Influence of Temperature and Wetness Duration on Infection of Strawberry Flowers by *Botrytis cinerea* and Disease Incidence of Fruit Originating from Infected Flowers

M. A. Bulger, M. A. Ellis, and L. V. Madden

Former graduate research associate and associate professors, Department of Plant Pathology, The Ohio State University, Ohio Agricultural Research and Development Center, Wooster 44691.

Salaries and research support provided by State and Federal funds appropriated to the Ohio Agricultural Research and Development Center and The Ohio State University. Additional funding supplied by the North American Strawberry Growers' Association. Journal Article 191-86.

We thank K. M. Reynolds and N. Lalancette for reviewing the manuscript.

Accepted for publication 27 February 1987 (submitted for electronic processing).

ABSTRACT

Bulger, M. A., Ellis, M. A., and Madden, L. V. 1987. Influence of temperature and wetness duration on infection of strawberry flowers by *Botrytis cinerea* and disease incidence of fruit originating from infected flowers. Phytopathology 77:1225-1230.

Strawberry flowers were inoculated with a conidial suspension of *Botrytis cinerea* at temperatures between 5 and 30 C and wetness durations up to 32 hr. There was an increased incidence of flower infection with increased wetness duration for all temperatures tested. Optimum temperature for flower infection was ~ 20 C, with 100% infection at 24 hr wetness. As with flowers, there was a general increase in the incidence of fruit infection resulting from flower inoculations with increased wetness duration at all temperatures. For inoculation of flowers at 20 C, the highest incidence of fruit infection (~ 60%) was recorded for 32 hr of wetness. Flower and fruit infection was greatly reduced above 25 and below 15 C for all wetness durations. Regression models were fitted to the data. A logistic model was chosen to describe the incidence of petal and stamen necrosis and incidence of fruit infections originating from infected flowers as functions of temperature and wetness duration. Coefficients of determination for the data combined from both runs of the experiment were 0.90, 0.87, and 0.92, respectively. A logarithmic model was used to describe the relationship between the incidence of petal necrosis and incidence of infected fruit from infected flowers. All models had significant coefficients and, based on an F test, the experimental runs were not significantly different.

Additional key words: *Fragaria* × *ananassa*, quantitative epidemiology.

Strawberry gray mold, caused by *Botrytis cinerea* Pers., is a major fruit rot disease of strawberry (*Fragaria* × *ananassa* Duch.) worldwide (4,12). Under favorable conditions of wetness and temperature during flowering and harvest, yield loss can exceed 50% (8).

The establishment of latent infections of floral parts is considered one of the major avenues for late-season rot of ripe fruit (6,8–11,13,14,17). Latent infection entails conidial germination and penetration of floral parts in which the mycelium remains quiescent until the fruit ripens, at which time Botrytis fruit rot may develop (4,22). Jarvis (10) found fruit rot incidence to be highly correlated with preharvest durations of relative humidity (RH) >80% and the amount of preharvest rainfall. Thus, environmental variables such as temperature and wetness duration likely play a critical role in the development of latent infections and subsequent rot of ripe strawberry fruit.

The objective of this study was to determine how wetness duration and temperature affect flower infections by *B. cinerea* and the subsequent rot of ripe fruit originating from infected flowers. Regression models were developed to quantify the relationship between disease incidence and the environmental variables.

MATERIALS AND METHODS

Plant and inoculum production and maintenance. An isolate of *B. cinerea* collected from a strawberry field in Wooster, OH, was used for infection studies. Pathogenicity was maintained by inoculating strawberry fruit on potato-dextrose agar (PDA) every 2 to 3 wk and reisolating it after the fungus had grown through the fruit and into PDA. Stock cultures were made by transferring mycelial plugs to PDA plates from the edge of actively growing cultures. Cultures were maintained at 20 C under continuous fluorescent light at 58 μEin m^{-2} s^{-1} for 14 days. Conidia for inoculations were collected by vacuuming from culture plates into a solution of Tween 40 (one drop per 1,000 ml) and deionized water. To remove debris, conidial suspensions were passed through two layers of tissue paper. Inoculum concentrations were determined by hemacytometer counts and inoculations made with suspensions adjusted to 10^5 conidia per milliliter. Open flowers were inoculated by spraying with an atomizer until runoff.

Strawberry plants (cultivar Midway) were grown in a soil mix of 1:2:2 (v:v:v) sand:peat:steam disinfested loam, which was fertilized every 2 wk with water soluble fertilizer (20-20-20, N-P-K) at 660 ppm. To minimize splashing, a semiautomatic drip irrigation system was used. Plants were watered only with deionized water.

Flowers were inoculated when they were between the white bud (sepals and petals open but not fully reflexed) and open flower stage (petals and sepals fully reflexed). Only primary and secondary flowers were tagged and inoculated.

