A Computer Simulation Model for Cercospora Leaf Spot of Peanut

G. R. Knudsen, H. W. Spurr, Jr., and C. S. Johnson

First author: research associate, Southern Region, ARS, USDA, Oxford Tobacco Research Laboratory, Oxford, NC 27565, and North Carolina State University, Raleigh, 27695-7616. Second author: research plant pathologist, ARS, USDA, and professor, North Carolina State University. Third author: assistant professor, Virginia Polytechnic Institute and State University, Blackstone 23824. Present address of first author: Department of Plant, Soil, and Entomological Sciences, University of Idaho, Moscow 83843.

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ABSTRACT

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A computer simulation model was developed to predict disease progression of Cercospora leaf spot of peanut (causal agents: *Cercospora arachidicola* and *Cercosporidium personatum*). The model was derived in part from an advisory system used for fungicide scheduling in North Carolina and Virginia. Basic infection rate was modeled as a function of hours of relative humidity > 95%, minimum temperature during the period of high humidity, amount of infectious tissue, and proportion of uninfected tissue remaining. Latent and infectious periods were treated as distributed delay processes, and host plant growth (increase in leaflet number) was described as a logistic process. The model was validated using independent weather and disease severity data from field trials with peanut cultivar Florigiant, in which *C. arachidicola* was the predominant pathogen. Simulated disease progress curves and periods of rapid disease increase were similar to those observed in field trials. The model effectively ranked four epidemics in terms of end-of-season disease severity and area under the disease progress curve.

Additional key words: disease forecast, early leaf spot, late leaf spot.

Early and late leaf spots of peanut (*Arachis hypogaea* L.), caused by *Cercospora arachidicola* Hori and *Cercosporidium personatum* (Berk. & Curt.) Deighton, respectively, are among the most serious diseases of peanuts (13,14). The two diseases exhibit similarities in symptom expression, epidemiology, and resultant yield loss and are usually referred to as Cercospora leaf spot or peanut leaf spot. Cercospora leaf spot is controlled by several fungicides; the most widely used in North Carolina is chlorothalonil. Fungicides are usually applied on a 10–14-day schedule beginning about 30–40 days after planting (12).

A leaf spot forecast system developed by Jensen and Boyle (7) and Parvin et al (11) is currently used to schedule fungicide sprays in North Carolina and Virginia (2,12). The forecast system uses hours of relative humidity (RH) > 95% and minimum temperature during the high relative humidity period to calculate a daily index representing likelihood of disease increase. The sum of 2 days' indices is usually sufficient to determine a fungicide spray advisory. The leaf spot advisory system is a management tool; it does not predict the effects of a control decision on subsequent disease severity.

A model to predict disease progress of Cercospora leaf spot would be a valuable research tool because it would provide a framework within which to evaluate effects of weather, control strategies, and host resistance on disease development. In this report, we describe a computer simulation model that predicts disease progression of Cercospora leaf spot over the course of a growing season. The model was validated in field trials in North Carolina and Virginia.

MATERIALS AND METHODS

Model description. The computer model simulates disease progress over the course of a growing season, with time steps of 1 day. Interactions between system components in the model are diagrammed in Figure 1. Disease severity was expressed as percentage of leaflets either defoliated or having one or more visible lesions (6,7). Plant growth, defined as increase in leaflet number, was modeled as a simple logistic function. Parameters for

