stem is called the 'crown'.

The primary center of origin of bananas is thought to be Malesia (Malaysia, Indonesia, the Philippines, Borneo, and Papua New Guinea). The top producing countries in 2017 were: India (30.4 million tons – Mt), China (11.4 Mt), Indonesia (7.2 Mt), Brazil, Ecuador, and the Philippines (each producing ca. 6 Mt), Angola (4.3 Mt), Guatemala and Colombia (each ca. 3.8 Mt), and Costa Rica and Mexico (each ca. 2.4 Mt) (Anonymous 2017). Ecuador is the main exporter to Europe and the United States, followed by Costa Rica and the Philippines.

Bananas are climacteric fruits, and the period between harvest and climacteric rise, the pre-climacteric life, is commonly called the 'green life' (Peacock and Blake 1970). The best estimator of the green life is physiological age, expressed as a sum of accumulated temperatures from flowering to harvest rather than days (Jullien et al. 2008). The physiological age of bananas (expressed in dd; i.e., degree days) has an impact on fruit susceptibility to postharvest decays and is an important factor for the proper timing of harvest. Other maturity indices are based on the age of the bunch, the interval between flowerings and harvesting, the filling of the fingers, or the color of the skin and pulp and brittleness of the flower end of the bunch (Hailu et al. 2013). Most of these criteria depend on the banana cultivar. Harvesting physiologically old fruit can induce ripening of all bananas in a container due to ethylene release (Liu 1976).

IMPORTANCE OF THE CROP

■ Banana is the most consumed fruit in the world. Based on production, trade, and consumption, the banana is ranking fourth after rice, wheat, and maize. About 105 Mt are produced each year in the tropics and subtropics. About 16 Mt enter the international market, with AAA cultivars dominating among fresh fruits; 70% of this production originates from Latin America. Dessert bananas are allowed to ripen and are eaten fresh. Cooking bananas have a high starch content and are consumed when green or ripe after boiling, frying, or roasting. Bananas provide carbohydrates for human nutrition either as starch in cooking bananas or as sugars in dessert bananas. The ripe banana is a good source of vitamin A, B6, and C, and has a high content of carbohydrates, fiber, and potassium. Bananas are low in protein and free of fat.

There are almost 1000 varieties of bananas in the world, subdivided into 50 groups. Among edible bananas, the triploid groups AAA, AAB, and ABB are the most common. Dessert bananas are found in all three groups, but the AAA 'Cavendish' genomic group

(with 'Gros Michel', 'Cavendish', and 'Lujugira-Mutika' subgroups) dominates in the international trade and constitutes approximately 50% of the world banana production. Plantains belong to the AAB group and cooking bananas to the ABB group, however, some cooking bananas belong to the other two genotype groups (Jones and Daniells 2019).

PRE- AND POSTHARVEST PRACTICES FOR MANAGING POSTHARVEST DISEASES

Integrated pre- and postharvest practices are needed to successfully manage fruit decays that occur from harvest to marketing and consumption, and these practices are similar for many of the diseases. Field sanitation in banana plantations is important, especially the removal of senescent or decaying leaves and flower parts which can act as sources of inoculum for fruit infection. Because fungi may colonize the bunch stalk, potentially leading to crown rot, early removal of flower parts in the field is essential (de Lapeyre et al. 2000). Some practices such as field trimming of false hands, some true hands, male buds, and external fruits of hands can accelerate fruit pulp filling. Subsequently, fruit more rapidly reach the physiological age necessary for a sufficient commercial grade and can be harvested sooner (Krauss and Johanson 2000; Lassois et al. 2010b). As mentioned above, fruit physiological age is the best estimator of 'green life' and thus, should be taken into account at harvest.

Plastic sleeves and treatments with prophylactics such as fungicides can reduce *Colletotrichum musae* contamination on developing bunches by 80% (de Lapeyre et al. 2000; Lassois et al. 2010b) and can also reduce other postharvest decays. At least five multi-site fungicides in the classes ethylene-bis-dithiocarbamates (e.g., mancozeb, propineb - FRAC Code M3), dimethyl-dithiocarbamates (e.g., thiram, metiram - FRAC Code M3), and chloronitriles (e.g., chlorothalonil – FRAC Code M5), as well as numerous single-site mode of action fungicides representing methyl benzimidazole carbamates (MBCs, FRAC Code 1), demethylation inhibitors (DMIs, FRAC Code 3), succinate dehydrogenase inhibitors (SDHIs, FRAC Code 7), anilinopyrimidines (APs, FRAC Code 9), quinone outside inhibitors (QoIs, FRAC Code 11), and quinone inside inhibitors (QiIs, FRAC Code 21) are registered as preharvest treatments for managing foliar diseases (mainly Sigatoka) in the field. Application of preharvest fungicides helps reduce disease and inoculum in the plantation by protecting leaves, stems, and fruit from infections and thus, also reduce postharvest decay. Still, the most efficient management method for crown rot, anthracnose, and other decays is the routine postharvest treatment of fruit with systemic fungicides (Lassois et al. 2010b). Use of the first systemic fungicides, the methyl benzimidazole carbamates (e.g., benomyl, carbendazim, thiabendazole), started in the late 1960s. They are classified as antimitotic compounds because they bind to β-tubulin, a spindle fiber protein, and inhibit mitosis of the fungal nucleus. Gradually, other fungicides such as imazalil, bitertanol, and prochloraz that inhibit a demethylation step in ergosterol biosynthesis (i.e., DMIs) were introduced (Johanson and Blazquez 1992; de Lapeyre de Bellaire and Nolin 1994).

Preventive Measures at Packing Stations

Sanitation practices include deflowering before the dehanding operation and involves removal of all floral parts that could potentially harbor inoculum. During fruit dehanding, large injuries and finger flexing resulting in creased stems may occur that can become main infection sites for wound pathogens (Fig. 26.10). If floral parts are not removed in the field, this is done at the packing station before bunch trimming, thus reducing the risk of contamination of the washing baths (Krauss and Johanson 2000; Shillingford 1976).

Maintaining water quality at the packing station is important to minimize fungal infections (Arneson 1971; Eckert and Ogawa 1985; Shillingford 1977; Slabaugh and Grove 1982). Spore accumulation in wash water can be reduced by regularly changing and de-latexing the water. It is recommended that the bath water be treated with active chlorine or quaternary ammonium disinfectants

FIG. 26.10. Creased stems of banana fingers and cut surfaces of crowns are infection sites. (Courtesy D. Edwards, UCANR—Reproduced by permission)

(where permitted) to reduce microbial contamination including human pathogens. The chlorine concentration has to be regularly monitored and adjusted to compensate for losses from volatilization and inactivation by latex or other organic matter. It is difficult to sanitize the water with chlorine when the water is recirculated in a closed system, and latex contamination gradually increases in the tanks. Therefore, non-recirculating sanitation rinses are preferred.

Postharvest Fungicide Applications

Some of the fungicide treatments applied in the field can also have an impact on postharvest decay development. There are only few fungicides approved for postharvest use, and among these, thiabendazole, imazalil, and prochloraz have been most commonly used in some countries (Johanson and Blazquez 1992). Additional compounds may become available in the future. Because postharvest fungicides generally have the same mode of action as those used in the field to control Sigatoka disease, this over-use of few modes of actions may lead to development of resistant strains.

Fungicides can be applied to harvested bananas using dips, sprays, or drenches (Fig. 26.11). Good coverage and wetting of the fruit have to be assured. The time between crown trimming and fungicide application is critical and should be as short as possible to protect the wounds. Bananas are usually treated in packing stations just after they are removed from the washing bath. Potassium aluminum sulphate (i.e., alum) is often included in the fungicide solutions to neutralize latex residues remaining on the crowns after washing. Some fungicides, however, such as thiabendazole and prochloraz are not compatible with alum.

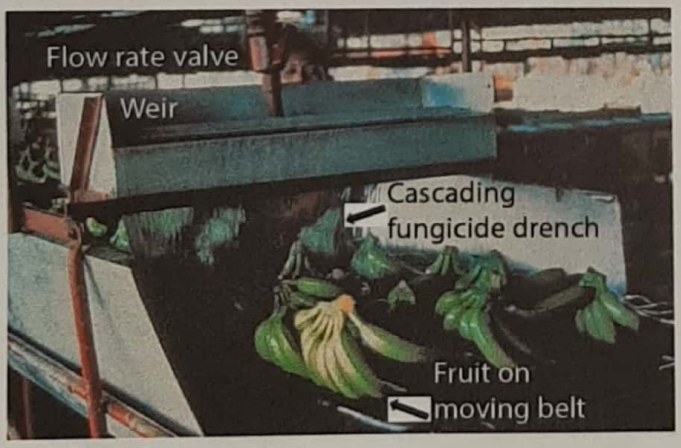

FIG. 26.11. Recirculating drench method of treating banana fruit to control crown rot. A weir is filled with a fungicide solution with an adjustable valve. The fungicide solution then cascades over the fruit as a drench treatment while fruit are conveyed on a moving belt. (Courtesy W. R. Slabaugh—© APS. Reproduced, by permission, from Ploetz et al., 1994.)

