Influence of Assessment Time and Modeling Approach on the Relationship Between Temperature–Leaf Wetness Periods and Disease Parameters of *Septoria glycines* on Soybeans

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ABSTRACT

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The disease severity and number of lesions of *Septoria glycines* were significantly influenced by temperature and length of leaf wetness period. In general, disease severity increased with increasing leaf wetness periods from 6 to 36 h at all assessment dates. The optimum temperature was 25 C, but disease symptoms were also observed at 15, 20, and 30 C. Similar trends were found for the number of lesions per square centimeter. Infection of soybean leaves by *S. glycines* led to premature senescence even at low disease severity. No disease threshold for early senescence could be determined, because leaf senescence was influenced by the location of the lesions on the leaf in addition to disease severity. Results from this study should be useful in determining favorable infection conditions in the field. The interpretation of the data sets for disease management or prediction was strongly influenced by the assessment date (7, 14, and 21 days after inoculation), and to a lesser degree by the choice of modeling approach (linear and nonlinear regression analysis). The number of lesions per square centimeter could only be modeled for data obtained on day 7. The inability to model the relationship at the later assessment dates was due to a decrease in lesion numbers caused by lesion merger at optimal temperature–dew period conditions and a further increase in lesion numbers at nonoptimal conditions. This was independent of the modeling approach. Disease severity was modeled successfully at days 14 and 21 by linear regression analysis, and at day 14 by nonlinear regression analysis. In general, both models described the data satisfactorily as determined through R^2 values (>0.85). Implications for disease management are discussed.

Brown spot of soybeans (*Glycine max* (L.) Merr.), which is caused by *Septoria glycines* Hemmi, is a foliar disease widely distributed throughout the soybean growing areas of the world (3,14,17). The primary inoculum originates from pycnidia that overwinter on plant debris in the field. The disease severity increases most rapidly during warm, moist weather and from lower to upper leaves (7). Conidia are spread primarily through splash dispersal during rain. Moderate to severe infection causes premature defoliation. Yield reductions from artificial and natural inoculations range from 12 to 34% and from 8 to 10%, respectively (7,9,10,16,19).

Resistance in soybeans to *S. glycines* has not been detected so far. Of approximately 3,600 introduction lines, all appeared susceptible when assessments were made late in the season (6,18). In the absence of host resistance, benomyl has been successfully used to control brown spot (9). The efficacy of benomyl applications was influenced by, among other factors, environmental ones favoring infection by the pathogen (1). The quantification of temperature and leaf wetness effects on infection is an important element of disease-management strategies in many host–pathogen systems. The infection levels predicted by such quantitative models are influenced, among other things, by the choice of data set used to develop the models (i.e., the time of variable assessment), as well as by the choice of modeling approach (linear vs. nonlinear models). This study, therefore, was designed to determine the quantitative relationship between temperature–leaf wetness period and infection of soybeans by *S. glycines,* and to investigate the influence of assessment time and modeling approach on the predicted infection levels.

MATERIALS AND METHODS

Plant production. Two seeds of the soybean cultivar Pella were planted in a mixture of steam disinfested sand–peat–loam (1:2:2,v/v/v) in 473-ml plastic containers. Plants were maintained on greenhouse benches, watered with deionized water, and fertilized biweekly with Peter's 20-20-20 (N-P-K) fertilizer. Sunlight was supplemented 12 h per day with 1,000-W high-pressure sodium lamps. Approximately 3 wk after planting, plants were thinned to one per pot to ensure uniformity. Greenhouse temperatures, as monitored by a hygrothermograph, ranged from 20 to 25 C with a relative humidity of 20–60%. Plants were maintained in a greenhouse until the second trifoliate was fully developed.

Inoculum production and culture maintenance. A population of *S. glycines* was obtained from infected leaf tissue collected at the Russell E. Larson Agricultural Research Center of Pennsylvania State University at Rock Springs. The fungus was maintained on potato-dextrose agar. Five days before inoculation, 14-day-old fungal colonies were macerated with the adhering agar substrate in a Waring blender. The resulting slurry was spread onto potato-dextrose agar in petri dishes. Plates were incubated for 5 days at 25 C under 12 h light/12 h dark. The light source was provided by two 20-W cool-white fluorescent lights 15 cm above the plates. This treatment resulted in the production of numerous pycnidia and conidia. After four consecutive transfers, new fungal material was obtained from the original culture from liquid nitrogen storage.

Inoculation. Fungal cultures were flooded with approximately 20 ml of distilled water containing 0.05% Tween 20. The surface of the colonies was gently rubbed with a camel hair brush to dislodge conidia. The suspension mixture was then strained through a double layer of cheesecloth to remove large particles, and adjusted to 125,000 conidia per milliliter with a hemacytometer. The second fully developed soybean leaves were inoculated with a Badger airbrush (Badger Air Brush Co., Franklin Park, IL) at 103 kPa. The target leaves were sprayed on the lower surface for 2 s from a distance of 15 cm. Leaves were allowed to dry before the start of the treatment.

Disease assessment. The number of lesions and the percent disease severity were assessed 7, 14, 21, and 28 days after inoculation. Prior to the first assessment, leaf area was measured nondestructively with a portable Li-Cor LI-3000 leaf area meter (Li-Cor, Inc., Lincoln, NE). Lesions per square centimeter (LSQ) were computed by dividing the total number of lesions per leaf

by the leaf area. Disease severity was assessed by superimposing each leaf with a clear plastic grid which was subdivided into small squares, each with an area of 4 mm^2. Disease severity was computed by dividing the number of squares times 0.04 by the leaf area. At the third and fourth assessment dates, the number of senescent leaves was determined. Four plants of identical age, subjected to the same treatments without inoculation, were used to obtain rates of senescence in the absence of fungal infection.

Experimental design. After inoculation, plants were placed in dew chambers (Percival Mfg. Co., Boone, IA). Plants were subjected to temperatures of 15, 20, 25, or 30 C and leaf wetness durations of 6, 12, 18, 24, and 36 h in all possible combinations. Five soybean plants were used per leaf wetness–temperature combination. The entire experiment was conducted four times. The sequence of treatments and the assignment of dew chambers within each treatment was random. Different temperatures required unequal time periods for the formation of dew on the leaves. Therefore, a misting system was engaged for 15 min after the beginning of the treatment to ensure rapid and uniform leaf wetness.

Model development. Both multiple regression and the Richards model were used to model the influence of temperature and leaf wetness period on the three variables. Models were evaluated based on the following criteria: 1) analysis of residuals, 2) significance of estimated parameters, 3) R^2 values, and 4) R^{*2} values between observed and the estimated back transformed values.

