Papaya Diseases and Integrated Control

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Abstract: Diseases are a significant limiting factor for papaya production. The nature and frequency of these diseases depend on local conditions and effective management depends on a thorough knowledge of the pathogen, host plant, environment, and their interaction. The precise identity of the causal agent is of paramount importance, and disease management options must be economical. Assessment of disease incidence or severity, and fruit loss are the key factors in determining the economics of disease management. In general, disease management strategies involve different practices that include plant resistance, and prophylactic and curative measures. The papaya diseases related have diverse etiologies, divided into those with biotic (infectious) and abiotic (noninfectious) etiologies that affect the fruit and those affecting the plant. We have attempted to emphasize procedures for diagnosis and control with detailed information on each. Descriptions of pathogens are provided in sufficient detail to assist professional diagnosticians in making accurate diagnosis. Fungicides used to control many of the diseases described in this chapter are mentioned for information proposes only. Legal restrictions and regulations of fungicide use vary among countries, and regulations within any country are subject to change over time. The information reported has been extracted from the available scientific literature and the authors' experience, and we hope that this publication will provide a helpful reference to growers, students, and professionals working with papaya.

1. Introduction

Diseases of papaya stand out economically because their presence can cause severe economic losses in production, sale, and exportation of fresh fruit that may reach in some cases 100%. In Brazil the principle diseases are viruses (Papaya ringspot virus and "meleira") in the field, and anthracnose and peduncular rot, in postharvest. Foliar diseases, such black spot, leaf spot and greasy spot, can also cause significant damage to fruits and reduce their commercialization when not controlled adequately.

In general, the importance of diseases of papayas varies with the region where they are grown, in function of climatic conditions; management of the orchard (cultural management practices) vector populations, inoculum density and destination of production for internal or external markets.

Post-harvest diseases are principally of three types: superficial rots, peduncular rots, and internal fruit rots, that reduce the quality of fruits and cause severe losses, reaching in some cases more than 90% depending on the conditions of harvest, transport and packing, and that may make totally impossible sale in importing markets.

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2. Fungal diseases

2.1 Anthracnose

Anthracnose is considered one of the principle postharvest diseases of papaya, occurring in all production regions in the world, having been noted to cause large losses in Brazil, Hawaii (USA) and Mexico. The fungus infects various parts of the plant but its greatest importance occurs in fruits, which become unfit for commercialization.

The latent infection, not detected at harvest, develops after harvest principally during transport of the fruits for export or local markets.

The absence of control measures and use of inappropriate postharvest procedures can result in production of up to 100% diseased fruits from some orchards.

2.1.1 Etiology

The causal agent of anthracnose, the fungus *Colletotrichum gloeosporioides* (Penz.) Penz. Sacc. in Penz., (teleomorphic phase *Glomerella cingulata* (Ston.) Spauld. & Schrenk., is a pathogen common in diverse tropical fruit plants, having been reported in the teleomorphic form in fruits and leaves (Ram, 1984; Costa *et al.*, 2001; Holliday, 1980).

The fungus forms subcuticular and subepidermal acervuli, separate or confluent with pinkish to orange conidial masses that cover the lesion centers, with setae, conidiophores septate, pale brown with hyaline conidia with obtuse ends or ellipsoidal with a rounded apex and a narrow, truncate base, aseptate, more or less guttulate and cylindrical.

Costa *et al.*, (2001) reported the occurrence of the perfect stage (*Glomerella cingulata*), with perithecia on various parts of the host (dead leaves and fruits), solitary or aggregated, globose to obpyriform, dark brown to black, and asci with 8 spores, clavate to cylindrical, with ascospores narrowly oval to cylindrical to fusiform.

It has not been possible to separate isolates of the fungus obtained from different symptoms frequently observed in the fruits and leaves of the papaya plant by morphological characteristics, however, the existence of ecotypes of *C. gloeosporioides* in relation to temperature was shown in other pathosystems in spite of the similarity of the DNA (Estrada *et al.*, 2000).

The occurrence of a new anthracnose disease on papaya fruits caused by *Colletotrichum capsici* was found at Miyako Island, Okinawa prefecture, located in the south-western part of Japan in 1994. These lesions were initially water-soaked, light brown spots. They gradually enlarged, became sunken and turned blackish brown. Numerous acervuli of anthracnose were produced on the fruit lesions. Conidia were falcate, and abundant black setae were found (Yagushi *et al.*, 1998).

2.1.2 Symptoms

The fungus may initially be established in flowers, penetrating through stigmas and scars left by petals and principally by superficial wounds on these tissues.

The greatest damage is on fruits where the fungus causes small, irregular, watersoaked spots that later enlarge and become dark in color. Infection by *C. gloeosporioides* can occur in any stage of development and remain quiescent until the fruits become mature, penetration being direct, through an infection "peg" or by wounds (Chau and Alvarez, 1983a).

When infected fruits begin ripening, small drops ("beads") of latex exuded on the surface are formed. The lesions in the fruits are round and deep, and can attain up to 5cm diameter. With development of the lesions, a pink growth can be observed, arranged in concentric rings of gelatinous aspect that later turn dark, consisting of the reproductive structures of the fungus. The internal tissue of the infected area is firm, with a white-grayish discoloration that turns brown (Fig.1).

The lesions spread easily to healthy tissues, leaving depressions in the fruit,

Figure1: Anthracnose lesion caused by *C. gloeosporioides* on papaya fruit, with the presence of a typical gelatinous mass of pinkish orange color.

and can coalesce forming a large irregular to circular lesions area on the surface of the fruit, up to 10 mm in diameter, sharply defined, and occasionally depressed. In the absence of control and/or under highly favorable climatic conditions, the presence of these lesions can be observed in fruits in the initial stages of maturity, including in the field.

Mature or damaged fruit is more susceptible to infection than immature fruit. Leaf infection does occur and initially appears as small, watersoaked spots of irregular shape. These lesions become brown, with gray-white centers that often fall out. The black, pin-cushion-like fruiting structures of the pathogen can be observed in these old lesion centers (Simone, 2002).

On petioles, dark lesions are formed with abundant formation of acervuli. The perithecia of the teleomorphic stage of the fungus also develop on the petioles (Fig. 2). Lesions on leaves are less frequent and when they occur, they are circular, with irregular borders and grayish centers, where “black spots”, which are the fruiting bodies (structures) of the fungus can be observed. In certain conditions the fungus does not penetrate deeply in the flesh of the fruit, In these cases superficial lesions of yellow brown

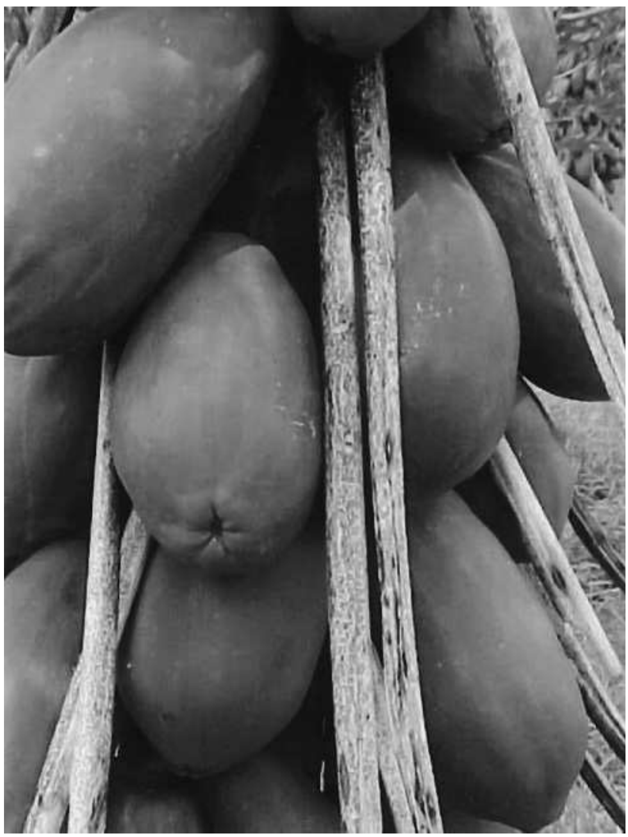

Figure 2: Lesions of anthracnose on petioles of papaya leaves with formation of perithecia of *Glomerella cingulata*, which serve as sources of inoculum of *C. gloeosporioides* underfield conditions.

color, often with a water soaked appearance on the margins occur, which are referred to as “chocolate spot” (Fig. 3).

2.1.3 Epidemiology

The infection generally occurs in the field, during the initial stages of development of the fruits, the pathogen remaining quiescent until the maturation phase of the fruit, when the symptoms become visible. Fruits and senescent leaves infected by *C. gloeosporioides*, principally the petioles, both on the plant as well as those that fall on the ground, represent important sources of inoculum of the pathogen. The production of ascospores in the petioles of dry leaves has been frequently observed but the importance of the sexual phase in the cycle of the disease still is not clear. In the field, the production of conidia occurs in large quantity principally in the petioles of senescent leaves and in mature fruits, these being considered the source of inoculum. However, additional studies to understand their epidemiological importance are required. Drops of rain and splashes of irrigation water are the principle sources of dissemination of inoculum of the pathogen.

When the conidia germinate (6-8 hours), they form appressoria (10-12 hours), on

Figure 3: Symptoms of chocolate spot on papaya fruit cv. Sunrise Solo.

the surface of the fruits and by enzymatic action penetrate the cuticle, the hyphae remaining subcuticular in a latent state until the beginning of maturation of the fruits, when growth resumes and symptoms of the disease appear.

Conidia dispersed in the initial phases of development of the fruits have less chance of survival than those that are deposited on developed fruits and that remain and form the quiescent infections or infect the fruits after harvest.

Termination of quiescence of the fungus is related to the action of antifungicidal compounds (Prusky, 1996), or to the production of ethylene in the maturation of the fruits that favors the germination and formation of appressoria of *C. gloeosporioides*

(Dickman, and Alvarez, 1983; Dickman et al, 1982 and 1983; Flaishman and Kolattukudy, 1994).

The mechanism of quiescence appears to represent a case of co-evolution of the pathosystem, bringing advantages to both the pathogen and host in the ecosystem. To the pathogen, it represents adaptation to the physiology of the host, and to the host, resistance to the pathogen, paralyzing the evolution of infection of the tissues of the fruit. This has the advantage of making possible development of the seeds, with liberation of the seeds occurring after maturation of the fruit and disintegration of the pulp by the pathogen and saprophytes (Arauz, 2000).

Climatic conditions that favor the incidence of anthracnose are temperatures near 28ºC, ranging from 20 to 30ºC, and relative humidity of the air more than to 95%. The conidia require water in the free state to germinate and are liberated from the acervuli only when the relative humidity is greater than 95% (Quimio, 1973). Severity of the disease depends on environmental conditions, being less severe in dry periods and lower temperatures. In the north of the Espírito Santo, Brazil, these conditions normally occur from April to August. In research conducted by *Incaper,* where monitoring of the disease in fruits during the period of July 1997 to March 1998 was conducted, the incidence of anthracnose was greater than 70% from September to March. The greatest incidence was reported in the months of November (94,44%), December (97,22%) and January (100%), the months of November and December having the greatest rainfall, with precipitation of 292 and 194mm, respectively (Costa *et al.*, 2002; Tatagiba *et al.*, 2002).

2.1.4 Control

The management of anthracnose in the field should begin with the choice of the field (planting) site, taking into consideration the prior history (old plantings), avoidance of excess humidity and conditions that favor the development of the disease, as well as cultural practices, the reduction of inoculum, chemical control and genetic resistance. Measures adopted during the phases of production and post-harvest processing of the fruits (care in handling, cleanliness of packaging and of the environment, control of storage temperatures, use of heat treatments and chemical control) influence the incidence and severity of the disease and when well managed, significantly reduce losses (Ventura, 1995, Ventura and Costa, 2002).

2.1.4.1 Cultural practices

i. Balanced fertilization and management of irrigation: Plants with unbalanced nutrition and water stress become predisposed to an increase in the severity of anthracnose. Studies conducted by *Incaper* verified that doses of boron and calcium greater than the amount required by plants, 0.77g of B and 50g of Ca/plant, contributed to an increase of approximately 70% in the incidence of anthracnose in fruits compared to doses of 0.06g of B and 2.5g of Ca/plant (Tatagiba *et al.*, 1998a). The incidence in fruits reached 100% with a content of boron in the plant of 50mg/kg; the average content generally required being 23mg/kg.

In another experiment conducted in the northern region of Espírito Santo by *Incaper*, where irrigation by microaspersion was utilized, a negative relation between the amounts of water utilized and the incidence of anthracnose was observed, demonstrating the possibility of irrigation management for control of the disease. The amount of 120mm reposition the evapo-transpiration (class A pan evaporation), besides contributing to the greatest production of fruits, was where the least incidence of anthracnose was observed. In the check without application of water, the incidence of the disease in fruits reached 100% (Tatagiba *et al.*, 2001).

ii. Elimination of sources of inoculum: For cleanliness of the orchards, removal of mature fruits and especially infected fruits and senescent leaves both on the plant and those fallen on the ground, is an important procedure. Elimination of infected fruits and leaves can contribute to the reduction of the initial inoculum and to the incidence of anthracnose in the fruits.

iii. Handling of the fruits: Papaya fruits should be harvested when the color of the skin changes from dark green to light green and when one yellow streak begins development from the base upwards. Fruits in this condition will continue to ripen normally after harvest. Those fruits harvested before this stage will fail to show complete ripening, and those harvested after are more susceptible to damage and bruising during handling. After harvest, the fruit are placed in single layers into shallow, light colored field crates, preferably containing a foam layer for cushioning. All stems should be trimmed after harvest to ensure that no stem to fruit rubbing occurs during transport to the packing facilities. Fruit should never be thrown or dropped. Field crates containing the fruit should be left in shaded conditions protected from the sun and rain while awaiting collection for delivery to a packing facility.

Avoid to the maximum causing injuries to the fruits during harvest, transport and storage. These injuries become ports of entry not only to anthracnose but also to other post-harvest diseases. Putting many fruits in the boxes of harvest in the field should also be avoided, the maximum being two layers.

Grading should be carried out as soon as possible after harvest, and fruit left under ambient conditions to continue ripening or placed at 10° to 12°C for cooling and storage. On arrival in the packing facility, fruit should be washed in water to remove latex and debris, then treated for postharvest disease control. Washing, treatment and grading can be carried out using mechanized or manual systems, depending on the volumes of fruits. Storage of unripe papaya below temperatures of 10°C will result in chilling injury. The symptoms are indicated by surface pitting, discoloration of the peel and flesh, incomplete ripening, poor flavor and increased susceptibility to disease.

iv. Sanitation of installations and equipment: Disinfestation of containers, equipment and storage installations is a practice very important to eliminate sources of inoculum. In the packinghouse a solution of chlorine (70-100 ppm) should be used for the required cleaning, and the fruits from the orchards should be washed and disinfested with a similar solution of chlorine adjusted to 70-100 ppm and pH of 6.0-7.5.

2.1.4.2 Chemical control in the field

Anthracnose can be controlled or reduced by pre-harvest sprays. Although symptoms of anthracnose are observed principally during the phases of transport and storage, control of the disease should begin in the field, with sprays applied during the period of fruiting, reaching flowers, and new and more developed fruits, combined subsequently with the treatments of post-harvest. The interval of application depends of the predominant climatic conditions of the region of production, varying from 7 to 28 days, being generally of 14 days. The fungicides most utilized are chlorothalonil, mancozeb and thiophanate-methyl. The best control is achieved by protective fungicides (chlorothalonil and mancozeb) sprays on the entire fruit and flower column, every 7-14 days during rainy periods and 14-28 days during dry conditions. In the north of the Espírito Santo, Brazil, bi-weekly and monthly applications of these fungicides, in a period of a year, provided levels of control of anthracnose that varied from 16 to 73%, 12 to 51% and 8 to 48%, respectively (Tatagiba *et al.*, 1997 and 1999). These results are similar to those obtained in other production regions of the world, where mancozeb was most effective, but no different statistically to captan and chlorothalonil (Solano and Aruz, 1995).

Few fungicides are registered officially for use on papaya. The use of fungicides on the planting should obey national legislation and also the demands of the importing countries, which normally follow the norms of the Codex (FAO/WHO) and of the EPA (USA).

Dithiocarbamate fungicides, including mancozeb, are effective in the control of the disease, but owing to the production of ethylenethiourea (ETU), are considered restricted in some countries, principally in the United States. A methyl dithiocarbamate like ferbam that does not produce ETU may be an alternative control but requires research to prove its effectiveness in the control of anthracnose.

Other fungicides like benomyl and prochloraz have been used in schedules of control of anthracnose, alternating with protective fungicides. The resistance of *C. gloeosporioides* to benomyl has been detected in the north of Espírito Santo (Ventura and Balbino, 1995) compromising the utilization of this fungicide. There are still no reports of resistance of *C. gloeosporioides* to prochloraz, but resistance in other fungi has been reported, and considering that this fungicide can be used in post-harvest treatment of fruits, its generalized use is not recommended in applications in the field in order to minimize the risk of selecting populations of the pathogen resistant to the fungicide. Other fungicides with potential to control the disease are being evaluated experimentally, but still have no official registration for papaya. As papaya is a sensitive plant, some fungicides can cause phytotoxicity, manifested by injuries to the leaves, on the skin of the fruits and, in some more extreme cases, in slowing of development, or even the death of plants. There have been reported occurrences of phytotoxicity with the fungicides pyrazophos (severe defoliation), and dinocap (causing foliar lesions) (Marin and Gomes, 2000). Fungicides of the triazol group can also cause phytotoxicity in plants and fruits of papaya when used in high doses (Table 1).

