Evaluation of a Model for Prediction of Postbloom Fruit Drop of Citrus

L. W. Timmer, Professor, and **S. E. Zitko**, Senior Biologist, University of Florida, Citrus Research and Education Center, 700 Experiment Station Road, Lake Alfred 33850

ABSTRACT

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Postbloom fruit drop, caused by *Colletotrichum acutatum*, produces orange-brown lesions on flower petals, abscission of fruitlets, and formation of persistent calyces (buttons). A previously developed model that predicts the percentage of flower infection 3 to 4 days in advance based on the current number of infected flowers and rainfall for the previous 5 days was evaluated in three navel and Valencia orange groves in 1993 and in five groves each in 1994 and 1995. There was a significant ($P \leq 0.05$) relationship between the predicted and observed percentages of affected flowers in seven of the nine cases in which sufficient disease developed to warrant fungicide applications ($R^2 = 0.38$ to 0.86). From one to three applications were made in each of these cases based on the model predictions. In many of the cases, the fungicide applications reduced the area under the curve for disease incidence on flowers and the number of buttons formed compared to the unsprayed controls. Fungicide applications increased fruit counts ($P \leq 0.10$) in eight of the nine cases with increases over the unsprayed controls ranging from 25 to 523%. Model predictions were accurate except when rain events were of short duration and tree canopies dried quickly. Model-based decisions on fungicide applications resulted in reduced disease, large increases in fruit production, and elimination of unnecessary sprays.

Additional keyword: *Colletotrichum gloeosporioides*

Postbloom fruit drop (PFD) affects citrus flowers, producing orange-brown lesions on the petals and inducing the abscission of fruitlets and the formation of persistent calyces commonly called buttons. The disease was described first in Belize (3) and has since appeared in most of the humid citrus-growing areas of the Americas (7). PFD was first reported in Florida in 1983 (6).

The causal agent was described originally as a strain of *Colletotrichum gloeosporioides* (Penz.) Penz. & Sacc. in Penz. Agostini et al. (1) identified three strains of *Colletotrichum* spp. on citrus: (i) FGG, the fast-growing gray strain that causes only postharvest anthracnose on fruit and whose characteristics are typical of *C. gloeosporioides*; (ii) SGO, the slow-growing orange strain that causes postbloom fruit drop; and (iii) KLA, the Key lime anthracnose strain, which was described originally as *Gloeosporium limetticola* R. E. Clausen, and which causes both PFD and lime anthracnose. There were only minor morphological differences between the SGO and KLA strains and neither was typical of *C. gloeosporioides* (1). Both had characteristics of *C. acutatum* J. H. Simmonds, but conidial shape was atypical for that species. Recent molecular studies have demonstrated that the SGO and KLA strains belong to *C. acutatum* (A. E. Brown, S. Sreenivasaprasad, and L. W. Timmer, unpublished) and we will refer to them as such in this paper.

A predictive model for PFD has been developed (9,10) to aid in the timing of fungicide applications. The purpose of this study was to evaluate the accuracy of the model predictions and determine the level of disease control and fruit production attained using application times based on the model.

MATERIALS AND METHODS

The predictive model developed previously was published in an incorrect form (9). The corrected equation is: $y = -7.15 + 1.2\sqrt{TD} + 0.44\sqrt{R\ x\ 100}$ where y = the predicted percentage of flowers affected at the next assessment, TD = the total number of affected flowers on 20 trees, and R = rainfall for the past 5 days (mm).

Experiments were conducted from 1993 to 1995 in groves of navel and Valencia oranges, the two cultivars most commonly affected by PFD (7). Sites and years are as follows: Arcadia, navel oranges in 1993, 1994, and 1995 and Valencias in 1994; Lake Placid, navels in 1993 and 1994 and Valencias in 1994; Frostproof, navels in 1993, 1994, and 1995; and Bowling Green, navels in 1995. All trees used in experiments were mature, fruit-bearing trees at least 15 years old. Five two-tree plots were used for the fungicide application treatments and five plots for the unsprayed controls. Treatments were arranged in a completely randomized design. Unsprayed guard trees and guard rows were located between the treated plots to minimize the effects of spray drift.