Storage of Bananas

Temperature, relative humidity, and atmospheric composition are the main environmental factors that influence storage disease development (Hailu et al. 2013). They may directly affect the pathogens, but they also have an indirect effect on the fruit by affecting its metabolism. Modification of these environmental factors can therefore extend the banana green life, and there is a direct relationship between ripeness and susceptibility to disease.

Storage Temperature and Postharvest Decay

Banana metabolism and decay development is reduced using refrigeration at the lowest possible temperature where physiological disorders of the fruit do not occur. Cooling should be continuous throughout storing, shipping, and marketing but for as short as possible. Containers for shipping of bananas are kept at 13 to 14°C because temperatures below 12°C provoke peel browning from injury and oxidization (Muirhead and Jones 2000). Fungal growth is reduced at 13°C, but some pathogens are still able to infect and slowly colonize the fruit. In one study, storage of bananas at 18°C resulted in significantly less crown rot, anthracnose, and cigar end rot, whereas decay was highest at 35°C (Azeem et al. 2016).

Relative Humidity

High relative humidity reduces water loss from the fruit by transpiration, ensuring a long green life. Green life is markedly reduced at a relative humidity of 30 to 40% or lower as a result of ethylene production by the fruit peel (Peacock 1973).

Atmospheric Composition

The composition of the atmosphere also determines metabolic activity of bananas. A modified atmosphere (MA) can be achieved by packing fruit in sealed plastic bags, whereas a controlled atmosphere (CA) is obtained by injecting nitrogen into storage rooms (Hailu et al. 2013). Some postharvest diseases can be partially controlled by packing bananas in MA (Bastiaanse et al.

2010). For MA, the balance between a lower O_2 and a higher CO_2 content depends on the extent of fruit respiration, bag permeability, and the composition of the ambient air. O_2 and CO_2 contents generally range from 1 to 10% and from 2 to 14%, respectively, depending on the quality and thickness of the plastic packaging. This modification in the gas composition reduces fruit respiration and endogenous ethylene synthesis. This can considerably increase the length of the preclimacteric phase and reduce postharvest diseases. According to some studies, storage life of bananas can be increased five times when stored in plastic film where the O_2 content is stabilized at about 2% and the CO_2 content at about 5%, and an ethylene scrubber is used compared to fruit stored without wraps (Hailu et al. 2013). High (>15%) CO_2 and low (<1%) O_2 levels are toxic to many postharvest pathogens. However, bananas cannot be stored at CO_2 levels above 7 to 12% or O_2 levels below 1 to 2% because detrimental physiological changes occur (Lassois et al. 2010b).

POSTHARVEST DISEASES

Bananas are affected by numerous fungal and some bacterial pathogens, and these pose major production constraints worldwide. In the field, Black Sigatoka (*Pseudocercospora fijiensis*, formerly: *Mycosphaerella fijiensis*) that affects leaves and Panama disease (*Fusarium oxysporum* f. sp. *cubensis*), a soil-borne wilting disease, are the most destructive. Panama disease has been largely controlled by the introduction of 'Cavendish' cultivars that are more resistant to infection. With few exceptions, postharvest diseases are mostly affecting the ripe fruit. Anthracnose and crown rot are by far the most damaging postharvest diseases of banana throughout the world (Ploetz et al. 1994). Diseases below are discussed in alphabetical order by their names.

Anthracnose

Level of Importance

Anthracnose is the most important postharvest disease in every banana-producing and -marketing country of the world and accounts for a majority of postharvest losses. Anthracnose can be a serious problem during shipping if fruit start to ripen prematurely. Minute quantities of ethylene produced by the pathogen and the host initiate the climacteric ripening process (Daundasekara et al. 2003).

Symptoms

Anthracnose is most commonly seen as a finger blemish or rot of ripening or ripe bananas and may affect the entire fruit. Initially, numerous small, brown, circular spots develop on the ripening fruit skin. These become enlarged, sometimes coalesce, turn charcoal-black in color, and are slightly sunken at advanced stages. Sometimes, anthracnose lesions are oval or lenticular in shape and are several centimeters in length. The sunken spots become deep depressions that are covered with pink masses of fungal conidia (Fig. 26.12A and B). The pulp immediately under the affected peel may turn brown and gelatinous. However, pulp symptoms are not typical for the disease. Large anthracnose lesions are the result of infections that are initiated at physical injuries to the skin. Circular spots start from pre-harvest quiescent infections of the intact skin during fruit ripening (Simmonds 1963). Anthracnose may also occasionally develop on green fruit as mostly dark brown to black, lenticular, slightly sunken lesions with a pale margin. The same fungus can also cause black end and tip rot.

Causal Organisms

Anthracnose of bananas traditionally has mostly been attributed to the Ascomycota fungus *Colletotrichum musae*, a member of the *C. gloeosporioides* species complex

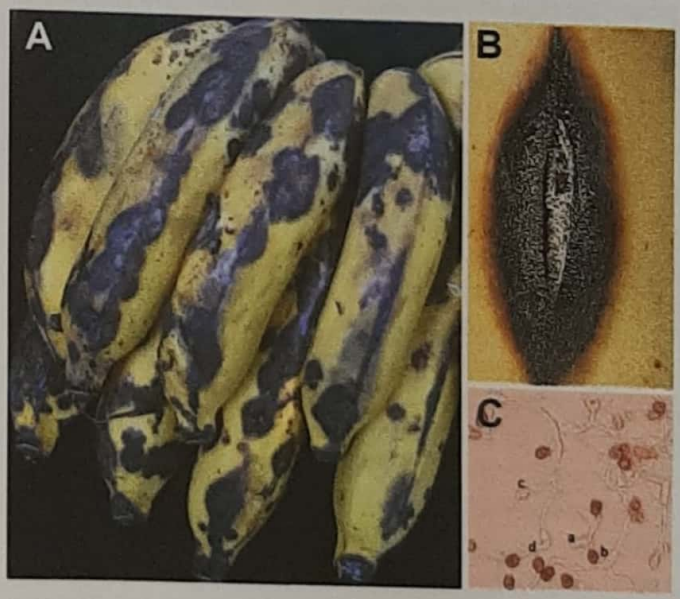

FIG. 26.12. **A,** Banana fingers heavily infected with *Colletotrichum musae*, showing typical sunken, charcoal-black anthracnose lesions; **B,** close-up of an anthracnose lesion; **C,** germinated conidia (a) of *C. musae* with dark (b) and hyaline (c) appressoria and (d) germ tubes. (A and C, Courtesy N. K. B. Adikaram—© APS; B, Courtesy D. Edwards, UCANR—Reproduced by permission)

(Su et al. 2011). This may be due to the reliance on host association to define species limits (Vieira et al. 2018). Based on morphological and molecular methods, additional species have been identified including *C. karsti* in the *C. boninense* species complex, *C. paxtonii* in the *C. acutatum* species complex, and *C. gloeosporioides sensu lato* (Intan Sakinah et al. 2013; Udayanga et al. 2013). In Brazil, among 431 isolates of *Colletotrichum* species obtained from banana fruit with anthracnose symptoms, the majority (93.5%) were assigned to *C. musae* and the remaining to *C. tropicale*, *C. theobromicola*, *C. siamense*, and a newly described species, *C. chrysophilum* (Vieira et al. 2018). Thus, *C. musae* may still be the main pathogen, however, other species may be of local importance.

Disease Cycle and Epidemiology

The disease cycle has been described for *C. musae* only, but likely is similar for the other species causing anthracnose. *C. musae* is present abundantly on transition leaves and diseased crop debris including flower parts and the last bunch bract. These serve as important sources of primary inoculum. Conidia are disseminated by rain, wind, and insects to fruit at early stages of maturity in the field where they germinate and form appressoria. Two types of appressoria, hyaline and dark, are formed (Fig. 26.12C) (Muirhead and Deverall 1981). The hyaline appressoria produce infection hyphae that penetrate into epidermal cells, leading to accumulation of phytoalexins (Brown and Swinburne 1980). The dark appressoria remain quiescent on the immature fruit (Swinburne and Brown 1983) and become active during fruit ripening causing anthracnose lesions. Kamo et al. (2000) showed that the level of hydroxyanigorufone phytoalexins in unripe fruit started to increase 1 to 2 days after inoculation with conidia of *C. musae* and, after its transient maximum, decreased during fruit ripening by exposure to ethylene. The level of phytoalexin production was much lower in ripe than in unripe fruit. In a 2013 report, a comprehensive analysis of the expression patterns of 15 pathogenesis-related proteins (*PR*; three *PR1*, one *PR2*, three *PR5*, one *PR10*, four chitinase, and three chitinase-like genes) and two *WRKY* transcription factors in banana fruit in relation to salicylic acid (SA)- and methyl jasmonate (MeJA)-induced resistance was done (Tang et al. 2013). The results indicated that the activation of banana *PR*s and WRKY genes by SA and MeJA, and WRKY transcription factors binding to *PR* promoters, may be attributed at least partially to SA- and MeJA-induced pathogen resistance in the host.

