Analysis of Disease Progression and the Randomness of Occurrence of Infected Plants During Tobacco Black Shank Epidemics

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ABSTRACT

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The logistic, Gompertz, monomolecular, Bertalanffy-Richards ($n = 2$), and Weibull models were tested for goodness-of-fit to disease progression data for 50 epidemics of tobacco black shank induced by *Phytophthora parasitica* var. *nicotianae* between 1974 and 1980. Based on coefficient of determination (R^2) values and subjective evaluation of residual plots, the logistic model was most frequently appropriate (28 times), and the Gompertz was the next most frequently appropriate model (14 times) for describing these data. Runs analyses were performed on disease incidence data from three locations in 1980 and one location in 1981 to test for randomness of occurrence of symptomatic plants. A nonrandom pattern of symptomatic plants was found very infrequently (<10% of all cases examined) suggesting that *P. parasitica* var. *nicotianae* was not spreading along rows in a nonrandom fashion and resulting in symptom expression during the years of study. Since inoculum spread in this instance is probably not a plausible biological explanation for the appropriateness of the logistic model for describing disease symptom development during black shank epidemics, hypotheses concerning root expansion into inoculum sources and environmental effects are suggested to account for this appropriateness of the logistic model.

Black shank is a widespread, destructive disease induced in tobacco (*Nicotiana tabacum* L.) by *Phytophthora parasitica* var. *nicotianae* Dast. (Breda de Haan) Tucker (19). This disease is amenable to assessment for epidemiological investigations (8) because plants are spaced at regular intervals within regularly spaced rows under normal cultural practices and the pathogen induces characteristic visual symptoms on stems and leaves.

The quantitative analysis of plant disease epidemics induced by soilborne pathogens is reported infrequently in phytopathological literature. The hypotheses of Vanderplank (34), Bald (2), and the discussion of Zadoks and Schein (37) concerning root diseases induced by soil-inhabiting pathogens suggest that the monomolecular model, $dy/dt = r(1-y)$, may be appropriate for describing epidemics of root diseases and that these diseases may be of either the simple interest or monocyclic type. Campbell and Powell (8), however, evaluated both the logistic, $dy/dt = ry(1-y)$, and monomolecular models (34) for describing field epidemics of tobacco black shank and determined that the logistic model was more appropriate for describing these epidemics. Stack (33) concluded that common root rot of spring wheat and barley was more appropriately described by the logistic model. Campbell et al (7) also concluded that snapbean hypocotyl rot was not well described by the monomolecular model. No biological explanation has been provided for the results of the studies cited; however, Huisman (15) provided a biological interpretation for the logistic behavior of diseases induced by soilborne pathogens. Pfender (25) has recently emphasized the need to distinguish between the shape of the disease progress curve and the biological nature of the disease cycle.

In addition to an appropriate model for describing disease progression, knowledge of the type of disease pattern and spread within a field is vital in interpreting disease progression data (6) and in providing plausible biological interpretations concerning the appropriateness of disease progression models (4). Crowe and Hall (9) and Scott (29) obtained empirical evidence for plant-to-plant spread of mycelium of *Sclerotium cepivorum* inducing white rot of onion. Similarly, Huang and Hoes (14) found plant-to-plant spread of mycelium of *Sclerotinia* in stands of sunflower.

Morrall and Verma (22) suggest that the possibility of occasional secondary alloinfection between adjacent plants in a field by a soilborne pathogen could be investigated using Vanderplank's "doublet method." Madden et al (21) evaluated tests for randomness of infected plants including doublet analysis and concluded that ordinary runs analysis is the preferred method for determination of the pattern of infected plants. A random pattern suggests that at the time of observation the pathogen has not spread from one plant to an adjacent plant or, in the case of a soilborne pathogen, that inoculum distribution is random. A nonrandom pattern suggests that plant-to-plant spread has occurred or that inoculum is spatially aggregated. This nonrandom pattern of inoculum for *P. parasitica* var. *nicotianae* has recently been demonstrated by Ferrin and Mitchell (10).

The objectives of this study were to compare several growth models for describing the progression of disease symptoms during tobacco black shank epidemics and to determine if diseased plants occur randomly or nonrandomly within rows in the field over time.

