L. W. Timmer
University of Florida, Citrus Research and Education Center, Lake Alfred

J. P. Agostini
Instituto Nacional de Tecnología Agropecuaria, Montecarlo, Misiones, Argentina

S. E. Zitko
University of Florida, Citrus Research and Education Center, Lake Alfred

M. Zulfiqar
Agriculture Research Institute, Tarnab, Peshawar, Pakistan

Postbloom Fruit Drop, an Increasingly Prevalent Disease of Citrus in the Americas

Until recent years, postbloom fruit drop (PFD) of citrus was a problem limited to a few valleys where citrus is grown in Belize in Central America. The first description of the disease and its causal organism was published by Fagan in 1979 (13). However, characteristic symptoms of the disease had been reported as early as the 1950s in Belize. Few subsequent reports appeared, and the disease seemed to be of limited, local importance.

Currently, the disease appears to occur widely throughout the humid tropics and subtropics of the Americas. Schwarz et al (24) in 1978 reported the disease in Misiones in Argentina. Denham (10) reported that the disease had been observed on the Caribbean island of Dominica and in Panama by 1976 and in Brazil by 1977. Orozco Santos and Gonzalez Garza (22) studied the disease in Mexico in the mid-1980s, and it was probably present there much earlier. PFD is common in Costa Rica, and outbreaks occurred in the mid-1980s and the disease was widespread by 1990 in Jamaica and the Dominican Republic (L. W. Timmer, *personal observations*). In most cases, PFD appeared suddenly where it had been unknown previously. Where frequent rain occurred during the bloom period, crop loss approached 100% in some locations. However, the disease has occurred sporadically and has been only a minor problem in some years and locations.

Dr. Timmer's address is University of Florida, Citrus Research and Education Center, 700 Experiment Station Road, Lake Alfred, FL 33850.

Florida Agricultural Experiment Station Journal Series No. R-03567.

In Florida, PFD appeared first on Tahiti limes (*Citrus latifolia* Tanaka) in 1983 in southwestern production areas of the state (21). Subsequently, the disease was observed on sweet orange (*C. sinensis* (L.) Osbeck) and other citrus throughout the state. Major outbreaks in 1988 and again in 1993 caused concern among growers.

Symptomatology

The disease appears first as necrotic peach to orange-colored lesions on open flowers (Fig. 1A). Although unopened and even pinhead flower buds may be affected (Fig. 1B), petals on open flowers are more susceptible to infection (1,11, 13). Whole flower clusters may be attacked, leaving entire branches with orange to brown petals clinging to inflorescences (Fig. 1C). After petal fall, the calyces and floral disks, which normally abscise if no fruit is set, usually remain attached to the twig (Fig. 1D). These persistent structures, commonly called buttons, survive for the life of the twig. The buttons are characteristic of the disease and are not known to be produced by any other disorder. Leaves surrounding infected flowers are often distorted, with twisted laminae and enlarged veins (Fig. 1D).

Colletotrichum spp. on Citrus

The fungus *Colletotrichum gloeosporioides* (Penz.) Penz. & Sacc. in Penz. has been associated commonly with citrus in virtually all humid growing regions. *C. gloeosporioides* usually is considered to be a saprophyte producing acervuli on dead or senescent tissue but may be a weak parasite. Conidia from these acervuli germinate on living tissue to form appressoria and quiescent infections (7,33). To complete its life cycle, the fungus invades senescing tissue and produces acervuli. Brown (7,8) found that *C. gloeosporioides* produced postharvest anthracnose on fruit that had many appressoria and was exposed to stresses such as ethylene degreening after harvest. The strain of *C. gloeosporioides* that causes postharvest anthracnose has been designated as the fast-growing gray (FGG) strain (5,25). The FGG strain is not pathogenic to flowers or to living vegetative tissues of citrus and is not responsible for PFD (5).

Anthracnose and wither tip of Key lime (*C. aurantifolia* (L.) Swingle) were described originally by Clausen (9) in 1912. This disease produces necrotic spots on young leaves, twigs, and fruit of Key lime and, if severe, blights entire shoots. The causal agent was described originally as *Gloeosporium limetticola* R.E. Clausen, but this group has been revised and the pathogen is now considered a form of *C. gloeosporioides* (26,30). This fungus was reported to attack no citrus other than Key lime (16), but only vegetative tissues were inoculated.

