

Influence of Temperature and Wetness Duration on Infection of Immature and Mature Strawberry Fruit by *Colletotrichum acutatum*

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ABSTRACT

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Immature and mature strawberry fruit were inoculated with a conidial suspension (2.5×10^4 conidia/ml) of *Colletotrichum acutatum* and incubated under various wetness (free water) durations ranging from 0.5 to 51 hr at constant temperatures of 4, 6, 10, 15, 20, 25, 30, and 35 C. After drying, plants were moved to a greenhouse where incidence of fruit infection was recorded daily for 8 days. No infection occurred at 4 or 35 C on immature fruit or at 4 C on mature fruit. Generally, fruit disease incidence increased with increased wetness durations, but on mature fruit at 35 C, disease incidence decreased over time from a maximum of 39%.

Additional keywords: *Fragaria* × *ananassa*, quantitative epidemiology.

Optimum temperature for infection on both immature and mature fruit was between 25 and 30 C, with greater than 80% disease incidence after 13 hr of wetness. A regression model using the logit of disease incidence as the dependent variable accurately described infection level as a function of wetness duration (W) and temperature (T). Terms in the model were W , WT , WT^2 , and WT^3 , and all estimated parameters were significant. Coefficients of determination for combined data from three repetitions of the experiment were 0.71 and 0.83 for immature and mature fruit, respectively.

Anthracnose of strawberry (*Fragaria* × *ananassa* Duch.), caused by *Colletotrichum acutatum* Simmonds, was first reported by Simmonds in 1965 (23). The disease was first found in Ohio in 1985 (7). In addition to causing a fruit rot (3,7,9,20,23,24,26–28), *C. acutatum* also can infect the crown (26), leaves, stolens, and petioles of strawberry (3,27). The disease has become a major problem in strawberry production regions in Florida and California (15). Yield losses of greater than 50% have been reported (20,27).

Little has been reported on the epidemiology of strawberry anthracnose caused by *C. acutatum* (23,27,28). Epidemiological studies have been published on *C. gloeosporioides* (1,6,8,10–13, 16–18,31)(morphologically similar to *C. acutatum*), *C. gramini-cola* (4,25), *C. lindemuthianum* (29), and *Glomerella cingulata* (12), but not on strawberry. In 1957, Sturgess (28) studied temperature and wetness durations in the field in relation to the

incidence of anthracnose of strawberry. He indicated that sufficient periods of free moisture on the fruit surface were required for infection and disease increase. Sturgess (28) further suggested that neither favorable temperatures nor rainfall alone was sufficient for the development of fruit rot epidemics. Other than these observations, little information is available on the effect of environment on this disease of strawberry.

Objectives of this study were to determine effects of temperature on mycelial growth and effects of temperature and wetness duration on infection of immature and mature strawberry fruits. Regression analysis was used to quantify controlled-environment results on fruit disease incidence and predict infection level under field conditions.

MATERIALS AND METHODS

Culture maintenance and inoculum production. The isolate of *C. acutatum* used was obtained from a naturally infected straw-

berry fruit collected near Mt. Vernon, OH, in 1985. To maintain pathogenicity, fruit were inoculated and the fungus was reisolated every 2 wk. Conidia were removed from infected fruit and streaked on potato-dextrose agar (PDA) and incubated in the dark at 25 C for 2-4 days (23). Inoculum was prepared by transferring conidia from culture plates and suspending them in deionized water. Concentration was adjusted with the aid of a hemacytometer (30). All inoculations were made with a conidial suspension of 2.5×10^4 conidia/ml. Stock cultures were stored at 5 C on PDA.

Effect of temperature on mycelial growth in vitro. To determine the effect of temperature on mycelia growth in vitro, 6-mm-diameter disks were cut with a cork borer from the margin of actively growing cultures of *C. acutatum* on PDA (23). Disks were placed in the center of petri dishes (100 × 15 mm) on PDA and then incubated in the dark at constant temperatures of 5, 10, 15, 20, 25, 30, and 35 C. Colony diameter was measured at the widest point daily for 7 days. Five plates were used at each temperature, and the experiment was repeated once.