2.1.4.3 Post-harvest treatment

Beyond sprays in the orchard, post-harvest treatment of the fruits should proceed in complementary form, as a means of controlling the quiescent mycelium in the fruits and

Table 1: Evaluation of fungicides used by growers to control papaya diseases in relation to phytotoxicity to papaya plants.

	Active Ingredient	Commercial Formulation	Dose (% p.c.)	Injury to the Leaves [/1]
1.	azoxystrobin	Azoxystrobin 80 WG	0,01	0
2.	benomyl	Benlate 50 WP	0,08	0
3.	captan	Captan 50 WP	0,20	0
4.	chinomethionat	Morestan 25 WP	0,06	1
5.	chlorothalonil	Daconil 75 WP	0,20	0
6.	copper	Reconil 588	0,40	0
	oxychoride	WPRecop 84 WP	0,25	0
7.	dinocap	Karathane 25 WP	0,10	1
8.	diniconazole	S3308L 12,5 WP	0,04	0
9.	sulfur	Kumulus 80 WP	0,40	0
10.	fenarimol	Rubigan 12 EC	0,06	0
11.	mancozeb	Dithane M 45	0,20	0
12.	propiconazole	Tilt 250 EC	0,07	2
13.	pyrazophos	Afugan 30 EC	0,06	3
14.	tebuconazole	Folicur 200 EC	0,25	2
15.	thiabendazole	Tecto SC 450	0,10	0
16.	thiophanate-methyl	Cercobin 70 WP	0,07	0
17.	triforine	Saprol	0,10	0

Source: Liberato *et al.* (1999); Marin (1988); Marin and Gomes (2000).

[1/] Scale of rating varying from 0 the 3, being 0=plants without symptoms of phytotoxicity; 1=injury light with small burned areas; 2=leaves chlorotic, with borders and apexs burned; and 3=leaves strongly injured and/or severe defoliation.

protection from secondary infections during the storage and transport to consumer markets.

i. Cleaning of the fruits in water: The fruits should be washed in water to remove soil and other adhering residues, and can also receive a treatment with chlorine for disinfestation. The peduncles should be trimmed and the fruits should be carefully packed when dry with the part of the peduncle down and wrapped in dry paper. This procedure can reduce infection by *C. gloeosporioides* and other fungi that cause post-harvest rots. After cleaning, the fruits should be carefully submerged in tanks of hot water for hydrothermal treatment.

ii. Hydrothermal treatment: Heat treatments have been used to control fungal diseases and insect infestation of papaya fruits for many years. The primary obstacle to the widespread use of heat to control postharvest fruit diseases is the sensitivity of the fruits to the temperatures required for effective treatment with no visible fruit damage. Specialized equipment for temperature maintenance and water circulation is necessary as fluctuations in temperature will reduce the effectiveness of the treatment and may damage the fruit (Couey, 1989).

Hydrothermal or hot-water treatment consists of the immersion of the fruits in hot water of 48°C (± 1°C) for 20 minutes, followed immediately by immersion in cold water of 8°C (± 1°C) for an equal period (Fig. 4). Other combinations of temperature-time of immersion are being tested for effectiveness in the control of post-harvest rots of papaya, as for example the combinations 54°C for 3 minutes and 66°C for 20 seconds.

However, the treatment with hot-water, described initially is that which meets the phytosanitary demands for exportation, since it also includes the capability to kill the eggs and larvae of fruit flies. The principal disadvantage of this treatment is the necessity of a system of heating with precision to maintain the temperature of the water constant during the twenty minutes, because temperatures less than 47°C do not exert the control desired and greater than the 49°C can cause scalding in the fruits. In spite of the existence of research associated with this method and gamma radiation to control post-harvest diseases, only the hydrothermal treatment has been utilized in commercial scale. A treatment by double immersion (30 minutes at 42ºC, followed by 20 minutes at 49ºC) was used in the Hawaii with excellent results, when combined with regular application of fungicides in the field (Alvarez and Nishijima, 1987). Excessive heating or delay in the cooling of fruits can inhibit the normal process of maturation of fruits, provoking in many cases scalding, facilitating colonization by other fungi, and affecting the commercial quality of the fruits.In Hawaii (Lay -Yee *et al.*, 1998) the effect of disinfestation of fruits cv. Waimanalo Solo was studied under commercial conditions using forced air treatment of different temperatures followed by application of the fungicide prochloraz. Fruits treated with temperatures between 47.5ºC and 48.5ºC caused the least loss of firmness of the pulp. Temperatures greater than 49.5ºC tended to cause a loss of the firmness of the pulp of the fruits.

This effect appears to be associated with a decrease in the enzyme polygalacturonase, responsible for the catalyzation of the hydrolysis of pectins, components of the cell wall (Chan *et al.*, 1981; Lay –Yee, *et al.*, 1998). Scalding of the skin also tends to increase with increase in temperature of treatment especially when lasting more than 60 minutes. In general, the thermal treatment did not alter the incidence of peduncular rots, but was associated with an increase of other rots, reflecting the reduction of the resistance of the tissues of the fruits to infection of secondary microorganisms. The immersion of the fruits in prochloraz, 0.25%, after the thermal treatment, reduced the incidence of rots without affecting the quality of the fruits (Lay -Yee *et al.*, 1998).

iii. Post-harvest chemical treatment: In fruits for exportation, principally by ship, after hydrothermal treatment, a treatment with fungicide is recommended, with the objective of increasing the effectiveness of control of the fungal diseases. The use of the two treatments combined (hydrothermal and chemical) is an alternative more advantageous in the control of anthracnose in post-harvest, respecting always the limits of tolerance for chemical residues.

The existence of latent infections in the fruits explains the post-harvest appearance of the disease even when protection of the fruits is provided, since the fungicides apparently protect the fruits from new infections, but do not eradicate the subcuticular hyphae that are quiescent (Alvarez and Nishijima, 1987).

The most effective fungicides utilized actually are prochloraz (50ml p.c./100 L of water), and thiabendazole (200g p.c./100 L of water). This fungicide was used in Hawaii, applied in a concentration of 4-8 g/L with wax of carnaúba (Couey *et al.*, 1984). Concentrations greater than those recommended should not be used to avoid the occurrence of phytotoxicity on the surface of the fruits and conform to limits of tolerance for residues established by the importing countries (Liberato *et al.*, 1999). Prochloraz in the EC formulation can present phytotoxicity in the fruits when used in concentrations greater than 250 mg l^{-1} a.i.

The fungicide benomyl has been used extensively for control of anthracnose of papaya in post-harvest, but studies conducted in the Espírito Santo proved its low effectiveness owing to the occurrence of populations of *C. gloeosporioides* with resistance to this fungicide (Ventura and Balbino, 1995).

After the chemical treatment, the fruits can be immersed in solution of water with wax, in the proportion of 1/1, for approximately 4 seconds.

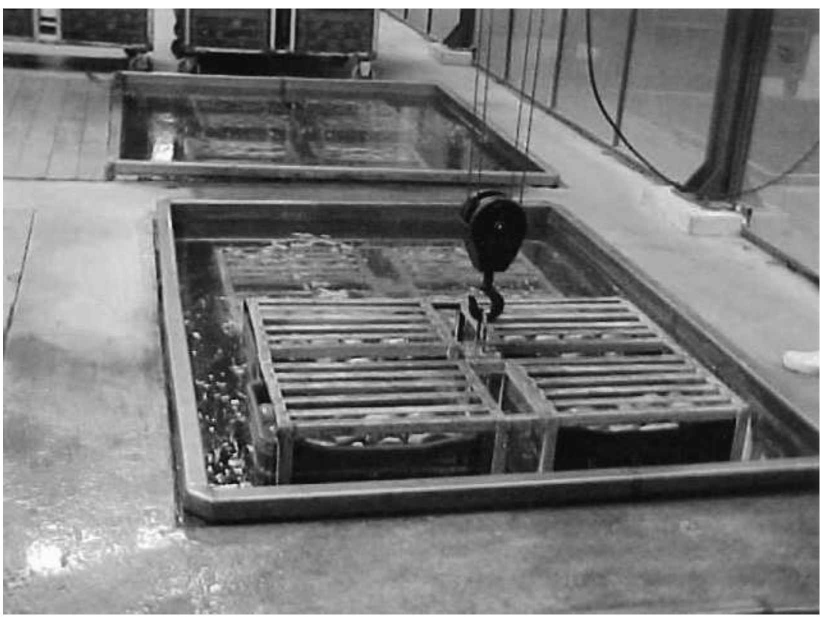

Figure 4 : Hot-water treatment of papaya fruits to control postharvest diseases

Recently the use of 1-methyl cyclopropene (1-MCP), a blocker of ethylene receptor sites, has demonstrated great effectiveness in slowing the maturation of fruits and reducing the incidence and severity of anthracnose during the storage.

iv. Genetic resistance: The commercial cultivars of papaya actually planted are susceptible to anthracnose. However, preliminary results of laboratory research demonstrated that the cv. Golden was more susceptible than other cultivars of the groups Solo and Formosa (Rodrigues *et al.*, 2001). Recent attempts to use genetic engineering to transfer resistance genes into papaya show promise.

2.2 Black spot

Black spot or leaf spot is the most common disease of papaya, occurring in both commercial and domestic orchards, as well as in isolated plants located in backyards and road margins. When it occurs with great severity in the leaves the disease can affect the development of the plants, especially those that are younger. The economic importance of the disease however is most noted when it occurs in the fruits, on which it causes lesions, reducing their value commercially, as verified in some orchards in the north of Espírito Santo and south of Bahia. It is the principal foliar disease of the culture, particularly when it occurs in the initial phase of the establishment of the orchard.

2.2.1 Etiology

The etiological agent of black spot is the fungus *Asperisporium caricae* (Speg.) Maubl., which presents subepidermal mycelium and well developed stroma, erumpent and subepidermal, forming short conidiophores closely packed together and covering the surface of the stromata, unbranched, hyaline to olive brown, smooth, with several prominent conidial scars at the apex. The conidia are solitary, dry, ellipsoidal, pyriform or clavate, almost always bicellular (uni-septate), hyaline to pale brown, distinctly verrucose, and measuring 14-26mm x 7-10mm (Holliday, 1980).

2.2.2 Symptoms

The disease occurs on the leaves and on the fruits. On upper surface of leaves, characteristic symptoms consist of round, light-brown (tan) necrotic spots, encircled by a yellow halo (Fig.5). On the lower surface of the leaves, in the areas corresponding to the spots, the powdery growth of the fungus of gray to black color can be observed (Fig. 6). In some cases, over these, a pale mycelium produced by a fungal hyperparasite of the pathogen may be observed. When it occurs, coalescence of the lesions is a common cause of leaf senescence and defoliation of the plants. Abundant spotting causes defoliation and over 50% leaf fall can occur. Young leaves generally do not present symptoms.

In the fruits, the presence of circular areas of watery aspect are observed initially, that, with evolution of the disease become brown in color, prominent, with pale points, and that may attain 5mm of diameter (Fig. 7). These lesions generally are epidermal and do not reach the pulp of the fruit, causing only a hardening of the skin of the part affected.

2.2.3 Epidemiology

Black spot occurs with greatest intensity under conditions of temperatures between 23 to 27ºC, with strong winds and high rainfall or overhead irrigation. The incidence is seasonal, and most infection occurs in late winter and spring. These conditions favor development of the lesions and dispersion of spores from older leaves, considered the principal sources of inoculum, and where the disease occurs initially, being disseminated subsequently to the younger leaves. The penetration of the fungus is stomatal and macroscopic symptoms are visible between 8-10 days after inoculation (Holliday, 1980). Fruits can be infected when still green, and the lesions resulting from the eruption of the stromas will emerge completely at the beginning of maturation, liberating new spores when the fruit is totally mature.

An epidemiological curve of the disease in different cultivars showed it to be least in *C. cauliflora* and greatest in the cv. Sunrise Solo line 72/12, while a cultivar of the group Formosa and the cvs. Baixinho of Santa Amália, Sunrise Solo, and Santa Bárbara had an intermediate comportment.

2.2.4 Control

Figure 5: Symptoms of black spot caused by *Asperisporium caricae* on the upper surface of papaya leaves, showing the characteristic yellow halo.

2.2.4.1 Cultural practices

Aimed at the reduction of the source of inoculum, older leaves with high severity of the disease should be eliminated. This practice can be carried out at the same time as the operation of sprout removal, which is begun generally 30 days after transplanting the seedlings.

2.2.4.2 Chemical control

Applications of fungicides to control black spot should be begun as soon as the first symptoms of the disease are observed, when the plants still are in the initial phase of growth. In monitoring of the plants, older leaves on which the lesions initially occur should be observed. The first five months after planting, when the plants are most susceptible, is the period most critical for control of the disease in the leaves, which is influenced by climatic conditions.

The fungicides normally recommended for anthracnose also have presented effective control of black spot. However, during periods of climatic conditions highly favorable for the disease, principally periods of prolonged rain, these fungicides have not been effective for control of the disease.

In these cases, fungicides in the triazole and strobilurin groups should be utilized based on their greater effectiveness observed in experimental evaluations. The interval of application of these fungicides varies with climatic conditions and manage-

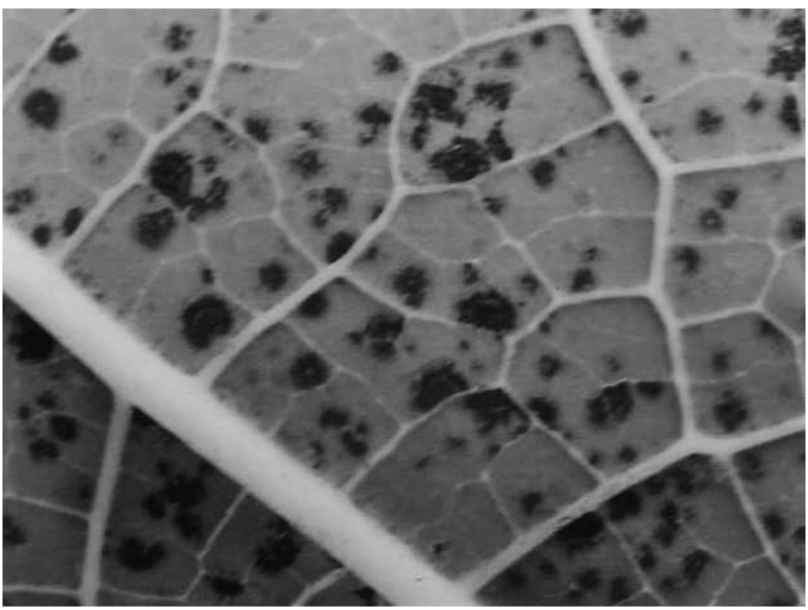

Figure 6: Black spot lesions on the lower leaf surface with the presence of fruiting structures of the fungus *Asperisporium caricae*

ment of the orchard. In drier periods of the year the interval between applications (sprays) may be as much as 30 days. If effective control of the disease in the leaves is not achieved its control in the fruiting phase becomes difficult, with losses in the commercial quality of the fruits occurring.

2.3 Powdery mildew

Papaya powdery mildew is a disease of general occurrence, especially in very shady nurseries and in colder months of the year, as noted in the regions north of Espírito

Santo and south of Bahia, Brazil, between the months of June and September. When it occurs with high severity, the disease can cause damage in the leaves, affecting photosynthesis and consequently the commercial quality of the fruits as well as slowing the development of the plants. In plants in the nursery a total loss of leaves may occur resulting in death of the plants.

2.3.1 Etiology

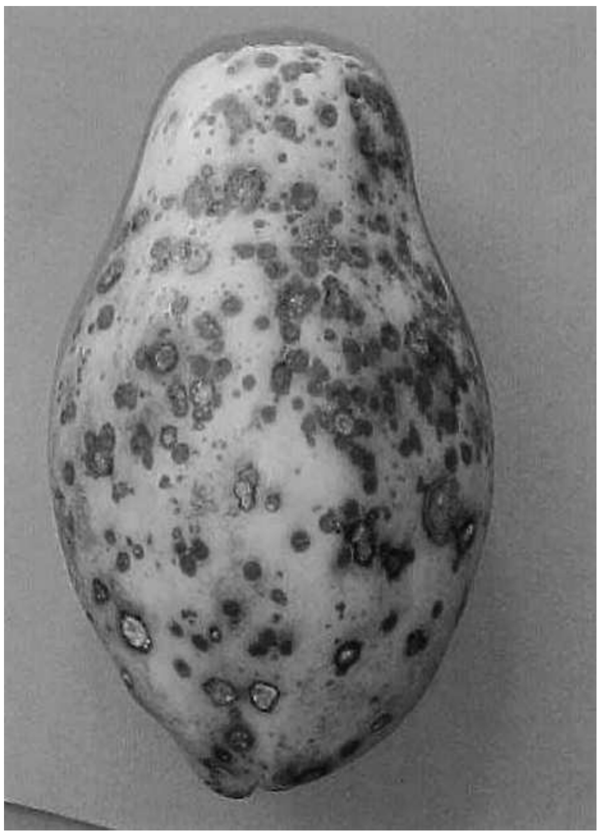

Figure 7: Black spot lesions on papaya fruits caused by *Asperisporium caricae*

Three species of *Oidium* have been reported and described as causing papaya powdery mildew: *O. caricae* (conidia elliptical, 24-30µm x 17-19 µm), *O. indicum* Kamat (conidia barrel shaped, 31-47µm x 12-33µm) and *O. caricae-papayae* Yen (conidia 36-44.4µm x 15.6-21.6µm). Another powdery mildew *Ovulariopsis papayae* van der Bijl. (teleomorph: *Phyllactinia* sp.), with conidia 14-23µm x 60-90µm, has also been described.

