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Development of *Cercosporidium personatum* in Three Peanut Canopy Layers[1]

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ABSTRACT

Disease progress of late leafspot of peanut caused by *Cercosporidium personatum* was monitored on the variety 'Florunner' in unsprayed plots and in plots sprayed weekly with chlorothalonil (343 μg active ingredient/ ml H_2O). The canopy was divided into three vertical semicircular leaf layers, each 15 cm high. Proportions of visible disease (x_v) and defoliation (d) per leaf layer were estimated with a modified Horsfall-Barratt rating system. Total disease (x_t) in each leaf layer and per plant were calculated with the equation

$$x_t = [(1\text{-}d) * x_v + d].$$

Apparent infection rates (sensu Vanderplank) were calculated for x_v, d, and x_t in each leaf layer and for the total plant canopy. Rates of disease increase and defoliation on sprayed and unsprayed tissue were not significantly different (P = 0.05). Disease severity in plots sprayed with chlorothalonil usually lagged three to five days behind disease in unsprayed plots. The disease components, x_v, d, and x_t were greatest in the bottom canopy layer. The rate of defoliation in the top leaf layer of unsprayed plots was significantly slower (P = 0.05) than that in the other two leaf layers, possibly because of dilution by the new plant growth. Chlorothalonil sprays reduced initial disease but did not reduce the rate of increase of *C. personatum*.

Key Words: *Arachis hypogaea* L., late leafspot, epidemiology.

The peanut (*Arachis hypogaea* L.) is the thirteenth most important crop plant grown for food in the tropical, subtropical, and warm temperate zones of the world (9). Peanut production is limited by numerous plant diseases. The most prevalent of these diseases are the early and late leafspots caused by *Cercospora arachidicola* Hori (*Mycosphaerella arachidicola* Jenkins) and *Cercosporidium personatum* (Berk. & Curt.) Deighton (*M. berkeleyii* Jenkins), respectively. Worldwide economic losses are estimated at 15 to 50 percent of final yield annually (5, 8).

Lesions resulting from both fungi develop on leaves, petioles, stems, gynophores, and pods. These diseases can defoliate the plant and cause a reduction in yield. Defoliation results in the reduction of the dry weight of stems, pods, and seeds (2).

Chemicals are widely used for control of Cercospora leafspots (10, 11, 13). However, there are few reports on the effect of these chemicals on the rate of disease progress throughout a growing season (4). Fungicides generally slow the rate of disease progress and do not affect the initial level of disease (1, 15).

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Jenkins (8) reported that *C. personatum* was the more destructive of the two fungi, particularly late in the season (September through harvest) and on late maturing varieties. Late leafspot causes more rapid defoliation than early spot (8, 16). In Florida, naturally occurring epidemics of *C. personatum* are generally more destructive than those resulting from *C. arachidicola* (Berger and Plaut, unpublished).

Infection and subsequent defoliation by *C. personatum* occurs first in the lower canopy and progresses upwards. Disease assessments in previous reports always have been made on a whole plant basis (3, 6, 8). However, it is difficult to evaluate precisely the contribution of disease in a canopy layer to the whole plant, particularly when disease distribution on the plant is nonuniform. Provided the peanut plant foliage canopy could be divided into several layers, assessed and then reassembled, a more accurate disease evaluation might be made. The use of three canopy layers for disease assessment may provide more accurate disease assessment and better definition of disease progress.

Defoliation is usually excluded in disease evaluations because of numerous difficulties, although its importance in yield reduction has been recognized (2). The loss of a single leaflet may be of equal photosynthetic importance to the plant as an equivalent loss of leaflet area due to a lesion. The integration of visible disease with defoliation would provide a more realistic assessment of disease stress on the peanut plant.

In this paper three concepts of the development of late leafspot are reported: 1) the division of the peanut plant into three canopy layers for a more accurate disease assessment; 2) the mathematical integration of the proportion of visible disease and of defoliation to obtain a single value for total disease; and 3) the observed effects of a protectant fungicide on the apparent infection rate of *C. personatum*.

