Field Spread of Anthracnose Fruit Rot of Strawberry in Relation to Ground Cover and Ambient Weather Conditions

L. V. MADDEN, Professor, L. L. WILSON, Research Assistant, and M. A. ELLIS, Professor, Department of Plant Pathology, Ohio State University, Ohio Agricultural Research and Development Center, Wooster 44691

ABSTRACT

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.

Strawberry plots of the everbearing cultivar Tristar were established in each of 2 yr in Ohio. Ground cover between and within rows consisted of plastic, straw, or bare soil. Fruit infected by *Colletotrichum acutatum* (cause of anthracnose fruit rot) were introduced immediately before a rain episode in all plots except controls (which had no soil cover). Seven days after the rain, fruit disease incidence in row segments within 61 cm of the inoculum source was 0.19, 0.07, 0, and 0 for plastic, soil, straw, and uninoculated control plots, respectively, in 1990; in 1991, incidence was 0.16, 0.07, 0, and 0 for plastic, soil, straw, and controls, respectively. In general, disease incidence declined with distance from the inoculum source, an indication that the introduced infected fruit were the source of spores for rain splash dispersal. Cumulative incidence of disease at the end of the season was consistent with results for 7 days after infestation. Disease incidence in the plastic and soil plots was related to weather variables using stepwise regression analysis. The best relationship was based on the product of four terms: rain amount (cm), days from introduction of inoculum minus 6, an index of infection (0–1) based on wetness duration starting with a rain episode and average temperature during the wetness period, and an ordered distance from the spore source (1–3; 1 for closest, 3 for greatest distance and separated by a row of plants). Results confirm previous controlled studies with a rain generator that surface topography or ground cover greatly affects dispersal of spores by rain splash and that the use of straw mulch reduces disease incidence.

Anthracnose of strawberry (*Fragaria* × *ananassa* Duchesne) is caused by several *Colletotrichum* species (21). Both the fruit and vegetative parts of the plants are susceptible. Although *Colletotrichum* species generally are considered warm weather (tropical and subtropical) pathogens (4,9), one species, *C. acutatum* J.H. Simmonds, is being found in increasing frequency in more northern regions of the United States such as Ohio (9). *C. acutatum* can infect the stolons, petioles, and leaflets (9), but in commercial fields, fruit rot is of major economic importance. In fact, infection of the vegetative parts of plants in Ohio has seldom been observed (M. A. Ellis, *unpublished*). Once established in a northern planting, the pathogen can overwinter in infected fruit (25). Overwintering in vegetative plant parts in warmer climates also has been demonstrated (5).

Epidemics of anthracnose result, in part, from the rain splash dispersal of conidia (12,29). Other workers have observed that increase in anthracnose of mango and citrus, caused by a related species (*C. gloeosporioides*), is positively associated with amount of rainfall and environmental conditions after rain episodes that favor infection (4,7,19). Using a rain simulator (generator), we have shown that substantial spore dispersal and subsequent infection of strawberry fruit by *C. acutatum* can occur with rains of less than 15 min duration (16). Splash droplets are known to travel very short distances (generally <10 cm) (6,11,26,27); therefore, movement of inoculum from a lesion to a new infection site requires the multiple resplashing of spore-carrying droplets (12). One would expect the ground cover or surface topography to greatly influence the splashing of droplets (15); this was demonstrated with the rain simulator (14,28). The greatest dispersal, as measured by infected fruit or fungal colonies on a selective medium, occurred with a plastic ground cover; the least dispersal occurred with straw. Bare soil was intermediate for spore dispersal. Additionally, when the inoculum source and sampling plates were positioned on raised blocks (46 cm above a soil surface), so that the ground cover was virtually eliminated, dispersal was reduced by more than 95% compared with spore movement over soil (13).

Plastic and straw are commonly used as mulch in different parts of the United States (1). Because of the potential effects of these mulches on the dispersal of *C. acutatum* and spread of anthracnose, we decided to confirm the rain simulation results under field conditions. The major objective of this study was to evaluate the effect of ground cover on the spread of strawberry anthracnose in Ohio. The secondary objective was to determine the relationship between fruit disease incidence and ambient weather conditions.

MATERIALS AND METHODS

Plot establishment. Field plots were established at the Ohio Agricultural Research and Development Center, Wooster, in an area where strawberries had never been grown. Each plot consisted of three 1.52-m-long rows with 76 cm between rows (Fig. 1) and approximately 10 cm between plants. Distance between plots was 2 m. Transplants of the everbearing (day-neutral) cultivar Tristar were hand-planted on 16 May 1990 and 3 May 1991. For the first 8 wk after transplanting, flowers were manually removed from all plants to ensure greater fruit set and vegetative growth.

A total of 12 plots were established, four plots randomized in each of three blocks. In three plots (one per block), the ground was covered by 6-mil clear plastic mulch in which slits were made for the plants. In three other plots, straw was applied to the entire area to a depth of 6 cm, including the aisles and spaces between plants within rows. The remaining six plots consisted of no ground cover (i.e., bare soil), three of which were used as no-inoculum controls. The area between plots was covered with straw (6 cm deep). Weeds were removed by hand from all plots throughout the growing season.

