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# Postharvest Diseases of Papaya

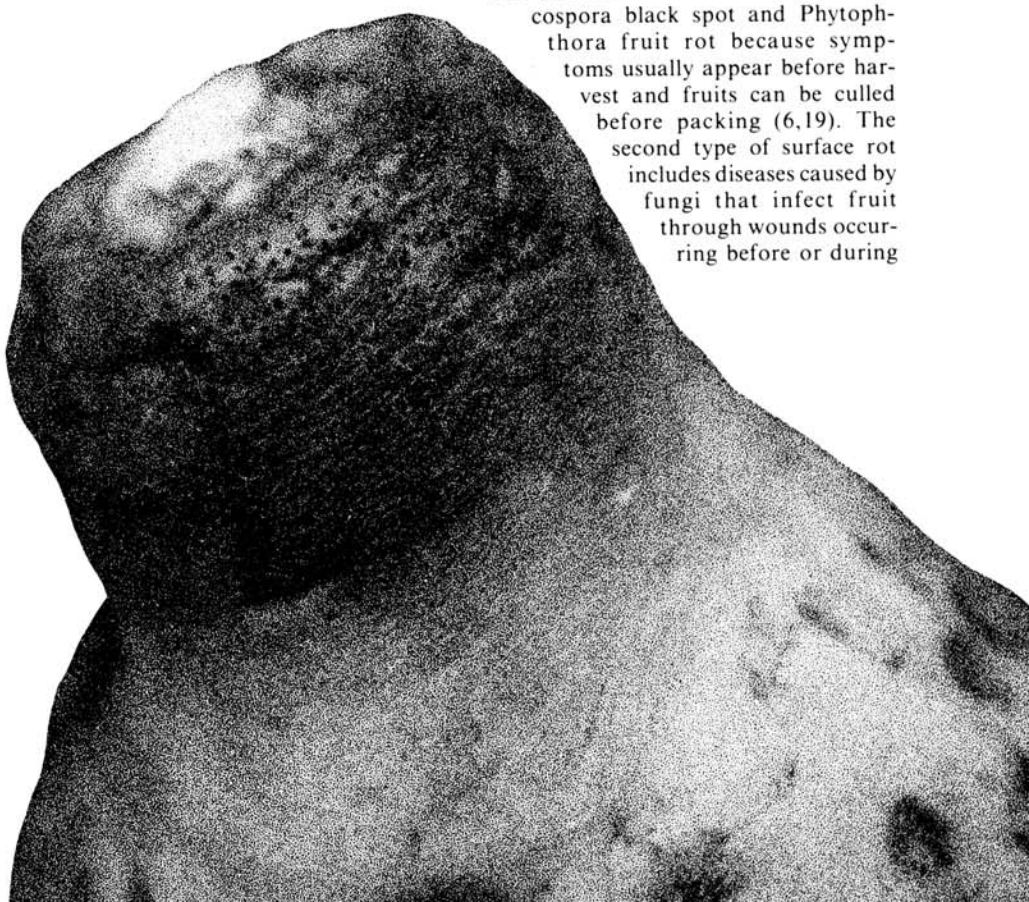
The papaya (*Carica papaya* L.), a native of tropical America, is grown throughout the tropics and subtropics for its melonlike fruit, which is usually eaten fresh. The acropetally produced fruits are clustered near the top of small (2–8 m), single-stemmed, herbaceous trees. New flowers are formed continuously; thus, a single hermaphroditic tree will have flowers and fruit in all stages of development. In Hawaii, the interval from anthesis to harvest ranges from 22 to 26 weeks. Harvesting begins about 12–15 months after seeding and continues until the trees become too tall (5–6 m) for efficient harvesting.

