A Model Based on Temperature and Leaf Wetness Duration for Establishment of Alternaria Leaf Blight of Muskmelon

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ABSTRACT

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We inoculated muskmelon leaves with a conidial suspension of *Alternaria cucumerina* and subjected the leaves to various controlled environments to determine the influence of temperature and leaf wetness duration on disease establishment. Disease severity, as measured by a "number of lesion area equivalents" method, increased with increasing wetness duration (2–24 h) at all temperatures tested (12–30 C). The optimum temperature was 18 C, and at least 8 h of leaf wetness were required for significant disease establishment at temperatures of 15–21 C. Analysis of variance with orthogonal polynomial contrasts and also regression analysis were used to determine the functional relationship between disease severity and temperature and leaf wetness duration. The resulting polynomial response surface provided a close fit to the observed data.

Alternaria leaf blight, which is caused by *Alternaria cucumerina* (Ellis & Everh.) J. A. Elliott, is one of the most important foliar diseases of muskmelon (*Cucumis melo* L. var. *reticulatus* Naudin) in Indiana and other midwestern and eastern states. In Indiana, the disease has been responsible for yield reductions of nearly 50% in situations in which control measures were ineffective or absent (23). In the absence of control measures, the potential for severe defoliation and crop loss is great because of numerous secondary disease cycles arising from primary inoculum in crop debris. Defoliation can adversely affect fruit quality by predisposing fruit to sun scald (14). The potential for increased fruit respiration of defoliated vines can reduce soluble solid content and fruit sweetness (1). Resistance to *A. cucumerina* is not available in commercially acceptable cultivars. Therefore, growers must rely on repeated applications of fungicides for acceptable disease management. Protective fungicides, such as chlorothalonil, mancozeb, and fixed copper fungicides, are applied on a 7- to 10-day schedule even though environmental conditions are not always favorable for disease increase.

An investigation of the environmental factors that influence the infection component of the disease cycle is critical to the understanding of factors responsible for disease establishment and spread. Chandler and Thomas (5) reported that at least 8 h of continuous leaf wetness were necessary for establishing significant levels of disease on muskmelon leaves under controlled environment conditions. Temperature and leaf wetness duration have been correlated with leaf blight severity in the field (9,18,24), but "cause and effect" relationships and the interaction of these two environmental factors on the infection process were not determined. The objective of this study was to quantify the influence of temperature and leaf wetness duration on the severity of Alternaria leaf blight on muskmelon leaves under controlled environment conditions. Results will form the basis for the future development of a fundamental disease forecasting system for scheduling protective fungicide applications.

MATERIALS AND METHODS

Plant production. Muskmelon seed (cv. All Star, Harris Moran Seed Company, San Juan Bautista, CA) was planted into alternate cells of 196-cell plastic cavity trays containing a soilless potting mix (Vegetable Plug Mix, W. R. Grace & Co., Cambridge, MA). Each cell was 7 cm deep and 2.5 cm in diameter. Seedlings were transplanted 9–11 days later into 11-cm-diameter plastic pots containing sand, soil, and potting mixture (Vaughan's Basic Blend, Downers Grove, IL) in a 2:1:1 (v/v/v) mixture. Each pot contained one plant. Fertilizer (RAPIDGRO Plant Food 23-19-17, Chevron Chemical Co., San Ramon, CA) was applied at a rate of 3.4 g/L via irrigation water 2 days after plants were transplanted and weekly thereafter. Light intensity in the greenhouse ranged from 70 to 400 $\mu E \cdot m^{-2} \cdot s^{-1}$ during a 14-h (on average) photoperiod. Temperatures in the greenhouse ranged from 18 to 40 C but were mostly 24–30 C.

Inoculum production and inoculation. For each inoculation, inoculum was increased from recent single spore isolates that were obtained from lesions on plants from the greenhouse. All isolates originated from an isolate of *A. cucumerina* (Latin 8721) from an infected muskmelon leaf collected in a local field of cv. Superstar in 1987. Sporulation was induced by transferring mycelia from the edges of acidified potato-dextrose agar (APDA) cultures to agar containing 20% V8 juice. All cultures were maintained under a 12-h photoperiod at 24 ± 2 C. Conidial suspensions were prepared by flooding the surface of 5- to 8-day-old V8 juice agar cultures with deionized water. Suspensions were filtered through two layers of surgical gauze and adjusted to 7,500 spores per milliliter with the aid of a hemacytometer.

Muskmelon plants were inoculated at the four-leaf stage when the fourth true leaf was still expanding rapidly. Plants were selected for inoculation when the third and fourth leaves had leaf widths at the midrib midpoint in the ranges of 9–12 cm and 3–8 cm, respectively. The conidial suspension was applied to runoff onto the surface of the second and third leaves with a manually operated plastic sprayer. We assessed spore viability by spraying the suspension onto APDA and estimating the percentage of spore germination after 6–8 h.

