# A Critical-Point Yield Loss Model for Cylindrocladium Black Rot of Peanut

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Journal Series Paper 8791 of the North Carolina Agricultural Research Service, Raleigh.

A portion of a thesis submitted by the first author in partial fulfillmant of the PhD requirements.

The authors wish to thank Joyce Hollowell, Mark Black, Kevin Jones, Barbara Shew, Chuck Johnson, and Simon Hau for assistance in field experiments. North Carolina peanut growers whose cooperation in this study is greatly appreciated include: Everett Byrd, Wade Byrd, Braxton Cobb, Johnny Ross, James Worsley, and the staff of the Peanut Belt Research Station, Lewiston, NC.

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Accepted for publication 8 June 1983.

#### ABSTRACT

Pataky, J. K., Beute, M. K., Wynne, J. C., and Carlson, G. A. 1983. A critical-point yield loss model for Cylindrocladium black rot of peanut. Phytopathology 73:1559-1563.

Three approaches were used to estimate peanut yield loss due to Cylindrocladium black rot for two commercial cultivars and two advancedgeneration breeding lines which were evaluated in 10 field trials in four North Carolina counties in 1980, 1981, and 1982. Results from trials in stratified fields and established plots, and from Cylindrocladium black rot simulations, were similar for a highly susceptible commercial cultivar (Florigiant), a moderately resistant commercial cultivar (NC 8C), and a highly resistant breeding line (NC 18229). A critical-point model for predicting incidence of Cylindrocladium black rot approximately 1 wk before digging explained disease incidence-yield relationships when yield was expressed as the percentage of the maximum yield at a location. Critical-point models for Florigiant, NC 8C, and NC 18229 were: Y = 98.8 $-72.7 X(r^2=0.84)$ ;  $Y=100.2-74.5 X(r^2=0.79)$ ; and  $Y=98.3-66.6 X(r^2=0.79)$ = 0.57), respectively, in which Y = predicted percentage maximum yield and X = incidence at the critical rating date. In trials where epidemics developed naturally, mean incidence 1 wk before digging ranged from 0.21 to 0.74, 0.17 to 0.53, and 0.13 to 0.33 and mean predicted yield loss ranged from 15 to 53%, 13 to 40%, and 9 to 22% for Florigiant, NC 8C, and NC 18229, respectively. A single critical-point model was not applicable over locations for NC 18016, which appeared to be less agronomically stable than the other lines evaluated in this study.

Additional key words: Arachis hypogaea, Cylindrocladium crotalariae, disease loss assessment, epidemiology.

Cylindrocladium black rot (CBR) of peanut (Arachis hypogaea L.), which is caused by Cylindrocladium crotalariae (Loos) Bell and Sobers, induces a peg, pod, and root rot that was first described in Georgia in 1965 (2). Currently, CBR is of economic importance in North Carolina and Virginia. Preliminary analysis of disease development in the field indicates that CBR is a monocyclic disease for which microsclerotia are the primary, soilborne inocula (20). Ascospores and conidia are formed but appear to be of little importance in epidemic development (18).

Following the discovery of CBR in North Carolina and Virginia in 1970 (5), evaluations were made of three disease control tactics: chemicals, crop rotations, and resistance. Initial studies of chemical control indicated that only the wide-spectrum soil biocides were effective against C. crotalariae (1,19). Fumigation of large acreages did not seem biologically or economically practical; however, recent investigations suggest that in-row applications of certain soil fumigants may effectively reduce CBR, particularly when combined with the use of partially resistant cultivars (J. E. Bailey, personal communication).

Initial investigations of crop rotations suggested that 2- or 3-yr rotations to nonhost crops would reduce the percentage of soil samples from which C. crotalariae was recovered (17,19). Quantitative evaluations of the microsclerotia population dynamics of C. crotalariae indicated decreases following 2-yr rotations to tobacco, corn, cotton, or fallow, but populations remained too high to grow susceptible commercial peanuts (13). Consequently, economic control of CBR must include other control tactics in addition to rotations.

