Spatial and Temporal Aspects of Epidemics of Cylindrocladium Black Rot in Resistant and Susceptible Peanut Genotypes

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ABSTRACT

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Epidemics of Cylindrocladium black rot were monitored in Martin County, NC, in 1986 and 1987. Seven peanut genotypes were used: susceptible cultivar, Florigiant; moderately resistant cultivar, NC 8C; three new genotypes selected for CBR resistance, NC Ac 18414, NC Ac 18416, and NC Ac 18417; and highly resistant breeding lines NC Ac 18016 and NC 3033. Each genotype was planted in 36 plots representing a range of inoculum densities of the pathogen, *Cylindrocladium crotalariae*. Disease progress was better described by the logistic model than by the monomolecular or Gompertz models for most genotypes. Rates of disease progress, determined by regression of the logit transformation of disease incidence on time, were similar in the moderately resistant genotypes NC 8C, NC Ac 18414, and NC Ac 18417 and susceptible cultivar Florigiant but were slower in NC Ac 18416, NC Ac 18016, and Nc 3033 than in Florigiant. Epidemics began 1.5–2 wk later in NC 8C, NC Ac 18416, and NC Ac 18417 than in Florigiant and were delayed further in NC Ac 18016 and NC 3033. Delay of onset of epidemics was more important than rate of disease progress in comparing effects of moderately resistant and susceptible genotypes on disease progress.

Additional keywords: Arachis hypogaea, epidemiology, soilborne fungi.

The use of disease progress curves in the epidemiology of diseases caused by root pathogens has been very limited relative to their common uses in the study of epidemics of foliar diseases (10). Disease progress curves of root diseases reflect the interactions of inoculum density, spread, and spatial patterns of inoculum of the pathogen, root growth, plant spacing and density, level of resistance in the host and conduciveness of the environment over time (10). By accounting for the effects of one or more of these factors, it is possible to determine more reliably the effects and importance of other specific factors.

Cylindrocladium black rot, caused by the soilborne fungus *Cylindrocladium crotalariae* (Loos) Bell and Sobers (4), is a major root disease in peanut (*Arachis hypogaea* L.) producing areas of North Carolina and Virginia (6,29,30). Disease severity is greatly affected by peanut genotype (20,21,25), the environment (24,33), and by inoculum density of the pathogen (7,8,25,35). Management of black rot depends largely on the use of moderately resistant cultivars, and programs established to develop resistant cultivars have produced genotypes that represent a range or resistance from very susceptible to highly resistant to the pathogen.

Microsclerotia are the primary propagules for dispersal, overwintering and inoculum (28,31) of *C. crotalariae*. Microsclerotia are produced in infected root tissues and are released into the soil upon subsequent degradation of the root tissue. Practices that reduce the density (3,9,13,27) and efficiency (6,7,8,33) of inoculum are used to complement host resistance. Inoculum of *C. crotalariae* typically is found in nonrandom or clustered patterns (17), which complicates epidemiological field studies and evaluation of new resistant genotypes. In areas of high inoculum density, even highly resistant genotypes may be severely diseased (20,21,25), thus the normal phenotype may be obscured by inoculum density effects. Assessing number and spatial patterns of microsclerotia present in the soil before planting facilitates determining the effect of these factors on disease progress and may allow differentiation from the effects of other factors such as host resistance.

Diagnostically distinct, aboveground symptoms of black rot include chlorosis, wilting, and plant death. Symptoms typically appear midway through the growing season and may increase rapidly as the season progresses. Monitoring the appearance of shoot symptoms provides an indirect, nondestructive means of estimating disease progress in the roots. The combination of the availability of genotypes representing a range of resistance to a root pathogen, the ability to determine the spatial patterns and levels of initial inoculum of the pathogen for use in the experimental design, and the opportunity to relate these factors to the appearance of symptoms in plant shoots is one that is not often encountered in epidemiology of root diseases.

Because of the ability to use spatial patterns and pathogen inoculum density levels in field experiments, the effects of host resistance and inoculum density of *C. crotalariae* on black rot disease progress can be examined. The purpose of this study was to examine the effects of host resistance and density of inoculum of *C. crotalariae* on development of CBR epidemics in genetically diverse peanut genotypes.

MATERIALS AND METHODS

Experiments were conducted in different fields in Martin County, NC, in 1986 and 1987 as previously described (14). In each field, four contiguous quadrants (11 × 76.8 m) were established with two quadrants each in an area of the field planted in peanut and two in an adjacent area planted in corn the previous

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year. Each of the four quadrants was further divided into three groups (11 × 25.6 m) consisting of 21 contiguous plots (3.7 × 3.7 m). Soil samples were taken as previously described (14) and assayed for inoculum of *C. crotalariae* as described by Phipps et al (23). With those estimates of inoculum density, three groups of seven plots, each representing high, medium, and low inoculum density classes, were established in each 21-plot group using a nested experimental design previously described (14). Seven peanut genotypes were used, representing a range of resistance to the pathogen. Genotypes included susceptible cultivar Florigiant; moderately resistant cultivar, NC 8C; two highly resistant breeding lines, NC Ac 18016 and NC 3033; and three moderately resistant genotypes, NC Ac 18414, NC Ac 18416 and NC Ac 18417. Each genotype was planted in 36 plots representing a range of inoculum density of the pathogen (14). Planting dates were 7 May 1986 and 2 May 1987.