Twice weekly during the bloom period, which may extend from late January into April, diseased and healthy flowers were counted on each tree in the experiment. If available, up to 200 flowers that had recently opened or were about to open were observed on each tree. The average number of flowers per tree, up to a maximum of 200, was used as an index of the status of the bloom on the basis of 0 to 100. The total number of diseased flowers per tree was used in the equation along with the rainfall for the last 5 days to predict the percentage of diseased flowers at the next assessment. Since the model uses total number of diseased flowers on 20 trees, the number of diseased flowers on the 10 trees was multiplied by two and used for TD in the equation. Rainfall data were obtained from gauges located in the grove or from cooperating growers. Fungicide applications were made to the treatment plots if all the following previously established criteria (9,10) were met: (i) the model predicted a percentage of flower infection greater than 20%; (ii) bloom present on the trees represented a significant portion of the crop (>75 on the relative scale used here); and (iii) no application had been made in the last 14 days.

Sprayed trees were treated with benomyl (Benlate 50WP) only in 1993 at 0.48 g a.i./liter. In 1994 and 1995, sprayed trees were treated with benomyl at 0.36 g a.i. plus ferbam (Carbamate 76W) at 0.91 g a.i./liter (Fig. 1). Fungicides were applied with a handgun sprayer at pressures of 1,500 to 1,700 kPa using from 20 to 30 liters of spray material per tree depending on tree size.

Predicted and observed percentages of blossom blight were calculated for each date at each location. Linear regression analysis was conducted to assess the agreement between the predicted and observed values at each date. Only data from the control plots were used for these analyses and results are presented as adjusted R^2 values.

The area under the curve (AUC) for the percentage of diseased flowers in percent-days was calculated in each replication in the sprayed and control plots over the entire

Corresponding author: L. W. Timmer
E-mail: lwt@gnv.ifas.ufl.edu

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bloom period represented in Figure 1 at each site. The AUC gave an indication of the ability of the fungicide treatment to reduce the blossom blight phase of the disease.

In June, after bloom and after the period of normal physiological fruit drop, the number of buttons and fruit was counted on all trees. The total number of buttons and fruit was counted on 12 0.75-m long branches, three per quadrant on each tree. The data were expressed as the average numbers of buttons or fruit per 12 branches per tree. Student's *t* test was used to compare differences between treated