MATERIALS AND METHODS

Disease progression. Regional small-plot tests were established yearly in North Carolina to evaluate tobacco breeding lines and commercial cultivars in soil naturally infested with *P. parasitica* var. *nicotianae*. Tobacco transplants (20–22 for each line or cultivar) were planted in each of three randomly located rows with a plant spacing of 0.56 m and 1.1 m between rows. An initial stand

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count was made 7–10 days after transplanting. The number of plants either severely (irreversibly) wilted or killed by black shank was recorded every 2 wk from 4–12 wk (1975 and 1978) or 4–14 wk (1974, 1976, 1977, 1979, and 1980) after transplanting. Plant death was attributed to black shank if the lower stem of an irreversibly wilted plant was blackened. Presence of regular disking of pith tissues was confirmed in randomly selected plants. Disease incidence was calculated as: total cumulative number of severely wilted or dead plants due to black shank/total plant number at base stand count. The 1974, 1977, 1979, and 1980 tests were performed at the black shank nursery at the Upper Coastal Plain Research Station at Rocky Mount, NC. The 1975, 1976, and 1978 tests were performed at the black shank nursery near the Oxford, NC, Tobacco Research Station.

Epidemics selected for analysis are given in Table 1. Criteria for selection of epidemics for analysis were: in most cases a final disease incidence (y_f) >0.50 (seven epidemics with y_f <0.50) and at least four intervals during the time of evaluation in which y was not zero.

Data were combined for the three rows of a line or cultivar within a year and disease incidence data were transformed using the transformation appropriate for the monomolecular, Gompertz, logistic, or Bertalanffy-Richards (n = 2) models (1,3,20). Transformed data were then tested for goodness-of-fit by the respective model using the General Linear Model procedure (PROC GLM) of the Statistical Analysis System (27,28). Coefficients of determination (R^2) and subjective evaluation of plots of residuals were used to indicate the appropriateness of a given model for describing the data. A specific model was considered inappropriate if a readily discernible pattern (eg, U-shaped or inverted U-shaped) was apparent in the plot of standardized residual values versus predicted values (23, especially pages 99–112). Disease progression data were also characterized by fitting the Weibull distribution function (24). The scale (b) and shape (c) parameters of the Weibull model were estimated by using a maximum likelihood technique after the location parameter (a) was set at the time when the first disease assessment was made, less 1 day.

Test for randomness of infected plants. Tobacco black shank was studied for randomness of infected plants in three fields in 1980 and in one field in 1981. The position and presence or absence of symptoms for each plant within each row was recorded. In 1980, assessments were made 13 times from 29 May to 20 August on four cultivars in a field of sandy loam soil in Robeson County, NC (Southern Coastal Plain). Tobacco cultivars utilized were Coker 319, NC 2326, Speight G-28, and McNair 944. The cultivars Coker 319 and NC 2326 have a low level of resistance to black shank; cultivars Speight G-28 and McNair 944 have a higher level of resistance to this disease. At this site, 10 replications of randomly arranged two-row plots were planted for each cultivar. Row width was 1.2 m with a plant spacing of 0.54 m. At one field in Edgecombe County, NC (Upper Coastal Plains Research Station, sandy loam soil) similar data were collected 12 times on 16-plant rows from 15 May to 5 August 1980 for each of the four cultivars mentioned above. At this location, row spacing was 1.1 m with 0.56 m between plants and five replicates were used. Similar data were collected on 27 June and 31 July 1980 and on 25 May, 15 June, and 15 July 1981 on each 20-plant row of each cultivar in the Regional Small Plot Test at Rocky Mount, NC. Standard commercial practices for tobacco culture were followed at all locations.

Disease progression in fields used for runs analyses was analyzed using procedures similar to those indicated above. Data from the replicates were averaged prior to fitting of disease progress models.

Initial inoculum density as determined by a selective medium assay utilizing a wet-sieving extraction procedure (18,32) ranged from 0.04 to 0.33 propagules per gram dry soil (ppg) with a mean of 0.21 ppg at the Robeson County site and from 0.09 to 0.16 ppg with a mean of 0.13 ppg at the Edgecombe County site. This inoculum density for *P. parasitica* var. *nicotianae* was sufficient to induce black shank epidemics under field conditions (18). Initial inoculum density was not determined for the Regional Small Plot test.