Preparation of the inoculum source. Infected strawberry fruit were used as the source of inoculum. Preparation followed procedures described in Yang et al (28) and Madden et al (16) and are summarized here. Immature (white) fruit were detached from greenhouse-grown plants, rinsed with deionized water, surface-sterilized in 70% ethanol for 1 min, rinsed again with deionized water, and placed on elevated screens in 5-L plastic containers. The pedicel of each fruit was inserted through the mesh of the screen and immersed in deionized

Accepted for publication 7 June 1993.

water.

Fruit were inoculated by being sprayed with a suspension (5×10^4/ml) of *C. acutatum* conidia until runoff. Then, the containers were sealed and incubated at 26 C for 24 hr, at which time the lids were removed. Fruit were incubated for another 7 days (26 C) to ensure lesion development and sporulation. With this protocol, lesions covered the entire fruit surface after the total of 8 days. Using methods described in Madden et al (16), mean conidia density was 3.4×10^8 per fruit.

Disease spread. Immediately before a rain episode, five infected fruit were placed in a tight cluster, between rows one and two, 30 cm from the beginning of the rows (Fig. 1). Ripe fruit from all plants were removed so that only immature fruit were exposed to splashed inoculum. The infected fruit were placed in all plots except the three control plots and were removed 24 hr after the end of the rain.

Each row was divided into three sections, 30, 61, and 61 cm in length (Fig. 1), for disease assessment. Starting 7 days after initial introduction of infected fruit, all ripe fruit were hand-picked from all plants every 3–4 days. The harvested fruit in row sections labeled 1–3 were removed from the plot and visually assessed for anthracnose fruit rot. The three numbers in Figure 1 represent ordered "distances" from the original infected fruit. The numbers refer to: 1) fruit up to 61 cm from the source, in rows adjacent to the source; 2) fruit from 62 to 122 cm away, in adjacent rows; and 3) fruit from 114 to 167 cm away, separated from the source by a row of plants. (These distance codes were chosen because rain simulation results previously had shown the effects of distance and plant barrier on spore dispersal [29].) Sections of rows rather than exact distances were used so that there would be sufficient fruit to observe for anthracrose symptoms. In the first section of each row (not labeled in Fig. 1), harvested fruit were placed on the ground in the row section. Infected fruit in these row sections served as the inoculum source for dispersal after the primary dispersal event. The sampling technique mimicked the typical commercial harvesting practice of picking all ripe fruit, which also permitted an appraisal of dispersal from the row ends throughout the seasons. (In commercial fields, pickers frequently do not pick diseased fruit.) By removing fruit, secondary spread outside the row ends was minimized.

Disease incidence (y) for each labeled distance (Fig. 1), treatment, replication (block), and sampling time was calculated as the proportion of fruit with visual symptoms of anthracnose. The cumulative disease incidence (Y) at any given sampling time was determined by summing the number of diseased fruit (X) and total ripe fruit (N) up to time t and calculating $Y = X/N$. Because new fruit were produced throughout the growing season, Y could increase or decrease between any two sampling times, depending on the number of new infections and fruit set that occurred.

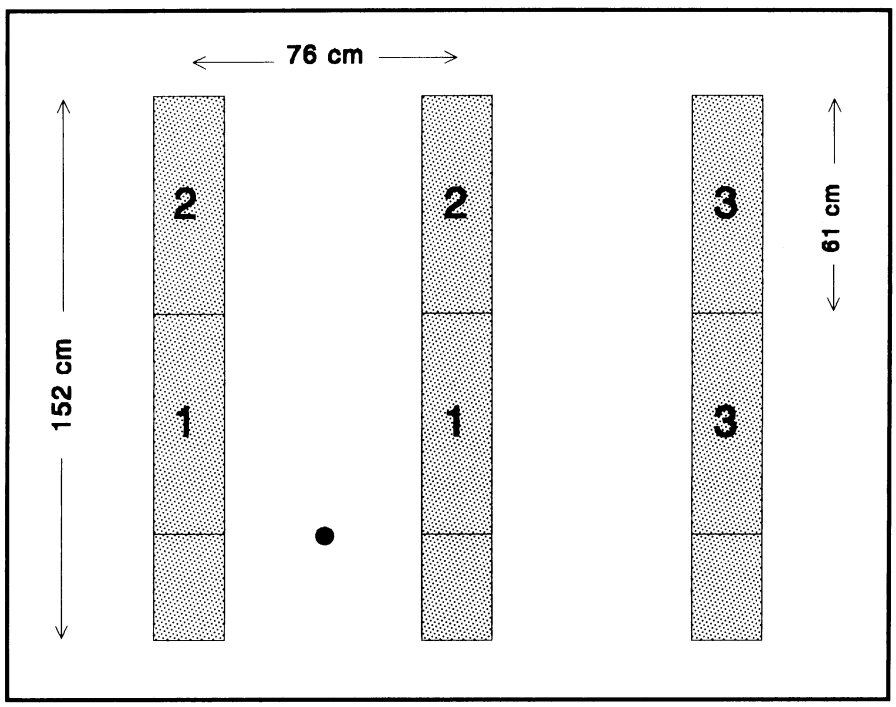

Fig. 1. Schematic diagram of the strawberry plots used to study the spread of anthracnose. Rectangles represent rows of strawberry plants. Numbers refer to sampling locations (data from all fruit were pooled for each number). Closed circle between first and second rows represents the position of the introduced (initial) inoculum source, i.e., five fruit infected by *Colletotrichum acutatum*.