C. musae can also infect fruit through small peel wounds without forming quiescent infections, usually during or after harvest. These infections continue to develop without a quiescent period and cause typical anthracnose lesions.

Critical Biological and Environmental Factors That Favor Disease

Humid weather and warm temperatures (25 to 30°C) favor rapid growth of *C. musae* and development of the disease. The relative humidity has to be above 86% for germination of conidia. Studies are underway to identify the underlying mechanisms and key genetic factors involved in the decline of fruit resistance during ripening. Reduction in phytoalexin levels (Abayasekara et al. 2013; Brown and Swinburne 1980) and softening of cell walls during fruit ripening result in the activation of quiescent *C. musae* infections and anthracnose development.

A study comparing the effect of physiological fruit age on susceptibility to wound anthracnose indicated that bananas of the same grade (i.e., fruit diameter) but with lower accumulated temperature sums were less susceptible to *C. musae* (Chillet et al. 2006). Additionally, soil-climate conditions also affected susceptibility; fruit grown in cooler highland areas were less susceptible than fruit of the same physiological age from lowland plantations.

Management

Field, harvest, and packinghouse practices

It is difficult to completely prevent infection of young fruit in the plantation, but any management strategy contributes to a reduced disease incidence. Strict sanitation procedures have to be followed. Dead leaves must be regularly cut and removed from plants and destroyed. Removal of flower debris and other tissues that harbor the pathogen is beneficial. Bunches must be harvested carefully at the proper stage of maturity and handled carefully without causing injuries or bruising. Bunches must be well protected with foam padding during transport to the packinghouse. Fruit should be cooled to 13°C soon after harvest and stored at this temperature to slow anthracnose development and ethylene production. Lower temperatures increase the probability of chilling injury. The packinghouse must be kept clean and free of bunch debris and rejected fruit. Bunches should be dehanded and washed in clean water containing detergent and chlorine to remove dirt and latex (sap) exuding from the cut crown. The water should be changed frequently. Special precautions must be taken to cope with bleeding sap. Sap should be allowed to drain from the freshly cut crown before washing.

Preharvest treatments

Preharvest spray treatments of developing bananas with salicylic acid or Bion®, a fruit resistance inducer, 9 and 11 weeks after bunch emergence has been reported to delay the incidence and reduce severity of anthracnose during ripening. Treated fruit showed higher activity of chitinase and β-1,3-glucanase (Wanigasekara et al. 2014).

Postharvest treatments

Fruit to be exported are routinely washed and dipped in a systemic fungicide (e.g., thiabendazole, prochloraz, imazalil) before they are packed and cooled for shipment (Johanson and Blasquez 1992). In situations where chemical control is necessary, postharvest treatment with a systemic fungicide is more effective than using other treatments such as mancozeb (Diedhiou et al. 2014).

Biological treatments

Anthracnose development by *C. musae* was significantly reduced in banana fruit field-infected with the freckle fungus, *Phyllosticta musarum*. Freckle infection is associated with several biochemical and cellular defense responses including accumulation of phytoalexins and upregulation of chitinase and β-1,3-glucanase (Abayasekara et al. 2013). Freckles are pin-head sized, superficial infections in the banana peel which do not expand during fruit ripening. The development of anthracnose may be reduced by allowing a certain amount of freckle infection in the peel, provided that there are no quarantine restrictions for freckle disease.

Black-End Rot (Stem End Rot), Tip Rot

Level of Importance

Black-end rot occurs on ripening fruit worldwide, but is generally of minor importance. It may become a problem when fruit are ripened as singles because the pathogen enters through large wounds. When fruit are ripened in clusters, hands, or on the bunch, the disease occurs where finger stalks have been bent and bruised by rough handling. In contrast, crown rot (which is discussed below) occurs on hands of bananas where a large number of injuries are inflicted on the crown during dehanding.

Causal Organisms

The main pathogen of black-end rot is *Colletotrichum musae* that also causes anthracnose. Black-end rot can also be caused by *Nigrospora sphaerica* that is also involved in squirter disease, and *Fusarium* spp. Tip rot of banana in Jamaica was reported to be caused by *Musicillium theobromae* and *Fusarium* spp. (Meredith 1965).

Symptoms

Black-end rot is characterized by a black decay of the finger stalk and adjacent tissues (Fig. 26.13). The infected stalk is first brown discolored before it turns black. The darkened peel at the fingertip may be separated from the sound yellow peel by a narrow, water-soaked, greenish-brown ring. When only part of the finger is affected, the decay is usually confined to the peel. In severe cases, the decay extends to the shoulders of the fruit and into the pulp. When *C. musae* is the causal agent, the blackened peel is unusually soft and moist, and the outer skin layer lifts off easily when rubbed. Additionally, anthracnose symptoms may develop as circular to oval, brown or black, sunken spots on the peel, and the spots may coalesce and cover large areas of the fingers. These symptoms should not be confused with ageing spots that develop on ripe fruit. Pink conidial masses may cover the decay at advanced stages of disease. With *N. sphaerica* infections, the outer skin is firmer, and there may be a dense mat of white fungal growth. Additionally, an area of water-soaked peel may extend beyond the blackened area. The pulp immediately next to the stalk may be dark and water-soaked. *Fusarium* spp. cause a drier lesion with shrinkage of the stalk. A sparse growth of gray fungal hyphae may be visible. Mixed infections and infections with secondary fungal growth are common. Squirter disease is a watery rot that develops from the stalk-end of ripe bananas along the center of the pulp, and the skin may show a slight bluish discoloration. The affected fruit, when squeezed, spurts out the pulp. The disease is most common when individual fingers are packed.

Disease Cycle and Epidemiology

Black-end rot occurs when conidia of *C. musae* that are present in the flower remnants infect the fruit tip during ripening. The fungus readily colonizes dead and dying banana leaves, flowers, and fruit. Conidia are formed in large numbers in acervuli and are disseminated by wind-driven rain and perhaps insects. Quiescent infections

FIG. 26.13. Black end rot of 'Seeni' banana caused by *Colletotrichum musae*. (Courtesy N. K. B. Adikaram—© APS)

are established in bunches of green fruit about 20 to 40 days after emergence. These infections will start actively growing when the bunch starts to ripen and cause typical lesions at the tip of the fruit. With high inoculum levels of the pathogen on plant surfaces, large wounds occurring at the crown during dehanding or on the stems of fingers can also be colonized after harvest and cause crown and stem end rot, respectively.

Management

Control measures for black-end rot are similar as for anthracnose. Bunches should be bagged as soon as the male bud has been removed, usually when the fingers are horizontal. Inoculum levels can be reduced by removing senescent and dead leaves and the remains of flowers from the plantation. During harvest and packing, banana bunches should be handled carefully to avoid injuries. Sanitation practices should be employed in packing areas and include removal of remains of leaves, flowers, and rejected fruit. When trimming the crown of the bunches, a clean, surface-sterilized knife should be used to obtain a smooth cut surface. In commercial production, hands of bananas are often treated with postharvest fungicides such as MBC (e.g., thiabendazole) or DMI (e.g., imazalil, prochloraz) compounds before they are put into boxes. No resistant varieties are known for this disease.

Cigar-End Rot

Level of Importance

Cigar-end rot is an economically important disease in Central and West Africa (Ploetz et al. 1994). The disease also occurs in the Canary Islands, India, South Africa, South America, Sri Lanka, the West Indies (Meredith 1965; Wardlaw 1931), Iran, and Egypt (Ed-Halaly et al. 1954).

Symptoms

The infection can occur in young bunches, causing necrosis at the pistillate end of the fruit. One or all fingers on a hand may be infected. The first symptoms are a localized darkening and wrinkling of the peel at the tip. The skin becomes folded and shrunken as the infection spreads slowly along the fingers; gray conidia are formed on the shriveled stalk-end of the fruit. A black band and a narrow chlorotic region between infected and healthy tissues border the darkened area. At advanced stages of the disease, the finger has the ashen-gray appearance of a burnt cigar (Fig. 26.14A). In Trachysphaera tip rot, the surface of the lesion becomes covered with white spores that turn pink or brown. The pulp tissue shows a characteristic dry rot and eventually is reduced to a dry fibrous mass. A wet rot can occur in the presence of secondary microorganisms. In Musicillium tip rot, the pulp is typically gray and fibrous and gray, powdery spore masses are present on the lesion surface (Fig. 26.14B and C).