O. caricae known in Brazil since 1898 and reported in other production areas of papaya in the world, presents mycelium hyaline, septate, with haustoria developing in the interior of the epidermal cells of the host. The conidia are hyaline and granular, barrel shaped, and formed in chains of three to five or more spores. *O. papayae*, observed in the north of Espírito Santo, Brazil, presents erect, multiseptate conidiophores, originating from cylindrical hyphae (Liberato *et al.*, 1995 and 1996). The conidia are large, subclavate and isolated in the apex of the conidiophores.

2.3.2 Symptoms

Symptoms caused by *O. caricae* begin in the leaves, with a light yellow-green discoloration of irregular outline and of dark-green margins. White powder-like growth of the causal fungus can develop on leaf undersides. This is usually not a severe problem but some leaf drop can occur (Simone, 2002). With evolution of the disease, the discolored patches become covered with a powdery white mass made up of structures of the fungus (mycelium and conidia), present on the upper and lower leaf surfaces. The leaves may become yellowish (chlorotic), present a generalized drying and subsequently fall. With the exception of the younger leaves, all of the leaves may be affected by the fungus, the older leaves being more susceptible. Beyond the leaves, the pathogen can in certain conditions also be observed in the stem, flowers, pedicels and fruits.

The fungus *O. papayae* causes symptoms similar to those incited by *O. caricae*. On the upper surface of the leaf chlorotic areas that evolve to yellowish spots delimited by principle veins, round (or with irregular margins), of approximately 0.5 cm diameter and that coalesce reaching great foliar area, may be observed. On the lower surface of the leaf a powdery mass of pale color, corresponding to the yellowish spots on the upper surface, is observed (Fig. 8). In contrast to *O. caricae* these signs of the fungus occur normally on the lower surface of the leaf, and are rarely observed on the upper surface. Sometimes the white patches appear on the fruits. In the nurseries seedling plants are especially susceptible to attack and may be seriously affected. Young infected leaves of the seedlings dry up prematurely and drop down.

2.3.3 Epidemiology

The disease occurs principally in the colder and drier months of the year, which for the northern region of Espírito Santo, Brazil, corresponds to the months of May to September, when the average temperature varies from 21- 24ºC and the relative humidity of the air is lower than 70% and the weather is cloudy (Anonymous, 2002).

For germination of the conidia, a brief period of high relative humidity is required, but not the presence of water in the free state. The great masses of spores produced on infected leaves are readily spread by wind currents to healthy plants. Year-round production of papaya permits uninterrupted reproduction of the fungus and continuous presence of the disease in an active state (Pernezny and Litz, 1993). The disease is more serious in orchards with systems of drip (or micro) irrigation, since sprinkle irrigation is unfavorable for the fungus.

2.3.4 Control

For both genera of fungi that cause powdery mildew, control is achieved by the application of specific fungicides. The sprays should be applied when conditions are favorable for the occurrence of the disease, principally if these conditions occur for prolonged periods. The product most utilized is wettable sulfur or sulfur dust, applied at biweekly intervals after the start of the appearance of the first symptoms. When climatic conditions are highly favorable, the interval may be less, generally weekly. Sprays with wettable sulfur have not been effective when symptoms in the plants are severe, or that is, when the area of the leaf has signs of the fungus of more than 25%. This fact reinforces the importance of carrying out constant monitoring of the disease in the orchard. Sulfur application should be avoided in the hottest periods of the day (temperature greater than 24° C) to prevent phytotoxicity.Fungicides of the benzimidazole group, like thiophenate-methyl, benomyl and carbendazim, also have been utilized by producers to control powdery mildew in the northern region of Espírito Santo; however,

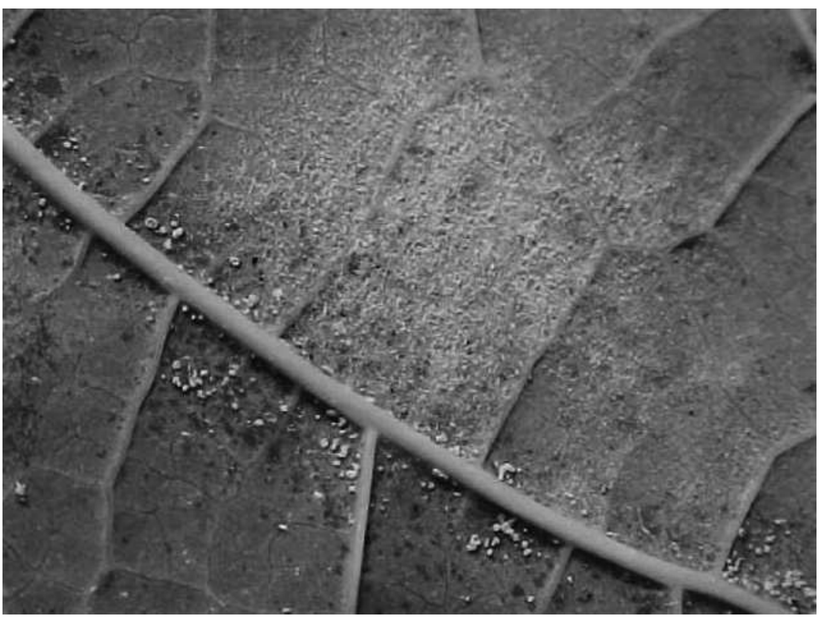

Figure 8: Symptoms of powdery mildew caused by *Ovulariopsis papayae* on the lower surface of the a papaya leaf

they have not been effective.

In the nursery, the fungicide triflumizole (15g c.p./100 l), an imidazol, was effective for control of papaya powdery mildew. In the field, triflumizole was less effective, but not significantly different than sulfur (Tatagiba *et al.*, 1998b).

2.4 *Ascochyta* leaf spot, dry rot and stem end rot of fruits

Ascochyta leaf spot, or leaf blight is a disease that can cause losses of approximately

30% in yield, as observed in some orchards of the northern region of Espírito Santo, principally during periods of high frequency of rain and when outbreaks of mites (*Tetranychus urticae*; two-spotted spider mite) which cause lesions in the leaves that serve as ports of entry for the fungus occur.

2.4.1 Etiology

The disease is caused by the fungus *Phoma caricae-papayae* (Tarr.) Punithalingam (teliom.= *Mycosphaerella* sp. (sin.= *Ascochyta caricae-papayae*), that can infect both the leaves and the fruits.

The etiological agent was for a long time reported as being of the genus *Ascochyta*, but taxonomic studies of the pathogen led to the transfer to the genus *Phoma*, by virtue of the predominance of unicellular conidia and presence of conidiogenous cells phialidics. In *Ascochyta* the conidia are septate (bicellular), and show (exhibit) annellidic ontogeny. The difficulties in the classification of fungi like *Phoma* were discussed by Holliday (1980) and Punithalingam (1980).

In the lesions, both on the leaves and fruits (dry rot), are formed smooth pycnidia and perithecia (100-180µm x 70-200µm), dark brown to black and flask shaped to oval, characteristic of the genera *Mycosphaerella*, this being the teleomorph (Alvarez and Nishijima, 1987).

The genera *Phomopsis* also can occur in papaya with similar symptoms on the fruits, but this fungus forms pycnidia in which two types of conidia are produced, the a conidia (6.4-8.0 x 2.7-3.1µm), hyaline, fusiform and unicellular, and the filiform b conidia (13.7-20.0 x 1.0-1.8µm), while *P. caricae-papayae* does not form the b conidia.

2.4.2 Symptoms

The fungus preferentially infects younger leaves and fruits but also was reported to cause spots on flowers and young fruits.

Penetration is highly favored by the presence of injuries on the borders of leaves, where the round lesions that present a dark-brown coloration and pycnidia arranged in concentric rings are generally observed (Fig. 9). As they evolve, the lesions may attain 4cm diameter, develop a blighted appearance and become broken (or wrinkled), principally in the older leaves. In the more advanced stage, when the conditions are highly favorable, the disease can cause a severe blight of the leaves in the apex of the stem leaving the plants unproductive and in some cases provoking the death of the apex. Under humid conditions, it is common to see light-colored tendrils of pycnidiospores oozing from pycnidia (Nishijima, 1998b).

Infections on flowers and young fruits are initially brown, becoming dark and sunken. Initial infection of stem-end rot is through the broken peduncle, where early symptoms are a slight browning. With growth, the infected area is delineated by a narrow, light brown, translucent margin and as the infected tissue ages, it becomes wrinkled, dry, and black.

In the fruits, the lesions generally occur in the phase of maturation, near the peduncle, turning the tissue black, wrinkled and dry, and frequently covered by a spongy mass of gray color that tends to increase in size, with the formation of pycnidia separate and embedded in the diseased tissue as the lesions age. The lesions evolve rapidly from the base of the peduncle to the pericarp and mesocarp of the fruits, principally after the beginning of maturation, reaching and affecting the seeds (Fig. 10).

The stem-end rot caused by *Mycosphaerella* sp. begins with a translucent zone around the peduncle that becomes brown. With the evolution of the disease the lesion becomes dark (black), with dry aspect, while maintaining translucent margins. In the internal tissues of the fruit the formation of white mycelium that reaches to and colonizes the seed cavity can be observed.

After *C. gloeosporioides*, the fungus *P. caricae-papayae* is considered the most important post-harvest pathogen of papaya.

Figure 9: Lesion of *Phoma caricae-papayae* on a papaya leaf, showing the concentric rings of pycnidia.

2.4.3 Epidemiology

Climatic conditions that favor the incidence of ascochyta spot are temperatures between 21 and 26ºC and high relative humidity (>90%). In the leaves the disease is favored by the occurrence of strong winds. In fruits, handling in harvest and post-harvest causes injuries that enable the penetration of the pathogen. The fungus colonizes senescing leaves and petioles, and produces abundant fruiting structures (pycnidia) in dead leaves that serve as a source of primary inoculum in the field.

In older leaves, the presence of the burn of the leaves associated with the damage caused by tetranychid mites, principally the two-spotted spider mite (*Tetranychus urticae* Koch), owing to the wounds that they cause to the leaves, generally can be observed.

Infection by *P. caricae-papayae* in mature fruits (more susceptible), requires a superficial wound to begin infection and a minimum of 6 hours of continual wetness (Sanchez *et al.*, 1991). In green fruits, the optimal temperature for infection is 21ºC, in the presence of water in the free state; and in the mature, the range of 18 to 24ºC enables the greatest development of lesions.

The epidemiology of the disease needs to be better studied to determine the importance of the teleomorph, and obtain data of the quantity of pseudothecia and perithecia in samples collected from orchards, both in lesions of the leaves, and, principally, in the fruit. In isolates of the fungus cultivated *in vitro* (Costa and Ventura,

Figure 10: Stem end-rot of papaya fruit (cv. of group Solo) caused by *Phoma caricae-papayae*. Longitudinal cross section of papaya fruit showing the colonized tissues.

INCAPER, unpublished), the formation of perithecia has been observed, confirming the homothalic characteristic of the fungus, conforming to that described by Honda and Aragaki (1983).

2.4.4 Control

Wounds created during harvest and postharvest handling are quickly colonized during storage if conditions for infection exist. Also because fruit are susceptible to chilling injury at temperatures below 7°C, normal storage and shipping temperatures are suitable for spore germination and infection (Nishijima, 1998b). The chemical control measures and principally, management, recommended to control anthracnose and black spot have presented effectiveness for the control this disease. Fungicide sprays decrease inoculum levels in the field. The fungicide prochloraz showed effective postharvest control of the disease in experimental studies, when applied by immersion of the fruits.

In Hawaii, Nishijima (1998b) reported that hot water treatment at 48°C for 20 min. also reduced stem-end rot, and the double hot water dip and vapor heat quarantine treatments also control the disease on fruits.

Based on epidemiological characteristics of the disease, the use of sprinkle irrigation favors the sporulation of the fungus and its dissemination in the orchard. The removal of infected leaves, petioles, and fruits is recommended for management of the disease. Equally, the prevention of injuries to the fruits during harvest is recommended. In an evaluation of genetic resistance, the germplasm of *C. papaya* and *C. cauliflora* was susceptible to the fungus. The species *C. gaudotiana* (Tr. Et Pl.) was resistant to *P. caricae-papayae* but presented susceptibility to the fungus *C. gloeosporioides* (Sanchez *et al.*, 1991).

2.5 Greasy or Brown spot

The disease greasy spot or brown spot, also known as *Corynespora* spot, can occur on the stem, fruit, petiole, and leaf of papaya. It is caused by a widespread fungus that also causes the disease often referred to as "target spot" in various hosts. The greatest incidence of the disease is observed in plants more than four months old and in the colder months of the year, when a great number of lesions in the leaves are observed, especially the older leaves, that often yellow and fall. Heavy infection results in premature defoliation with losses in yield and possibly fruit quality.

Lesions on fruits and stems are much less frequent than on the leaves, but in recent years the incidence in fruits has increased in all the cultivars in Espírito Santo, Brazil, lowering sales of the fruits.

2.5.1 Etiology

The disease is caused by the fungus *Corynespora cassiicola* (Berk. & Curt.) Wei., which produces conidiophores erect, simple or occasionally branched, straight or slightly curved, pale to brown, smooth, septate, monotretic, with up to nine successive cylindrical proliferations (110-850mm x 4-11mm). The conidia are solitary or catenate, variable in shape, obclavate to cylindrical, straight or curved, subhyaline to rather pale olive brown having generally 4-20 pseudosepta (40-220mm x 9-22mm) with a dark "hilum" in the form of a kidney (Holliday, 1980). In culture, the fungus produces mycelium gray to olive-brown with growth superficial and into the media, with an absence of stromatas. *C. cassiicola* from infected papaya leaves in Espírito Santo, was cultured on potato dextrose agar (PDA). On PDA the isolates exhibited variability in colony color and growth rates and the optimum temperature for growth was 28°C.

The pathogen rapidly colonized dead papaya leaves and produced ca. 9,000 conidia per g (dry weight) of leaf tissue on the plant. An average of 44,500 conidia per g of leaf tissue was produced on leaf debris from the soil beneath papaya plants (Kingsland, 1985). The fungus is reported as a pathogen in more of 70 species of plants. Isolates of different hosts are morphologically similar. However, they present significant polymorphism when the DNA is analyzed by RAPD-PCR, enabling separation into a genetic group isolates of papaya in relation to those of other hosts, with this group also not pathogenic to tomato and eggplant (Sliva *et al.*, 1998).

2.5.2 Symptoms

Older leaves are most likely to be affected. The symptoms differ greatly depending on climatic conditions and the part of the plant infected. On leaves, the infection occurs principally on the lower surface (Oluma and Amuta, 1999) and the lesions are generally small, of 2 to 3mm diameter, circular, white and encircled by a yellow halo (Fig. 11). In conditions of high humidity, on older leaves, larger lesions reaching 6mm diameter of irregular shape and of light-brown color may arise (Fig. 12). *C. cassiicola* forms conidia on both upper and lower leaf surfaces, but masses of spores are most evident on the latter. Examination of lower leaf surfaces with a hand lens for a dark growth of the fungus hyphae and spores is important for diagnosis of this disease (Pernezny and Litz, 1993).

On the petiole and stem, the lesions are red-brown with the center dark, elliptical, measuring 3 to 5mm in length, with width constant of about 2mm. In the fruits, even green, very small circular spots (1mm) appear, that may evolve rapidly (Fig. 13). The lesions are depressed and with, a dark center_where the structures of the fungus are observed. The lesions may coalesce reaching a great area of irregular form on the fruit.

2.5.3 Epidemiology

Temperatures between 20 to 24ºC and high relative humidity favor infection of the pathogen, which is disseminated by the wind. *In vitro* the optimum temperature for mycelial growth is 28ºC, with a drastic reduction of radial growth occurring at temperatures greater than 32ºC, and no growth of the fungus occurring at 8ºC and 40ºC (Melendez and Pinero, 1970).

Plants with water or nutritional stress become more predisposed to the occurrence of the disease, principally the stem of the plants. The use of sprinkle irrigation generally favors the severity of the disease by forming a microclimate highly favorable for pathogen infection.

Papaya cultivars vary in their responses to infection by *C. cassiicola*. In Brazil cv. Tainung 01 develops very high levels of greasy spot during the months of January and February, when the temperature is greater than 25ºC and humidity is high (Andrade *et al.*, 2002).

The fungus *C. cassiicola* has a wide geographic distribution and has been reported in different plants including tomato, soybean, bean, cassava, rubber and cucurbits. *Commelina benghalensis* a weed of wide geographic distribution and frequently present in papaya orchards, was reported as a host of *C. cassiicola* in Brazil, and may constitute an important source of inoculum, principally when climatic conditions become favorable (Silva and Souza, 1999).

2.5.4 Control

To reduce the quantity of inoculum in the orchard, the removal of senescent and highly infected leaves is recommended. No specific fungicides registrations exist for the disease in Brazil. However, the fungicides mancozeb and chlorothalonil, employed to control anthracnose, reduce inoculum levels, and provide satisfactory control.

