Melanose and Phomopsis Stem-End Rot of Citrus

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Cause

The fungus *Diaporthe citri* causes melanose and stem-end rot. The superficial blemish on fruit associated with melanose creates a cosmetic problem on fruit intended for the fresh market. Juice quality and quantity are not affected. Stem-end rot of fruit occurs 10-20 days after harvest during storage or transit most commonly in the latter part of the harvest season (Jan. -June). Because fruit intended for processing are usually not held for more than one week, stem-end rot is of little consequence on fruit for processing. Melanose and stem-end rot are of consequence only for fresh market citrus.

The fungus *Diaporthe citri* produces spores called ascospores (sexually produced) and pycnidiospores (asexually produced). This latter spore type is the part of the life cycle that provides most of the inoculum for disease and often is referred to as *Phomopsis citri*. Ascospores are formed within a vesicle-like structure on decaying wood on the soil or on dead branches remaining on the tree or in brush sites. These spores are produced in relatively small numbers and contribute slightly to disease potential within a grove. Their main contribution to disease development relates to spread of the fungus over long distances because ascospores are windborne.

Pycnidiospores, on the other hand, are produced abundantly on dead branches within a flask-shaped structure (pycnidium). Because they are released within a mucilage, they provide for short distance spread within a tree or from one tree to an adjacent tree by rain or irrigation splash. Even more restricted movement of these spores can occur by rain or irrigation water washing over infected branches and dripping onto leaves, fruits and twigs below, carrying the spores in a passive fashion. Nursery trees could be infected with the melanose fungus and carry inoculum to the new site. Spores of this fungus are produced on dead wood only, not on melanose pustules found on leaves, fruit, or live branch tissue. Therefore, freeze-damaged citrus trees, older groves, and poorly maintained groves with much dead wood should be considered high melanose incidence areas. All types of citrus are susceptible. Other hosts for this fungus have not been identified.

Leaves become resistant to infection once they are fully expanded. Fruit rind becomes resistant about 12 weeks after petal fall, but the later infection occurs during that 12 week period the smaller will be the resulting pustules. Thus, even though suitable weather conditions exist for infection after late June, melanose will not infect the fruit rind after that time except in years when the bloom is later than normal.

Temperature and moisture conditions during

periods of leaf expansion and during the first 12 weeks after petal fall regulate disease severity. After a spore lands on susceptible tissue, a period of 10-12 hours of moisture is required for infection at 77°F (25°C) while at 59°F (15°C), 18 to 24 hours of wetness are necessary for infection. Thus, extended wet periods resulting from afternoon rain showers plus dew periods in May and June coupled with warmer temperatures during these months create favorable weather for infection. In contrast, rainfall prior to May in central and south Florida is associated with fast-moving cold fronts that are quickly followed by temperatures less favorable (too cool) for infection. Also, winds behind the front quickly dry surface moisture on plant tissue. At temperatures between 75-82°F (24-28°C) initial symptoms can occur 4 to 7 days after infection.

When temperature and moisture are considered in relation to rind susceptibility, May and June are normally high-hazard months for melanose infection. Occasionally, March or April can support favorable weather for melanose infection. The shoots of the spring growth flush usually emerge in March during unfavorable weather for infection. Should a late bloom occur, fruit remain susceptible into July, a month that can be expected to be a highhazard period because of favorable weather.

Symptoms

Leaf symptoms begin as tiny watersoaked specks that become depressed in the center with a surrounding translucent, yellow area that is not depressed (Fig 1). Later, the leaf cuticle ruptures and a gummy substance is exuded which turns brown and hardens (Fig. 2). The yellowish margin disappears and the hardened gummed areas will have a sandpaper-like feel. Infected areas on the leaf may be scattered, aggregated or in streaks, depending on where water transported the spores prior to infection. Heavily infected leaves become pale green to yellow, can be distorted in shape (Fig. 3), and may fall from the tree. Melanose is seldom severe on the spring growth flush. When it does occur on this flush, the pustules are usually few in number and little or no leaf drop occurs. On the summer growth flushes, melanose can be severe enough to cause serious defoliation, particularly in years following freeze-induced twig dieback.

