Temporal and Spatial Dynamics of Postbloom Fruit Drop of Citrus in Florida

J. P. Agostini, T. R. Gottwald, and L. W. Timmer

First and third authors, graduate research assistant and professor, University of Florida, IFAS, Citrus Research and Education Center, 700 Experiment Station Road, Lake Alfred 33850; second author, research plant pathologist, USDA, ARS, U. S. Horticultural Research Laboratory, Orlando 32803.

Florida Agricultural Experiment Station Journal series R-02623.
Accepted for publication 28 January 1993.

ABSTRACT

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.

Temporal progress of postbloom fruit drop of citrus in Florida was studied in four experimental plots of 25–81 3- to 4-yr-old trees of Valencia sweet orange in Gainesville and Lake Alfred and of Pineapple sweet orange and Ruby Red grapefruit in Hastings. Blossoms of central focal trees in each plot were inoculated with the slow-growing orange strain of *Colletotrichum gloeosporioides*. Incidence of trees and of open blossoms with postbloom fruit drop was monitored over time. Directionality of spread from a point source was measured in four contiguous quadrants of each plot. Disease gradients were estimated on four transects radiating from a central focal tree. Disease increased in intensity and spread in space following rain events in all four plots. The temporal increase of postbloom fruit drop expressed as proportion of affected trees or blossoms was fit better by a Gompertz model than by linear, monomolecular, logistic, or exponential models. Rate parameters of 0.05, 0.11, 0.05, and 0.04 gompits/day for disease incidence on trees and 0.07, 0.05, 0.04, and 0.02 gompits/day for disease incidence on flowers were found for the Gainesville, Lake Alfred, Hastings-sweet orange, and Hastings-grapefruit, respectively. Directional spread was apparent in Lake Alfred and spread was associated with a single rain event accompanied by northerly winds. Little indication of directionality in spread of disease was noted in the other three plots. There was some evidence of development of secondary foci in some plots. Spread appeared to be primarily by windblown rain.

Citrus postbloom fruit drop is caused by a slow-growing orange strain of *Colletotrichum gloeosporioides* (Penz.) Penz. & Sacc. in Penz. (8). The fungus infects petals of citrus trees where it produces peach-to-orange necrotic spots and later induces fruit drop and formation of persistent buttons consisting of the calyx and floral disk. The fast-growing gray strain of *C. gloeosporioides* is saprophytic on citrus and does not infect healthy flowers or produce persistent buttons (1,3). The disease was first described in Belize in 1979 (8), although it was present much earlier. Postbloom fruit drop was first reported in Florida in 1983 (15), and it is now widespread in the humid tropics of the Americas (6, and L. W. Timmer, *unpublished*).

Open flowers are very susceptible to infection with unopened bloom and pinhead blossoms much less severely affected (7,8). Postbloom fruit drop affects most citrus species and cultivars and has been reported on Valencia and Navel oranges, grapefruit, tangelos, and Key (West Indian) lime in Central America and the Caribbean (8), on sweet oranges and Tahiti lime in Florida (15), and observed on many orange cultivars, lemons, limes, grapefruits, and mandarins in Florida and the Caribbean (L. W. Timmer, *unpublished*).

Abundant conidia are produced on diseased petals under moist conditions, and these are splash dispersed to healthy flowers. Disease is most severe when high rainfall with prolonged leaf wetness occurs during bloom (7,8,10), and peaks in the number of conidia collected in spore traps occurred at these times (10). The disease is usually most severe in the lower portion of the tree canopy, presumably due to washing of the spores downward from affected flowers (7). The optimal temperature range for conidial germination was 20–25 C (7,9) and disease severity was greatest during periods with temperatures from 19–30 C in Belize (8).

No studies have been conducted on the development of disease or spread of the pathogen in citrus orchards. The objectives of this study were to determine the rates of spread of postbloom fruit drop and to ascertain the effect of environmental conditions on pathogen dispersal. The dynamics of disease progress and disease spread were investigated in four artificially inoculated plots of young citrus trees.

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MATERIALS AND METHODS

Field experiments. All field plots were isolated and located at least 500 m from other citrus and at least 10 km from orchards affected by postbloom fruit drop.

Gainesville experiment. A plot of 25 trees of Valencia sweet orange (*Citrus sinensis* (L.) Osb.) was established 5 February 1990 in Gainesville, FL, by planting 3-yr-old, screenhouse-grown trees in a 5 × 5 square planting with 5 m between trees and between rows. The trees were irrigated manually three times a week. Disease assessment was made twice weekly during the bloom period of 9 February through 15 March 1990. Disease incidence was assessed on every sample date as the proportion of trees with diseased flowers and as the number of open flowers with lesions divided by the total count of the number of open flowers per tree. Final fruit set and the number of buttons were assessed approximately 3.5 mo after bloom when normal, physiological fruit drop was complete.