PFD was attributed originally to a specialized form of *C. gloeosporioides* (13) that is now referred to as the slow-growing orange (SGO) strain (5,25). The SGO strain is readily distinguished from the FGG strain. The FGG strain grows faster and forms gray-pigmented colonies in culture, has larger conidia that are mostly rounded at the apex and base, produces setae in culture and on host tissue, and has lobulate appressoria averaging 6.0 × 8.4 μm (5). In contrast, the SGO strain is slower growing and produces mostly white mycelia with orange conidial masses, has smaller conidia mostly fusiform at the apex, rarely produces setae, and forms smaller, clavate appressoria averaging 4.7 × 6.1 μm. A medium and procedure have been developed to selectively isolate and differentiate the SGO and FGG strains (4).

Morphologically, the Key lime anthracnose (KLA) strain is difficult to

distinguish from the SGO strain (5), differing only in having smaller, round appressoria and more crenate colonies with brighter pink conidial masses.

The FGG strain produces only postharvest anthracnose on fruit and does not cause PFD or Key lime anthracnose (5). Surprisingly, the KLA strain produces all of the symptoms of PFD when flowers of sweet orange and other species are inoculated (5). Conversely, SGO strains from PFD-affected sweet orange petals usually cause only mild chlorotic spotting of young Key lime leaves. It appears, on the basis of the pathogenicity of the different strains, that the SGO strain may have arisen from the KLA strain and that PFD may be the result of invasion of plantings of other citrus species by the KLA strain. Key limes are widely grown in tropical areas but in humid climates, because of the severity of KLA, are mostly confined to dooryard plantings. Once introduced into orchards of sweet orange, the KLA strain may cause PFD and then lose its pathogenicity to Key lime. If the SGO strain is the result of escape of the KLA strain from dooryard plantings of Key limes to orchards of other citrus, the relatively sudden widespread appearance of PFD might be explained.

Liyanage et al (20) found that the SGO and FGG strains also differed in ribosomal DNA and in chromosome number. Cutinases of these two strains

Fig. 1. Symptoms of postbloom fruit drop: Necrotic lesions on (A) open flower and (B) unopened flower buds, (C) severely affected flowers with petals still clinging to the floral base, and (D) persistent calyces (buttons) formed as a result of flower infection.

differ greatly (19), and Gantotti and Davis (17) found several differences in other isozymes as well. Unfortunately, KLA strains were not included in these studies. The SGO and KLA strains might warrant taxonomic separation from other *C. gloeosporioides* strains because of their great morphological, pathological, and genetic differences. Any reclassification, however, should await determination of the relationship of the KLA and SGO strains to similar strains from other hosts (6,18).

Disease Cycle of PFD

Conidia of the SGO strain are produced in abundance in acervuli on infected flowers (Fig. 2A). These conidia are splashed onto surrounding leaves and twigs and on the buttons that remain after the blossom period. Originally, the SGO strain was thought to reproduce primarily on dead tissues much as the FGG strain does (11). However, the two strains behave quite differently on the leaf surface. When conidia of the FGG strain are applied to the leaf surface under favorable conditions, most germinate and form appressoria. With the SGO strain, many conidia survive ungerminated for at least a month (1,3), but once spores germinate, most form appressoria (Fig. 2B), and inoculum remains available to attack later blooms even after petals have fallen.

The behavior of the appressoria of the FGG and SGO strains differs dramatically. Appressoria of the FGG strain produce quiescent infections that develop only when the leaf tissue weakens or senesces. Appressoria of the FGG strain do not respond to moisture or to applications of nutrients such as petal extracts (1,3,35). In contrast, in the presence of petal extract and moisture, appressoria of the SGO strain germinate and produce hyphae and conidia on the leaf surface without producing acervuli (Fig. 2C).