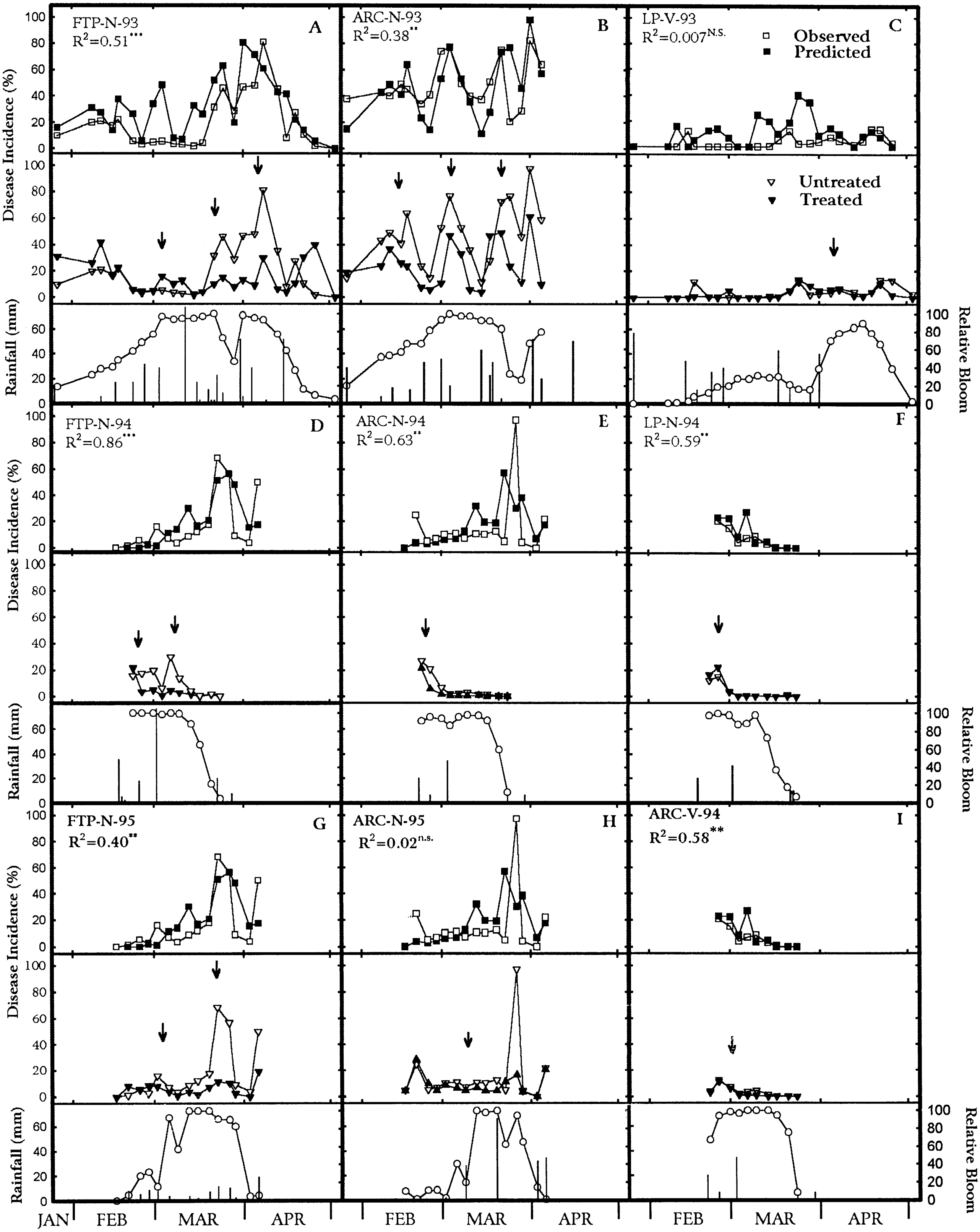

Fig. 1. Incidence of postbloom fruit drop caused by *Colletotrichum acutatum* in different locations and years on navel (N) and Valencia (V) oranges; FTP = Frostproof; ARC = Arcadia; LP = Lake Placid. **(A–H)** Top panels: Observed percentages of affected flowers in the control plots for each evaluation date (□) and those percentages that had been predicted (■) for that date based on information available 3 to 4 days earlier. Middle panels: Disease incidence in plots sprayed with benomyl or benomyl + ferbam (▼) and in unsprayed controls (▽). Arrows indicate the dates of applications. Bottom panels: Relative bloom on a scale of 0 to 100 (line graph); 100 = at least 200 open flowers per tree; rainfall (vertical bars).

and control plots for the AUC for blossom blight, and for button and fruit counts.

RESULTS

Disease incidence in the navel orange plots in 1993 was high with up to 80% of the flowers affected at times during the bloom (Fig. 1A,B). However, incidence was low in the Valencia orange block (Fig. 1C). In the navel orange plots, there was generally good correspondence between predicted and observed values with significant R^2 values ($P \le 0.05$). However, on two dates in late February and early March at Frostproof, predicted values greatly exceeded the observed (Fig. 1A,B). These predictions were associated with high rainfall that fell in a short period in advance of a cold front, which allowed tree canopies to dry quickly. Lack of correspondence of predicted and observed values in the Valencia orange plots at Lake Placid was primarily due to lack of inoculum (Fig. 1C). Overpredictions of disease incidence occurred nearly every time it rained when no diseased flowers were present.

In 1994, disease incidence was high initially but decreased sharply when almost no rain fell after peak bloom (Fig. 1D–F,I). The correspondence between observed and predicted disease incidence was high with significant R^2 values at all sites (Fig. 1D–F,I). One overprediction, associated with a rain event of short duration, was observed in early March in the navel and Valencia orange blocks at Arcadia and the navel orange block at Lake Placid (Fig. 1E,F,I).

In 1995, bloom began late and occurred over a short period with few rain events (Fig. 1G,H). Disease distribution was erratic and data were highly variable. For example, in the navel orange plots at Arcadia, disease incidence on the flowers on unsprayed control trees ranged from 0 to 68% on 23 March. The relationship between observed and predicted values was significant at the Frostproof location but not at Arcadia (Fig. 1G,H).