Lake Alfred experiment. Three-year-old trees of Rohde Red Valencia sweet orange trees grown in the screenhouse were planted on 11 February 1991 at the Citrus Research and Education Center, Lake Alfred, FL. The plot consisted of 49 trees in a 7 × 7 square planting with 5 m between trees and rows. The trees were irrigated for 1 h by microsprinklers three times a week. Assessment of postbloom fruit drop in this plot was made twice weekly from 25 March through 30 April 1991. Disease was assessed as above on all trees in the plot.

Hastings experiments. Existing plots of 4-yr-old trees of Ruby Red grapefruit (*Citrus paradisi* Macf.) and Pineapple sweet orange (*C. sinensis* (L.) Osb.) were used in this experiment. The grapefruit plot was a 9 × 9 square planting and the Pineapple sweet orange plot was a rectangular planting with eight rows and nine trees per row. Both plots were planted with 5.9 m between rows and 4.4 m between trees, with a total of 81 and 72 trees, respectively, in the plots of grapefruit and sweet orange. Rows were oriented in an east-west direction. Plots were irrigated for 1 h by microsprinklers about twice weekly. Assessment of postbloom fruit drop was made twice weekly from 27 March through 30 April 1991. Disease incidence was assessed by the presence or absence of disease on all trees in both plots. Incidence of disease on flowers was assessed on the six trees surrounding the central focal tree

and on each of the four trees of the northwest, northeast, southwest, and southeast transects (Fig. 1). The number of fruit set and the number of buttons formed as a result of postbloom fruit drop in the transects were recorded as above.

Inoculation of central focal trees. A single-spore isolate of the slow-growing orange strain of *C. gloeosporioides,* originally isolated from blighted blossoms in a Valencia orange grove near Lake Placid, FL, was grown on potato-dextrose agar in the dark at 27 C. Conidia were washed from the surface of 7- to 10-day-old cultures with sterile water and counted with a hemacytometer, and the concentration was adjusted to 2×10^5 conidia per milliliter of sterile water. Flowering 3- to 4-yr-old trees of Valencia sweet orange growing in a screenhouse were sprayed to runoff with the conidial suspension using a hand pump sprayer. After inoculation, the trees were covered with plastic bags for 48 h to maintain high relative humidity. At the time of bloom initiation in each field plot, a single inoculated Valencia orange tree with active postbloom fruit drop on the flowers was planted in the center of each plot to act as a focal point of disease within the plot. In the grapefruit plot, the inoculated tree was set immediately adjacent to the center tree.

Climatic data. All data on temperature, wind speed and direction, and rainfall were obtained from the National Weather Service network. All of the information for the experiments at Gainesville and Lake Alfred was available for the respective sites. Rainfall and temperature were recorded at the Agricultural Research Center in Hastings for the experiment there.

Analysis of disease progress. Models for disease progress over time were tested for each plot for disease incidence of trees and flowers. Incidence of diseased trees (y) was defined as the average proportion of diseased trees from the four quadrants in each experimental plot at each sampling date. In the test at Lake Alfred, quadrants of trees were delimited by the transects running northeast to southwest and northwest to southeast through the central focal tree; thus, four quadrants north (N), east (E), west (W), and south (S) were formed (Fig. 1A). Quadrants in the plots of grapefruit and the Pineapple sweet orange in Hastings were formed by the trees delimited by the transversal lines through the focal tree in the east-west and north-south directions; thus, northwest, southwest, northeast, and southeast quadrants were formed (Fig. 1C and D). Trees on the delimiting line between quadrants were included in both quadrants, and the central focal tree was considered the common corner of each quadrant.