Figure 3 depicts the disease cycle of PFD. Conidia are produced in acervuli on flower petals and are splash-dispersed during the current bloom periods. Citrus often has secondary blooms during the spring and early summer, depending on climatic conditions. Free conidia on the leaf surface may be a source of inoculum for those flowers. The appressoria on vegetative surfaces are the primary survival structures between blooms. When bloom begins the following spring, nutrients washed from the first flowers onto the leaves stimulate germination of appressoria and production of conidia without forming acervuli. These conidia are splash-dispersed to flowers, where they penetrate petals directly without forming appressoria (35) to reinitiate the cycle. In vitro, the SGO strain readily colonizes detached, senescent leaves and produces acervuli. In nature, we have not observed acervuli of the SGO strain on dead leaf tissue or recovered high

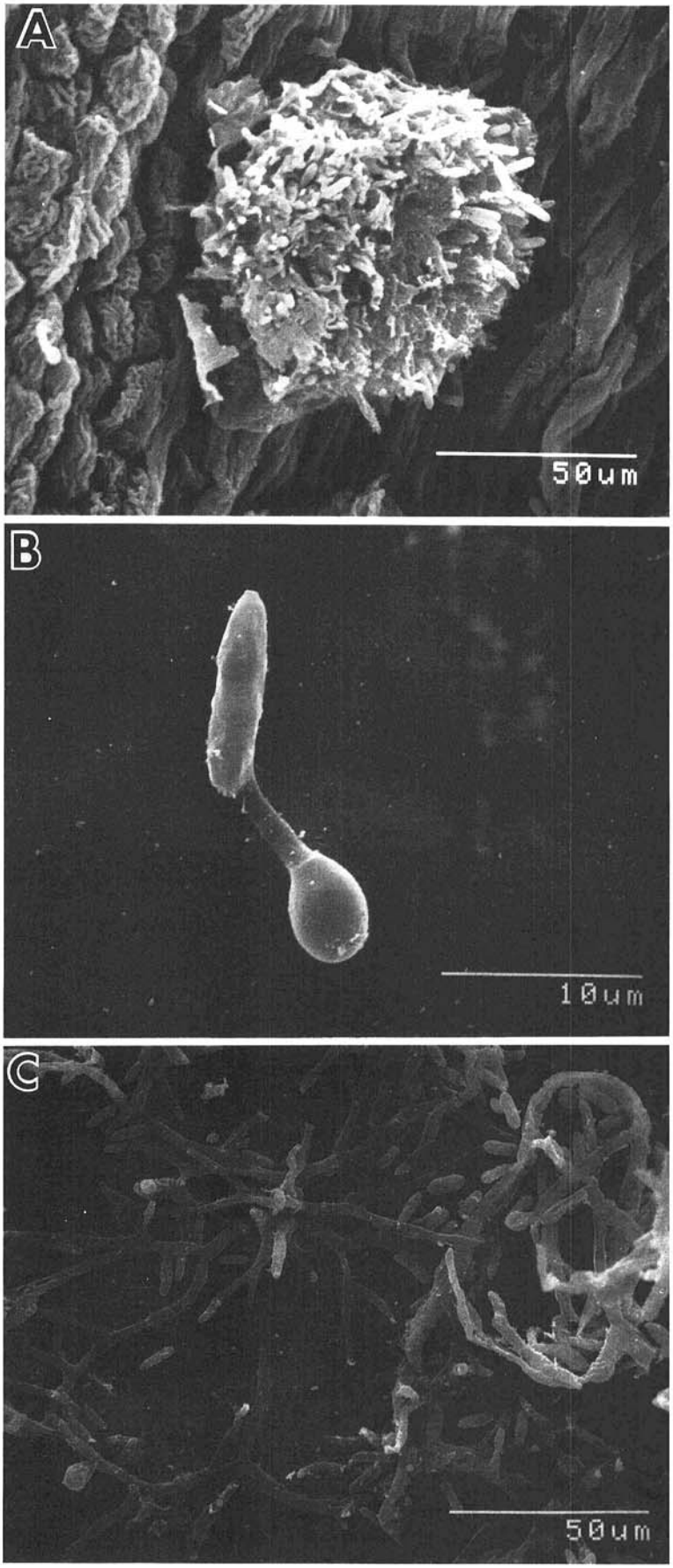

Fig. 2. Scanning electron micrographs of structures of *Colletotrichum gloeosporioides* on citrus: (A) An acervulus on an infected flower petal, (B) a conidium on the leaf surface germinating to form an appressorium, and (C) production of conidia on the leaf surface without acervulus formation in response to treatment with flower extracts under moist conditions.

populations from dead or senescent vegetative tissue. At this point, we are uncertain as to what extent survival of the SGO strain in nature might be due to colonization of dead tissues in the tree canopy. Ascospores of *Glomerella cingulata* (Stoneman) Spauld. & H. Schrenk have been observed in spore traps and perithecia found in dead leaves in Belize (14) and perithecia have been produced on sterilized leaves in Florida (5), but the teliomorphic stage is not believed to play a major role in the disease cycle.

