Path Coefficient Analysis of the Effect of Rainfall Variables on the Epidemiology of Phytophthora Blight of Pepper Caused by *Phytophthora capsici*

J. H. Bowers, R. M. Sonoda, and D. J. Mitchell

First and third authors: Department of Plant Pathology, University of Florida, Gainesville 32611; second author: Agricultural Research and Education Center, P.O. Box 248, Ft. Pierce, FL 34954. Present address of first author: Department of Plant Pathology, The Ohio State University, Ohio Agricultural Research and Development Center, Wooster 44691.

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ABSTRACT

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Field plots were established in Delray Beach, FL, in the spring and fall of 1984, the spring of 1985, and the fall of 1986 to quantify disease progress and the effect of rainfall and temperature variables on Phytophthora blight of pepper caused by *Phytophthora capsici*. From point sources of inoculum (diseased plants), the incidence of disease was observed to spread outward over time from the central, primary foci. Disease progress was observed to be influenced by rainfall and the movement of water over the soil and plastic mulch. Path coefficient analysis was conducted to determine which rainfall variables had relatively large, direct, or indirect effects on the incidence and the rate of disease progress without the confounding influences of multicollinearity. The cumulative amount of rainfall had the largest, absolute direct effect on disease progress and was a large component of the indirect effects of the other variables in three of the trials. A cumulative rain intensity index had the largest, absolute direct effect in one trial. The cumulative number of days with rainfall, the cumulative daily average temperature, and chronological time had far lesser effects, indicating their lack of influence on disease progress. The amount of rainfall also had the largest, direct effect on the rate of disease progress when calculated between disease assessment dates and was the largest component of the indirect effects of the other variables in all four trials. The other rainfall and temperature variables had relatively less influence on the rate of disease progress. The average rates of symptom expression were 0.14, 0.14, 0.20, and 0.27 per unit per centimeter of rainfall for the four trials.

Phytophthora blight of pepper (*Capsicum annuum* L.), caused by *Phytophthora capsici* Leonian (20), was first noted on peppers in Florida by Weber (40) during the 1930–1931 winter growing season in the Homestead area. Phytophthora blight has occurred sporadically in Florida and continues to be a serious threat to pepper production in south Florida. Heavy losses were reported during the 1982–1983 growing season when record-setting rainfall in south Florida was associated with a severe epidemic of *P. capsici* on several vegetable crops (5). Little, however, has been reported on the quantitative epidemiology of the disease.

Most species of *Phytophthora* require water for dispersal of sporangia and zoospores and for subsequent infection processes (11,12). Water has been reported to be an important factor in the dissemination of propagules of *Phytophthora* spp. (14,16,18, 19,29,31) and zoospores have been reported in surface water above infested soil (10,28,34,36). Rainfall, irrigation, and the cyclic nature of the soil water status in the field have been reported to be associated with disease development in pathosystems involving *Phytophthora* spp. (13,17,30,32,35,42). Ferrin and Mitchell (13) reported that cycles in the increase of mortality of tobacco plants infected with *P. parasitica* Dast. var. *nicotianae* (Breda De Haan) Tucker were more pronounced when the increases in mortality were expressed in terms of soil water status than chronological time. Schlub (33) reported that rainfall and soil moisture correlated better with disease incidence than did variables describing humidity, temperature, or calendar days in the *P. capsici*-pepper pathosystem. Water movement over the surface of the soil during periods of soil flooding was implicated in diseases caused by *P. capsici* (5,33).

The behavior of the interaction between the pathogen and host is favored, modified, or inhibited depending on the environment over time in which the interaction takes place (6,39). For diseases caused by *Phytophthora* spp., water is probably the most important aspect of the environment for disease development since most species require water in order to complete their life cycles (11,12). But selection of water variables to measure and their relationships to disease have depended on the viewpoints of the various investigators (5,13,17,33). The determination of which variables or measurements of rainfall best describe the effect of rainfall on disease progress has remained questionable. Most analyses of the effect of environmental variables on disease progress have employed multiple regression techniques, and the selection of environmental and meteorological variables to incorporate into a final multiple regression model is often accomplished by using stepwise model-building algorithms (6–8,17,30).

Weather patterns affect a range of variables with the result that many meteorological and environmental variables selected for analysis are highly correlated. The problem is, then, how to determine which variables exert a relatively large influence on disease while recognizing the effects of multicollinearity. Path coefficient analysis is one method whereby the direct effects of an independent variable on a dependent variable can be separated from the indirect effects of the independent variable on the dependent variable by virtue of being highly correlated with still another independent variable (9,15,21,37,41). The direct contribution of an independent variable to the variation observed in the dependent variable can be determined without the confounding influences caused by multicollinearity.

Path coefficient analysis, termed generally and herein as path analysis, was investigated as a method to identify rainfall and temperature variables having large, direct influences on the variation of the incidence and rate of disease progress of Phytophthora blight of pepper caused by *P. capsici*. The polycyclic nature of the pathosystem was also investigated with experimentation designed to follow the spread of disease from initial foci. Portions of this research have been previously reported (3,4).

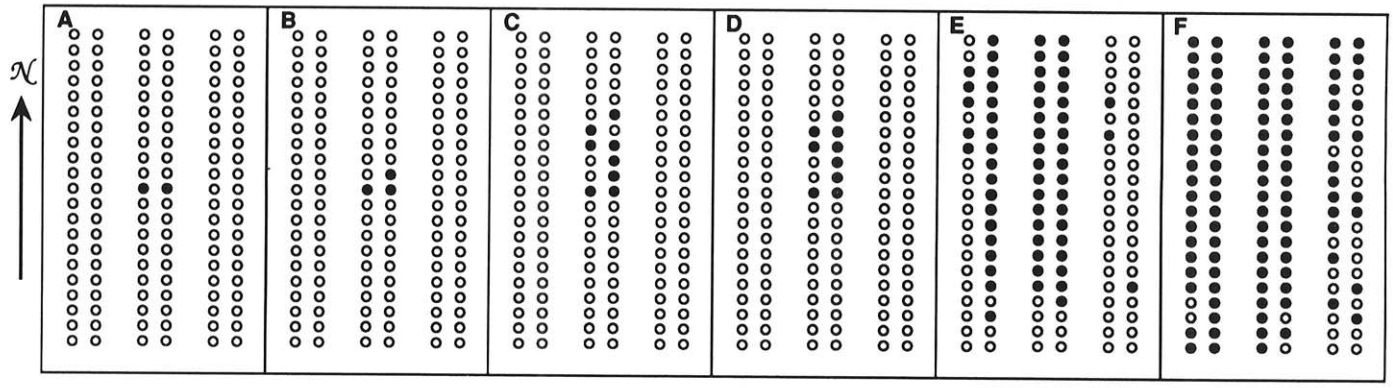

Fig. 1. Diagrammatic representation of spread of Phytophthora blight of pepper (caused by *Phytophthora capsici*) in a characteristic subplot from the spring 1984 trial; open circles represent healthy plants and darkened circles represent diseased plants: A, B, C, D, E, and F represent the distribution of diseased plants 0, 1, 2, 3, 6, and 7 wk, respectively, after the introduction of the two diseased plants, and the arrow indicates north.

