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POSTBLOOM FRUIT DROP OF CITRUS—SYMPTOMS, DISEASE CYCLE AND CONTROL[1]

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Abstract. **Postbloom fruit drop (PFD) appeared in Florida in 1983 and has since become widespread in the state. The disease appears as necrotic spots on petals and produces fruit drop and the formation of persistent buttons. PFD is caused by the slow-growing orange (SGO) strain of *Colletotrichum gloeosporioides* which is readily distinguished pathologically, morphologically and physiologically from the common, saprophytic fast-growing gray strain. However, the SGO strain is nearly identical to the strain of *C. gloeosporioides* which causes Key lime anthracnose. All isolates from Key lime tested caused PFD. The fungus is spread primarily by rain splash of conidia produced on infected petals. It appears to overwinter as appressoria on the surface of leaves, twigs and buttons which germinate at flowering to produce new conidia. The only fungicide currently registered for control of PFD is benomyl. An equation has been developed based on the current number of affected flowers and rainfall for the past five days to predict disease incidence and assist in timing of fungicide applications.**

In 1983, a new disease—postbloom fruit drop (PFD) hit Florida citrus groves for the first time. Initially, it was confined to Tahiti lime plantings and a few sweet orange groves in the Immokalee area. Subsequently, outbreaks occurred in various areas with damage ranging from minor to locally severe. Rainfall was high during the bloom period in 1988 and damage was more widespread and more severe. The causal agent, a strain of *Colletotrichum gloeosporioides* (Penz.) Penz. and Sacc. in Penz., had become widely distributed and significant outbreaks occurred on the East Coast, South Ridge, and North Ridge production areas. Rainfall during the blossom period was minimal in many areas in subsequent years and outbreaks were localized. In 1993, the bloom period was extended and rainfall abundant throughout citrus production areas through March. Losses were severe particularly on Navel oranges and some Valencia plantings, but some damage occurred on most citrus types.

The disease was described first in Belize by Fagan (1979) and most of what was known about PFD was from research conducted there (Denham, 1979; Denham and Waller, 1981; Fagan, 1971, 1979, 1984a, 1984b). Following the 1988 outbreak, my laboratory initiated an extensive research program to determine the nature of the causal organism, the disease cycle, the effects of environmental factors on the disease and to develop effective control measures. Much of that research is summarized herein.

Symptoms

Symptoms of the disease first appear as peach to brown-colored necrotic spots on petals of flowers which

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have recently opened or are about to open. If the disease is severe, entire flower clusters become brown, even pinhead bloom may be attacked, and the petals remain clinging to the inflorescence. Subsequently, petals and small fruitlets drop leaving the calyx and floral disc. These structures, commonly called buttons, persist for the life of the twig. These buttons are characteristic of PFD and are not known to be produced by any other disorder.

PFD symptoms are relatively easily distinguished from normal petal senescence and drop. Normal petals dry from the tip toward the base of the petal and become a light tan color in contrast to the peach or brown color of PFD affected petals. Circular necrotic spots typical of PFD almost never occur on normal petals. Healthy petals abscise readily whereas diseased petals cling to the inflorescence.

The Causal Agent

Postbloom fruit drop is caused by a specialized strain of *Colletotrichum gloeosporioides* [perfect stage: *Glomerella cingulata* (Stonem.) Spauld and Schrenk] now known as the slow-growing orange (SGO) strain (Agostini et al., 1992; Sonoda and Pelosi, 1988). It differs considerably from the common saprophytic form of the fungus which has always been common in Florida citrus groves. The common form now known as the fast-growing gray strain (FGG) is readily differentiated from the SGO strain by fungal morphology (Agostini et al., 1992), physiology (Gantotti and Davis, 1991; Liyanage et al., 1993), genetic characters (Liyanage et al., 1992) and by pathogenicity (Agostini et al., 1992).

Interestingly, the SGO strain is almost identical to the fungus originally described as *Gloeosporium limetticolum* Clausen which causes anthracnose of Key lime (Clausen, 1912). That fungus is now included taxonomically within *C. gloeosporioides* (Sutton, 1980). Isolates of this fungus from Key lime produce blossom blight and persistent buttons just as do SGO strains isolated from PFD affected flowers. SGO isolates do not produce typical Key lime anthracnose symptoms, but cause mild chlorotic spotting on young Key lime leaves (Agostini et al., 1992). We have speculated that postbloom fruit drop may have developed when the fungus moved from Key limes into sweet orange groves and attacked blossoms (Agostini et al., 1992). Thus, widespread appearance of the disease in many countries in Central and South America and the Caribbean may be attributable to movement of the fungus from Key lime rather than to separate introductions from foreign sources.

The Disease Cycle

The SGO strain produces large quantities of single-celled conidia in a gelatinous mass in acervuli, pin-cushion-like structures, on the surface of diseased petals. These spores are dispersed by rain splash to nearby healthy bloom. Infection occurs within 12 hr and symptoms appear in 2-3 days. By 5 days after infection, newly affected petals are producing large quantities of inoculum. With the large number of conidia produced and a short cycle, PFD can very quickly become epidemic in a grove under favorable environmental conditions.