Disease assessment. Disease severity was assessed as the area of leaf tissue that was necrotic because of disease. A new method for assessing the severity of Alternaria leaf blight on muskmelon leaves is described. When the area of necrotic leaf tissue was less than approximately 20% of the healthy leaf area, disease severity was estimated by a "number of lesion area equivalents" method. This method involved counting the number of lesions in different size categories and then expressing disease severity

as the number of lesions equivalent to the size of those lesions in the smallest size category. The formula for this "number" is

$$\text{LAE} = C1 + (10 \times C2) + (C3_{\text{area}}/0.2) \quad (1)$$

in which LAE is the number of *C1* lesion area equivalents per leaf; *C1* is the number of lesions with diameter ≤1 mm; *C2* is the number of lesions with diameter >1 mm and ≤5 mm; and $C3_{\text{area}}$ is the sum of the areas (mm^2) of lesions with diameter >5 mm.

The number of lesion area equivalents per leaf (LAE) was divided by the leaf area to express disease severity as LAE per square centimeter of leaf area. Leaf area was measured electronically with a LI-3000 leaf area meter (LI-COR, Lincoln, NE). The constant value of 10 in Equation 1 was the median *C2* lesion area (=2 mm^2) divided by the median *C1* lesion area (=0.2 mm^2). These values of the median lesion areas were determined in a preliminary experiment (K. J. Evans, *unpublished*). We estimated the sum of the area of lesions in *C3* by measuring the approximate dimensions of each lesion (diameter or length and width). The constant value of 0.2 mm^2 in Equation 1 was the median *C1* lesion area. If a leaf was 100% necrotic, then the theoretical maximum value of LAE was 500 (=100 mm^2/0.2 mm^2) per square centimeter of leaf area. This method was of practical use when disease severity was less than approximately 100 LAE/cm^2 of leaf area.

Controlled environment study. To induce infection, we kept inoculated plants continuously wet at various temperatures in a Percival dew chamber (Model I-35D, Boone, IA). Temperature treatments were 12, 15, 18, 21, 24, 27, or 30 C. Temperatures to be tested were chosen in random order. The temperature within the dew chamber was monitored with a −20 to 110 C mercury thermometer (Fisher Scientific, Springfield, NJ). The temperature was maintained within an observed range of ±1 C. A total of 18 muskmelon plants were used for each temperature treatment. Three plants were removed from the dew chamber after exposure to 2, 4, 8, 12, 16, or 24 h of leaf wetness and placed in a growth chamber (Environmental Growth Chambers Model M-31, Chagrin Falls, OH) at the same temperature as the dew chamber. The temperature within the growth chamber was monitored with another mercury thermometer that was previously calibrated with the dew chamber mercury thermometer. Plants were completely randomized in both the dew chamber and growth chamber. A leaf wetness sensor (DP223, Omnidata International Inc., Logan, UT) within the growth chamber was gently misted with water on transfer of inoculated plants from the dew chamber to the growth chamber so that the length of time the plants remained wet could be recorded. Drying times varied from 45 min to 3 h, depending on the relative humidity within the growth chamber. The relative humidity within the chamber appeared to be closely related to the ambient conditions within the room housing the growth chamber. There was no apparent correlation between the length of drying time and experimental treatment. We developed the statistical model for disease establishment by using preassigned treatment levels of leaf wetness duration.

All inoculated plants were kept in darkness for the first 24 h. After 24 h, the growth chamber was reset to 27 C and to a 16-h photoperiod at 90 $\mu E \cdot m^{-2} \cdot s^{-1}$ to promote symptom expression. Disease severity was assessed, as described previously, on the third leaf only of each plant 5 days after inoculation. A single leaf position was selected for disease assessment, because there appeared to be age-related differences in disease severity between the second and third true leaves (Fig. 1).

The entire procedure of testing 42 temperature and leaf wetness duration combinations was repeated, and this represented the second replication. The experimental design was classified as a generalized split-plot design with whole units arranged in a randomized complete block design. The whole unit was temperature, and replications of this unit were blocks in time. The subunit was leaf wetness duration, and there were three repeats of the subunit within the whole unit, giving a generalized randomized complete block design for each temperature level (22).