Number and position of dead and wilted plants in each plot were determined and recorded each week until 20 wk after planting as described previously (14). Percent disease incidence (apparent incidence) was calculated by dividing the number of dead and wilted plants by the total plant stand per plot. Disease progress was plotted as apparent incidence (percent dead and wilted plants) versus time.

Linearized forms of the monomolecular (36), Gompertz (5), and logistic (36) models were evaluated for goodness of fit to the disease progress data for each genotype in each quadrant and across all quadrants. Criteria for goodness of fit included examination of the graph of disease incidence over time, coefficient of determination (R^2) values for regression of transformed data for each of the models on time, and plots of standardized residuals vs. expected values of those regressions for each model (11). These were determined for each genotype for each quadrant.

The linearized form of the logistic model was judged to be the most appropriate model in most of the cases considered each year. Thus, for comparison purposes, this model, with the assumption $Y_{max} = 1.00$ where Y is the proportion of symptomatic plants, was used to determine the rate of disease progress for each genotype in each quadrant. Inoculum density classes nested in three subdivisions per quadrant were incorporated into the regression analysis. Intercept values of the regression equation of logit transformed disease incidence on time were used as an indication to of epidemic onset. Slope of regressions of transformed disease incidence data on time for the different genotypes were compared using reduced versus full model F tests (19).

Rate of disease progress also was calculated for each plot. Because of delayed onset of symptoms compared to Florigiant, the first week of the epidemic was omitted for NC Ac 18414 in both years, the first 2 wk were omitted in NC 8C, NC Ac 18417 in both years, and the first 4 wk in 1986 and the first 3 wk of the epidemics in 1987 were omitted for NC Ac 18016 and NC 3033. Rates calculated for each plot were used in examining correlation between rate of disease progress and initial inoculum density.

Weeks until observation of first symptoms were analyzed with analysis of variance (34). Statistical significance of genotype effects was evaluated by single-degree-of-freedom contrast statements and by Fisher's protected LSD (34). Differences referred to in the text were significant ($P = 0.05$) unless otherwise stated. Pearson's correlation coefficients (34) were also calculated for the association between initial inoculum density and time until first symptoms and rate of disease progress.

RESULTS

The experimental design was successful in representing the different genotypes in similar ranges of inoculum, and was sufficient to account for the clustered pattern of inoculum of *C. crotalariae* (14). Examination of the disease progress curves revealed the genotype effects on black rot epidemics were consistent in both years, both across and within the different quadrants (Figs. 1–3). Average inoculum density for quadrants 1–4 was 2.26, 2.11, 4.63, and 7.29 and 5.17, 6.03. 2.42, and 1.99 in the two fields in 1986 and 1987, respectively (Figs. 2 and 3). Effects of level of inoculum of *C. crotalariae* on disease progress were not consistent between the 2 yr. In 1986, disease incidence was highest throughout the epidemic in quadrants with highest mean initial inoculum density (quadrants 3 and 4). Disease incidence was greater overall in 1987 than in 1986 for all genotypes (Fig. 1) and was similar in the 2 yr in quadrants with highest mean initial inoculum density (Fig. 3). The general shape of the disease progress curves of the moderately resistant genotypes was similar to that of disease progress curves of Florigiant (Figs. 1–3). This relationship was consistent across quadrants in both years. Based on examination of disease progress curves, epidemics began earlier in Florigiant than in other genotypes.

The logistic model was most appropriate for describing disease progress in plots of Florigiant, NC 8C, NC Ac 18417, and NC Ac 18414 in both years (Table 1). The Gompertz model described disease progress as well as or better than the logistic model in quadrants 1 and 2 for these genotypes in 1986. The Gompertz model was also best among the models examined for describing disease progress in NC Ac 18417 in quadrant 3 in 1986; however,