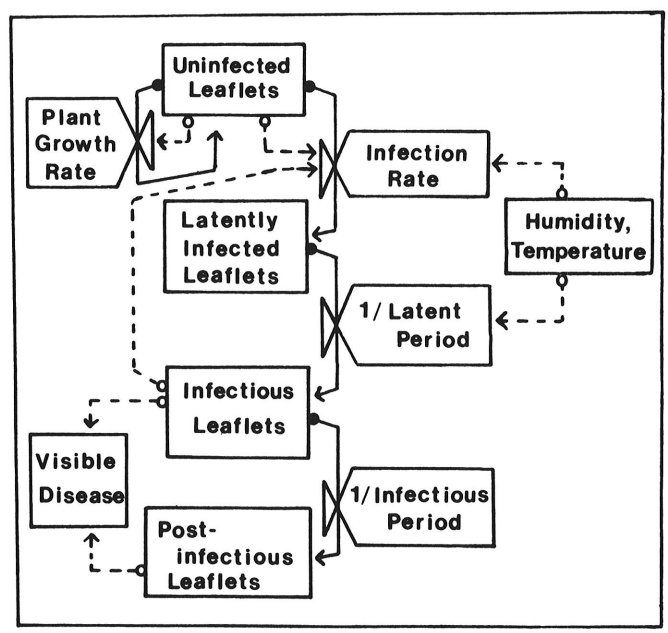

Fig. 1. State-Rate diagram for a computer simulation model of Cercospora leaf spot of peanut cultivar Florigiant. Major state variables are shown in rectangular boxes; other boxes represent rate variables. Flows of material (e.g., transfer of lesions from latent to infectious state) are indicated with solid arrows. Flows of influence or information (e.g., the effect of weather on infection rate) are indicated with dashed arrows.

this function were estimated from a model describing top growth of peanut plants (16) and from field counts of peanut leaflet numbers (Knudsen and Spurr, *unpublished*).

Because daily relative humidity and temperature summaries have been and continue to be obtained from many locations for use in the leaf spot advisory system, these weather parameters were chosen to drive the simulation model. Multiple regression method was used to develop a second-degree polynomial function with cutoff points that closely approximated (r^2= 0.97) the daily leaf spot index table of Parvin et al (11). Parvin et al used the sum of current and previous days' indices as an indication of the favorability of weather conditions for disease increase. In our model, we used a linear function of this sum to estimate the infection rate: R = newly infected leaflets per leaflet with sporulating lesions per day. Slope and intercept values for this function were estimated by running the simulation model with different parameter values and comparing the model's predictions with disease progress data from epidemics in several years (2,6,7). Parameters were calibrated to minimize sums of squares of residuals (observed minus predicted) and to accurately mimic patterns of disease increase.

Latent period (defined as the time in days from infection of a leaflet until sporulation from that leaflet) and infectious period (duration of spore production) were modeled as distributed delay processes (3); this method imposes a distribution around the mean developmental time (delay) for latent or infectious periods. Mean latent period was estimated daily as a linear function of minimum temperature during high relative humidity periods. Latent period has been reported to be temperature-dependent and to range from 10–21 days (6,7,14). On this basis we made the simplifying assumption that the relationship is linear with a latent period of 10 days at 22 C and 21 days at 19 C and that latent period is never less than 8 days.

The other distributed delay parameters (mean infectious period and number of age classes for latent and infectious periods) were estimated from published observations (6,14) and by calibrating the model as previously described. Mean infectious period was assumed to be 8 days. Lesions on infectious leaflets were assumed to become visible at the onset of the infectious period and to remain capable of initiating new infections, under conducive weather conditions, for the duration of the infectious period. Leaflets with postinfectious lesions (including defoliated leaflets) were not a source of inoculum in the model, but were included in the assessment of disease severity.

TABLE 1. Variables and constants used in the simulation model[a]

Variable	Description
Driving variables	
$HRH_{(t)}$	Daily hours of RH >95%
$TMP_{(t)}$	Minimum temperature during high RH period
State variables	
$LFLT_{(t)}$	Total number of leaflets
$INF_{(t)}$	Newly infected leaflets daily
$LAT_{(t)}$	Total, latently infected leaflets
$LL_{(j,t)}$	Latently infected leaflets in age class j
$SPL_{(t)}$	Total, leaflets with infectious lesions
$SL_{(j,t)}$	Infectious leaflets in age class j
$VIS_{(t)}$	Total, leaflets with visible leaf spots
$DIS_{(t)}$	Percent disease; DIS = (VIS/LFLT) × 100
Rate variables (units = day^{-1})	
R	Daily infection rate (newly infected leaflets)
$\delta 1_{(j)}$	Rate of age class change, latently infected leaflets
$\delta 2_{(j)}$	Rate of age class change, infectious leaflets
Auxiliary variables, constants	
$INDEX_{(t)}$	Daily weather index
p	Mean latent period (days)
KL	Index of dispersion around p; KL = 9
i	Mean infectious period (days)
KS	Index of dispersion around i; KS = 2
LFMAX	Maximum number of leaflets (6×10^4 per 10-m row)