Causal Organisms

Trachysphaera fructigena, an Oomycota organism, and *Musicillium theobromae* (syn. *Verticillium theobromae*) cause cigar end rot in Central and West Africa (Ploetz et al. 1994). *T. fructigena* has not been reported in the western hemisphere, but *M. theobromae* occurs in both hemispheres (Jones and Stover 2019). The disease caused by *M. theobromae* was first reported by Dhingra et al. (1970) in India. *T. fructigena* can also cause a destructive rot in West and Central Africa (Maramba and Clerk 1974).

The mycelium of *T. fructigena* is non-septate, and asexual reproduction is by conidia. Conidiophores are erect and usually produce one terminal conidium, or they are complex with a whorl (or several whorls) of conidia on branches arising from the main hypha. Conidia are spherical, 13 to 48 µm in diameter, echinulate, thin-walled,

FIG. 26.14. Cigar end rot symptoms on ripe banana fruit. **A,** Symptoms on an Asian banana variety caused by *Trachysphaera fructigena*; **B** and **C,** symptoms on ripe banana fingers from the Western Hemisphere caused by *Musicillium theobromae*. (A and B, Courtesy N. K. B. Adikaram—© APS; C, Courtesy D. Edwards, UCANR—Reproduced by permission)

hyaline, and have a 10- to 30-µm long pedicel. Inside the host, thick-walled conidia ('chlamydospores') may be produced. Pyriform, thick-walled oogonia (24 × 40 µm) have sac-like outgrowths, and each contains a thin-walled oospore. Antheridia are amphigynous.

Koch's postulates with *M. theobromae* were fulfilled in inoculations of wounded and non-wounded green bananas (Masudi and Bonjar 2012). Incubation at 23°C and high relative humidity resulted in typical cigar end rot symptoms after 30 days. Cultures of the pathogen grow rapidly on potato dextrose and malt extract agar at 23°C. The mycelium is white, flocculose, compact or sparse, and becomes olivaceous gray-brown after 1 to 2 weeks. Conidiophores are verticillately branched, and ellipsoidal to sub-cylindrical conidia (3 to 8 µm × 1.5 to 3 µm) arise singly at the apices of the phialides. Chlamydospores and microsclerotia are absent.

Disease Cycle and Epidemiology

The incidence of disease increases during periods of high humidity and rainfall. Conidia of *M. theobromae* are wind-disseminated and infect dying flower parts. The source of *T. fructigena* inoculum is not known. In West Africa, the incidence of cigar end rot is highest along plantation borders and at higher elevations (Tezenas du Montcel and Laville 1977). The high incidence of postharvest cigar end rot is likely due to packing of banana finger-bunches in plastic bags during shipping and storage that creates a favorable environment with high relative humidity where contaminating spores on the fruit surface can cause infection (Masudi and Bonjar 2012).

Fruit infected in the field with *T. fructigena* continue to rot after harvest unlike those infected with *M. theobromae* where premature ripening and postharvest rotting do not occur. New infections by *T. fructigena* can develop during dehanding and delatexing or in contaminated ripening rooms. New infections typically develop on freshly cut crowns and on fruit injuries (Ploetz et al. 2003).

Management

Preharvest

Frequent removal of dead flowers followed by plastic sleeving of the entire developing fruit bunch with perforated polyethylene (30 to 40 µm thick) helps to reduce the disease (Ploetz et al. 1994). Bracts and dead flower parts that accumulate in the bags should be removed after a few weeks. Sanitation is helpful in reducing *M. theobromae* inoculum in the field. This includes removal of all dying or dead leaves from plants routinely but especially in the rainy season and burning or burying infected plant parts away from the plantation. Spraying with a recommended fungicide (e.g., mancozeb) as soon as fruit are formed is effective in controlling the disease, especially during peak infection periods.

Postharvest

Sanitation of the packing station to avoid contamination of fruit with fungal spores is essential to reduce postharvest infection and rot. Infected fruit should be removed prior to dehanding to avoid contamination during dehanding and delatexing tank water with spores of the pathogens. It has been suggested that avoiding packaging of bananas during storage and shipping in plastic may reduce postharvest losses from infection by *M. theobromae* (Masudi and Bonjar 2012).

Crown Rot

Level of Importance

Crown rot of dehanded (hands are separated from the bunch) bananas is a major cause of losses during storage and marketing. It is considered one of the most serious and common postharvest and post-packaging diseases of banana (Khan et al. 2001). All commercial cultivars of dessert bananas are known to be susceptible to crown rot.

Symptoms

The infection takes place on the exposed surface of the crown of dehanded bananas. The exposed crowns of hands turn black and rot (Fig. 26.15A). Usually,

FIG. 26.15. Severe crown rot symptoms in dehanded 'Poovan' banana during storage. **A,** Multiple bunches with symptoms; and **B,** close-up. (A, Courtesy N. K. B. Adikaram—© APS; B, Courtesy D. Edwards, UCANR—Reproduced by permission.)

the rot is confined to the crown, but as fruit continue to ripen, the decay advances rapidly down to the fruit stalks causing fruit to separate from the hand. Fungal colonization gives the affected crown surface a whitish to gray appearance. The rot may advance to the finger stalks (Fig. 26.15B) causing the fingers to drop off when handled. Finger stalks may also be infected directly in the absence of crown rot if the fingers are injured by bending (Meredith 1971). With severe infections, whitish fungal mycelium and reproductive bodies develop on the rotted crown, finger stalks, and finally on the fingers (Griffee and Burden 1976).

Causal Organisms

Crown rot is typically a disease complex that is caused by several fungi. The pathogens involved depend on the locality, time of the year, and possibly other factors (Meredith 1971). Fungi that have been associated with banana crown rot include *Fusarium chlamydosporum* (syn. *F. sporotrichioides*), *F. incarnatum* (syn. *F. pallidoroseum* and *F. semitectum*), *F. neocosmospora*, *F. oxysporum*, *F. verticillioides* (syn. *F. moniliformae*), other *Fusarium* spp., *Musicillium theobromae* (syn. *Verticillium theobromae*), *Lasiodiplodia theobromae*, *Penicillium coryophilum*, *Acremonium* sp., *Nigrospora sphaerica*, *Colletotrichum musae*, and *Thielaviopsis musarum* (Griffee and Burden 1976; Jones 1991; Jones and Muirhead 2019; Knight 1982; Martin et al. 1996). Additionally, *Thielaviopsis paradoxa* (syn. *Chalara paradoxa*, *Ceratocystis paradoxa*) is a common crown rot fungus in Colombia and Ecuador on fruit kept at 14.4°C during shipping and storage and also at higher temperatures (Greene and Goos 1963). In the Dominican Republic, the etiological agents of crown rot were ranked based on their presence and pathogenicity as *F. incarnatum*, *C. musae*, *F. verticillioides*, *F. sacchari*, and *L. theobromae* (Kamel et al. 2016).

Disease Cycle and Epidemiology

Many of the fungi causing crown rot survive on plant debris in the plantation. Conidia on decaying flowers, bracts, stalks (Finlay et al. 1992), as well as transitional and normal leaves are carried by wind or rain splash to the fruit surface. At the time of harvest, infection occurs through the cut ends of the dehanded crowns. Most dehanded bananas are dipped into water as they are cut from the main stalk to remove the latex. The spores on the surface contaminate the washing water and packinghouse equipment and enter the dehanding crown cuts. Fruit stored for long periods at the market are more prone to develop crown rot (Stover 1972).

Management

Cultural methods and sanitation of banana plantations

Most species involved in the fungal crown rot complex are saprophytes on senescent banana tissues, especially on decomposing leaves. Old leaves in the banana plantation may contain inoculum that contaminate fruit. Inoculum pressure can be reduced by regular removal of senescent leaves nearby fruit. Banana floral parts are also inoculum sources, especially for *C. musae* and several *Fusarium* species (Knight 1982). Thus, early removal of flower parts in the field is also important for reducing bunch contamination by the pathogens (de Lapeyre 2000).

Plastic sleeving of bunches

No detailed studies have been done to assess the impact of sleeving on crown rot development. Still, it is known that bunch sleeving with perforated plastic film protects bunches from fungal contamination, and a reduction by over 80% has been found for *C. musae*. Thus, sleeving may directly reduce crown contamination in the field (de Lapeyre 2000), but sleeved fruit also release fewer spores into washing water during packing operations.

Maturity stage at harvest

Bananas should be harvested at an age that will ensure a sufficient pre-climacteric life, and they should reach the ripening rooms unripe (green). The fruit physiological age should therefore be considered when determining the harvest date. Some practices such as field trimming of false hands, some true hands, male buds, and external fruits of hands can accelerate the fruit pulp filling rate. These practices can reduce the physiological age that bananas need at harvest to enable them to reach a sufficient commercial grade.