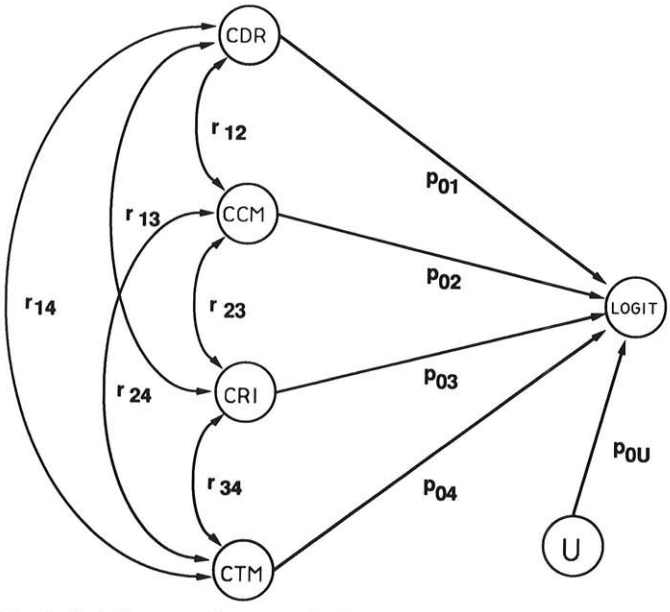

Fig. 2. Path diagram with paths of influence of cumulative environmental variables on Phytophthora blight of pepper (caused by *Phytophthora capsici*) disease progress (the logistic transformation of disease proportion, LOGIT); environmental variables consisted of cumulative number of days with rainfall (CDR), cumulative amount of rainfall measured in centimeters (CCM), cumulative values of a rain intensity index (CRI) calculated as the amount of rainfall divided by the number of days of rain between disease assessment dates and summed over time, the cumulative summation of the daily average temperature (CTM), and the residual (U); p_{ij} = path coefficients, r_{ij} = correlation coefficients.

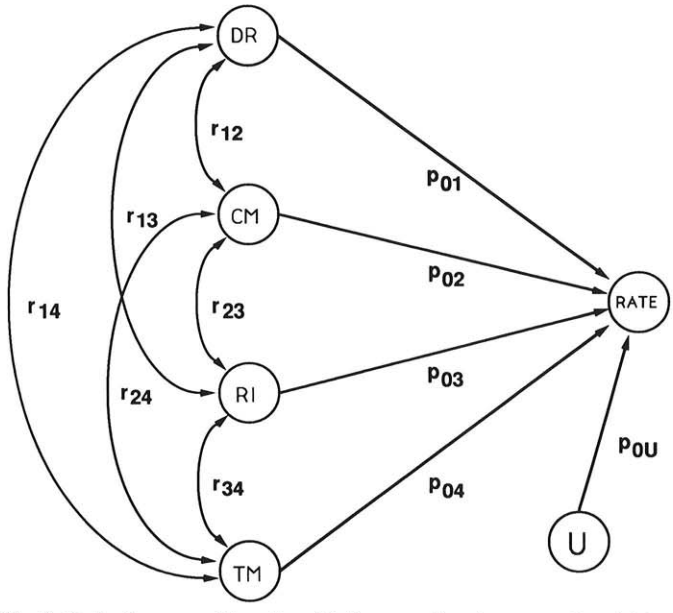

Fig. 3. Path diagram with paths of influence of environmental variables on the rate of change of the logistic transformation of Phytophthora blight of pepper (caused by *Phytophthora capsici*) disease proportion between assessment dates (RATE); environmental variables consisted of the number of days with rainfall between disease assessment dates (DR), the amount of rainfall measured in centimeters between disease assessment dates (CM), a rain intensity index calculated as the centimeters of rainfall divided by the number of days with rainfall between disease assessment dates (RI), the summation of the daily average temperature between disease assessment dates (TM), and the residual (U); p_{ij} = path coefficients, r_{ij} = correlation coefficients.

MATERIALS AND METHODS

Field plots were established in Delray Beach, Palm Beach County, FL, at the research farm of the Asgrow Seed Company in the spring and fall of 1984, the spring of 1985, and the fall of 1986. The experiment was conducted in two field plots that were separated by a drainage canal. The larger plot (A) measured approximately 0.078 ha and the smaller plot (B) measured 0.051 ha. The soil was a Myakka sand (sandy, siliceous, hyperthermic Aeric Haplaquod) (2) with an organic matter content of 0.95% and a pH of 6.6 in water (Soil Testing Lab and Analytical Research Lab, IFAS, University of Florida, Gainesville). Six-week-old pepper plants (*C. annuum* 'Early Calwonder') were transplanted into raised beds covered with white plastic mulch. The beds consisted of double rows spaced 0.47 m apart with 0.28 m between plants within a row. Bed centers were 1.83 m apart with approximately 0.6 m between beds. All plant locations were marked on a field map. The field plots were fumigated with 327 L of Vorlex (Nor-Am Chemical Co., Wilmington, DE) per hectare between trials. The field plots did not have a history of Phytophthora blight.

Six-week-old pepper plants were each inoculated in the greenhouse by adding 10^4 sporangia in 1 ml of water to a flooded planting mix containing one plant in a Styrofoam cup. Sporangia from six A1 and six A2 isolates of *P. capsici* from southern Florida were pooled and used as inoculum. The planting mix was kept flooded overnight and then drained. Seven to ten days later symptoms were present on all plants. The diseased pepper plants were transplanted to the plots 1 wk after the healthy plants had been transplanted, except in the trial conducted during the spring of 1984 when diseased plants were transplanted at the same time as the healthy plants. Two diseased plants were transplanted into the same transplant hole with a healthy plant to ensure the establishment of the initial disease focus.