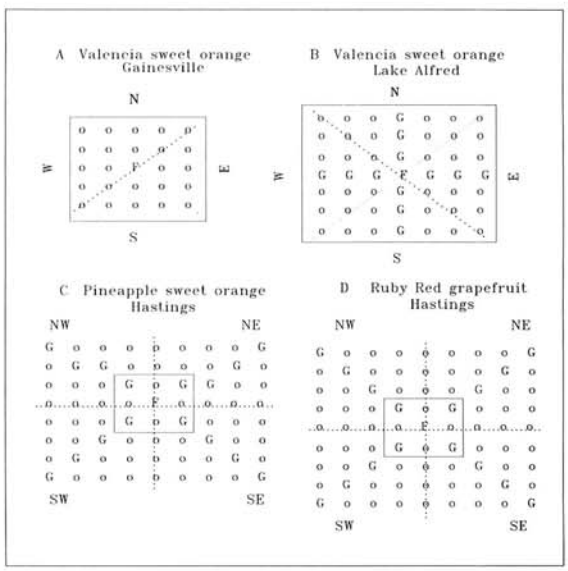

Fig. 1. Diagrams of the four experimental plots illustrating the number and position of trees. The central focal tree (F) and trees used to assess disease gradients (G) as percentage diseased blossoms are designated in each plot. Trees within the boxes formed by solid lines were those used to assess the percentage of blighted blossoms over time. Dotted lines separate the N, E, W, and S quadrants in the Gainesville (A) and Lake Alfred (B) experiments and the NW, NE, SE, and SW quadrants in the two Hastings experiments (C,D).

Because various linearizing transformations are undefined at some levels of y as initial disease (y_0), y for disease incidence trees was defined as equal to the number of diseased trees plus 0.5 and divided by the total number of trees in the quadrant (5). The appropriateness of the exponential, linear, monomolecular, logistic, and Gompertz models was examined for disease incidence of trees and flowers from each plot by linear regression analysis of transformed values over time (4,5,14,19). The appropriateness of each model was determined on the basis of the coefficient of determination (R^2), the standard error of the parameter estimates, the correlation coefficient when observed values were plotted against the back-transformed predicted values (r), and by the plots of residuals to examine patterns (5).

Disease gradients over time were calculated for the Lake Alfred test and for the plots of Ruby Red grapefruit and Pineapple sweet orange at Hastings. Since the Gainesville plot was smaller, no attempt was made to analyze disease gradients at that location. Slopes of the disease gradients (k) were calculated by plotting Gompertz transformations of the incidence of flowers with disease against the natural logarithm (ln) of the distance from the focus at several sampling times. In Lake Alfred, gradients of disease incidence on flowers were assessed on all of the trees in the transverse lines crossing the central focal tree coinciding with the four cardinal directions (north, south, west, and east) (Fig. 1B); whereas in the plots of Ruby Red grapefruit and Pineapple sweet orange at Hastings, disease gradients were assessed on all of the trees in the four transects (northeast, southeast, northwest, and southwest) running from the focal point (Fig. 1C,D). The slopes of the disease gradients for the different directions of the same plots at different times were compared by the Student's t test.

The rate of disease spread (v) was evaluated for the Valencia sweet orange plot in Lake Alfred and the two plots in Hastings. The rate parameter was calculated as $v = (k/b)s$, in which k = the Gompertz rate parameter, b = the slope of the disease gradient, and s = an arbitrary distance (5, 10, or 15 m) from focus toward the edge of the planting (5).

Aggregation of diseased trees in each experiment within and across rows was calculated by ordinary runs analysis at each sampling date (11) in the plots at Lake Alfred and Hastings. Lloyd's index of patchiness (11) was also calculated at each sampling date as an additional measure of aggregation for the plots at Lake Alfred and Gainesville where a complete census of disease incidence was available.

Isolation of *C. gloeosporioides.* A composite sample of leaves and healthy or diseased flowers was collected at days 11 and 22 after inoculation from each tree on the gradient lines at each of the experimental plots (Fig. 1) to examine the dissemination of the slow-growing orange strain of *C. gloeosporioides* from the focus. In the laboratory, 1-cm-diameter disks were cut from leaves and flower petals. A 0.5-g subsample of each tissue was transferred to a 125-ml flask containing 20 ml of sterile water. Flasks were shaken on a rotary shaker for 15 min and dilution plated onto three petri plates containing a semiselective medium composed of potato-dextrose agar, streptomycin, and copper hydroxide (2). Plates were incubated at 18 C for 4–5 days and transferred to 27 C for 1 day. Slow-growing orange colonies were counted and data expressed as the number of propagules per gram of fresh weight.

RESULTS

Disease development at the experimental sites. At the Gainesville site, a rain event shortly after setting the diseased focal tree brought about some initial spread of the fungus. Disease increased rapidly with rains from 8–15 days postinoculation (Fig. 2A). Disease increased slowly during a dry period from days 15–21. At the end of the bloom period, disease had been detected on over 50% of the trees with about 20% of the total blossoms diseased.

In the experiment at Lake Alfred, rainfall associated with a

slow-moving cold front on days 3–6 postinoculation spread the inoculum, initially resulting in a sharp increase in disease (Fig. 2B). Winds of 2.4 m/s from the northwest (309° east of magnetic north) were noted during a rain event on 30 March. Disease developed rapidly and about 75% of the total trees and over 25% of the total flowers had been affected by the end of the flowering period.