Packing station sanitation

Protective measures implemented in the packing station are aimed at keeping the crowns of freshly trimmed bananas away from all inoculum sources. To ensure efficient crown rot control, it is thus essential to keep the packing station and adjoining facilities clean. Plant waste in the vicinity of the banana packing area that could be inoculum sources must be eliminated. It has been shown that trimming clusters in a clean environment rather than in the field can reduce crown rot incidence by 50%.

Banana crown trimming

Bananas should be trimmed with a clean stainless-steel blade. Less effective ways of trimming the crowns or ripping off the hands significantly increases the level

of fruit contamination. Tissue fragments on the surface of the crowns dry and quickly become senescent, thus providing an ideal site for rot development (Lassois et al. 2010a,b). Moreover, the tips of banana trimming knives are rounded to avoid fruit injuries. It is also important to cut wide crown sections containing as much crown tissue as possible, a technique that seems to enhance crown resistance to rot and seldom leads to the spread of rot into the fruit pedicels.

Chemical treatments

The disease can be reduced by fungicide and other treatments. Postharvest application of the protective systemic fungicide imazalil to the exposed crown is the most common commercial control practice for banana crown rot (Cox 1996; Lassois et al. 2010b; Shillingford 1976). Recycling drench applications are commonly used (Fig. 26.11). In inoculation studies, this latter fungicide was more effective than azoxystrobin or boscalid (Daniel et al. 2018). Alum by itself or in combination with vacuum packaging has been used to reduce the incidence of crown rot without the use of fungicides for up to 28 days (Kamel et al. 2018; Siriwardana et al. 2016). Vacuum packaging by itself (Esguerra et al. 2017), essential oils (particularly basil oil) (Anthony et al. 2003) have been used effectively to reduce crown rot during cold storage (13°C).

Non-chemical treatments

Attempts have been made to manage crown rot by cultural, physical, and biological methods as alternatives to synthetic fungicides (Lassois et al. 2008). A considerable reduction of crown rot was achieved by treatment with antagonistic bacteria (de Costa and Subasinghe 1998), mycoparasites (Krauss et al. 1998), hot water dips, or rapid cooling soon after dehanding (Green and Goos 1963), as well as by CA and MA storage. Under commercial conditions, hot water treatments have not been successful in reducing crown rot and additionally, these treatments delayed ripening (Lassois et al. 2010b).

Application of a water-soluble fraction of papaya (*Carica papaya*) latex, which contains several hydrolytic enzymes including chitinase, on the cut surface of the crown approximately one hour after dehanding was shown to prevent crown rot development (Indrakeerthi and Adikaram 2011).

Resistant varieties

Most dessert bananas for export are clones that belong to the 'Cavendish' group and thus, there is little genetic diversity among commercial varieties. Breeding of bananas is difficult because triploid varieties are generally sterile. In the past, banana genetic improvement programs were mainly focused on resistance to Sigatoka and Panama diseases. Two 'Gold Finger' banana hybrids (i.e., FHIA 1 and FHIA 2) were partially resistant to infection by some crown rot fungi including *F. incarnatum*, *F. verticillioides* (syn. *F. moniliforme*), and a *Penicillium* sp. (Marin et al. 1996). In inoculation studies done by others, however, these two hybrids were more susceptible to crown rot caused by *F. incarnatum* and *C. musae* compared with varieties of 'Cavendish' (Perez Vicente and Hernandez 2002). In the latter study another genotype, FHIA-23, was identified that was more resistant. Organoleptic characteristics of FHIA hybrids that were introduced in the late 1980s, however, were not accepted by consumers because they differed from those of 'Cavendish' (Lassois et al. 2010b).

Deightoniella Fruit Speckle and Black Tip

Level of Importance

The disease is also known as swamp spot, black tip, and tip-end rot (Stover 1972). Hosts include banana and relatives in the Musaceae, e.g., *M. textilis*, *Ensete ventricosum*, as well as *Heliconia*, *Strelitzia*, and *Costus* species. It is a relatively minor disease in well-managed banana plantations and is only important during the wet season. The disease has been reported worldwide including Australia, Africa, Asia (India, and Sri Lanka), Central and South America, and Oceania. There have been sporadic severe outbreaks of Deightoniella fruit speckle and black tip in Jamaica (Meredith 1961), Cuba (Leiva-Mora et al. 2013), and India.

Symptoms

Speckles develop on fruit at all stages of maturity and are most abundant towards the tips of the fingers. They are reddish-brown to black, up to 2 mm in diameter, and have a dark green halo (Fig. 26.16). The diseased area is bordered by a narrow pale yellow or gray margin. Old lesions tend to rupture, and pale brown fungal growth may develop under humid or wet conditions. Black tip symptoms consist of a slowly advancing black lesion at the flower end of one or more fingers. The pathogen does not produce spores on fruit. The distribution of spots on the fruit suggests that the infective spores are disseminated by rain, but no studies are available on the epidemiology of this disease.

Causal Organism

Deightoniella fruit speckle is caused by *Corynespora torulosa* (synonyms *Deightoniella torulosa*, *Brachysporium torulosum*) (Jones and Stover 2019). The same pathogen also causes Deightoniella leaf speckle. Spores of *C. torulosa* are commonly present in the airborne microflora in plantations.

Management

Black tip can be managed by removing leaf litter and prunings at frequent intervals (14 to 28 days) and by improving drainage in the field to reduce humidity. The disease is worse under poor growing conditions and low plant nutrition. Plantations with adequate mulching (but not banana leaves) and sucker management have less disease. Symptoms develop before harvest, and affected fruit arriving at the packing station can be sorted out and removed from the marketing chain. Sleeving of the developing bunches may also provide control of the disease. Generally, the fungicides used to control black Sigatoka will also reduce Deightoniella fruit speckle. Protectant fungicides that include mancozeb, copper hydroxide, chlorothalonil, and banana misting oil are effective to control the disease on leaves and fruit. If Black Sigatoka is present, systemic fungicides such as the triazoles (e.g., propiconazole, fenbuconazole, tebuconazole) and QoIs (e.g., azoxystrobin) are effective against both diseases (Vawdrey et al. 2010). If the latter fungicides are used, it is important to rotate among the different modes of action to prevent the build-up of resistant strains of the fungus. No more than two applications of the same mode of action fungicide should be made before changing to another group. Under drier conditions, mancozeb can be used alone.

FIG. 26.16. Speckle lesions on banana fruit caused by *Deightoniella torulosa*. (Courtesy R. H. Stover—© APS. Reproduced, by permission, from Ploetz et al. 1994.)

Freckle Disease

Level of Importance

Freckle is a disease of banana and plantain leaves and fruit and was first reported in Hawaii in 1917 (Carpenter 1918). Subsequently, it was found in several countries in Asia, the Pacific Islands, the Americas (Wardlaw 1961), and more recently in Australasia-Oceania and South and Southeast Asia (Abayasekara et al. 1993). The disease damages the fruit skin, reducing market quality and size of the fruit. In Sri Lanka, freckle disease is a major problem in the production of high-quality bananas (Abayasekara et al. 1993).

Symptoms

Freckles appear on leaves, fruit stalks, and fruit as soon as the bunch has emerged. They are initially minute (approximately 0.25 mm in diameter), gray-brown, circular spots surrounded by a water-soaked 1.5 mm wide halo (Fig. 26.17A). As fruit mature, freckles increase in size up to 2 mm in diameter and turn dark brown or black (Fig. 26.18). The individual freckles are an aggregation of pycnidia or perithecia of the pathogen in the upper cell layers of the banana peel (Fig. 26.17C). They protrude from the host surface and feel rough to the touch. Conidia and ascospores (Fig. 26.17B,D) are released by rain splash. Freckles do not expand into coalescing lesions during ripening, but they may completely cover the fruit by the time of harvest (Fig. 26.18). Symptoms also include characteristic patterns of spotting, streaks or circular areas that coincide with the movement of rainwater and dew.

Causal Organisms

Freckle disease of leaves and fruit of bananas and plantains was originally described to be caused by *Phyllosticta musarum*. Previous attempts to isolate the fungus in culture were unsuccessful in Hawaii (Meredith 1968) and Sri Lanka (Abayasekara 1998). Based on morphological and molecular data from a global set of banana isolates (Wong et al. 2012; Wu et al. 2014), several species are now considered to be causal pathogens of freckle disease: *P. musarum*, *P. cavendishii*, *P. maculata* (syn. *G. musae*; based on Wong et al. 2012), *Guignardia stevensii* (no conidial states), and *P. musaechinensis*. Additional species have been described as endophytes of banana, and these include *P. capitalensis*, *P. cocoicola*, *P. musae*, *P. musicola*, and *G. sydowiana* (Brown et al. 1998; Photita et al. 2001, 2002). *G. musae* was originally described as the sexual state of *P. musarum* but is now associated with *P. maculata* (Wong et al. 2012).