Controlled environmental studies. Wetness periods were induced by placing inoculated plants in a controlled environment chamber (Environmental Growth Chambers, Chagrin Falls, OH) with a constant temperature between 5 and 30 C and continuous wetness and light (220 μEin m^{-2} s^{-1}). In the growth chamber, plants were kept in a clear plastic mist chamber containing a Hermidifier mister (Hermidifier Co., Lancaster, PA). Enough plants were placed in the growth chamber so that 30 flowers per wetness duration could be monitored for symptoms. Generally, five to eight plants were required to achieve 30 flowers. Plants were arranged in the chamber such that flowers were not in contact with each other. Plants were removed randomly from the mister at 5-hr intervals from 5–30 hr or at increments of 2, 4, 8, 16, and 24 hr. Plants were dried in another growth chamber (330 μEin m^{-2} s^{-1}) that was set at the same temperature as the mist chamber. Temperature and wetness were monitored continuously with thermistors (Fenwall Electronics, Ashland, MA) and a wetness sensor coated with white latex paint connected to a data logger (CR-21, Campbell Scientific, Logan, UT). On placement of plants into the dry chamber, another wetness sensor was sprayed with

deionized water so that plant drying time could be estimated. Approximately 5 hr after plants were dry, they were moved to a greenhouse maintained at 24 ± 1 C and 50–70% RH. Four days after inoculation, necrosis of flower petals, sepals, and stamens was assessed and recorded. Proportion of flowers with necrotic parts was determined for each plant and for all plants combined. To confirm the presence of *B. cinerea*, necrotic flower parts were occasionally incubated on a basal medium selective for *Botrytis* spp. (15).

After the incidence of flowers with necrotic parts was recorded, plants were maintained in the same greenhouse for 2 to 3 wk. Ripe fruit that developed from inoculated flowers were harvested. Fruit were surface disinfested by washing in distilled water with Tween-40 (10 drops per 1,000 ml), rinsing in distilled water, immersing in 1% sodium hypochloride for 1 min, and rinsing them again in distilled water. Disinfested berries were placed on 6.4-mm-mesh galvanized metal screens within disinfested plastic moisture chambers (4 L). Fruit were positioned so that they were not in contact with each other. High relative humidities (95–100%) were maintained by placing moistened tissue paper in the bottom of the chamber. Fruit were incubated at 20 C, and the number with gray mold symptoms was visually assessed after 6 or 7 days. These incubation conditions were chosen to favor symptom development if the fruit were infected. Observations on fruit infection were made corresponding to flowers inoculated at four constant temperatures, 15, 20, 25, and 30 C. Determination of gray mold incidence was attempted for additional temperatures <15 C, but high flower abortion and other problems prevented assessment of fruit infection. The constant temperature treatments were tested in a random order. The experiment was repeated twice.

Data analysis. Regression analysis was used to describe the effect of temperature (T) and wetness duration (W) during flowering on the level of infection (Y) of resulting ripe fruit and flowers (based on petal, sepal, and stamen necrosis). Desirable model properties were: an optimal relationship between Y and T, such that Y increases to a maximum and then decreases; Y increases with increasing W; and Y cannot be greater than 1.0 or less than 0.0 regardless of the value of W (5). A logistic model and a generalization of the Analytis 'Beta' model (4) were evaluated to describe the relationships listed above.

The regression models were evaluated based on the following criteria: randomness and normality of the residuals, significance of estimated parameters, coeffficient of determination (R^2), i.e., goodness of fit between observed and predicted transformed Y values, coefficient of determination adjusted for degrees of freedom (R_a^2), and R^{*2}, goodness of fit between the back-transformed predicted values and observed Y values. These criteria were applied to each experimental run and the combined data.

The generalized form of the logistic model can be written as:

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

in which $\ln(Y/(1-Y))$ is the logit of Y, and $f(T,W)$ consisted of linear combinations of some of the following terms: W, T, T^2, T^3, WT, WT^2, and WT^3. The generalized Analytis 'Beta' model (5) is of the form

$$Y = pt^m(1-t)^n\, W^q, \quad (2)$$

which can be rewritten as

$$\ln(Y) = \ln(p) + m\ln(t) + n\ln(1-t) + q\ln(W) \quad (3)$$

in which $t = (T - T\text{min})/(T\text{max} - T\text{min})$ and p, m, n, and q are unknown parameters. Tmin and Tmax were set equal to 4 and 33 C, respectively (6). An F test was used to determine whether the regression results of the two runs of the experiment were significantly different.

Several simple models were evaluated for describing the relationship between fruit infection and the incidence of flower infection. Petal, sepal, and stamen necrosis were evaluated for predicting fruit infection level.

RESULTS

In general, there was an increase in the incidence of flower infection with increases in wetness duration over all the temperatures tested (Figs. 1 and 2). Highest levels of flower disease incidence occurred around 20 C. Up to 100% of the flowers had petal necrosis by W = 24 hr (Fig. 1). Minimal petal necrosis occurred at 5 C for all the wetness durations tested. Incidence of flowers with necrotic stamens was much lower than flowers with necrotic petals. Optimum temperature for stamen necrosis was about 20 C, but even at 30 C there were an appreciable number of flowers with necrotic stamens. There was much greater variability in sepal necrosis, with a less clear relationship to T and W.

As with flowers, there was a general increase in the incidence of fruit with disease symptoms resulting from flower inoculations with increases in W (Fig. 3). At 20 C during the flower inoculation wetness period, the highest level of disease incidence of fruit for 32 hr wetness was ~ 60% (Fig. 3). At 30 C during flowering, the resultant incidence of ripe fruit infection was very low for all wetness periods tested (<10%).