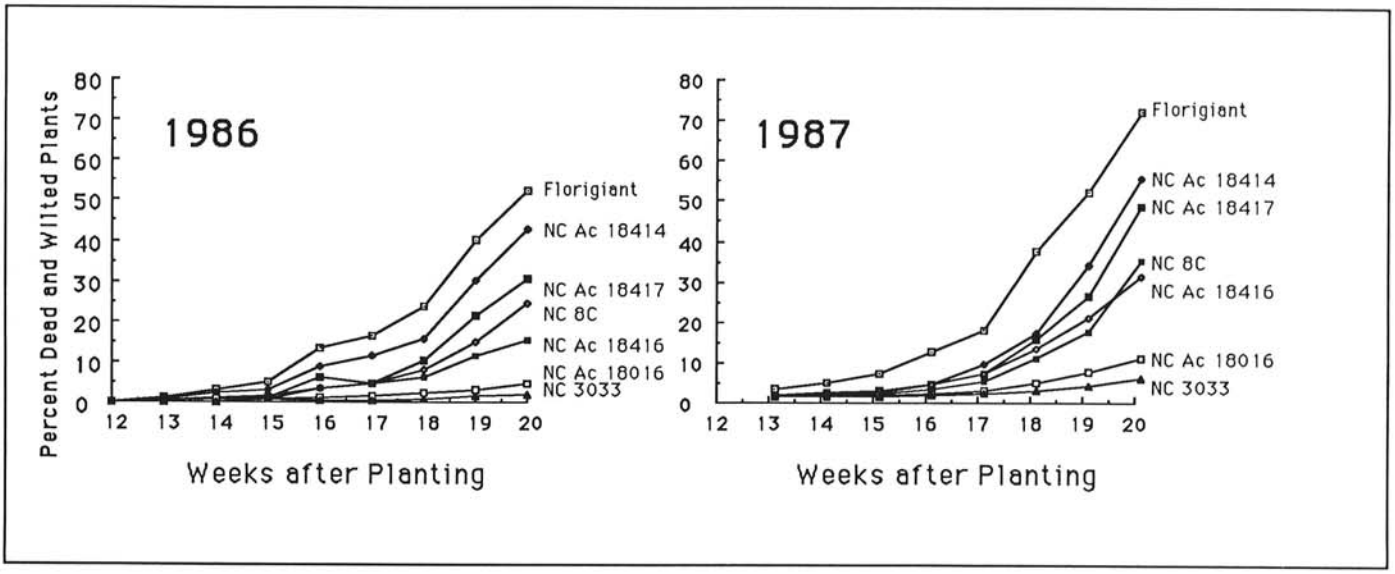

Fig. 1. Disease progress curves for Cylindrocladium black rot in seven peanut genotypes planted in a range of inoculum density levels of *Cylindrocladium crotalariae* in 1986 and 1987.

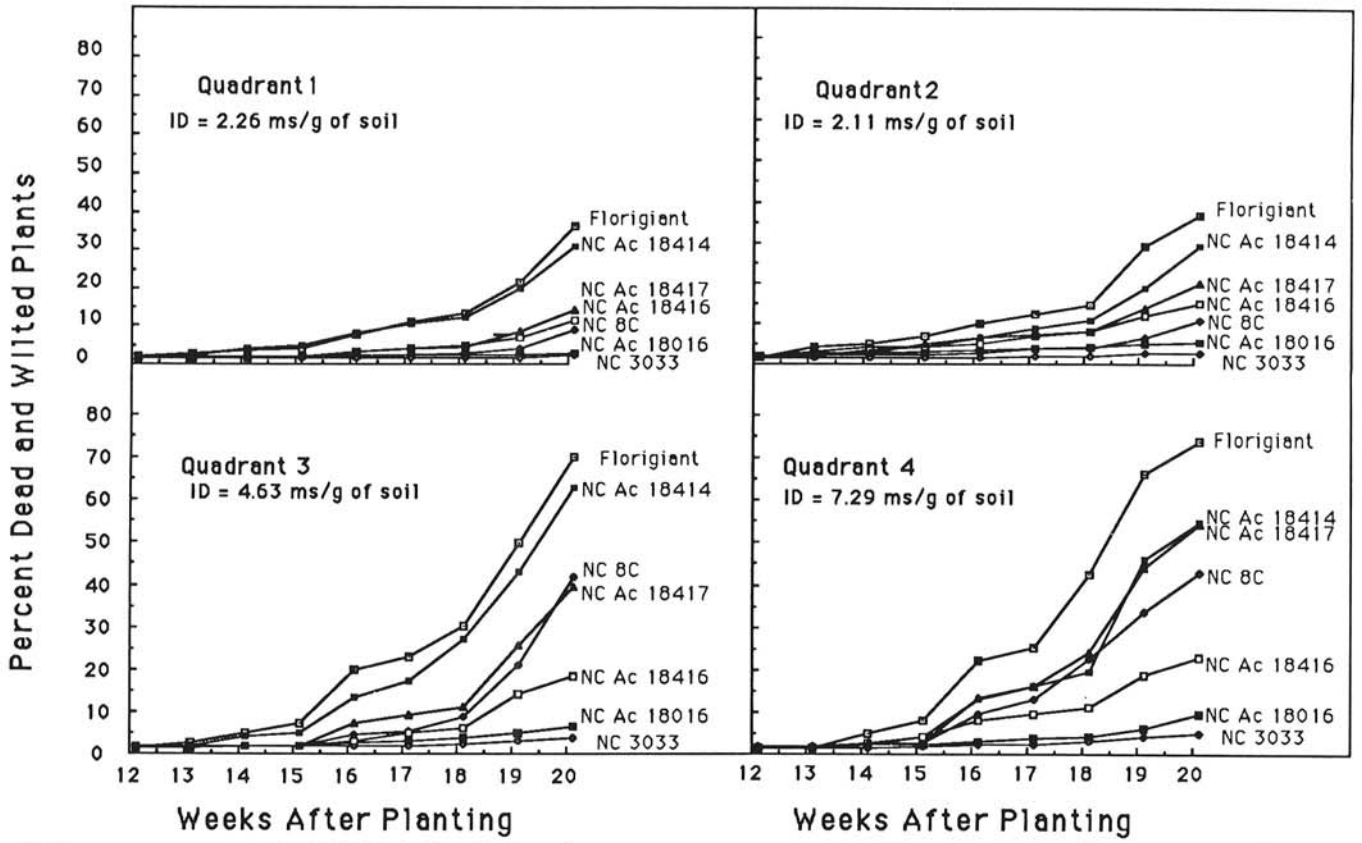

Fig. 2. Disease progress curves for Cylindrocladium black rot in seven peanut genotypes planted in quadrants with different initial inoculum density of *Cylindrocladium crotalariae* in 1986.