P. musarum s. lato has a more narrow distribution in India and Thailand than previously described. *G. stevensii* has only been identified from Hawaii. *P. maculata* is present in Australia, Malaysia, Indonesia, Papua New Guinea, the Philippines and the South Pacific islands; whereas *P. cavendishii* occurs in Australia, East Timor, Hawaii, India, Indonesia, Malaysia, the Philippines, Sri Lanka, Taiwan, Vietnam, and some Pacific islands (Wong et al. 2012). *P. cavendishii* is the only species infecting 'Cavendish' AAA bananas, but like the other *Phyllosticta* species, it also infects bananas in the AAB and ABB groups.

Single or clusters of black globose to pyriform pycnidia are present in the freckles (Fig. 26.17D) and produce conidia. Spermogonia and spermatia (pycniospores) may also be found. The sexual stage of the pathogens produces ascostroma and asci (Fig. 26.17).

FIG. 26.17. **A,** Bananas with freckle symptoms caused by *Phyllosticta musarum;* **B,** asci with ascospores; **C,** transverse section through freckled skin showing ascostroma; and **D,** pycnidia and conidia of the pathogen. (Courtesy N. K. B. Adikiram—© APS)

FIG. 26.18. Severe freckle symptoms on fruit of 'Bluggoe' (ABB) banana. (Courtesy D. R. Jones—© APS. Reproduced, by permission, from Ploetz et al. 1994.)

Epidemiology

The epidemiology of freckle disease has been described for the pathogen *Phyllosticta musarum* (syn. *Phyllostictina musarum*) in Hawaii (Meredith 1968) that now is recognized as *G. stevensii* or *P. cavendishii*. The disease is common on leaves and fruit of 'Dwarf Cavendish' and other varieties in Hawaii. On fruit, symptoms may appear 2 to 4 weeks after the bunch has opened and become more severe as maturity is approached. The disease is usually confined to older leaves on affected plants. Freckled tissue contains numerous pycnidia of the pathogen, and the disease was experimentally reproduced by inoculating leaves and fruit with conidia (Meredith 1968). Spores of the pathogens are spread short distances by wind and rain, as well as dew. The fungi are spread over large distances with the movement of infected fruit, leaves, and suckers used for planting. Pathogen survival is thought to be in diseased leaves, although it is not known for how long. Conidia germinate after 3 to 6 h in a film of water on the banana peel, and appressoria form after 18 to 30 h. Penetration of the epidermis occurs by an infection hypha that develops from the appressorium 24 to 96 h after inoculation (Meredith 1968). The progressive increase in severity of freckle as fruit mature is due to

repeated infections by conidia rather than by enlargement of original lesions. Some banana clones, including 'Gros Michel', appear to be resistant to the fungus.

Infection by *P. musarum* was reported to induce several defense responses in the banana peel which include accumulation of five phytoalexins, upregulation of chitinase, β-1,3-glucanase, and phenyl ammonium lyase activity, as well as cell wall lignification (Abayasekara et al. 2013). Some of these responses in immature fruit persisted throughout the ripening period, and this may have been responsible for restricting the development of anthracnose. Thus, development of anthracnose, caused by *C. musae*, was significantly reduced in ripe *P. musarum*-infected 'Embul' (Mysore, AAB) bananas as compared to fruit without such infections.

Management

Field sanitation practices such as removing infected leaves and bagging newly emerged bunches as well as application of a fungicide starting at bunch emergence have provided good control of freckle disease (Abayasekara et al. 1993).

Lasiodiplodia Finger Rot (Botryodiplodia Finger Rot)

Level of Importance

The disease has been found on ripe fruit during storage in most banana growing areas of the world but is not very common. Severe cases of Lasiodiplodia finger rot have been reported from parts of India, Central and South America, the Philippines, and Taiwan (Ploetz 2003; Snowdon 1990; Srivastava and Tandon 1971).

Causal Organism

Lasiodiplodia finger rot is caused by *Lasiodiplodia theobromae*, a fungus well known in the tropics as a wound pathogen on banana during storage. This pathogen and several other fungal species were shown to be endophytic in banana leaves, and the possibility of these endophytes becoming pathogens has been discussed (Zakaria and Aziz 2018). Random amplified polymorphic DNA (RAPD) markers have been used to determine the genetic diversity among 12 isolates of *L. theobromae* collected from different banana cultivars in India (Sangeetha et al. 2012). There was a high degree of genetic variability with a maximum similarity index among isolates of 80% and a minimum index of 29.4%. These results warrant further taxonomic studies on isolates of the species infecting banana. The pathogen has a broad host range, and isolates from banana, cocoa, mango, and yam all infected cocoa and mango (Twumasi et al. 2014). The authors of this latter study concluded that mixed- or inter-crop systems that have been adopted in some areas will not be effective in managing Lasiodiplodia fruit rots on plantation farms.

Symptoms

Surface growth of grayish-black mycelial masses on the stalk end is characteristic for the disease (Fig. 26.19A). The infection usually starts from the persistent perianth or stem-end and progresses with a brownish-black discoloration of the peel. The pathogen invades fruit through wounds or bruises and spreads rapidly into the pulp, turning it into a black, watery mass. The infected skin becomes black and soft (Fig. 26.19A,B), is eventually encrusted due to pycnidial production, and is sometimes wrinkled. Rotting of the pulp is comparatively faster than that of the skin. In maturing or mature fruit, two-thirds or even the entire finger may be affected.

The disease may be associated with fruit spots and blemishes and causes an extensive tip rot in most banana varieties. Since the fungus grows very rapidly at temperatures present in the tropics, it may cause considerable rotting in the course of a few days. At high humidity, mycelium may be seen on the infected area (Raut and Ranade 2004).

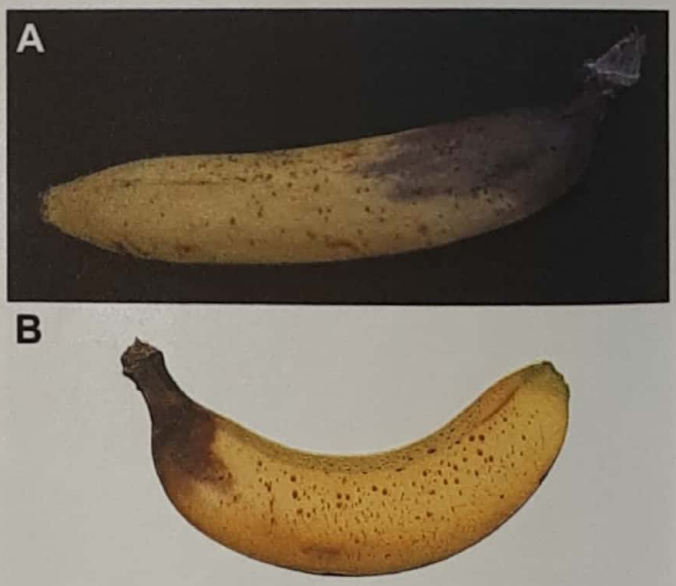

FIG. 26.19. **A** and **B**, Lasiodiplodia finger rot caused by *Lasiodiplodia theobromae* resulting from infection at the stalk end of the fruit. (A, Courtesy N. K. B. Adikaram—© APS; B, Courtesy D. Edwards, UCANR—Reproduced by permission)

Epidemiology

The pathogen is a common inhabitant of decaying banana tissues. Conidia are disseminated by wind and water, and infection occurs through tissues at the flower end of the finger or through wounds. The fungus grows slowly at less than 20°C. Optimum fungal growth and fruit decay occur between 25°C and 30°C (Ploetz et al. 2003).

Management

Stalk-end and stem-end diseases can result from mechanical damage to the pedicel by flexing during harvest and handling, creating entry points for decay fungi. Therefore, care should be taken during all handling to prevent any injuries. Removal of the affected portion of the hand reduces spread of the disease. Fruit temperature should be rapidly reduced after harvest, and storage of bunches at 10°C reduces disease incidence. Over-mature fruit should be excluded from export.

Preharvest treatments with mancozeb, carbendazim, or propiconazole, as well as dipping harvested fruit in carbendazim, propiconazole, or imazalil are highly effective (Nath et al. 2014, 2015). Additionally, sprays with garlic clove and cinnamon leaf extracts, *Pseudomonas fluorescens*, or *Bacillus subtilis*, as well as covering of bunches with blue polythene (polyethylene) reduced banana finger rot disease under field conditions (Nath et al. 2014).

Pedicel or Stalk Rot

Level of Importance

Pedicel rot is seen commonly on fruits of 'Poovan' dessert banana in some parts of India, particularly in New Delhi. The disease considerably lowers the market value and also causes quantitative fruit losses. The disease has not been reported elsewhere.