Materials and Methods

Peanuts (*Arachis hypogaea* L. var. 'Florunner') were planted in rows 1 m apart on June 7, 1978, (day 0) at the University of Florida agronomy farm, Newberry, FL. Experimental plots consisted of two parallel rows, 3 m in length, which were established by roguing soon after seedling emergence. A randomized complete block design with four replications was used. Two peanut rows bordered each experimental plot. All plants received standard cultural practices, including adequate fertilization. Plants were overhead irrigated when necessary, receiving 2.5 cm of water per irrigation. The entire experimental plot area was bordered by 'Florunner' peanuts on the East, North, and South, and by soybeans (*Glycine max* L.) on

the West. During 1977, peanuts were also grown in the plot area; blue lupine (*Lupinus* sp.) was used for the winter cover crop.

A total of ten approximately weekly applications of chlorothalonil fungicide (Bravo® 6F, Diamond Shamrock, Cleveland, Ohio, 44110) were initiated on day 22 and terminated on day 84. A uniform application rate of chlorothalonil at 343 μg active ingredient/ml water was maintained with a back-pack sprayer (Weed Systems, Inc., Gainesville, Florida, 32611) using pressurized carbon dioxide gas as a propellant. The concentration per volume of fungicide was one-half of the commercial recommendation (12) and was applied at approximately one-half the recommended time interval to facilitate coverage of new plant tissue.

Plants were exposed to naturally occurring *C. personatum*. The first symptoms of leafspot was observed on day 37 on the border rows.

Disease Assessment.

Each plot in the peanut canopy was randomly divided into three vertical semicircular leaf layers, henceforth referred to as the top, middle, and bottom leaf leayers (Fig. 1). The arcs separating each leaf layer were 15 cm apart (Fig. 1). Using several criteria, disease was assessed weekly. Assessments were made in both rows of the experimental plots for each of the three leaf layers beginning on day 64. Visible disease, (x_v), including necrosis and chlorosis of a leafspot per total leaflet area, and defoliation (d) due to late leafspot were estimated with the Horsfall-Barratt rating scale (7) modified by within class categories to obtain more accurate intraclass ratings (Berger unpublished). Total disease (x_t) in each leaf layer and on total plot was calculated by the equation

$$x_t = [(1-d) * x_v + d].$$

The values of the individual disease components, x_v, d, and x_t for the total canopy were obtained by calculating the mean of the three canopy laryers.

The values of x_v, d, and x_t in each leaf layer and for the total plant canopy were logistically transformed with the equation

$$f(x)=\log_e\left(\frac{x}{1-x}\right)$$

(14, 17). The function is called the logit of x. In this paper, the values of x_v, d, and x_t were so transformed. The apparent infection rate, r sensu Vanderplank (14) is the slope of the linear regression line, often termed the logit line, determined by plotting logit (x) against time. The equation:

$$r=\frac{1}{(t_2-t_1)} * (\text{logit } (x_2)-\text{logit } (x_1))$$

where t=time, $x_2=x_v$, d, or x_t at time 2, and $x_1=x_v$, d, or x_t at time 1,

was used to calculate the apparent infection rate for logit (x_v), logit (d), and logit (x_t), respectively (14, 17).

The delay in time (Δt) represents the time needed in a treated plot to reach a given severity compared to the time in an untreated plot. This delay in time between the plotted lease squares regression lines under all experimental treatments was calculated using the equation:

$$\Delta t=\frac{1}{r}(\text{logit } (x_u)-\text{logit } (x_s))$$

where r = apparent infection rate,

$x_u=x_v$, d, or x_t

under unsprayed conditions, and

$x_s=x_v$, d, and x_t

under sprayed conditons.