In each plot, eight subplots consisting of three beds and 126 plants with 21 plants per row (Fig. 1A) were selected for the tests and an initial disease focus was established in the center of each subplot. In each plot, the subplots were treated as replicates. At weekly intervals the incidence of diseased plants, those showing symptoms of incipient wilt or the presence of a characteristic purplish-black lesion, was noted on the plot map.

Daily rainfall data were obtained from the Asgrow Research Farm from a weather station located adjacent to the field plots. Temperature data were obtained from the National Oceanic and Atmospheric Administration (NOAA) weather station at the West Palm Beach airport, which was located approximately 25 km north of the field plots (1). Partial temperature data obtained from the Asgrow Research Farm were found to be similar to the data published by NOAA. Therefore, the more complete data from NOAA were used in the analysis. Pathogen incubation periods of 1–7 days were assumed and the analysis conducted using a lag time of 1–7 days to identify correlations between rainfall and temperature data and new appearances of disease. The incubation period was defined as the time from ingress of the pathogen until symptom development. Infection was assumed to occur on each day with rainfall. For example, if the rain data were lagged for an incubation period of 4 days, then any rainfall event, with associated infection, occurring 1–3 days before a disease assessment day (t_i) would be included in the analysis for the next disease assessment day (t_{i+1}) since, by definition of an incubation period, symptom expression would not yet be observable.

The effects of rainfall and temperature variables on disease progress were studied using the methods of path analysis (21,41). Path analysis revolves around the path diagram (Fig. 2). The arrangement of the variables and the direction of the arrows in the path diagram are solely dependent on the causal relationships among the variables as envisioned by the investigator on theoretical or experimental grounds. The path diagram in Figure 2 is similar to a multiple regression model with several predictor variables and a dependent variable.

Path analysis is based on the decomposition of correlation coefficients into direct and indirect effects and the complete determination of a variable by other variables. The direct effect of one variable on another, indicated by a single-headed arrow, is measured by the path coefficient p_{0i}, which is simply a standardized, partial regression coefficient. The squared path coefficient gives the fraction of the variance of Y_0 (dependent variable) that can be accounted for by the variance of the X variable (independent variable), and it is a measure of the direct influence of the X variable on the variance of Y_0. Indirect effects are the effects of one X variable on the dependent variable through another X variable by virtue of the two X variables being correlated (r_{ij}). In the path diagram, correlations between variables are denoted by curved double-headed arrows. The residual, U, accounts for experimental error and variables not included in the model.

Two dependent variables were examined, the logistic transformation of disease proportion (y) [LOGIT = $\ln(y/(1-y))$] and the rate of change in logits between disease assessment dates (t)

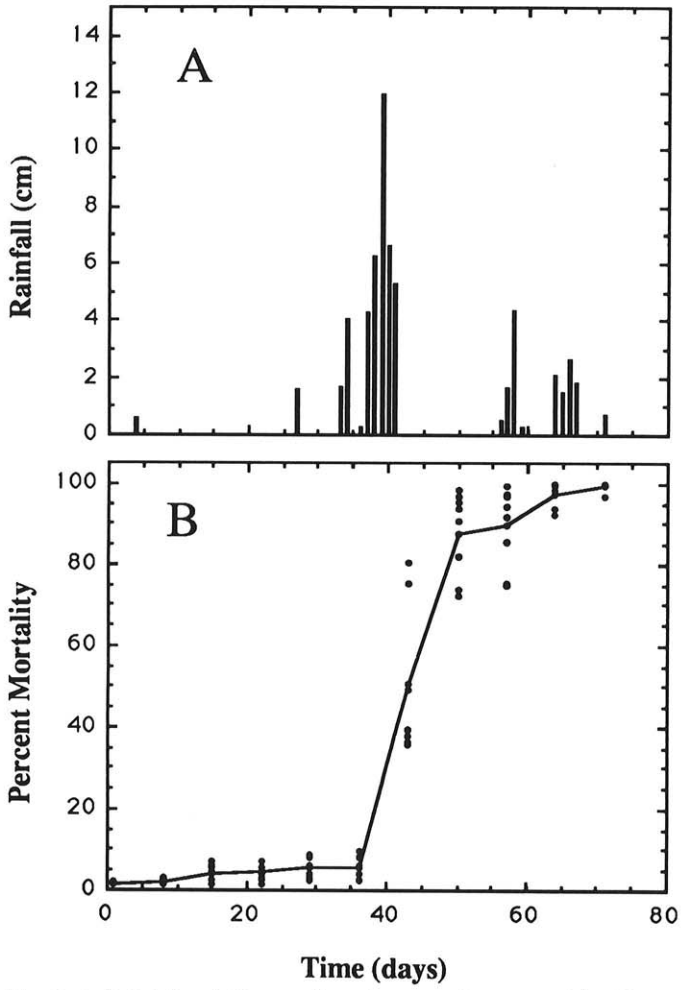

Fig. 4. A, Rainfall and, **B,** mortality of pepper plants caused by *Phytophthora capsici* over time during the field trial conducted in the spring of 1984 in Delray Beach, FL; each point represents the percentage of dead plants in each subplot.