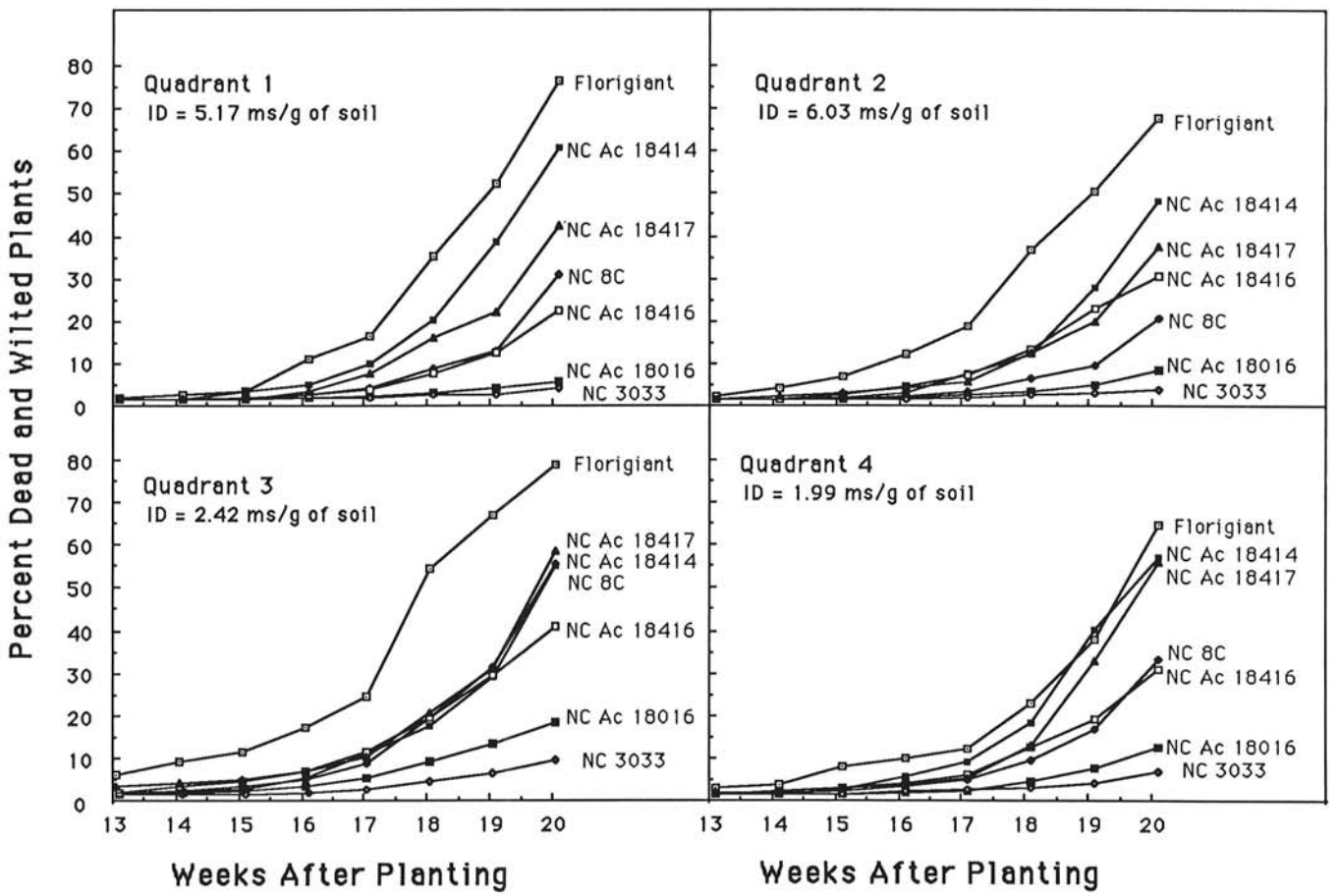

Fig. 3. Disease progress curves for Cylindrocladium black rot in seven peanut genotypes planted in quadrants with different initial inoculum density of *Cylindrocladium crotalariae* in 1987.

the logistic model best described disease progress in Florigiant, NC 8C, and NC Ac 18414 in quadrant 3 and for all four of these genotypes in quadrant 4. The logistic model also best described disease progress in Florigiant, NC 8C, NC Ac 18414, and NC Ac 18417 in all four quadrants in 1987 except for NC Ac 18417 in quadrant 2. In this exception, the Gompertz model gave the best fit. Overall, the Gompertz model was most appropriate for description of disease progress in NC Ac 18416 in both years, based on evaluation of plots of residuals, although R^2 values for the Gompertz and logistic models were similar. The Gompertz model best described disease progress in NC Ac 18416 in quadrants 1 and 4 in 1986 and in all individual quadrants in 1987. The monomolecular model described disease progress in NC Ac 18416 in quadrant 2 and the logistic model in quadrant 3 in 1986. The proportion of the variation in the data accounted for by any model was very low in NC Ac 18016 and NC 3033 in 1986. In 1987, overall disease progress of NC Ac 18016 and NC 3033 was best described by the Gompertz model.