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Sources of CBR resistance have been identified (3,4,7,23,25) and a resistant cultivar, NC 8C, has been developed and released (24); however, resistance is inoculum density dependent and incomplete (12). At moderate or high microsclerotial populations, the resistant cultivar and breeding lines are severely diseased.

Several tactics have been integrated into a CBR management program. The current management strategy is to reduce microsclerotial populations to levels at which commercial peanuts can be profitably grown in fields infested with C. crotalariae. To estimate the disease levels which result in maximum profits for growers, yield loss functions and control cost functions should be compared. Currently, no extensive evaluations of yield loss due to CBR have been reported, although estimates of incidence and yield were published in a preliminary report on this disease (5).

The objectives of this study were to estimate yield loss due to CBR for susceptible and partially resistant peanut cultivars and to develop yield loss functions that can be used in evaluations of management tactics. Evaluations of quality and value reductions due to CBR have been reported (10).

### MATERIALS AND METHODS

Cultivars and breeding lines. Two commercial peanut cultivars, Florigiant and NC 8C (NC 3139 × Florigiant), and two advanced generation breeding lines, NC 18016 (NC 9088 × NC 3033) and NC 18229 (NC 3033 × NC 2), were evaluated. Florigiant, which is widely grown in North Carolina and Virginia, is highly susceptible to CBR. NC 8C is moderately resistant. NC 18016 and NC 18229, which are not agronomically acceptable because of low yields and small seed size, are highly resistant.

Field experiments. Trials were conducted in 10 fields in four North Carolina counties during the 1980, 1981, and 1982 growing seasons. In all trials, normal peanut production practices were

followed by growers (21). At all locations, yields were measured from plots that were two rows wide (0.91 m row width) and 6.1 m long. A total of 541 yield plots were evaluated.

Natural CBR epidemics of various characteristics and a simulation of epidemics were obtained by using three approaches to field experiments: established plots, stratified fields and a field simulation.

In the established plot method, growers' fields were identified in which CBR epidemics were beginning to develop in late July. Trials were established in late July or early August in areas of these fields that were relatively uniform for soil properties but varied for the occurrence of CBR. A rectangular area of the field was divided into 18–30 individual two-row yield plots. The epidemics that developed in individual plots within the larger rectangular areas varied in rate and incidence. Each established plot trial consisted of a single cultivar. Adjacent fields of Florigiant and NC8C were used at one location. Four established plot trials were conducted for Florigiant: in Bladen County in 1980 and 1981, in Edgecombe County in 1981, and in Bertie County in 1982. Two established plot trials were conducted for NC8C: in Bladen County in 1980 and in Bertie County in 1981.

In the stratified field approach, growers' fields that were known to be infested with *C. crotalariae* were assayed for microsclerotia (14) in the early spring. Selected fields were stratified into 20 or 21 blocks and each of the two cultivars and breeding lines were planted in each block. Each stratified block represented a replication of a randomized complete block design except that inoculum density (ie, disease treatments) varied within, as well as among, blocks. To prevent genotypic competition, only the middle two rows of each four-row plot were assessed for yield loss. Stratified trials were conducted in Bladen and Martin counties in 1981 and in Bladen County in 1982.

In the field simulation of CBR, the experimental design was a randomized complete block with four replications of a four by five factorial treatment design. The factorial consisted of the two cultivars and two breeding lines and four simulated epidemics plus a control. The four epidemics were simulated by using hand-held pruning shears to sever varying numbers of peanut taproots approximately 2 cm below the soil surface. Taproots were severed at various times during the growing season according to four disease progress curves. Disease progress curves describing the simulated epidemics (Fig. 1) were selected by using the Weibull function (11) to analyze 60 epidemics studied during the 1980

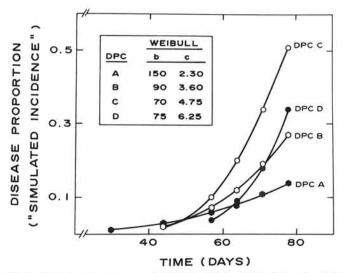


Fig. 1. Disease progress curves (DPC) fit with the Weibull function (11) describing simulated Cylindrocladium black rot epidemics on peanuts: disease proportion (Y) =  $1 - e^{-[(X-a)/b]^c}$  in which X = time in days, a = 13 at 15 July 1981 and 22 July 1982, and b and c are variable parameters as given. These progress curves were derived from analyses of 60 epidemics studied in 1980 and are representative of some of the epidemics that were observed in established plot and stratified field trials in 1980, 1981, and 1982.

growing season. Field simulations were conducted at the Peanut Belt Research Station in Bertie County in 1981 and 1982. Four-row plots were also used in the simulation trials to prevent genotypic competition.