Data analysis and model development. Analysis of variance (ANOVA) and regression techniques were used to determine the functional relationship between disease severity (Y in LAE/cm^2 of leaf area) and temperature (T, C) and leaf wetness duration (W, h). The linear model was

$$Y_{ijkl} = m + R_i + T_j + a_{(ij)} + W_k + TW_{jk} + b_{(ijk)} + E_{(ijk)l} \quad (2)$$

in which, Y_{ijkl} is the response of the $ijkl$th individual subunit; m is the overall mean; R_i is the effect of the ith replication (random), $i = 1, 2$; T_j is the effect of the jth temperature (fixed), $j = 1, \ldots, 7$; $a_{(ij)}$ is the random effect of the ijth whole unit, including RT_{ij} interaction effect, if present (error term for T); W_k is the effect of kth duration of leaf wetness (fixed), $k = 1, \ldots, 6$; TW_{jk} is the interaction effect of jth temperature with kth wetness duration; $b_{(ijk)}$ is the random effect of the ijkth subunit with respect to replication, temperature, and wetness duration, including RW_{ik} and RTW_{ijk} interaction effects, if present (error term for W and TW); and $E_{(ijk)l}$ is the random effect of the lth subunit in the ijkth combination, $l = 1, \ldots, 3$. If RW_{ik} and RTW_{ijk} equal zero, then σ_b^2 equals σ_E^2.

We performed analyses of variance by using the Statistical Analysis System (SAS) (20). The relationship between the standard deviation of Y and the mean Y, at each level of W, was linear, suggesting a logarithmic transformation. Therefore, the data were transformed to $\log_{10}(Y + 1)$ before analysis. Sums of squares for temperature, leaf wetness duration, and temperature by leaf wetness duration interaction were partitioned by using orthogonal polynomial coefficients computed by the SAS (IML) ORPOL function.

From the significant sources in the analysis of variance table, the model for the response surface can be written in terms of orthogonal polynomial coefficients. The model can be converted into an expression in terms of T and W by algebraic substitution for the orthogonal polynomial coefficients (22). Instead, we used SAS REG (20) to approximate the response surface by specifying all the terms required from the orthogonal polynomial model, including all lower order terms whether significant or not. We computed the regression equation by using the observed mean for each temperature and leaf wetness duration combination. One criterion for an acceptable regression model was that the predicted response surface should agree with the expected biological response. Other criteria for an acceptable model included goodness of fit between the observed and predicted values, randomness and normality of residuals, and R^2 and adjusted R^2 values. When regression procedures for model development have been used by

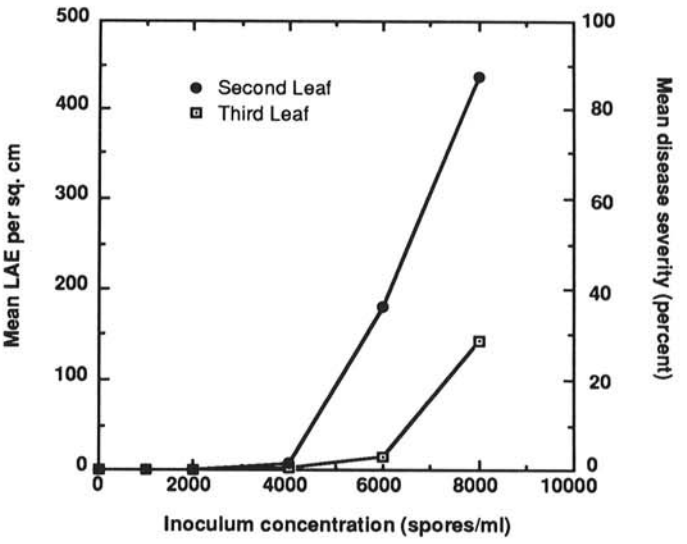

Fig. 1. The response of the second and third true leaves of muskmelon plants to increasing inoculum concentration of *Alternaria cucumerina*. Plants were exposed to 24 h of leaf wetness at 18 C. Disease severities greater than 30% were estimated visually. Each point represents the mean of three plants.

other researchers (2,8), one criterion for selecting independent variables for the regression equation was that the estimated parameters must be significant according to Student's t tests. This criterion did not need to be satisfied in this regression because variables were selected based on their significance in the analysis of variance.

The 80% confidence intervals for population means were constructed about predicted means and were obtained by adjusting those calculated by the procedure of SAS REG (20). We used the 80% confidence intervals instead of the 95% intervals to reduce the frequency of overlap and to improve the clarity of the graphical presentation. The confidence intervals were adjusted to utilize the average error variance in the ANOVA, rather than the residual variance in the regression model. The error variances in the ANOVA were used because they were pure errors and were independent of any specific model. The ANOVA error variance, when available, is commonly used for testing the regression components (22). The adjusted limit of error (ALE_{ij}) for the ith temperature and the jth wetness duration, being one-half of the 80% confidence interval for the population mean, was calculated as