Plant production and maintenance. Strawberry plants, cultivar Midway, were obtained from Brittingham Nursery, Salisbury, MD, and Lewis Strawberry Nursery, Inc., Rocky Point, NC. They were stored at 0-2 C before planting. Plants were grown in the greenhouse in 15-cm-diameter pots in 1:2:2 (v:v:v) sand:peat:steam-disinfested loam. Plants were fertilized every 2 wk with Peter's 20-20-20 (N-P-K) fertilizer at 660 ppm (2). Plants were watered only with deionized water. Fluorescent lights were used to supplement sunlight and maintain 14 hr of light. Greenhouse temperature ranged from 15 to 27 C. Plants flowered approximately 3 wk after transplanting, and immature fruit were ready for inoculation studies at approximately 5 wk. Fruit matured about 1 wk later.

Growth chamber and greenhouse studies. Attached fruit were inoculated (2.5×10^4 conidia/ml) by spraying with an atomizer to runoff (2,14). Plants with inoculated fruit then were placed into a controlled-environment chamber (Rheem Manufacturing Company, Asheville, NC) maintained at a constant temperature under continuous wetness and light ($180 \mu\text{E m}^{-2} \text{s}^{-1}$) (2). Inside the chamber, plants were kept in a mist chamber (plastic-covered wood frame) containing two Hermidifier misters (Hermidifier Co., Lancaster, PA). Five plants were randomly removed from the mist chamber at postinoculation times ranging from 1 to 48 hr, depending on the temperature studied. After removal, plants were immediately placed in a similar chamber without free moisture (drying chamber) and maintained at the same temperature and light until all plant parts were dry. Additionally, attached, inoculated fruit were placed directly into the drying chamber to achieve a very short wetness duration. Temperature and surface wetness, in both chambers, were continuously monitored by thermistors (Fenwall Electronics, Ashland, MA) and printed-circuit wetness sensors (Wong Laboratories, Cincinnati, OH) (coated with white latex paint) interfaced to a microprocessor datalogger (model CR-21, Campbell Scientific, Logan, UT) for storage and later analysis (2). A wetness sensor, located in the drying chamber, was sprayed with deionized water when plants were placed in the chamber to estimate fruit drying time in this chamber. Fruit dryness was confirmed with visual observation in many cases. Drying time was added to the wetness duration in the mist chamber to determine total wetness duration. Dried plants with inoculated fruit were placed in the greenhouse and watered with deionized water through a trickle irrigation system. A thin rubber tube (1-mm-diameter opening) with a metal emitter was placed at the crown of each plant to prevent water from splashing during watering. In addition, a meshed (1-mm-diameter opening) wire screen was placed over the top of the pot and around the crown of each plant. The screen prevented fruit from contacting the soil. These techniques proved to be very effective in preventing undesired wetting of the fruit surface.

Inoculations were made at temperatures of 4, 6, 10, 15, 20, 25, 30, and 35 C and at wetness durations ranging from 0.5 to 51 hr (including drying time) at each temperature. The experimental design was a split plot, with temperature as the whole

plot and wetness duration as the subplot. Immature (beginning to lose chlorophyll and turn whitish [5]) and mature (beginning to turn red) fruit were inoculated in separate experiments. For each set of inoculations, fruit on five uninoculated plants served as controls. Uninoculated control plants remained in the mist chamber until the last (longest wetness duration) inoculated plants were removed. Then the uninoculated plants were removed. Incidence of fruit infection was recorded at 8 days from time of inoculation. The experiments were conducted three times.

Data analysis. Regression analysis was used to characterize the effect of temperature (T) and wetness duration (W) on the proportion of immature and mature strawberry fruit infected (Y). Two factors had to be taken into account in the regression model: an optimum relationship between Y and T , such that Y increases to a maximum and then decreases; and Y increases with increases in W , but Y cannot be greater than 1.0 or less than 0.0, regardless of the value of W (2,14). The logistic and other models were evaluated. The logistic model can be written as follows:

$$\ln(Y/[1 - Y]) = f(T, W) \quad (1)$$

in which $\ln(Y/[1 - Y])$ is the logit of Y , and $f(T, W)$ is a linear function of the following terms that were tested in all combinations: W , T , WT , T^2 , T^3 , WT^2 , WT^3 . An all-possible-subsets regression was used to eliminate nonsignificant terms.