Until recently, the means of survival of the SGO strain between bloom periods was unknown. The saprophytic FGG strain produces appressoria, small thick-walled structures, on the surface of living leaves or other tissues and penetrate the surface two or three cells deep. These quiescent infections can persist indefinitely but the fungus does not begin to grow until the tissue becomes senescent (Brown, 1977; Whiteside, 1988). The FGG strain then colonizes the dying tissue and produces conidia in acervuli on dead tissue. Conidia of the SGO strain germinate to form appressoria on the surface of living tissues as do those of the FGG strain. These appressoria on the leaves persist for long periods but the number decline with time in the absence of flowers (Agostini, 1992; Agostini and Timmer, 1992). In contrast to the FGG strain, appressoria of the SGO strain germinate in the presence of moisture and blossom extracts to produce a few conidia which are borne singly on hyphae (Agostini, 1992; Zulfigar, 1993). These few spores then serve as inoculum for the first few flowers which appear in the spring reinitiating the disease cycle. Thus, we believe that the SGO strain survives on the surface of living tissue rather than colonizing dead tissue as the FGG strain does. After severe outbreaks, high concentrations of inoculum persist on the buttons, leaves and twigs around infected flower clusters.

Environmental Effects

The optimum temperature for growth of the SGO strain in culture is 75-80F (Agostini et al., 1992; Fagan, 1979). In the grove, low temperatures slow disease development but also slow blossom development. Thus, cool temperatures do little to reduce final disease severity. In contrast, warm, dry conditions speed flower development and fruit set while restricting disease progress.

PFD is highly dependent on moisture. Free water and high humidity are needed for infection but, more importantly, rain splash and wind-blown rain are needed for dispersal of conidia. We have observed some infection of flowers associated with heavy dews and fogs, but rainfall is essential for development of epidemics. Fagan (1979), in Belize, found a consistent association of disease severity with periods of high rainfall. Likewise, in Florida, disease incidence rises rapidly several days after a rain if sufficient inoculum is present (Timmer and Zitko, 1992, 1993b). PFD declines quickly after 10 days to two weeks of dry weather.

Disease Control

Adequate control of PFD is highly dependent on timely applications of fungicides during the bloom period. Based on our studies of factors which affect disease severity, we have developed an equation for prediction of disease incidence 3-4 days in advance (Timmer and Zitko, 1993a,b). The factors used in the equation are the number of diseased flowers present currently on 20 trees and the total rainfall which has occurred during the past five days. The other factors considered—leaf wetness, temperature, relative humidity, and wind speed—played only minor roles or had no effect on disease development. This predictive equation and its use have been described in detail elsewhere (Timmer and Zitko, 1993a,b).

Currently, we recommend fungicide applications be initiated when there is sufficient bloom present to represent a significant portion of the crop and the amount of disease predicted by the above model is greater than 20%. Benomyl (Benlate 50WP), the only fungicide currently registered for control of PFD, is effective when properly used.

This fungicide acts by three means: i) it protects already open flowers from infection, ii) being systemic, it is capable of eliminating already existing infections, and iii) most importantly, it reduces the inoculum on diseased flowers thus protecting flowers which have not yet developed. An application is usually effective for at least 10-14 days at which time another assessment must be made as to whether a second application is needed. Fungicide applications need to be initiated early in the bloom if conditions indicate. If the disease is allowed to reach high levels, it becomes very difficult to control and the possibility of selecting strains resistant to benomyl is great.

Many fungicides have been evaluated for control of PFD. In tests in Belize, benomyl and captafol (Difolatan 80 Sprills) were the most effective; whereas, captan and maneb showed lower levels of activity (Fagan, 1971, 1984b). In laboratory tests, most of the sterol biosynthesis inhibiting fungicides were active at very low concentrations (Zitko and Timmer, 1992) and propiconazole (Tilt) has shown some effect in field tests in Florida (Timmer and Zitko, unpublished). Most other currently available fungicides have little or no activity against this disease.

The current recommendation is to apply Benlate at 2 lb/acre. Applications can be made with ground equipment or by air (Fagan, 1984b; Timmer and Zitko, 1991) if there is a need to cover large acreages in a short period. The combination of Benlate at 1.5 lb/acre plus 5 lb/acre of Carbamate 76W (ferbam) has been effective for disease control (R. M. Sonoda, personal communication). The addition of ferbam helps reduce the possibility of selecting benomyl-resistant strains.

Other measures have sometimes proved helpful in reducing PFD severity. Overhead irrigation promotes the disease and switching to undertree microsprinklers has reduced disease problems in some groves. Declining trees tend to be severely affected by PFD because they often flower off-season and maintain high levels of inoculum. Removal of these declining, nonproductive trees prior to the bloom appears to have facilitated control in some groves.