The polynomial regression model used in analysis was of the form

$$Y = f(W, T) \tag{1}$$

where Y is the arcsine-transformed disease severity or the log-transformed lesion count, and $f(W, T)$ is a linear function of temperature (T) and leaf wetness duration (W). The model was developed by using all possible combinations of temperature and leaf wetness period. The analysis was performed using the SAS PROC GLM (13) with the MAXR option. An F test (8) indicated no significant differences among the four experimental runs. The data were therefore combined for the analysis.

The procedure used for model development based on the Richards function of the form

$$Y = k(1 + e^{-rW})^{1/(1-m)} \tag{2}$$

where Y is the number of lesions per square centimeter or disease severity (%), k is the maximum value of these variables at a given temperature, r is the rate parameter, W is the duration of leaf wetness, and m is the shape parameter, was similar to the procedure described by Lalancette et al (5). The first step consisted of modeling the upper asymptote of disease severity–lesions per square centimeter (k) as a function of temperature (T) through the use of second- or third-order polynomial equations, as follows:

$$k = b_0 + b_1 T + b_2 T^2 + b_3 T^3 \tag{3}$$

The b's are regression coefficients. Regression analysis was performed on the full and reduced models.

In the second step, the linearized, nonintercept version of the form

$$\ln[(y/k)^{1-m} - 1] = -r^* W \tag{4}$$

was fitted to the data for each temperature separately. The minimum wetness duration for infection (4 h) was subtracted from the dew period so that the curves would begin at the origin. A range of m values was used to achieve the best possible fit.

In the third step, the rate parameter estimates were regressed against a cubic function of temperature of the form

$$r = b_0 + b_1 T + b_2 T^2 + b_3 T^3 \tag{5}$$

Only the pooled data were used for the analysis in this step.

The final step consisted of substituting the rate parameter function (equation 5) for r in the Richards model:

$$\ln(y/k)^{1-m} - 1 = b_1 W + b_2 \times W \times T + b_3 W \times T^2 + b_4 W \times T^3 \tag{6}$$

The function coefficients were estimated for the pooled data and the separate replicates. A standard F test was used to determine if the data could be pooled. Since the predicted upper asymptote of y, k was lower than the observed asymptote in some cases, a fixed value was added to avoid the logarithm of a negative number.

Disease development over time. The disease development over time was modeled by fitting the linearized version of the logistic equation for each temperature–leaf wetness combination separately. The equation had the form

$$\ln Y/(0.16 - Y) = b_0 + r_L T \tag{7}$$

where 0.16 is the upper asymptote of disease severity (Y), b_0 is the intercept, T is the time in days since inoculation, and r_L is the infection rate based on the logistic model. Only observations

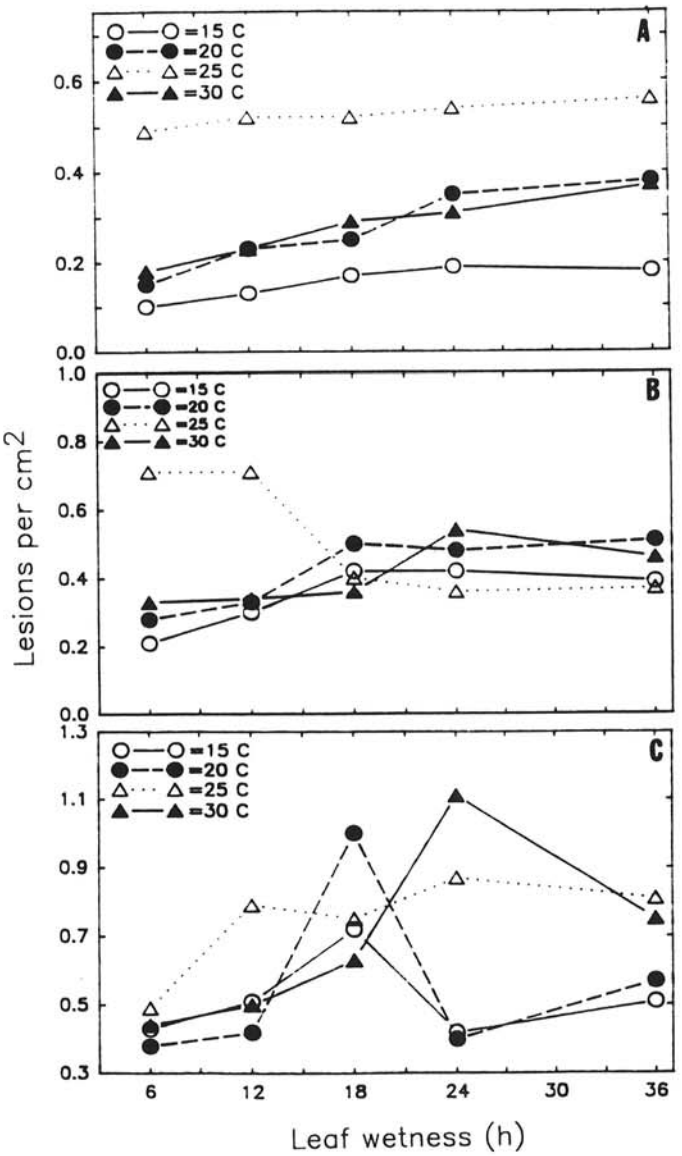

Fig. 1. Number of lesions per square centimeter of *Septoria glycines* as a function of leaf wetness period for temperatures from 15 to 30 C. Each point is an average of observations made on 20 plants (4 experimental replications, 5 plants per replication). Assessment times: **A** = 7 days, **B** = 14 days, and **C** = 21 days.

from days 7, 14, and 21 were used, because most leaves were desiccated at day 28. Regression models were developed that relate the estimated r_L value to the temperature and leaf wetness conditions during inoculation. Models were tested for temperature within each leaf wetness period and vice versa.

Comparison of the percentage of senescent leaves. The percentage of senescent leaves was compared between inoculated and noninoculated control plants. The comparisons were based on data obtained at day 28.

RESULTS

The number of lesions per square centimeter was influenced by temperature and leaf wetness period at the first assessment date (Fig. 1A). In general, the number of lesions increased with increasing leaf wetness periods independent of temperature. The optimum temperature was 25 C, followed by 20 and 30 C. The least favorable temperature was 15 C. At assessment dates 14 (Fig. 1B) and 21 (Fig. 1C), the situation was less clear. Lesion number seemed to increase until 18 or 24 h of leaf wetness, then decrease. At 25 C, the number of lesions decreased after 12 h of leaf wetness. At assessment date 21, no trend in regard to temperature or leaf wetness was discerned.

Disease severity was influenced by temperature and leaf wetness period. The disease severities at day 7 were generally low (maximum disease severity = 2.3% at 25 C and 36 h of leaf wetness). At day 14, disease severity increased with increasing leaf wetness periods (Fig. 2A). The strongest influence of leaf wetness period was observed at 25 C. In general, 25 C was more favorable than 30 C, followed by 20 and 15 C, independent of leaf wetness period. At the third assessment date, the trends were similar to the ones observed at day 14. Disease severities were about twice as high (Fig. 2B) and increased with increasing leaf wetness periods. The ranking of temperatures from most to least favorable was 25, 30, 20, and 15 C.