$$ALE_{ij} = AC \times LE_{ij} \quad (3)$$

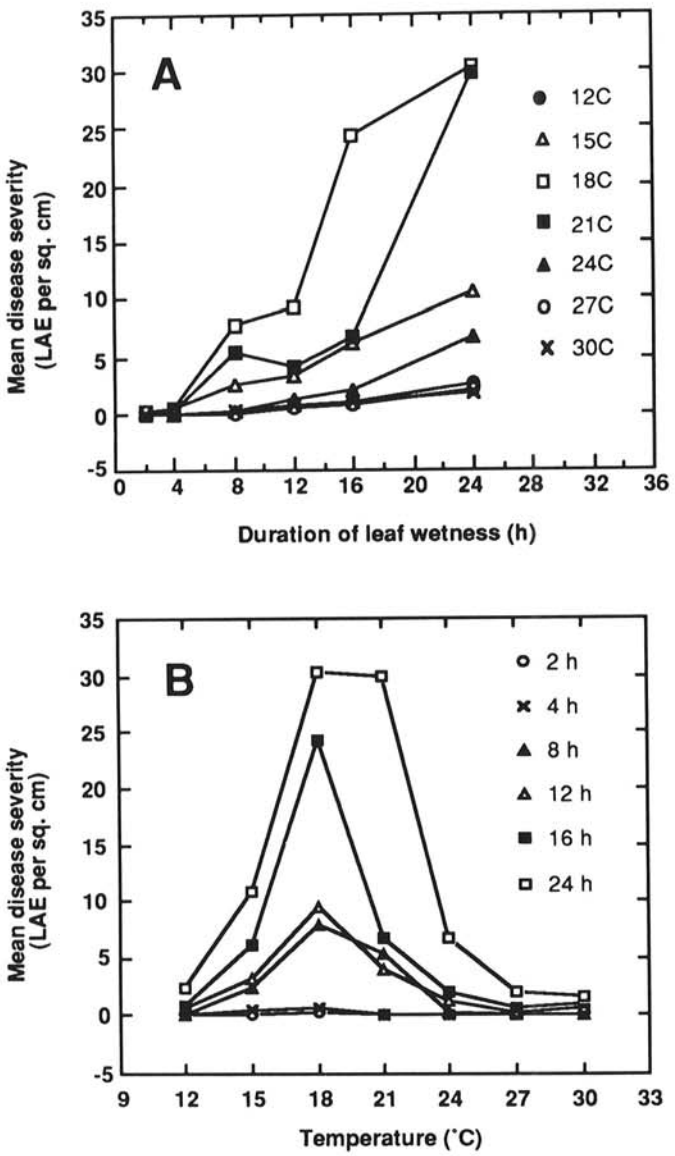

Fig. 2. The observed mean disease severities on the third leaf of muskmelon plants inoculated with a conidial suspension of *Alternaria cucumerina* at **A,** different temperatures and **B,** leaf wetness durations.

in which AC is the adjustment constant, $(t_{anova}/t_{reg}) \times ([s^2_{anova}/r]^{1/2}/[s^2_{reg}]^{1/2})$:

t_{anova} is the t value (2-tailed, $\alpha = 0.20$) for df for error(a) plus df for error(b) ($df = df_a + df_b = 5 + 30 = 35$);

t_{reg} is the t value (2-tailed, $\alpha = 0.05$) for df for residual mean square in regression analysis (i.e., number of observations minus number of parameters fitted; $df = 42 - 12 = 30$);

$s^2_{anova} = (df_a \times MS_a + df_b \times MS_b)/(df_a + df_b)$,
MS_a = mean square of error(a), and MS_b = mean square of error(b);

$r = 6$ = number of observations in the means used in the regression analysis;

s^2_{reg} = regression residual mean square;

LE_{ij} = limit of error for the ijth population mean, being one-half of the 95% confidence interval for the population mean, calculated by the procedure of SAS REG.

RESULTS

In general, there was an increase in disease severity with increase in leaf wetness duration at all temperatures tested (Fig. 2A). Disease severity on individual leaves ranged from 0 to 82 LAE/cm^2. The maximum observed mean disease severity occurred after 24 h of wetness duration at 18 C. Infection was observed at all treatment combinations tested, although mean disease severity was less than 1 LAE/cm^2 for 2 and 4 h of wetness duration for all temperatures (Fig. 2B). Disease severity remained below 1 LAE/cm^2 for up to 8 h of wetness duration at 24 C, and for up to 12 h of wetness duration at 12, 27, and 30 C.