Disease assessments. Incidence of CBR was determined every 7-20 days from mid-July until late September or early October when the peanuts were dug. Except for established plots in Bertie County in 1982, at least five assessments were made for each trial with at least three assessments after 1 September when epidemics were rapidly developing. Due to the lack of a standardized growth stage key for peanuts, disease assessments from various locations were compared on the basis of number of days before digging. In each plot the number of dead and wilted plants due to C. crotalariae was measured. Symptomatic plants from border rows were destructively sampled and their roots were incubated in moist chambers to confirm the presence of C. crotalariae. Perithecia of the perfect stage, Calonectria crotalariae (Loos) Bell and Sobers, were also noted on many dead and wilted plants. Incidence of CBR was expressed as proportions by dividing the number of infected plants (dead and wilted) by the total number of plants per plot. Disease ratings were also taken for southern stem rot (caused by Sclerotium rolfsii Sacc.) which occurred in some plots. Stem rot was rated as "hits" per plot similar to ratings described by Rodriguez-Kabana et al (16).

Statistical evaluation of models. Linear, quadratic, and cubic critical-point models based on the incidence of CBR and area-under-the-curve (AUC) models were evaluated by least-squares regressions for each trial. Multiple-point models were not extensively evaluated due to collinearity of disease assessments which would result in unstable parameters for regression equations (22). F-statistics were examined to compare the overall significance (P < 0.05) of models and the significance (P < 0.05) of polynomial terms. Coefficients of determinations ( $r^2$ ) estimated the proportion of variation in yield that was explained by disease assessments. Significance (P < 0.05) of partial regression coefficients were evaluated with t-statistics. Residuals were analyzed for homogeneity, linearity of the model, and outliers. A test for lack of fit was conducted for the field simulation trials where an estimate of error variance could be obtained.

Yields from individual trials were converted to percentage maximum yield for a cultivar at a location with the intercepts of the appropriate regression equation as the best estimate of maximum yield. Regressions of percentage of maximum yield on incidence which included trials (location and year) and cultivars as qualitative variables were conducted and tested for homogeneity of parameters.

### RESULTS

Disease assessments. CBR epidemics of various characteristics (rate and incidence) developed naturally in individual plots of established and stratified trials without interplot interference due to the soilborne nature of the pathogen and the monocyclic nature of the disease. Symptoms were first observed in late July or early August. Initial incidence was below 0.02 in the stratified trials and below 0.10 for all established plot trials except Bertie County in 1982. At approximately 1 wk before digging, incidence varied among trials and among plots within trials (Figs. 2 and 3). Over all trials, the range of incidence in individual plots approximately 1 wk before digging was: 0-1.0 for Florigiant, 0-0.89 for NC 8C, 0-0.76 for NC 18229, and 0-0.43 for NC 18016. The range of mean incidence for trials was: 0.21-0.74 for Florigiant, 0.17-0.53 for NC 8C, 0.13-0.33 for NC 18229, and 0.11-0.20 for NC 18016. In all trials except the stratified field at Martin County in 1981 and established plots of NC 8C, there were plots in which no CBR symptoms were observed.

A low amount of southern stem rot (less than five "hits" per plot) was observed in individual plots of many trials. For the stratified field at Bladen County in 1981, many plots had more than 10 stem rot "hits" and stem rot incidence was equal to or greater than CBR incidence. Many plants were infected by both fungi.