2.6 Foot rot and *Phytophthora* rot of fruits

Foot rot, also known as root rot, stem rot, *Phytophthora* blight or *Phytophthora* rot, is

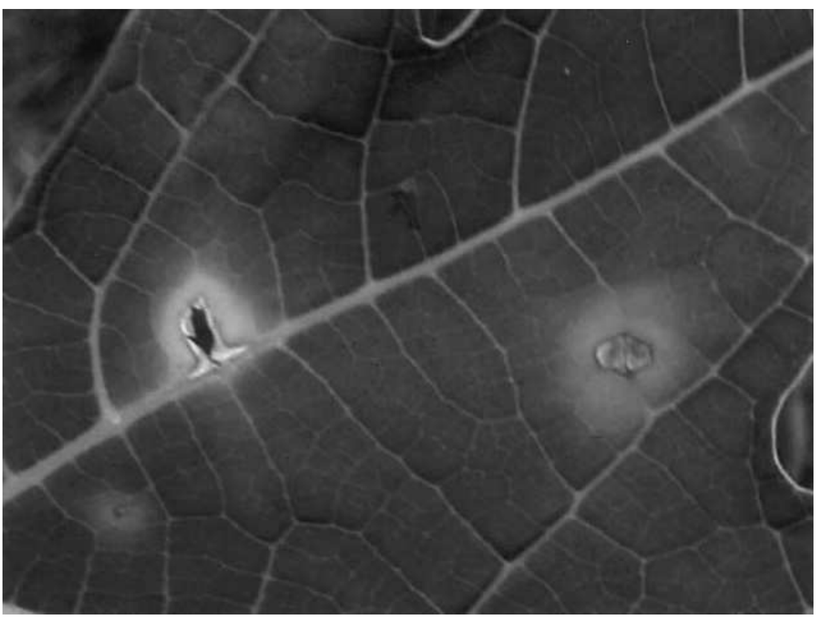

Figure 11:Lesions of greasy leaf spot (*Corynespora cassiicola*) on papaya leaves, showing the characteristic white center and with yellow halo of the lesions.

reported in all of the regions of cultivation of papaya, occurring principally in rainy periods and in heavy, excessively wet and poorly drained soils. The damage can be observed in the roots, collar, fruits and apical region of the plant. Besides causing the death of plants when it causes severe rot in the collar, *Phytophthora* rot can also cause great losses when occurring in the fruits during periods of intense rain.

2.6.1 Etiology

The etiological agent of the disease is the fungus *Phytophthora palmivora* (Butler)

Butler, which has a wide host range. The pathogen produces abundant, terminal, prominently papillate sporangia (50-33μm), in well-developed sympodia. Sporangia when mature fall away from the sporangiophores, within which are formed biflagellate zoospores (10-40). Chlamydospores produced in infected papaya tissues are thick walled (Ko, 1998).

2.6.2 Symptoms

The disease appears with greatest frequency in the collar region of the plants, where watery spots that subsequently coalesce and which may have a gummy exudation in the lesionous area, may be observed. Water soaked lesions appear on the bark just above the ground level. The parenchyma tissues are destroyed, but the vascular tis-

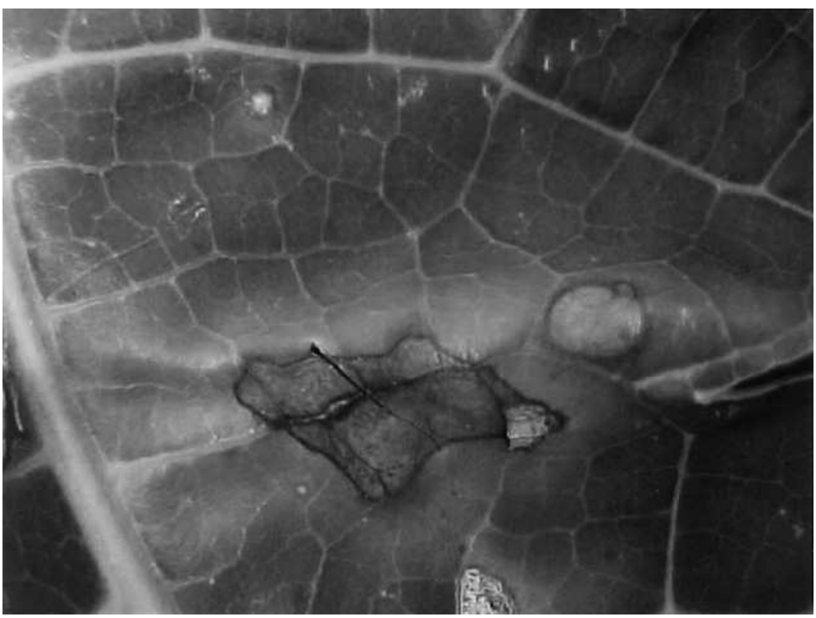

Figure 12 : Large irregular leaf spot symptoms caused by *C. cassiicola* on papaya leaves.

sues remain intact. In poorly drained soils, the pathogen attacks lateral roots and later extends to the taproot. The whole root system may becomes brown, soft, and shredded, and trees become stunted. The root decay has a foul odor. Leaves turn yellow, wilt and hang limply around the stem, living only a few small leaves at the apex of the plant, and the tree dies. As a result of infection in the collar, symptoms of yellowing of leaves, premature fall of fruits, wilt of the top, lodging, and death of the plant may be observed (Fig. 14). In advanced stages of the disease the plants may be knocked down by strong winds. If conditions are not favorable, the lesion may not develop, and the plant may recuperate, but production is compromised. Normally associated with the rot that occurs in the collar, symptoms of rot in the roots of the papaya can be observed, occurring with greatest frequency up to three months after emergence of the plants. Symptoms similar to those caused by the root rot, are observed in rot of the collar. With age, plants become more resistant to infection of the roots.

Lesions may also occur on the apical region of the plants and on the column of fruits. Initially, small, discolored areas arise on the stem, principally where fruits are located, which frequently fall prematurely. These areas increase in size, encircling the stem, killing the upper part of the plant, and in periods of high rainfall, causing the death of the plant. In the fruits, principally mature and near mature, a rot of the tissues which become covered by white, cottony mycelium may be observed, the disease receiving the name of “Papai Noel” (Santa Claus) in the Espírito Santo, Brazil (Fig. 15).

2.6.3 Epidemiology

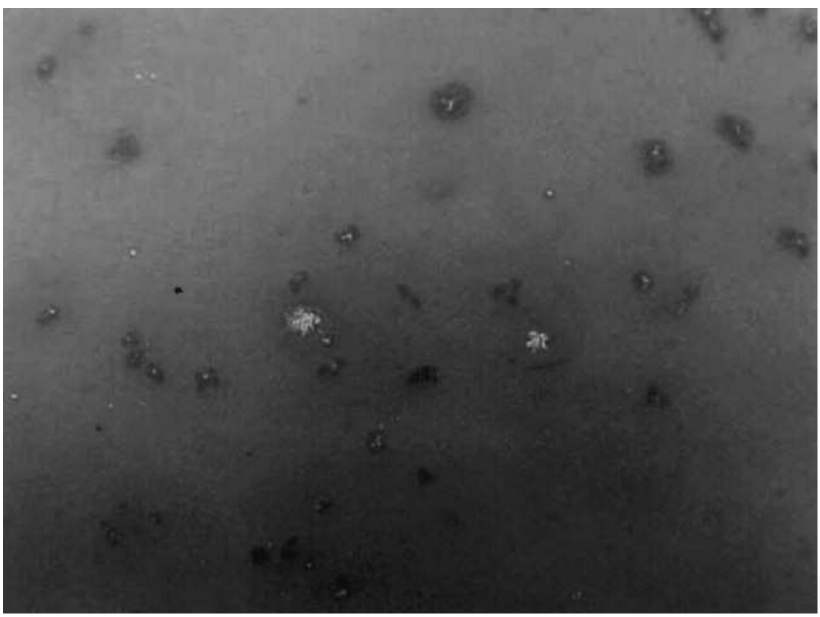

Figure 13: Symptoms of greasy spot on papaya fruits

Disease is more severe on young plants. Cool, wet environmental conditions with high soil moisture favor disease development. *P. palmivora* may be introduced into the orchard from infected seedlings produced in nurseries where phytosanitary care has not been observed, by irrigation water, and sporangia disseminated by wind. High humidity, and temperature from 28-32°C, poorly drained soils, and injuries constitute factors important for initiating the disease. Sporangia and zoospores can survival in the soil for short periods while the chlamydospores, which are the principle structures of resistance, can survive for long periods, in the soil and in fallen fruits. When in water, these germinate to produce sporangia and liberate zoospores, serving as the principal source of inoculum for the infection of plant roots in subsequent plantings.

Rain and the wind are important for dissemination. Drops of rain are necessary for the liberation of the sporangia from the infected plant material or soil. The mobility of the zoospores and their attraction to roots of the papaya plant increase with free water, which also augments the severity of the disease. The pathogen produces a great number of sporangia on the surface of diseased tissues, principally when the temperature is near 25ºC, and this is an important source of inoculum in the development of epidemics. Generally, production of sporangia does not occur when the temperature is

Figure 14: Orchard of papaya cv. Sunrise Solo with high incidence of plants infected by *Phytophthora*. Note, wilt of the canopy of the plants and abscission of the fruits.

less than 15ºC or more than 35ºC. The microbial population of the soil, principally protozoa, significantly reduces the rate of growth of the pathogen (Ramirez *et al.*, 1998).

In experimental studies to evaluate resistance under controlled conditions, the least development of lesions in fruits occurred on the cv. Sunrise Solo, when compared with the genotypes CMF002, CMF007, CMF083 and CMF033, the last two being the most susceptible (Lima *et al.*, 2000).

2.6.4 Control

In the control of foot rot, principally the measures of escape, exclusion, and eradication of the disease are recommended: i. avoid planting in excessively clay soils, with poor drainage and in regions with high rainfall. ii. planting on mounds reduces the incidence of the disease; iii. cultivate papaya in soil where the pathogen has not been reported; iv. utilize sterilized soil for seedlings; v. utilize healthy seeds treated with fungicides; vi. avoid injuries to the plants during cultural treatments; vii. remove diseased plants and fruits from the orchard.

The use of organic material and the enrichment of the soil with microorganisms, particularly in nurseries, is an important practice to prevent and reduce the reproduction of the fungus.

Figure 15: Symptoms of *Phytophthora palmivora* on fruit cv. Sunrise Solo, with growth of white mycelium.

When conditions are highly favorable for the disease, apply preventative sprays directed to the region of the collar of the plants, as well as to the column of fruits, with fungicides based on chlorothalonil or metalaxyl; and proceed with surgery to the stem, removing the tissue affected and treating the local with cupric paste (1 kg of copper sulfate + 2 kg of lime + 10 liters of water). This measure is only recommended for small and superficial lesions. In case of attack only on the apical region that affects the fruits, sprays with specific fungicides should be realized immediately to reduce the loss of fruits. Fruit rot can be controlled with fungicides (Simone, 2002).

Root rot of seedlings caused by *P. palmivora* can be controlled in replanted fields with the virgin soil technique, in which soil from areas where papaya has never been grown is placed in planting holes that are about 30 cm in diameter and 10 cm deep, The roots of plants are protected by the virgin soil during the susceptible stage (Ko, 1987). Cultural practices are also important in the management of root rot. The disease incidence on mature plants during rainy periods can be greatly reduced by improving drainage in orchards.

2.7 Damping off

"Damping off" occurs generally in sporadic form in plants in seedbeds or sacks in nurseries, but may also occur in seedlings recently transplanted in the field. In the nurseries, papaya seedling roots are very susceptible during the first three months after seedling emergence. Infection of roots in this period resulted in yellowing of the leaves, defoliation, and death.

2.7.1 Etiology

Various soil fungi are responsible for the appearance of the disease, most prominently the genera *Pythium, Phytophthora, Sclerotium, Rhizoctonia* and *Fusarium*, which may occur individually or in association. Two species of *Pythium* were recorded on papaya: *P. aphanidermatum* (Edson) Fitzp. with a rapid growth on agar media at 34-36°C, and *P. ultimum* Trow with optimal growth in vitro at 28°C or less (Holliday, 1980). Knowledge of the etiological agent associated with the disease is important in the choice of effective fungicide or the tactic of appropriate control.

2.7.2 Symptoms

Symptoms of the disease are noticed initially in the form of a watery spot in the region in the collar of the plants that increases in size provoking the destruction of the tissues and, in consequence, lodging and death of the seedlings occurs. In the south of Bahia, Brazil, the fungus *Pythium* sp. with white mycelium, well developed and wooly, infected plants between 4 and 36 months of age. Symptoms were underdevelopment, yellowing of the leaves and subsequently, abscission of the older leaves with newer leaves remaining on the extremities of the plants and mummification of the fruits also occurring. In these plants, the root system presented a death of the lateral roots, with a zone of reddish coloration occurring in the region of transition between the healthy and diseased parts (Ram *et al.*, 1983).

2.7.3 Epidemiology

The incidence of damping off is favored by clayey, poorly drained soils with inadequate aeration, and by seeding densely and deeply. High temperatures and rainy periods contribute to an increase in the severity of the disease. The use of mulch also can significantly favor the incidence of the disease in young plants, independent of the cultivar (Elder *et al.*, 2000).

2.7.4 Control

Various measures should be adopted to control damping off, beginning with the choice of the local of establishment of the nursery, which should be in a well ventilated location, free of flooding, with good exposure to the sun, away from roads and fields of papaya, and with water for irrigation, following technical recommendations for construction of the nursery (Marin and Gomes, 2000). Plastic sacks (transparent or dark) preferentially should have the dimensions of 9 to 15 cm width by 12 to 18 cm height, with a minimum thickness of 0.06 cm and with holes in the lower part for drainage of water (Marin and Gomes, 2000). Sacks with height less than 12 cm are not recommended since that can cause deformation of the roots in the bottom of the sack, subsequently compromising the development of the plants.

The preparation of the substrate to fill the sacks is of fundamental importance. It should be composed of two to three parts of surface soil (humus) sieved and one part well-composted stable manure, also sieved (Marin and Gomes, 2000). The origin of the humus should be of sandy-clay and organic soils, avoiding clayey soils that easily become waterlogged during irrigation, prejudicing the development of the plants and predisposing them to the action of the pathogens responsible for damping off. It is important to make a chemical analysis of the substrate and enrich the mixture with the addition of fertilizer if needed, generally simple superphosphate and potassium chlorate. The soil of seedbeds or for filling of sacks should be sterilized by fumigation or heat treatment, and the seeds should be treated with fungicides. Irrigation should be moderate with water free of contamination. Temperature has a very important influence on the time required for germination of the seeds, being generally 9 to 18 days with a temperature average of 25.4ºC and of 12 to 21 days at 23.3ºC. In conditions of the north of Espírito Santo germination occurred between 7 and 12 days during the months of October to March and between 12 and 17 days during the colder months from April to September (Marin and Gomes, 2000). Seeds that germinate 20 days after seeding present low vigor and development of the plants, and are more susceptible to the diseases.

If mulch is used it should be kept at least 30 cm away from the stem of the plants (Elder *et al.*, 2000). Organic fertilizer should be well composted and its application should precede the addition of mineral fertilizer. Stable manure presents the risk of being contaminated with herbicides, like 2,4-D, which is highly phytotoxic to the papaya plant (Marin and Gomes, 2000). When the first symptoms of damping off appear, application of a specific fungicide to the plants is recommended.

2.8 Other fungal diseases and rots in postharvest

2.8.1 *Lasiodiplodia* Fruit Rot or Stem End-Rot

Besides anthracnose, other postharvest rots may cause great damage to fruits of papaya. Principal of them is lasiodiplodia fruit rot, stem end-rot or peduncular rot, which arises after harvest on the region of the cut of the peduncle, affecting the basal part of the fruit, generally at the beginning of maturation. Stem-end rot was attributed principally to the fungus *Phoma caricae-papayae*, and subsequently has also been associated with other genera of fungi like *Lasiodiplodia* (Syn. = *Botryodiplodia*), *Phomopsis* and occasionally *Fusarium*.

The main etiological agent of this disease is the fungus *Lasiodiplodia theobromae* (Pat.) Griffon & Maubl. (syn.: *Botryodiplodia theobromae* (Pat.), which presents rapid growth, and which generally rots and totally mummifies the fruit. Actually, the involvement of other fungi with the disease is also known, including the species *Colletotrichum gloeosporioides*, *Mycosphaerella* sp. (anam.: *P. caricae-papayae*), *Alternaria alternata*, *Stemphylium lycopersici* and more frequently, *Rhizopus stolonifer*. Depending on the fungus involved, variable symptoms develop at the stem end as the fruit ripen. The fungi responsible for peduncular rot can also incite lesions in other regions of the fruit, producing spots or rots with symptoms characteristics to each species.

Lesions caused by *Lasiodiplodia theobromae* are dark with a wide margin of watery tissue and a surface wrinkled owing to the eruption of the pycnidia. Pockets of growth of mycelium occur in the tissues of the infected parenchyma. In a longitudinal cut of the fruit, the vascular tissue is dark, differing from infections of *Mycosphaerella,* which are translucent and those of *Stemphylium,* which are reddish-brown.

With exception of *R. stolonifer*, all of the other fungi can begin the process of infection in the field, causing peduncular rot alone or in different combinations, after the harvest, and during the transport and storage of the fruits. For *L. theobromae* a temperature of 30ºC, and high relative humidity, are the conditions favorable for infection by the pathogen, which colonizes the peduncular region of the fruits, developing abundant superficial mycelium of gray color with intense sporulation. The pycnidia are ostiolate, globose to sub-globose and the conidia are initially hyaline, unicellular, thick walled, and oval, with granular protoplasm. Mature conidia are two celled and light brown, generally measuring 21.7-30.1 x 11.7-18.3 µm with a central septum that turns dark.