In 1993, the model triggered three fungicide applications in the navel orange plots at Arcadia, each of which reduced disease incidence to about the same degree during the bloom period (Fig. 1B). Fluctuations in disease incidence in the control were due to periods of no or low rainfall. In contrast, the three fungicide sprays triggered by the model at Frostproof eliminated a peak in the disease that occurred between mid-March and mid-April in the control trees (Fig. 1A). The Valencia trees at Lake Placid bloomed late (Fig. 1C). A single fungicide application was made in response to a prediction prior to peak bloom, but no rain fell in April and disease did not develop further.

In 1994, single fungicide applications were triggered early by high inoculum levels and some rain, but very little rain occurred after early March and disease incidence declined generally (Fig. 1D–F,I). Only at Frostproof was a second application indicated.

Table 1. Effect of application of benomyl or benomyl plus ferbam on the area under the curve (AUC) for flower infection by *Colletotrichum acutatum* and the number of buttons and fruit produced as a result of postbloom fruit drop

Year	Location/cultivar	No. of sprays	AUC[a]	Buttons/12 branches/tree	Fruit/12 branches/tree	Increase (%)[b]
1993	Frostproof/navels	3	316 NS[c]	136**	27.1†	261
		0	369	237	7.5	
	Arcadia/navels	3	333**	324 NS	14.8†	131
		0	624	351	6.4	
	Lake Placid/Valencias	1	45 NS	80 NS	21.4†	53
		0	65	116	14.0	
1994	Frostproof/navels	2	19.3†	134 NS	19.5†	65
		0	67.1	151	11.8	
	Arcadia/navels	1	12.9*	65***	16.2**	25
		0	34.9	135	13.0	
	Lake Placid/navels	1	25.1 NS	41***	22.1***	63
		0	18.3	88	13.6	
	Arcadia/Valencias	1	15.9 NS	95*	17.0 NS	. . .
		0	22.4	163	17.8	
	Lake Placid/Valencias	0	1.0 NS	24 NS	50.8	. . .
		0	0.5	16	45.7	
1995	Frostproof/navels	2	19.6**	74**	29.9**	523
		0	49.6	209	4.8	
	Arcadia/navels	1	24.7 NS	66 NS	27.2†	101
		0	30.8	102	13.5	
	Lake Placid/navels	0	6.1 NS	34 NS	18.9**	52
		0	8.3	9	12.4	
	Bowling Green/navels	0	1.0 NS	6 NS	1.7 NS	. . .
		0	3.6	1	1.5	
	Arcadia/Valencias	0	3.0 NS	16 NS	15.5 NS	. . .
		0	2.4	15	13.1	

[a] AUC for incidence of flower infection in percent-days.

[b] Percentage of increase in fruit production over the nonsprayed control.

[c] All figures are means of five two-tree plots. NS = not significant; †, *, **, *** = significant at $P \le$ 0.1, 0.05, 0.01, and 0.001, respectively.

In 1995, fungicide sprays triggered by a rain event in early March greatly depressed a peak in the disease brought about by a prolonged rain in mid-March in the navel groves at Frostproof and Arcadia (Fig. 1G,H).

Fungicide applications triggered by the model significantly reduced the AUC for blossom blight in some, but not all, cases (Table 1). In certain locations (e.g., navels at Frostproof in 1993, Fig. 1A), disease incidence was higher in the sprayed than in untreated plots early and late in the bloom period. Since high disease levels occurred during times outside of peak bloom and before the first fungicide application, high AUC values were observed that did not necessarily reflect the total amount of damage that occurred.

Fungicide sprays also reduced the number of buttons formed compared with the nonsprayed plots in many cases. The most dramatic effect of the fungicide applications was to increase fruit counts. In eight of nine cases in which fungicides were applied, significant ($P \le 0.10$) increases in fruit production were observed, with increases ranging from 25 to over 500% (Table 1).