Criteria used to evaluate the models were: 1) graphical appraisal of the randomness and normality of the residuals; 2) value of coefficient of determination (R^2), that is, agreement between observed and predicted transformed values; 3) coefficient of determination adjusted for degrees of freedom (R^2_a); 4) R^2 , goodness of fit between back-transformed values and observed Y ; 5) significance of the estimated parameters; and 6) standard deviation around the regression line (s) (2,14).

Field studies. To validate results from studies with controlled-environment chambers, field studies were performed during the 1987 and 1988 growing seasons at two sites near Wooster, OH. Cultivar Earliglow was grown at one site and cultivar Tristar was grown at the other. Attached immature and mature fruit were inoculated by spraying plants with a conidial suspension of *C. acutatum*, as previously described. Uninoculated control fruit were sprayed with deionized water until runoff. Inoculations were made during natural dew periods and during overhead irrigation. The irrigation system consisted of three garden sprinklers mounted at a height just above the plant canopy. Wetness and temperature were continuously monitored with electronic sensors placed in the plant canopy within 30 cm of the inoculated fruit and attached to a CR-21 datalogger, as previously described.

During 1987, 17 and 13 separate inoculations of immature and mature fruit, respectively, were done, and in 1988, 15 and 20 inoculations, respectively, were done. After drying, approximately

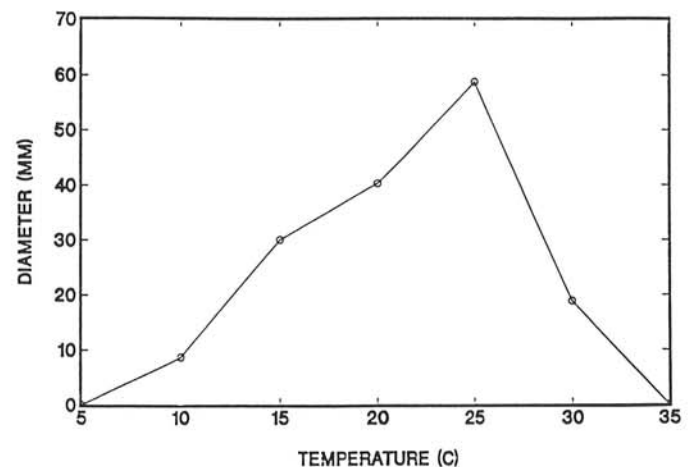


Fig. 1. Effect of temperature on mycelial growth of *Colletotrichum acutatum* on potato-dextrose agar. Results represent the mean of two replications. Results were virtually identical for each repetition.

20 fruit were randomly detached from inoculated plants at intervals ranging from 1 to 24 hr postinoculation. Uninoculated controls remained attached until the last (longest wetness duration) inoculated fruit were detached. Inoculated and uninoculated fruit from each wetness duration were placed on metal screens with their stems placed through the mesh (6-mm opening) of the screen. Screens with fruit then were placed inside a 5-L plastic container (without lid) containing 700 ml of deionized water. Stem tips of detached fruit were immersed in the water to reduce desiccation. Visual observations confirmed that fruit surfaces remained dry. The containers were placed in the dark in an incubator maintained at 25 C. Fruit infection incidence was recorded at 8 days from the time of inoculation.

RESULTS

No growth of *C. acutatum* in vitro was observed at 5 or 35 C (Fig. 1). Mycelial growth increased with increased temperature from 10 to 25 C, then declined sharply at 30 C.

Infection of immature fruit. For immature fruit, no observable infected fruit occurred at 4 or 35 C. At all other temperatures studied, generally there was an increase in the level of disease incidence with increased wetness duration (Fig. 2). Mean percentage of infected fruit was less than 10% for all temperatures with less than 7 hr wetness duration. At 6 C, incidence was 6% after 40 hr wetness duration and increased to only 13% after 50 hr. At 10 C, after 34 hr wetness duration, incidence reached 22%. At 25 and 30 C, less than 3% infection was observed at