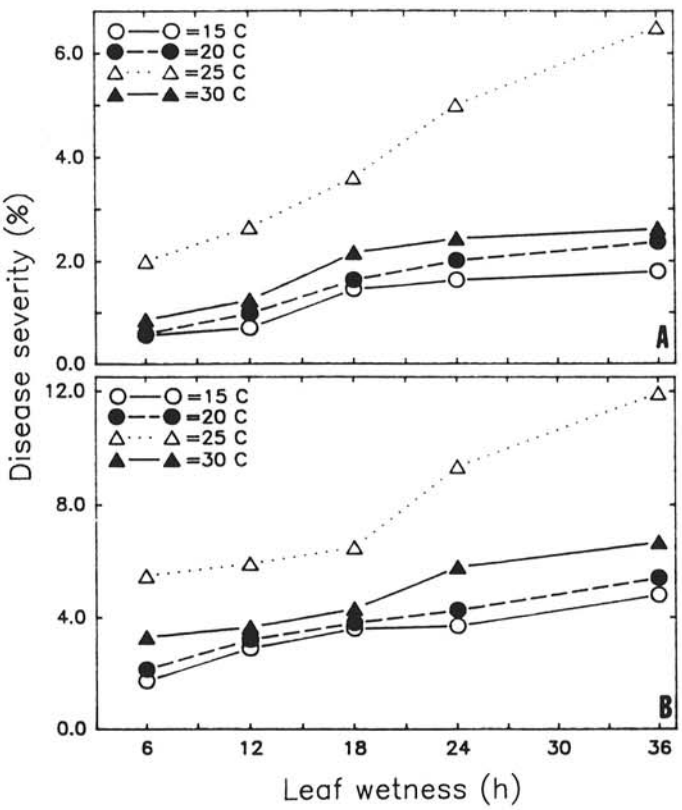

Fig. 2. Disease severity (% leaf coverage) of *Septoria glycines* as a function of leaf wetness period for temperatures from 15 to 30 C. Each point is an average of observations made on 20 plants (4 experimental replications, 5 plants per replication). Assessment times: **A** = 14 days, **B** = 21 days.

Model development. *Linear regression models.* Day 7: The influence of temperature and leaf wetness period on the number of lesions per square centimeter (LSQ) could be modeled for the first assessment date only. Polynomial regression resulted in the equation of the form

$$Y = 12.269 - 2.379^{*}T + 0.124^{*}T^{2} - 0.002^{*}T^{3} + 0.019^{*}W \quad (8)$$

where Y is the log-transformed LSQ, T is the temperature in Celsius, and W is the leaf wetness period in h. All coefficients were significant at $P = 0.001$. There was no significant ($P = 0.05$) difference among the replications based on an F test of

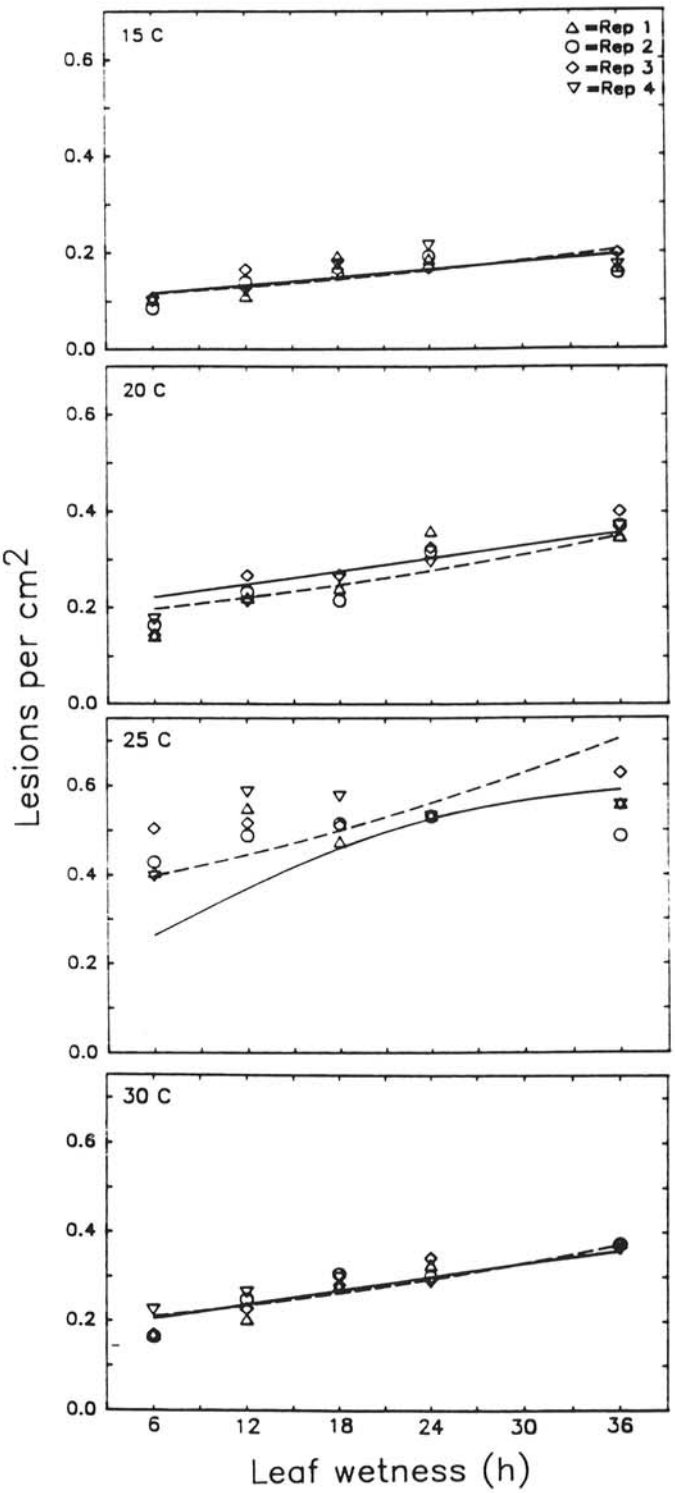

Fig. 3. Prediction of the number of lesions per square centimeter (assessment day 7) of *Septoria glycines* for various temperatures and leaf wetness durations. Dashed line is regression model, solid line is Richards model.

full and reduced models. The data from the four runs were therefore combined. The equation indicated a complex relationship between Y and temperature (linear, quadratic, and cubic terms). The relationship of Y with leaf wetness period was linear. The influence of leaf wetness period, although highly significant, is small compared to temperature. An interaction term between the two parameters was nonsignificant ($P = 0.05$). The equation had an R^2 value of 0.90 and an R^{*2} value 0.94. The model described the observed disease severities closely at 15, 20, and 30 C (Fig. 3). At 25 C, the model tended to underestimate at 6 and 12 h of leaf wetness and overestimate at 36 h. Based on the shape of the curves, LSQ generally increased with increasing leaf wetness periods.