Orchard and postharvest diseases are very important in reducing yield and market quality of papaya and are primarily responsible for the losses that occur during shipment of the fruit (3,4,9,11). Postharvest losses of 10–40% in surface shipments and of 5–30% in air shipments are not unusual. In a weekly inspection of 100 hot-water-treated fruit from five packinghouses during 1986, 24% of 16,985 fruit in simulated air shipments were unmarketable (H. M. Couey, *personal communication*). Losses due to diseases ranged from 1 to 93%, depending on postharvest handling and packing procedures. The diseases are of three general types: fruit surface rots, stem-end rots, and internal fruit infections. The purpose of this article is to describe and illustrate the major postharvest diseases of papaya and to outline the general methods of disease control.



## Fruit Surface Rots

There are two general types of surface rots of papaya. The first includes the diseases caused by fungi that infect intact, immature, green fruit still attached to the tree. Anthracnose, chocolate spot, Cercospora black spot, and Phytophthora fruit rot are examples. Our discussion does not include Cercospora black spot and Phytophthora fruit rot because symptoms usually appear before harvest and fruits can be culled before packing (6,19). The second type of surface rot includes diseases caused by fungi that infect fruit through wounds occurring before or during



"A single isolate of *Colletotrichum gloeosporioides* can produce both anthracnose and chocolate spot, but little is known about why some lesions remain superficial while others advance into the fruit parenchyma."