At the Hastings site, no rain occurred until day 5 following the setting of the diseased focal trees (Fig. 2C and D). Frequent rains after day 10 resulted in some disease increase, but fewer than 40% of the trees in each plot had been affected by the end of the bloom period. Cumulative proportion of flowers affected was less than 20% in the plot of Pineapple sweet orange and even lower in the plot of Ruby Red grapefruit where flowering was more scattered.

Analysis of disease progress. All four disease progress models accounted for a high percentage of variation in the incidence of diseased trees and diseased flowers (Table 1). A linear model for disease progress was also evaluated for incidence of flowers with disease but it accounted for a low percentage of the variation in most plots (data not shown). When back-transformed predicted values were correlated with the original observations, the Gompertz model provided the best fit in nearly every case. Only for the incidence of diseased flowers at the Gainesville plot was the correlation coefficient higher with the monomolecular model. When plots of the residuals were examined, patterns were less apparent when the Gompertz model was used than with other models. Thus, the Gompertz model was selected for additional analyses. For this model, the dependent (y) variable is $-\ln(-\ln(y))$ and the independent variable is time (t). The estimated rate parameters of the Gompertz model (k) for both disease incidence of trees and disease incidence of flowers were higher at Gainesville and at Lake Alfred compared to the two plots in Hastings (Table 1).

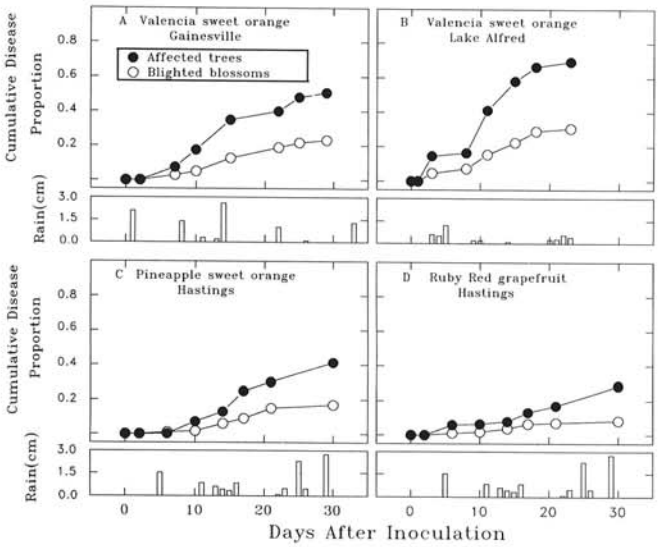

Fig. 2. Cumulative proportion of the total number of trees affected by postbloom fruit drop and cumulative proportion of the blossoms affected at each sample date and rainfall in each of the experimental plots.

Disease gradients. The Gompertz spread model with $\ln(x)$ as the independent variable (x = distance in meters from the infection focus) was chosen to describe the disease gradients in each plot because of the appropriateness of the Gompertz model to describe temporal data from the same plots. Initial disease gradients at Lake Alfred were steeper than at later assessment dates (as indicated by b, the slope of gompits versus ln (x)), demonstrating that a higher density of disease incidence occurred near the focal tree early in the epidemic (Table 2). Overall, disease gradients became flatter over time and disease incidence was greater further from the inoculated focus in the southerly and easterly directions compared to the northerly or westerly direction. Disease gradients on the southerly and easterly lines remained high during the epidemic. At day 8, the greatest number of trees were affected by postbloom fruit drop in the southerly direction (Table 2); and at day 11, the highest population of the pathogen was detected in trees in the southern sector (Table 3). At day 11, trees to the south and the east had flatter gradients (Table 2). Trees to the west had flattened gradients by day 15 but, based on the low correlation coefficient at this time ($r = -0.77$), this may have been due to establishment of secondary foci. At day 22, high levels of disease incidence and large numbers of propagules were detected on trees to the south, east, and west (Table 3). However, propagule concentrations were much lower on those trees than on the focal tree at that time. During the epidemic, predominant winds were from the southwest and southeast in the Lake Alfred area. However, during the hours when rain was

TABLE 1. Linear regression analyses of four models for the transformed incidence of postbloom fruit drop caused by *Colletotrichum gloeosporioides* on flowers and on trees affected over time in the four experimental sites