Day 14: The influence of temperature and leaf wetness period on disease severity (assessment day 14) was best described by a polynomial regression model of the form

$$Y = 2.523 - 0.369^{*}T + 0.017^{*}T^{2} - 0.000274^{*}T^{3} + 0.000256^{*}T^{*}W - 0.000003^{*}T^{*}W^{2} \quad (9)$$

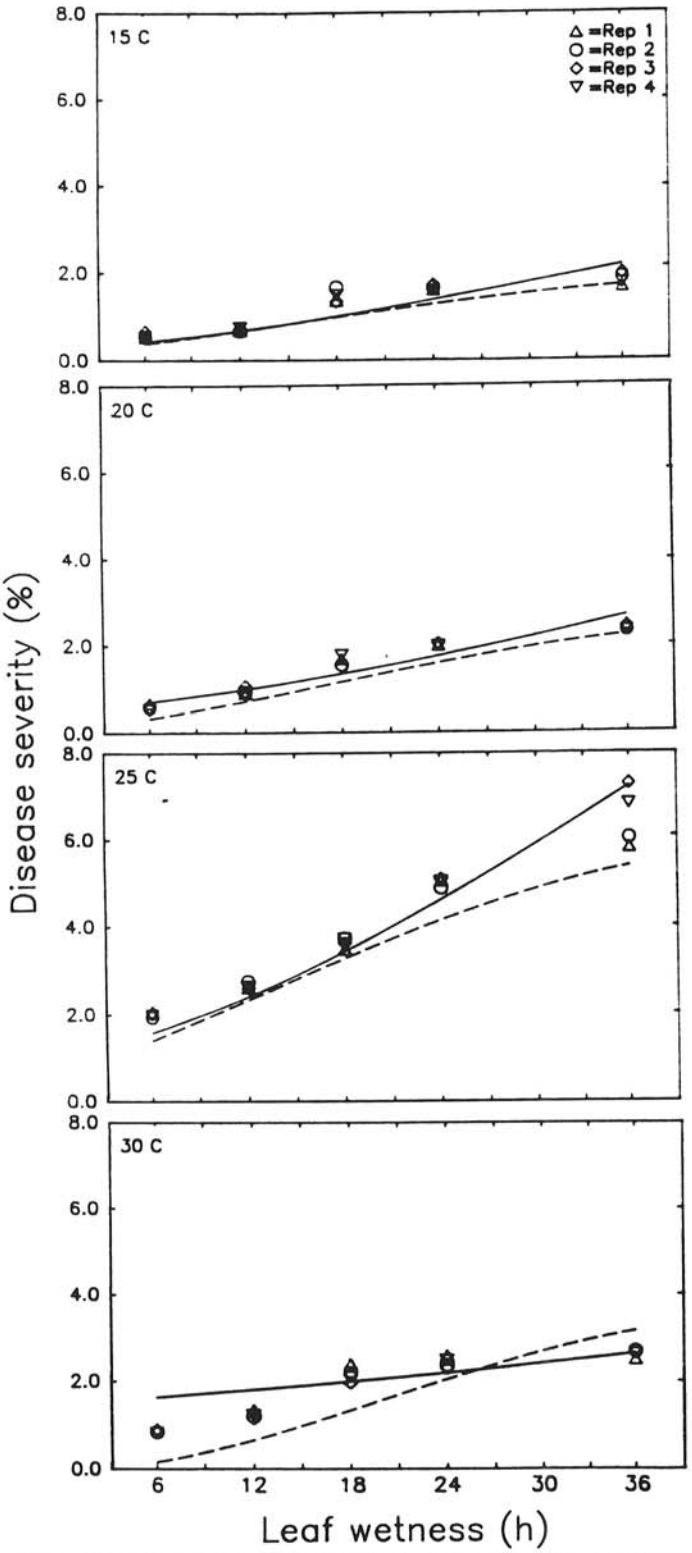

Fig. 4. Prediction of the disease severity (assessment day 14) of *Septoria glycines* for various temperatures and leaf wetness durations. Dashed line is regression model, solid line is Richards model.