Rates of disease progress as determined using the slope (k) of the logistic model (Table 2), were similar in Florigiant, NC 8C, NC AC 18417, and NC Ac 18414 across all quadrants in both years (Fig. 4). In these genotypes, rates were similar in each quadrant in both years except in quadrant 1 in 1986 where NC 8C had a rate slower than those of Florigiant and NC Ac 18414, and in quadrants 3 and 4 in 1987, where disease progress in Florigiant and NC Ac 18414, tended to be slower than in NC 8C, NC Ac 18417, and NC Ac 18414 (Table 2). Disease progressed more slowly in NC Ac 18416 than in these genotypes in both years. Within quadrants, rates of disease progress in highly resistant genotypes, NC Ac 18016 and NC 3033 were less than that in any genotype except NC Ac 18416. Rates of disease progress were higher for Florigiant, NC 8C, NC Ac 18417, NC AC 18414, and NC AC 18416 in quadrants 3 and 4 than in quadrants 1 and 2.

Overall incidence of Cylindrocladium black rot increased more rapidly in 1987 than in 1986 (Fig. 4, Table 2). However, rates of disease progress in quadrants with high average inoculum density were similar in 1986 and 1987 for Florigiant, NC 8C, NC Ac 18414, and NC Ac 18416. Rates in these genotypes in quadrants with low levels of inoculum were much higher in 1987 than in 1986 (Table 2). Disease progressed faster in both NC Ac 18016 and NC 3033 in 1987 than in 1986.

Epidemic onset, as determined by examination of the disease progress curves, intercept of the regression of logit transformed disease incidence on time, and time from planting until appearance of first symptoms, was delayed in all plots with more resistant genotypes. Specific results here and subsequent discussion will address time of appearance of symptoms, although all three methods showed similar trends. First symptoms in moderately resistant NC Ac 18417, NC Ac 18416, and NC 8C appeared an average of 1.5–2.6 wk later than in Florigiant in both years (Table 3). Epidemics in these genotypes began later than those in Florigiant in all quadrants in both years except in quadrant 4 in 1986 and quadrant 2 in 1987. In both cases, time until first symptoms for NC Ac 18417 and Florigiant was not different, although symptoms appeared slightly, but not significantly, later in NC Ac 18417. In 1986, time until first symptoms in NC Ac 18414 was not different from that of Florigiant. In 1987 black rot epidemics began later in NC Ac 18414 than in Florigiant in all quadrants. Except in quadrant 1, disease onset in NC Ac 18414 in 1987 was similar to that in NC 8C, NC Ac 18416, and NC Ac 18417. Epidemics were delayed further in NC Ac 18016 and NC 3033. In 1986, across all quadrants, epidemics began later in NC Ac 18016 than any other genotype except NC 8C and NC 3033. Appearance of first symptoms was later in NC Ac 18016 than in NC 8C across all quadrants and in quadrants 1 and 4 in 1987. Time until onset was greater in NC 3033 than in NC Ac 18016 across all quadrants in both years and in quadrant 1 in 1986 and quadrants 1,3 and 4 in 1987.

In 1986, when all plots were considered, rate of disease progress was correlated ($P = 0.05$, $n = 34$) with inoculum density of *C. crotalariae* in Florigiant ($r = 0.49$, $P = 0.01$), NC Ac 18417 ($r = 0.38$, $P = 0.02$) and NC Ac 18414 ($r = 0.49$, $P = 0.02$) across all quadrants. Positive correlation between these factors was detected in quadrant 1 in NC 8C ($r = 0.72$, $P = 0.03$). Significant correlation ($P = 0.05$) of rate of disease progress across all quadrants was not detected in any genotype in 1987. Correlations

TABLE 1. Coefficients of determination for fit of monomolecular, logistic, and Gompertz disease progress models to Cylindrocladium black rot epidemics in resistant and susceptible peanut genotypes in 1986 and 1987

	Model		
Genotype	Monomolecular	Logistic	Gompertz
1986			
Florigiant	0.20	0.65	0.48
NC 8C	0.16	0.42	0.37
NC Ac 18417	0.14	0.47	0.40
NC Ac 18414	0.18	0.63	0.47
NC Ac 18416	0.29	0.37	0.39
NC Ac 18016	0.11	0.09	0.10
NC 3033	0.12	0.12	0.13
1987			
Florigiant	0.41	0.68	0.58
NC 8C	0.25	0.68	0.54
NC Ac 18417	0.32	0.63	0.54
NC Ac 18414	0.39	0.77	0.68
NC Ac 18416	0.40	0.59	0.61
NC Ac 18016	0.26	0.41	0.43
NC 3033	0.35	0.33	0.37