Symptoms

Infection initially takes place on the inner surface of the stalk of one or two corner fingers in the inner whorl of a hand, facing the main stem of the bunch. The symptoms appear during ripening, first as small blackish-brown necrotic lesions at the mid-point of finger stalks. They extend around the stalk and also downwards to the fingers as diffused pale brown areas. At advanced stages, the infection covers over 50% of the stalk. The lesions rarely extend upwards into the crown, and about half of the stalk towards the crown remains unaffected (Fig. 26.20). Decay of a pale color extends into the upper half of the stalk-end of fruit. Whitish mycelial growth develops mainly on the dark necrotic region. Sometimes, the infected stalks become dry, hard, and shrink. The fingers remain attached to the crown. The exposed crown often remains unaffected, even if all finger stalks are infected. Sometimes, however, the crown region may also become infected and develop crown rot (Adikaram *unpublished data*).

Causal Organisms

Musicillium theobromae (syn. *Verticillium theobromae*) has been predominantly associated with stalk rot. This pathogen also causes cigar end rot. Additionally, a *Fusarium* species has been detected in diseased tissues (Adikaram

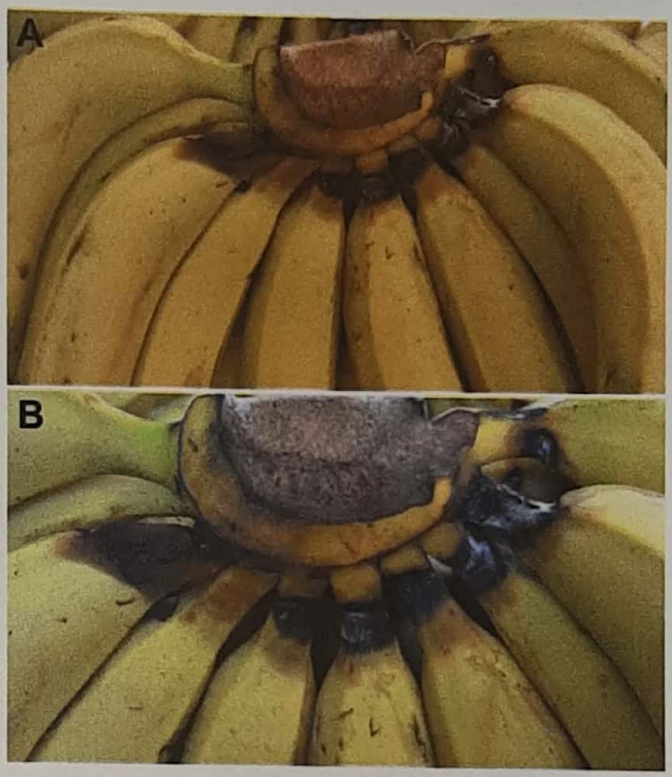

FIG. 26.20. 'Poovan' banana hands with **A,** early symptoms of pedicel or stalk rot caused by *Musicillium theobromae*; and **B,** an advanced stage with extensive fungal colonization of pedicel and crown. (Courtesy N. K. B. Adikaram—© APS)

FIG. 26.21. Thielaviopsis rot of banana fruit caused by *Thielaviopsis paradoxa*. (Courtesy UCANR—Reproduced by permission)

unpublished data). Moreover, symptoms similar to pedicel or stalk rot have also been described on 'Cavendish' bananas, and the causal agent was identified as *Thielaviopsis paradoxa* (Fig. 26.21) (Cooke et al. 2009; Jones and Stover 2019).

Critical Biological and Environmental Factors That Favor Disease

Flexing that occurs during handling of dehanded bananas after harvest can damage the pedicel and provide injuries for fungal infection. Warm and dry climatic conditions favor the development of disease.

Management

Careful handling of fruit after harvest to avoid flexing and treatment of the pedicel and exposed crown with a systemic fungicide (e.g., an imidazole) are strategies to minimize disease incidence.

Pitting Disease (Johnston Fruit Spot)

Level of Importance

Pitting disease was first reported in 1931 in England on banana fruit shipped from Brazil (Tomkins 1931). The disease was subsequently described by Johnston (1932) and, therefore, is also referred to as 'Johnston fruit spot'. Pitting disease has also been found in other parts of South America, as well as Central America, Australia, Africa (Madagascar), and Asia (Snowdon 1990). In India, pitting disease occurs sporadically but may cause significant economic losses under favorable weather conditions during epidemic years (Ganesan et al. 2017). When the rainy season coincides with a susceptible fruit stage (usually more than 60 days old after bunch emergence), pitting may occur on 50% of the fruit. The disease is most serious on 'Cavendish' AAA cultivars. The causal pathogen also causes a leaf spot (blast) on banana. Pitting disease is of minor importance when a strict management program for foliar diseases in plantations is followed.

Symptoms

Symptoms start to develop as fruit reach maturity in the field or after harvest. Lesions are minute reddish spots that enlarge, turn brown, are typically surrounded by a reddish brown or blackish ring, and the tissue within the ring becomes a sunken dark pit 4 to 6 mm in diameter (Fig. 26.22) (Jones and Stover 2019). Some authors (Kim et al. 1987), however, reported lesions up to 20 mm in diameter. The center of the pit may split, but the damage does not extend into the pulp (Jones and Stover 2019). Thus, although the fruit does not rot, there is a significant loss in fruit quality. During transport and ripening, the number of pits may increase. Small pits may also develop on pedicels and crowns, and these may result in finger drop. The pathogen does not sporulate on fruit lesions but does on leaf lesions (Jones and Stover 2019).

Causal Pathogen

The disease was originally described to be caused by *Pyricularia grisea*, but the pathogen was subsequently identified as *P. angulata* in some parts of the world (Jones and Stover 2019; Kim et al. 1987). Molecular identification of Indian isolates also confirmed the pathogen as *P. angulata* (Ganesan et al. 2017). On potato dextrose agar, the fungus typically produces pale, delicate floccose, loosely interwoven colonies that are initially creamish-white in color and later turn light brown. Conidia are three-celled and ovate to obpyriform in shape, 20 to 22 µm × 6 to 9 µm in size, hyaline to pale brown, thin-walled, and have a small protuberant hilum. They are produced solitary at the ends of denticles of conidiophores (Ganesan et al. 2017). Conidial sizes were described slightly different by others (e.g., 6.0 to 34.0 µm × 7.0 to 12.0 µm; Kim et al. 1987).

Disease Cycle and Epidemiology

The disease is most serious under high rainfall conditions when conidial inoculum is produced abundantly on senescing and dead banana leaves and bracts and is

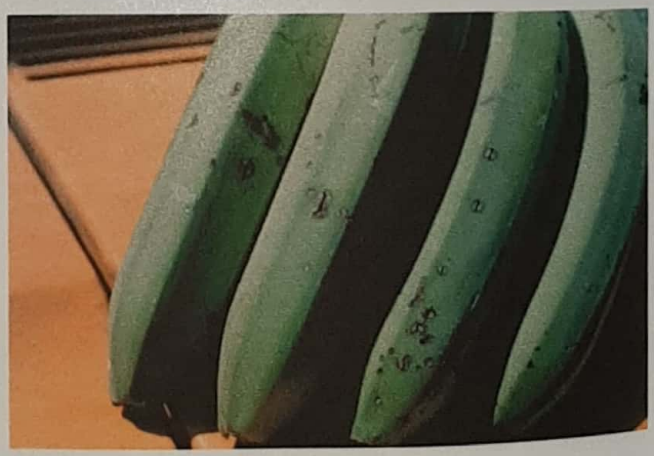

FIG. 26.22. Pitting disease (Johnston spot) lesions on banana fruit caused by *Pyricularia grisea*. (Courtesy R. H. Stover—© APS. Reproduced, by permission, from Ploetz et al., 1994.)

disseminated by wind and rain splash. Under field conditions, conidia germinate and form appressoria on fruit within 4 to 8 h at high humidity. The optimum temperature for infection is 24°C to 26°C (Jones and Stover 2019). Infections at early fruit development remain quiescent until fruit near maturity or after harvest. Unacceptable levels of pitting can develop from quiescent infections during transit and ripening. In the field, symptoms begin to develop ca. 70 days after bunch emergence. The disease does not progress during dryer environmental conditions.

Management

Dead and dying banana tissues should be removed from the plantation at regular intervals, especially during the rainy season. Before sleeving, fruit bunches can be sprayed with a protective fungicide. Benomyl, thiophanate-methyl, and especially maneb and mancozeb have shown efficacy against the disease (Guyon 1970). At the packing station, all symptomatic fruit should be culled.

OTHER DISEASES

Brown spot caused by *Cercospora hayi* has been reported from South and Central America and the Philippines. The disease occurs on peduncles, fruit crowns, and fingers. Spotting is more prevalent on the inner whorl of fingers and only occurs more than 50 days after fruit emergence. Spots (5 to 6 mm in diameter) are centered on stomata (Ploetz et al. 2003). They are light tan brown and have irregular margins surrounded by a water-soaked halo (Fig. 26.23). Lesions do not increase in size or number during ripening and lack aerial mycelium and sporulation. Dead banana leaves and weeds are primary sources of inoculum and conidia are produced within 16 h at 23 to 26°C and high humidity (Ploetz et al. 2003). The disease is no longer considered a problem because fungicides that are used for management of other foliar diseases are highly effective against *C. hayi*.