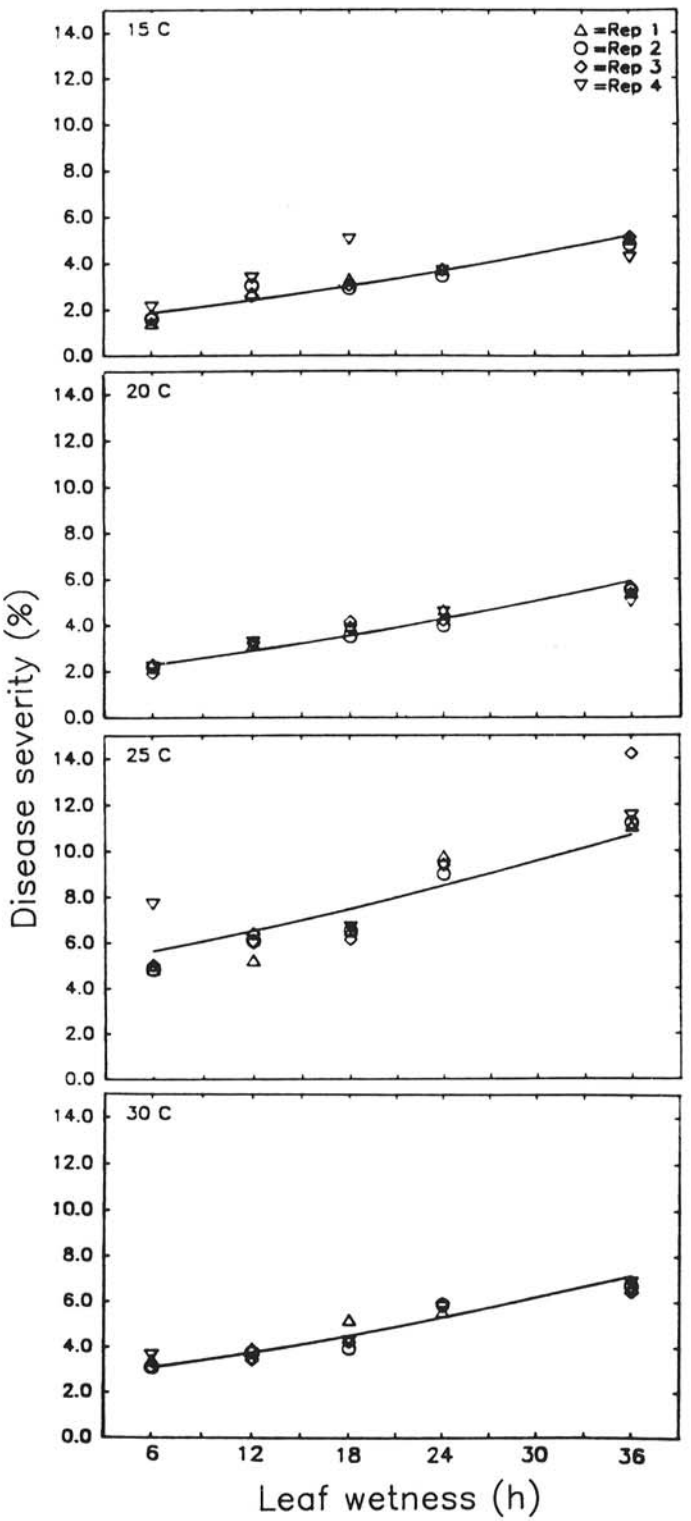

Fig. 5. Prediction of the disease severity (assessment day 21) of *Septoria glycines* for various temperatures and leaf wetness durations. Solid line is regression model.

where Y is the arcsine-transformed disease severity. All coefficients were significant at $P = 0.01$. The model had an R^2 value of 0.94. Based on an F test, data were pooled among replications. The interaction between T and W was significant, and the R^{*2} value was 0.86. The equation modeled disease severity very closely at 15 and 20 C (Fig. 4). At 25 C, the disease severities were underestimated at 24 and 36 h of leaf wetness. At 30 C, the equation underestimated the observed values at 6, 12, 18, and 24 h, and overestimated at 36 h of leaf wetness.

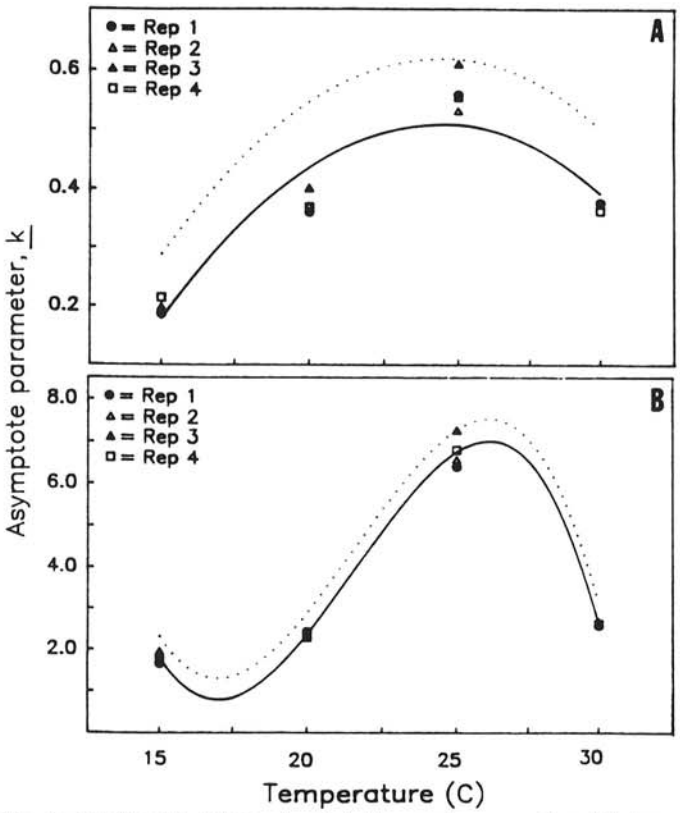

Fig. 6. Relationship between k and **A,** the maximum number of lesions per square centimeter (dotted line represents the predicted value of $k + 0.11$), and **B,** the maximum disease severity (dotted line is $k + 0.0052$) for the pooled data of the four replications.

Day 21: The relationship between the arcsine-transformed disease severity (Y) and T and W at assessment day 21 was best described by a polynomial regression model of the form

$$Y = 2.731 - 0.396^{*}T + 0.019^{*}T^2 - 0.00029^{*}T^3 + 0.003^{*}W \quad (10)$$

with an R^2 value of 0.92. Based on an F test between full and reduced models, the data from the four replications were combined. All coefficients were significant at $P = 0.01$. Disease severity increased with increasing dew periods. No significant interaction term between the two independent variables was obtained when the significance level was fixed at $P = 0.05$. The R^{*2} value was 0.95. The equation described the data very closely at 15, 20, and 30 C (Fig. 5). At 25 C, it slightly overestimated the disease severities at 12 and 18 h of leaf wetness, and slightly underestimated at 24 and 36 h.

Richards model. Day 7: Estimation of k and r parameters. The purpose of the first step of model development was to establish a relationship between the maximum value of LSQ (at each temperature) and temperature. The maximum LSQ observed (assessment day 7) was at 25 C (Fig. 6A). At 20 and 30 C, the values of k observed were approximately equal, with 15 C being the lowest. A quadratic function described the relationship between k and T with an R^2 value of 0.86 (Table 1). The regression equation was highly significant when based on the reduced model. The equation underestimated the k values observed at 25 C. Therefore, 0.11 was added when results from the equation were used as input in the next modeling step.

In the second step, the rates obtained through equation 4 were regressed against temperature. A cubic equation provided the best fit (Table 2), with an R^2 value of 0.80. The estimated coefficients were significant at $P < 0.01$. The observed values for the rate parameter r were highest at 25 C (Fig 7A). The r values for the other temperatures were close, with 30 C being slightly higher.

In the third step, the linearized, nonintercept version of the Richards model was regressed against a linear set of T and W (see equation 3) of the form obtained in the previous step (Table 3). The resulting nonlinear relationship between LSQ and T, W was

$$LSQ = k \times (1 + e^{-p})^{1/(1-m)} \quad (11)$$

where $k = -1.716 + 0.182^{*}T - 0.003^{*}T^2 + 0.11$, $p = -2.771^{*}W + 0.408^{*}W^{*}T - 0.019^{*}W^{*}T^2 - 0.000029^{*}W^{*}T^3$, and $m = 1.75$.

TABLE 1. Polynomial regression of the maximum number (k) of lesions per square centimeter and disease severity (% leaf coverage) on temperature[a]

Model variable	Assessment time (day)	df[b] error	SS[b] error	R^2 value	F P value	Partial regression coefficients and P value b_0	b_1	b_2	b_3
Reduced LSQ[c]	7	13	0.039	0.86	<0.01	−1.72	0.182	−0.03	...
						<0.01	<0.01	<0.01	...
Reduced DSQ[d]	14	12	5.07×10^{-5}	0.98	<0.01	1.47	−0.219	0.01	-1.65×10^{-4}
						<0.01	<0.01	<0.01	<0.01

[a] $k = b_0 + b_1T + b_2T^2 + b_3T^3$.
[b] df = Degrees of freedom; SS = sum of squares.
[c] Lesions per square centimeter.
[d] Disease severity (% leaf coverage).