Plants exposed to 12 C in the second replication exhibited symptoms of chill injury. In the first 24 h after inoculation, the margins of most leaves were curled, and the whole leaf was somewhat flaccid. Disease severity on some leaves exposed to 16 and 24 h of leaf wetness was 150–250 LAE/cm^2. This magnitude of disease severity was much higher than observed in the 18 C treatments and was possibly due to the injured host tissue. The

TABLE 1. Analysis of variance for $\log_{10}$ (LAE/cm^2 + 1) on the third leaf of muskmelon plants inoculated with a conidial suspension of *Alternaria cucumerina* at various combinations of temperature and leaf wetness duration[a]

Source of variation	df	MS[b]	P
Replication	1	0.0925	...
Temperature (T)	6		
T-linear (l)	1	2.8115	0.04
T-quadratic (q)	1	5.9502	0.01
T-cubic (c)	1	3.6850	0.02
Lack of fit	3	0.2118	0.64
Error (a)	5[c]	0.3555	...
Wetness duration (W)	5		
W-linear (l)	1	22.7170	0.4×10^{-11}
W-quadratic (q)	1	0.0606	0.27
Lack of fit	3	0.0335	0.55
$T \times W$	30		
T-l $\times$ W-l	1	1.1726	0.2×10^{-4}
T-l $\times$ W-q	1	0.0892	0.18
T-q $\times$ W-l	1	2.1729	0.1×10^{-6}
T-q $\times$ W-q	1	0.1366	0.10
T-c $\times$ W-l	1	0.4000	0.01
T-c $\times$ W-q	1	0.6135	0.1×10^{-2}
Lack of fit	24	0.0390	0.68
Error (b)	30[c]	0.0470	...
Plant-to-plant error	156[c]	0.0261	...

[a] LAE is the lesion area equivalent.
[b] Mean square.
[c] Degrees of freedom reduced to account for 18 missing values estimated.

18 observations were eliminated from the analysis, and, instead, estimated observations were calculated from those of the first replication by adjusting for the difference between the mean of the second replication and the mean of the first replication of all other temperatures. We reduced degrees of freedom in the ANOVA table (Table 1) to account for the missing values.

The ANOVA identified significant linear, quadratic, and cubic relationships between $\log_{10}(Y+1)$ and temperature (T) and a significant linear relationship between $\log_{10}(Y+1)$ and wetness duration (W). There were also significant treatment interaction components. Therefore, the response to T was not consistent for all levels of W.

Polynomial model. The influence of temperature and wetness duration on the severity of Alternaria leaf blight of muskmelon under controlled environment conditions was described by the following model:

$$\begin{aligned}\text{Log}_{10}(Y+1) = {} & b_0 + b_1T + b_2T^2 + b_3T^3 + b_4W + \\ & b_5W^2 + b_6TW + b_7TW^2 + b_8T^2W + \\ & b_9T^2W^2 + b_{10}T^3W + b_{11}T^3W^2 \end{aligned} \quad (4)$$

in which Y is the disease severity in LAE/cm^2 of leaf area, T is the temperature, and W is the leaf wetness duration. The parameter estimates, $b_0, \ldots, b_{11}$, are listed in Table 2. Because the interaction component T-cubic $\times$ W-quadratic was highly significant, three lower order nonsignificant terms, W-quadratic, T-linear $\times$ W-quadratic, and T-quadratic $\times$ W-quadratic, were included in the regression model. Therefore, the number of parameters in the regression model was increased from nine to twelve. The high correlation between independent variables resulted in nonsignificant parameter estimates for T-linear, T-quadratic, and T-cubic. One or more of these terms could have been eliminated from the model by a backward elimination procedure, but their elimination would have had little effect on the response surface. The R^2 and adjusted R^2 were 0.96 and 0.95, respectively. The residual mean square of the model was 0.0093, and a random pattern of residuals was observed across the range of predicted means. Figure 3 shows that the predicted curves closely fit the observed values at each temperature.

Although there was an increase in $\log_{10}(Y+1)$ with increase in W at all T, the shapes of the predicted curves varied with the value of T (Fig. 4A). Bell-shaped curves were produced when $\log_{10}(Y+1)$ was plotted against temperature (T) (Fig. 4B). The curve flattened with decreasing values of duration of leaf wetness (W). According to the model, disease severity was at a maximum at 17.8 C after 24 h of leaf wetness (the maximum experimental level). The optimum temperature range for disease establishment was 15–21 C on the basis of the closeness of the predicted values in this temperature range for a given wetness duration. Within this same temperature range, at least 8 h of leaf wetness were required for significant disease establishment.

The model predicted an increase in $\log_{10}(Y+1)$ when T increased from 27 C to 30 C between 4 and 16 h of wetness duration (Figs. 4B,5). However, this response may not represent a biological response because there appeared to be no real increase in disease severity as shown in Figure 2B.