In PDA culture media, the fungus has rapid growth, with the formation of aerial mycelium of cottony aspect, initially white and that subsequently becomes greenish-gray to black, with the formation of pycnidia solitary or grouped over dark stromas (Queiroz *et al.*, 1997). The fungus *L. theobromae* has an extensive range of hosts and in papaya, besides stem-end rot, it was also reported as the etiological agent of a rot of the stem of the papaya 'Sunrise Solo' in the state of Alagoas, Brazil (Queiroz *et al.*, 1997). No specific control measures have been developed, but orchard sprays with protective fungicides should reduce inoculum levels and disease incidence. A hot-water postharvest dip (48°C for 20 min) also is effective in controlling this disease (Nishijima, 1998a).

2.8.2 *Alternaria* fruit spot

Alternaria fruit spot is characterized by production of black, circular to oval lesions covered by a mass of black conidia and/or by the white mycelium of the pathogen. The causal agent of this disease is the fungus *Alternaria alternata* (Fr.:Fr.) Keissler, which can cause significant damage, especially in dry environments and after refrigeration, the temperature optimal for development of the disease being 25ºC.

Conidiophores are brown and up to 50 mm x 3-6mm. The conidia are also brown, formed in long chains, measuring 20-60 mm x 9-18 mm, with surface smooth or verruculose, and with a short beak. The fungus can be encountered colonizing senescent petioles, from which the spores can contaminate the fruits during the harvest, being in this way a principal source of inoculum. The lesions are generally restricted to the surface of the fruits and do not cause extensive rotting of the flesh.

Chemical treatments in the orchards and thermal in postharvest generally are sufficient to the control the disease. The disease rarely is observed in fruits that do not undergo refrigeration, but fruits that are kept in cold storage (*e.g.*, 10ºC for 14 days), may experience high disease incidence (Nishijima, 1998a).

2.8.3 *Fusarium* fruit rot

Fusarium fruit rot is associated with various species of *Fusarium*, (*F. verticillioides*

Figure 16: Lesion of *F. solani* on the surface of a papaya fruit. Note the mycelium that has formed on the surface of the lesion.

and *F. equiseti*), but the most common of them is *Fusarium solani* (Mart.) Appel & Wollenw. emend. Snyder & Hansen, that occurs sporadically in the fruits of papaya when these begin to mature, producing small, superficial and dry lesions, subsequently forming a mass of white and very compact mycelium.

F. solani is considered a weak pathogen in fruits of papaya, requiring a stressing factor or injury for its establishment. It is frequently a secondary invader, generally associated with the lesions of *Colletotrichum*, *Corynespora* and *Phoma*.

The lesions of *Fusarium* on the fruit surface (Fig. 16) are generally 1.5-2.0 cm in diameter, and depressed, with the formation of white mycelium occurring on the surface of the lesion (Quimio,1976; Rodrigues *et al.*, 2001; Saxena and Sharma, 1981). The fungus is also known to cause a rot of young (3-5 cm long) papaya fruits especially during wet weather. The fungus enters the seed cavity through the blossom end where it quickly spreads within the fruit and causes the fruit to abort and fall from the tree. In PDA medium, colonies of *F. solani* are white to cream colored, usually with sparse aerial mycelium. Abundant macroconidia are produced in cream colored sporodochia, from long monophialides or branched conidiophores with a distinct notched basal cell. Microconidia are present, generally single-celled, oval to ellipsoidal formed in false-heads on monophialides. Chlamydospores also are formed in specific medium and host tissues, singly and in pairs (Ventura, 2000).

The disease is favored by temperatures near 25°C with rainy weather when conditions of high humidity prevail. In Mexico *F. solani* has been reported to cause a seedling collar rot. It is a weak pathogen requiring some kind of predisposing factor that stresses or injures the fruit before it becomes established (Quimio,1976). No specific control measures have been developed, but preventive fungicide field sprays reduce inoculum levels and are effective in controlling the incidence of the disease.

2.8.4 Wet Fruit Rot

Watery rot, caused by the fungus *Phomopsis carica-papayae* Petr. & Cif. is not frequent in papaya, but when occurs it causes severe losses. The fruit rot occurs most frequently as a stem-end rot, but any part of the fruit can be infected. Initially, a wrinkling of the tissue is observed, that later becomes translucent, of color light-yellow-green color. There is a yellowing of the infected area advancing rapidly to the interior of the fruit, reaching the seed cavity (Nishijima, 1998c). The pathogen requires wounds to infect the fruits, and the disease usually develops on ripened fruits. The part infected can be removed from the fruit easily. With the evolution of the infection, there is a formation of black pycnidia on the surface, beginning in the center of the lesion. In isolates of *Phomopsis* sp. from papaya, two types of conidia are produced in the pyncidia: α-conidia (6.4-8.0 μm x 2.7-3.1 μm), hyaline, fusiform and unicellular, non-septate; and β-conidia (13.7-20.0 μm x 1.0-1.8 μm). The conidial dimensions differ from those of *P. caricae-papayae*, which also does not form β-conidia on host (Nishijima, 1998c).

The fungus sporulates on dried petioles that remain attached to the tree, and during rainy periods, conidia are discharged and deposited on fruit surfaces (Nishijima,1998c).

Control of wet fruit rot like other papaya postharvest diseases, must begin in the field with management procedures (Ventura and Costa, 2002). Senescent and dead leaves should be removed from trees, because they become an inoculum source. Nishijima (1998c) recommends that the removal of leaves is best accomplished by periodically cutting the petioles of leaves that drop below horizontal about 30 cm from the stem and removing them about a week later after the abscission zone forms but before the petiole stub has dried. Fungicide field sprays reduce inoculum levels and are effective in controlling the disease.

2.8.5 *Stemphylium* fruit rot

Stemphylium rot, caused by the fungus *Stemphylium lycopersici* (Enjoji) W. Yamamoto (syn.: *S. floridanum* Hannon & G.F. Weber), normally colonizes and is restricted to tissues of the fruit where wounds are found. Lesions incited by the pathogen are initially small, circular, dark brown color, and subsequently evolve acquiring oval forms with the margins reddish to purple. In the center of the lesion white-gray mycelium and dark-green conidia are formed. Infection by *S. lycopersici* is favored by the presence of injuries in the fruits often times caused by thermal treatment or storage at low temperatures (Chau and Alvarez, 1983b).

The optimal temperature for mycelial growth is 26°C, and sporulation occurs at 10-14°C, *in vitro* under continuous fluorescent light, but 8-10 hr of darkness is required for conidia to form. No sporulation occurs above 30°C (Nishijima, 1998a). Wounding and prolonged cold storage should be avoided. Fungicide field sprays reduce inoculum levels and are effective in controlling the disease.

2.8.6 *Rhizopus* soft rot

Rhizopus rot is a common disease in post-harvest, observed during storage and transport of the fruits in more advanced stages of maturation, but rarely detected in the field. The disease, caused by *Rhizopus stolonifer* (Ehrenb. ex Fr.) Vuill. (syn.: *R. nigricans* Ehrenb.), can cause losses of more of 50% in some shipments.

The pathogen presents great saprophytic capability and only penetrates the fruit through injuries that occur during harvest and post-harvest handling. Colonization of the tissues occurs rapidly, causing a soft and watery rot that affects all of the fruit.

On breakage of the cuticle, the fruit is covered by a mass of prominent, gray mycelium, with black, macroscopic sporangia. Conditions favorable for development of the disease are temperature around 20 to 25°C and relative humidity of 70 to 90%. *Rhizopus*, in contrast to other fungi that cause peduncular rots, disseminates rapidly between the fruits in boxes or even within containers, provoking rot of the fruits in a few days (Alvarez and Nishijima, 1987). The treatments suggested for rhizopus control are the same as those recommended for control of anthracnose, most prominent being sanitation of installations in the packinghouse (Alvarez and Nishijima, 1987).

2.8.7 Internal Rot or Blight

Internal rot of the fruits, also called blight, is a disease that has sporadic occurrence and is caused by fungi that colonize the seed cavity and form a mass of mycelium and spores.

The principle fungi associated with this disease are *Cladosporium* sp., *Penicillium* sp., *Fusarium* sp. and *Alternaria* sp. that penetrate to the seed cavity through the stylar canal in the fruit that remains open after flowering and development of the fruits (Alvarez and Nishijima, 1987; Liberato *et al.*, 1996; Ventura and Santos, 1981). The infected fruits generally present precocious and irregular maturation and are generally discarded in the operations of selection/packaging in the packinghouse.

Considering that the origin of the entry of the fungi is the lack of closure of the stylar canal, which may be a disorder of genetic origin, collection of seeds of plants that present the problem is not recommended.

F. solani is also know to cause a rot of young papaya fruits (3-5cm long) especially during wet weather. The fungus enters the seed cavity through the blossom end, spreads within the fruit, and causes the fruit to abort and fall from the tree (Nishijima, 1998a). Sprays with fungicides in the orchards reduce the inoculum of the fungi, ensuring that the disease has a sporadic occurrence and is of little economic importance (Alvarez and Nishijima, 1987).

3. Diseases caused by viruses

Papaya viruses cause serious reductions in fruit production, even totally destroying affected orchards. Although more than ten different viruses have been reported in papaya worldwide (Table-2), only four are considered of most importance: Papaya ringspot virus, Papaya lethal yellowing virus, Papaya droopy necrosis virus, and the Meleira or sticky disease virus, which is currently being characterized.

3.1 Papaya Ringspot Virus or Mosaic

Papaya ringspot virus also known as papaya mosaic and papaya distortion ringspot, is

Table 2: Viruses of papaya, by family and genus, in main growing regions worldwide.

Family	Genus	Virus species
Bunyaviridae	*Tospovirus*	*Tomato spotted wilt virus*, TSWV
	Tenuivirus	*Papaya mild yellow leaf virus*, PMYLV
Geminiviridae	*Begomovirus*	*Papaya leaf curl virus, PaLCuV*
	Bigeminivirus	*Croton yellow vein mosaic virus, CYVMV*
Potyviridae	*Potyvirus*	*Papaya ringspot virus*, PRSV-p
	Potyvirus	*Papaya leaf distortion mosaic virus*, PLDMV
Rhabdoviridae	*Rhabdovirus*	*Papaya apical necrosis virus, PANV*
Tombusviridae[1]	*Carmovirus*[1]	*Papaya lethal yellowing virus*, PLYV
NE[2]	*Potexvirus*	*Papaya mosaic virus*, PapMV
NE[2]	NE[2]	*Papaya meleira virus* (PMeV)

[1] Molecular research indicates homology with the Family *Sobemoviridae* and genus *Sobemovirus* (Silva *et al.*, 2000). [2] Not established. Molecular characterization of virus genome is in development

one of the most important and destructive diseases of papaya, being one of the factors limiting development of its cultivation worldwide. In Brazil, the disease was recorded in 1935 (Bitancourt, 1935). Mosaic became the principle phytosanitary problem of papaya in the state of São Paulo, which was responsible for most of the papaya produced in Brazil in the late 70's and early 80's, but accounted for less than 1% of the total papaya production by the mid 90's, leading to migration of papaya cultivation to other states. The disease is widespread in the Central and South America, Asia, Australia and is found in the papaya-growing areas of the U.S.A., including Hawaii, Florida and Texas. Losses are very high when rouging of infected plants is not carried out.

3.1.1 Etiology

Papaya ringspot virus is caused by a virus of the family *Potyviridae*, Genus *Potyvirus*, whose flexuous rod-shaped particles are about 760-800nm long by 12nm diameter. The nucleic acid consists of a linear positive single stranded RNA with 10.326pb and the genomic organization proposed consists of the VPg-5' leader, 63K *NT*, 52K *HC-Pro*, 46K, 72K *CI*, 48K *NIa*, 59K *NIb*, 35K cp, 3'-ncr and poly (A) tract (Yeh *et al.*, 1992).

There are two distinct biotypes of papaya ringspot virus: Papaya Ringspot Virus – Papaya biotype (*Papaya ringspot virus* - PRSV-p), and Papaya Ringspot Virus – biotype of cucurbits or watermelon (*Papaya ringspot virus*- PRSV-w). The papaya biotype infects papaya, cucurbits and *Chenopodiaceae* family, while the watermelon biotype infects only cucurbits and does not infect papaya, being previously known as *Watermelon mosaic virus 1* (WMV-1).

PRSV biotypes P and W cannot be distinguished on the basis of divergence in their *cp* sequences. When the nucleotide sequence of geographically different PRSV isolates of P and W types were compared, *cp* sequences which diverged more than 12% at the amino acid level were not found (Souza Jr. and Gonsalves, 1999). Both viruses cause local lesions in *Chenopodium quinoa* and *C. amaranticolor*, plants used as indicators of the viruses. PRSV-p has been transmitted experimentally by sap inoculation, grafting, and by more than 25 aphids species.

3.1.2 Symptoms

Early symptoms appear first on young leaves in the crowns of plants. The leaves exhibit a yellowing and later present an aspect of mosaic, green areas mixed with areas of yellow shade of variable form and size and well defined outline, resulting in a diminution in the rate of growth of the plants, loss of vigor, and decreased fruit yield.

Mottling and intense deformations and blisters may occur characterized by elevated areas of normal green color in contrast with remaining yellow areas (Fig. 17). In plants infected by severe strains of the virus, the leaves are greatly distorted and may develop a fingerlike or shoestring appearance._In the region of the stem, irregular watersoaked spots with an oily appearance may be present, these symptoms being greatly characteristic of the disease. Streaks on the petioles are usually lighter colored and blend into normal color of the petiole. The canopy of the diseased plants becomes smaller with the progress of the disease, the leaves remaining being of reduced size and often deformed.

The fruit surface may present indistinct small concentric rings, very distinct, with the center green (Fig. 18). The first ringspots are dark green to tan and about 1mm in diameter. In the later stages, several spots may join to form irregular shaped spots up to 8mm in diameter and the rings may become necrotic and pale. Fruit yields in infected plants are markedly lower and fruits that mature after the tree is infected may appear normal but are of much low quality and generally unmarketable.Care must be taken to avoid confusing the symptoms of mosaic with those caused by broad mite attack, which are similar. In the case of broad mite infestation, the leaves present thicker veins, are deformed and rugose and reduced to almost veins only, and, on the leaf lamella, the green "islands" typical of mosaic are not observed (Fig. 17). Tree yellowing may be caused by low nitrogen levels, which cause a general loss of color. Thus, some growers

Figure 17: Symptoms of PRSV-p infection on papaya leaves showing the mosaic and green "islands".

mistakenly believe that fertilizer will cure the yellow color occurring on infected trees. It is important to note that before a tree is tagged as being infected with the virus, specific symptoms must be first identified. The yellowing associated with the PRV is a veinal chlorosis, which is very different from the general yellowing associated with nitrogen deficiency. PRV-p symptoms are most severe on younger leaves as compared to N deficiency, which is most severe on older leaves.

3.1.3 Epidemiology

Papaya ringspot virus can be transmitted mechanically and by grafting. However, it is thought that aphid transmission is the most important mechanism for disease spread in the field.

PRSV-p may be transmitted naturally from infected plants to healthy ones by more than 20 species of aphids that feed on the leaves, in a non-persistent form. _In other words, the virus is acquired and transmitted by the vectors in seconds. In Brazil, at least six species have been proven to transmit this virus under experimental conditions: *Myzus persicae* (green peach aphid), *Aphis gossypii* (cotton aphid), *A. fabae* (black bean aphid), *A. coreopsidis*, *Aphis* sp. and *Toxoptera citricidus* (black citrus aphid).

Worldwide, twenty-three aphid species have been tested experimentally and are considered to be vectors of the virus (Mark Culik, INCAPER/CNPq, personal communication, 2002). Although aphids do not colonize the papaya plant, they are attracted by

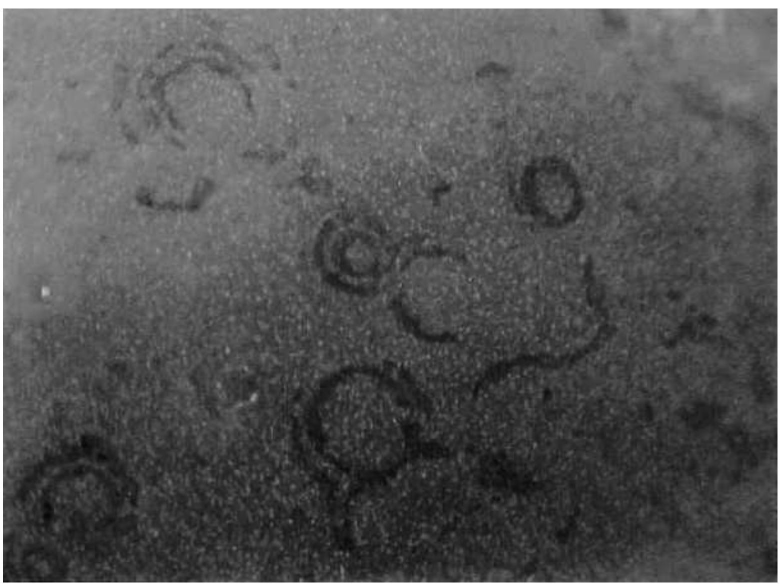

Figure 18: Small concentric rings on the surface of fruits, characteristic of infection by papaya ringspot virus (PRSV-p).

the color of the leaves and the virus is transmitted at the moment of the host recognition or "test probe", when the aphids are searching for a desirable host. By feeding for only a few seconds, an aphid can pick up the virus or infect a plant and in this way_the disease can spread rapidly once infected aphids enter a papaya orchard (Nishijima *et al.*, 1989).