The relationship between the incidence of fruit (Y) and flower (X) infection was not linear (Fig. 4). Petal necrosis was found to be a better predictor of fruit disease incidence than either stamens or sepals (data not shown). When incidence of flowers with diseased petals was low ($X < 0.05$), the level of fruit infection was greater than flower necrosis. This indicated that some flowers apparently were infected even though they did not show symptoms. At higher levels of petal necrosis, fruit disease incidence was less than flower

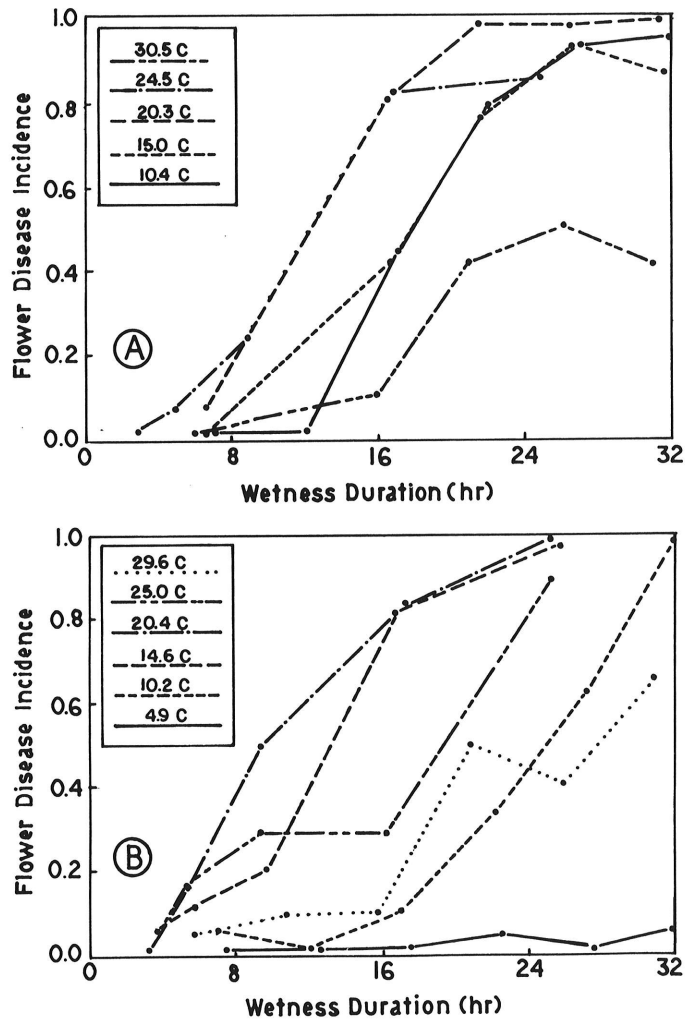

Fig. 1. Proportion of flowers with petals infected by *Botrytis cinerea* for wetness durations between 0–35 hr at temperatures between 4.9 and 30.5 C (see figure label). Results are for runs 1 **(A)** and 2 **(B)** of the experiment. Each point represents the mean of 30 flowers.

infection (Fig. 4). As petal necrosis approached 1.0, the incidence of fruit infection rapidly rose. The best regression model describing Y as a function of X was of a logarithmic form:

$$Y = b_0 + b_1 \ln(1/(1 - X)). \quad (4)$$

The coefficient of determination (R^2) equaled 0.80. The intercept (b_0) was significantly different from zero, indicating the higher observed fruit than flower infection at low flower disease incidence (Table 1). The b_1 coefficient was highly significant ($P < 0.01$). An F test indicated no significant difference in the regression results for the two experimental runs ($P > 0.10$).

Stamen and sepal necrosis were poor predictors of fruit infection. Fitting equation 4 to the data resulted in R^2 values equal to 0.43 and 0.49 for sepals and stamens, respectively. Residual plots also exhibited nonrandom patterns for these latter regressions.

A logistic model of the form:

$$\ln(Y/(1 - Y)) = b_0 + b_1 WT + b_2 W + b_3 WT^3 \quad (5)$$

was chosen to best describe incidence of fruit infection based on temperature and wetness durations during flowering. Equation 5 also was used to describe the effect of T and W on petal and stamen necrosis. The b values are estimates of unknown parameters; all estimates were significant ($P < 0.05$). There was no significant difference ($P > 0.10$) in the regression results between the two runs for petals, stamens, or fruit. The model indicated a linear and cubic relationship between T and logits. There was an interaction between W and T, and W and T^3. For fruit infection, R^2, R_a^2, and R^{*2} for the combined data were 0.79, 0.78, and 0.92, respectively (Table 2). For petal and stamen necrosis incidence, R^{*2} for combined data were 0.90 and 0.87, respectively (Tables 3 and 4). The parameter b_2 consistently was negative, but this did not