Disease incidence data were analyzed for each row and date by using ordinary runs analysis (22). A run was defined as a succession of one or more identical symbols, which are followed or preceded by a different symbol or no symbol at all in an ordered sequence of two types of symbols (12), eg, diseased and not diseased. Runs analyses were performed on each row in the Edgecombe and Robeson County fields. Runs analyses were also performed on all

TABLE 1. Final disease incidence, number of non-zero intervals for y (change in disease), acceptable fits of models to disease progression data and Weibull model b and c parameter estimates for 50 epidemics of tobacco black shank induced by *Phytophthora parasitica* var. *nicotianae*

Line or cultivar	Final disease incidence	No. of non-zero points for y	Model(s) giving acceptable fit[y]	Weibull parameter estimates	
				b	c
1974					
Hicks	0.97	5	G,L	49.97	2.82
NC 2326	0.27	5	B,G,L	109.50	2.85
Virginia Gold	1.00	5	B,G,L,M	35.92	2.03
1975					
Coker 319	0.60	5	-[z]	78.83	3.10
Coker 347	0.38	5	M	117.81	1.66
McNair 944	0.27	4	B,G,L,M	117.70	2.47
NC 79	0.36	5	B,G,L,M	96.68	2.26
NC 95	0.32	5	B,M	150.78	1.26
NC 2326	0.60	5	B,G,L	80.21	2.25
Virginia 115	0.35	5	-	95.08	3.30
1976					
Hicks	0.93	6	L	82.01	3.45
1977					
Coker 86	0.68	5	L	75.40	3.38
Coker 347	0.71	6	L	75.43	3.39
Hicks	0.78	5	L	58.73	2.58
NC 79	0.91	6	L	59.13	2.96
NC 95	0.60	5	L	79.04	2.82
Speight G140	0.67	6	-	77.95	3.24
Virginia Gold	0.91	6	B,G,M	49.86	1.60
Virginia 115	0.92	6	-	66.42	4.27
1978					
Coker 298	0.43	4	G,L	67.76	3.73
Coker 347	0.93	5	L	49.31	3.11
Coker 411	0.72	4	G	54.41	2.58
McNair 944	0.55	4	L	60.69	6.88
NC 95	0.93	4	L	47.92	4.09
NC 2326	0.88	4	B,G	43.67	2.18
Speight G140	0.87	4	L	53.53	5.37
1979					
Coker 78-98 mm	0.82	6	L	72.30	2.99
Coker 411	0.68	5	G,L	101.06	2.27
Hicks	1.00	4	G,L,M	38.16	1.61
McNair 3207	0.66	6	G,L	88.50	2.40
NC 95	0.69	6	-	76.39	3.36
NC 744	0.84	6	L	76.72	2.39
NC 2326	0.71	6	B,G,L	90.21	1.71
NC 7537	0.89	5	-	83.81	2.83
NC 7556	0.68	6	-	81.21	3.69
Speight G140	0.56	5	-	90.62	2.51
1980					
Coker 48	0.68	4	-	59.64	8.04
Coker 79-211S	0.74	5	G,L	52.83	4.54
Coker 298	0.65	5	-	67.50	5.70
Coker 347	0.91	5	-	56.38	6.26
Coker 411	0.79	5	-	56.16	4.35
Coker 7651	0.63	4	-	60.45	6.64
NC 67 USDA	0.58	5	-	66.56	3.64
NC 89	0.98	5	L	45.80	3.77
NC 95	0.93	5	L	48.86	4.62
NC 2326	0.98	5	L	47.50	4.15
PD 81	1.00	5	-	45.79	3.82
Reams 8201	0.97	5	L	46.96	5.16
Speight G140	1.00	5	-	47.36	4.98
Virginia 79	1.00	5	L	46.34	3.61

[y] The models used were the integrated forms of the following: logistic (L) $dy/dt = ry(1-y)$, Gompertz (G) $dy/dt = ry(-\ln y)$, monomolecular (M) $dy/dt = r(1-y)$, Bertalanffy-Richards (B) $dy/dt = ry(1-\sqrt{y})$, Weibull $dy/dt = c/b\,[(t-a)/b]^{c-1} \cdot e^{-[(t-a)/b]^c}$.