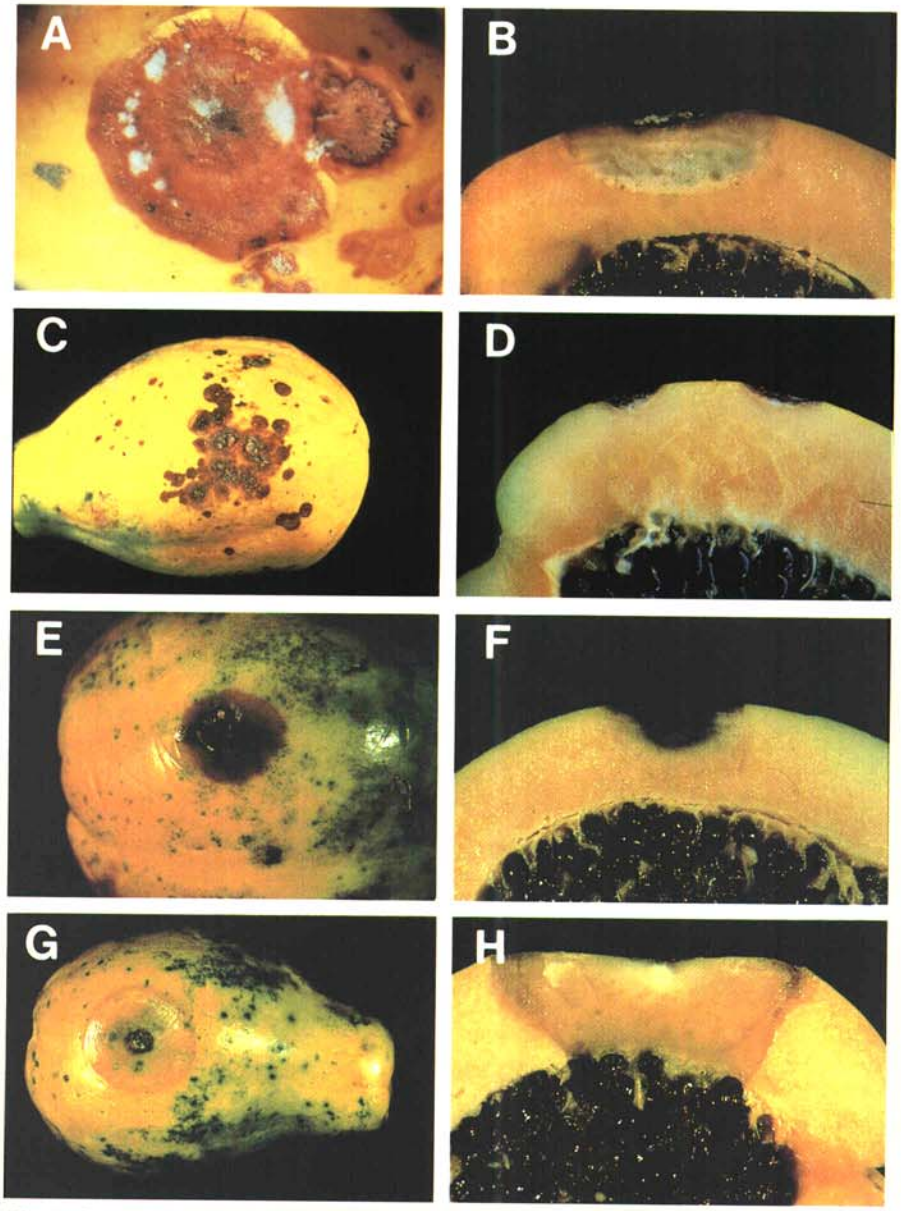



**Fig. 1. Common surface rots of papaya fruit: (A) Sunken anthracnose lesion caused by *Colletotrichum gloeosporioides*. (B) Cross section of anthracnose lesion showing grayish white discoloration of papaya flesh. Firm callose tissue forms at the border of the soft, semicircular lesion. (C) Chocolate spot lesions ranging from minute superficial spots (left) to large sunken lesions with water-soaked margins (center). (D) Cross section of chocolate spot lesions showing limited penetration into fruit parenchyma. (E) *Mycosphaerella* lesion with light brown, translucent margin. (F) Cross section of *Mycosphaerella* lesion showing a layer of firm, black tissue below the infection site. (G) Soft, translucent *Phomopsis* lesion with black pycnidia at center. (H) Cross section of rapidly expanding *Phomopsis* lesion showing progress of decay into the seed cavity.**

harvest. The organisms involved typically are weak pathogens, such as *Mycosphaerella, Phomopsis, Alternaria, Stemphylium, Fusarium*, and *Guignardia*.

**Anthracnose.** This disease is caused by *Colletotrichum gloeosporioides* (Penz.) Sacc. Infections usually are initiated in the field at early stages of fruit development, but the pathogen remains quiescent until the fruit reaches the climacteric phase (13). The fungus may penetrate the fruit surface directly with an infection peg (8). An extracellular cutinolytic enzyme is produced, enabling the pathogen to enter green, unwounded fruit. Infection can be inhibited by an antiserum to cutinase and by several organophosphorous cutinase inhibitors (14,15).

When infected fruits begin to ripen, beads of latex are exuded at the fruit surface, and small water-soaked spots appear. As the infection advances, a circular, sunken lesion with translucent, light brown margins forms. The fungus produces light orange or pink spore masses in the central portion of the lesion (Fig. 1A). Internal tissue in the infected area is firm with a grayish white discoloration that later turns brown (Fig. 1B). A layer of callose forms in the parenchyma cells, permitting the infected area to be lifted free of the fruit surface as a plug (24).

*C. gloeosporioides* was first considered to be a wound pathogen of papaya (24), but direct penetration of the cuticle and establishment of latent infections were later demonstrated in laboratory and field studies (8,13–15). The existence of latent infections explains why field sprays often showed delayed effectiveness in reducing postharvest disease; fruit lesions were not reduced until 8 weeks or more after orchard sprays had been initiated (5,22). The fungicides apparently protected fruit from new infections but did not eradicate the subcuticular quiescent hyphae within the fruit.

**Chocolate spot.** Minute, superficial reddish brown lesions are the initial symptoms of this disease (Fig. 1C). As the fruit ripens, lesions may remain superficial (Fig. 1D) or enlarge and become sunken with water-soaked margins. Anthracnose and chocolate spot have been described as separate diseases, and different symptom types were attributed to different physiological races of *C. gloeosporioides* (18). Since this initial description, M. Aragaki (*personal communication*) has determined that a single fungal isolate can produce both symptom types, but little is known about the factors that cause some lesions to remain superficial while other lesions advance deeply into the parenchyma of the fruit.