Apparently, appressoria of the SGO strain lose viability with time, since populations of the strain decline gradually in the absence of bloom (1,3). Repeated flower infections seem to be needed to maintain inoculum levels. PFD is less likely to be severe in cooler citrus areas where trees bloom for only a short period each spring. In tropical areas where trees bloom nearly year-round, however, PFD can be devastating. Also, PFD is often more serious on limes, which bloom more sporadically than orange or mandarin (*C. reticulata* Blanco) cultivars. PFD is very severe on declining trees and on young trees, which are more likely to bloom off-season.

Environmental Effects and Epidemiology

Fagan (13,14) in Belize and Timmer and Zitko (29) found that PFD was associated with periods of high rainfall during the bloom period. Disease was often most severe in the lower canopy where rainfall had dispersed inoculum from infected flowers above.

Optimum temperatures for growth of the fungus in culture are 24–27 C (5,13). However, the SGO strain grows well at temperatures down to 15 C. Low temperatures, while slowing disease development, also slow blossom development. Thus, low temperatures may delay the epidemic but do not necessarily affect the final outcome. High temperatures speed blossom development and, if they occur during dry periods, help avoid disease.

In Florida, we utilized multiple regression analyses to develop predictive equations for disease incidence (29) and found that the most important parameters in disease prediction were the number of affected blossoms already on the tree (inoculum availability) and rainfall during the previous 5 days. Leaf wetness and temperature played relatively minor roles, and relative humidity had no effect. Thus, while moisture was essential for infection, dispersal of inoculum to healthy flowers by the impact of raindrops was more important in determining disease incidence.

Disease progress in time was best fit by a Gompertz pattern when central focal trees were inoculated in young citrus plantings (2). Disease spread primarily by rain splash or by wind-driven rain. There was some indication of development of secondary foci that could be attributable to spread by insects. Bees, fruit flies, and other insects that visit flowers are known to carry conidia of the pathogen (23), but their importance in disease spread under natural conditions is unknown.

Mechanisms for long-distance transmission of the pathogen remain an enigma. PFD has appeared in orchards well removed from other plantings. We suspect the fungus may be carried to some extent by harvesting crews on equipment that is moved from orchard to orchard. Infected petals may be carried on picking sacks, clothing, or equipment. This could explain spread in Valencia oranges, which are often harvested during the bloom period, but appears less likely with other cultivars. Some spread may also occur on vegetative material carried from orchard to orchard on equipment of any type. Since the pathogen can be acquired by bees, it is conceivable that transmission may also occur by movement of hives from area to area.

The disease may have originated in widely separated locations in the tropics by spread from Key limes affected by anthracnose. This does not appear to be a likely explanation for the appearance of the disease in central and north Florida. First, very few Key limes were planted in these areas and, second, almost all were destroyed by the freezes of the 1980s.