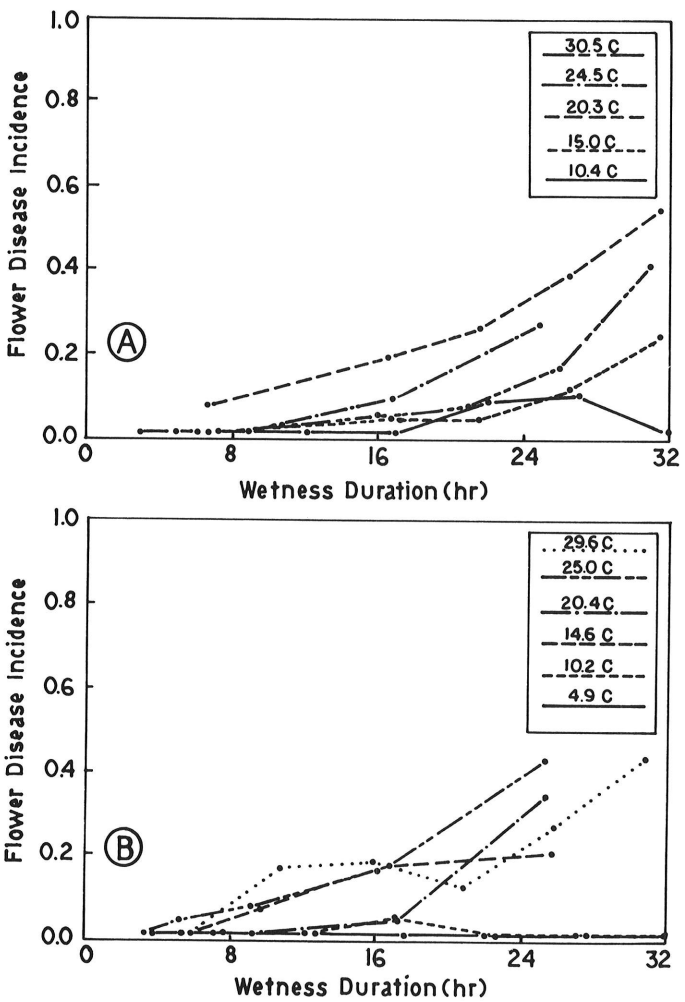

Fig. 2. Proportion of flowers with stamens infected by *Botrytis cinerea* for wetness durations between 0–35 hr at temperatures between 4.9 and 30.5 C (see figure label). Results are for runs 1 **(A)** and 2 **(B)** of the experiment. Each point represents the mean of 30 flowers.

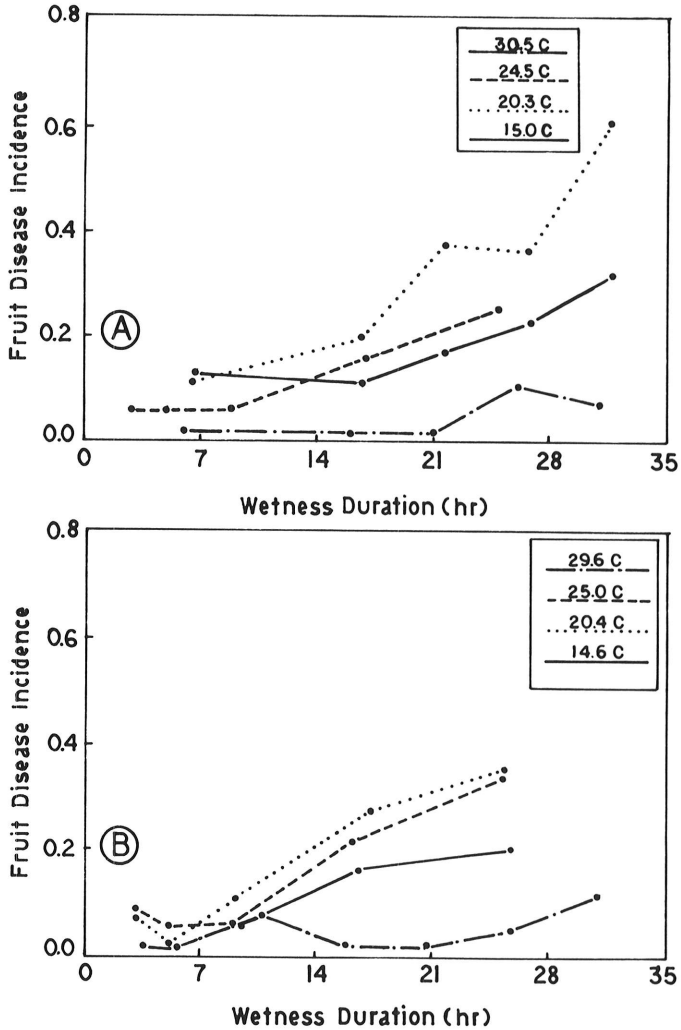

Fig. 3. Proportion of strawberry fruit infected by *Botrytis cinerea* originating from inoculated flowers for wetness durations between 0–35 hr at temperatures between 15.0 and 30.5 C (see figure label). Results are for runs 1 **(A)** and 2 **(B)** of the experiment. Each point represents the mean of 20–30 fruit.

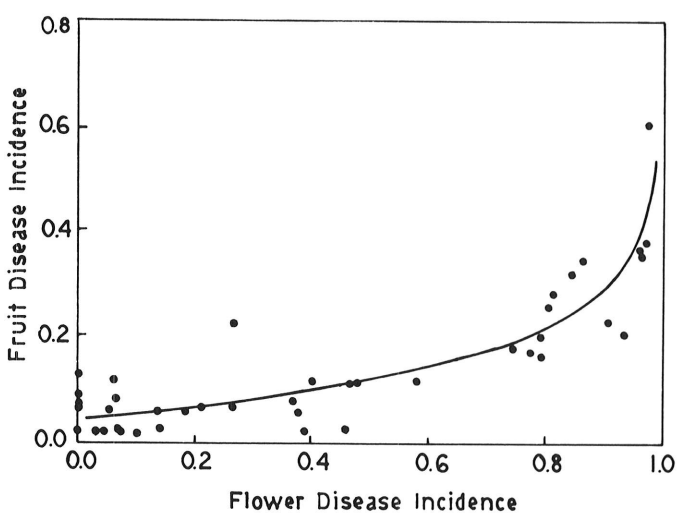

Fig. 4. Relationship between the incidence of flowers with petal necrosis and incidence of ripe strawberry fruit infected by *Botrytis cinerea*. The predicted curve was generated from equation 4 using the estimated parameters in Table 1 for the combined data.