Fruit symptoms are of two types. The melanose symptom occurs in the field. Melanose symptoms on fruit are similar to those found on leaves, but there is a greater tendency for the diseased areas to be streaked from the top to the bottom of the fruit with regards to fruit orientation on the tree (Figs. 4 & 5). Fruit infected when young may remain small and abscise prematurely. Late infection produces small pustules (Fig. 6). If the severity is such that solid patches of blemish are produced, the fruit surface can crack producing a roughened condition sometimes called mudcake melanose (Fig. 7). Mudcake melanose occurs if infection takes place soon after petal fall (usually early April). Melanose on fruit can be distinguished from rust mite injury by the presence of a roughened surface with melanose and a smooth rind blemish with rust mite injury. Color is not a reliable characteristic for distinguishing melanose from rust mite blemish.

Stem-end rot is the second fruit symptom associated with this disease and it occurs as a postharvest decay in the fresh market trade where long-term handling, storing and transporting are involved. Hyphae (growth filaments) of this fungus can be present in necrotic tissue of the calyx; then, after harvest, penetration can occur through the abscission area during periods of suitable temperatures and moisture. Symptoms usually begin to appear after 10 days. The infected area is soft, tan or brown (Fig. 8). The internal core of the fruit becomes infected and turns dark. Later season fruit are more apt to have stem-end rot than early season fruit.

Control

Control of melanose and stem-end rot is necessary for fruit intended for the fresh market only. Melanose is best controlled by using a combination of techniques. Pruning and burning dead wood will reduce inoculum (spore supply). Regardless of how conscientiously the prune and burn technique is carried out, fungicide spraying will still be necessary to produce blemish-free fruit. Fungicides could be applied prior to the initiation of spring growth, which generally occurs in late February. Because no susceptible tissue (leaf or fruit) is present at this time, a fungicide applied while trees are dormant must be able to either inhibit spore production or inactivate spores that are produced on dead wood. The fungicide applied during dormancy must remain stable over time and be redistributable. These two characteristics will be beneficial in allowing the fungicide to remain toxic to the spores over time and move in water with the spores. For example, Difolatan had these characteristics when used at high but costly rates. Copper fungicides effectively protect fruit rind tissue from infection as postbloom sprays but are ineffective as a dormant spray because they do not reduce spore numbers and they are not redistributed sufficiently. Benlate reduces spore reduction to some extent but it does not redistribute effectively. Also it does not function as well as a protectant fungicide against melanose. Resistance to Benlate now exists in this fungus and thus its effectiveness of any degree has been lost. Therefore, Benlate is not adequate for melanose control. For current fungicide recommendations obtain a copy of the Florida Citrus Pest Management Guide (SP-43).

Stem-end rot is reduced by utilizing numerous control measures in the field during production and during harvest and postharvest handling periods. Because the calyx of immature fruit on the tree is also infected during periods of melanose infection, fungicide sprays and removal of dead wood (source of spores) for melanose control will contribute to Phomopsis stem-end rot control. Such beneficial effects may not show up until the fruit is harvested (possibly months later) and stored at cooler temperatures. Warm temperatures associated with ethylene degreening after harvest are more favorable for development of *Diplodia natalensis*, the other stem-end rot fungus, than for growth of *Phomopsis*. *Phomopsis* becomes more prevalent than *Diplodia* later in the season from January to June when naturally colored fruit do not require degreening. Also, the incidence of Phomopsis stem end rot will increase in fruit grown on older trees where more inoculum is likely to be present.

In the packinghouse the use of fungicides in a postharvest application are necessary regardless of prior control measures. Application of fungicides with water is superior to the incorporation of fungicides in the wax. Throughout harvest and postharvest periods, fruit should not be handled in a rough fashion.

Figure 1. Early leaf spots of melanose.

Figure 2. Pustules of melanose in leaf.

Figure 3. Melanose in grapefruit leaves associated with source of spores on dead branches above leaves.

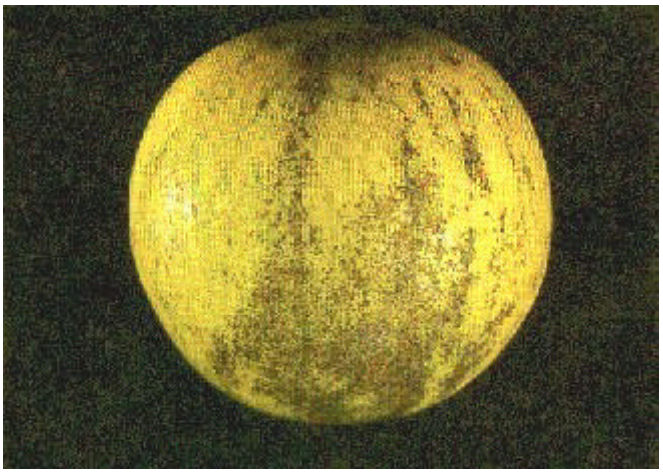

Figure 4. Streaked melanose on grapefruit caused by spores moving in water down the fruit.