Field simulation. Preliminary analyses of the simulation trials were conducted to determine the lag period necessary to adjust for the time at which peanut taproots were severed until the time at which these simulation treatments would have a similar effect on peanut yield as CBR. These analyses were based on comparisons of regressions from simulation trials of yield or "simulated incidence" (proportion of plants with severed roots) to regressions from stratified field and established plot trials of yield on CBR incidence. Based on regression coefficients and intercepts, "simulated incidence" was similar to CBR incidence when taproots had been severed for approximately 3 wk. For example, if 18% of the peanut plants in a plot had their roots severed by 10 September, the effect on peanut yield was similar to 0.18 CBR incidence on 1 October. The 3-wk adjustment period was appropriate in both 1981 and 1982 even though environments were different in Bertie County for the 2 yr. After the 3-wk adjustment was made for simulation treatments, data were analyzed for yield loss.

**Yield loss assessments.** All possible critical-point models and an AUC model were evaluated for individual trials. For most trials, more than one model adequately described disease incidence-yield relationships. For example, yield (in kilograms per hectare) of NC 8C in the stratified trial in 1982 was explained by incidence approximately 3 wk before digging ( $Y = 4078 - 3413 X, r^2 = 0.76$ ), 1 wk before digging (Y = 4467 - 3352 X,  $r^2 = 0.87$ ), and AUC (Y = 0.87) 4000-120.3 X,  $r^2=0.80$ ). The critical-point model using incidence approximately 1 wk before digging gave a relatively good fit to all sets of data (Figs. 2-4) except for the stratified field in Bladen County in 1981 where stem rot was severe (data not presented) and for NC 18016 in the stratified trial in Martin County in 1981. When stem rot and CBR ratings were included in a multiple regression model for the stratified trial at Bladen County in 1981, a significant proportion of variation in yield still was not explained by the model. Therefore, data from the 1981 Bladen County trial and from the Martin County stratified trial for NC 18016, where regressions of yield on disease assessments were not significant, were not included in subsequent analyses.

When critical-point regressions of individual trials were combined and analyzed by multiple regressions with "dummy" variables for cultivars and locations trials (location by year), firstorder interactions were significant. This indicates that regression coefficients of yield on incidence differed among cultivars and

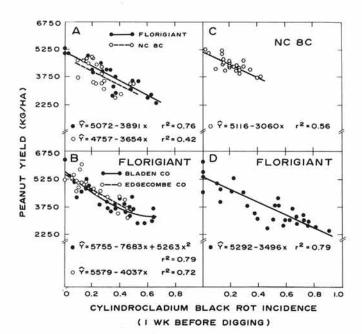


Fig. 2. Regressions of peanut yield (kilograms per hectare) on the incidence of Cylindrocladium black rot on peanuts ~1 wk before digging for two cultivars evaluated in a total of five established plot trials: A, Bladen County, 1980; B, Bladen and Edgecombe counties, 1981; C, Bertie County, 1981; and D, Bertie County, 1982.

trials. Similarly, when these analyses were conducted with percentage maximum yield as the dependent variable, the cultivar × trial interaction term was significant, which indicates that the two cultivars and the two breeding lines did not respond similarly in different trials. Consequently, data were sorted by cultivar and regressions of percentage maximum yield on incidence were

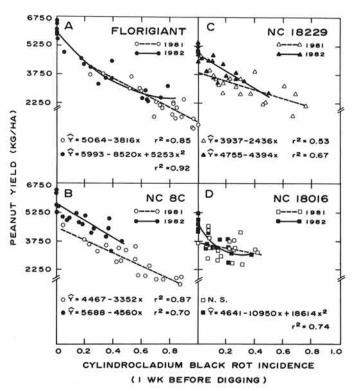


Fig. 3. Regressions of peanut yield (kilograms per hectare) on Cylindrocladium black rot incidence approximately 1 wk before digging two peanut cultivars and two advanced generation breeding lines evaluated in stratified field trials in Martin County in 1981 and Bladen County in 1982: A, Florigiant; B, NC 8C; C, NC 18229; and D, NC 18016.