Environmental monitoring. Environmental data were collected with a micrologger (CR-21, Campbell Scientific, Logan, UT). Air temperature was monitored every minute with a Campbell model 101 sensor located under a radiation shelter at canopy height (20 cm) and positioned within a plant row. Averages for 10-min intervals were stored. Total rainfall in each 10-min interval was determined with a tipping bucket rain gauge (model RG 2501, Sierra-Misco, Berkeley, CA) placed adjacent to a strawberry row. Surface wetness (from rain or dew) was estimated with a Campbell model 231 wetness sensor coated with two layers of white latex paint and positioned within a row at a height of 3 cm. Voltage for wetness was monitored every minute, and 10-min averages were stored. Sensors were calibrated every month.

Weather variables were calculated for each rain event. These included: total rain amount (mm); wetness duration, starting with a rain and ending when the sensor recorded dry (a period that could be much longer than the rain duration); and average temperature during the wetness period. Maximum and minimum daily temperatures also were calculated.

Data analyses. The experimental design was a repeated-measures factorial (17), with ground cover and block as randomized (crossed) factors, ordered distance from the source (1–3 in Fig. 1) as a spatially repeated measure, and sampling time as a temporally repeated measure. Analysis of variance (ANOVA) was used to determine the effect of these factors and their interactions on disease incidence. To stabilize variances, y was transformed to $y^* = \arcsin(y^{1/2})$ prior to ANOVA. Because of unequal number of fruit (n) in the row sections at any time, weights for ANOVA were chosen and equaled n. Data were also pooled across blocks to represent mean disease incidence or cumulative incidence at a given time. Variability was represented as the standard error of y or Y, which was calculated using the formula for a binomial variable (2).

The relationship between y at any time and weather variables was assessed using regression analysis. To obtain a linear scale for disease, y was transformed with the multiple infection transformation (8), $y' = \ln(1/[1 - y])$. For each sampling time, y' was related to weather variables from 6–11 days earlier, because latent period for anthracnose fruit rot is between 6 and 11 days (L. V. Madden, *unpublished*). For example, y for day 32 would be related to environmental data from days 21 to 26. Because more than a single rain event can occur over these partially overlapping 5-day periods, the following variables were calculated: maximum amount of rain among the rain events during each period, minimum amount of rain, total rain, maximum

wetness duration, minimum wetness duration, total wetness duration, average temperature for these three possible wetness periods, and average daily maximum and minimum air temperatures. Using a combination of observed wetness duration and temperature, an index of (predicted) infection (i; 0–1) was calculated using equation 3 in Wilson et al (24), which was based on inoculations of immature fruit under constant environmental conditions. The i index was transformed as $v = \ln(1/[1 - i])$ for analysis to provide a linear scale. All second and higher-order interactions of these variables also were calculated.

Stepwise regression (forward selection method) was used to determine the variables or combination of variables that provided the best prediction of y'. Criteria used for model selection was a maximum coefficient of determination (R^2) adjusted for degrees of freedom (R_a^2), significant estimated parameters ($P < 0.05$), and a random residual plot (2). Regressions were performed for each year separately, first only for the closest distance to the spore source (1 in Fig. 1), and then for the entire data set. In the latter case, the ordered distance variable ($s = 1, 2, 3$) and its interactions with the above variables were considered in the stepwise regression procedure. MINITAB (18) was used for all regression analyses and ANOVA.

RESULTS

Ground cover, 1990. In 1990, infected fruit were introduced into the field plots on 16 July, immediately before a rain episode that totaled 26 mm. With a wetness duration of 8.2 hr and an average temperature of 18.8 C (Table 1), an index of (predicted) infection (24) of 0.10 was obtained.

Seven days after the initial dispersal of conidia in 1990, infected fruit were found in all plastic and soil plots but not in the control plots (Fig. 2). Infected fruit were found only in one of the three straw-cover plots. For both plastic and bare soil, the highest incidence (y) was for the closest distance to the inoculum source, as one expects for dispersal gradients (2). At 7 days, mean y across all distances was 0.10, 0.05, 0.007, and 0.0 for plastic, soil, straw, and the control, respectively. There were similar results for cumulative disease incidence at the end of the season (labeled "final" in Fig. 2). Bare soil and a plastic ground cover had similar disease levels, and there was a gradient of Y from $s = 1$ to $s = 3$. Cumulative incidence was very low in the straw plots, and only a few infected fruit were eventually found in the controls (representing less than 0.5% of all fruit). Infected fruit in the straw and control plots were restricted to one or two of the three blocks.

Because disease incidence was mostly zero for straw and control plots, and because one or more blocks had no infected fruit to place in the row ends after introduced source fruit were removed, ANOVA was done only for the plastic and bare soil data. Also, using treatments with all or mostly zeros will result in biased estimates of error terms and F statistics (17). Distance from the source ($P = 0.03$), sampling time ($P < 0.001$), and the interaction of time and distance ($P = 0.01$) significantly affected transformed disease incidence (y^*). Ground cover (plastic vs. bare soil; straw not included) and other interactions were not significant ($P > 0.10$). The distance effect was due to the decline in y with ordered distance from the original source. Time was significant because of the change in y over the sampling period. Figure 3 shows the multiple infection transformation of y (y') in relation to time. During some sampling times, incidence was nearly zero for all distances and ground covers (e.g., from $t = 14$–25 days). Highest mean y' reached about 0.8 (corresponding to $y = 0.55$). The significant interaction indicated that the change in incidence over time was not the same for each distance. For instance, incidence at distance 3 ($s = 3$; see Fig. 1) varied much less over time than incidence at the closest distance ($s = 1$).