**Dry rot.** Dry rot is caused by a *Mycosphaerella* sp. that is unable to penetrate the cuticle enzymatically and thus is associated with mechanical

injuries. Small wrinkles in the fruit surface are the first symptoms, and lesions with brown, translucent margins develop later (Fig. 1E). A layer of hard tissue may form just below the infection site, separating the darkened parenchyma tissue from the epidermal portion of the papaya fruit (Fig. 1F).

The imperfect (pycnidial) stage of *Mycosphaerella* sp. was previously designated *Ascochyta caricae* Pat. (18,19), then *A. caricae-papayae* (Tarr) (7), but the fungus later was transferred to *Phoma caricae-papayae* (Tarr) Punith. (23). Both ascospores and conidia are capable of infecting wounded fruit surfaces (7). In some areas of the tropics, notably India and Brazil, fruit surface lesions are common; in Hawaii, the pathogen usually causes a stem-end rot.

**Wet rot.** Fruit lesions caused by *Phomopsis* sp. occur infrequently but cause extensive damage (19). The entire infected area is soft and translucent, and black pycnidia may form at the central portion of the lesion (Fig. 1G). A wet rot proceeds rapidly from the surface into the fruit cavity (Fig. 1H), and the infected tissue can be lifted free from the rest of the fruit. This fungus also is frequently associated with stem-end rots of papaya.

**Alternaria fruit spot.** This disease is characterized by circular to oval black lesions that become covered with black spore masses of *Alternaria alternata* (Fr.) Keissler (Fig. 2A). Lesions are usually restricted to the surface of the fruit and do not cause extensive rotting of the parenchyma tissues. Refrigeration during surface shipment enhances disease development, and symptoms rarely develop on unrefrigerated fruit.

Alternaria fruit spot previously was a major disease on fruit grown in papaya orchards in relatively dry areas of Maui (5). *Alternaria* was found to colonize senescing petioles, and large numbers (13,700–36,900 spores per fruit) were found on fruit surfaces at the time of picking; thus, petioles appeared to be the major inoculum source (I. W. Buddenhagen, *unpublished*). Infection was reduced with biweekly orchard sprays (5) and by postharvest hot-water treatments (I. W. Buddenhagen, *unpublished*).

**Stemphylium fruit spot.** Small, round, dark brown lesions are early symptoms of *Stemphylium* infections. Lesions later enlarge and develop reddish brown to purple margins (Fig. 2B). Dense, dark-green spore masses cover the lesions, and a white to gray mycelium forms at the lesion center. The pathogen, *Stemphylium lycopersici* Yamamoto (= *S. floridanum* Hannon & Weber), is primarily a wound pathogen and usually occurs on fruit damaged by heat or refrigeration (10,17).

**Fusarium rot.** Small dry lesions develop on the fruit surface and are later covered by a white, rather compact mycelial mat (Fig. 2C). The pathogen