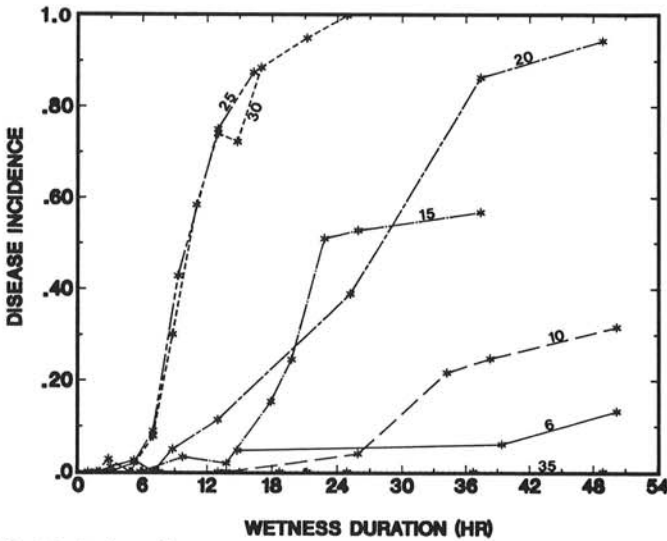


Fig. 2. Infection of immature strawberry fruit by *Colletotrichum acutatum* for wetness durations between 0.5 and 51 hr at temperatures between 6 and 35 C (labeled on the lines). Results represent the mean from three repetitions of the experiment.

TABLE 1. Estimated parameters from equation 2 for temperature (T) and wetness duration (W) effects on infection of immature strawberry fruit by *Colletotrichum acutatum*, including coefficients of determination (R^2), R^2 adjusted for degrees of freedom (R^2_a), coefficient of determination for the back-transformed infection levels (R^{*2}), and the standard error about the regression line (s)

	Estimated parameters ^y					R^2	R^2_a	R^{*2}	s
	b_0	b_1	b_2	b_3	b_4				
Replication 1	-3.83 ^z (0.32)	0.32 (0.10)	-0.069 (0.020)	0.0052 (0.0012)	-0.98×10^{-4} (0.21×10^{-4})	0.642	0.596	0.555	0.990
Replication 2	-4.12 (0.27)	0.22 (0.08)	-0.050 (0.016)	0.0043 (0.0009)	-0.86×10^{-4} (0.16×10^{-4})	0.755	0.727	0.722	0.839
Replication 3	-3.98 (0.26)	0.33 (0.15)	-0.065 (0.024)	0.0046 (0.0011)	-0.86×10^{-4} (0.12×10^{-4})	0.774	0.740	0.790	0.697
Combined experiments	-3.70 (0.18)	0.33 (0.06)	-0.069 (0.011)	0.0050 (0.0006)	-0.93×10^{-4} (0.99×10^{-5})	0.670	0.658	0.711	1.110

^yEstimated parameters for equation 2 corresponding to the intercept (b_0), W , WT , WT^2 , and WT^3 . Numbers in parentheses correspond to the parameters' standard deviations.

^z b_0 is the value of $\ln(Y/[1 - Y])$ when $T = 0$ and $W = 0$.

less than 5 hr. After 17 hr, incidence increased sharply to 88%, and by 25 hr reached 100%. No infection was observed on any uninoculated control fruit.

Infection of mature fruit. As with immature fruit, generally there was an increase in infected mature fruit with increased wetness duration at most temperatures studied (Fig. 3). No infected fruit were observed at 4 C. Unlike immature fruit, however, infected mature fruit were observed at 35 C. At short wetness durations (<2 hr), relatively high disease incidence (about 39%) occurred at 35 C; incidence then rapidly decreased with increasing wetness duration (Fig. 3). By 48 hr, no infected fruit were observed. At 6 C, incidence was only 11% at 38 hr but increased to 25% after 51 hr. At 20 C, incidence increased from 4% at 1 hr to 85% after 25 hr. At 25 and 30 C, incidence increased sharply to 97 and 89% after 13 hr, respectively. No infection was observed on any uninoculated control fruit.