Ground cover, 1991. Infected fruit were introduced into the plots in 1991 on 6 August, immediately before a 15-mm rain. Wetness duration was longer and average temperature was higher than in 1990 (Table 1), resulting in a substantially higher index of (predicted) infection (i).

Infected fruit were found in all plastic and bare soil plots 7 days after initial spore dispersal (Fig. 2), with no or very few infected fruit at the greatest distance from the source. No infected fruit were found in the straw or control plots. Average disease incidence across all distances was 0.11 for plastic and 0.05 for soil at 7 days. By the end of the experiment, only a few infected fruit were observed in the straw or control plots, and not in all blocks (Fig. 2). For plastic, there was a decline in cumulative disease incidence with distance by the final assessment (see "final" in Fig. 2) as well as at intermediate times (*data not shown*). For the bare soil, cumulative disease incidence was lowest at the greatest distance.

As in 1990, ANOVA was conducted only for the plastic ground cover and bare soil. ANOVA indicated a significant effect of distance ($P = 0.002$), time ($P < 0.001$), and the interaction of time and distance ($P = 0.01$). The significance and interpretation of these factors and interaction are the same as for 1990. Unlike the first year of the experiment, however, ground cover (plastic vs. soil) was significant ($P = 0.03$), with plastic

Table 1. Wetness periods (W) starting with rain events, average temperature during the wetness periods (T), amount of rain, and index (0–1) for infection of strawberry by *Colletotrichum acutatum* based on T and W (i)[a]

Days	Wetness duration (hr:min)	Temperature (C)	Rain (mm)	i
1990				
0	8:20	18.8	26	0.10
5	9:20	20.3	5	0.15
6	17:40	19.8	30	0.47
8	10:00	18.7	37	0.13
11	2:00	18.9	1	0.03
12	9:30	17.5	1	0.10
13	9:00	18.4	1	0.11
16	5:40	20.8	9	0.08
24	3:30	19.9	1	0.05
25	10:10	20.3	39	0.18
27	11:50	18.5	20	0.16
28	6:00	14.9	20	0.04
31	7:00	18.2	1	0.08
32	13:00	17.7	31	0.16
34	10:50	13.3	10	0.05
1991				
0	20:10	20.4	15	0.65
7	9:40	19.5	1	0.14
11	4:50	21.2	3	0.07
12	1:30	21.9	1	0.03
16	21:20	17.6	18	0.42
22	4:10	20.3	1	0.06
25	22:00	20.5	16	0.74
27	24:30	17.9	10	0.58
28	14:40	16.0	2	0.13
36	14:00	21.7	1	0.40
38	16:50	21.0	19	0.52
42	19:20	19.9	49	0.56

[a]Calculated using equation in Wilson et al (24) for immature fruit. Time is measured as days from introduction of inoculum.

having a higher incidence than bare soil. Other interactions were not significant ($P > 0.05$). The highest y' incidence was 1.38 (exceeding the scale used in Fig. 3), corresponding to $y = 0.75$.

Weather variables and disease incidence. At least one rain episode occurred 6–11 days before each sampling time during both years of the study. These episodes varied greatly in amount of rain, as well as the wetness duration and average temperature during the wetness period (Table 1). The index of infection (i) ranged between 0.03 and 0.74. New infected fruit were found at each sampling time in the plastic and bare soil plots, although mean incidence was near zero for some times and greater than 0.5 (or $y' > 0.8$) for other times at the closest distance to the inoculum source ($s = 1$).

Low values of disease incidence normally were preceded by low values of i, low rain amounts, and short wetness durations. For instance, between days 11 and 24 in 1990, rains were less than 10 mm and i was less than 0.12; y' was subsequently low between days 14 and 28 (Fig. 3). High y' later in the season was associated with increases in rain amount in 1990 and with increases in rain amount and i in 1991. A major exception to this trend was for weather data on days 6 and 8 in 1990; a high y' did not immediately follow relatively high i and rain amounts.

Several weather variables were significantly correlated ($P < 0.05$) with y', although the correlation coefficients were all low (<0.5). In general, maximum wetness duration and maximum rainfall amount for the rain events 6–11 days before each disease sampling time had higher correlations than the minimum or total values for these weather variables. The weather variables also were intercorrelated so that stepwise regression analysis resulted in models with only one or two variables for predicting y'. As with ANOVA, regression analysis was limited to data from plastic ground cover and bare soil plots.

When data from only the closest distance ($s = 1$) were analyzed, the regression model that satisfied the selection criteria and was consistent between years comprised only one independent variable (Table 2). The variable (vrt') was a product (interaction) of three terms: 1) transformed index for fruit infection (v, dimensionless) (24), which is based on wetness duration and temperature during the wetness period; 2) maximum rain amount (r, cm); and 3) time from the initial introduction of infected fruit minus 6 (t', days). At the first sampling (7 days), $t' = 1$ and the term reduces to vr. As the season progresses, t' increases to reflect the buildup of inoculum in the row ends. The product vr suggests that rain and a favorable environment for infection were both necessary for new infected fruit.

Although the regression equation had the same significant term for both years, the estimated coefficient for vrt' was higher for 1990 than for 1991 (Table 2). This shows a greater change in y' with changes in any of the three terms of vrt' in 1990 compared with 1991. For both years, R^2 values were relatively low, indicating the high proportion of variation in y that was not explained by the weather variables.