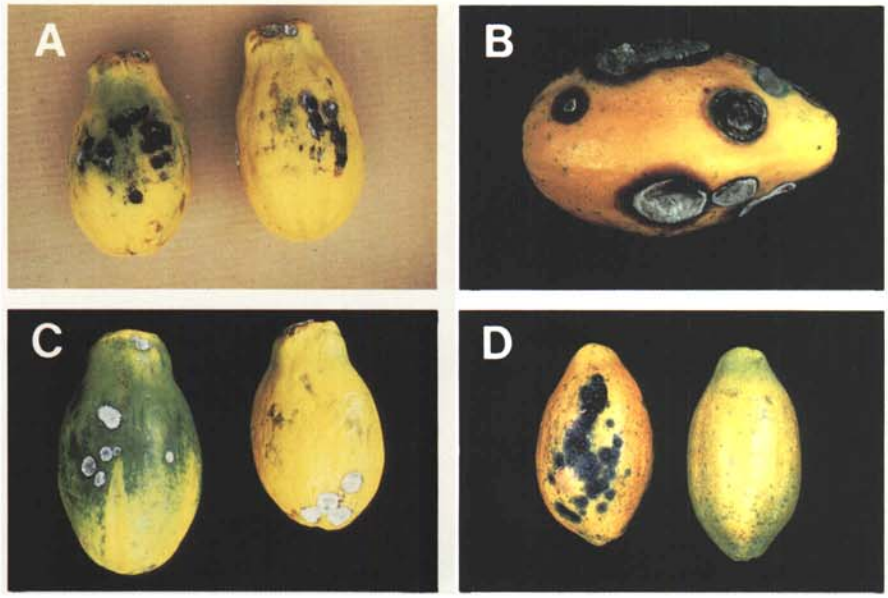



**Fig. 2. Infrequent surface rots of papaya fruit: (A) Lesions caused by *Alternaria alternata* showing black spore masses. (B) *Stemphylium* lesions characterized by reddish brown margins and grayish white mycelium. (C) Dry fruit rot caused by *Fusarium solani* in which compact white mycelial mats form over the lesions. (D) Greenish black lesions associated with *Guignardia* sp.**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was identified as *Fusarium solani* sensu Snyd. & Hans. (19). The disease occurs sporadically on fruit after harvest.

**Guignardia spot.** Sunken, greenish black lesions occasionally observed on fruit surfaces (Fig. 2D) are associated with *Guignardia* sp. (18). Little is known about this fungus on papaya. This disease was frequently seen when papayas were preheated in hot water (42 C) for 40 minutes during postharvest fruit fly disinfestation, but the incidence subsided after the preheating time was reduced to 30 minutes.

## Stem-end Rots

Stem-end rots of papaya occur when fungi invade the severed peduncle after harvest. Spores may also invade through crevices between the peduncle and the papaya flesh or invade through small wounds that occur at harvest. Stem-end rot initially was attributed only to *Ascochyta* sp. (18). Later, other genera, including *Botryodiplodia, Phomopsis*, and occasionally *Fusarium* (19), were identified in diseased tissues. We now know that several other fungi, including *A. alternata, S. lycopersici, C. gloeosporioides*, and *Mycosphaerella* sp. (5,7,10), also may cause stem-end rots when inoculated alone or in various combinations. The most common stem-end rots are described and compared here.

Stem-end rot caused by *Mycosphaerella* sp. is initially characterized by a translucent zone around the peduncle. At early stages, only a slight browning of the peduncle is apparent as the fungal hyphae invade the vascular tissue. As the infection advances, the lesion margin remains translucent while the remaining infected tissue becomes black, wrinkled, and dry (Fig. 3A,B). White mycelium forms at the stem end at an advanced stage of infection.

Infections caused by *Botryodiplodia theobromae* Pat. have a wide margin of water-soaked tissue (Fig. 3C) and a rough surface caused by an irregular pattern of erumpent pycnidia (19). Pockets devoid of parenchyma tissue form in the infected area and later become filled with mycelium. In longitudinal section, the infected vascular tissue has a bluish black discoloration resembling infections by *Mycosphaerella* (Fig. 3D). In contrast, infections caused by *S. lycopersici* are characterized by a reddish brown discoloration of the parenchyma tissue, and margins of diseased and healthy tissue are bright red to purple.