	Disease incidence—trees				Disease incidence—flowers			
Model	R^2 [a]	r [b]	Intercept (y_0) ± SE	Rate (k) ± SE	R^2 [a]	r [b]	Intercept (y_0) ± SE	Rate (k) ± SE
Valencia sweet orange (Gainesville)								
Monomolecular	86.0	91.8	0.01 ± 0.03	0.02 ± 0.001	86.0	98.8	−0.01 ± 0.01	0.01 ± 0.05
Logistic	83.7	96.5	−2.84 ± 0.15	0.10 ± 0.008	75.7	91.7	−4.59 ± 0.43	0.30 ± 0.03
Gompertz	86.9	98.1	−1.10 ± 0.06	0.05 ± 0.003	83.9	96.2	−1.59 ± 0.08	0.07 ± 0.04
Exponential	80.0	94.0	−2.85 ± 0.13	0.08 ± 0.007	74.0	91.3	−4.60 ± 0.43	0.29 ± 0.03
Valencia sweet orange (Lake Alfred)								
Monomolecular	69.2	86.8	−0.07 ± 0.108	0.05 ± 0.007	83.2	83.9	−0.01 ± 0.01	0.01 ± 0.01
Logistic	84.1	94.3	−2.89 ± 0.213	0.17 ± 0.014	82.3	96.7	−3.71 ± 0.18	0.14 ± 0.01
Gompertz	80.3	98.1	−1.17 ± 0.141	0.10 ± 0.009	85.3	97.3	−1.35 ± 0.05	0.05 ± 0.01
Exponential	82.2	91.5	−2.81 ± 0.152	0.11 ± 0.010	80.5	97.0	−3.74 ± 0.15	0.13 ± 0.01
Pineapple sweet orange (Hastings)								
Monomolecular	71.9	81.2	0.01 ± 0.04	0.02 ± 0.002	51.8	86.1	−0.02 ± 0.01	0.01 ± 0.01
Logistic	78.2	84.2	−4.00 ± 0.22	0.13 ± 0.001	78.3	85.7	−4.89 ± 0.19	0.12 ± 0.01
Gompertz	84.4	94.3	−1.42 ± 0.07	0.05 ± 0.003	71.7	98.4	−1.64 ± 0.07	0.04 ± 0.04
Exponential	73.1	79.9	−3.46 ± 0.19	0.10 ± 0.010	79.4	91.2	−4.98 ± 0.18	0.11 ± 0.01
Ruby Red grapefruit (Hastings)								
Monomolecular	79.3	91.3	−0.01 ± 0.02	0.01 ± 0.001	47.7	93.4	−0.01 ± 0.01	0.03 ± 0.05
Logistic	87.2	97.0	−3.80 ± 0.12	0.10 ± 0.006	72.7	96.5	−4.73 ± 0.13	0.08 ± 0.08
Gompertz	89.3	97.8	−1.39 ± 0.04	0.04 ± 0.002	67.7	97.3	−1.57 ± 0.04	0.02 ± 0.02
Exponential	85.3	85.2	−3.77 ± 0.11	0.09 ± 0.006	73.4	95.5	−4.73 ± 0.13	0.08 ± 0.08

[a] R^2 = Coefficient of determination for the transformed disease percentages. Disease percentages were transformed by $\ln(1/(1-y))$, $\ln(y/(1-y))$, $-\ln(-\ln(y))$, and $\ln(y)$ for the monomolecular, logistic, Gompertz, and exponential transformations, respectively.
[b] r = Correlation coefficient of the back transformed predicted values regressed against the original observations to test models.

falling, prevailing winds were from the north to northwest. At the end of the epidemic, the highest percentages of buttons were detected to the south, east, and west of the focal plant (Table 3).

At Hastings, regardless of the wind-driven rainfall that occurred early in the epidemics, initial disease gradients were not significantly different in any direction in either Pineapple sweet orange or the Ruby Red grapefruit (Table 2). Disease gradients began to flatten slightly after day 11 in both plots. Although no major directionality was observed in the disease gradients, at day 15, the highest percentages of blighted flowers occurred in the southwest and southeast portions of both plots and in the northwest portions of the grapefruit plot. However, secondary foci formed early in the epidemic in the southeast direction of both plots and correlation coefficients for the Gompertz-transformed disease rating vs. distance were low at 15–30 days (Table 2).

Numbers of propagules per gram fresh weight of flowers from the focal trees on day 11 were very high in both plots, increased slightly with continuing flowering in Lake Alfred on day 22, but declined with flower senescence in Hastings by day 22 (Table 3). High populations of the pathogen were detected in most of the trees in most of the transects of both plots at day 11, except the southeast transect of the Pineapple sweet orange plot and the northeast transect of the grapefruit plot, respectively (Table 3). The numbers of propagules in the southeast and northeast directions in the plot of Pineapple sweet orange increased by day 22. Although disease gradients increased with time, the numbers of propagules were lower on grapefruit and remained constant or decreased on Pineapple sweet orange at day 22. Except for the focal plant, few buttons were recorded after 3 mo, and no significant differences were detected among transects in either plot.