When data from all locations were analyzed with stepwise regression, two terms were significant ($P < 0.05$). These were vrt' (as with the simpler situation above) and the product of vrt' and ordered distance from the source, $vrt's$ (Table 2). The negative coefficient estimated for the four-way interaction represents the decline in y' with distance from the original spore source. The second form of this equation in Table 2 shows how this model is analogous to the one for the close distance ($s = 1$) case, with a regression coefficient for vrt' dependent on s. This equation shows that there are greater changes in y' with changes in vrt' close to the source compared with farther away, which can be seen in Figure 3. R^2 values were somewhat higher for the full data set compared with that using only the closest distance to the source.

DISCUSSION

The spread of anthracnose fruit rot of strawberry, resulting from rain splash dispersal of conidia, was found in this field study to be highly dependent on

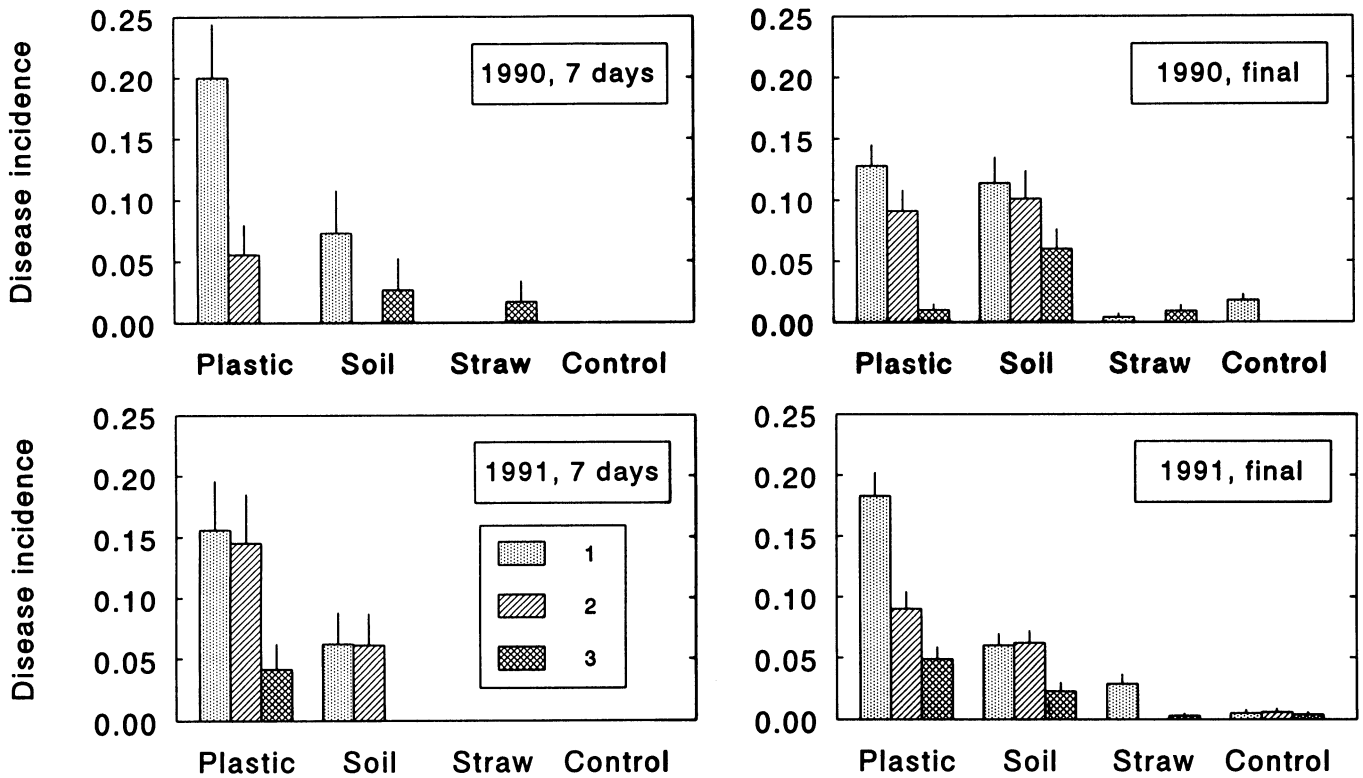

Fig. 2. Proportion of strawberry fruit infected by *Colletotrichum acutatum* in field plots with bare soil, plastic, or straw ground cover in each of 2 yr. Three numbered bars refer to sampling locations (see Fig. 1). Graphs at left are for the first sampling (7 days after inoculum introduction). Graphs at right are for the final cumulative disease incidence, i.e., total infected fruit divided by total fruit produced for the entire assessment period. Vertical lines are standard errors. Control plots did not have introduced inoculum.

ground cover or surface topography. Compared with plastic ground cover or bare soil, there was very little spread in plots with a straw ground cover. This confirms results with a rain simulator for splash dispersal of *C. acutatum* in which the intensity, duration, and timing of rain episodes could be controlled (16,28,29). The large difference between straw plots and the others with an initial spore source (plastic and soil; see Fig. 2) was maintained throughout the growing seasons as new fruit were produced by the everbearing cultivar.