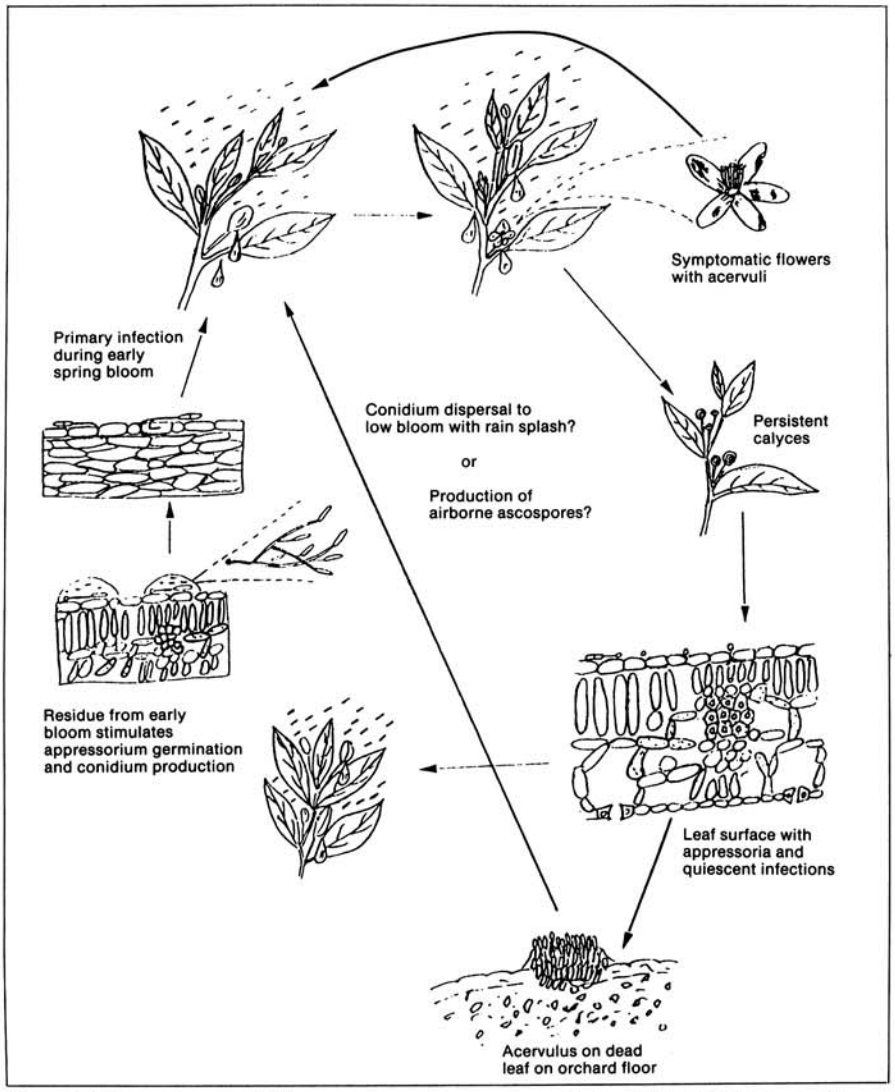

Fig. 3. Disease cycle of the slow-growing orange strain of *Colletotrichum gloeosporioides*, the cause of postbloom fruit drop.

Species and Cultivars Affected

PFD has been observed on almost all species and cultivars of citrus, and none is known to be resistant to the disease. In pathogenicity tests (5), there was little difference in the rate of lesion expansion or disease severity on petals of different citrus species. However, controlled comparisons have not been conducted with different inoculum concentrations.

In the field, disease often varies widely with the citrus species. Navel oranges, which have profuse bloom and often flower out-of-season, especially in warmer climates, are severely affected. In Florida, Valencia oranges have been more frequently damaged than early and

midseason orange cultivars. Damage on a given species or cultivar can vary from year to year, depending on whether the majority of the bloom occurred during rainy or dry periods.

Cultivars also have different physiological responses to disease, irrespective of bloom period. Most orange and grapefruit cultivars respond to petal infection by producing persistent buttons. In contrast, infection of many types of tangerines and tangerine hybrids results in abscission of the entire peduncle, and few persistent buttons are formed.

Effects on Production

On citrus trees that set fruit, the percentage of flowers reaching maturity is low and variable but usually ranges from 0.5 to 2.0%. Flowers affected by PFD form persistent buttons regardless of whether they would have set and matured fruit. Thus, judging damage by the number of persistent buttons may result in overestimating yield losses. Under Florida conditions, it seems reasonable to expect that, with light infestations, no more than two fruit are lost for each 100 buttons present. In addition, it appears that trees compensate for PFD losses by shedding less fruit during the normal period of physiological drop in May and June (28). Also, trees with fewer fruit produce larger fruit (28), which helps to compensate for losses to PFD. We have observed up to 20% blossom blight caused by PFD with no consequent losses in yield (28).