TABLE 2. Polynomial regression of the rate parameter of the Richards model (r) on temperature[a]

Model variable	Assessment time (day)	Model	SS[b] error	R^2 value	F P value	Partial regression coefficients and P value b_0	b_1	b_2	b_3
LSQ[c]	7	Reduced	0.0003	0.80	<0.01	−2.706	0.038	−0.018	0.0002
						<0.01	<0.01	<0.01	<0.01
DSQ[d]	12	Reduced	3.75×10^{-5}	0.98	<0.01	−0.918	0.122	−0.005	8.7×10^{-5}
						<0.01	<0.01	<0.01	<0.01

[a] $\ln[(y/k)^{1-m} - 1] = -r^{*}W$.
[b] SS = sum of squares.
[c] Lesions per square centimeter.
[d] Disease severity (% leaf coverage).

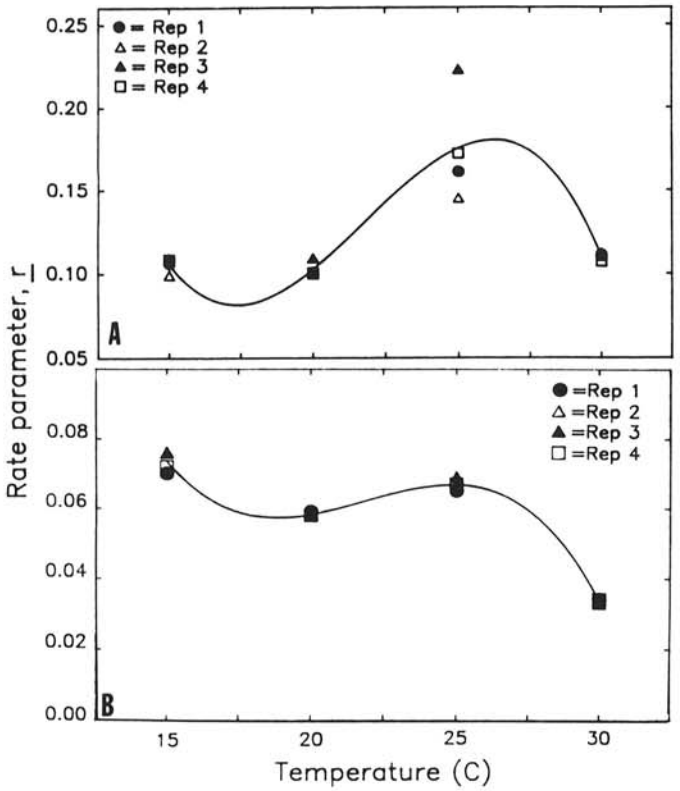

Fig. 7. Relationship between the rate parameter (r) of the Richards function and temperature for the pooled data. Values of r were generated by fitting the Richards function to the data for each temperature. **A,** the variable lesions per square centimeter at day 7; **B,** the variable disease severity (%) at day 14.

The back transformed estimates of the LSQ were highly correlated with the observed values ($R^{*2} = 0.90$). The estimated values were very close at 15, 20, and 30 C. At 25 C, the equation underestimated at 6, 12, 18, and 24 h of leaf wetness (Fig. 3).

Day 14: The procedure to model disease severity at day 14 was identical to the procedure for day 7. The equation relating k and T (equation 3) included a cubic term and had an R^2 value of 0.99. It underestimated the maximum disease severity at 25 C (Fig. 6B). Therefore, 0.0052 was added to the predicted k values when the equation was used as input in the remaining modeling steps.

The regression of the rate parameter (equation 5) on temperature resulted in an equation having linear, quadratic, and cubic terms (Table 2) with an R^2 value of 0.98. The highest r value was observed at 15 C, the lowest at 30 C (Fig. 7B). The r values at 20 and 25 C were intermediate.

The linearized, nonintercept version of the Richards function was then regressed against a linear combination of T and W (see equation 6). The resulting equation was highly significant ($P < 0.01$) and had an R^2 value 0.93 (Table 4). The F test between full and reduced models was nonsignificant. Data were therefore pooled.

The resulting nonlinear relationship between disease severity (Y) and temperature and leaf wetness period had the form

$$Y = k^*(1 + e^{-p})^{1/(1-m)} \quad (12)$$

where $k = 1.467 - 0.219^*T + 0.01^*T^2 - 0.00016^*T^3 + 0.0052$, $p = r^*W = -0.945^*W + 0.129^*W^*T - 0.006^*W^*T^2 + 0.0000927^*W^*T^3$, and $m = 1.3$. The estimated and observed disease severities were highly correlated ($R^{*2} = 0.93$). The equation estimated the disease severities closely, with the exception of 30 C, where severities were overestimated at 6 and 12 h of leaf wetness (Fig. 4).

TABLE 3. Estimation of the number of lesions per square centimeter of *Septoria glycines* as a function of temperature and leaf wetness duration by fitting the Richards model[a]

Model	df[b] error	SS[b] error	R^2 value	F P value	Partial regression coefficients and P value			
					b_0	b_1	b_2	b_3
Rep 1	16	4.97	0.79	<0.01	−2.39	0.350	−0.016	2.54×10^{-4}
					<0.01	<0.01	<0.01	<0.01
Rep 2	16	4.81	0.75	<0.01	−1.67	0.246	−0.011	1.80×10^{-4}
					<0.01	<0.01	<0.01	<0.01
Rep 3	16	4.48	0.90	<0.01	−4.15	0.616	−0.029	4.56×10^{-4}
					<0.01	<0.01	<0.01	<0.01
Rep 4	16	8.60	0.72	<0.01	−2.87	0.420	−0.020	3.06×10^{-4}
					<0.01	<0.01	<0.01	<0.01
Reduced	76	28.35	0.77	<0.01	−2.77	0.408	−0.019	3.00×10^{-4}
					<0.01	<0.01	<0.01	<0.01

[a] $r = b_0 + b_1T + b_2T^2 + b_3T^3$.
[b] df = Degrees of freedom; SS = sum of squares.

TABLE 4. Estimation of the disease severity (% leaf coverage) of *Septoria glycines* as a function of temperature and leaf wetness duration by fitting the Richards model[a]

Model	df[b] error	SS[b] error	R^2 value	F P value	Partial regression coefficients and P value			
					b_0	b_1	b_2	b_3
Rep 1	16	0.573	0.94	<0.01	−0.746	0.102	−0.004	7.50×10^{-5}
					0.02	0.02	0.02	0.01
Rep 2	16	0.737	0.93	<0.01	−0.978	0.133	−0.006	9.38×10^{-5}
					0.01	0.01	0.01	0.01
Rep 3	16	0.549	0.95	<0.01	−1.10	0.151	−0.007	1.07×10^{-4}
					<0.01	<0.01	<0.01	<0.01
Rep 4	16	0.714	0.93	<0.01	−0.959	0.132	−0.006	9.48×10^{-5}
					0.01	0.01	0.01	<0.01
Reduced	76	2.638	0.93	<0.01	−9.45	0.129	−0.006	9.28×10^{-5}
					<0.01	<0.01	<0.01	<0.01

[a] $r = b_0 + b_1T + b_2T^2 + b_3T^3$.
[b] df = Degrees of freedom; SS = sum of squares.