TABLE 2. Slope (k), intercept (i), and estimates and coefficients of determination (R^2) for regression of logit [$\ln(y/(1-y))$] transformation of Cylindrocladium black rot incidence on time in seven peanut genotypes

	Quadrant 1			Quadrant 2			Quadrant 3			Quadrant 4		
Genotype	k	i	R^2	k	i	R^2	k	i	R^2	k	i	R^2
1986												
Florigiant	0.70(±0.06)	−7.2(±0.4)	0.62	0.64(±0.05)	−6.3(±0.3)	0.63	0.96(±0.06)	−7.4(±0.4)	0.75	1.13(±0.07)	−8.0(±0.4)	0.78
NC 8C	0.50(±0.09)	−8.2(±0.6)	0.35	0.61(±0.10)	−8.4(±0.1)	0.41	1.06(±0.09)	−10.1(±0.6)	0.71	0.91(±0.11)	−8.5(±0.7)	0.51
NC Ac 18417	0.65(±0.10)	−8.7(±0.6)	0.43	0.64(±0.10)	−7.5(±0.6)	0.40	0.98(±0.07)	−9.1(±0.4)	0.77	1.01(±0.10)	−8.5(±0.1)	0.62
NC Ac 18414	0.72(±0.07)	−7.2(±0.4)	0.62	0.55(±0.06)	−6.1(±0.3)	0.59	0.98(±0.08)	−8.0(±0.4)	0.71	0.99(±0.08)	−8.1(±0.5)	0.70
NC Ac 18416	0.59(±0.09)	−8.0(±0.6)	0.40	0.44(±0.11)	−6.3(±0.7)	0.21	0.80(±0.10)	−9.4(±0.6)	0.50	0.72(±0.10)	−7.6(±0.6)	0.48
NC Ac 18016	0.19(±0.15)	−6.9(±1.1)	0.21	0.27(±0.17)	−6.2(±1.2)	0.05	0.46(±0.16)	−7.8(±1.2)	0.16	0.60(±0.18)	−8.4(±1.3)	0.21
NC 3033	0.24(±0.09)	−8.3(±0.6)	0.15	0.43(±0.13)	−8.6(±1.0)	0.19	0.25(±0.16)	−7.7(±1.2)	0.05	0.58(±0.15)	−8.9(±1.1)	0.27
1987												
Florigiant	1.20(±0.07)	−8.1(±0.07)	0.80	1.03(±0.08)	−7.1(±0.5)	0.66	0.92(±0.07)	−5.4(±0.4)	0.67	0.84(±0.06)	−6.0(±0.3)	0.70
NC 8C	1.12(±0.09)	−10.0(±0.5)	0.74	1.00(±0.06)	−7.6(±0.4)	0.81	1.10(±0.10)	−8.4(±0.6)	0.67	0.98(±0.08)	−8.8(±0.5)	0.69
NC Ac 18417	1.15(±0.10)	−9.7(±0.6)	0.69	0.94(±0.09)	−8.3(±0.6)	0.66	1.01(±0.09)	−7.6(±0.6)	0.66	1.19(±0.11)	−9.3(±0.7)	0.64
NC Ac 18414	1.22(±0.08)	−9.4(±0.5)	0.78	1.10(±0.06)	−9.1(±0.3)	0.85	1.04(±0.06)	−8.2(±0.4)	0.79	1.16(±0.09)	−9.0(±0.5)	0.71
NC Ac 18416	0.94(±0.08)	−9.1(±0.5)	0.68	0.85(±0.09)	−7.9(±0.5)	0.62	0.81(±0.07)	−6.7(±0.4)	0.66	0.79(±0.08)	−7.4(±0.5)	0.59
NC Ac 18016	0.79(±0.15)	−9.7(±1.1)	0.40	0.86(±0.14)	−9.8(±1.0)	0.47	0.67(±0.13)	−7.2(±0.9)	0.38	1.07(±0.14)	−11.0(±1.0)	0.59
NC 3033	0.87(±0.16)	−11.1(±1.1)	0.42	0.67(±0.17)	−9.9(±1.2)	0.27	0.80(±0.12)	−9.0(±0.8)	0.53	0.77(±0.13)	−9.2(±1.0)	0.42