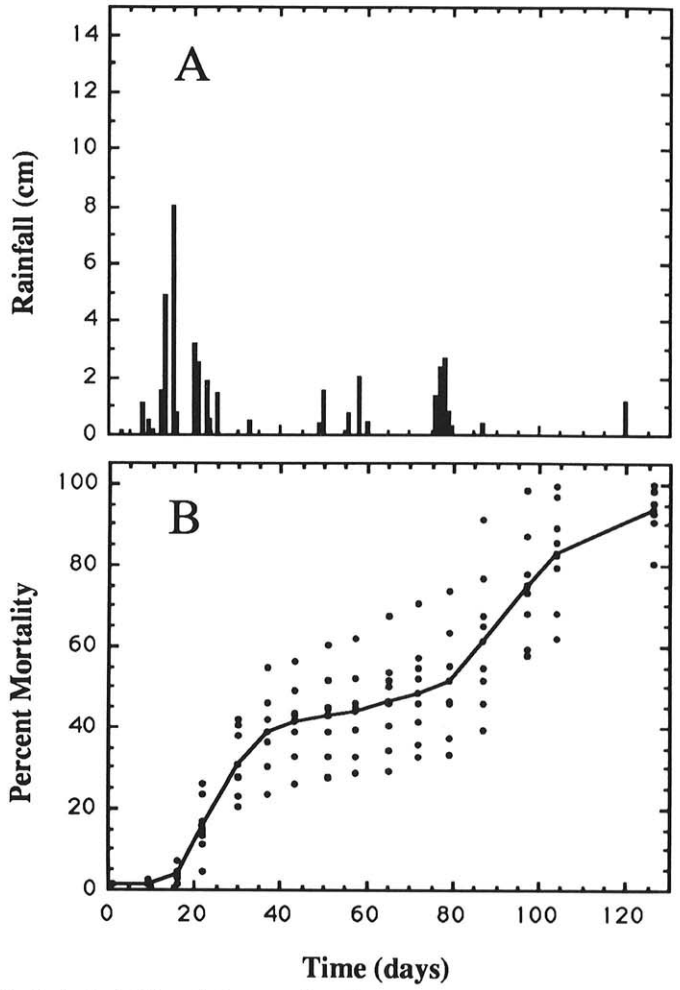

Fig. 5. A, Rainfall and, **B,** mortality of pepper plants caused by *Phytophthora capsici* over time during the field trial conducted in the fall of 1984 in Delray Beach, FL; each point represents the percentage of dead plants in each subplot.

[RATE = (LOGIT$_i$−LOGIT$_{i-1}$)/(t_i− t_{i-1})], in which the subscript refers to a given time. The independent variables used in the analysis are described as: 1) the number of days with rainfall between disease assessment dates (DR) and cumulative days with rainfall over the growing season (CDR); 2) the amount of rainfall measured in centimeters between disease assessment dates (CM) and cumulative rainfall (CCM); 3) a rain intensity index (RI) calculated as the centimeters of rainfall between disease assessment dates divided by the number of days of rainfall during the same time period and cumulative RI values (CRI); and 4) the daily average temperature (TM) calculated by averaging the minimum and maximum temperature each day and summing over the time between disease assessment dates and cumulative daily averages (CTM). The rate of rainfall, defined as the centimeters of rainfall divided by the number of days between disease assessment dates, and the frequency of rainfall, defined as the number of days with rainfall divided by the number of days between disease assessment dates, were exactly correlated with the amount of rainfall in centimeters and the number of days with rainfall, respectively, and were not used in the analysis. The number of calendar days between disease assessment dates was found to be exactly or almost exactly correlated with the daily average temperature and was not considered after preliminary analysis. Data from cumulative variables were used in the model with LOGIT as the response variable and data from individual time periods between disease assessment dates were used in the model with RATE as the response variable in the manner depicted in Figures 2 and 3, respectively. Separate analyses were run for each trial of the experiment.

RESULTS

The pattern of disease development in all trials indicated that the incidence of diseased plants spread outwardly from the primary foci and that *P. capsici* dispersed from the initially infected plants in a manner that indicated the plant-to-plant spread of inoculum and disease (Fig. 1). Generally, disease progressed down the row before bed-to-bed movement occurred. The direction of inoculum dispersal and disease progress was in the direction of the prevailing winds, which usually came out of the southeast.

Disease incidence was always greater than 80% at the conclusion of each trial, with many subplots having incidences of 100% (Figs. 4–7). Disease spread from the two infected plants that were introduced into each subplot to 100–124 other plants in the subplot. Runs analysis of the data from each subplot in each trial at various assessment dates indicated a general aggregation or clumping of diseased plants; this also implied plant-to-plant dispersal of inoculum and disease (13,24,35).

Increases in disease incidence were observed to be associated with rainfall in all trials of the experiment. Several consecutive days of heavy rainfall coincided with increases in disease incidence in the spring of 1984 (Fig. 4) and fall of 1984 (Fig. 5). Consecutive days with lesser amounts of rainfall were associated with increases in disease incidence in the spring of 1985 (Fig. 6), and two single days of heavy rainfall in the fall of 1986 (Fig. 7) coincided with the initiation of the epidemics. Trials of the experiment were conducted during different rainfall patterns in both timing and intensity. Rainfall patterns in the spring and fall 1984 consisted of clusters of several days with heavy rainfall. Rainfall patterns

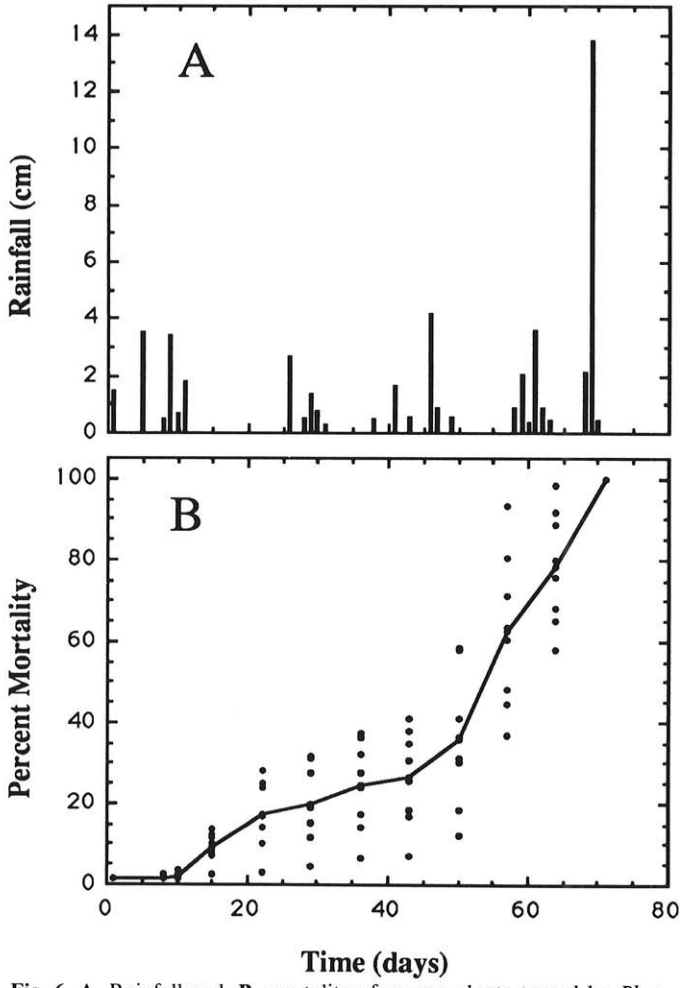

Fig. 6. A, Rainfall and, **B,** mortality of pepper plants caused by *Phytophthora capsici* over time during the field trial conducted in the spring of 1985 in Delray Beach, FL; each point represents the percentage of dead plants in each subplot.