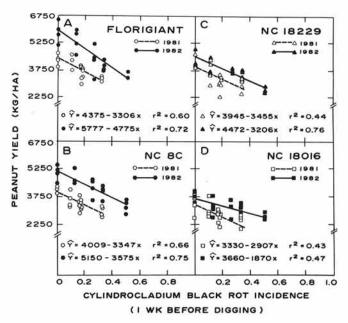


Fig. 4. Regressions of peanut yield (kilograms per hectare) on simulated Cylindrocladium black rot incidence approximately 1 wk before digging two peanut cultivars and two advanced generation breeding lines in Bertie County in 1981 and 1982: A, Florigiant; B, NC 8C; C, NC 18229; and D, NC 18016.

evaluated for individual cultivars. For Florigiant, NC 8C, and NC 18229, there were no significant disease  $\times$  trial interactions. Thus, a single regression equation could be used for all trials (locations and years) to relate percentage maximum yield and incidence 1 wk before digging (Fig. 5). For NC 18016, the interaction term was significant, and regression equations differed for each of the trials. For the 1981 and 1982 Bertie County trials and the 1982 Bladen County trial the equations were  $Y = 99.7 - 87.3 \ X$ ,  $Y = 99.4 - 51.0 \ X$ , and  $Y = 96.4 - 113.4 \ X$ , respectively.

## DISCUSSION

A critical-point model using CBR incidence approximately 1 wk before digging explained the disease incidence-yield relationships for Florigiant, NC 8C, and NC 18229 when yield was expressed as the percentage of the maximum at a given location. By expressing vield as the percentage of the maximum yield at a location, comparisons among years, locations, and cultivars, and comparisons to other peanut-pathogen or host-pathogen systems could be made more easily. These comparisons of percent or proportional relationships are similar to the concept of elasticity which is used by economists to compare relationships of items that have dissimilar units. For the two commercial cultivars, there were losses of ~7.5% of the maximum yield for each 10% CBR incidence (Fig. 5). For the more resistant breeding line, NC 18229, yield loss was ~6.7% for each 10% incidence (Fig. 5). These results are similar to the preliminary findings of Garren et al (5) in which a yield loss relationship for Florigiant of: Y = 5082 - 3925 X (in which Y =predicted yield in kilograms per hectare and X = proportion visually infected plants at harvest) can be derived from the limited

Based on mean incidence at the critical-point rating date in the stratified field and established plot trials, mean predicted yield loss due to CBR in these studies ranged from ~15 to 53% for Florigiant, 13 to 40% for NC 8C, and 9 to 22% for NC 18229. These percentages can be converted to losses of ~850-2,685 kg/ha for Florigiant, 740-1,785 kg/ha for NC 8C, and 430-1,300 kg/ha for NC 18229 by using the intercepts of regressions as the best estimates of maximum yield at a location. Similarly, if a peanut grower can reasonably estimate the average maximum yield per hectare of a field in the absence of CBR and the amount of CBR that may occur in that area of the field, the critical-point yield loss models can be used in economic evaluations of disease management tactics. Most growers' estimates of yield and disease incidence have been based on previous experiences in a particular field. Therefore, further research on the value of late season disease assessments for

predicting disease in subsequent years would be useful. The critical-point models can also be applied in conjunction with disease survey data to estimate yield losses due to CBR for large acreages. By using infrared aerial imagery, Powell et al (15) estimated that CBR incidence on 18 September 1974 was from 0.001 to 0.44 in 31 fields totaling 204.4 ha in Southhampton County, VA. Mean incidence for the entire 204.4 ha was 0.08. The critical-point model derived for Florigiant in our study suggests that yield loss in these fields was approximately 6.95% if peanuts were dug in late September. If the mean yield of these fields was 4,500 kg/ha, then peanut yields were reduced a total of 63,925 kg because of CBR.

Two genetically controlled host characteristics that can complicate the development of widely adopted yield loss models for any host-pathogen system were evident from our yield loss studies of peanut and *C. crotalariae*. The lack of agronomic stability (the ability of a genotype to perform equally well over environments relative to an environmental mean) may prevent the development of widely adopted models for specific genotypes; and, resistance mechanisms that affect disease incidence-yield relationships may result in different models for different genotypes.