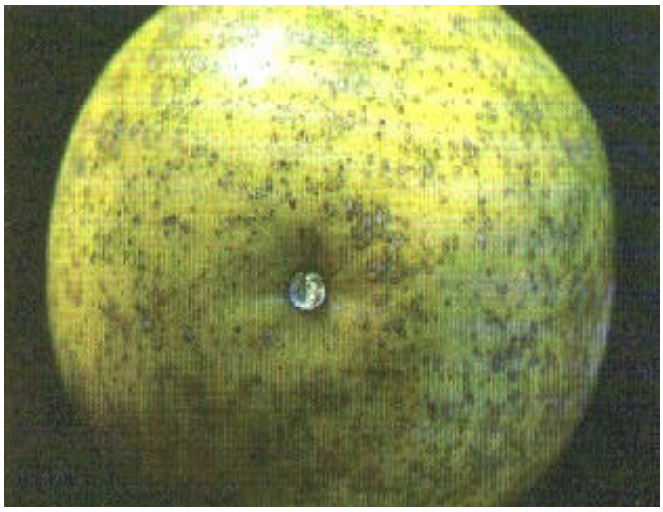

Figure 5. Generalized distribution of melanose on grapefruit when infection occurs in May or June.

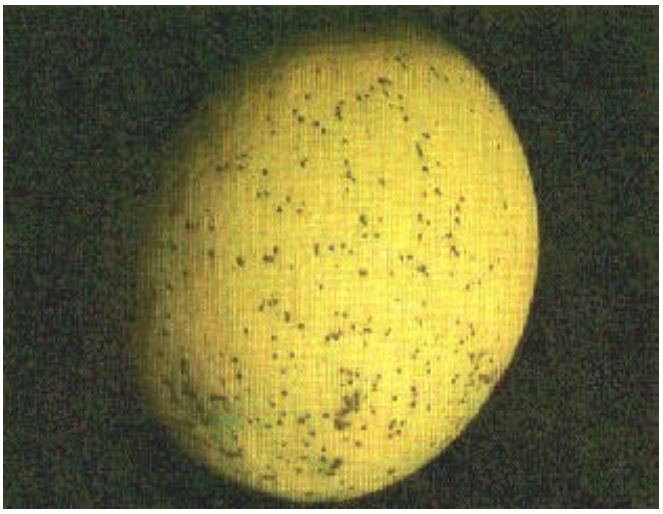

Figure 6. Smaller melanose pustules associated with infection occurring while fruit rind is becoming less susceptible.

Figure 7. Mudcake melanose.

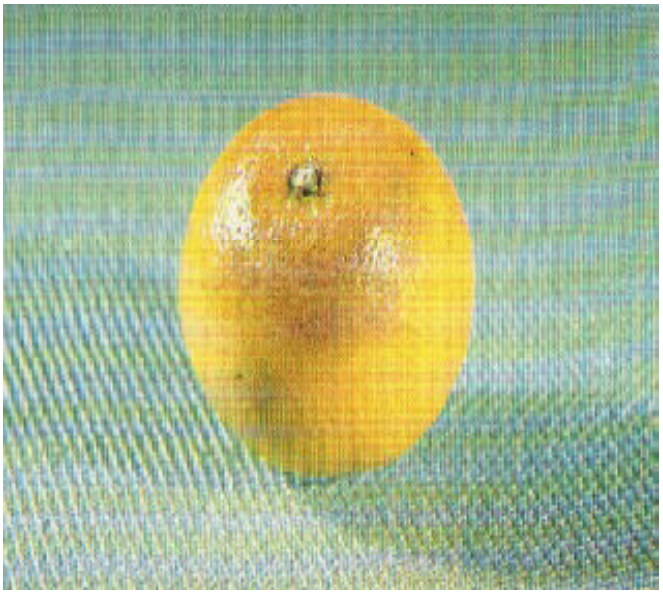

Figure 8. Stem-end rotsymptom.