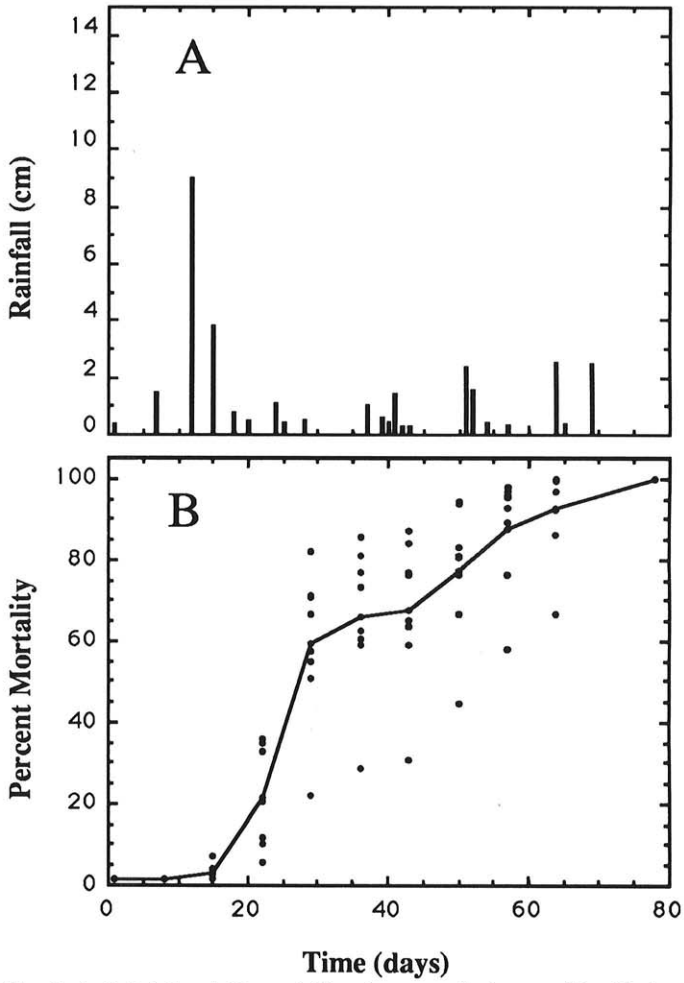

Fig. 7. A, Rainfall and, **B,** mortality of pepper plants caused by *Phytophthora capsici* over time during the field trial conducted in the fall of 1986 in Delray Beach, FL; each point represents the percentage of dead plants in each subplot.

in the spring of 1985 and fall of 1986 were generally distributed evenly over time with lesser amounts of rainfall per day and only a few days with heavy rainfall. Weather patterns during the experiments were subtropical with daily average temperatures ranging from 21 to 28 C with approximately 100% relative humidity every night. The weather during the trial in the fall of 1984 was relatively drier and cooler, especially during the latter stages of the epidemic; accordingly, the time required to achieve high levels of disease incidence was longer.

Preliminary analysis of the data attempted to construct multiple regression models to predict disease progress based on rainfall and temperature variables using stepwise regression techniques (J. H. Bowers and D. J. Mitchell, *unpublished*). Significant models were found, but they could not be validated using the model validation techniques of Coakley et al (8). Additionally, the models were poor predictors of disease incidence when tested against disease and rainfall data not included in the development of the models (often 20% over- or under-predicting disease incidence during the middle of the epidemic). Further analysis indicated that the independent variables in the various models often were highly correlated (Tables 1–4). The regression coefficients were not reflecting any inherent effect of an independent variable on disease progress, but only a marginal effect given whatever other correlated independent variable(s) was included in the model (25). The rain variable(s) affecting the progress of the epidemic could not be determined based on regression analyses using stepwise model-building techniques because of the confounding effect of multicollinearity, which resulted in the inability to validate the models and the poor predictive ability of the models.

Path analysis was used to determine which variable(s) had a relatively large direct and indirect effect or influence on disease progress (Figs. 2 and 3). Preliminary results indicated that a lag time of 5 or 6 days resulted in the highest coefficients of determination and are the data presented.

Correlation coefficients among disease progress data (LOGIT) and the cumulative environmental variables are presented in Tables 1 and 2. Correlation coefficients among the rate of disease progress (RATE) and environmental variables calculated between disease assessment dates are presented in Tables 3 and 4. Correlations were generally greater for the cumulative environmental variables and LOGIT than for the variables calculated between disease assessment dates and RATE, for which some correlation coefficients were not significant ($P > 0.05$). Graphic analysis indicated that low correlation coefficients were due to variation within the data and not to any deviation from linearity.

TABLE 1. Correlation coefficients among Phytophthora blight of pepper (caused by *Phytophthora capsici*) disease progress data and cumulative environmental variables for an experiment conducted in the spring of 1984[a] (upper right diagonal) and the fall of 1984[b] (lower left diagonal)

	LOGIT[c]	CDR[d]	CCM[e]	CRI[f]	CTM[g]
LOGIT	...	0.954[h]	0.958	0.954	0.904
CDR	0.896[h]	...	0.980	0.975	0.936
CCM	0.893	0.974	...	0.997	0.903
CRI	0.896	0.992	0.988	...	0.915
CTM	0.873	0.966	0.896	0.946	...

[a] Rainfall and temperature data were lagged to account for a pathogen incubation period of 5 days.
[b] Rainfall and temperature data were lagged to account for a pathogen incubation period of 6 days.
[c] The logistic transformation of disease incidence [$\ln(y/(1-y))$].
[d] Cumulative days of rain.
[e] Cumulative centimeters of rain.
[f] Cumulative rain intensity index calculated as the amount of rainfall divided by the number of days of rain between disease assessment dates and summed over time.
[g] Cumulative values of the daily average temperature.
[h] All correlations were significant at $P = 0.01$.