Tissue infected by *Phomopsis* sp. first wrinkles, then becomes translucent and light green to yellow (Fig. 3E). A band of water-soaked tissue advances very rapidly from the infection site toward the fruit cavity (Fig. 3F), and the infected portion often can be lifted free from the

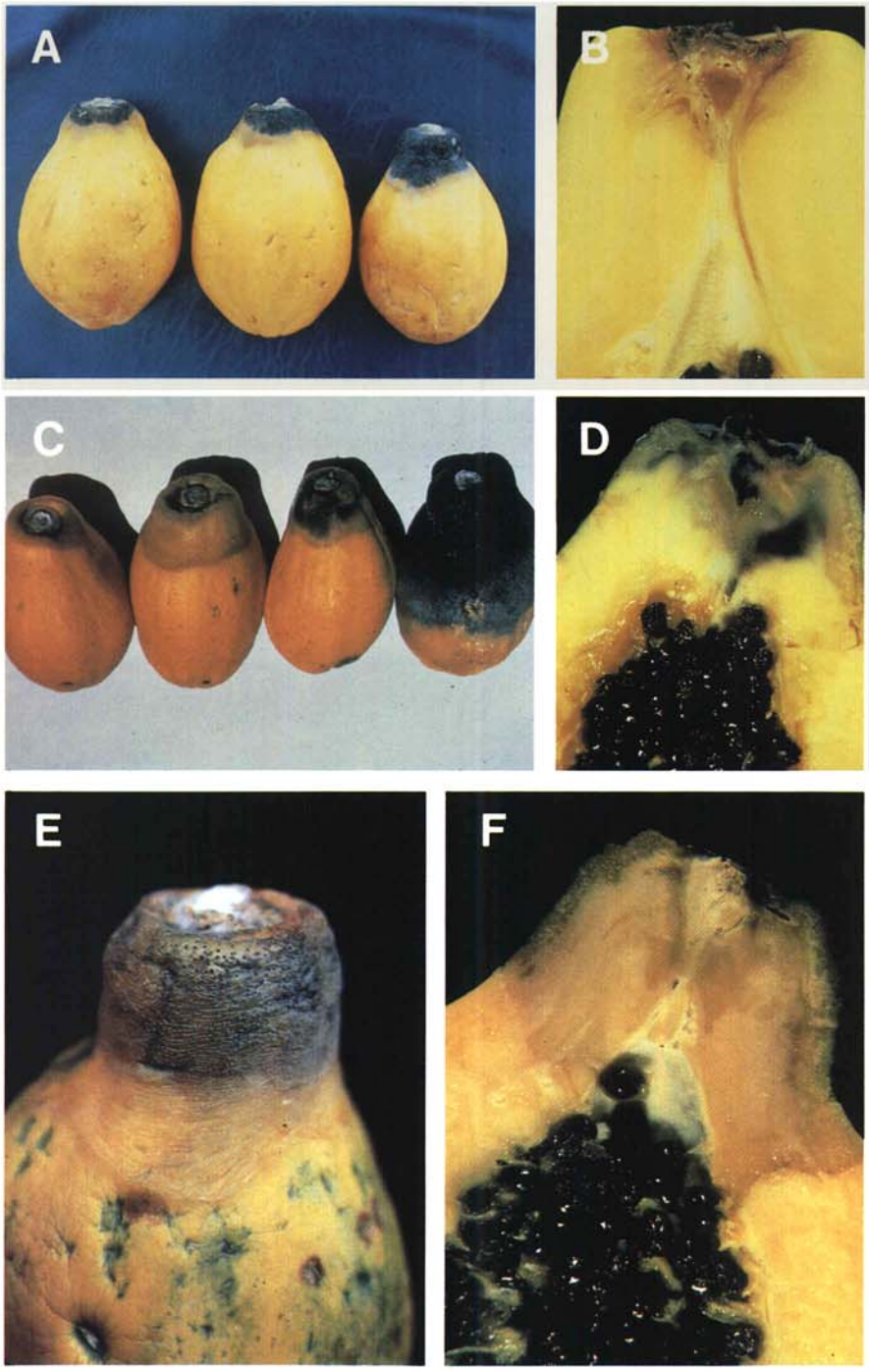


**Fig. 3. Stem-end rots caused by various fungi: (A) Stages of stem-end rot caused by *Mycosphaerella* sp. showing black, infected tissues with brownish, translucent margins. (B) Penetration of vascular bundles by *Mycosphaerella* sp. (C) Successive stages of infection by *Botryodiplodia theobromae*. (D) Longitudinal section of stem end showing bluish black discoloration characteristic of infections by *Mycosphaerella* sp. and *B. theobromae*; fungus has penetrated the vascular bundles. (E) Wrinkled stem-end tissue characteristic of infection by *Phomopsis* sp. (F) Phomopsis decay showing soft, light-brown, translucent parenchyma tissue.**

rest of the fruit. Pycnidia usually form on the fruit surface of advanced infections.