The aphids are usually found more numerous on alternate hosts of the virus than on papaya, which is not normally a host plant of aphids. The insects usually move into papaya fields as a result of high populations on alternate hosts, following prevailing wind directions. The disease can spread quickly if there is a source of infected plants in or around the orchard. Rain and the wind are factors important in the movement of the aphids within the orchard or between neighboring orchards, facilitating the dissemination.

In fertilization of the orchards, balanced nutrition of the plants is very important, especially the relation N/K. Deficiency of N significantly favors incidence of the disease (Vallejo, 1999). The virus can also be spread by mechanical means, such as tools contaminated with sap of infected plants, but this is not considered an epidemiologically important method of transmission. Tools can be cleaned of virus by dipping them in chlorine bleach solution 10% (Nishijima *et al.*, 1989). PRSV-p may be transmitted mechanically to other species of the genus *Carica* and also to different species in the family *Cucurbitaceae*, but there is no evidence that it is transmitted in seeds.

Papaya ringspot virus presents a very rapid speed of dissemination from the focal point of the disease, and all of the plants of an orchard may be infected after a period of 3-7 months, as may be observed in areas with high winged aphid populations. The plants may be infected in any phase of vegetative development, and the symptoms appear between 2-3 weeks after inoculation. However, research in India demonstrated a significant effect of the season of planting on the incidence of disease in the plants (Singh and Singh, 1998). Plants infected at a very young age never produce fruit but rarely die prematurely. There are reports, however, that some isolates from Taiwan cause wilting and sometimes death of the plants (Nishijima *et al.*, 1989).

3.1.4 Control

Once established in a papaya orchard, the virus is rapidly disseminated and difficult to control as a consequence of its nonpersistent mode of transmission by aphids. Attempts to reduce disease levels by applying insecticides have not been successful. To control papaya ringspot virus the use of roguing and a combination of different methods is required (Costa *et al.*, 2000; Ventura and Costa, 2002). Considering that commercial varieties resistant to mosaic do not exist and control attempts by use of tolerant varieties and cross protection with attenuated strains of the virus have not shown success in the control of the disease, it has been possible to live economically with the virus using preventative methods and management of the culture aimed at reducing its dissemination:

i. Carry out periodic visits to the orchard and cut down all infected plants (roguing) as soon as symptoms of mosaic can be recognized. After plants dry or die they no longer will attract aphids;
ii. The trees should be cut as low as possible and do not allow regrowth from the stumps of infected trees. If necessary spray the stumps with a herbicide (glyphosate) to prevent regrowth;
iii. Install nurseries and orchards as far as possible from other orchards, especially if mosaic occurs in the region;
iv. Eliminate the presence of cucurbits (such as pumpkin, melon, watermelon, cucuber, squash or others), which are hosts of the virus as well as hosts of the aphid vectors, in and near the orchard. Aphids in combination with an infected plant in an orchard will result in the rapid spread of the virus;
v. Realize balanced fertilization, and maintain the orchard clean to prevent the forma

-tion of aphid colonies in weeds;

vi. Avoid planting rows in the same direction as predominant winds, which can favor dissemination of aphids into and within the orchard and to nearby orchards.

vii. Destroy abandoned orchards, principally those with plants infected by the virus. One diseased tree can infect a whole neighborhood;

viii. Install the orchard in regions where lower populations of aphids, vectors of PRSV p, occur;

ix. Buy only papaya plants that have been grown under conditions that will prevent infection of seedlings or grow your plants from certified seeds.

Some growers delay cutting infected trees to enable harvest of fruits on the fruit column Although it is a financial loss to growers when they cut down infected trees, it is the only way to manage the disease. Once a tree is infected with the virus, there is no cure. The longer an infected tree is left standing, the greater is the likelihood that the virus will spread to adjacent trees. This will certainly only result in a greater financial loss to the grower in the long run. The virus can easily spread not only within ones own orchard but also to neighboring orchards. It is critical for growers and field workers to learn to identify the early symptoms of the virus. Those in the field daily will have the greatest opportunity to identify infected trees early. Delay for someone else to identify and destroy infected trees will enable the virus to spread to additional trees. The best way to manage the virus is quick identification and immediate destruction of infected trees.

The measures related above, principally the systematic roguing of plants with mosaic, when employed by all growers in a production region, can give very satisfactory results, as has been noted. This has been demonstrated in the north of Espírito Santo, Brazil, since 1994 when an “Eradication of Papaya Mosaic” campaign supported by the Ministry of Agriculture was implemented in this state. Legislation provides for closing, partially or totally, farms where the presence of papaya ringspot virus has been officially diagnosed and proven; with the immediate eradication of all reported foci of disease, and elimination of the infected plants. Also, it obligates property owners, renters or occupants of whatever title, to eliminate at their expense abandoned orchards, and host plants of aphids, in and near commercial orchards, as well as to install nurseries and orchards as far as possible from other orchards where the disease has been found.

The campaign initially presented an educative character, informing the growers of the importance of the elimination of plants with PRSV-p as soon as symptoms were observed and what was mandated by the legislation. In orchards where the virus was detected, the grower received an official notification of the occurrence of PRSV-p in the orchard, establishing a time for eradication.

After the specified time, officers of the Ministry of Agriculture returned to the farm notified to verify if the grower had complied with the legislation. If not, an interdiction was issued, prohibiting the movement of any papaya vegetative material from the farm. If, the grower still did not adhere to the requirements imposed, compulsory eradication of the orchard was carried out by police action. Considering that non-compliance contributes to the maintenance and diffusion of disease, the producer is subject to the penalties of Brazilian law, for the crime of diffusion of diseases.

The positive result of the campaign, which made possible the continuation of commercial production of papaya in Espírito Santo, was a function of the frequency of inspections of orchards by Ministry of Agriculture officers, in view of the difficulty of some growers to adhere to requirements of roguing to control of the disease, principally in periods of high papaya prices.

In 1998, of 161 farms inspected, 58 (35%) were notified, 21 (13%) interdicted and 3 were eradicated compulsorily. Of the 5.052ha of papaya cultivated in this year, 771ha (15,3%) were eradicated. In 1999, a greater consciousness of the producers to comply with the legislation was noted, in that, of 168 farms inspected, 37 (22%) were notified and only 5 (3%) were interdicted. Sources of resistance to PRSV-p in cvs. of *C. papaya* have not been found, but other species like *C. cauliflora*, *C. pubescens and C. quercifolia* have resistance to the virus. However the interspecific hybridization of these species with *C. papaya* have not been viable due to the interspecific reproductive barriers. Pre- and post-zygotic barriers have severely limited the use of this approach in breeding programs (Souza Jr and Gonsalves, 1999).

In Australia, hybrids of *C. papaya* x *C. cauliflora* have been evaluated in the greenhouse and field, and present resistance to two Australian strains of the virus, but have however problems of survival and sterility in some plants, possibly due to aneuploidia (Magdalita *et al.*, 1997). Papaya cultivars tolerant to PRSV-p were also developed in breeding programs in Florida (USA) and Taiwan, using in both cases, polygenic tolerance, originating from the dioecious papaya cv. Cariflora, selected by R. Conover (Conover *et al.*, 1986). These tolerant genotypes were susceptible to PRSV-p, but fruit and leaf symptoms were milder, and infected trees produced reasonable quantities of fruits. Solo-type papaya cultivars with resistance or tolerance have not been developed (Nishijima *et al.*, 1998a).

Cross protection is a technique attempted in different countries with reports of different degrees of success (Rezende and Costa, 1993; Lima *et al.*, 2001). This technique involves the use of a mild virus strain to protect plants against the infection of a severe strain of the same virus that causes economic damage (Rezende and Costa, 1993). Mutant mild strains showed promising results in greenhouse experiments, but when tested in field plots success was limited under severe disease pressure. The level of protection is variable and depends on the geographic region from where the virus used was obtained. Cross protection has been reported to be moderately successful in Hawaii and Taiwan, but not successful in Brazil and Thailand (Nishijima *et al.*, 1998a; Rezende and Costa, 1993).

In recent years, there has been intensified research with molecular biology aimed at producing transgenic papaya plants expressing the PRSV coat protein gene (*cp*), that resistance to PRSV-p under field conditions, which has opened up a new control measure. The first studies to develop a transgenic papaya plant resistant to the disease were conducted in the beginning of the decade of 1990, through a cooperative project between Cornell University, the University of Hawaii and the UpJohn corporation of the USA, resulting in the line of plants known as 55-1, that express the gene of the coat protein (cp) of an isolate of the virus obtained in Hawaii (Fitch *et al.*, 1992).

The RO clones of the 55-1 line maintain their resistance to PRSV-p, and the quality of the fruits and characteristics of the plants are similar to those of 'Sunset' from which they originated (Gonsalves *et al.*, 1998).

In the field evaluations, the transgenic genetic materials were denominated 'UH SunUp' (line 55-1 homozygous for the gene of the coat protein) and 'UH Rainbow' (hybrid from the cross of 'UH SunUp' and the non-transgenic cv. Kapoho,), which retained resistance to the virus, obtained approval from official American organs (APHIS, EPA and FDA) for cultivation and commercialization, and received good acceptance by the papaya producers in Hawaii (Gonsalves *et al.*, 1998).Despite resistance to the virus of Hawaii, however, when these plants were inoculated with virus of other geographic regions, including Brazil, they demonstrated susceptibility (Tennant *et al.*, 1994; Souza Jr., 2000).

In Brazil, EMBRAPA, through of the Center of Cassava and Fruits in Bahia and more recently the Center of Genetic Resources and Biotechnology, established a partnership with Cornell University to develop transgenic plants resistant to the Brazilian virus, and these plants have also showed resistance to isolates of the virus of Hawaii and Thailand (Souza Jr., 2000). The mechanism of resistance of the papaya plants that express the non-translatable version of the *cp* gene of the virus is mediated by the RNA and not by the protein, and is influenced by the stage of development of the plants and by the concentration of inoculum; the influence of these factors being minimal, however, relative to the effects of the genetic dose and of the degree of homology between the (trans) *cp* gene and the *cp* gene of the virus used in the inoculation (Souza Jr., 2000).

The experience with transgenic papaya in Hawaii is discussed from the technical and the grower/consumer perspective by Ferreira (2001).

Currently there is much concern over the potential ecological impact of transgenic plants. One concern is the risk of viral recombination. Discussion of possible risks associated with to the use of genetically modified organisms (GMOs) has intensified in recent years, raising the concern of bio-safety, principally of food and environmental order. The genetically engineered virus gene in papaya may mix with the genome of other viruses which infect the papaya, creating new and more potent disease-causing viruses. Scientists do not fully understand such interactions, and it is therefore still unclear how readily, if at all, new viruses would arise (Kaesuk-Yoon, 1999; Swain and Powell, 2001).

In the case of transgenic papaya developed in Brazil, the genes utilized have been the *cp* gene of the virus that causes the disease, and the marker gene *nptII*, and the analysis of risks should therefore consider the interaction *C. papaya* x *cp* x *nptII* (Souza Jr., 2000). As the transgenic papaya plants of the Brazilian program express the gene of the coat protein in a non-translatable form, that does not produce the protein, heteroencapsidation of the incoming virus with coat protein produced by transgenic papaya, should not occur, however, as no studies exist with other viruses that occur in the papaya plant, this possibility should be addressed and evaluated.

In the same way, gene flow or vertical transfer of the (trans) gene to other cultivars and hybrids should be considered, principally in the programs of genetic improvement and production of seeds.

Some farmers are reluctant to plant genetically engineered papayas because of market concerns. Typically 35 to 40 per cent of the Hawaiian papaya harvest is exported to Japan. Genetically engineered papayas have not yet been approved for sale in Japan (Kaesuk-Yonn, 1999; Swain and Powell, 2001).

EMBRAPA Genetic Resources and Biotechnology has developed papaya plants resistant to Brazilian isolates of PRSV-p, but because of questions of bio-safety and patent of the genotypes, these cultivars still are being evaluated and their commercial utilization is not permitted. Possibly, in the future, these transgenic cultivars will be available to growers and will offer a more permanent solution for control of papaya ringspot virus.

3.2 "Meleira" or Sticky Disease

Meleira also known sticky disease, is considered the most important disease of papaya and the major phytosanitary problem for its cultivation in Brazil. The first reports on this disease date from the late 1980's, when it was observed in orchards in the south of Bahia and north of Espírito Santo, Brazil (Rodrigues *et al.*, 1989a). Since then, the disease has also been found in the states of Ceará, Minas Gerais, Paraíba and Rio Grande do Norte.

Initially, meleira was localized in small areas, but today, it can be detected in all orchards of papaya in the north of Espírito Santo and in some municipalities of the states of Bahia, Minas Gerais, Ceará, and Pernambuco. The disease is characterized by intense exudation of latex in fruits that oxidizes and darkens making them totally unsuitable for commercialization, as well as compromising their flavor. In the north of Espírito Santo, while the percentage of plants eradicated with mosaic (PRSV-p) is about 2% in well-managed orchards, meleira typically infects at least 20% during the economic cycle of culture. In some orchards where rouging was not carried out, an incidence of the disease of up to 100% was recorded by the time the plants reached the harvest phase and were only 12-15 months old.

Although the occurrence of meleira had been noted by producers since the decade of 1970, this problem worried no one for the most part, until the middle of the following decade, when it was reported causing losses in commercial orchards in Teixeira de Freitas, in the south of Bahia. Meleira had been observed since 1984 in the state of Espírito Santo, but was only reported officially in 1989, when it was proven by means of epidemiological studies to be in fact a disease with biotic etiology (Rodrigues *et al.*, 1989a and 1989b).

3.2.1 Etiology

Initially, symptoms of the disease were attributed to a deficiency in the absorption of calcium or boron resulting from a lack of water or imbalance of these elements in the soil (Nakagawa *et al.*, 1987).

The biotic etiology of this disease was confirmed after the development of the anticipated typical symptoms in papaya plants followed inoculation of healthy plants with latex collected from symptomatic fruits (Rodrigues *et al.*, 1989a; Kitajima *et al.*, 1993; Ventura *et al.*, 2001a). Also, the association meleira with a biotic agent was studied by the monitoring dispersal of the disease in commercial orchards, which indicated involvement of a pathogen (Rodrigues *et al.*, 1989b; Maffia *et al.*, 1993). Transmission electron microscope studies of the latex of leaves, stems, and fruits of plants with symptoms of meleira indicate that the disease is of viral etiology, with the presence of large numbers of isometric particles of approximately 50nm diameter, and the occurrence of a double strand of RNA (dsRNA), of approximately 6×10^6 daltons, in latex and extract of leaves and fruits of affected plants. Ultrafine tissue sections revel that these isometric particles are restricted to lactiferous vesicles (Kitajima *et al.*, 1993).

The efficiency of the virus purification protocol was confirmed after successful purification of the viral particles, and the RNA of approximately 12 kb long was observed after removing the proteins from the virus particles. The double stranded nature of the RNA was confirmed in a 1% agarose gel, as 6×10^6 daltons dsRNA purified directly from leaves (Souza Jr. *et al.*, 2002; Zambolim *et al.*, 2000). Virus purification from infected plants confirmed the viral etiology of the disease, with the inoculation of healthy papaya plants that developed symptoms after inoculation (Zambolim *et al.*, 2000). Studies demonstrated that the dsRNA is found in leaves, stem, fruits, flowers and roots of papaya.

In 2001, researchers of thr EMBRAPA Genetic Resources and Biotechnology Center, Department of Plant Pathology/Federal University of Viçosa-UFV, INCAPER and the Federal University of Espírito Santo-UFES, joined efforts and initiated collaborative work towards the characterization of the virus genome, as well as the development of protocols for early and widespread diagnosis.

3.2.2 Symptoms

Exudation of latex from fruits, spontaneous or provoked by injuries, which appears to be more fluid than the typically milky latex found in healthy plants, and that oxidizes and becomes dark, is the most common symptom of meleira (Fig. 19).

In severe cases, the extensive exudation gives a sticky aspect to the fruit, from which the name of the disease originates.The latex of fruits of a plant with meleira presents a clear watery aspect, due to its lower viscosity and lack of coagulation, that darkens with greater facility than that of healthy fruits (Ventura *et al.*, 2001b). The exudation of latex also occurs from edges of young leaves in the top of the plant and with oxidation provokes small light-brown necrotic lesions on the leaf tips (Fig. 20). These symptoms, although not always observed, permit the identification of the disease in young plants. However, they should not be confused with the lesions caused by the blight of the leaves caused by the fungus *P. caricae - papayae*.

In infected fruits, in advanced stages of the disease, irregular light-green areas are observed on the surface, slightly resembling symptoms of deficiency of micronutrients, principally boron. Farmers must be careful not to confuse symptoms of meleira in fruits with those caused by mechanical damage. Thus, it is important to note the viscosity of the latex to obtain a secure diagnosis of the disease.

3.2.3 Epidemiology

The causal agent of meleira was efficiently transmitted by injection of the latex of diseased plants into the stem of healthy papaya, obtaining disease symptoms approximately 45 days after inoculation. Mechanical transmission by friction using latex was not effective in papaya and other plant species, requiring a continuation of research regarding transmission and alternative hosts.

Recent studies provide strong evidence of the existence of an aerial vector associated with the disease. The silverleaf whitefly (*Bemisia argentifolii* Bell. & Perring), also known as *B. tabaci* biotype B, has been associated experimentally, in controlled

Figure 19: Typical symptoms of the meleira (sticky disease) on fruits with the characteristic flow of oxidized latex

conditions, with the transmission of meleira (Vidal *et al.*, 2000).