Once PFD reaches moderate to severe levels, effects on yield may be great and losses may be underestimated. When inoculum concentrations are high, flower buds and entire clusters are infected and abscise without formation of buttons. In tropical areas, trees severely affected by PFD will later bloom again, producing a late crop somewhat compensating for yield losses. In more temperate areas, however, such off-season bloom does not produce marketable fruit. In navel oranges in Florida, for example, severe attacks have resulted in low yields of off-bloom and oversized fruit that were not marketable. If yields are reduced below certain levels by PFD, the cost of harvest per box increases and the crop may be abandoned.

Our observations indicate that a single lesion on a flower petal is sufficient to induce fruit drop and button formation. Usually, once the petals have fallen from healthy flowers and the fruit has set, no further fruit loss occurs. In some cases, however, fruit that set early in the season and are up to 1 cm in diameter may abscise if PFD affects a later, adjacent flower cluster.

Disease Control

Investigators in Belize (12,15) found that benomyl and captafol were the most effective fungicides for control of PFD. Used alone or in combination, these agents achieved a high degree of control, but up to four applications were often needed. Captan, maneb, and other contact fungicides reduced disease but did not provide a high level of control (12,15). In Florida, benomyl and captafol provide control, but only benomyl is registered for use on PFD. In laboratory tests, the sterol biosynthesis inhibiting fungicides substantially reduce growth of the SGO strain at low concentrations (34) and thus are promising for future use. The greasy spot pathogen (*Mycosphaerella citri* Whiteside) (31) and the citrus scab pathogen (*Elsinoe fawcettii* Bitancourt & Jenk.) (32) have developed resistance to benomyl, and the potential for the SGO strain to develop resistance also exits, although none has been detected to date.

Perhaps the most critical factor in effective, low-cost control of PFD is deciding whether and when to spray. Repeated preventive sprays are effective but costly and may not increase yield if little PFD develops. Delaying sprays can result in excessive buildup of inoculum on early bloom, making the disease difficult to control and increasing the probability of selecting resistant strains.

The number of persistent buttons remaining from the previous year provides some indication of disease potential in the coming bloom. The number of buttons counted in January 1992 were correlated with disease severity of the subsequent bloom ($r = 0.85$) (L. W. Timmer and S. E. Zitko, *unpublished*). However, those factors were not correlated in 1993 because extensive rain during bloom produced high levels of infection even where the disease was a minor problem in 1992.

Blocks of trees with persistent buttons need to be monitored twice weekly to assess the amount of bloom and the percentage of flowers affected. We (29) developed the following equation to predict disease 3–4 days in advance: $y = -7.15 + 1.28\ (TD)^{1/2} + 0.44\ (R \times 100)^{1/2}$, where y = predicted percentage of flowers affected, TD = total number of flowers affected on 20 trees, and R = rainfall total (mm) for the last 5 days.

We recommend that sprays be applied when more than 20% disease is predicted and the current bloom represents a significant proportion of the total crop. An application is usually effective for 10–14 days, after which another determination should be made as to the advisability of a second application. The blossom period on Florida citrus may extend up to 8–10 weeks on some cultivars, and as many as five applications have been needed to maximize yields (28). Aerial applications have proved effective, so a fungicide can be applied quickly over large areas should the need arise (15,27).

Other measures found useful by some growers in reducing disease pressure are replacement of overhead sprinklers with undertree microsprinklers and removal of declining trees, often heavily affected by PFD, prior to bloom. These measures are seldom effective when used alone but decrease inoculum production, making the disease easier to control with fungicides.