TABLE 2. Correlation coefficients among Phytophthora blight of pepper (caused by *Phytophthora capsici*) disease progress data and cumulative environmental variables for an experiment conducted in the spring of 1985[a] (upper right diagonal) and the fall of 1986[b] (lower left diagonal)

	LOGIT[c]	CDR[d]	CCM[e]	CRI[f]	CTM[g]
LOGIT	...	0.855[h]	0.901	0.911	0.879
CDR	0.877[h]	...	0.988	0.984	0.992
CCM	0.925	0.911	...	0.997	0.975
CRI	0.920	0.890	0.997	...	0.974
CTM	0.896	0.990	0.934	0.923	...

[a] Rainfall and temperature data were lagged to account for a pathogen incubation period of 6 days.
[b] Rainfall and temperature data were lagged to account for a pathogen incubation period of 5 days.
[c] The logistic transformation of disease incidence [$\ln(y/(1-y))$].
[d] Cumulative days of rain.
[e] Cumulative centimeters of rain.
[f] Cumulative rain intensity index calculated as the amount of rainfall divided by the number of days of rain between disease assessment dates and summed over time.
[g] Cumulative values of the daily average temperature.
[h] All correlations were significant at $P = 0.01$.

TABLE 3. Correlation coefficients among the rate of disease progress of Phytophthora blight of pepper (caused by *Phytophthora capsici*) and environmental variables calculate between disease assessment dates for an experiment conducted in the spring of 1984[a] (upper right diagonal) and the fall of 1984[b] (lower left diagonal)

	RATE[c]	DR[d]	CM[e]	RI[f]	TM[g]
RATE	...	0.865[h]	0.830	0.697	0.238
DR	0.531[i]	...	0.787	0.647	0.308
CM	0.685	0.828	...	0.957	0.199
RI	0.629	0.793	0.948	...	0.168
TM	0.056	0.031	−0.010	0.145	...

[a] Rainfall and temperature data were lagged to account for a pathogen incubation period of 5 days.
[b] Rainfall and temperature data were lagged to account for a pathogen incubation period of 6 days.
[c] The change in the logistic transformation of disease incidence [LOGIT, $\ln(y/(1-y))$] between assessment dates [$\text{LOGIT}_i - \text{LOGIT}_{i-1})/(t_i - t_{i-1})$].
[d] The number of days of rain between disease assessment dates.
[e] The amount of rain in centimeters between disease assessment dates.
[f] A rain intensity index calculated as the amount of rainfall divided by the number of days of rain between disease assessment dates.
[g] The daily average temperature between disease assessment dates.
[h] Values greater than 0.234 and 0.305 in absolute value are significant at $P = 0.05$ and 0.01, respectively.
[i] Values greater than 0.180 and 0.235 in absolute value are significant at $P = 0.01$, respectively.

TABLE 4. Correlation coefficients among the rate of disease progress of Phytophthora blight of pepper (caused by *Phytophthora capsici*) and environmental variables calculate between disease assessment dates for an experiment conducted in the spring of 1985[a] (upper right diagonal) and the fall of 1986[b] (lower left diagonal)

	RATE[c]	DR[d]	CM[e]	RI[f]	TM[g]
RATE	...	0.308[h]	0.579	0.559	−0.009
DR	0.261[h]	...	0.868	0.715	0.360
CM	0.670	0.123	...	0.890	0.130
RI	0.610	−0.089	0.975	...	−0.057
TM	0.278	0.247	0.193	0.196	...

[a] Rainfall and temperature data were lagged to account for a pathogen incubation period of 6 days.
[b] Rainfall and temperature data were lagged to account for a pathogen incubation period of 5 days.
[c] The change in the logistic transformation of disease incidence [LOGIT, $\ln(y/(1-y))$] between assessment dates [$\text{LOGIT}_i - \text{LOGIT}_{i-1})/(t_i - t_{i-1})$].
[d] The number of days of rain between disease assessment dates.
[e] The amount of rain in centimeters between disease assessment dates.
[f] A rain intensity index calculated as the amount of rainfall divided by the number of days of rain between disease assessment dates.
[g] The daily average temperature between disease assessment dates.
[h] Values greater than 0.234 and 0.305 in absolute value are significant at $P = 0.05$ and 0.01, respectively.

Because of possible serial correlation in the dependent variable LOGIT, the correlations of LOGIT and the independent variables may appear to be higher than what they actually are and the significance levels may appear to be too low. However, analysis in which the dependent variable LOGIT and the independent variables were corrected for serial correlation using the first difference method (23) indicated that, although the actual values in the path analysis differed slightly, the interpretation of the results did not change. Therefore, the results are presented uncorrected for serial correlation. The analyses with the dependent variable RATE avoids any problems with serial correlation.

The results of path analysis for the effects of the cumulative environmental variables on LOGIT are presented in Table 5 for the four trials. Within the trials conducted in the spring of 1984, fall of 1984, and fall of 1986, the cumulative amount of rainfall in centimeters (CCM) had the highest direct effect on disease progress (p_{02} = 1.874, 2.127, and 4.619, respectively). The cumulative values of the rain intensity index (CRI) had the highest direct effect on disease progress in the spring of 1985 (p_{03} = 2.207). In each trial, CCM and CRI had relatively large, but opposing, influences on disease progress, as evidenced by their different signs, in both direct and indirect effects. The analysis indicated that CCM influenced disease progress more than CRI in three of the four trials based on larger, absolute direct effects, and that CRI was found to have larger, absolute, indirect effects via CCM than its direct effect on disease progress. The opposite situation occurred in the spring 1985 trial. The cumulative rain intensity index (CRI) had a larger, absolute direct effect than CCM and that CCM had a larger, absolute indirect effect via CRI than its direct effect on disease progress.