Another common and severe postharvest disease is caused by *Rhizopus stolonifer* (Ehr. ex Fr.) Lind, which at times is the most destructive of the postharvest pathogens. The fungus invades through wounds and rapidly rots the entire fruit, leaving intact only the enclosing cuticle. When the fungus breaches the cuticle, infected fruits become covered by a mass of coarse gray mycelium with black macroscopic sporangia (Fig. 4). In contrast to the other pathogens, *R. stolonifer* is capable of spreading quickly to other fruit in a container, and an entire carton of fruit may be rotted within a few days. Nevertheless, with careful sanitation and avoidance of wounds, the disease may be kept under control.

## Internal Fruit Infections

Internal "smut" is a term for fungal spore masses that fill the fruit cavity. The disease occurs sporadically when the blossom end of the fruit is not completely sealed (Fig. 5). Fungi such as *Cladosporium* sp., *Penicillium* sp., and *Fusarium* spp. may enter through the narrow passage leading into the seed cavity and destroy the seed as well as the surrounding tissue. Infected fruits usually have a small hole at the blossom end, often with a light green halo. Fruit with such symptoms usually ripen unevenly and are culled before packing operations. The anatomical disorder apparently is of genetic origin, and careful seed selection usually is sufficient to circumvent this problem. Seed is collected only from trees in which the disorder does not occur.

Two bacterial diseases also cause sporadic damage of papaya fruit. External symptoms are absent, and the diseases can be observed only after fruits are cut open. Purple-stain, caused by pigment-producing strains of *Erwinia herbicola* (Loehnis) Dye (21), is characterized by violet to purple streaks in the vascular tissue and latex ducts surrounding the seed cavity (Fig. 6A). The parenchyma tissue becomes translucent and later rots, producing an offensive odor and taste.

Internal yellowing disease, caused by *Enterobacter cloacae* (Jordan) Hormaeche & Edwards, is a similar bacterial disease. The infected fruit flesh is translucent with a bright yellow to lime-green discoloration (Fig. 6B). *E. cloacae* has been isolated from papaya fruit, hot-water treatment tanks, papaya blossoms, and the gut and crop of the oriental fruit fly (*Dacus dorsalis* Hendel); bacterial strains isolated from these sources reproduced internal yellowing disease (K. Nishijima, *unpublished*). Because of the sporadic occurrence and lack of external symptoms, modes of

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**Fig. 4. Watery fruit rot caused by *Rhizopus stolonifer*. Black masses of sporangia cover the surface of an infected fruit.**

**Fig. 5. Internal "smut" caused by *Cladosporium* sp.**

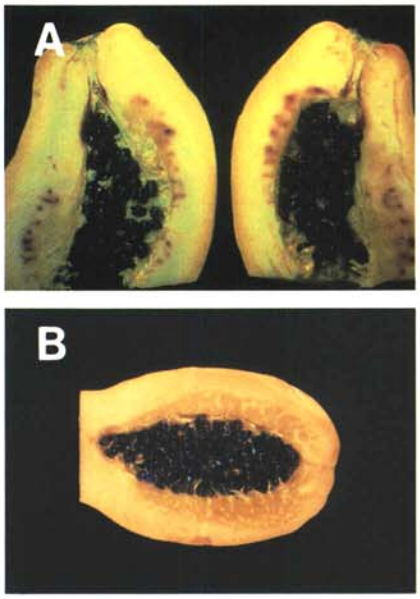


**Fig. 6. Internal fruit rots caused by bacteria: (A) In purple-stain fruit rot caused by pigment-producing strains of *Erwinia herbicola*, latex ducts and vascular tissue surrounding the seed cavity are discolored. (B) Internal yellowing disease caused by *Enterobacter cloacae*.**

**Fig. 7. Effect of hot-water treatment on papayas harvested from the same field and selected for uniformity. Fruit on the left were treated with hot water for 30 minutes at 48 C and show no stem-end rot, although ripening is slightly retarded. Fruit on the right were not treated.**

infection and spread of these diseases are poorly understood.