TABLE 2. Estimated parameters from Equation 4 for temperature and leaf wetness duration effects on the severity of Alternaria leaf blight of muskmelon

Parameter	Parameter estimate	Standard error
b_0	2.403002	2.24
b_1	−0.386617	0.35
b_2	0.018483	0.017
b_3	−0.000277	0.27×10^{-3}
b_4	−1.818693	0.43
b_5	0.056367	0.016
b_6	0.286222	0.067
b_7	−0.008811	0.0025
b_8	−0.013609	0.0033
b_9	0.000427	0.12×10^{-3}
b_{10}	0.000204	0.52×10^{-4}
b_{11}	−0.000006547	0.2×10^{-5}

DISCUSSION

Temperature and leaf wetness duration were clearly important factors in the establishment of disease on muskmelon leaves by *A. cucumerina*. A high proportion of the variability of mean disease severity was accounted for by the components in the polynomial model. The analysis of variance provided the tool for directly selecting variables in the model rather than using a regression procedure for selecting variables to build the model. Regression was used to fit the response surface, and the regression on the treatment means allowed the information from SAS REG to be used directly in the computation of confidence intervals for the response surface.

We adopted an empirical approach to model building to describe the shape of the response surface, which was similar to descriptions of infection models for other diseases (2,3,6,8,13,21). The polynomial model was simply a statistical model for fitting curves to the data. It was possible to draw conclusions about the biological relevance of the shape of the response surface, but the parameter estimates were a function of the controlled environment. That is, the response to temperature showed the typical bell-shape curve seen in many other biological responses to temperature, but the magnitude of the response may vary under different experimental conditions. Other empirical, statistical models, such as the Richards function (3,13,19,26) contain constants (e.g., rate and asymptote parameters) that yield information of biological interest. However, these other types of functions have a more rigid nonlinear mathematical structure that is difficult

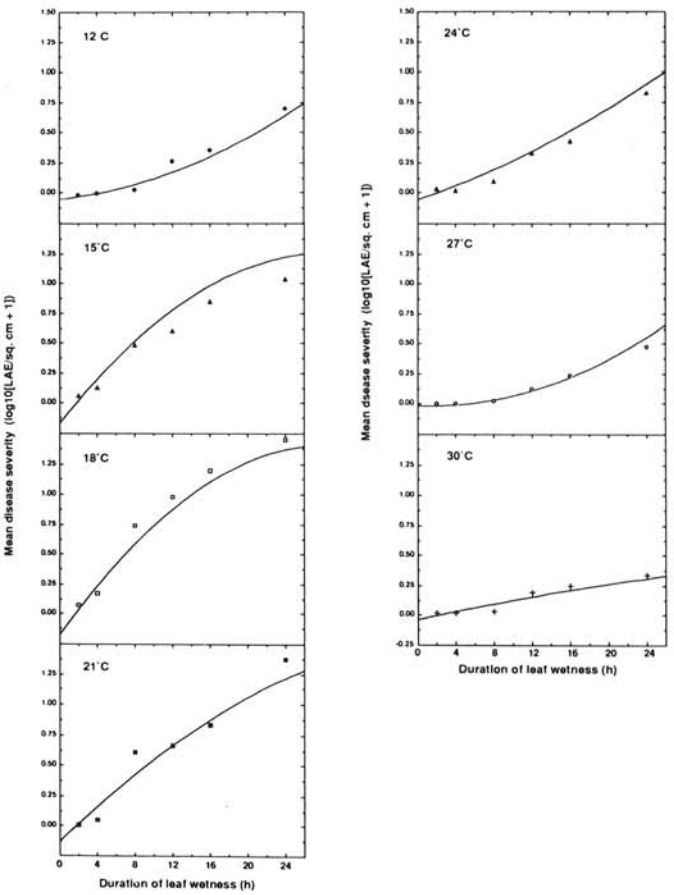

Fig. 3. Observed and predicted mean severities of Alternaria blight of muskmelon at various combinations of temperature and leaf wetness duration. The solid lines were generated by the polynomial model described in Equation 4. The data points represent observed mean disease severities.

to deal with statistically (26). Both types of functions may not be applied outside the range of temperatures and leaf wetness durations used to derive the equation. It was concluded that the choice of a polynomial model was adequate for the modeling objectives of this study.

The additional wetness exposure of plants due to drying time was considered a type of experimental bias. For the purposes of model application, the direction of the bias could lead to an underestimate of the wetness period required for significant disease establishment. The drying time was an additional period of wetness exposure, but the quality of the wetness period, in terms of spore germination and penetration, was probably different from the quality of the wetness period created in the dew chamber. Drying time was not added to the treatment level of wetness duration because of this difference. In addition, there are practical difficulties in quantifying accurate drying times for each leaf. Drying time according to the leaf wetness sensor was correlated with actual drying time, but the drying time was an average for all plants in the chamber. Practical difficulties aside, the main reason for not incorporating drying time in the model relates to the proposed field application of the model. We propose to use this statistical model to develop a weather-based disease forecaster for scheduling protective fungicide applications. The forecaster will predict general disease increase rather than the degree of symptom expression after an infection period. Under this system, a fungicide application is timed after some critical level of cumulative weather events. In terms of disease prediction in the field, the bias of the statistical model may sometimes lead us to predict more disease than that which actually occurs. However, there are two reasons why this problem may not manifest itself in the practical application of the disease forecaster. First, there is a drying time associated with leaf wetness periods observed in the field. A leaf wetness sensor often records a "dry" reading when some leaves in the lowest levels of the canopy are still wet. The calibration and positioning of the leaf wetness sensor will affect the duration of the measured wetness period. Second, the proposed decision rule for a fungicide application will not rely on a weather event observed during a single 24-h period. The decision rule, based on cumulative weather events, will be determined from field experiments. There may be flexibility to adjust the decision rule to compensate for any "on-average" overestimation of disease increase. The grower may even benefit by a conservative disease prediction, because the disease risk associated with extended spray intervals is likely to be reduced.