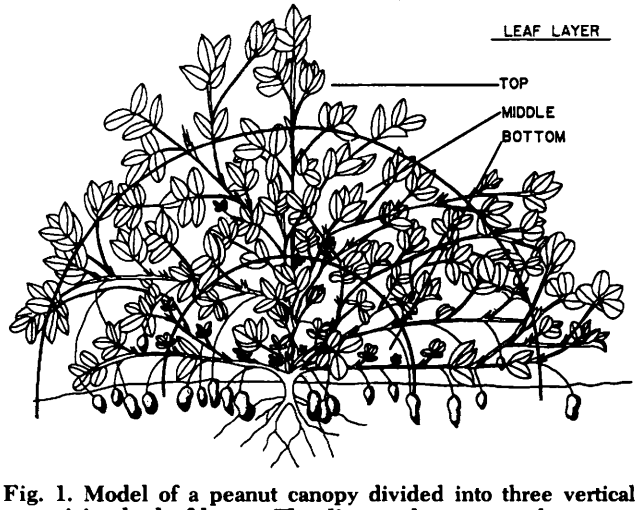

Fig. 1. Model of a peanut canopy divided into three vertical semicircular leaf layers. The distance between each are separating the canopy leaves is 15 cm.

Results and Discussion

After the initial appearance of focal infection of *C. personatum* in border rows, the pathogen spread uniformly throughout the plot area. There was not significant variation among replicates for any measured parameter on any observation date.

The logistically transformed disease progress curves for unsprayed tissue are given in Fig. 2A-C. The amount of visible disease (x_v) was usually greatest in the bottom canopy layer and least in the top layer. The rates of increase of x_v in the three canopy layers were not significantly different (P = 0.05) for days 64-78 (Fig. 2A). Defoliation of the lower leaves beginning around day 80 caused a confounding of the x_v rating for the bottom layer on day 84. Defoliation lagged behind the x_v by about seven days. The rate of defoliation (Fig. 2B) was slowest in the upper canopy layer possibly because of the influence of new plant growth diluting the total defoliation for this layer. Defoliation rates were faster than rates for progress of visible disease during all calculated time periods; defoliation made up the major component of total disease for days 78-84. Total disease was greatest in the bottom canopy layer and least in the top layer; the middle layer had intermediate values (Fig. 2C).

Disease progress in plots sprayed weekly with chlorothalonil (Fig. 3A-C) was similar to disease development in unsprayed plots, i. e., x_v, d, and x_t were usually greater in the bottom canopy layer, intermediate in the middle layer and least in the top layer.

The comparison of disease development in unsprayed plots and in plots sprayed weekly with chlorothalonil is best seen in the disease summary for the total canopy (Fig. 4A-C, Table 1). In general, the disease progressed at similar rates in unsprayed and sprayed plots. The exception, shown in Fig. 4A, is due to the significant decrease in visible

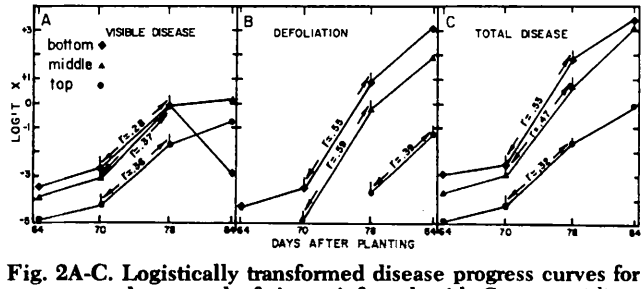

Fig. 2A-C. Logistically transformed disease progress curves for unsprayed peanut leaf tissue infected with *Cercosporidium personatum*. Figures A, B, and C represent three disease components including visible disease (x_v), defoliation (d), and total disease (x_t) respectively in three canopy layers.

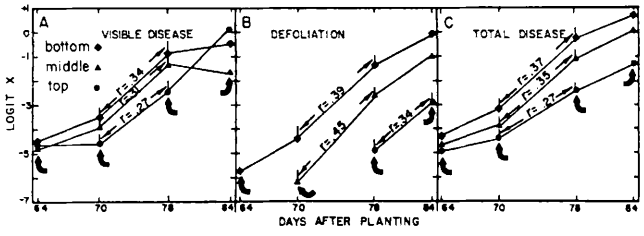

Fig. 3A-C. Logistically transformed disease progress curves for peanut tissue infected with *Cercosporidium personatum* sprayed with chlorothalonil at 343 μg active ingredient/ml water. Figures A, B, and C represent three disease components including visible disease (x_v), defoliation (d), and total disease (x_t) respectively for three canopy layers. Arrows indicate spray dates.