Day 21: The observed disease severities at day 21 could not be modeled using the Richards function. Even though k was related to temperature with statistical significance, the relationship between r and temperature (see equation 5) was nonsignificant. The r values were approximately 0.1, independent of temperature.

Disease progress and conditions during infection. The disease progress over time was modeled based on the logistic equation of the form

$$\ln[Y/(0.16 - Y)] = b_0 + r_L T \quad (13)$$

where Y is disease severity, T is time in days, and 0.16 is the maximum disease severity observed. Significant regression models could not be obtained when regressing r_L against leaf wetness period for each temperature separately. When regressing r_L against temperature for each leaf wetness period separately, significant regression models were obtained for 24 and 36 h of leaf wetness (Fig. 8) with all coefficients significant at $P < 0.01$ and R^2 values of 0.91 and 0.65, respectively. In general, r_L values were higher at the 36-h leaf wetness period compared to the 24-h (Fig. 8). The maximum r_L value is at 25 C, followed by 30 and 15 C, with the minimum at 20 C.

Influence of disease severity on senescence. At the fourth assessment (day 28), 80.9% of the inoculated leaves were senesced averaged over all treatment combinations, whereas only 25.4% were senesced in the control group. In general, rates of senescence were higher with extended dew periods and at 25 C. The differences were, however, statistically nonsignificant ($P = 0.05$).

DISCUSSION

Both temperature and leaf wetness period had a significant effect on the number of lesions per square centimeter of *S. glycines* assessed at day 7. The observed optimum temperature of 25 C was close to the optimum for germination and germ tube length as reported by Peterson and Edwards (11). Even though the lesion number increased with increasing leaf wetness periods, the effect of temperature was more pronounced (Fig. 1). When comparing the predictive abilities of the two models (Richards and linear regression), close agreement was observed for 15, 20, and 30 C (Fig. 3). The curve slope (relative rate of increase) suggested increasing values (LSQ) for leaf wetness periods beyond the assessed range. At 25 C, the slope of the curve based on the regression model increased with an extended leaf wetness period, whereas the relative rate of the curve based on the Richards model decreased.

The number of lesions at days 14 and 21 were not significantly influenced by temperature and leaf wetness duration. This is due to increasing as well as decreasing lesion numbers in certain treatments. At less favorable conditions during infection, the number of lesions increased over time. With the exception of 25 C, this was observed at leaf wetness periods of 6, 12, and 18 h. The second trend was observed at favorable infection conditions (24 or 36 h of leaf wetness, 25 C) (Fig. 1). The underlying cause of the decrease was the merging of lesions and/or the reduction in disease-free tissue. The disease severity (day 14) in relation to temperature and leaf wetness duration was successfully modeled with both techniques. Both models had high R^2 values, and the coefficients were highly significant. In the regression model, the influence of temperature was complex, as seen by the inclusion of linear, quadratic, and cubic terms. The influence of leaf wetness period was expressed as a linear and quadratic interaction with temperature. When comparing the predicted and observed values (Fig. 4), close agreement between the two models occurred at 15 and 20 C. At 25 C, the situation was similar to the one observed in modeling LSQ, with the slope reversed. The curve based on the Richards model had an increasing relative rate with increased leaf wetness, whereas the polynomial model had a decreasing one. At 30 C, the Richards model curve was essentially flat, whereas the polynomial model rate indicated a more rapid increase in disease severity.

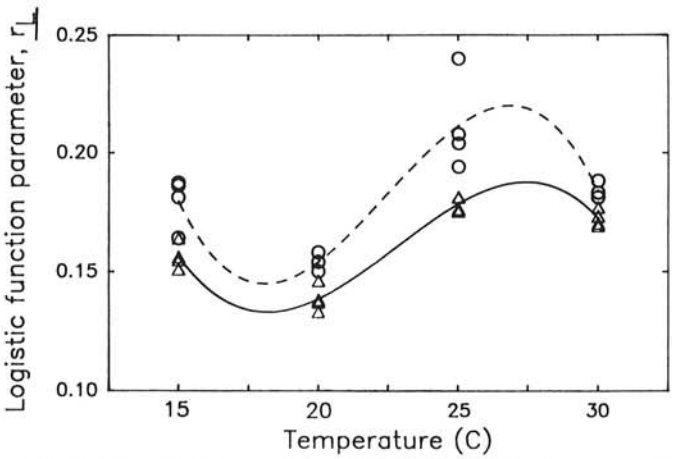

Fig. 8. Relationship between the r_L value of the logistic function and temperature (equation 13 in text). ○ = 36 h of leaf wetness period; △ = 24 h of leaf wetness period.

At assessment day 21, the Richards function could not be fitted due to the lack of a significant relationship between temperature and r. A similar situation was observed by Imhoff et al (4) for the germination of bean rust (*Uromyces phaseoli*) urediospores. This could be caused by the limitation on lesion expansion due to merger with surrounding lesions. The regression model, however, fitted the data well ($R^2 = 0.92$). The observed and predicted values were close over all temperatures and leaf wetness periods. When comparing the regression models obtained for days 14 and 21, certain similarities could be observed. The influence of temperature is represented by linear, quadratic, and cubic terms at both assessment dates. Additionally, the estimates of the coefficients for the variables were very close.

The optimum temperature (25 C) for infection and disease severity observed in this study was close to the optimum temperature of 28 C observed by Peterson and Edwards (11). The difference could be explained by the use of different temperatures in the studies, namely 15, 20, 25, and 30 C vs. 20, 24, 28, and 32 C. Similarly, maximum and minimum temperatures were close. Disease severity in their study increased with increasing dew periods of up to 120 h. Direct comparison of observed disease severities between the two studies was not possible because of the use of an index by Peterson and Edwards. They stated, however, that little disease developed in less than 48 h of dew. Again, use of an index limited direct comparison. Results from this study showed that even short periods of dew are sufficient to cause disease that can lead to early defoliation compared to noninoculated controls. The ability of the fungus to lead to premature defoliation at low disease severities might be related to toxin production, as evidenced by the chlorotic area surrounding the actual lesion. Under field situations, the disease severities resulting from these inoculations could be higher because of the influence of dew periods on disease progress after the infection process has ended. Ross (12) determined that daily dew periods after the initial postinoculation dew period led to a significant increase in disease severity. This was especially noticeable on plants exposed to relatively short postinoculation dew periods (<6 h). Dew formation in the greenhouse was not observed.