The selection of host tissue type and the LAE disease assessment method produced a wide range of disease severities, even though the observed maximum disease severity was less than 20% of the theoretical maximum of 100% necrotic leaf area. We selected this disease severity range to enable separation of individual lesions so that the measurement related more closely to the infection process than the lesion expansion phase of disease establishment. The term "disease establishment" is used as a general term to describe the outcome of infection and lesion expansion. Given that the model is not strictly an infection model, it is called disease establishment to account for the unavoidable confusion between infection and lesion growth.

The chill injury observed in the second replication of the 12 C treatment predisposed the plant to greater disease. In both replications of 12 C, there was approximately a 12 C decrease in temperature when plants were transferred from the warm greenhouse to the cool dew chamber. The entire experiment was conducted during the months of May to September. Plants for the first replication of 12 C were raised in the month of June, whereas plants for the second replication of 12 C were raised

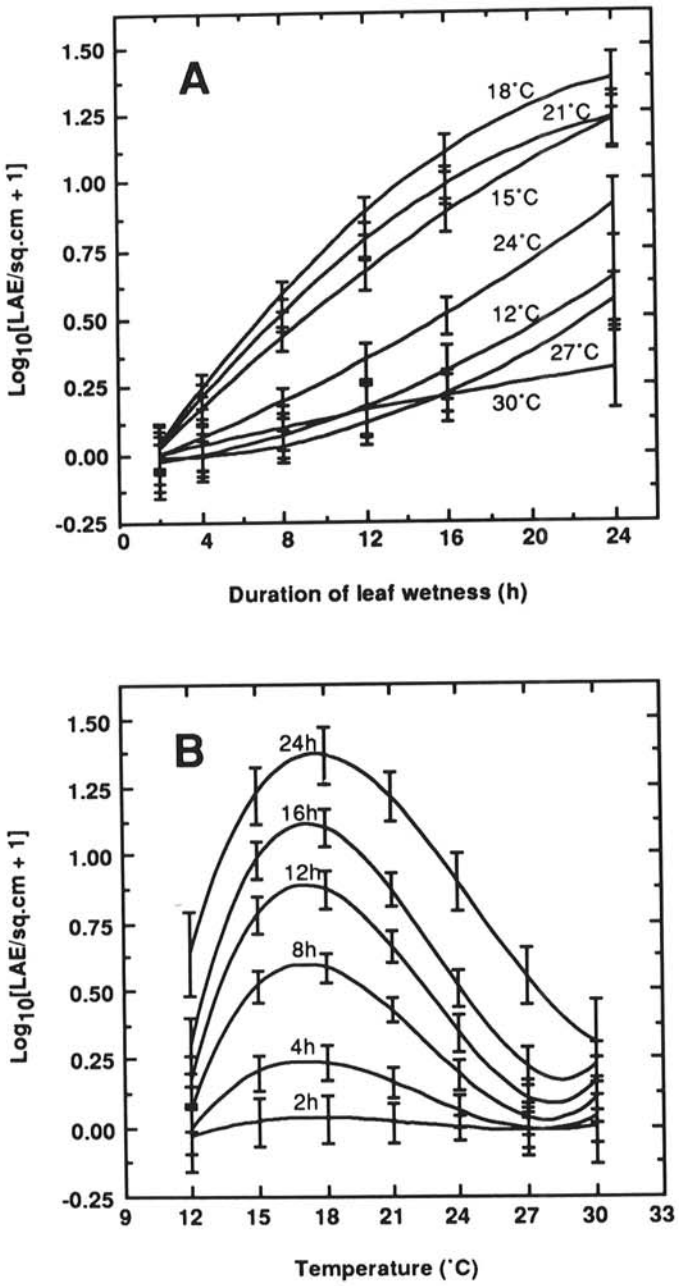

Fig. 4. Prediction curves for Alternaria blight of muskmelon based on the polynomial model for **A**, different temperatures and **B**, leaf wetness durations. The error bars are the 80% confidence intervals for the predicted means calculated by Equation 3.