[a] Parameters i, KL and KS used in simulations were obtained by the curve-fitting procedures described in the text, as was the equation to calculate daily values of R. Other model parameters and equations were obtained from published literature or experiments as described in the text.

Variables used in the simulation model are listed in Table 1. Model equations were:

Plant growth:

$$LFLT_{(t)} = LFLT_{(t-1)} + \{LFLT_{(t-1)} \times 0.1\,[(LFMAX - LFLT_{(t-1)})/LFMAX]\} \quad (1)$$

Disease progress:

$$INDEX_{(t)} = -1.9 + 0.004\,TMP - 1.823\,HRH + 0.031 \times [TMP \times HRH]; \quad (2)$$

(0<INDEX<1); (approximation of index table of Parvin et al)(11)

$$R_{(t)} = -3.6 + 1.6[INDEX_{(t)} + INDEX_{(t-1)}]; \quad 0< R <1; \quad (3)$$

(linearized function of 2-day index sums)

$$INF_{(t)} = R_{(t)} \times SPL_{(t)} \times CORR; \quad (4)$$

(where correction factor $CORR = 1 - \{[LAT_{(t)} + VIS_{(t)}]/LFLT_{(t)}\}$; thus, newly-infected leaflets = rate × "infectious leaflets" × proportion leaflets uninfected).

Latently infected leaflets (distributed delay model): for j= 1 to KL; KL = 9

$$LL_{(j,t)} = LL_{(j,t-1)} + \delta 1_{(j+1,t-1)} - \delta 1_{(j,t-1)} \quad (5)$$

$$\delta 1_{(j,t)} = LL_{(j,t)} \times KL \times p^{-1}, \text{ and } \delta 1_{(KL+1)} = INF \quad (6)$$

$$LAT_{(t)} = \Sigma_{j=1}^{KL} LL_{(j,t)} \quad (7)$$

(total leaflets with latent infections).

Infectious leaflets (distributed delay model): for j= 1 to KS; KS = 2

$$SL_{(j,t)} = SL_{(j,t-1)} + \delta 2_{(j+1,t-1)} - \delta 2_{(j,t-1)} \quad (8)$$

$$\delta 2_{(j,t)} = SL_{(j,t)} \times KS \times i^{-1} \text{ and } \delta 2_{(KS+1,t)} = \delta 1_{(1,t-1)} \quad (9)$$

$$SPL_{(t)} = \Sigma_{j=1}^{KS} SL_{(j,t)}; \quad (10)$$

(total leaflets with infectious lesions).

Visible disease:

$$VIS_{(t+1)} = VIS_{(t)} + \delta 1_{(1,t)}; \quad (11)$$

(defoliated or spotted leaflets).

The simulation model was written in the computer languages BASIC and Pascal and implemented on an IBM microcomputer.

Model performance. The simulation model was validated using disease progress data obtained in 1982, 1983, and 1984, from field plots in Lewiston and Rocky Mount, NC (8), and Newsoms, VA. The peanut cultivar Florigiant was used, and plots received no fungicide treatment. Validation data sets were independent (i.e., from different years and/or locations) from those used for model development. Planting date, number of plots assessed, and method of disease assessment varied between locations, as indicated in Table 2. Weather data were obtained from hygrothermographs installed at field sites. Weather data from 1983 were not available from the Rocky Mount location, and data from Lewiston were used to drive the simulation model for that year. Simulation runs were initialized for each validation experiment as follows: The first observation of mean disease severity greater than or equal to 1% and the date of observation were input to the model. An equal

proportion of disease was assumed to be latent at that time. The model then predicted disease severity for each remaining day of the season. Mean observed percent disease for each assessment date and 95% confidence intervals around sample means were plotted against model predictions.