indicate a decline in Y with increase in W. To see this, equation 5 can be rewritten as:

$$\ln(Y/(1-Y)) = b_0 + W(b_1 T + b_2 + b_3 T^3). \quad (6)$$

Logit of Y (or Y) increases with W whenever $b_1 T + b_3 T^3$ is greater than $-b_2$, and this was met by our data. The predicted levels of Y are plotted for temperatures between 10 and 30 C by using parameters for the combined data (Figs. 5–7). These figures show a monotonic increase in Y with an increase in W for petals, stamens, and fruit.

Incidence of sepal necrosis was not well predicted by T and W, although equation 5 was the most appropriate model. However, residuals were not random, and the coefficients of determination were low. For instance, R^2, R_a^2, and R^{*2} were 0.44, 0.41, and 0.52, respectively, for the combined data.

The generalized Analytis model did not fit the fruit or flower data as well as equation 5. Residuals had a strong pattern, and coefficients of determination were lower than equation 5. For example, R^2, R_a^2, and R^{*2} for the Analytis model fit to the combined petal necrosis data were 0.65, 0.64, and 0.76, respectively.

DISCUSSION

Postinoculation temperature and wetness duration at flowering were found to be significant factors influencing infection by *B. cinerea* of strawberry flowers and also ripe fruit originating from infected flowers. Equation 5 accurately predicted disease incidence of fruit and flowers based on T and W at the inoculation of flowers. Coefficients of determination (R^2) indicated that a high proportion of the variability in logits of fruit or flower disease incidence was accounted for by the independent variables in the model. The coefficients of determination adjusted for degrees of freedom (R_a^2) for the models decribing disease incidence of fruit, petals, and stamens based on T and W during flowering were very close to the R^2 values. This indicated significance of terms in the model; in contrast, a lower R_a^2 would infer a redundancy of terms in the model. The logistic model for fruit, petal, and stamen disease incidence had R^{*2} values of 0.92, 0.90, and 0.87 for the combined data, respectively. These values indicate that the high proportion of variability in the Y's was accounted for by the model.

Optimum temperature observed for petal necrosis and gray mold of ripe fruit was ~ 20 C, with highest disease incidence occurring between 15 and 25 C. Based on equation 5, the optimum T equals $(-b_1/3b_3)^{1/2}$. For fruit gray mold, the optimum was calculated as 21.1 C; for petal necrosis it was 20.4 C. At a given T and W, the incidence of stamen necrosis was always lower than petal necrosis. This difference may be because of a larger petal surface intercepting more inoculum or because the incidence of stamen necrosis was more difficult to detect visually.

The incidence of fruit infection had a logarithmic relationship (Eq. 4) with the incidence of petal necrosis. The R^2 for the logarithmic model was 0.80, for the combined data. The R_a^2 and the R^{*2} statistics were not applicable in evaluating equation 4, since there was only one independent variable and the Y values were not transformed. Except at low levels of petal necrosis (5% or less), disease incidence of fruit was less than the petals. Even at 80% petal necrosis, for instance, predicted incidence of fruit gray mold was only 22%. Environmental conditions during the period between postinoculation drying of the flowers and the

TABLE 1. Estimated parameters for equation 4 that relate the proportion of flowers with petal necrosis (X) to the incidence of ripe strawberry fruit (Y) infected by *Botrytis cinerea*, together with the coefficients of determination (R^2) and the standard error about the regression line (s)

	Estimated parameters[y]			
	b_0[z]	b_1	R^2	s
Test 1	0.0448 (0.0161)[z]	0.0951 (0.0138)	0.715	0.058
Test 2	0.0406 (0.0186)	0.1138 (0.0113)	0.850	0.059
Combined data	0.0416 (0.0121)	0.1076 (0.0085)	0.805	0.058

[y]Estimated parameters for equation 4 corresponding to Y-intercept (b_0) and slope (b_1).
[z]Numbers in parentheses are the standard deviations of the estimated parameters.

TABLE 2. Estimated parameters for a logistic model (equation 5) that describes the proportion of ripe strawberry fruit infected by *Botrytis cinerea* as a function of temperature (T) and wetness duration (W) at flower inoculation, together with the coefficients of determination (R^2), R^2 adjusted for degrees of freedom (R_a^2), coefficient of determination for the back-transformed infection levels (R^{*2}), and the standard error about the regression line (s)

	Estimated parameters[y]							
	b_0[z]	b_1	b_2	b_3	R^2	R_a^2	R^{*2}	s
Test 1	−3.698 (0.254)	0.0287 (0.0081)	−0.2615 (0.1157)	-2.1×10^{-5} (2.4×10^{-6})	0.748	0.704	0.867	0.607
Test 2	−3.303 (0.249)	0.02 (0.005)	−0.1669 (0.0717)	-1.5×10^{-5} (1.9×10^{-6})	0.846	0.817	0.933	0.50
Combined data	−3.526 (0.172)	0.0228 (0.0044)	−0.1949 (0.0622)	-1.7×10^{-5} (1.9×10^{-6})	0.794	0.777	0.916	0.548

[y]Estimated parameters corresponding to $WT(b_1)$, $W(b_2)$ and $WT^3(b_3)$. The numbers in parentheses are the standard deviations of the estimated parameters.
[z]b_0 is the value of $\ln(Y/(1-Y))$ when $T=0$ and $W=0$; value of Y at these conditions is equal to $1/(1+\exp(-b_0))$.