The cumulative number of days with rainfall (CDR) and the cumulative daily average temperature (CTM) had far lesser effects than the other variables on disease progress in all trials (Table 5). Direct effects were sometimes less than the residuals, which measure the experimental error and the effect of variables not included in the analysis. The high correlations between CDR and LOGIT and CTM and LOGIT were due to the fact that CDR and CTM were highly correlated with CCM and CRI, as evidenced by larger indirect effects via CCM and CRI than direct effects. Dropping CDR and CTM from the analysis did not change the results to any extent and resulted in only slightly lower coefficients

TABLE 5. Path coefficient analysis of the relationships between Phytophthora blight of pepper (caused by *Phytophthora capsici*) disease progress data and cumulative environmental variables

Pathways of association	Spring 1984[a]	Fall 1984[b]	Spring 1985[b]	Fall 1986[a]
CDR[d] vs. LOGIT[c]				
Direct effects, p_{01}	−0.079	−0.865	−0.071	−1.912
Indirect effects				
via CCM[e]	1.836	2.072	−1.189	4.209
via CRI[f]	−1.105	−1.521	2.172	−3.460
via CTM[g]	0.301	1.210	−0.026	2.040
Total correlation	0.954[h]	0.896	0.885	0.877
CCM vs. LOGIT				
Direct effects, p_{02}	1.874	2.127	−1.203	4.619
Indirect effects				
via CDR	−0.077	−0.842	−0.070	−1.743
via CRI	−1.130	−1.515	2.201	−3.877
via CTM	−0.290	1.123	−0.026	1.925
Total correlation	0.958	0.893	0.901	0.925
CRI vs. LOGIT				
Direct effects, p_{03}	−1.133	−1.534	2.207	−3.887
Indirect effects				
via CDR	−0.077	−0.858	−0.070	−1.702
via CCM	1.870	2.102	−1.200	4.607
via CTM	−0.294	1.185	−0.026	1.902
Total correlation	0.954	0.896	0.911	0.920
CTM vs. LOGIT				
Direct effects, p_{04}	0.321	1.253	−0.027	2.061
Indirect effects				
via CDR	−0.074	−0.835	−0.071	−1.893
via CCM	1.692	1.907	−1.173	4.314
via CRI	−1.036	−1.451	2.149	−3.587
Total correlation	0.904	0.873	0.879	0.896
Residual, p_{0U}	0.265	0.394	0.402	0.364
Coefficient of determination	0.929	0.845	0.839	0.868

[a] Rainfall and temperature data were lagged to account for a pathogen incubation period of 5 days.
[b] Rainfall and temperature data were lagged to account for a pathogen incubation period of 6 days.
[c] The logistic transformation of disease incidence [$\ln(y/(1-y))$].
[d] Cumulative days of rain.
[e] Cumulative centimeters of rain.
[f] Cumulative rain intensity index calculated as the amount of rainfall divided by the number of days of rain between disease assessment dates and summed over time.
[g] Cumulative values of the daily average temperature.
[h] All correlations were significant at $P = 0.01$.

TABLE 6. Path coefficient analysis of the relationships between the rate of disease progress of Phytophthora blight of pepper (caused by *Phytophthora capsici*) between assessment dates and environmental variables

Pathways of association	Spring 1984[a]	Fall 1984[b]	Spring 1985[b]	Fall 1986[a]
DR[d] vs. RATE[c]				
Direct effects, p_{01}	0.361	−0.120	−0.929	−0.890
Indirect effects				
via CM[e]	0.951	0.969	1.114	0.641
via RI[f]	−0.448	−0.322	0.064	0.412
via TM[g]	0.001	0.004	0.058	0.098
Total correlation	0.865[h]	0.531[i]	0.308[h]	0.261[h]
CM vs. RATE				
Direct effects, p_{02}	1.209	1.170	1.284	5.206
Indirect effects				
via DR	0.284	−0.099	−0.806	−0.110
via RI	−0.663	−0.385	0.080	−4.503
via TM	0.001	−0.001	0.021	0.077
Total correlation	0.830	0.685	0.579	0.670
RI vs. RATE				
Direct effects, p_{03}	−0.693	−0.406	0.090	−4.620
Indirect effects				
via DR	0.234	−0.095	−0.664	0.079
via CM	1.156	1.109	1.142	5.073
via TM	0.001	0.019	−0.009	0.078
Total correlation	0.697	0.627	0.559	0.610
TM vs. RATE				
Direct effects, p_{04}	0.002	0.130	0.162	0.396
Indirect effects				
via DR	0.111	−0.004	−0.334	−0.220
via CM	0.241	−0.012	0.167	1.006
via RI	−0.117	−0.059	−0.005	−0.905
Total correlation	0.238	0.056	−0.009	0.278
Residual, p_{0U}	0.408	0.714	0.702	0.671
Coefficient of determination	0.833	0.491	0.507	0.549

[a] Rainfall and temperature data were lagged to account for a pathogen incubation period of 5 days.
[b] Rainfall and temperature data were lagged to account for a pathogen incubation period of 6 days.
[c] The change in the logistic transformation of disease incidence [LOGIT, $\ln(y/(1-y))$] between assessment dates [$\text{LOGIT}_i - \text{LOGIT}_{i-1})/(t_i - t_{i-1})$].
[d] The number of days of rain between disease assessment dates.
[e] The amount of rain in centimeters between disease assessment dates.
[f] A rain intensity index calculated as the amount of rainfall divided by the number of days of rain between disease assessment dates.
[g] The daily average temperature between disease assessment dates.
[h] Correlation coefficients within a column greater than 0.234 and 0.305 in absolute value are significant at $P = 0.05$ and 0.01, respectively.
[i] Correlation coefficients within a column greater than 0.180 and 0.235 in absolute value are significant at $P = 0.05$ and 0.01, respectively.

of determination, thus indicating their lack of influence on disease progress. The variables in the model explained 92.9, 84.5, 83.9, and 86.8% of the variation observed in disease progress for the four trials (Table 5).

Similar results were obtained when the effects of environmental variables on the rate of disease progress between assessment dates were analyzed (Table 6). The amount of rainfall in centimeters between disease assessment dates (CM) had the largest direct effect on RATE in each trial. The other variables had lesser direct effects and larger indirect effects via CM than direct effects, even though significant correlations existed between the environmental variables and RATE. The model for the spring of 1984 explained 83.3% of the variation in the rate of disease progress between assessment dates. The model for the other three trials, however, only explained 49.1–54.9% of the variation in the rate of disease progress between assessment dates. Other factors not included in the model accounted for 45.1–50.9% of the variation.

The average rates of symptom expression with a time lag of 5 or 6 days, as calculated from the logistic transformation of disease proportion, were 0.13, 0.05, 0.08, and 0.12 per unit per day and 0.14, 0.14, 0.20, and 0.27 per unit per centimeter of rainfall for the four trials.