In the plants subjected to infestation with the silverleaf whitefly, monthly monitoring for the presence of dsRNA particles was carried out, with detection occurring six months after infestation and the appearance of the characteristic symptoms of exudation of latex in the fruits in 8 months (Vidal *et al.*, 2000).

The involvement of leafhoppers, principally of the genera *Solanasca* is also being investigated. The presence of dsRNA, with molecular weight similar to that associated with meleira was detected in samples of gramineae species *Trichacne insulares* and *Brachiaria decunbens*, present in orchards of papaya affected by meleira in the south of Bahia (Barbosa *et al.*, 1998).

Field observations, based on careful monitoring and monthly mapping of the distribution of diseased plants, demonstrated that the dispersion of meleira was along the lines of planting, presenting an aggregated arrangement of diseased plants. This evidence suggests the possibly that cultural practices could be responsible for dissemination of the disease within the orchard, because these practices are normally executed following the direction of the plant rows. Fruits of papaya from of plants infected by the virus present greater predisposition to infestation by fruit flies, principally the species *Ceratitis capitata,* possibly by the alteration of the levels of benzyl

Figure 20: Early symptoms of necrotic lesions caused by meleira virus on young leaves

isothiocyanate (BITC), present in the fruits (Martins, *et al.*, 1999). Research developed by INCAPER demonstrated that the fly only occurs in fruits of plants more than three months after the appearance of disease symptoms, and is absent in areas where systematic rouging is carried out. The correct execution of a “systems approach” for production in the form carried out in the state of Espírito Santo, and which has become an important measure for the production of fruit for exportation, shows effectiveness even in orchards where meleira occurs.

3.2.4 Control

The roguing of infected plants is recommended until more specific control procedures have been developed. The following measures are also recommended, with the objective of reducing dissemination of the disease as well as preventing or delaying its introduction into areas where the disease has not been noted:

i. Carry out weekly inspections in orchards and eliminate diseased plants (roguing), as soon as the first symptoms of meleira are detected (Ventura *et al.*, 2001b);
ii. Install nurseries and new orchards as far as possible from other orchards, especially those with a history of disease;
iii. Do not collect seeds from diseased plants and orchards with high disease incidence;
iv. Disinfect all material used in the process of thinning and harvesting fruits; try to reduce injuries to plants during cultural treatments;
v. Manage the vegetation under the plants, maintaining rows clean and the area between rows trimmed to diminish the variety of weed species;
vi. Eliminate all orchards (diseased and healthy) at the end of the economic cycle of production to eliminate sources of inoculum;
vii. Destroy abandoned orchards, principally those with plants infected by the virus.

One diseased tree can infect a whole neighborhood. The Brazilian Ministry of Agriculture, by a Federal decree, specifies that farms of the state of Espírito Santo where official diagnosis has proven the presence of papaya meleira virus will be notified of the disease occurrence, and infected plants will be immediately eliminated as recommended for control of papaya ringspot virus.

3.3 Papaya Lethal Yellowing Virus

Lethal Yellow of Solo papaya (*Papaya lethal yellowing virus* – PLYV) was initially reported in papaya plants in the state of Pernambuco, Brazil, in 1983. Later, the disease was reported in the states of Bahia, Paraíba, Rio Grande do Norte and Ceará, being a virus little known but that is spreading in northeastern Brazil (Lima *et al.*, 2001).
Because most cases of this virus have been encountered in individual plants or small orchards, little information exists about its economic importance for production. In a survey in Rio Grande do Norte, a disease incidence of 40% was reported (Kitajima *et al.*, 1982a and 1982b).

However, based on reports made by those who have observed the problem, it is believed that papaya lethal yellowing virus may bring great damage to production if it is disseminated to major, commercial growers.

3.3.1 Etiology

The disease is caused by *Papaya lethal yellowing virus* (PLYV), a virus with isometric particles, of diameter in the range of 29-32nm, and that occurs in high concentration in the tissues of affected plants.

Studies conducted with an isolate obtained from papaya plants from Natal and Rio Grande do Norte, demonstrate that the virus is made up of a piece of single stranded RNA of ca. $1{,}6 \times 10^6$ Da, with a single protein capsid of 36 kDa (Kitajima *et al.*, 1982b; Kitajima, 1999).

The virus was initially considered a possible member of *Tombusviridae* Family, genera *Carmovirus*, but recent molecular research shows a homology of ca. 51% of the gene polymerase nucleotidic sequences and the VPg with the Family *Sobemoviridae* and genera *Sobemovirus*. Also the sequences of *cp* gene show homology of ca. 44% with *Sobemovirus* (Silava *et al.*, 2000).

3.3.2 Symptoms

The symptoms in Solo papaya begin with a yellowing of young leaves of the top third of the stem, which later may fall. Longitudinal depressions and necrotic lesions may be observed on the leaf petioles and on the bottom side in the leaf veins, respectively. Fruits have an intense exudation of latex and round chlorotic spots. With evolution of the disease, the stem becomes twisted and the leaves chlorotic. Subsequently, the yellowed leaves wilt and dry, leading to plant death. In plants of the cv. Caiano, the foliar symptoms develop in the same manner as in the cvs. of the Solo group. However, twisting of the stem and death of the plant do not occur.

In fruits, circular spots are observed on the skin, initially light green and later yellowing. Maturation of the pulp is delayed and appears generally stony.

3.3.3 Epidemiology

The virus is transmitted mechanically from papaya to papaya. Seeds of fruits from infected plants have been tested by ELISA, indirectly verifying the presence of the virus on the surface of the seeds (Camarch *et al.*, 1997). These results suggest the recommendation to not utilize seeds of diseased plants, as well as the risk of introducing the disease in uninfested production areas through seeds.

Transmission between plants appears to depend on insect vectors still not identified. However, its form of dispersion indicates a low effectiveness of these vectors. The virus has the capability of surviving in soils of the rhizosphere of infected plants and infecting healthy seedlings when planted in these soils (Camarch and Lima, 1997). The virus also is dispersed by irrigation water (Camarch *et al.* 1997).

3.3.4 Control

Although this is a disease that has received little study, there are recommendations for control, including measures of general scope, employed to minimize dissemination within those states where it has been found, as well as to avoid or delay its introduction into areas where it still has not been noted.

Avoid movement of seedlings and seeds between states, principally those originating from those states where the disease occurs. Efforts to control the disease consists of roguing and a combination of other management methods:

i. Systematically eradicate affected plants through periodic inspections in orchards where the disease is present;
ii. Buy only papaya plants that have been grown under conditions that will prevent infection of seedlings or grow your plants from certified seeds.

iii. Install nurseries and orchards as far as possible from other orchards, especially if the disease is known to occur in the area;
iv. Manage soil and irrigation of orchards taking into consideration survival of PLYV in the soil and water
v. Tools used on infected plants may be cleaned of virus by dipping them in chlorine bleach solution 10%;
vi. Eradicate old papaya plants so they will not serve as a source of inoculum of the pathogen.

3.4 Papaya Droopy Necrosis and Papaya Apical Necrosis

Papaya droopy necrosis virus has been reported in Florida (USA) and a similar virus disease causing apical necrosis was reported in Venezuela. The viruses have similar particle morphology, and both can cause the same symptoms (Wang and Conover, 1983; Lastra and Quintero, 1981).

3.4.1 Etiology

The diseases are caused by rhabdoviruses with similar sized particles of 87-98nm x 180-254nm, for *Papaya droopy necrosis virus* (PDNV) and 80-84nm x 210-230nm for *Papaya apical necrosis virus* (PANV), aggregated and detected in the parenchyma cells in vascular tissues (Lastra and Quintero, 1981; Zettler and Wang, 1998).

3.4.2 Symptoms

The earliest symptoms of PDNV are the drooping and curvature of the leaves in the plant upper crown, which is rounded and has a distinct bunchy appearance with short internodes. The youngest leaves at the apex are pale yellow, sharply cupped downward, do not expand properly and may also be marginally necrotic. The petioles are shorter and more stiff than normal. Flowers of diseased plants abort, and fruit set ceases. The stem tip becomes very short, necrotic, and the plant eventually dies (Pernezny and Litz, 1993).

3.4.3 Epidemiology

The disease incidence is relatively low when compared with other papaya viruses, and is considerably reduced by avoiding overlapping and successive crops and by promptly roguing infected plants (Wang and Conover, 1983; Zettler and Wang, 1998). The disease usually increases in severity in the winter.

The diseases is mechanically transmissible. No natural vector or alternative host has been identified for PDNV, but papaya seedlings exposed to the leafhopper *Empoasca papaya* that had fed on PANV infected plants developed symptoms. However particles of the rhabdovirus were not detected in these plants (Zettler and Wang, 1998).

3.4.4 Control

Since field incidence is low at present, the only practical means of control is to avoid overlapping and successive crops and realize prompt roguing of symptomatic plants.

4. Diseaes caused by Phytoplasma

4.1 Dieback and Yellow Crinkle

In Australia papaya may be infected by a phytoplasma causing the disease known as by "dieback", "yellow crinkle" and "papaya mosaic" (Gibb *et al.*, 1996 and 1998). Papaya dieback disease has become one of the most serious factors limiting papaya production in Australia (Queensland), with annual plant losses often greater than 5%, and up to 100% during epidemics. The disease was first recorded in Queensland in the early 1920s (Cook, 1975; Glennie and Chapman, 1976), about the time the crop was first grown commercially.

4.1.1 Symptoms

Diseased plants present apical leaves with a slightly chlorotic appearance that soon after begin to shrivel. The older leaves develop a chlorosis and may fall.

The development of translucent areas in the extremities of the leaves of the crown and necrosis reaching the principle veins resulting in a "claw like" aspect may also be seen.

Dieback-affected papaya plants were characterized by a discoloration of the contents of laticifers, while the anatomy of sieve elements was healthy in appearance until the necrotic stages of the disorder were reached. Laticifer discoloration was not always associated with the presence of phytoplasma in affected tissue, as determined by polymerase chain reaction (PCR), using primers based on the 16S rRNA gene and 16S-23S intergenic spacer region (Siddique *et al.*, 1998).

The production of fruits is affected by the development of a greening of the floral parts and appearance of phyllody (transformation of the flowers into leaf-like structures). Plants with the "mosaic" symptoms present wet lesions in the stem, petioles and fruits.

4.1.2 Etiology

Over the years, many investigations failed to conclusively link nutrient deficiencies/toxicities, nematode damage or a pathogenic microorganism to the disease. Although viruses, bacteria and fungi were not consistently detected by conventional techniques, the progression of disease symptoms did seem to indicate a plant pathogen affecting the phloem tissues of diseased plants.

In 1998, three phytoplasma-related diseases of papaya (*Carica papaya*), dieback, yellow crinkle, and mosaic, were recognized within Australia (Guthrie *et al.*, 1998). Phytoplasma DNA was detected using the polymerase chain reaction (PCR) with primers specific for phytoplasmas in general, and for the stolbur group of phytoplasmas. Phytoplasma DNA was detected in a range of plant tissues, including roots, but not in mature leaves, which would act as photoassimilate sources (Siddique *et al.*, 1998). Positive results for PCR tests for phytoplasmas were consistently obtained for plants affected by dieback and yellow crinkle, and no amplification was obtained from DNA extracts from a number of asymptomatic papaya plants. This was the first consistent evidence for the association of a pathogenic organism with both dieback and yellow crinkle. Similar results were obtained by other research groups and confirmed that it was phytoplasma DNA that was being amplified from tissues of diseased plants (Gibb *et al.*, 1996; Davis, *et al.*, 1996; White *et al.*, 1997). In these studies, although PCR tests indicated the presence of phytoplasma DNA, phytoplasma cells were not observed in transmission electron microscopy studies of mature sieve elements of dieback affected leaf, stem, or fruit tissue from plants at various stages of symptom expression.

In some cases membrane-bound structures, similar in shape and size to phytoplasma cells, were observed within vacuoles of cells in the phloem tissue of leaves displaying tissue breakdown in the form of a water-soaked appearance to veins ("X-Y" patterning). But, the structures were interpreted as autophagic vesicles or latex vesicles in immature laticifers. In contrast, phytoplasmas were readily observed in papaya leaves displaying symptoms of yellow crinkle. Siddique *et al.* (1998), concluded that phytoplasma cells are present in very low titer in dieback-affected tissues and that, while the plant appears to limit proliferation of the dieback-associated pathogen, this defense strategy is ultimately unsuccessful because it is associated with a rapid decline of the papaya plant.

4.1.3 Epidemiology

Phytoplasmas are spread from plant to plant by phloem-feeding insects, typically leafhoppers and planthoppers. It was transmitted by the leafhopper *Orosius argentatus* Evans, but attempts to transmit the disease by mechanical inoculation have been unsuccessful. The pathogen was also transmitted by dodder (*Cuscuta australis* Hook.), but not by grafting from papaya to papaya (Cook, 1975).

The dieback-associated phytoplasma was detected 1 week prior to or the same week as symptom expression, while phytoplasma DNA was detected between 3 and 11 weeks prior to expression of mosaic symptom. Lateral shoot regrowth on the lower stem of plants that had suffered dieback disease failed to generate stolbur-specific PCR products (Guthrie *et al.*, 1998).

In the field young plants are more susceptible than older plants, and most extensive losses are experienced in the first 12 months following planting (Cook, 1975).

4.1.4 Control

To minimize losses that can range from 20 to 100% of production, producers in Australia utilize roguing and a practice of the cutting the plants at approximately 0.75m of height (ratooning) as soon the first symptoms of the disease appear.

This strategy of management is based on the principle of that the phytoplasma is located in the upper part of the plant, and ratooning and rouging reduce its potential dissemination (Guthrie *et al.*, 1998). Therefore, ratooning of dieback-affected plants and removal of yellow crinkle or mosaic-affected plants are suggested for the management of the disease.

4.2 Bunchy Top

Papaya bunchy top has been observed throughout much of the Caribbean region, from Cuba southward to Trinidad, since it was first reported in Puerto Rico in 1931 (Cook, 1975). Although the occurrence of the disease appeared to be restricted to the Caribbean islands, recent observations suggest that it is present in Central America and the northern part of South America. The disease has not been reported in Hawaii and Brazil. Bunchy top is a devastating disease and can severely limit commercial papaya production (Nishijima, *et al.* 1998a).

4.2.1 Symptoms

The first symptom of bunchy top is a faint mottling of the upper leaves. The laminae of infected leaves progressively become more chlorotic, especially in interveinal areas, and may eventually exhibit marginal necrosis. Leaves and petioles show reduced growth, and become rigid. Internodes become progressively shortened, and petioles tend to assume a more horizontal position. Oily appearing spots are often present in the upper parts of stems and in petioles. Apical growth ultimately ceases, which, with the shortening of internodes, imparts a "bunchy top" appearance to affected plants. With the cessation of apical growth and decline of plant vigor, the oldest leaves progressively abscise, leaving fewer, more stunted leaves at the top. Eventually, plants may be entirely killed; however, new shoots may sprout from the lower, non-affected portion of stems.

If fruits are set on infected plants, their flavor may be bitter due to the disease (Cook, 1975, Nishijima, 1998a). In plants with the dieback syndrome the absence of latex exudate from wounds was observed (Cook, 1975, Webb and Davis, 1987) and failure of latex to exude from wounds on diseased leaves, petioles, stems or fruits is a common characteristic of this disease.

4.2.2 Etiology

Although bunchy top was originally thought to be caused by a virus, the disease is most likely caused by a phytoplasma. The pathogen has been observed in the phloem of infected plants using transmission electron microscopy, and infected plants treated with tetracycline-based antibiotics exhibited a remission of symptoms (Rezende and Costa, 1993; Story and Halliwell, 1969). Although the absence of latex flow from fresh puncture wounds in leaves, petioles, stems, and fruits was considered diagnostic for bunchy top, a more recent study indicates that this is not a reliable diagnostic characteristic. The presence of phytoplasma in plants with foliar symptoms of bunchy top and normal latex flow was confirmed by electron microscopy.

4.2.3 Epidemiology

Two leafhoppers, *Empoasca papayae* Oman and *E. stevensi* Young, transmit the bunchy top agent, and the occurrence of the disease coincides largely with that of these vectors (Haque and Parasram, 1973; Seín and Adsuar, 1947; Story and Halliwell, 1969). *Empoasca papayae* has most frequently been associated with the disease, but in Trinidad, where *E. papayae* is not established, *E. stevensi* was found to be a vector. *Empoasca papayae* has rarely been found on other plant species and is the only leafhopper known to breed on papaya. *Empoasca stevensi* was originally described from specimens collected from papaya in Florida in 1940, but little is known about the ecology and distribution of this species. Interestingly, both bunchy top and *E. papayae* are not known to occur in Florida (Haque and Parasram, 1973; Story and Halliwell, 1969; Webb and Davis, 1987).

Bunchy top also can be also transmitted by grafting. Symptoms usually begin to appear 30 to 45 days after inoculation. Some papaya cultivars are more tolerant to the disease than others, but immunity is not known. The degree of tolerance affects the rate and severity of symptom expression. However, it is not known whether vector preference and the ability to transmit the bunchy top phytoplasma are affected by cultivar differences.

4.2.4 Control

Currently, the use of tolerant cultivars is the only practical means of control of bunchy top, but it has limited application. Where bunchy top occurs, tolerant cultivars can only be grown commercially in locations with low disease pressure. Disease pressure varies with geographic location, presumably due to insect vector ecology. Application of insecticide to reduce the incidence of leafhopper vectors may be beneficial (Nishijima *et al.*, 1998a).

In the past, the disease has been managed through a program involving leafhopper control throughout the productive life of the crop and the removal of sources of inoculum. The latter was attained by rouging infected trees or the topping of infected plants below the point where latex exudes after wounding. Axillary shoots that develop after topping are often free of the disease (Cook, 1975).