Literature Cited

1. Agostini, J. P. 1992. Etiology and epidemiology of postbloom fruit drop of citrus. Ph.D. dissertation. University of Florida, Gainesville.
2. Agostini, J. P., Gottwald, T. R., and Timmer, L. W. 1993. Temporal and spatial dynamics of postbloom fruit drop of citrus in Florida. Phytopathology 83:485-490.
3. Agostini, J. P., and Timmer, L. W. 1992. Population dynamics and survival of *Colletotrichum gloeosporioides*, the cause of citrus postbloom fruit drop. (Abstr.) Phytopathology 82:1084.
4. Agostini, J. P., and Timmer, L. W. 1992. Selective isolation procedures for differentiation of two strains of *Colletotrichum gloeosporioides* from citrus. Plant Dis. 76:1176-1178.
5. Agostini, J. P., Timmer, L. W., and Mitchell, D. J. 1992. Morphological and pathological characteristics of strains of *Colletotrichum gloeosporioides* from citrus. Phytopathology 82:1377-1382.
6. Bonde, M. R., Peterson, G. L., and Maas, J. L. 1991. Isozyme comparisons for identification of *Colletotrichum* species pathogenic to strawberry. Phytopathology 81:1523-1528.
7. Brown, G. E. 1975. Factors affecting postharvest development of *Colletotrichum gloeosporioides* in citrus fruits. Phytopathology 65:404-409.
8. Brown, G. E. 1977. Ultrastructure of penetration of ethylene-degreened Robinson tangerines by *Colletotrichum gloeosporioides*. Phytopathology 67:315-320.
9. Clausen, R. E. 1912. A new fungus concerned in wither tip of varieties of *Citrus medica*. Phytopathology 2:217-236.
10. Denham, T. G. 1979. Citrus production and premature fruit drop disease in Belize. PANS 25:30-36.
11. Denham, T. G., and Waller, J. M. 1981. Some epidemiological aspects of postbloom fruit drop (*Colletotrichum gloeosporiodes*) in citrus. Ann. Appl. Biol. 98:65-77.
12. Fagan, H. J. 1971. Pathology and nematology in British Honduras. Pages 10-21 in: Annual Report of Citrus Research Unit, University of West Indies.
13. Fagan, H. J. 1979. Postbloom fruit drop, a new disease of citrus associated with a form of *Colletotrichum gloeosporioides*. Ann. Appl. Biol. 91:13-20.
14. Fagan, H. J. 1984. Postbloom fruit drop of citrus in Belize: I. Disease epidemiology. Turrialba 34:173-177.
15. Fagan, H. J. 1984. Postbloom fruit drop of citrus in Belize: II. Disease control by aerial/ground spraying. Turrialba 34:179-186.
16. Fulton, H. R. 1925. Relative susceptibility of citrus varieties to attack by *Gloeosporium limetticolum* (Clausen). J. Agric. Res. 30:629-635.
17. Gantotti, B. V., and Davis, M. J. 1991. Detection of pectinase isozyme polymorphism in *Colletotrichum gloeo-*

sporioides. (Abstr.) Phytopathology 81:1170.
18. Jeffries, R., Dodd, J. C., Jeger, M. J., and Plumbey, R. A. 1990. The biology and control of *Colletotrichum* species on tropical fruit crops. Plant Pathol. 39:343-366.
19. Liyanage, H. D., Köller, W., McMillan, R. T., Jr., and Kistler, H. C. 1993. Variation in cutinase from two populations of *Colletotrichum gloeosporioides* from citrus. Phytopathology 83:113-116.
20. Liyanage, H. D., McMillan, R. T., Jr., and Kistler, H. C. 1992. Two genetically distinct populations of *Colletotrichum gloeosporioides* from citrus. Phytopathology 82:1371-1376.
21. McMillan, R. T., Jr., and Timmer, L. W. 1989. Outbreak of citrus postbloom fruit drop caused by *Colletotrichum gloeosporioides* in Florida. Plant Dis. 73:81.
22. Orozco Santos, M., and Gonzalez Garza, R. 1986. Caída de fruto pequeño y su control en naranja 'Valencia' en Veracruz. Agric. Tec. Mex. 12(2):259-269.
23. Peña, J. E., and Duncan, R. 1990. Role of arthropods in the transmission of post bloom fruit drop. Citrus Ind. 71(4):64-69.
24. Schwarz, R. E., Klein, E. H. J., and Monsted, P. 1978. Fungal infection of citrus flowers: Probable cause of abnormal fruit drop in the Parana mist zone of Misiones, Argentina. (Abstr.) Page 130 in: Int. Congr. Plant Pathol. 3rd.
25. Sonoda, R. M., and Pelosi, R. R. 1988. Characteristics of *Colletotrichum gloeosporioides* from lesions on citrus blossoms in the Indian River of Florida. Proc. Fla. State Hortic. Soc. 101:36-38.
26. Sutton, B. C. 1980. The Coelomycetes. Commonwealth Mycological Institute, Kew, England.
27. Timmer, L. W., and Zitko, S. E. 1991. Aerial applications of fungicide for control of postbloom fruit drop. Citrus Ind. 72(12):26-27.
28. Timmer, L. W., and Zitko, S. E. 1992. Timing of fungicide applications for control of postbloom fruit drop of citrus in Florida. Plant Dis. 76:820-823.
29. Timmer, L. W., and Zitko, S. E. 1993. Relationships of environmental factors and inoculum levels to the incidence of postbloom fruit drop of citrus. Plant Dis. 77:501-504.
30. von Arx, J. A. 1970. A revision of the fungi classified as *Gloeosporium*. Bibl. Mycol. 24:1-203.
31. Whiteside, J. O. 1980. Tolerance of *Mycosphaerella citri* to benomyl in Florida citrus groves. Plant Dis. 64:300-302.
32. Whiteside, J. O. 1980. Detection of benomyl-tolerant strains of *Elsinoë fawcetti* in Florida citrus groves and nurseries. Plant Dis. 64:871-872.
33. Whiteside, J. O. 1988. Symptomless and quiescent infections by fungi. Page 30 in: Compendium of Citrus Diseases. J. O. Whiteside, S. M. Garnsey, and L. W. Timmer, eds. American Phytopathological Society, St. Paul, MN.
34. Zitko, S. E., and Timmer, L. W. 1992. Evaluation of fungicides in vitro for control of *Colletotrichum gloeosporioides* from citrus, 1991. Fungic. Nematicide Tests 47:335.
35. Zulfiqar, M. 1993. Infection and survival of *Colletotrichum gloeosporioides* on citrus. M.S. thesis. University of Florida, Gainesville.