DISCUSSION

Phytophthora blight of pepper was shown to be a polycyclic disease in an experiment specifically designed to follow the spread of disease from a central focus (26,38). Disease spread from two infected plants transplanted to the center of each subplot to 100–124 other plants in the subplot (Fig. 1). Shew (35) and Pfender and Hagedorn (27) also demonstrated plant-to-plant spread of disease with experimentation designed to follow disease development from foci of diseased plants in stands of healthy plants in previously noninfested fields. MacKenzie et al (22) concluded that several diseases caused by species of *Phytophthora* are accommodated by the compound continuous interest epidemic model as described by Vanderplank (38). Although this model is consistent with polycyclic epidemics, Pfender (26) cautioned that the nature of the disease cycle must be determined before an appropriate model can be chosen.

In the present study, the only detectable initial inoculum present in the field at the beginning of each trial was associated with the transplanted diseased plants. The soil was fumigated before each trial, and diseased plants not visually associated with the initial foci of diseased transplants were not observed in any trial; this was true even for plants around the edges of the field where the fumigation might not have been expected to be fully effective. Midway through the various trials, the transplanted diseased plants that served as the initial inoculum source were completely dried and necrotic, and they were not even present in some subplots. These plants were no longer contributing to the epidemic, yet disease progress continued via the production of secondary inoculum on subsequently infected plants. Masses of sporangia that were observed on infected fruit late in the season visually indicated the production of secondary inoculum on infected plants.

Initial disease spread in the present study was within the same rows as the diseased transplants. However, disease spread was not always serially down the row; sometimes two or three plants between diseased plants showed no symptoms. However, plants between diseased plants were almost always diseased at the next assessment date. Runs analysis of the data always indicated a clumping of diseased plants supportive of plant-to-plant spread. Shew (35) observed asymptomatic tobacco plants between symptomatic plants at the conclusion of his experiments; however, roots from asymptomatic plants plated on a selective medium were infected with *P. p. nicotianae*, indicating that the dispersal of the pathogen was continuous down the row from the initially infected plant. This mode of dispersal may have occurred in the present study. Ferrin and Mitchell (13) also presented evidence for the increase and dispersal of secondary inoculum of *P. p. nicotianae* based on the temporal change in the nonrandom pattern of plant mortality.

Transitory inoculum dispersed in wind-driven rain and running water was hypothesized to account for the pattern of disease observed in the present study, but no direct quantitative data supporting this hypothesis was obtained. Nevertheless, after several days of rain in one trial, lesions were observed on those plant parts that were lying in water on the top of the plastic mulch. This implied that inoculum was carried in moving water along the surface of the plastic mulch. The data are thus consistent with the hypothesis of plant-to-plant spread and the polycyclic nature of the disease.

Path analysis indicated that, even though the four field trials were conducted under different patterns of rainfall with different frequencies and intensities of rainfall, the amount of rainfall, measured in centimeters, had a greater direct effect on disease progress in three trials and the rate of disease progress in all trials than did the other variables. Although the number of days with rainfall was highly correlated with disease progress, its direct influence on the variation of disease progress was relatively small. These relationships would not be evident based only on the analysis of correlation coefficients. These results confirm observations from the field that indicated that the amount of rainfall, with associated flooding, is of critical importance for an increase in disease development by providing a vehicle for inoculum dispersal. More days of rain, but in lesser amounts that do not result in surface water accumulation, may not be as important to disease progress as are the number of days with heavy rainfall. Thus, further research should focus on the amount of rainfall in a given time frame. This also has important implications for disease management. Irrigation, if necessary, should be kept to a minimum or at a low rate with frequent, light applications, as opposed to heavy applications that promote flooding and surface water accumulation.

The amount of rainfall and the rain intensity index were found to have opposing influences on disease development in all trials, even though these variables were significantly correlated ($P < 0.01$) with disease progress and the rate of disease progress. A negative effect may not seem biologically correct in this system, but it can be explained by the analogy that while the coefficient of determination must be positive, certain of its components may be negative (21). This situation occurs only when both variables are included in the model. Since each has a large, direct effect by itself, the negative contribution to the determination of disease progress and the rate of disease progress may be due to the high correlation ($P < 0.01$) between the rainfall variables themselves. The negative sign disappears when one or the other variable is removed from the model. Interestingly, the coefficient of determination is only reduced slightly when either of these variables are removed, also indicating their large, but opposing influences on disease progress. Consequently, both variables should not appear in further analyses. The cumulative amount of rainfall in centimeters (CCM) should be used in subsequent analysis because it had a large, positive effect on disease progress in three of four trials and it can be measured directly.

Disease incidence was assessed weekly in the present study and the data were lagged to account for various pathogen incubation periods based on weekly time frames. A time lag of 5 or 6 days resulted in the highest coefficients of determination. However, this represents just the optimum observed in this study. A time lag of 2–7 days also resulted in acceptably high coefficients of determination. This is probably the result of variable disease development in the field, which has not been studied in this pathosystem, and the fact that individual rain events were not segregated in the analysis. The actual time infection occurred was not known because the actual time during the day that the rain event occurred was not evident in the manner the data were acquired. The rain data were received from the Asgrow staff as only the amount of rain during a 24-hr period. The number of rain events, the intensity of rain, and the duration of each rain event were not known. To further characterize the effect of the environment on disease progress, further studies should include data accumulation for individual rain events, the amount of rain-

fall, the intensity of rainfall, and the duration of each rain event, as well as leaf wetness duration, relative humidity, and soil-water matric potential. Disease assessment should be made daily and the data analyzed to determine the conditions necessary for infection, symptom development, the production of secondary inoculum, and the incubation and latent periods of the pathogen as they occur in the field. Attempts should also be made to document the dispersal of inoculum in the field.

Path analysis uncovers relationships that would otherwise remain hidden based on the analysis of correlation coefficients. Correlation models do not express any causal relationships, but they can be useful in analyzing causal relationships (25). Not only must the correlation between an independent variable and the dependent variable be examined, but the correlations among all the independent variables must also be examined. Path analysis will be useful to determine which variables exert a large, direct effect on disease development and, therefore, the variables to include in further studies. The analysis should prove to be a very useful tool in the analysis of epidemiological data.

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