Area under the disease progress curve (AUDPC) for each observed epidemic was estimated by making linear interpolations between disease levels on successive assessment dates, and then calculating the area under the curve for each replicate. Units for AUDPC were thus "percent days." For simulated epidemics, AUDPC values were calculated by integrating percent day values for each day of the simulated growing season.

RESULTS

Observed and predicted disease progress curves obtained in validation experiments are shown in Figure 2. Confidence intervals (95%) around observation means for each date are indicated. Also shown are cumulative daily disease index values for each season. Predicted disease progress curves closely matched patterns of disease increase observed in the field, although predicted disease levels throughout the season did not always fall within 95% confidence intervals around sample means. Final disease levels (last observation date) and AUDPC values for observed and simulated leaf spot epidemics are shown in Figure 3. The simulation model correctly ranked the four epidemics in terms of disease severity. For AUDPC, the model incorrectly predicted a higher value for Lewiston 1984 than for Lewiston 1982, but otherwise ranked the epidemics correctly.

DISCUSSION

In the locations and years represented by validation experiments, weather conditions varied considerably. For example, 1982 and 1984 were relatively favorable years for disease development, whereas the weather in 1983 was unusually cool and dry, and thus less favorable for leaf spot development. The model accurately mimicked patterns of disease development observed in the field under these different weather conditions. Predicted disease levels during the growing season generally fell within or close to 95% confidence intervals around observation means, and periods in which disease increased rapidly were identified. As shown by Figure 2, periods of predicted rapid disease increase did not always coincide with periods in which the cumulative daily index was increasing rapidly; this results from the model's requirements for not only disease-conducive weather but also a source of inoculum (i.e., sporulating lesions) for infection to occur. In fields where peanuts have been grown in previous years, leaf spot is almost invariably present. In that case, the simulation model may be arbitrarily initialized with a very low level of disease

TABLE 2. Design of field experiments to validate the simulation model

Year	Location	Planting date	Number of plots assessed[a]	Assessment method[b]
1982	Lewiston, NC	5/7	7	A
1983	Rocky Mount, NC	5/18	7	B
1984	Lewiston, NC	5/18	7	B
1984	Newsoms, VA	5/10	5	C

[a] Lewiston and Rocky Mount, NC: 4-row plots, 4 m long were assessed. Plots were isolated from each other on the ends by two 3-m strips of fallow ground and a 9.2-m strip of field corn planted between the fallow areas. Plots were isolated on the sides by 8.2 m of field corn.

Newsoms, VA: 4-row plots, 5 m long were assessed. Untreated plots were laid out in a randomized block design with other, fungicide or biocontrol agent-treated plots.

[b] Assessment methods:

A: The proportion of leaflets visibly infected was estimated (by multiple observers) from observations made on 61-cm sections of each row in each plot. Two stems were randomly selected from each of the four 61-cm sections of row, and proportion of defoliated leaflets was determined. Total percent disease was calculated as: % disease = {% defoliated + [(1-proportion defoliated leaflets) × % leaflets with leafspots]}.

B: Same as 1982, except only one 61-cm section of row was assessed on the two center rows of each plot.

C: Five stems were randomly picked from the two center rows in each plot, and percentage of leaflets either visibly infected or defoliated was determined.