[z] Hyphen indicates no model gave an acceptable fit to the data.

rows in the Regional Small Plot test in which at least four and not more than 18 diseased plants occurred. Runs analyses were performed by using the Minitab Statistical Computing System (26). In cases where the number of plants was less than 20 ($N<20$), tables (12,13) were used for determining level of significance; minimum sample size was 16 plants. A significance level of $P=0.05$ was used in all tests.

RESULTS

Disease progression. Final disease proportion (y_{max} = 1.00) varied among years and cultivars (Table 1). The logistic model was the model most frequently judged to be appropriate for describing disease progression data. The logistic model was found to describe adequately the data 28 times, the Gompertz 14, the Bertalanffy-Richards nine, and the monomolecular eight times (Table 2) based on coefficients of determination and residual plot evaluation. Coefficients of determination (R^2) were consistently above 90% for the selected model. In 1974 and 1975, it was difficult to choose the most appropriate model; in most cases from these years more than one model or even all of them statistically described the data equally well. In 1977 and 1978, this problem was not encountered, but in 1980 only six of 14 epidemics were adequately described by one of the nonflexible models considered.

When a cultivar or line appeared in three or more years in regional small plot tests, an attempt was made to compare the appropriateness of particular models for consistently describing the disease progression data over years. If one model regularly described disease progression on one cultivar while another model was consistently more appropriate for use with another cultivar, this could indicate a basic difference in the characteristics of disease progression between the two cultivars. In three of five instances that were considered, the logistic model predominated, while for NC 2326 the Gompertz model was consistently the best choice (Table 3).

A range of Weibull model shape parameter (c) values was obtained. In cases where a monomolecular model was considered appropriate for several curves, c was generally low and the upper limit was also low. Also, when the logistic model predominated as a model choice (1977–1980) greater c values were included in the range of c values (Table 1).

Disease progression of black shank in 1980 (Fig. 1) in fields used for runs analyses (Edgecombe and Robeson counties) were consistently well described by the Gompertz model (Table 4). In several cases, other models were also appropriate. Disease progressed more rapidly, as indicated by rate parameter values, on cultivars in Robeson County than on cultivars grown in Edgecombe County.

Randomness of infected plants. In most cases, infected plants occurred at random within rows at all dates examined. In the Edgecombe County field (2 July–6 August 1980) in only three instances out of 240 were the number of observed runs within a row less than the number of runs expected given the disease incidence level (Table 5). In the Robeson County field there were several cases in which there were fewer runs than expected for random disease occurrence; however, 12 of 22 cases could be accounted for as the occurrence of only two adjacent plants exhibiting symptoms within the row. Two adjacent plants exhibiting symptoms will consistently indicate a departure from randomness in the runs analysis. In the Regional Small Plot tests which included a number of cultivars and lines, results for 1980 and one sample in 1981 were similar to those from other sites. In general, fewer than 10%, and in most cases fewer than 5%, of all rows of plants analyzed had a nonrandom occurrence of diseased plants as indicated by the number of runs observed being less than the number expected (Table 5). Nonrandom occurrence of plants within a row could not be attributed to any one particular cultivar or line, but occurred (for example) in three different cultivars in the Edgecombe County field on 9, 16, and 23 July 1980.

TABLE 2. Frequency of models giving acceptable fit to tobacco black shank disease progress data and Weibull parameter estimates for epidemics

Year	Curves analyzed	Number of times model gave acceptable fit			
		Logistic	Gompertz	Mono-molecular	Bertalanffy-Richards (n = 2)
1974	3	3	3	1	2
1975	7	3	3	4	4
1976	1	1	0	0	0
1977	8	5	1	1	1
1978	7	5	3	0	1
1979	10	6	4	1	1
1980	14	5	0	1	0

TABLE 3. Model judged most appropriate for describing tobacco black shank disease progress curve when a cultivar was present in analysis for at least three years

Cultivar	Year						
	1974	1975	1976	1977	1978	1979	1980
Hicks	L or G[y]	–[z]	L	L	–	LGM	–
NC 2326	B,L,G	G	–	–	G	L or G	+
NC 95	–	M	–	L	L	+	L
Coker 347	–	M	–	L	L	–	+
Coker 411	–	–	–	–	G	L or G	+

[y] L = logistic, G = Gompertz, M = monomolecular, B = Bertalanffy-Richards (n = 2); when two or more models are listed, we could not distinguish between appropriateness of the models based on coefficient of determination and residual plot.