The rate of disease spread (v) for the Valencia sweet orange plot in Lake Alfred and the Ruby Red grapefruit plot in Hastings tended to increase during the middle of the epidemic then decrease at the end of the assessment period for all distances, whereas the rate continued to increase throughout the epidemic for the Pineapple sweet orange plot in Hastings for all distances (Table 4). Rate of disease spread was somewhat greater in the Valencia plot at Lake Alfred compared to the two Hastings plots.

The number of runs of diseased trees within and across rows increased and Z values declined in all plots as the epidemics progressed. Despite this tendency, there was significant aggregation ($Z < -1.64$) only on the last evaluation date in the Ruby Red grapefruit plot at Hastings.

When disease incidence on flowers was analyzed using Lloyd's index of patchiness, there was significant aggregation at the first sample date at the Gainesville plot (LIP = 62.2) and in Lake Alfred (LIP = 77.3). However, these values declined rapidly and on the last sample date patchiness indices were only 0.19 in Gainesville and 0.90 in Lake Alfred.

DISCUSSION

When inoculum was present and susceptible tissue was available, postbloom fruit drop increased after rainfall periods. Occurrence of rain also influenced the disease spread through pathogen dispersal in all experimental plots. The largest estimated epidemic rate (k) for the disease incidence on flowers was in the plot of Valencia sweet orange in Gainesville and was probably due to the higher amounts and more uniformly distributed rainfall during the flowering there than at the other locations. Little increase in disease occurred after dry periods, even when susceptible open flowers were available. These results agree with those of previous reports (1,7,18). Alternating wet and dry periods, mainly in the plot of Valencia sweet orange at Lake Alfred, resulted in fluctuations in the disease gradients and resulted in increases and decreases in disease incidence of open flowers. Similar changes have been observed in other diseases on perennial trees in tropical or subtropical climates (5). The assessment of postbloom fruit drop as disease incidence on open flowers provided more information than was derived from the estimation of disease incidence on trees. Disease incidence on open flowers was transformed as cumulative values of blighted open blossoms from the different assessment dates to evaluate models for the linearization of disease progress over time. The use of cumulative values did not overestimate disease because when postbloom fruit drop incidence was high, closed flowers as well as open flowers in the entire clusters of flowers become blighted (7,16).

The linearization of disease progress curves is useful to deter-

TABLE 2. Comparison of slopes (b) postbloom fruit drop disease gradients from a focal source on Valencia sweet orange at Lake Alfred, and on Pineapple sweet orange and Ruby Red grapefruit at Hastings

Days[a]	b[b]	r[c]	b	r	b	r	b	r
Valencia sweet orange (Lake Alfred)								
	South		North		East		West	
3	−1.39	−0.98	−1.22	−0.97	−1.38	−0.98	−1.08	−0.72
8	−0.83	−0.89	−1.31	−0.98	−1.08	−0.84	−1.11	−0.85
11	−0.73	−0.98	−1.21	−0.97	−0.65	−0.92	−1.00	−0.83
15	−0.55	−0.95	−1.07	−0.97	−0.44	−0.95	−0.85	−0.77
18	−0.70	−0.97	−1.25	−0.97	−0.62	−0.97	−0.85	−0.94
22	−0.76	−0.97	−1.33	−0.98	−0.70	−0.98	−0.92	−0.94
Pineapple sweet orange (Hastings)								
	Southwest		Northeast		Southeast		Northwest	
4	−0.96	−0.92	−0.96	−0.91	−0.77	−0.84	−0.99	−0.98
11	−0.67	−0.96	−1.04	−0.98	−0.54	−0.69	−0.97	−0.98
15	−0.65	−0.97	−0.93	−0.93	−0.52	−0.70	−0.98	−0.98
18	−0.58	−0.95	−0.91	−0.91	−0.48	−0.75	−0.97	−0.97
22	−0.56	−0.96	−0.81	−0.94	−0.43	−0.77	−0.77	−0.89
30	−0.51	−0.98	−0.75	−0.92	−0.36	−0.66	−0.75	−0.90
Ruby Red grapefruit (Hastings)								
	Southwest		Northeast		Southeast		Northwest	
4	−0.85	−0.92	−0.85	−0.92	−0.85	−0.92	−0.89	−0.98
11	−0.95	−0.98	−0.88	−0.98	−0.77	−0.80	−0.96	−0.97
15	−0.61	−0.80	−0.65	−0.92	−0.52	−0.70	−0.51	−0.84
18	−0.66	−0.80	−0.69	−0.89	−0.56	−0.72	−0.52	−0.84
22	−0.84	−0.88	−0.74	−0.94	−0.74	−0.78	−0.70	−0.91
30	−0.69	−0.87	−0.76	−0.94	−0.75	−0.76	−0.52	−0.97

[a] Number of days after the infected focal tree was placed in each plot.