Yield Effects

Losses to PFD tend to be overestimated because of the large numbers of buttons produced. Fungal infection induces the formation of a button regardless of whether the flower would have actually set a fruit or not. Since less than one percent of the flowers actually set fruit, a corresponding percentage of the buttons would have been expected to produce fruit. In addition, we have found that trees tend to compensate for fruit lost to PFD by dropping less fruit during the period of normal physiological drop in May or June (Timmer and Zitko, 1992). Trees with fewer fruit also tend to produce larger fruit. In some experiments, up to 20-25% blossom blight has been observed at some times during bloom without reducing final yield.

In contrast, when the disease becomes epidemic, yield loss may approach 100%. When inoculum levels are high, flower buds may be infected and abscise without the formation of buttons. When crop yields are low, greater fruit size may be detrimental rather than advantageous.

There are risks involved in both conservative and liberal approaches to control of PFD. If fungicides are applied at the first sign of disease and continued through the bloom at regular intervals, a high degree of control can be expected. But, in many blocks and in many years, no benefits would be realized. In addition, there would be an increased risk of selection of benomyl-resistant strains. On the other hand, PFD can be an explosive disease and a high percentage of the bloom may be affected in a short time. The disease becomes difficult to control if a high percentage of the bloom is affected. The consequences of a severe attack of PFD are great. If a large percentage of the crop is lost, all other inputs for irrigation, weed control, and fertilization will result in no gain for the year. Harvesting costs may also increase if the fruit crop is light. In addition, severe infection produces large amounts of inoculum for the subsequent crop year and may result in continuing problems.

I prefer to follow a conservative approach early in the bloom period to try to keep the disease from getting out of hand. Late in the bloom period once a substantial amount of fruit has been set, one can risk higher percentages of disease without risking the entire crop. Attention should be focused on those blocks with the most serious problems and several applications made if necessary. Widespread precautionary spraying of entire plantings with minor problems is not usually advisable.

PFD is different from most of the diseases we deal with in Florida. Close attention to the amount of bloom, the percentage affected by the disease and rainfall data is essential. With vigilance and timely applications of fungicide, PFD can be managed and some past disasters avoided.

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AN UPDATE ON POSTBLOOM FRUIT DROP IN THE INDIAN RIVER AREA AND RESULTS OF A FUNGICIDE TEST ON THE DISEASE

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Abstract. **Observations on the incidence of postbloom fruit drop (PFD) in 3 navel orange groves in the Indian River area from 1988 to 1993 are presented. The extent and severity of PFD in the Indian River area in 1993 is discussed. A combination of benomyl and ferbam applied in a commercial grove was more effective than benomyl alone in reducing blossom infection.**

Postbloom fruit drop (PFD) of citrus caused by *Colletotrichum gloeosporioides* (Penz.) Penz. & Sacc. in Penz. was first noticed in the southern portion of the Indian River citrus-growing area, in Martin county, in the spring of 1987. The disease was first reported in Florida, in southwest Florida, in 1984 (McMillan and Timmer, 1988). By the spring of 1988, the disease was observed in groves throughout Martin, St. Lucie and Indian River counties in an area encompassing over 90% of the citrus in the area. A severe outbreak of PFD occurred in the spring of 1993. Plantings of navel orange and to a lesser extent, Valencia orange, were the most often affected by the disease. No variety was unaffected. Incidence of postbloom fruit drop in several groves in Indian River county was monitored from spring 1988 through summer 1993 and a summary of the observations is presented. A summary of the spring 1993 PFD outbreak is also presented.

Initially, information developed in Belize, Central America by Fagan (1984) was used as a basis for fungicide applications in the Indian River area. Fagan had determined that of the compounds available in 1972, benomyl and captafol (Difolatan) were the most promising. The combination of benomyl and captafol appeared to be more effective than either alone. Captafol has since become unavailable. Timmer, more recently, (Timmer and Zitko, 1992) has found that benomyl can be effective under Florida commercial grove conditions. However, several applications may be required, especially when bloom periods are long. There is also a risk that continuous use of benomyl will result in the development of benomyl resistance, as in the case of benomyl resistance in the greasy spot and scab diseases of citrus in Florida. No other fungicides, effective against PFD are yet available. Sonoda et al. (In press) found in a small-scale field test that a greater reduction in the blossom blight phase of PFD resulted when ferbam was combined with benomyl. The use of combinations of fungicides, in addition to being more effective against a disease, may prolong the use of a fungicide when there is a risk of resistance development. Larger scale field testing of the combination of benomyl and ferbam was conducted in the spring of 1993 and the result of one of the studies is reported here.

Postbloom Fruit Drop in the Indian River Area From 1988 to the Present

Bloom 1988

PFD was severe in several plantings of navel oranges in the spring of 1988 and it was present on other citrus varieties in many groves in Martin, St. Lucie and Indian River counties. In the spring of 1988, 3 navel orange groves in Indian River county, severely affected by PFD, were selected to determine the effect 'buttons' (persistent calyces

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