Antibiotic therapy is an effective control measure from an experimental standpoint but has not been applied commercially. Drenching the soil around infected plants with chlortetracycline or tetracycline hydrochloride and drenching combined with root-dip treatments were both successful. The efficacy of foliar application or trunk injection has not been established (Cook, 1975; Rezende and Costa, 1993).

5. Diseases caused by bacteria

5. 1 Bacterial Leaf Spot

Bacterial leaf spot, was first reported in the state of Rio de Janeiro, Brazil, in 1955 but since then has appeared in other countries (Cook, 1975), and more recently in the state of Paraná, Brazil, in the region of Paranavaí, in plants both in the nursery and in the field (Funada *et al.*, 1998).

The causal agent is the bacterium *Pseudomonas carica-papayae* Robbs, gram-negative, rod-shaped, with three to six polar flagella. Colonies on nutrient agar medium are circular, flat, gray white, and fluorescent. Neither ammonia, indol or hydrogen sulfide is produced, and nitrates are not reduced, and the optimum temperature for growth *in vitro* is 23-29°C (Robbs, 1956).

Initially the lesions caused by the pathogen are minute, circular to angular and

Figure 21: Symptoms of bacterial leaf spot, with coalescence of spots

of dark-green color, and water-soaked spots first appear on the lower surface of the leaves (Robbs, 1956; Funada *et al.*, 1998). These lesions later became light brown, or of chestnut shade, translucent, 3 to 6mm in diameter, and coalesce to form irregular necrotic areas on affected leaves (Fig. 21). On the lower surface of the leaf, in the local corresponding to the lesion and under conditions of high humidity, a milky exudation made up of bacterial exudate may be seen.

The disease has irregular occurrence and is of no economic importance, but when it occurs in young plants it can cause the death of the plants, principally in the nursery. Control should be preventative by means of sprays with products based on copper fungicides. Removal of affected plant parts and the roguing of diseased plants are also recommended. Utilization of seeds from orchards where the disease occurs is not recommended.

5.2 Purple Stain Fruit Rot and Internal Yellowing

In Hawaii, two bacteria (*Erwinia herbicola* (Loehnis) Dye and *Enterobacter cloacae* (Jordan) Horn. & Ed.) can cause rot of the pulp of fruits that affect the internal flesh of ripening fruits without causing easily noticeable external symptoms. Only purple stain fruit rot associated with *E. herbicola* occurs in sporadic form in the north of Espírito Santo, Brazil.

Internal yellowing is caused by *E. cloacae,* a gram negative rod-shaped bacterium, with peritrichous flagella, oxidase negative, catalase positive, and facultatively anaerobic. Both pathogens remain quiescent during fruit development until fruit are ripe, when symptoms are expressed.

When harvested, fruits infected by purple stain do not present external symptoms that easily differentiate them from healthy; however, when cut, a softening of the pulp with intense red coloration is observed, with decomposition of the tissues occurring, and with in some cases the red color accentuated in the vascular tissue next to the seed cavity (Fig. 22). Infected tissues of the flesh of the fruit become soft, translucent, and rotted as the fruit ripens (Nelson and Nishijima, 1980). Purple stain appears to be most common during the winter months, although incidence of the disease is usually very low and sporadic (Nishijima *et al.*, 1987). In the field, before harvest, it is possible to identify the infected fruits, which present a precocious maturation (out of position or normal order) on the plant and irregular distribution of yellow color on the skin, frequently associated a light deformation of the fruit.

The internal yellowing rot caused by *E. cloacae* causes a softening of the pulp, around the seed cavity, usually near the calyx and middle sections of the fruit, that becomes translucent or with a bright yellow to lemon-green color, with a rotting odor. The bacterium is associated with oriental fruit fly *Dacus dorsalis* Hendel, which is attracted to the bacterium and possibly transmits it to papaya flowers (Jan and Nishijima, 1990; Nishijima, 1998d). This disease still has not been reported in Brazil.

Based on their sporadic occurrence, these bacteria have little economic importance, however, infected fruits can contaminate the thermal treatment tanks, as well as installations of the packinghouse. Therefore, the elimination of the diseased fruits and the sanitation of the installations are recommended (Nelson and Alvarez, 1980; Nishijima, 1998d).

6. Diseases caused by nematodes

In the culture of papaya, gall nematodes (*Meloidogyne* spp.) and the reniform nematode (*Rotylenchulus reniformis* Linford & Oliveira), tiny soil-inhabiting worms, are the most important nematode species, principally when the orchards of papaya are planted in sequence with other hosts of these species, which can affect the development of the plants and cause the formation of galls in the radicular system, reducing the capability for absorbtion of nutrients and water. In Espírito Santo, the damage until present is minor and reports of these nematodes are restricted to limited foci without economic importance.

6.1 Root-knot nematodes

Damage to the radicular system of the plants depends on the level of infestation of the soil. The larvae penetrate into the radicular system causing hypertrophy of tissues (giant cells) forming galls and consequently a reduction of the root system and stunting of plants, which may be more sensitive than normal to water stress, with fruits

Figure 22: Longitudinal cross section of papaya fruit showing symptoms of purple stain with severe internal red coloration of the flesh.

smaller than normal and more likely to have an off-flavor. The symptoms are much less severe in loam or clay soils.

The species *Meloidogyne incognita* (Kofoid & White) Chitwood has been identified most frequently provoking galls in papaya plants.

Other species, however, like *M. javanica* (Treub) Chitwood, *M. arenaria* (Neal) Chitwood and *M. hapla* Chitwood, also have been reported infesting papaya in different countries, but occur less often. *M. hapla* prefers cool temperatures and may damage papaya trees grown at higher elevations. The larvae of these nematodes can travel short distances in soil, finding and attacking papaya roots. Root penetration is by second-stage juvenile, generally occurring near the root tip, and when the female begins feeding in the central cylinder region of the root, giant cells are formed. During the process of formation of the galls or knots, the females remain sedentary, embedded in the tissues of the root. Each females may produce more than 350 eggs, which are maintained in oothecas, and can give rise to of 14 to 17 generations in a year. To confirm a diagnosis, one can cut into the galls and observe pearly looking, pear-shaped female nematodes embedded in the tissue.

Owing to the biology and mode of dispersion of the nematodes, preventative control associated with management practices is most recommended.

Often seedlings infested in the nursery only manifest symptoms of the nematodes later in the field, depending on climatic conditions, principally temperature and humidity, and physical and chemical characteristics of the soil.

The use of organic material, non-host plants, and antagonistic or suppressive plants are alternatives that should be used to reduce the population of nematodes in the soil. The effects of root knot can be partially alleviated by maintaining plants at optimum water and nutrition levels. No nematicides are registered for use on papaya plants in Brazil and USA, and some common systemic nematicides are phytotoxic to papaya.

6.2 Reniform nematodes

Rotylenchulus reniformis Linford & Oliveira infests many plants from the tropics to temperate regions and on papaya causes leaf chlorosis and in some cases, stunting of plants. The plants may show some wilting during periods of peak transpirational stress on plants and the fruit produced on diseased plants are smaller than normal and may be slightly soft (Holtzmann and McSorley, 1998). The nematodes also cause lesions in the cortical tissue of the roots, which can serve as ports of entry for other pathogens.

Careful observation of the washed roots with a lens (10x), makes possible identification of small lesions and the presence of points of sand like bodies in the roots, which are egg masses of the nematode.

Dissemination of the nematodes occurs principally through contaminated seedlings, by cultural treatments, and by the surface runoff of rain or through irrigation water. The juvenile phases penetrate into the cortex of the roots and remain sedentary with part of the body outside of the roots, acquiring the form of a kidney (hence the name reniform). The nematode feeds near the phloem in papaya roots, sometimes inducing the formation of giant cells and a complete life cycle requires about 25-30 days. Adults produce a gelatinous matrix containing egg masses with the appearance of “grains of sand” used in the field diagnosis.

Preplant soil treatments have been used to reduce nematode populations and have resulted in increased vigor and yields. Nematicides registered for use on papaya orchards do not exist in Brazil, and recommendations for control have to be based on management practices. Some common systemic nematicides are phytotoxic to papaya plants.

7. Miscellaneous and abiotic diseases

7. 1. "Vira cabeca" or Apical necrosis

"Vira cabeça" or apical necrosis is a devastating disease that can limit commercial papaya orchards and in recent years has increased in incidence in the south of Bahia and north of Espírito Santo, Brazil principally in the first year of development of the plants. It is very similar to bunchy top disease that was first reported in Puerto Rico in 1931 and now occurs in Central and South America.

Transmission electron microscopy has been used to observe the cells and tissues of infected plants, and PCR work with primers used for phytoplasm diagnosis indicated negative and inconsistent results (Kitajima, USP-personal communication). So, the identity of the disease etiology still is currently in doubt (unknown etiology), but the epidemiological evidence suggests the involvement of an infectious agent possibly transmitted by insects. Evaluation of mechanical transmission by insects and by grafting is being evaluated by researchers of Incaper.In Australia a disease with similar symptoms and epidemiological characteristics has been associated with a phytoplasm (*see* phytoplasma diseases).

7.1.1 Symptoms

The initial symptom of "vira cabeça" is a faint mottling of the upper leaves involving one or more young leaves. The leaves become chlorotic, especially in the interveinal areas, and eventually exhibit marginal necrosis. The petioles are reduced and assume a horizontal position, with the internodes shortened with fewer, stunted leaves. All of the leaves of the apex of the plant become chlorotic, with poor growth, and subsequently they dry and fall (Fig. 23). Sometimes oily spots appear in the upper, apical part of the plant stem. Apical growth ultimately ceases, necrosis and curvature of the apex occurs, and the plant apex dies.In the more advanced stages of the disease the plants loose their leaves, develop extensive apical necrosis and finally die.

7.1.2 Epidemiology and Control

Roguing of symptomatic plants is recommended to reduce the inoculum source and disease incidence.

7.2 Freckles or Physiological fruit spot

The quality of papaya fruits produced in the north of Espírito Santo and in the state of Bahia, Brazil, have been seriously compromised by a disturbance known as freckles or physiological fruit spot, also referred to in the international literature as "freckle spot" (Ishii and Holtzmann, 1963; Nishijima *et al.*, 1998a).

The incidence and severity of freckles, which affect the external appearance of the fruit skin, have caused great damage for exportation, with losses that reach 50% of the fruits destined for exportation, principally in the period of when sunny days prevail

(May to October), The freckles are a cosmetic disorder, since are superficial and do not affect the flesh of the fruits that become depreciated visually for the fresh market. The etiology of freckles still is not well known, but experimental evidence points to a physiological disturbance (Eloisa *et al.*, 1994; Nishijima, 1998a). Temperature, associated with water deficiency, solar radiation, and the physiological state of the plant, appear to influence the incidence and intensity of the spots. These conditions are present during the period of greatest occurrence of freckle in the north of the Espírito

Figure 23: Symptoms of “vira cabeça” on papaya tree.

Santo.

In other fruits, low temperatures and the limitation of nocturnal transpiration in the fruits increases the potential turgor in the pericarp causing cracks (Aloni *et al.*, 1998). Physiological disorders have been associated with an imbalance of calcium in vegetative cells (Bangerth, 1979; Zambolim and Ventura, 1993), principally in the tissues with a more elevated respiratory rate, having an accumulation of Ca^{++}.

In the north of Espírito Santo, it can be observed that more vigorous plants, with better foliation, and apparently without problems of water deficiency have presented the least intensity of spots in the fruits, suggesting that probably these plants have greatest resistance to the effect of the temperature on the fruits, suffering less from solar exposure. It is known that heat stress can cause a reduction in photosynthesis and respiration of the cells as well as the liberation of electrolytes.

The side of fruits exposed to the greatest solar radiation consistently presents the greatest intensity of spots. This occurrence may be associated with the temperature on the surface, causing cellular lesions and consequently exudation of latex below of the epidermis of the fruits. The increase in turgor pressure under conditions of thermal amplitudes (fluctuations of temperature) also can cause the rupture of lactiferous vessels in the mesocarp of the fruits, provoking the spots.

The symptoms are small superficial spots that range from points to 10mm of diameter, with appearance of gray or brown freckles on the skin of the fruits, varying in the form and number on the fruits, that coalesce and cause the fruit surface to appear russetted (Fig. 24).

They are observed only in the fruits of more than 40 days of age and intensify in the final phase of development of the fruits, principally near the point of harvest. The larger spots generally present darker coloration with miniscule cracks, showing in some cases, in the margins, a soaked appearance, and can coalesce, affecting up to 50% of the exposed surface of the fruits. In post-harvest, when the fruits mature the freckles present a greenish coloration, contrasting with the yellow of the skin, reducing commercialization of the fruits.

For the growers, while research studies are not conclusive, carrying out the correct management of irrigation, preventing the plants from suffering water stress, as well as proper fertilization, with an adequate balance of nutrients, to reduce physiological stress to the plant, is recommended.

Mechanical protection of the fruits on the plants with sacks of paper or plastic was reported as a practice that reduced the incidence of the spots (Eloisa *et al.*, 1994), but still requires complementary research for its recommendation on commercial scale in conditions such as those of Espírito Santo, Brazil. Genetic improvement should also include the selection of plants with resistance to this disorder.

7.3 Bumpy fruit

Bumpy fruit is a physiological disorder common in all papaya-growing regions of the world, and is associated with a deficiency of boron, considered among the micronutrients most limiting to cultivation of papaya (Oliveira, 1999; Wang and Ko, 1975).

The lack of this element induces the plant to produce fruits with bumpy appearance, with the drainage of latex in different points of the skin frequently occurring as a result of the localized deficiency, which paralyzes the growth of the fruit and causes the rupture of the lactiferous vessels. Adjacent unaffected tissue continues to grow, resulting in a misshapen, bumpy appearance (Figure 25).

This symptom however, should not be confused with the drainage of latex caused by “Meleira”. The seeds of fruits with symptoms generally are aborted or poorly developed, enabling a darkening of the vessels to be observed. Under severe deficiency situations, growth of trees may be affected causing a slight rosette effect and an associated stunting in height.

In Hawaii, plants with fruits showing the symptoms of deformation generally present levels of boron in the petioles of the leaves (calculated based on dry weight) lower than 20 ppm, and normal boron level are 25ppm (Nishijima, 1998a). In Brazil, INCAPER-Instituto Capixaba de Pesquisa, Assistência Técnica e Extensão Rural, conducted nutritional studies in papaya fields in the north of Espírito Santo, and defined

Figure 24: Symptoms of freckles or physiological spot in fruits of papaya cv. Sunrise Solo

the standards of macro and micronutrients in the papaya petiole and leaves, with a mean B content in the petiole of 23.10 ppm in the wet season and 25.20 ppm during the dry season, and in the leaf lamina, 42.68 ppm and 44.26 ppm in the wet and in the dry seasons, respectively (Costa, A. N., INCAPER, personal communication). Applications of 0.25% borax ($Na_2B_4O_7.10H_2O$) or a mixture of 0.25% of boric acid (H_2BO_3) with 17% of B in a spray on the foliage have corrected the deficiency of this element and the young fruits discontinue manifestation of the symptoms (Oliveira, 1999; Nishijima, 1998a).

Deformed fruits cannot be brought back to original shape but fruits produced after treatment have normal shape and size.

Applications of boric acid to the soil (approximately 6g) on the area of the projection of the canopy also corrected B deficiency and increased petiole boron levels to prevent of deficiency symptoms on fruits subsequently formed, (however, complementary leaf sprays are also recommended). After application of boron and observation of the disappearance of symptoms in new fruits, a new leaf analysis should be made to

Figure 25: Bumpy symptoms on fruits of papaya cv. Sunrise Solo line 72/12 caused by boron deficiency.

determine the level adequate _for the specific conditions of production and cultivar used, to serve as a reference standard for subsequent analyses that should be conducted periodically.

7.4 Carpellody or Cat-face

Carpellody or "Cat-face" occurs when the flowers develop in an abnormal form as a result of a transformation of the stamens into carpel-like structures, and the fusion of these to the ovary early in the development of the flower (Marin and Gomes, 2000). After opening, the flowers become distorted producing deformed fruits known as "cat-face" or carpellody fruits.

Before blooming the carpellodic flowers present a form similar to feminine flowers (with the appearance of a "candle flame"), but formed on the axil of the leaves, which is a characteristic of the hermaphroditic flowers.

The appearance of carpellodic fruits can range from those that resemble female fruits to those that are severely deformed with longitudinal ridges or seams. Carpellodic fruits are generally rounded rather than the more typical pyriform shape and are unmarketable.

It is believed to be an abnormality of genetic origin, and influenced by environmental factors, like low night temperature, elevated moisture, and excess nitrogen in the soil (Marin and Gomes, 2000; Nishijima, 1998a).Since carpellody is an inherited trait, careful seed selection and orchard management can reduce its occurrence. The Solo cultivars have a lower incidence of carpellody. These cultivars, because of many years of inbreeding, have lower incidence of carpellody but it does occur occasionally under conditions described above (Nishijima, 1998a; Arkle and Nakasone, 1984).

7.5 Pentandry

Pentandry is a genetic anomaly known in hermaphroditic plants that may be influenced by the environment, and can occur, occasionally, in plants with masculine flowers. The flower develops five short stamens internally with the filaments inserted in furrows on the wall of the ovary. The fruits develop rounded or oval, very similar to those formed of female flowers, but present five deep, longitudinal grooves that them gives an unmistakable characteristic, lowering sale of the fruits that loose their commercial value (Marin and Gomes, 2000).

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