[b] b = Slope generated by regressing $-\ln[-\ln(y)] = a + b\ln(x)$, where y = disease incidence in open flowers transformed by Gompertz, and x = distance from the focus in meters. Slopes were compared by paired Student's t tests and were significantly different for different directions in the same plots, $P \leq 0.05$.

[c] Correlation coefficients of back-transformed predicted values regressed against the original observations. All are significant ($P \leq 0.05$).

mine epidemic rates, to project future disease, and to make decisions about disease control strategies (4). The Gompertz model generally was superior to the other models tested to linearize the increase of postbloom fruit drop over time in most of the experiments when disease was assessed as disease incidence on trees over time. The logistic or exponential models appeared to be more appropriate to linearize disease progress curves in some experiments when postbloom fruit drop was assessed as disease incidence on open flowers. However, the Gompertz model provided the highest correlation coefficients of observed vs. predicted values, lower mean square errors of the regression, and fewer patterns in residual plots.

The use of numbers of buttons per tree resulting from flower infection has been a useful variable to determine the incidence of postbloom fruit drop and the effectiveness of fungicides on disease (17,18). In all experiments, the highest percentage of buttons (buttons/[buttons + fruit set] × 100) occurred in plots with a high incidence of infected flowers. The highest percentages of buttons were observed in the plots of Valencia sweet orange at Lake Alfred and Gainesville, and the lowest percentages occurred on Pineapple sweet orange and Ruby Red grapefruit in Hastings. High populations of *C. gloeosporioides* but few buttons per tree at the end of the epidemic at both plots in Hastings may have resulted from severe fruit drop of whole flower clusters without button formation.

The rate of disease spread (v) indicated more rapid movement in the plot of Valencia sweet orange than in the Pineapple sweet orange or Ruby Red grapefruit. In commercial orchards in Florida, postbloom fruit drop is more serious on Valencia oranges than on early or midseason oranges (e.g., Hamlins or Pineapples) or on grapefruit. Thus, these results concur with field experience, but these data must be interpreted with caution since the experiments were conducted under different conditions in simulated new plantings.

Since slow-growing orange strains of *C. gloeosporioides* were not detected from many random samples taken from the trees before trees were inoculated, it was assumed that the central, focal, and diseased trees were the only source of inoculum within the plots. Based on the disease gradients, disease incidence was greatest close to the focal trees. The presence of secondary foci at the southeast quadrant in the Hastings plots and the lower number of diseased trees in the northern quadrant at the Lake Alfred site indicated that factors such as wind or insects may be involved in the spread of *C. gloeosporioides*. The association of windblown rain with the spread of postbloom fruit drop has not been demonstrated previously for this disease. The highest concentration of disease in the plot at Lake Alfred occurred early in the south and east quadrants and appeared to be associated with windblown rains from the north-northwest of 2.4 m/s on 30 March 1991. Also, the lack of aggregation within rows in this plot could be the result of diagonal and across-row spread of postbloom fruit drop. Spore-carrying droplets of *Septoria nodorum* and *Pseudocercosporella herpotrichoides* have been collected up to 4 m from the inoculum source in rain tower experiments with wind speeds of about 2.5 m/s (11). Raindrops fragment into smaller droplets whose size depends on the size of the impacting drop, its velocity, and the nature of the surface (13). In citrus groves with trees at least 1 m tall, Gottwald et al (12) suggested that because raindrops fragment into smaller droplets, inoculum from lesions oozing bacteria of *Xanthomonas campestris* pv. *citri* did not reach even the closest neighboring trees in a new grove in the absence of winds because of the distance between individuals. However, during rainstorms with high winds, these small droplets could be carried predominantly downwind to neighboring trees. Diseased trees 1 m tall or higher in groves also elevate the foci of inoculum above the boundary layer of air close to the ground. This exposes inoculum to the turbulent air layers where dissemination of inoculum due to eddies is possible. The predominance of uniform, mild rains without strong winds or changes in wind direction during storms could have been the cause of lack of directionality of disease spread in the plots at Hastings.