Data analysis. A regression model using the logit of disease incidence adequately described the effects of temperature (T) and wetness duration (W) on disease incidence for each replication and for data combined from all replications (Tables 1 and 2). The same terms in the model, based on stepwise regression, were significant ($P < 0.05$) for both immature and mature fruit. However, data at 35 C for mature fruit were not analyzed with the other data because of the decline in incidence with increasing W . The chosen logistic regression equation can be written as follows:

$$\ln(Y/[1 - Y]) = b_0 + b_1W + b_2WT + b_3WT^2 + b_4WT^3 \quad (2)$$

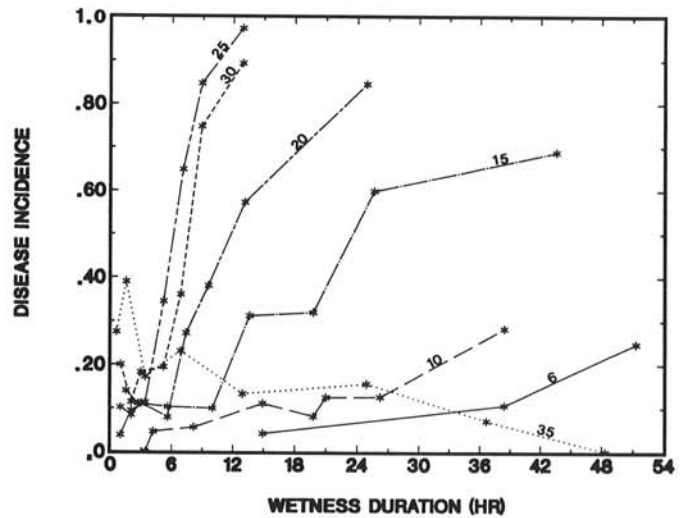


Fig. 3. Infection of mature strawberry fruit by *Colletotrichum acutatum* for wetness durations between 0.5 and 51 hr at temperatures between 6 and 35 C (labeled on the lines). Results represent the mean from three repetitions of the experiment.

in which the b_s are estimates of unknown parameters, all of which were significant ($P < 0.05$) (Tables 1 and 2). An F -test indicated that there was no difference in regression results among the three replications for immature or mature fruit data; therefore, the data for the three experiments could be combined.

Equation 2 indicated that T , W , and their interaction had a significant effect on disease incidence. There was a significant linear, squared, and cubic relationship between logits and T , a linear relationship between logits and W , and an interaction between W and all T terms. The interactions between W and the T terms indicate that change in logits (or Y) with change in T was not consistent for all W s. Although some of the b_s were negative, this does not indicate a decline in logits with increase in W . To see this, equation 2 can be rewritten as follows:

$$\ln(Y/[1 - Y]) = b_0 + W(b_1 + b_2T + b_3T^2 + b_4T^3) \quad (3)$$

The term in parentheses is the slope for the change in logit with W . The slope was positive for the temperatures analyzed, and, therefore, the equation demonstrated that there was a positive relationship between logits and W at the temperatures considered. There was less variability with the mature fruit data as indicated by the lower standard deviation (s) and higher coefficients of determination. The R^2 , R^2_a , and R^{*2} values for the combined data were 0.67, 0.66, 0.71 for immature fruit and 0.75, 0.74, 0.83 for mature fruit, respectively.

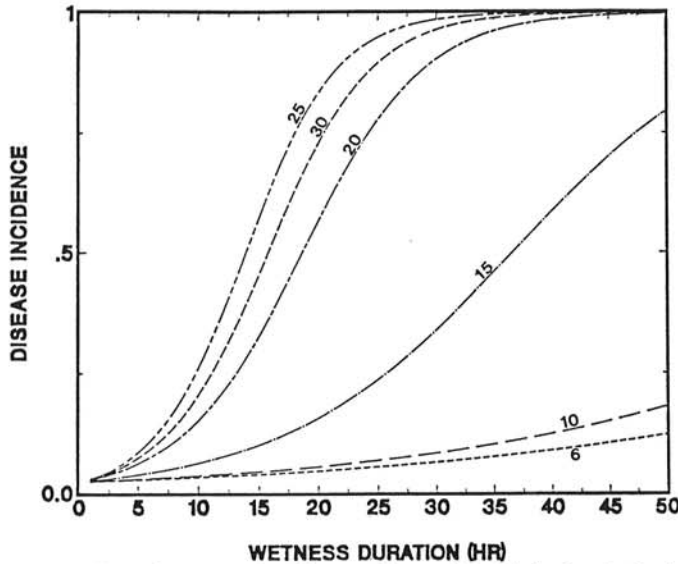


Fig. 4. Effect of wetness duration on the predicted infection level of immature strawberry fruit by *Colletotrichum acutatum* at temperatures of 6, 10, 15, 20, 25, and 30 C (labeled on the lines). Curves were produced using equation 2 with the estimated parameters shown in Table 1 for the combined data.