TABLE 3. Estimated parameters for a logistic model (equation 5) that describes the incidence of strawberry flowers with necrotic petals caused by *Botrytis cinerea* as a function of temperature (T) and wetness duration (W), together with coefficients of determination (R^2), R^2 adjusted for degrees of freedom (R_a^2), coefficient of determination for the back-transformed infection levels (R^{*2}), and the standard error about the regression line (s)

	Estimated parameters[y]							
	b_0[z]	b_1	b_2	b_3	R^2	R_a^2	R^{*2}	s
Test 1	−4.20 (0.306)	0.0342 (0.0029)	−0.130 (0.028)	-2.8×10^{-5} (2.7×10^{-6})	0.881	0.869	0.895	0.852
Test 2	−4.512 (0.494)	0.0212 (0.0055)	−0.015 (0.071)	-1.8×10^{-5} (3.9×10^{-6})	0.849	0.828	0.904	1.11
Combined data	−4.268 (0.274)	0.0294 (0.0024)	−0.0901 (0.274)	-2.35×10^{-5} (2×10^{-6})	0.860	0.850	0.901	0.990

[y]Estimated parameters corresponding to $WT(b_1)$, $W(b_2)$, and $WT^3(b_3)$. The numbers in parentheses are the standard deviations of the estimated parameters.
[z]b_0 is the value of $\ln(Y/(1-Y))$ when $T=0$ and $W=0$; value of Y at these conditions is equal to $1/(1+\exp(-b_0))$.

development of ripe fruit likely have a large influence on fruit disease incidence. We did not vary conditions after flowers dried in order to focus on the effects of temperature and wetness duration at the time of flower inoculation on fruit disease incidence. We believe that more variable temperature and moisture conditions during this period would have resulted in a lower proportion of fruit with gray mold symptoms.

It is well accepted that infection of strawberry flowers and the establishment of quiescent mycelium in floral parts by conidia of *B. cinerea* is one of the primary avenues for the later aggressive infection and rotting of ripe fruit (6,8–11,13,14,17,19,22). *B. cinerea* infects flower parts of several other crops, including raspberry (8), grape (16), apple (20,21), and macadamia nut (7). Strawberry pollen significantly enhances conidial germination of *B. cinerea* and germ tube elongation, and thus probably contributes to the development of latent infections (2). Powelson (17) isolated *B. cinerea* more frequently from necrotic strawberry petals, stamens, and sepals than from symptomless flower parts. He noted that removal of floral parts from one flower of a pair of flowers resulted in reduced fruit infection following inoculation with *B. cinerea*. Levels of fruit infection depended on the environment before and after flowering. A wet bloom and postbloom environment yielded the highest proportion of infected fruit, whereas dry bloom and postbloom conditions resulted in the lowest proportion of infected fruit. The open flower, white bud, and old senescent flower stages were the most susceptible to infection by *B. cinerea* (13). The green bud stage was relatively resistant (6,13). More recently, UV microscopy of strawberry pistils, stamens, petals, sepals, and receptacles infected by *B. cinerea* indicated that stamens may be the most important source of latent infection (3). Incidence of fruit infection has been found to be highly correlated with the preharvest (6–30 days) durations of RH > 80% and the amount of preharvest (11–30 days) rainfall (10), thus, strongly indicating that weather conditions during flowering are important in determining the level of gray mold fruit rot of strawberries. Jarvis and Borecka (13) found that fruit originating from flowers inoculated at open (petals fully reflexed), old (petals senescent), white bud (petals visible but not reflexed), and green bud stages, had fruit rot levels of 61, 54, 42, and 37%, respectively. However, no environmental data were recorded for their field study.

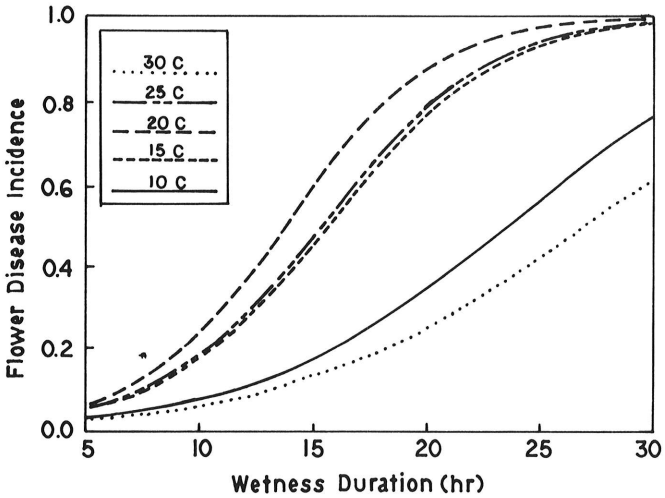

Fig. 5. Effect of wetness duration on the predicted proportion of flowers with petal necrosis caused by *Botrytis cinerea* at temperatures of 10, 15, 20, 25, and 30 C. Curves were generated using equation 5 with estimated parameters in Table 3 for the combined data.