Thus, water splash from rainfall or from overhead irrigation during the bloom period appears to be an important factor in

TABLE 3. Recovery of the slow-growing orange strain of *Colletotrichum gloeosporioides* from flowers by a selective isolation procedure from the transects of the plots

Focus or transect[a]	Propagules/g fresh wt[b] Day 11	Propagules/g fresh wt[b] Day 22	Buttons (%)[c]
Valencia sweet orange (Lake Alfred)			
Focal plant	163,300	360,000	93.5
North	0	17[d]	7.8[d]
East	11	16,177	44.7
South	40[d]	19,833	59.3
West	0	9,230	50.6
Pineapple sweet orange (Hastings)			
Focal plant	44,330	5,080	98.2
Northeast	7,166	10,400[d]	7.3
Southeast	235	5,258	13.1
Southwest	12,540	237	9.5
Northwest	10,667	1,550	10.0
Ruby Red grapefruit (Hastings)			
Focal plant	44,165	22,260	97.2
Northeast	0	620	20.0
Southeast	1,650	316	7.5
Southwest	2,650	200	14.5
Northwest	6,200	133	9.4

[a] Transects in each plot as indicated in Figure 1.

[b] Propagule numbers from flowers as determined for each tree by plating of tissue washes on a selective medium. Each data point is the mean of three trees except for the focal plant, where it is the mean of three samples.

[c] Percentage buttons determined by counting total numbers of buttons and fruit 3.5 mo after petal fall and expressed as buttons/(buttons + fruit) × 100. Each data point is the average of the three trees in each gradient line.

[d] Significant differences as determined by paired Student's t tests between transects only: Valencia sweet orange (Lake Alfred): Day 11, South significantly greater than North and West. Day 22, North significantly less than East and South. Buttons, North significantly less than East, South, and West. Pineapple sweet orange (Hastings): Day 22, Northeast significantly greater than northwest and southwest. All other values do not differ significantly, $P \geq 0.05$.

TABLE 4. Rates of disease spread at different times and distances from the focus of infection of *Colletotrichum gloeosporoides* in three experimental sites

Days	V_{5m}[a] ± SE	V_{10m} ± SE	V_{15m} ± SE
Valencia sweet orange (Lake Alfred)			
3	0.491 ± 0.044	0.491 ± 0.088	1.474 ± 0.133
8	0.601 ± 0.054	0.601 ± 0.108	1.802 ± 0.162
11	0.772 ± 0.069	0.772 ± 0.139	2.317 ± 0.208
15	0.687 ± 0.062	0.687 ± 0.124	2.062 ± 0.186
18	0.578 ± 0.052	0.578 ± 0.104	1.734 ± 0.156
22	0.539 ± 0.049	0.539 ± 0.097	1.617 ± 0.146
Pineapple sweet orange (Hastings)			
3	0.272 ± 0.016	0.272 ± 0.033	0.815 ± 0.049
8	0.311 ± 0.019	0.311 ± 0.037	0.932 ± 0.056
11	0.325 ± 0.019	0.325 ± 0.039	0.974 ± 0.058
15	0.340 ± 0.020	0.340 ± 0.041	1.020 ± 0.061
18	0.389 ± 0.023	0.389 ± 0.047	1.167 ± 0.070
22	0.422 ± 0.025	0.422 ± 0.051	1.266 ± 0.076
Ruby Red grapefruit (Hastings)			
4	0.233 ± 0.012	0.233 ± 0.023	0.698 ± 0.035
11	0.225 ± 0.011	0.255 ± 0.022	0.674 ± 0.034
15	0.349 ± 0.017	0.349 ± 0.035	1.048 ± 0.052
18	0.329 ± 0.016	0.329 ± 0.033	0.988 ± 0.049
22	0.265 ± 0.013	0.265 ± 0.026	0.795 ± 0.040
30	0.294 ± 0.015	0.294 ± 0.029	0.882 ± 0.044

[a] V_x = the rate of disease spread where x indicates the distance in meters from the focus of infection based on the integrated model $-\ln(-\ln(y)) = a^* - b \ln(s) + r_G t$, where y = disease incidence, b = the slope of disease incidence over distance s, t = time in days, and r_G = the Gompertz rate of disease increase.

the limited and localized spread of postbloom fruit drop in citrus groves of Florida. The effect of windblown rain on pathogen dispersal and resulting disease spread over distances of several meters has been demonstrated in this study. Further studies are needed to determine if rain driven by strong winds is responsible for the spread of postbloom fruit drop over long distances and to understand the spatial dynamics of the disease in larger plantings.

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