The effect of ground cover on splash dispersal likely is due to several mechanisms (12). Because trajectories of splash droplets are very short, multiple resplashings on the ground normally would be needed before a spore reaches an infection site. Therefore, greater loss of spore-carrying droplets through surface infiltration is expected with straw than with plastic. The transfer of kinetic energy from impacting raindrops to the splash droplets, and also the trajectories of droplets, may be affected as well (12,23). This is confirmed by the few studies that showed the effects of ground cover or surface topography on the splashing of water drops or the movement of droplets and/or spores over areas that varied in roughness (12,28). Smith and Spiers (22) have also observed in field studies with natural inoculum (in Mississippi) that the incidence of anthracnose crown rot, caused by *C. fragariae*, was lower with straw than with plastic mulch. Incidence of leather rot (caused by *Phytophthora cactorum*) in a commercial field of the strawberry cultivar Midway in Ohio was much higher in rows next to bare soil compared with straw (20), which agreed with other rain simulation tests (14).

Results using the rain simulator also showed greater splash dispersal of *C. acutatum* and *P. cactorum* with a plastic ground cover than bare soil (14,28,29). This was initially seen in the field studies (Fig. 2), but by the end of the season, differences were apparent in only one of the years. Variation in the environment over time as well as differences between field soil and the soil mix used with the simulator (both depth and bulk density) could have contributed to these results. Even with the rain simulator, large differences in the volume of splash droplets were not found between plastic and soil (12), indicating that multiple processes are involved in rain splash dispersal.

The predominant short-range nature of splash dispersal (6,11,26,27) was verified in this field study by the higher disease incidence near the original spore source ($s = 1$) compared with the more distant sampling locations ($s = 2$) or locations separated from the inoculum source by a plant-row barrier ($s = 3$). Plant barriers also have been shown to reduce dispersal in simulation tests (28). The disease gradient was, for the most part, maintained throughout the growing season, as shown by the cumulative disease incidence at the final sampling time (Fig. 2). Control plots, which were only 2 m from plots with introduced inoculum, had very few infected fruit throughout the season, and their location was not related to any particular sampling location. The ground between all plots

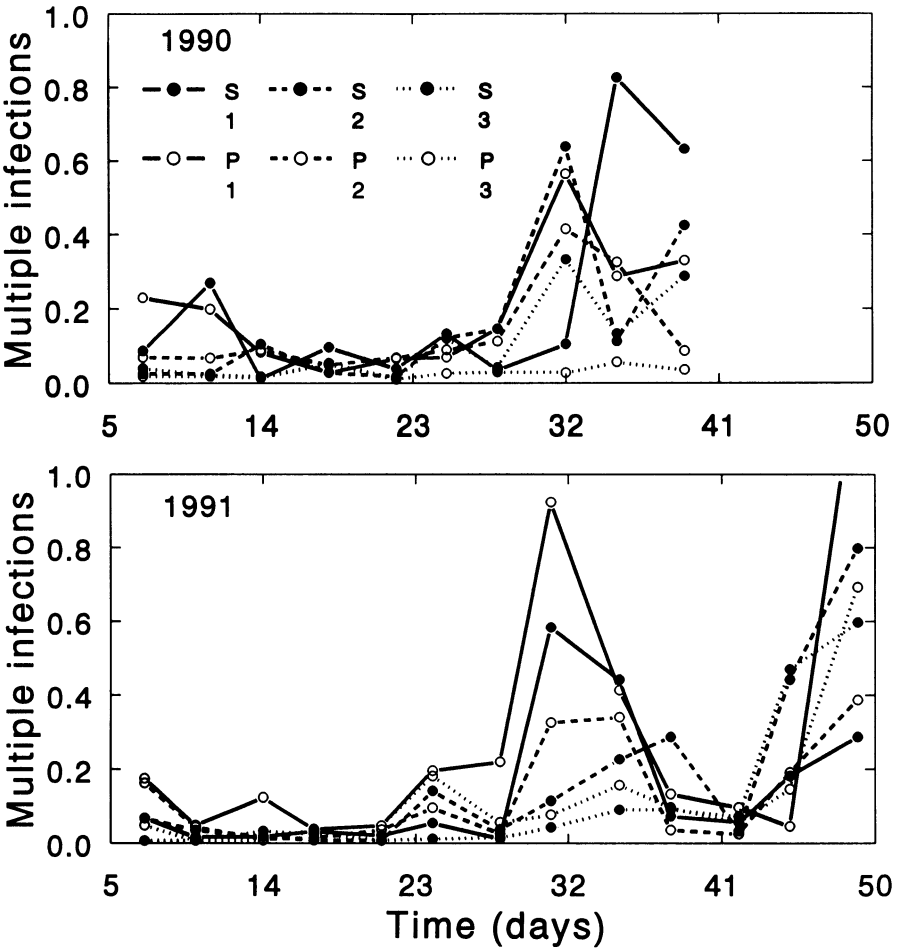

Fig. 3. Mean transformed proportion of strawberry fruit infected by *Colletotrichum acutatum* at each sampling time in plots with plastic ground cover (P ○) or bare soil (S ●). Three lines for each ground cover represent the three sampling locations (see Fig. 1), i.e., ordered distances from the original inoculum source. The multiple infection transformation (8) was used. At the final sampling for 1991, S-1 equaled 1.3.