L. W. Timmer

Dr. Timmer is a plant pathologist at the University of Florida's Citrus Research and Education Center in Lake Alfred. He received his B.S. degree in botany and plant pathology at Michigan State University in 1963 and his Ph.D. at the University of California, Riverside, in 1969. He spent 8 years at the Texas A&I University Citrus Center in Weslaco doing research, extension, and teaching on citrus diseases prior to joining the University of Florida in 1978. Currently, his primary research interests are in the study of fungal diseases of citrus. He has spent time in Argentina and traveled extensively in other Latin American countries in conjunction with various research projects and consulting assignments.

J. P. Agostini

Dr. Agostini is a plant pathologist with the Instituto Nacional de Tecnología Agropecuaria (INTA) in Montecarlo, Misiones, Argentina. He received his Ingeniero Agrónomo degree from the Universidad Nacional in Corrientes, Argentina, in 1976. He completed his M.S. degree on Phytophthora root rot of citrus in 1989 at the University of Florida and his Ph.D. on postbloom fruit drop in 1992 at the same institution. Both degrees were under the direction of L. W. Timmer. Prior to joining INTA in his current post, he worked as an extension agent in Formosa Province and in the citrus canker eradication program in Entre Ríos. His current program involves research and extension on all diseases of citrus.

S. E. Zitko

Mr. Zitko is a biological scientist at the University of Florida's Citrus Research and Education Center in Lake Alfred. He received his B.S. degree in agriculture in 1979 from Ohio State University and his M.S. degree from North Dakota State University in 1982. He spent 2 years in the Peace Corps working with citrus growers in Belize prior to joining the University of Florida in 1986. He spent 6 months in Concordia, Argentina, during 1988–1989 working on a citrus canker project. Most of his current research work is related to fungal diseases of fruit and foliage of citrus trees.

M. Zulfiqar

Mr. Zulfiqar is a research officer in plant pathology at the Agricultural Research Institute, Tarnab, Peshawar, Pakistan. He received his B.S. degree in 1986 from the North-West Frontier Province Agricultural University in Peshawar. In 1988, he completed his M.S. degree at the same institution working on root-knot nematodes on tomato. His M.S. degree from the University of Florida dealt with cytological studies of postbloom fruit drop of citrus and was conducted under the direction of L. W. Timmer. He completed his degree in 1993 and returned to his post in Pakistan, where he is responsible for research on diseases of fruit and vegetable crops.