FIG. 26.23. Brown spot on banana fruit caused by *Cercospora hayi*. (Courtesy R. H. Stover—© APS. Reproduced, by permission, from Ploetz et al., 1994.)

Diamond spot occurs in parts of America and the Philippines. The disease develops on the peel of green fruit as raised yellow spots 3 to 5 mm in diameter. Because infected cells can no longer expand at the same rate as healthy cells, lesions develop a longitudinal crack. Lesions have a yellow halo, become black, and are sunken and diamond-shaped (Fig. 26.24) (Ploetz et al. 2003). A complex of fungi is responsible for causing diamond spot and include a strain of *C. hayi* that is different from

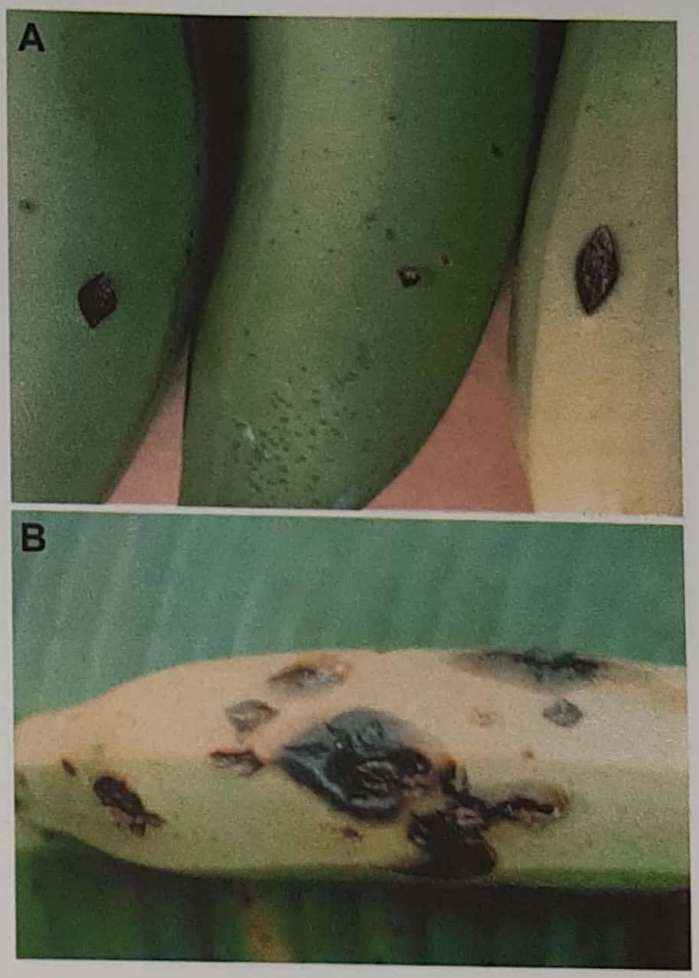

FIG. 26.24. **A,** Diamond spot on banana fruit caused by *Cercospora hayi* and a *Fusarium* sp. **B,** Severe diamond spot on maturing banana fruit in the Philippines. (A, Courtesy W. R. Slabaugh—© APS. Reproduced, by permission, from Ploetz et al., 1994; B, Courtesy R. H. Stover—© APS. Reproduced, by permission, from Ploetz et al., 1994.)

the brown spot strains and several *Fusarium* species (Fig. 26.25) (Ploetz 2003). These fungi grow on decaying leaves where they sporulate under moist conditions and are part of the air-borne microflora of banana plantations. Quiescent infections are common, and the disease develops during transit and ripening (Ploetz 2003). The disease is no longer a problem because of modern sanitation practices, pathogen exclusion, and because early fungicide applications followed by bagging of the developing bunch of fruit are highly effective management practices against the diamond spot pathogens.

Three **finger rots** of minor importance are **Dothiorella rot,** caused by *Neofusicoccum ribis* (syn. *Botryosphaeria ribis*) (Jones and Stover 2019), and **black heart,** caused by *Gibberella moniliformis* (anamorphic syn. *Fusarium verticillioides*), both reported from Israel (Chorin and Rotem 1961) and other locations (Jones and Stover 2019), and **Phytophthora rot,** caused by *Phytophthora nicotianae* var. *parasitica*, that has been reported from South Africa (Snowdon 1990). Dothiorella rot and black heart decays characteristically begin at the flower or tip-end and develop into finger rots. At high humidity, Dothiorella rot develops white to gray mycelium. Black pycnidia exuding white spore tendrils may form on the fruit epidermis (Jones and Stover 2019). Infected fingers with black heart appear healthy but when cut longitudinally show a dark brown discoloration (Snowdon 1990). Management of these two decays is obtained by sanitation practices with the early removal of dead flower parts from the developing fruit and application of fungicides such as zineb and copper (Jones and Stover 2019). Phytophthora rot begins at either the stem or blossom end of the fruit and is firm and black. Rarely a problem in the field, Phytophthora rot may develop after harvest when non-sanitized wash water is used at packing stations. Therefore, the disease can be managed by using sanitized water or by changing wash water frequently.

Fuzzy pedicel is a newly described postharvest disease of 'Cavendish' banana that was mainly observed in the United States on marketed single fingers (Tarnowski et al. 2010). Symptoms on fruit are light-gray mycelium on the surface of the cut end of pedicels or after fingers are placed in plastic shipping packages. Decay did not develop, but the disease is considered a significant aesthetic obstacle in the development of the single-finger product. Several *Sporothrix* and *Fusarium* species including *F. pseudocircinatum* that have not been reported previously from banana were obtained in isolations. Koch's postulates were successfully performed. The disease did not develop at typical shipping temperatures of 14°C but developed at marketing temperatures of 25°C (Tarnowski et al. 2010).

FIG. 26.25. Fusarium rot of banana fruit caused by *F. roseum*. (Courtesy UCANR—Reproduced by permission)

Rhizopus rot and **pink mold rot** caused by *Rhizopus stolonifer* and *Trichothecium roseum*, respectively, are decays that result from physical injuries to the peel. Generally, Rhizopus rot develops rapidly when fruit are marketed at high temperatures (Adisa 1983). White mycelium with stolons and rhizoids that produce sporangiophores and black sporangia on the fruit surface are typical signs of the pathogen on black, decayed fruit. The decay has been reported from India and Nigeria (Adisa 1983). Pink mold is a superficial decay where the pathogen produces pink spores and mycelium on the decay surface at high humidity (Srivastava and Tandon 1971).

Sooty molds and **sooty blotch** are superficial soilage problems on banana fruit during marketing. Sooty blotch has been reported from Australia and Costa Rica and is caused by *Chaetothyrina musarum*; whereas a number of fungi have been implicated to cause sooty mold and include *C. musarum*, *Cladosporium cladosporioides*, *Cladosporium atriellum*, *Leptoxyphium fumago*, and others that are found in Latin America (Jones and Stover 2019) and the Philippines (Ploetz et al. 2003). Under cool and wet environments, greenish-black or gray patches of mycelium and spores form on the concaved side of the crown at the stalk-end of the fingers. The pulp is not affected, but the fruit is stained and off-graded or not marketed. Sooty molds develop on exudates of aphids, mealybugs, and other insects that are commonly referred to as "honeydew" (Snowdon 1990). Management includes preharvest applications of insecticides and the use of insecticide-impregnated bags for covering the bunches as the fruit develop.

Squirter disease and **watery soft rot** are destructive diseases of the banana pulp. Watery soft rot is caused by *Sclerotinia sclerotiorum* and has been reported from Costa Rica (Snowdon 1990). Squirter disease is caused by *Nigrospora sphaerica* or *N. musae*; both have been reported from Australia and Asia (Jones and Muirhead 2019). Wang et al. (2017) indicated that based on phylogenetic relationships, *N. sphaerica* and *N. musae* are separate species. The disease can be a problem when single fruit are picked and can also be involved with Black-end rot

or tip rot (see above). The pathogen enters the cut stem of green fruit, and symptoms develop after removal of fruit from the ripening room. In early stages, a dark band forms on the surface of the fruit as the decay advances from the stem end, and the peel eventually becomes bluish tan (Ploetz et al. 2003). In advanced stages of decay, the pulp is liquified, and any pressure to the fruit results in the pulp squirting out (Snowdon 1990). Management strategies include harvesting bananas as hands instead of individual fingers and the use of postharvest fungicides.

Black Sigatoka caused by *Pseudocercospora fijiensis* (formerly *Mycosphaerella fijiensis*) is the most serious foliar disease of banana. The pathogen does not directly invade the fruit, however, fruit harvested from diseased plants may show buff-colored pulp, premature ripening, increased susceptibility to chilling injury, and development of abnormal flavor and aroma (Snowdon 1990).

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