Table 2. Best-fitting regression equations for describing the relationship between the multiple infection transformation of incidence of anthracnose fruit rot, caused by *Colletotrichum acutatum*, ($y' = \ln\{1/[1 - y]\}$), and variables representing ambient weather data, functions of weather data, interaction of these variables, and ordered distance from source,[a] together with the coefficient of determination (R^2), R^2 adjusted for degrees of freedom (R_a^2), standard deviation about the line (SD), and degrees of freedom (df)

Year	Equation[b]	R^2	R_a^2	SD	df
1990	$y' = 0.09\ (0.054)^c + 0.019\ (0.0047)v\ t'r$	0.49	...[d]	0.17	16
	$y' = 0.54\ (0.024) + 0.028\ (0.0044)v\ t'r - 0.0077\ (0.0019)v\ t'r\ s$ $= 0.54\ (0.024) + [0.028\ (0.0044) - 0.0077\ (0.0019)s]v\ t'r$	0.51	0.49	0.13	51
1991	$y' = 0.08\ (0.053) + 0.005\ (0.0010)v\ t'r$	0.48	...	0.22	24
	$y' = 0.05\ (0.020) + 0.006\ (0.0008)v\ t'r - 0.0010\ (0.0004)v\ t'r\ s$ $= 0.05\ (0.020) + [0.006\ (0.0008) - 0.0010\ (0.0004)s]v\ t'r$	0.61	0.60	0.15	75

[a] v = Index of predicted disease incidence based on wetness duration (starting with a rain episode) and average temperature during the wetness period (see Wilson et al [24]), t' = days from the introduction of infected source fruit minus 6, r = amount of rain (cm), and s = ordered distance from the inoculum source (see Fig. 1); v was calculated for the longest wetness event for days 6–11 before each disease assessment, and r was the maximum rain event for days 6–11 before each disease assessment.

[b] Equations based on data from the plastic ground cover and soil plots. First equation for each year is for disease incidence at the closest distance to the source ($s = 1$).

[c] Standard deviation of estimated regression parameter is in parentheses.

[d] Not appropriate for a single independent variable.

was covered with straw, further showing the short-range nature of splash dispersal and the effect of this surface on disease spread.

The removal of all ripe fruit throughout the growing seasons mimicked a typical harvesting practice. This technique also resulted in a mostly monocyclic (or simple interest disease) type of epidemic outside the row ends, in which most of the newly infected fruit resulted from inoculum at the row ends or from previously dispersed spores on the ground or on other plant parts. By removing fruit, it was then possible to directly relate new infected fruit to weather variables, at least for the plots with high enough disease incidence to allow comparisons (i.e., plastic ground cover and bare soil plots). About one-half of the variation in (transformed) disease incidence near the original inoculum source was attributable to earlier environmental conditions and time during the season. Our approach is similar to that taken by Fitzell et al (7) for infection of mango by *C. gloeosporioides*, where a regression equation (previously developed for fruit infection in relation to temperature and wetness duration in laboratory studies) was used to quantify conditions in the field for infection. The calculated index for infection in our study, however, was insufficient for predicting disease incidence of newly ripe fruit. The index had to be multiplied by the amount of rain and time during the season to result in an acceptable predictor for (transformed) disease incidence. Postbloom fruit drop of citrus, caused by *C. gloeosporioides*, also is associated with the combination of high rainfall and prolonged leaf wetness (a component of *i*) (3). The rainfall term in the derived regression model (*r*) reflects the importance of rain for dispersal; the time term (*t'*) reflects the buildup of inoculum throughout the season, so that less rain and lower *v* values are sufficient for the same predicted disease incidence as the season progresses. Although an acceptable model was obtained with this single three-way interaction variable, based on the criteria for regression model evaluation (2), only about one-half of the variability could be explained. Variation in fruit susceptibility over time, exact position of susceptible fruit, inoculum production, and latent period all would account for the unexplained variability.

The spread of anthracnose by the dispersal of conidia in the experimental plots with plastic ground cover and bare soil was further supported by the regression results. When data from all locations were analyzed, ordered distance from the original spore source was included in the derived regression models as an interaction term with *v*, *r*, and *t'*. The decline in (transformed) disease incidence with distance from the original source, therefore, depended on environmental conditions (i.e., *s* was multiplied by *vrt'*). Viewed another way, the change in (transformed) disease incidence with changes in weather variables depended on distance. Changes were large near the spore source and much smaller at the greatest distance, likely reflecting the number of spores at the different distances. The final model is an empirical generalization of Jeger's simple-interest model 1 for disease spread (10). Use of models such as those in Table 2 should help identify periods when substantial disease increase will occur.

In the southern United States and California, use of the double-hill plastic mulch system is routine for strawberry production (1,9). In the northern United States, matted rows with straw mulch is the normal practice. In some states (e.g., North Carolina), production is switching from straw to plastic. Notably, the highest incidence of anthracnose is occurring where plastic is used (R. D. Milholland, *personal communication*). Because of previous observations and tests with *P. cactorum* (14), we have recommended that growers use a deep layer of straw mulch to control leather rot of strawberry (15). Based on the results from rain simulation (28,29) and field experiments (Fig. 2), we would now make the same recommendation for control of anthracnose.

ACKNOWLEDGMENT

Salaries and research support provided by state and federal funds appropriated to the Ohio Agricultural Research and Development Center, Ohio State University. Manuscript No. 103-93.