Estimated model parameters for the combined data (Tables 1 and 2, bottom line) were used to calculate predicted values of $\ln(Y/[1 - Y])$ and then Y for temperatures between 6 and 30 C for immature fruit and 10 and 30 C for mature fruit over wetness durations from 0.5 to 50 hr. Figures 4 and 5 show the consistent increase in Y with increase in W for immature and mature fruit, respectively. The greatest errors were for immature fruit at 10 and 20 C (Fig. 4). The temperature at which maximum Y occurred was about 27 C and did not depend on wetness duration.

Field studies. The logistic equation with the coefficients from the controlled-environment study was used to predict disease incidence from field inoculations. Because of variable temperature, average temperature during the wetness duration was used in the equation. For the total of 65 inoculations, wetness durations ranged from 0 to 25 hr, and average temperatures ranged from 8 to 30 C. For short wetness durations (≤ 11 hr), the hourly temperature ranges during a given inoculation were between 0.4 and 6.0 C; for long durations (> 11 hr), temperature ranges were between 5 and 14 C. Predicted disease incidence values were regressed on observed incidence from equation 2 to validate controlled-environment results. An unbiased result would produce an intercept of 0 and a slope of 1. Figure 6 demonstrates the predicted proportion of field-inoculated immature strawberry fruit versus the observed proportion of immature infected fruit. The estimated equation was as follows:

$$\text{Predicted} = 0.04 + 1.03(\text{observed}) \quad (4)$$

The intercept of 0.04 was not significantly different from 0 ($P > 0.05$), and the slope of 1.03 was not significantly different from

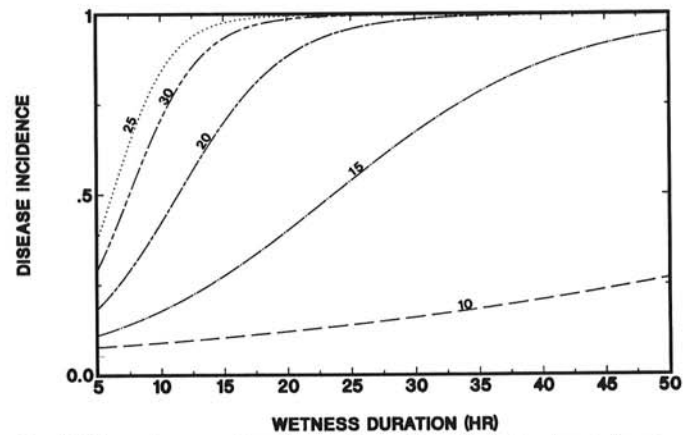


Fig. 5. Effect of wetness duration on the predicted infection level of mature strawberry fruit by *Colletotrichum acutatum* at temperatures of 10, 15, 20, 25, and 30 C (labeled on the lines). Curves were produced using equation 2 with the estimated parameters shown in Table 2 for the combined data.

TABLE 2. Estimated parameters from equation 2 for temperature (T) and wetness duration (W) effects on infection of mature strawberry fruit by *Colletotrichum acutatum*, including coefficient of determination (R^2), R^2 adjusted for degrees of freedom (R^2_a), coefficient of determination for the back-transformed infection levels (R^{*2}), and the standard error about the regression line (s)

	Estimated parameters ^y					R^2	R^2_a	R^{*2}	s
	b_0	b_1	b_2	b_3	b_4				
Replication 1	-3.23 ^z (0.28)	0.29 (0.12)	-0.069 (0.028)	0.0056 (0.0020)	-1.0×10^{-4} (0.41×10^{-4})	0.803	0.770	0.819	0.729
Replication 2	-3.02 (0.18)	0.24 (0.06)	-0.063 (0.016)	0.0053 (0.0011)	-0.98×10^{-4} (0.22×10^{-4})	0.894	0.879	0.925	0.545
Replication 3	-2.21 (0.15)	0.08 (0.02)	-0.019 (0.036)	0.0016 (0.0021)	-0.21×10^{-4} (0.37×10^{-4})	0.887	0.863	0.935	0.863
Combined experiments	-2.66 (0.11)	0.17 (0.05)	-0.042 (0.013)	0.0034 (0.0009)	-0.57×10^{-4} (0.17×10^{-5})	0.748	0.739	0.838	0.739

^y Estimated parameters for equation 2 corresponding to the intercept (b_0), W , WT , WT^2 , WT^3 . Numbers in parentheses correspond to the parameters' standard deviations.