[z] A (–) indicates that a cultivar was not present in the data analyzed for that year; a (+) indicates the presence of the cultivar in the test, but no model gave an acceptable fit.

DISCUSSION

Changes in characteristics of disease progression are related to increase or decrease in amount of available inoculum, alteration of host susceptibility or a change in the conduciveness or suppressiveness of the environment for disease development. For some epidemics induced by foliar pathogens, the effects of altering one disease component can be fairly well predicted and an

TABLE 4. Characterization by least squares regression analysis of disease progression data in 1980 for four tobacco cultivars in two locations utilized in runs analyses

Location in NC and cultivar	Acceptable model[x]	b_0[y]	b_1[y]	$S(b_1)$[y]	R^2
Edgecombe County					
Coker 319	Gompertz	−2.11	0.041	0.0022	97.0
NC 2326	Bertalanffy-Richards[z]	−0.68	0.034	0.0020	96.3
	Gompertz	−2.27	0.044	0.0029	95.6
Speight G28	Gompertz	−2.63	0.041	0.0030	94.6
McNair 944	Gompertz	−2.38	0.037	0.0020	95.4
Robeson County					
Coker 319	Gompertz	−2.94	0.059	0.0040	96.5
NC 2326	Mono-molecular	−0.79	0.033	0.0020	96.3
	Bertalanffy-Richards	−0.81	0.041	0.0020	97.9
	Gompertz	−2.68	0.056	0.0031	97.0
Speight G28	Gompertz	−2.98	0.050	0.0020	98.7
McNair 94	Gompertz	−3.09	0.051	0.0029	96.7

[x] Model acceptance was judged on magnitude of coefficient of determination (R^2) value and presence or absence of a readily discernible pattern in the plot of standardized residuals versus predicted values.

[y] b_0 is an estimate of the y-intercept, b_1 is an estimate of the slope, and $S(b_1)$ is the standard error of the estimate b_1. b_1 represents the rate parameters for each model.

[z] Bertalanffy-Richards model with n = 2.

explanation found for differences in disease progress curve characteristics (5,30,35,36). Our understanding of the relationships among factors which influence epidemics of root diseases induced by soilborne pathogens is not as complete. Thus, these initial analyses of disease progression for tobacco black shank may provide testable hypotheses for future research.

Inoculum of *P. parasitica* var. *nicotianae* is not present in soil in a static reservoir. Rather, populations of this pathogen increase greatly in the rhizosphere of host plants during the growing season (11,18). Black shank is, therefore, potentially not a monocyclic disease during a growing season.

The role of spatial aggregation or nonrandom occurrence of inoculum of *P. parasitica* var. *nicotianae* recently reported by Ferrin and Mitchell (10) in determining pattern of symptomatic plants within rows is unclear. Our results from ordinary runs analyses on data of the pattern of symptomatic plants indicate a random occurrence of disease within rows under the conditions of our study. This implies that the pathogen occurs randomly or at least that disease is expressed randomly within rows. The scale of spatial aggregation may be a determining factor. Future information on this scale would be useful.

Our results also imply that the pathogen is not spreading down rows or that if it spreads down rows, it does not induce symptoms in a nonrandom manner. Unlike the soilborne pathogens studied by Crowe and Hall (9), Scott (29), and Huang and Hoes (14) in which fungal mycelium was the principle means of primary infection and