LITERATURE CITED

1. Bringhurst, R. S. 1991. The future of the strawberry industry in North America. Pages 19-24 in: The Strawberry Into the 21st Century. A. Dale and J. J. Luby, eds. Timber Press, Portland, OR.
2. Campbell, C. L., and Madden, L. V. 1990. Introduction to Plant Disease Epidemiology. John Wiley & Sons, New York.
3. Denham, T. G., and Walker, J. M. 1981. Some epidemiological aspects of postbloom fruit drop disease (*Colletotrichum gloeosporioides*) in citrus. Ann. Appl. Biol. 98:65-77.
4. Dodd, J. C., Estrada, A. B., Matcham, J., Jeffries, P., and Jeger, M. J. 1991. The effect of climatic factors on *Colletotrichum gloeosporioides*, casual agent of mango anthracnose, in the Philippines. Plant Pathol. 40:568-575.
5. Eastburn, D. M., and Gubler, W. D. 1990. Strawberry anthracnose: Detection and survival of *Colletotrichum acutatum* in soil. Plant Dis. 74:161-163.
6. Fitt, B. D. L., McCartney, H. A., and Walklate, P. J. 1989. The role of rain in dispersal of pathogen inoculum. Annu. Rev. Phytopathol. 27:241-270.
7. 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.
8. Gregory, P. H. 1973. The Microbiology of the Atmosphere. 2nd ed. Leonard Hill, London.
9. Howard, C. M., Maas, J. L., Chandler, C. K., and Albregts, E. E. 1992. Anthracnose of strawberry caused by the *Colletotrichum* complex in Florida. Plant Dis. 76:976-981.
10. Jeger, M. J. 1983. Analysing epidemics in time and space. Plant Pathol. 32:5-11.
11. Macdonald, O. C., and McCartney, H. A. 1987. Calculation of splash droplet trajectories. Agric. For. Meteorol. 39:95-110.
12. Madden, L. V. 1992. Rainfall and the dispersal of fungal spores. Pages 39-79 in: Advances in Plant Pathology. Vol. 8. J. H. Andrews and I. Tommerup, eds. Academic Press, London.
13. Madden, L. V. 1993. Aggregation of *Colletotrichum acutatum* in response to simulated rain episodes. J. Phytopathol. 138:145-156.
14. Madden, L. V., and Ellis, M. A. 1990. Effect of ground cover on splash dispersal of *Phytophthora cactorum* from strawberry fruits. J. Phytopathol. 129:170-174.
15. Madden, L. V., Ellis, M. A., Grove, G. G., Reynolds, K. M., and Wilson, L. L. 1991. Epidemiology and control of leather rot of strawberries. Plant Dis. 75:439-446.
16. Madden, L. V., Wilson, L. L., Yang, X., and Ellis, M. A. 1992. Splash dispersal of *Colletotrichum acutatum* and *Phytophthora cactorum* by short-duration simulated rains. Plant Pathol. 41:427-436.
17. Milliken, G. A., and Johnson, D. E. 1984. Analysis of Messy Data. Vol. 1. Lifetime Learning Publishers, Belmont, CA.
18. MINITAB. 1989. MINITAB Reference Manual, Release 7. Minitab Inc., State College, PA.
19. Peak, C. M., Fitzell, R. D., Hannah, R. S., and Batten, D. J. 1986. Development of a microprocessor-based data recording system for predicting plant disease based on studies on mango anthracnose. Comput. Electron. Agric. 1:251-262.
20. Reynolds, K. M., Madden, L. V., and Ellis, M. A. 1988. Effect of weather variables on strawberry leather rot epidemics. Phytopathology 78:822-827.
21. Smith, B. J., and Black, L. L. 1990. Morphological, cultural, and pathogenic variation among *Colletotrichum* species isolated from strawberry. Plant Dis. 74:69-76.
22. Smith, B. J., and Spiers, J. M. 1986. Influence of mulch and irrigation types on strawberry anthracnose-crown rot. (Abstr.). HortScience 21:946.
23. Walklate, P. J., McCartney, H. A., and Fitt, B. D. L. 1989. Vertical dispersal of plant pathogens by splashing. Part II. Experimental study of the relationship between raindrop size and the maximum splash height. Plant Pathol. 38:64-70.
24. Wilson, L. L., Madden, L. V., and Ellis, M. A. 1990. Influence of temperature and wetness duration on infection of immature and mature strawberry fruit by *Colletotrichum acutatum*. Phytopathology 80:111-116.
25. Wilson, L. L., Madden, L. V., and Ellis, M. A. 1992. Overwinter survival of *Colletotrichum acutatum* in infected strawberry fruit in Ohio. Plant Dis. 76:948-950.
26. Yang, X., Madden, L. V., Reichard, D. L., Fox, R. D., and Ellis, M. A. 1991. Motion analysis of drop impaction on a strawberry surface. Agric. For. Meteorol. 56:67-92.
27. Yang, X., Madden, L. V., Reichard, D. L., Wilson, L. L., and Ellis, M. A. 1992. Splash dispersal of *Colletotrichum acutatum* and *Phytophthora cactorum* from strawberry fruit by single drop impactions. Phytopathology 82:332-340.
28. Yang, X., Madden, L. V., Wilson, L. L., and Ellis, M. A. 1990. Effects of surface topography and rain intensity on splash dispersal of *Colletotrichum acutatum*. Phytopathology 80:1115-1120.
29. Yang, X., Wilson, L. L., Madden, L. V., and Ellis, M. A. 1990. Rain splash dispersal of *Colletotrichum acutatum* from infected strawberry fruit. Phytopathology 80:590-595.