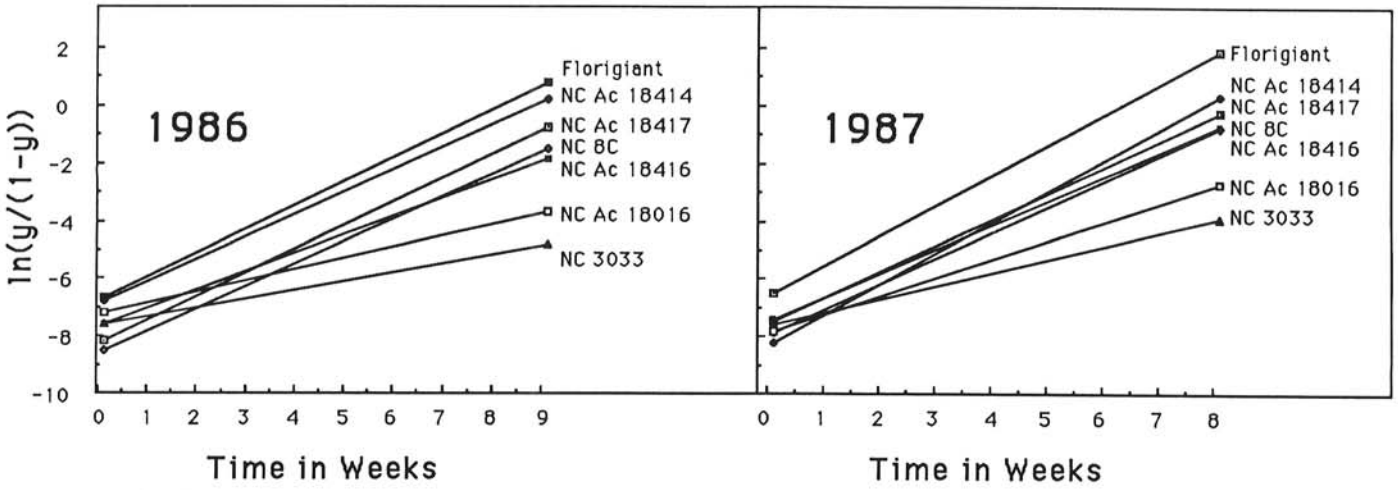

Fig. 4. Regression lines of logit $\ln[Y/(1 - Y)]$ transformation of Cylindrocladium black rot incidence on time in seven peanut genotypes in 1986 and 1987.

TABLE 3. Effect of resistance and susceptible peanut genotypes on time until observation of first symptoms of Cylindrocladium black rot in 1986 and 1987 field studies

	Weeks until first symptom				
Genotype	1[a]	2	3	4	Total
1986[b]					
Florigiant	14.6	13.6	14.1	14.1	14.1
NC 8C	17.0	16.1	16.4	15.6	16.7
NC Ac 18417	16.9	15.3	15.6	14.8	15.6
NC Ac 18414	14.7	13.9	14.4	14.6	14.4
NC Ac 18416	16.4	15.3	17.0	15.3	16.0
NC Ac 18016	18.1	16.4	17.2	16.7	17.1
NC 3033	20.0	19.0	18.8	17.4	18.9
LSD ($P = 0.05$)	2.3	2.0	1.5	1.7	0.96
1987[c]					
Florigiant	15.1	14.2	12.7	13.4	13.9
NC 8C	16.7	16.5	15.2	15.8	16.1
NC Ac 18417	16.6	15.5	14.4	15.6	15.5
NC Ac 18414	16.2	16.1	15.3	15.8	15.9
NC Ac 18416	16.6	16.0	14.7	15.3	15.6
NC Ac 18016	17.9	17.2	16.0	17.8	17.2
NC 3033	19.1	19.6	17.0	17.3	18.3
LSD ($P = 0.05$)	1.3	1.6	1.3	1.6	0.72

[a] Quadrant.
[b] Planting date = 2 May 1986.
[c] Planting date = 7 May 1987.

were detected in Florigiant ($r = 0.77$, $P = 0.02$) in quadrant 3, in NC Ac 18414 ($r = 0.76$, $P = 0.02$) in quadrant 4, and in NC Ac 18416 ($r = 0.76$, $P = 0.02$) in quadrant 2.

Initial inoculum density of *C. crotalariae* was negatively correlated with number of weeks from planting until appearance of first symptoms in NC Ac 18417 ($r = -0.36$, $P = 0.03$) across all quadrants and in NC Ac 18016 ($r = -0.80$, $P = 0.01$) in quadrant 4 in 1986. In 1987, correlation between time of onset and inoculum density was detected only in NC Ac 18414 ($r = -0.65$, P = 0.05) in quadrant 1, and in NC Ac 18016 ($r = -0.66$, $P = 0.05$) in quadrant 4.

DISCUSSION

Nonrandom or clustered inoculum is common for soilborne pathogens, and unless the spatial pattern of inoculum is taken into account, erroneous conclusions and interpretations may result from experiments on root disease epidemics (10). Final disease levels may be lower overall if inoculum is clustered than if evenly dispersed, and greatest variance in disease severity levels may be encountered (12). This problem may be of greatest importance in investigations of the effects of genotypes with different levels of partial resistance to a pathogen on disease progress. The peanut genotypes used in this study represented a range of levels of resistance to *C. crotalariae*. Even in genotypes with high levels of resistance, disease may progress rapidly if inoculum density is very high. By determining the pattern of inoculum in the field, and using inoculum estimates in our experimental design, we were able to address the effects of both genotype and inoculum density by characterization of disease progress curves.

Description of black rot disease progress in peanut by the logistic and model should not be interpreted as evidence for secondary spread of the pathogen but simply as a mathematical function describing the combined effects of many factors. Pfender (22), Campbell (12), and Campbell and Madden (11) discuss the invalidity of inference to the disease cycle from interpretation of the disease progress curve. Increasing incidence of black rot-symptomatic plants over time (i.e., disease progress) is a function of the number of infected roots, (inoculum dispersion and density related), stage of plant growth when infected, conduciveness of soil environment (moisture, temperature), and level of host resistance. Secondary reproduction and spread of the pathogen are not important in black rot disease development during a growing season in the field (28,31). The logistic model, however, described the increase in disease incidence in both susceptible and moderately resistant genotypes and was useful in description of the epidemics. The Gompertz model fit as well as the logistic model in many cases, and similarly, could have been used for comparison of the genotypes. Had comparisons been restricted to the most resistant genotypes in this study, the Gompertz model probably would have been a better choice than the logistic model.