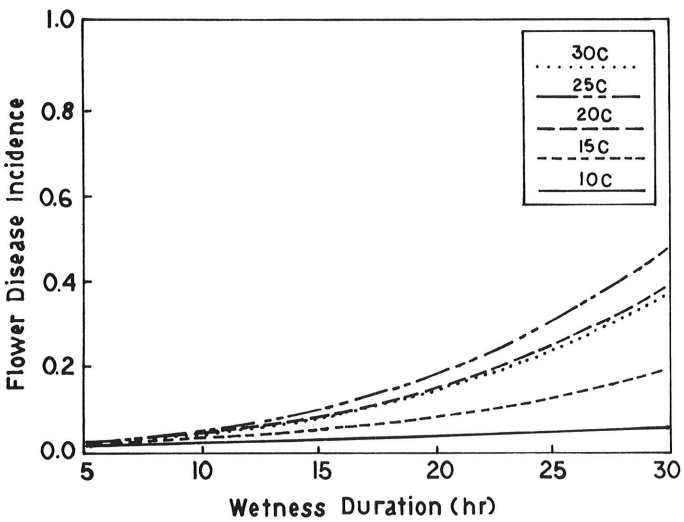

Fig. 6. Effect of wetness duration on the predicted proportion of flowers with stamen necrosis caused by *Botrytis cinerea* at temperatures of 10, 15, 20, 25, and 30 C. Curves were generated using equation 5 with estimated parameters in Table 4 for the combined data.

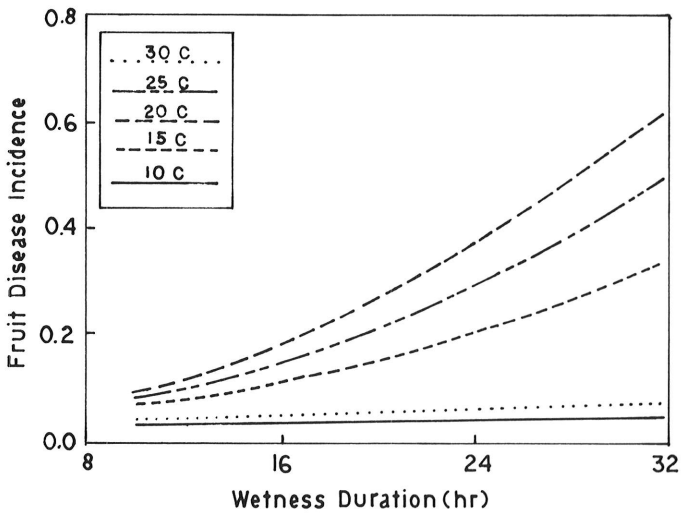

Fig. 7. Effect of wetness duration and temperature at flower inoculation on the predicted proportion of ripe strawberry fruit infected by *Botrytis cinerea*. Curves were generated for tempertaures of 10, 15, 20, 25, and 30 C using equation 5 with the estimated parameters in Table 2 for the combined experiments.

TABLE 4. Estimated parameters for a logistic model (equation 5) that describes the incidence of strawberry flowers with necrotic stamens caused by *Botrytis cinerea* as a function of temperature (T) and wetness duration (W), together with 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]							
	b_0[z]	b_1	b_2	b_3	R^2	R^2_a	R^{*2}	s
Test 1	−4.192	0.0104	−0.0588	-4.4×10^{-6}	0.769	0.745	0.799	0.681
	(0.245)	(0.0023)	(0.0222)	(2.1×10^{-6})				
Test 2	−4.64	0.0172	−0.1151	-1×10^{-5}	0.837	0.815	0.910	0.593
	(0.263)	(0.0029)	(0.0376)	(2.1×10^{-6})				
Combined data	−4.367	0.0127	−0.0703	-6.8×10^{-6}	0.781	0.769	0.870	0.658
	(0.181)	(0.0016)	(0.0182)	(1.3×10^{-6})				

[y] Estimated parameters corresponding to $WT(b_1)$, $W(b_2)$, and $WT^3(b_3)$. The numbers in parentheses are the standard deviations of the estimated parameters.
[z] b_0 is the value of $\ln(Y/(1-Y))$ when $T = 0$ and $W = 0$; value of Y at these conditions is equal to $1/(1 + \exp(-b_0))$.

Previous to this study, no quantitative data were available that demonstrated the effect of a range of temperatures and wetness periods on the establishment of infection of strawberry flowers by *B. cinerea* and the subsequent fruit infections arising from these infected flowers. However, similar studies have been done for *B. squamosa*, which causes leaf blight of onion (1,18). Onion leaf lesions were evident after 6 hr of wetness at temperatures of 15, 20, and 25 C (1). Optimum temperature for lesion development was 20 C, which was the same as found for strawberry fruit and flower infection in this study. Ramsey and Lorbeer (18) reported that the minimal, optimal, and maximal temperatures for onion floret blight by *B. cinerea* were 12, 21, and 31 C. Except for the minimum of 12 C, these temperatures are similar to the estimated cardinal temperatures for strawberry flower and fruit infection.

In Ohio, extended wet periods commonly are coincident with temperatures between 15–25 C that favor infection of strawberry flowers (L. V. Madden, *unpublished*). Therefore, fungicide applications during flowering are necessary for adequate control of gray mold. At present, there are no fungicides with postinfection or curative activity for control of gray mold on strawberry. If such a fungicide is developed in the future, it could be used in a postinfection spray program where applications are made in response to predicted infection periods. Fungicides currently being used are protectants that usually are applied on a 5- to 7-day schedule without consideration of infection periods or high disease risk situations. A disease prediction or forecasting system based on our data could permit growers to improve their timing of protectant fungicide applications. In seasons with excessive high risk periods, the application interval may be reduced and the number of sprays increased. During dry seasons the number of applications may be reduced.