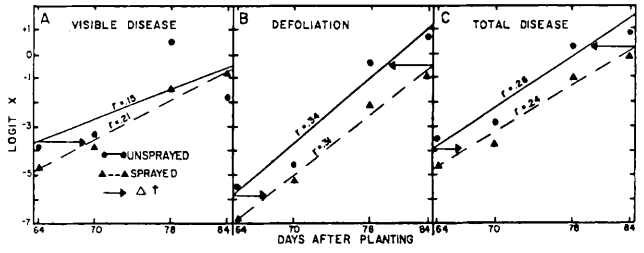

Fig. 4A-C. Least-squares regression lines for unsprayed and sprayed (343 μg chlorothalonil a. i./ml water) peanut tissue infected with *Cercosporidium personatum*. Figures A, B, and C represent three disease components, visible disease (s_v), defoliation (d), and total disease (x_t) respectively for the total canopy. Total canopy values are the mean of three leaf layers with four replications. The delay in time (t) is represented by arrows while r is equal to the slope of the regression line.

Table 1. The linear equation, correlation coefficient (least squares linear regression analysis) and delay in time (Δt) values for the total canopy. Three disease components (visible disease (x_v), defoliation (d), and total disease (x_t)) by two treatments sprayed and unsprayed) are presented. Data are diagrammed in Fig. 4A-C. Values are the average of four replications.

Disease component treatment	Linear Equation ($y = rx^d + b$)	Correlation coefficient (R)	Delay in time (Δt)[b] Initial	Final
Visible disease (x_v)				
sprayed[a]	y=.21X-18.26	.98	5.25	1.00
unsprayed	y=.15X-13.39	.69		
Defoliation (d)				
sprayed	y=.31X-26.79	.99	3.60	5.00
unsprayed	y=.34X-27.61	.98		
Total Disease (x_t)				
sprayed	y=.24X-20.08	.99	3.25	5.00
unsprayed	y=.26X-20.49	.96		

[a] Weekly application of chlorothalonil (343 μg active ingredient/ml water).

[b] $\Delta t = \frac{1}{r}$ (logit[c] X unsprayed - logit X sprayed).

[c] logit = ln $(\frac{X}{1-X})$.

[d] r = Vanderplank's term for the slope of the linear regression line.

disease on unsprayed tissue on day 34. As a result, the slope of the line decreased and correlation coefficients for the regression line are relatively poor. In all other cases, the severity of x_v, d, and x_t in sprayed plots lagged three to five days behind the disease in unsprayed plots. This delay in time (Δt) between sprayed and unsprayed did not vary greatly over the period 64-84 days. We interpret this lack of change in Δt to mean that the fungicide was only effective at reducing initial inoculum and it was not effective at slowing the epidemic when x >.01. The lack of control of *C. personatum* with weekly sprays of chlorothalonil was not typical of commercial practice or other experimental results (4). With the spraying system used, poor fungicide coverage of the lower surfaces of leaves may have occurred which allowed those areas to remain unprotected. In addition, the frequency of fungicide application allowed new plant growth to remain unprotected for a period sufficient to allow infection by the fungus.

The epidemic rates which we observed (r = .25-.6) would be considered extremely fast for a leaf spotting fungus (14). These rates are in the range observed for cereal rusts and potato late blight in epidemics that have led to substantial yield losses. For a leaf spotting fungus to proceed at such a pace, a fully susceptible host, an aggressive pathogen, and a favorable environment must have occurred.

Our division of the plant canopy into three layers provided greater definition of disease progression of *C. personatum*. Defoliation was identified with clarity as the major component of total disease severity. This quantification will lead to more accurate charcterization of this disease in a simulation model; a procedure which may allow the development of more efficient control strategies.

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