^z b_0 is the value of $\ln(Y/[1 - Y])$ when $T = 0$ and $W = 0$.

1.0. The R^2 value was 0.624 and the equation was significant at $P < 0.01$. Figure 7 demonstrates the predicted proportion of field-inoculated mature strawberry fruit versus the observed proportion of mature infected fruit. The estimated equation was as follows:

$$\text{Predicted} = 0.06 + 1.08(\text{observed}) \quad (5)$$

The slope of 1.08 was not significantly different from 1.0 ($P > 0.05$), but the intercept of 0.06 was significantly different from 0 ($P < 0.05$), indicating a slight overprediction. The R^2 value was 0.895, and the equation was significant at $P < 0.01$. For immature fruit, more variability was observed than in similar studies on mature fruit. This was reflected by the considerably higher R^2 for mature fruit than immature fruit. Field site or cultivar had no effect on the relationship between observed and predicted disease incidence.

DISCUSSION

Results of this study indicate that temperature and wetness durations have a direct effect on the infection of both mature and immature strawberry fruit by *C. acutatum*. Disease incidence for mature fruit generally was higher than that of immature fruit at the same temperature and wetness duration, indicating that mature fruit appear more susceptible. Except at 4 and 35 C, disease incidence generally increased with increased wetness duration. Additionally, at each wetness duration, disease incidence generally increased with increased temperature from 6 to 25 C (near optimum). This agreed with the mycelial growth results. Similar temperature and wetness relationships have been reported for other fungal pathogens of strawberry fruit that are common in Ohio (2,14,21,22).

This study showed that the logistic model (equation 2) was adequate for describing disease incidence at different combinations of wetness duration and temperature for immature fruit from 6 to 35 C and for mature fruit from 6 to 30 C. Coefficients of determination (R^2) for the three replications of 0.80–0.89 for mature fruit and 0.64–0.77 for immature fruit indicate that a moderately high proportion of the variability of logits was accounted for by temperature and wetness duration. Coefficients of determination adjusted for degrees of freedom (R^2_a) were close to R^2 values, indicating that the estimated parameters were significant. Low values of R^2_a relative to R^2 would have indicated redundancy and suggest that some terms (for example, W) of the model may not be essential (19). Finally, except for replication 1 with immature fruit, the agreement between observed and predicted disease incidence values (R^{*2}) (that is, back-transformed

logits) was also fairly high. The model was of the same form as that used by Grove et al (14) and Bulger et al (2) for describing the effect of temperature and wetness duration on infection of strawberry fruit and flowers by *Phytophthora cactorum* (Leb. & Cohn) Schroet and *Botrytis cinerea* Pers., respectively. The optimum temperature for *C. acutatum*, however, was found to be higher than that for those pathogens.

In agreement with the mycelial growth data, there was no fruit infection below 6 C or, for immature fruit, at 35 C. At 35 C, infection level of mature fruit was about 39% after 1.5 hr and then declined to 0% after 48 hr. We propose possible explanations for this phenomenon. First, mature fruit appeared to be physically damaged by the 35 C temperature. This could result in relatively high disease incidence at short durations. The fruit then appeared to rapidly decompose and lose color at long wetness durations. These changes were not observed on immature fruit at 35 C. Mature fruit were physically, and probably physiologically, changed by the 35 C temperature and long wetness durations, possibly producing an adverse effect on the infection process. A second, related, possibility is that the high temperature had a cumulative (dosage) effect over time on the pathogen. The fruit surface likely had an initial temperature less than 35 C, resulting in some spore germination and the initiation of the infection process. As the fruit temperature increased to 35 C, mortality then would have occurred over time. The more susceptible mature fruit, when combined with the effect of prolonged periods of high temperature on fungal physiology, probably resulted in the observed infections at this high temperature.