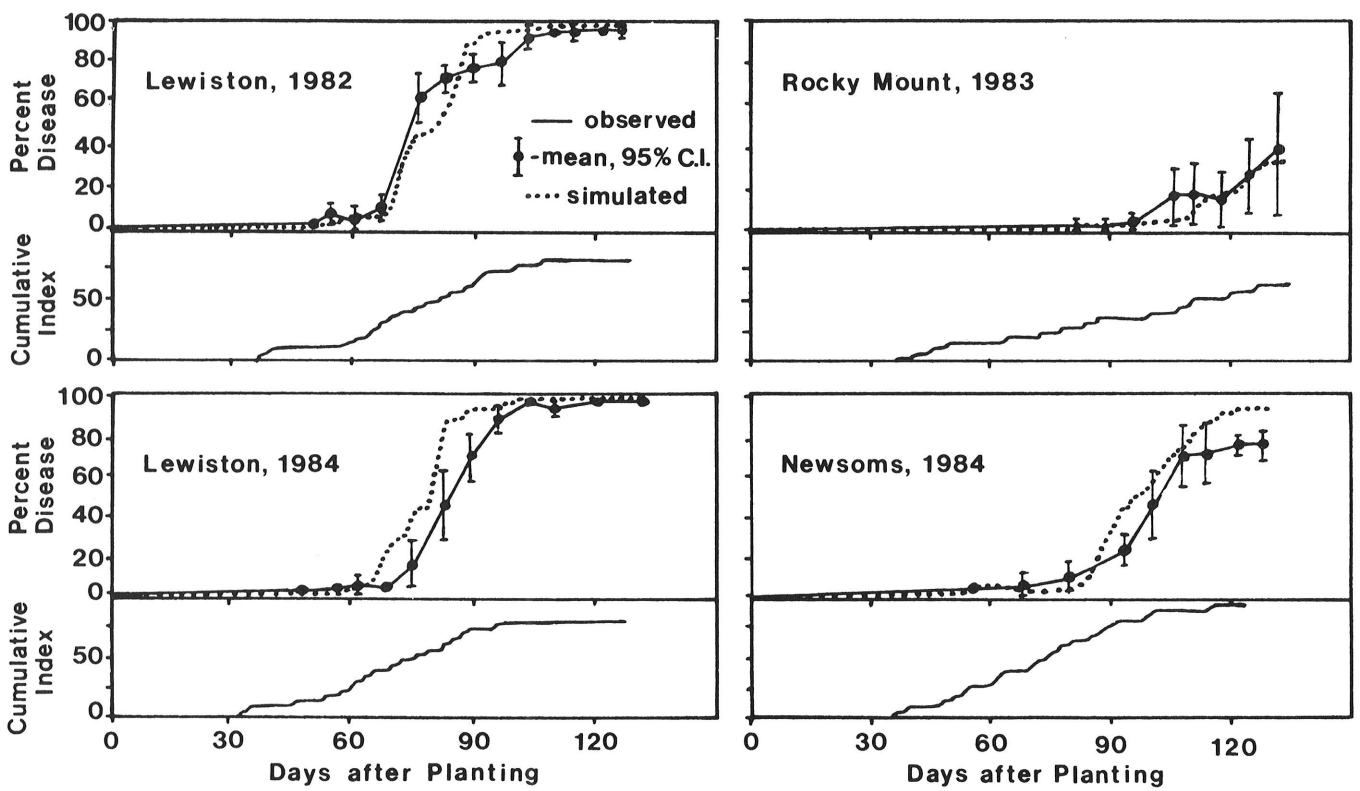

Fig. 2. Model validation: observed and simulated disease progress curves for Cercospora leaf spot on peanut cultivar Florigiant in four separate field trials. Vertical lines represent 95% confidence intervals around means for each observation date.