Many of the fungicides currently being registered in the United States have a range of usage rates recommended on the label. For example, vinclozolin is registered for control of strawberry gray mold at the rates of 1.12–2.24 kg/ha. The label states the 1.12-kg rate should be used when low disease pressure is predicted and 2.24-kg rate should be used when the foliage is dense and disease pressure is high. A disease predictive system could be useful in aiding growers in determining when disease pressure is high. Equation 5 may be used to identify the risk of the development of flower infections, leading to a given level of fruit infection. Field studies are planned to test the validity of equation 5 under natural conditions where postinoculation conditions are highly variable and refine regression parameter estimates where necessary. It should be noted that the transition from a latent infection to an aggressive infection probably is influenced by environmental and host factors during ripening and, therefore, this is an area that needs further investigation.

LITERATURE CITED

1. Alderman, S. C., and Lacy, M. L. 1983. Influence of dew period and temperature on infection of onion leaves by dry conidia of *Botrytis squamosa*. Phytopathology 73:1020-1023.
2. Borecka, H., and Millikan, D. F. 1973. Stimulatory effect of pollen and pistillate parts of some horticultural species on the germination of *Botrytis cinerea* spores. Phytopathology 63:1431-1432.
3. Bristow, P. R., McNicol, R. J., and Williamson, B. 1986. Infection of strawberry flowers by *Botrytis cinerea* and its relevance to gray-mould development. Ann. Appl. Biol. 109:545-554.
4. Coley-Smith, J. R., Verhoeff, K., and Jarvis, W. R. 1980. The Biology of *Botrytis*. Academic Press, London, England. 318 pp.
5. Grove, G. G., Madden, L. V., Ellis, M. A., and Schmitthenner, A. F. 1985. Influence of temperature and wetness duration on infection of immature strawberry fruit by *Phytophthora cactorum*. Phytopathology 75:165-169.
6. Hennebert, G. L., and Gilles, G. L. 1958. Epidemiology de *Botrytis cinerea* Pers. sur fraisers. Meded. Landbouwhogesh. Opzaekingsstn. Staat Gent. 23:864-888.
7. Hunter, J. E., Rohrbach, K. G., and Kunimoto, R. K. 1972. Epidemiology of Botrytis blight of macadamia racemes. Phytopathology 62:316-319.
8. Jarvis, W. R. 1962. Infection of strawberry and raspberry fruit by *Botrytis cinerea* Pers. Ann. Appl. Biol. 50:569-575.
9. Jarvis, W. R. 1962. The epidemiology of *Botrytis cinerea* Pers. in strawberries. Pages 258-262 in: Proc. Int. Hortic. Congr. 1961.
10. Jarvis, W. R. 1964. The effect of some climatic factors on the incidence of grey-mould of strawberry and raspberry fruit. Hortic. Res. 3:65-71.
11. Jarvis, W. R. 1969. The phenology of flowering in strawberry and raspberry in relation to grey-mould control. Hortic. Res. 9:8-17.
12. Jarvis, W. R. 1977. *Botryotinia* and *Botrytis* Species: Taxonomy, Physiology, and Pathogenicity. Monograph 15. Research Branch, Canada Department of Agriculture. 195 pp.
13. Jarvis, W. R., and Borecka, H. 1968. The susceptibilty of strawberrry flowers to infection by *Botrytis cinerea* Pers. Hortic. Res. 8:147-154.
14. Jordan, V. W. L. 1978. Epidemiology and control of fruit rot caused by *Botrytis cinerea* on strawberry. Pflanzenshutz.-Nachr. Bayer (Am. Ed.)
15. Kritzman, G., and Netzer, D. 1978. A selective medium for the isolation and identification of *Botrytis* spp. from soil and onion seed. Phytoparasitica 6(1):3-7.
16. McClellan, W. B., and Hewitt, W. B. 1973. Early Botrytis rot of grapes: Time of infection and latentcy of *Botrytis cinerea* Pers. in *Vitis vinifera* L. Phytopathology 63:1151-1157.
17. Powelson, R. L. 1960. Initiation of strawberry fruit rot caused by *Botrytis cinerea*. Phytopathology 50:491-494.
18. Ramsey, G. R., and Lorbeer, J. W. 1986. The role of temperature and free moisture in onion flower blight. Phytopathology 76:612-616.
19. Ramsey, G. R., and Lorbeer, J. W. 1986. Pathogenicity of *Botrytis* species on onion umbels and scapes under controlled conditions. Phytopathology 76:604-612.
20. Sitterly, W. R., and Shay, J. R. 1960. Physiological factors affecting the onset of susceptibility of apple fruit to rotting by fungus pathogens. Phytopathology 50:91-93.
21. Tronsmo, A., Tronsmo, A. M., and Raa, J. 1977. Cytology and biochemistry of pathogenic growth of *Botrytis cinerea* in apple fruit rot. Phytopathol. Z. 89:208-215.
22. Verhoeff, K. 1974. Latent infections caused by fungi. Annu. Rev. Phytopathol. 12:99-110.