A statistically significant relationship between disease severity and senescence could not be established. The position of lesions at the top or bottom of the leaf, as well as lesion distribution, i.e., over the entire leaf surface or concentrated in a restricted area, could explain the absence of a relationship.

The relationship between the r_L value (logistic model) and temperature or leaf wetness period was significant only when r_L was regressed on temperature for the 24- and 36-h leaf wetness periods (Fig. 8). The r_L values were higher at 36 h of leaf wetness compared to 24 h at all temperatures. Within each leaf wetness period, the highest values were observed at 25 C. The following conclusions could be drawn from this situation. Temperature and leaf wetness period during the infection process influenced the rate of disease increase (r_L) after the infection process was com-

pleted, but only at long leaf wetness periods. Temperature seemed to have a stronger influence on the rate of increase. The increase at 15 C, which is greater than 20 C but less than 30 C, could be a function of initial slowing of development due to low temperature, and subsequent faster relative increase under greenhouse conditions identical to the other treatments. This situation will be investigated in detail in further experimentation.

The choice of modeling approach, i.e., linear regression models vs. nonlinear models, has been discussed (2,5); and the the nonlinear approach was identified as superior. In this study, no clear conclusions could be drawn. Both modeling approaches described the data well as measured by the R^2 values, although the shape of the curves at certain temperatures can lead to different conclusions regarding the effect of extending dew periods outside the tested range. Venus and Causton (15) compared the use of the Richards function and polynomial exponentials (polynomial function of the natural logarithm of a growth attribute in relation to time). They concluded that, based on statistical criteria, there are no grounds for choosing one or the other. Third degree polynomials containing the same number of parameters as the Richards function generally fitted the data better. They identified the main advantage of the Richards function in analyses where relative growth rates were used. One problem encountered with the use of the Richards function in this study was the use of cubic function in the modeling of the asymptote k (Fig. 6B) and rate parameter (Fig. 7A). The shape of the curve suggests a decrease in the parameter values between 15 and 20 C, which is biologically unlikely. A potential solution to this problem would have been the use of nonlinear functions to model the relationship. However, when judging the fit of the resulting curves (Figs. 3 and 4), this did not seem necessary.

The choice of quantitative model had little effect on the predicted LSQ or disease severity values, which were generally very close and should therefore be of minor importance in the development of disease forecasting–management systems. R^{*2} values could be used as unbiased criteria for the choice of model.

Lesion counts are considered a superior parameter to disease severity in describing infection success. In this study, the effect of assessment time on the predictive ability of the resulting quantitative models has the potential to bias disease management–forecasting systems. This is due mainly to the underestimation of terminal lesion counts at nonoptimal infection conditions and short assessment dates. This could be especially important for diseases with low apparent infection rates or for diseases that propagate in a discontinuous manner. *S. glycines* is a good example of the latter. Liberation and dispersion of conidia is mainly related to rain events. Disease developing at later dates (i.e., 14, 21, or 28 days after inoculation) will contribute to the spread of the pathogen in the same manner as disease present on day 7 if no intermittent rain event has taken place. Equations describing the increase in lesion numbers over time based on infection conditions and initial counts (similar to equation 6) could be used to reduce this bias.

Results from this study should be helpful in explaining the higher disease levels observed in spring and fall due to the more favorable temperatures observed in the field during that time. The shorter dew period requirements explain the survival in the field of the fungus in absence of extended periods of leaf wetness (>48 h). These lower levels of disease severity are important because they can lead to premature defoliation and because they constitute the inoculum base for epidemic outbreaks following protracted periods of leaf wetness and/or rain.

LITERATURE CITED

1. Backman, P. A., Rodriguez-Kabana, R., Hammond, J. M., and Thurlow, D. L. 1979. Cultivar, environment, and fungicide effects on foliar disease losses in soybeans. Phytopathology 69:562-564.
2. Carisse, O., and Kushalappa, A. C. 1990. Development of an infection model for *Cercospora carotae* on carrot based on temperature and leaf wetness duration. Phytopathology 80:1233-1238.
3. Hemmi, T. 1940. Studies on Septorioses of plants. VI. *Septoria glycines* Hemmi causing the brown spot disease of soybean. Mem. No. 470, Coll. Agric. Kyoto Imp. Univ., Phytopathol. Ser. 9.
4. Imhoff, M. W., Main, C. E., and Leonard, K. J. 1981. Effect of temperature, dew period, and age of leaves, spores, and source pustules on germination of bean rust urediospores. Phytopathology 71:577-583.
5. Lalancette, N., Ellis, M. A., and Madden, L. V. 1988. Development of an infection efficiency model for *Plasmopara viticola* on American grape based on temperature and duration of leaf wetness. Phytopathology 78:794-800.
6. Lim, S. M. 1979. Evaluation of soybean for resistance to Septoria brown spot. Plant Dis. Rep. 63:242-245.
7. Lim, S. M. 1980. Brown spot severity and yield reduction in soybean. Phytopathology 70:974-977.
8. Neter, J., and Wasserman, W. 1974. Applied linear statistical models. R. D. Irwin Inc., Homewood, IL.
9. Pataky, J. K., and Lim, S. M. 1981. Efficacy of benomyl for controlling Septoria brown spot of soybeans. Phytopathology 71:438-442.
10. Pataky, J. K., and Lim, S. M. 1981. Effects of row width and plant growth habit on Septoria brown spot development and soybean yield. Phytopathology 71:1051-1056.
11. Peterson, D. J., and Edwards, H. H. 1982. Effects of temperature and leaf wetness period on brown spot disease of soybeans. Plant Dis. 66:995-998.
12. Ross, J. P. 1982. Effect of simulated dew and postinoculation moist periods on infection of soybean by *Septoria glycines*. Phytopathology 72:236-238.
13. SAS Institute. 1985. SAS User's Guide: Statistics. Version 5 ed. SAS Institute, Cary, NC.
14. Sinclair, J. B., ed. 1982. Compendium of Soybean Diseases. American Phytopathological Society, St. Paul, MN.
15. Venus, J. C., and Causton, D. R. 1979. Plant growth analysis: The use of the Richards function as an alternative to polynomial exponential. Ann. Bot. 43:623-632.
16. Williams , D. J., and Nyvall, R. F. 1980. Leaf infection and yield losses caused by brown spot and bacterial blight disease of soybean. Phytopathology 70:900-902.
17. Wolf, F. A., and Lehman, S. G. 1926. Brown spot disease of soybean. J. Agric. Res. 33:365-374.
18. Young, L. D., and Ross, J. P. 1978. Resistance evaluation and inheritance of a nonchlorotic response to brown spot of soybean. Crop. Sci. 18:1075-1077.
19. Young, L. D., and Ross, J. P. 1979. Brown spot development and yield response of soybean inoculated with *Septoria glycines* at various growth stages. Phytopathology 69:8-11.