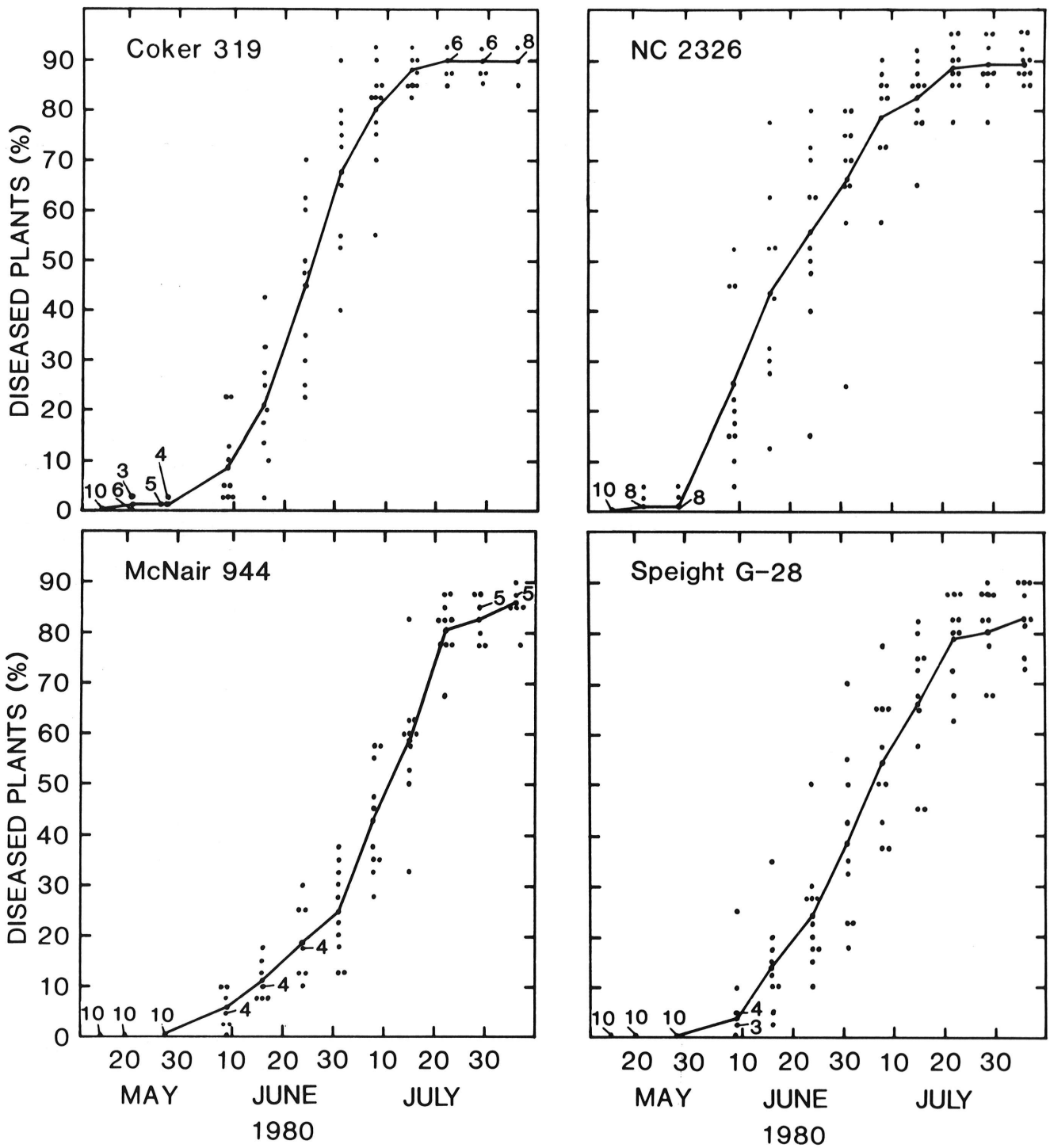

Fig. 1. Disease progression of black shank on four tobacco cultivars in Robeson County, NC, in 1980. Each point represents disease incidence in a given replication. The solid line represents the mean disease for the ten replications.

pathogen spread, the primary infectious and transportable propagule of *P. parasitica* var. *nicotianae* is the zoospore which requires free water for movement (19). The sites where our data were collected were well drained and standing water would not normally be present for long periods of time in the field during the growing season. When soil flooding occurred, however, inoculum could be spread down the rows. Shew (31) recently reported that *P. parasitica* var. *nicotianae* is capable of spreading down a row from a point source of host plant inoculation during one growing season in the absence of cultivation. He found that although tobacco plants down the row from an inoculated plant were infected by *P. parasitica* var. *nicotianae*, not all infected plants were symptomatic. Distance of spread was related to level of host resistance to black shank. If zoospore movement occurred in our study, deposition of zoospores and resultant infection sites must have been random within rows or the lack of symptom expression made the effect of this zoospore movement indiscernible.