## General Measures for Control of Postharvest Diseases

**Measures in the field.** Because most postharvest diseases begin in the field, control measures must also begin in the field. Reduction of inoculum and application of protective fungicides are the most effective approaches to disease control, and various chemicals have been tested for this purpose (16). For papaya, best control is achieved by frequent sprays of mancozeb or chlorothalonil, beginning at first fruit set, about 6–8 months after planting. The entire fruit and flower column is sprayed once every 7–14 days during rainy periods and 14–30 days during dry conditions. A surfactant is added to the spray for more efficient coverage, and a sticker is also used whenever rainfall of 25 mm per week or more is anticipated.

Removal of all infected and discarded fruit is essential for reducing the inoculum level of postharvest pathogens. Although removing senescing leaves from the field is not practical, they should be removed from the tree on a regular basis to provide an unobstructed path between the sprayer and the fruit column and because such leaves serve as a source of inoculum in the immediate vicinity of the fruits.

Infection by fungi that cause stem-end rot occurs through and around the severed peduncle sometime after picking (7). Field sprays substantially reduce the inoculum level but do not eliminate stem-end rot infections (5). Adequate control is achieved only when field sprays are combined with postharvest hot-water or fungicide treatments (1,2,11,12).

**Resistant varieties.** Kapoho Solo, the major export cultivar, shows no substantial resistance to the described postharvest diseases. It continues to be grown because it is well adapted to the main papaya-growing area on the island of Hawaii and because it has superior horticultural and marketing qualities. Sunrise Solo, the other export cultivar, has some resistance to infection by *C. gloeosporioides* (20). Its resistance is sufficient to preclude spraying for these diseases except in the wettest areas, but it is highly susceptible to blight caused by *Phytophthora palmivora* Butl.

**Measures after harvest.** Hot-water immersion or spray followed by application of fungicides in wax substantially reduces postharvest decay even for extended storage during surface shipment (3,9,11,12). Hot water treatment also retards ripening (Fig. 7). According to federal quarantine regulations, papayas for export to the U.S. mainland must be less than one-fourth ripe and must be disinfested for fruit flies within 18 hours of harvest with a double hot-water immersion treatment consisting of an initial 30-minute immersion at 42 C, followed by a 20-minute immersion at 49 C. The double-dip treatment provides excellent control of postharvest diseases of papayas when coupled with regular field fungicide sprays.

Excessive heating or delayed posttreatment cooling can inhibit the normal ripening process or scald the fruits, allowing rapid colonization and serious postharvest disease problems. Storage and shipping temperature for papayas should be at or near 10 C, with as little fluctuation as possible.

Daily sanitation of the packing line and the water tanks is necessary to minimize reinoculation of the hot-water-treated fruits and particularly to reduce *Rhizopus* infections. Equipment and containers may be disinfested with quaternary ammonium compounds or calcium hypochlorite. Chlorine levels in cold water tanks are maintained at 70–100 ppm at pH 6.0–7.5 to ensure sufficient chlorine to kill contaminating organisms.

A number of experimental chemicals have been tested as postharvest fungicide treatments to supplement orchard sprays (1,2,16). Either benomyl or thiabendazole is effective as a postharvest treatment (2). The most common postharvest treatment for surface- and air-shipped papaya is thiabendazole applied at 4–8 g per liter with a carnauba wax (3,11,12).

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