The inability of a single model to explain yield loss over trials for NC 18016 was probably related to the agronomic instability of this line. At the stratified field in Martin County in 1981, where regressions of yield on disease assessments were not significant for NC 18016, most of the data points falling above the regression line were from plots located in the north and east portions of the field and those data points below the regression line were from plots located in the south and west areas of the field. When residuals from regressions for Florigiant, NC 8C, and NC 18229 were evaluated, no patterns were observed that related residuals to the location of plots in the field. Therefore, it appears that NC 18016 was more sensitive to field variation than were the other lines. Similarly, NC 18016 was the only line for which the cultivar × trial interaction was significant in the evaluation of percentage maximum yield. These results suggest that NC 18016 is not agronomically stable and that agronomic stability may be necessary in order to develop widely adapted yield loss models.

A yield loss model may also be affected by different mechanisms of resistance. Resistance mechanisms that affect disease incidence-yield relationships, in contrast to those that affect inoculum density-disease relationships, may account for the subtle difference in yield loss models among the two commercial cultivars, Florigiant and NC 8C, and the resistant breeding line, NC 18229. Coffelt and Garren (4) have suggested that different genetic mechanisms may control pod and root resistance to C. crotalariae. Since ratings of incidence are based on wilt symptoms, resistant

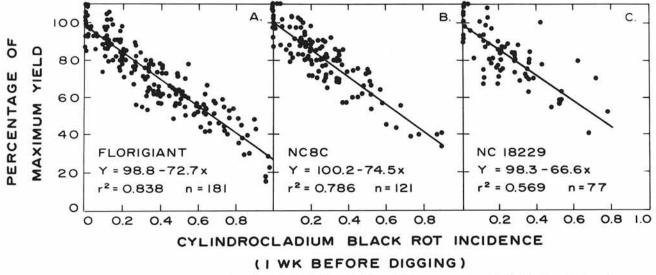


Fig. 5. Regressions of percentage maximum peanut yield on Cylindrocladium black rot incidence approximately 1 wk before digging: A, a susceptible cultivar, Florigiant; B, a moderately resistant cultivar, NC 8C; and C, a resistant breeding line, NC 18229.

lines with similar levels of root rot resistance could differ in their yield loss relationships if they differed in their level of pod rot resistance. Although there were only two commercial cultivars evaluated in this study, previous evaluations of commercial Virginia-type peanuts indicate a similarity of all commercial cultivars to Florigiant in their susceptibility to CBR (6,19). Therefore, the yield loss function derived for Florigiant in this study should be valid for economic evaluations of control tactics for the current commercial cultivars. However, as other resistant lines are released, yield loss relationships may need to be reevaluated, especially if high levels of pod rot resistance occur.

To obtain adequate data from which to develop yield loss models, epidemics of various characteristics and control plots must be established (8,9). Many yield loss studies dealing with foliar pathogens have used chemical sprays or genotypes nearly-isogenic for resistance in order to create these epidemics. However, these methods are not available for CBR and several other diseases caused by soilborne pathogens. The three approaches used to obtain epidemics of various characteristics in this study can be used for other soilborne pathogens as long as the advantages and disadvantages associated with each method are considered.

The advantages of the stratified field approach were that all lines could be evaluated in the same field; fields could be stratified so that assessments of field variation could be made; and, initial inoculum density could be measured. Disadvantages were that we were not assured of obtaining disease-free control plots, such as with the Martin County trial, and multiple pest problems could confound results, such as with the Bladen County trial in 1981. Established plot trials allowed greater control of variability in the amount of disease in the trial, and therefore increased the probability of a control plot. However, disadvantages of established plot trials were that differences among cultivars were confounded with differences among fields and the initial stages of the epidemics were not measured. Simulation trials allowed all lines to be evaluated in a replicated trial that included controls and also allowed the estimation of error variance. However, comparison of results from simulations to data from actual diseased plots were necessary to determine if treatments simulated the effects of disease. With the exception of stratified trials at Bladen County in 1981 and the variable results for NC 18016, results from each of the methods used in this study were similar, which suggests that each of the three approaches for determining yield loss due to CBR was appropriate. Nonetheless, by using all three methods we feel that there was a broader basis about which to make inferences from our data and that we guarded against the disadvantages associated with any single approach.

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