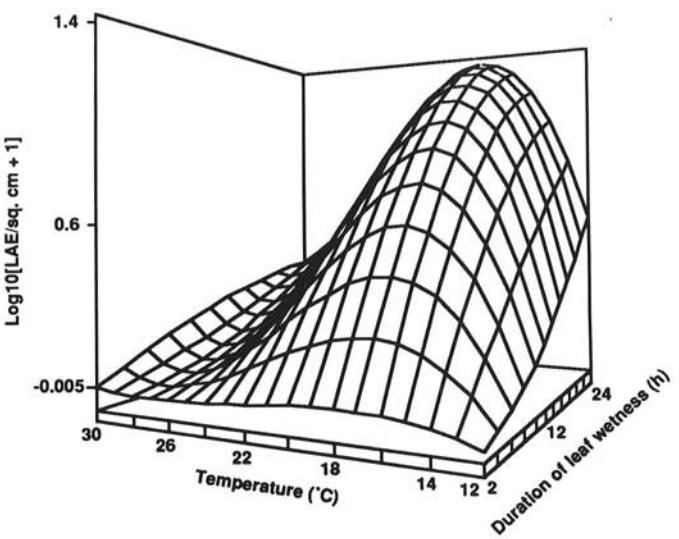

Fig. 5. A response surface based on the polynomial model (Eq. 4) describing the influence of temperature and leaf wetness duration on disease severity on the third leaf of muskmelon plants inoculated with a conidial suspension of *Alternaria cucumerina*.

in the month of August. The difference in the time of year that the plants were raised in the greenhouse may have contributed to differences in plant vigor and thus differences in predisposition to chill injury. Other types of leaf tissue injury can also predispose the plant to greater disease (5). Tissue injury appears to enhance the infection process of *A. cucumerina*, which necessitates the maintenance of healthy plants in controlled studies.

The results of this research reflect the findings of many studies that have investigated the role of wetness periods in disease, namely that the duration of leaf wetness needed for disease establishment depends on temperature (10). Our model is consistent with the concept that the duration of leaf wetness determines the amount of spore germination and host penetration, whereas temperature determines the rapidity and extent of infection (10,13). At least 8 h of leaf wetness were required for significant disease establishment of *A. cucumerina* at the optimum temperature of 18 C. This result was consistent with the controlled environment studies of Chandler and Thomas (5) who also found that 8 h of leaf wetness (temperature not specified) were necessary for significant levels of infection of muskmelon leaves by *A. cucumerina*. An optimum value for the wetness period was unobtainable within the range tested. It is likely that when temperature becomes limiting, extended leaf wetness periods have potential for compensation (16).

Thomas (24) observed the importance of dew periods in the development of Alternaria leaf blight epidemics in Texas. Delaying fungicide applications until continuous leaf wetness periods reached at least 8 h during the growing season resulted in acceptable disease control and at least a one-third reduction in the number of applications on the basis of crop phenology. Severe epidemics resulting in 100% disease severity in experimental field plots were observed when temperatures during wetness periods longer than 8 h were in the range of 16–23 C (K. J. Evans and R. X. Latin, *unpublished*). In controlled environment studies, Carmody et al (4) and Thomas et al (25) established disease on muskmelon plants by using leaf wetness periods longer than 12 h and temperatures in the range of 19–23 C. These environmental conditions for disease establishment were also consistent with the findings of this study.

Temperature optima for infection and lesion expansion are likely to be different (7). We incubated all plants at the relatively high temperature of 27 C to promote symptom expression after infection. In fact, daily maximum temperatures during the Indiana growing season often exceed 27 C. Jackson (9) observed that Alternaria leaf blight on cucurbits in Florida was severe when daily temperatures were in the range of 20–32 C. Prasada et al (18) observed that disease was most severe on watermelons in India when daily temperatures were in the range of 25–30 C. These reported temperature ranges provide limited information for comparative purposes because they were not related specifically to muskmelons, wetness periods, or a specific component of the disease cycle.

The measure of disease severity in this study was not simply a measure of fungal colonization but a measure of fungal colonization relative to host growth. In the absence of disease, the area of the third true leaf can expand by up to 40% over the course of the experiment. Differences between the response at 27 and 30 C, although not statistically meaningful, might be explained by differences in the relative growth between host and fungus during the incubation period. It was concluded that temperature settings of 27 and 30 C should be treated as equivalent conditions for disease establishment under controlled conditions.

Although the model developed here applies strictly to the controlled environment conditions used in this study, relative differences between sets of environmental factors could be used to develop a daily index of environmental favorability for disease increase in the field (6,11,12,15,17). The development of a weather-based forecaster for minimizing unnecessary sprays is warranted for this economically important crop. Research on the correlation of environmental indices and disease increase and the development of a decision rule for the scheduling of protective fungicide applications continues.

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