Differences in disease progress between susceptible Florigiant and moderately resistant peanut genotypes appear to be due more to delay in the onset of epidemics in resistant genotypes than to reduction of rate of disease progress. Although reduction of rate of disease progress was observed in the moderately resistant genotype NC Ac 18416, and in other moderately resistant genotypes in specific quadrants, the effect of genotypes NC 8C and NC Ac 18417 on disease incidence was attributable to a delay in onset of the epidemic.

The delay of epidemics in NC 3033 and NC Ac 18016 compared with moderately resistant genotypes suggested that either fewer roots were infected or disease developed more slowly after root infection. In these genotypes and NC Ac 18416, however, rate of disease progress was also reduced. These differences may reflect a greater efficiency in the ability of these plants to minimize root disfunction by excluding the fungus from the vascular tissue. Mechanisms of resistance not found in NC 8C, NC Ac 18417, and NC Ac 18414 may occur in the highly resistant genotype. Further investigation into the effects of host genotype on rate of root colonization and decay are needed.

Regardless of the mechanisms involved, delay of onset of black rot epidemics is important in explanation of differences in performance of moderately resistant genotypes and Florigiant. Relative differences in onset were consistent regardless of initial inoculum or final disease incidence observed in each genotype. This delay of the black rot epidemic may be even more important when moderately resistant genotypes are used in conjunction with cultural (33) and chemical (3,13) control practices. Incorporation of similar resistance into earlier maturing genotypes might further exploit delay of the epidemics.

Resistance has been reported to be closely associated with the host's ability to "wall off" the invading pathogen to prevent colonization of vascular tissue and disruption of water transport (15,16,18). Although susceptible Florigiant has preformed periderm barriers that impede colonization of its root tissue by *C. crotalariae*, they are more easily breached by the pathogen in Florigiant than in highly resistant NC 3033 (15). Florigiant also forms additional periderm layers in response to invasion by the pathogen (18), but formation is slower than in NC 3033. Delay in appearance of symptoms in the resistant genotypes, in comparison with Florigiant, most likely is due, at least in part, to greater rate of formation and greater efficiency of barriers to exclude the pathogen. Differences in response in disease incidence (14) and rate of disease progress to increased inoculum density observed among some genotypes may be caused by differences in the number of infection sites required to overcome defensive barriers in individual root systems (14).

Both peanut genotype and inoculum density of *C. crotalariae* may affect disease progress of black rot. Similar relative positions of the disease progress curves for the different genotypes in 1986 and 1987 suggested that differences between genotypes were due primarily to level of resistance. Significant correlations and regression equations describing the relationship between final incidence and initial inoculum density for six genotypes in 1986 (14) suggested that of disease progress may also be affected by initial inoculum density. In a favorable environment, rate of disease progress may increase and time until onset may decrease with increasing inoculum for a particular genotype. Correlations within the individual quadrants serve to emphasize the importance of consideration of spatial patterns of inoculum as well as average density of inoculum in fields used for epidemiological studies. However, detection of correlations between inoculum and disease progress in this study was the exception rather than the rule. Hence, correlations that were detected can be used as evidence that these factors can be related, but do not indicate that such relationships are universal. Other factors in the field such as intraplot patterns of inoculum, range of inoculum density encountered, and other pathogens or environmental conditions that also may occur in a nonuniform pattern in the field may affect our ability to detect such relationships.

A mathematical description of the relationship between inoculum density and disease can be developed, even in soils with clustered inoculum, if the spatial pattern of the inoculum is taken into consideration (10). Correlations between incidence of black rot and density of inoculum of *C. crotalariae* have been detected (14,35). In this study, inoculum density was found to be negatively and positively correlated with onset and rate, respectively, of black rot epidemics in some cases. However, these correlations were not detected in the majority of genotype and quadrant combinations. Relationships between inoculum density and rate of disease progress of root diseases have been reported in other systems (1,2,26). Significant correlations between rate of black rot disease progress and inoculum density in some genotypes and quadrants indicated that rate was related to inoculum density if the combination of genotype, inoculum range, and environmental conditions were favorable. Level of effective inoculum has been proposed as a possible cause of delay of onset of Phymatotrichum root rot epidemics in cotton (32). Significant negative correlations between inoculum density of *C. crotalariae* and time until appearance of first symptoms in moderately resistant genotypes and NC Ac 18016 suggest that inoculum density may also influence time of occurrence of black rot epidemics.

Knowledge of the effects of moderate levels of resistance on disease progress can also help to ensure the optimal use of those genotypes for disease management. Characterization of these effects in this test in which inoculum effects were also considered helps ensure that differences observed were due to resistance and not due to large differences in inoculum encountered.

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