DISCUSSION

The previously developed model was very useful in determining the need for and proper timing of fungicide applications for control of PFD. Predicted and observed percentages of diseased flowers were significantly related in seven of the nine cases in which sufficient disease developed to warrant fungicide sprays. Use of the model resulted in increases in fruit counts in eight of nine cases in which sprays were applied and unnecessary applications were avoided.

Our model differs considerably from those developed for diseases caused by *Colletotrichum* species on other crops. Most of those models are based on leaf wetness and temperature during the infection process (2,4,5). With PFD on citrus, temperature was not a significant factor in determining disease incidence (9), probably because temperatures are seldom outside of the optimum range for the fungus during the bloom period. Leaf wetness accounted for only a small portion of the variability in disease incidence (9). With anthracnose on strawberry, Madden et al. (5) found that the amount of rainfall was important for inoculum dispersal, as appears to be the case with PFD. With other systems (2,4) inoculum may be continuously available. However, with PFD, inoculum fluctuates greatly since it is produced on transient tissues, flower petals, thus making availability an important determinant of future disease incidence. Rainfall is probably as important in conidial dispersal as in providing leaf wetness.

Thus, with PFD on citrus, the factors that are significant are the presence and dispersal of inoculum rather than the conditions for infection, which are the significant factors in other systems.

Predictions for PFD were most accurate when disease was at low to moderate levels, at which times fungicide sprays would likely be most valuable. Because of the additive nature of the model, high rainfall in the absence of inoculum may result in inaccurately high predictions. However, since nearly 50 mm of rain would be required to trigger a fungicide spray when the number of diseased flowers is zero, this defect would seldom result in unnecessary fungicide applications. Likewise, inaccurate predictions may result when high levels of disease are present and no rain has fallen in the last 5 days. Again, however, this shortcoming is of little practical importance since the grove probably would have been treated earlier.

As observed in a few cases in this study, high rainfall in a brief period with rapid drying resulted in excessively high predictions when little disease actually developed. Thus, the model is conservative and could result in an occasional unnecessary spray but is unlikely to fail to indicate a necessary one.

Under favorable environmental conditions, PFD can be an extremely damaging disease (7,8). Using the fruit counts in sprayed plots as maximum yields, we found that PFD reduced yield 25 to 80% compared with the unsprayed control in this study. In reality, the disease was difficult to control on the isolated sprayed plots used in this test because they were surrounded by unsprayed guard and control trees. Disease control in adjacent blocks by growers following our schedule was generally better than we attained. Large increases in fruit production were realized as a result of fungicide applications scheduled by use of the model. We also successfully avoided making unneeded applications in locations where the disease never developed. Some improvements in the model, such as inclusion of leaf wetness or rainfall duration, may be helpful, but with proper scouting it is a useful tool in management of PFD. The model has already been widely adopted by Florida citrus growers. Early-season indicators for PFD severity in the coming season, such as the number of buttons remaining from the previous year and infection on scattered early bloom, are helpful in focusing scouting efforts (8) and can be used in conjunction with the model.

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LITERATURE CITED

1. 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.
2. Danneberger, T. K., Vargas, J. M., Jr., and Jones, A. L. 1984. A model for weather-based forecasting of anthracnose on annual bluegrass. Phytopathology 74:448-451.
3. 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.
4. Fitzell, R. D., Peak, C. M., and Darnell, R. E. 1984. A model for estimating infection levels of anthracnose disease of mango. Ann. Appl. Biol. 104:451-458.
5. Madden, L. V., Wilson, L. L., and Ellis, M. A. 1993. Field spread of anthracnose fruit rot of strawberry in relation to ground cover and ambient weather conditions. Plant Dis. 77:861-866.
6. 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.
7. Timmer, L. W., Agostini, J. P., Zitko, S. E., and Zulfiqar, M. 1994. Postbloom fruit drop, an increasingly prevalent disease of citrus in the Americas. Plant Dis. 78:329-334.
8. 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.
9. 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.
10. Timmer, L. W., and Zitko, S. E. 1993. Use of environmental factors and inoculum levels to predict postbloom fruit drop incidence. Citrus Ind. 74(11):4-5,7.
11. Timmer, L. W., and Zitko, S. E. 1995. Early season indicators of postbloom fruit drop of citrus and the relationship of disease incidence and fruit production. Plant Dis. 79:1017-1020.