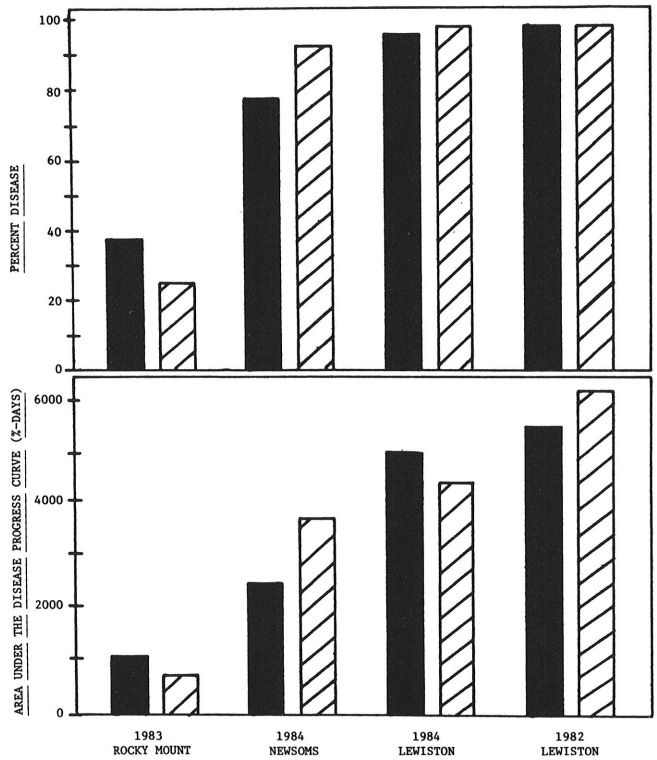

Fig. 3. Observed (shaded bars) and predicted (striped bars) end-of-season disease severity and area under the disease progress curve for Cercospora leaf spot epidemics on peanut cultivar Florigiant.

early in the season (e.g., 0.5% at 30–40 days after planting). However, for fields where peanuts were not previously grown, especially if distant from other peanut fields, this assumption would probably result in overestimation of disease. Thus, initializing the simulation with the first observation of disease greater than or equal to 1% enhances the robustness of the model.

More late leaf spot (*C. personatum*) was observed in 1983 than in other seasons, and because model parameters were derived from years in which early leaf spot predominated, it may be that model predictions are more accurate for that disease. *C. personatum* has been reported to cause more explosive epidemics than *C. arachidicola* (5), and changes in the relative importance of these two diseases have been observed in the southeastern United States in recent years (13). Further model validation for epidemics where late leaf spot is predominant are needed to test the model's performance in predicting late leaf spot epidemics.

The use of a distributed delay model to simulate latent and infectious periods offers an advantage over a simpler life-table type of model: Transfers between age classes are not synchronized for a particular cohort of lesions but have some of the variability that has been observed in the field (6,13). Although this method allows a very small proportion of individuals to remain within an age class for the entire simulation run, it is probably reasonable to assume that in nature, also, a finite number of new infections never become infectious lesions. Parameter values for the leaf spot simulation model were based partly on curve-fitting, and several components of the epidemic (sporulation, spore dissemination, spore arrival and germination, and penetration of the host) were simplified to: disease increase = f(relative humidity, temperature, and proportion of infectious tissue). The success of this approach depends on the fulfillment of two criteria. First, the model should make acceptably accurate predictions based on input data not used in the model's development and representing a range of possible weather patterns. The model's performance in validation experiments indicates fulfillment of this criterion, especially in mimicking disease progress and ranking seasonal weather patterns in terms of their effect on leaf spot. Second, the model should be a useful tool. We have used the model for development of chemical and biological control strategies for Cercospora leaf spot (9,10,15) because it provides a framework to evaluate effects of varying control agent efficacy, persistence, and timing of application on disease progression under different weather conditions.

Although weather conditions in North Carolina are often sufficiently warm and humid for leaf spot severity in untreated fields to reach 90% or more by mid-September, end-of-season disease severity may be less important than disease development earlier in the season in terms of impact on yield. Crop loss models have been based on disease severity observed at a single critical point, on multiple observations, or on AUDPC (4,8). Whereas multiple point or AUDPC models probably make better yield loss predictions than critical point models, they are also more time-consuming and expensive to develop. One advantage of the computer simulation model is that it estimates disease at many times over the season (literally, every day). Thus, the simulation model will be an efficient tool to evaluate the potential effects of disease control agents or rate-reducing resistance on yield.

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