A biological explanation of the appropriateness of the logistic model for describing epidemics induced by foliar pathogens is the polycyclic increase of pathogen inoculum during a growing season (34). Given the results of our runs analyses, however, the possible spread of inoculum down a row without the induction of symptoms would not account for the general appropriateness of the logistic model for describing disease progression for tobacco black shank. Two other explanations are plausible. One is that disease incidence increases over time as host roots grow out into infested soil and encounter increasing amounts of inoculum. The other would be that the time of infection for plants is of relatively short duration, but the expression of disease symptoms is environmentally conditioned and is variable, ie, incubation times vary.

Roots of many plants elongate in a manner which can be described by the logistic growth model (15). Although information is not specifically available on the growth of tobacco roots in our study, as roots ramify through soil, the likelihood of infection from an encounter with a propagule of *P. parasitica* var. *nicotianae* increases. The greater the volume of soil occupied by the root system, the greater the probability of infection. Thus, as the season progresses, more and more roots and, therefore, plants will become infected. Although it is not known how many specific infection sites must occur or what the location of such sites must be for symptom expression to occur, it can be hypothesized that if a "critical" root or a "critical" number of roots is infected, typical black shank symptoms will be induced. Huisman (15) has suggested that if root growth follows the logistic growth model, disease progress may also be described by this growth model. We believe this may be the case for tobacco black shank.

TABLE 5. Runs analyses to test for randomness of occurrence of plants within rows exhibiting symptoms of tobacco black shank at three locations

Date plant count made	No. of rows with observed runs <expected runs ($P = 0.05$)	No. of rows with observed runs not <expected runs ($P = 0.05$)	Average disease incidence (%)
Edgecombe County (Rocky Mount)			
2 Jul 1980	0	40	18
9 Jul 1980	1	39	31
16 Jul 1980	1	39	47
23 Jul 1980	1	39	60
30 Jul 1980	0	40	67
6 Aug 1980	0	40	77
Robeson County			
9 Jun 1980	7	77	11
16 Jun 1980	7	77	22
24 Jun 1980	8	76	35
1 Jul 1980	4	80	47
8 Jul 1980	4	80	62
15 Jul 1980	3	82	73
22 Jul 1980	0	84	83
Edgecombe County (Rocky Mount RSP Test)			
27 Jun 1980	2	43	48
31 Jul 1980	6	57	65
15 Jul 1981	6	66	43

The environmental hypothesis which Jacobi et al (17) proposed to account for the increase in symptom expression (disease incidence) over time, states that the plants are infected by *P. parasitica* var. *nicotianae* during the first 6–8 wk after transplanting into the field and that environmental conditions (usually in the form of water stress) become increasingly favorable for symptom development after the initial 6- to 8-wk period. Concomitant with the period of environmental stress is the physiological maturation of the host. The combination of these factors may be responsible for the rapid increase of tobacco black shank incidence which results in the appropriateness of a model which fits a sigmoid-shaped curve for describing disease progression.

The most suitable hypothesis may be the simultaneous operation of the above-mentioned mechanisms. This would provide a plausible biological/environmental explanation for the appropriateness of the logistic model for describing progression of disease incidence of tobacco black shank over several years on a number of breeding lines and commercial cultivars.

In this study, disease progression for cultivar NC 2326 was better described by an asymmetrical growth function (Gompertz) while for other cultivars (Hicks, NC 95, and Coker 347) the logistic model was more appropriate (Table 3). Differences were due to cultivars and not to growth conditions within a year. The appropriateness of a symmetric (logistic) or asymmetric (Gompertz) growth function may be due to differences in plant root growth, plant maturation, host response to environment, or other factors. The possible consistent differences in appropriateness of a growth model due to cultivar effects is intriguing and requires further investigation.

A knowledge of the appropriateness of certain models for describing disease progression and the biological/environmental explanations for the suitability these models will lead to a better understanding of epidemic development. This should enhance ability to forecast diseases and, ultimately, contribute to the development of better management strategies. Epidemics of root diseases may not differ in toto from epidemics of foliage and shoot diseases, but different explanations may be appropriate to account for similar phenomena in shoot and root pathosystems.

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