Field inoculations resulted in disease incidence values in general agreement with those predicted from the controlled-environment studies. Although there was considerable variability with immature fruit (Fig. 6), the regression results for predicted incidence in relation to observed incidence showed that model predictions were unbiased; that is, slope and intercept were not significantly different from 1 and 0, respectively. Although there was a closer agreement between observed and predicted incidence for mature than immature fruit (Fig. 7 versus Fig. 6), the regression slope was not different from 1, but the intercept was significantly greater than 0 for mature fruit. For all inoculations when no infected mature fruit were observed, the controlled-environment model predicted 6% disease incidence. Because the slope was not different from 1, this 6% average bias held for all disease levels. There was only one observation that was in serious error: Inoculation resulted in about 10% incidence, but the model predicted about 85% (Fig. 7). We cannot explain this outlier, but the sensor drying time may not have corresponded with actual fruit drying for this inoculation. The overall degree of correlation between predicted and observed disease incidence in the field was judged to be

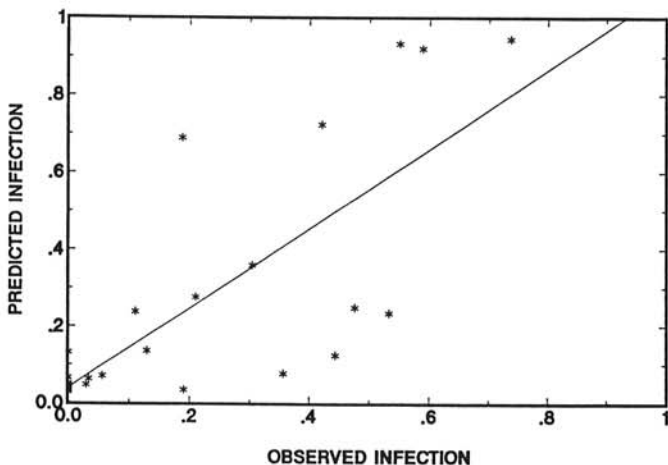


Fig. 6. Predicted proportion of infected immature fruit based on equation 2 (with the parameters listed in Table 1 for the combined tests) versus the observed proportion of field-inoculated strawberry fruit infected by *Colletotrichum acutatum* ($R^2 = 0.624$). Temperatures and wetness durations used in the equations were measured in the field.

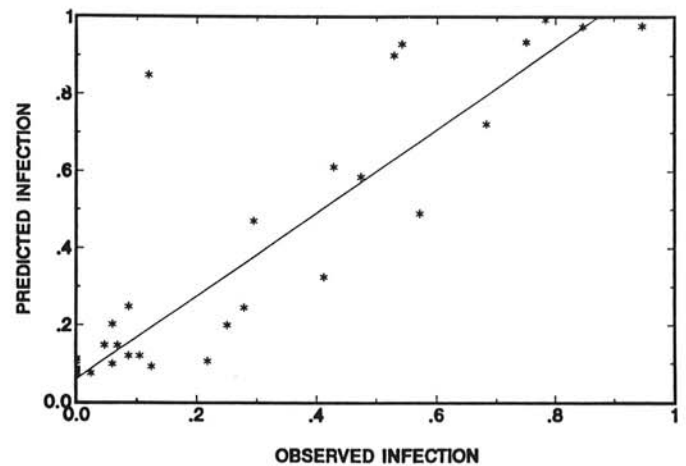


Fig. 7. Predicted proportion of infected mature fruit based on equation 2 (with the parameters listed in Table 2 for the combined tests) versus the observed proportion of field-inoculated strawberry fruit infected by *Colletotrichum acutatum* ($R^2 = 0.895$). Temperatures and wetness durations used in the equations were measured in the field.

acceptable considering the variability in temperature during each wetness period, radiation, and air movement in the field, as well as the potential variation in fruit susceptibility over time.

Few studies have been conducted on the effects of temperature and wetness duration on infection and other epidemiological processes of *C. acutatum* (23,27,28). Sturgess (27,28), in qualitative studies, found the optimum temperature for fruit infection to be between 23.9 and 29.4 C, and found it to be 28 C for mycelial growth. Simmonds (23) reported that 27.3 C was the optimum temperature for mycelial growth. Sturgess (28) also demonstrated the importance of fruit wetness periods for infection to occur. Our results for fruit infection as well as mycelial growth agree with those of Sturgess, Simmonds, and other researchers working on related pathogens (8,12,31).

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