Analysis of Progress of Alfalfa Leaf Spot Epidemics

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This research supported in part by a grant from the National Crop Loss Design Committee, USDA-CSRS.
Paper No. 10418 of the Journal Series of the North Carolina State Agricultural Research Service, Raleigh 27695-7601.
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We would like to thank K. J. Leonard, R. I. Bruck, and R. M. Moll for helpful comments in the preparation of this manuscript. We also thank J. P. Mueller, J. T. Green, and Dorsey Daniel for their cooperation in the experiments and Charles R. Harper, Brenda Dorman, Judy Sinn, Belinda Gaskins, and Angie Dorman for their assistance.
Accepted for publication 2 September 1987 (submitted for electronic processing).

ABSTRACT

Thal, W. M., and Campbell, C. L. 1988. Analysis of progress of alfalfa leaf spot epidemics. Phytopathology 78:389-395.

The progress of alfalfa leaf spot diseases, caused primarily by *Leptosphaerulina briosiana*, was monitored to compare characteristics of disease development among five cultivars in successive growth cycles within a growing season. The rate of disease progress and the shape of the disease progress curve were generally similar among cultivars but varied among growth cycles. Disease severity often decreased toward the end of an epidemic within a growth cycle. Defoliation and accumulation of debris toward the end of epidemics generally corresponded to decreased levels of visible disease near the time of harvest. A simple theoretical simulator, constructed to examine some of the unique factors that appear to influence disease development in the alfalfa leaf spot pathosystem, indicated that defoliation was not the sole cause of the decreased disease severity at the end of an epidemic.

Little information is available on the progress of alfalfa leaf spot diseases. Alfalfa (*Medicago sativa* L.) is a genetically heterogeneous, perennial crop. Stand life generally ranges from 4 to 6 yr, and the crop is harvested three to six times per year (3). This results in multiple growth cycles, which allow several epidemics to occur within a single growing season (15), and diseased tissue from one epidemic may serve as primary inoculum for a subsequent epidemic.

Several pathogens are capable of causing leaf spot diseases on alfalfa (2). *Stemphylium botryosum* Wallr., *Phoma medicaginis* Malbr. & Roum. var. *medicaginis* Boerema, *Leptosphaerulina briosiana* (Poll.) Graham & Luttrell, and *Cercospora medicaginis* Ell. & Ev. are the predominant leaf-spotting pathogens on alfalfa North Carolina (16). *S. botryosum, P. medicaginis*, and *L. briosiana* often produce similar symptoms in the field. Several of these pathogens may be isolated from the same leaf spot (10). *C. medicaginis* produces leaf spots that are usually distinct from those caused by the other three. *Pseudopeziza medicaginis* (Lib.) Sacc. also produces distinct lesions, but it is not commonly observed in North Carolina. The pathosystem is, thus, a complex one, involving at least four major diseases; however, we have considered the alfalfa leaf spot pathosystem as a whole, because the diseases often are symptomatically similar and are present simultaneously during different times in the growing season.

The alfalfa leaf spot pathosystem has several unique qualities. Plant growth is rapid and continuous throughout the epidemic, which may be shorter than 30 days. Defoliation, which occurs both naturally and as a result of leaf spot diseases (6), affects the observable disease severity. Defoliation may have an indirect effect on epidemics, since plant growth in subsequent growth cycles can be affected by the loss of photosynthetically active tissue in the current growth cycle. Sporulation occurs mainly on abscised leaves or, at least, on leaves that are senescing, particularly for leaf spots caused by *L. briosiana* (8). Older leaves are generally less susceptible to infection by *L. briosiana* than younger leaves (7).

The objectives of the present research were to characterize the progress of alfalfa leaf spot diseases; to examine the relationships among disease severity, defoliation, and accumulation of debris within an epidemic; and to compare epidemics among growth cycles and cultivars.

MATERIALS AND METHODS

Experimental design and data collection. Five cultivars were selected for study, on the basis of leaf spot resistance (14). The cultivars were ranked as most susceptible (Arc, WL 318), moderately susceptible (Cimarron, Pioneer 531), and least susceptible (Raidor) to leaf spot diseases present in North Carolina.

Experimental plots at two locations in Wake County, North

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Carolina, were established to monitor the progress of alfalfa leaf spot diseases. Five cultivars (Arc, Cimarron, WL 318, Pioneer 531, and Raidor) were assessed at the first location (Wake 1), and three cultivars (Arc, Cimarron, and WL 318) were assessed at the second location (Wake 2). The two locations were separated by approximately 12 km and had similar soil types.

Plots were arranged as three parallel rows, planted on 23-cm centers and separated from other plots by 31 cm. Plots at Wake 1 were established on 31 August 1983 and seeded at a rate of 28 kg/ha. Plots at Wake 2 were established on 8 September 1982 and seeded at a rate of 32 kg/ha. Plots at Wake 1 were 6.1 m long, and plots at Wake 2 were 1.8 m. Standard cultural practices for alfalfa were followed at both locations.

The experimental design was a randomized complete block with five replications at each location. The sampling unit for each location was a 30-cm section of the center row. Plots at Wake 1 were divided into two strata, and two units were randomly sampled from each stratum (Fig. 1). Two randomly selected units were sampled from each plot at Wake 2. Plots were generally sampled at 5- or 7-day intervals during the period from March to October, during 1984 and 1985. Individual plots were mechanically harvested when plants reached approximately 10% bloom. Harvested plant material was removed from the field at the time of cutting. Assessments were resumed after each harvest when regrowth was visible.

Several measurements were obtained for each sampling unit. A visual estimate of disease severity, based on the Horsfall-Barratt rating system (4), was made for the entire section of canopy when plants were small; separate ratings were obtained for the lower and upper halves of the canopy when plants were larger. Plant height and the total stem length defoliated were measured. The percentage of ground covered by leaf debris between the rows was estimated, using the Horsfall-Barratt scale. On each sampling date, the average growth stage of plants was recorded (5).

Stems were removed from experimental plots on sampling dates throughout the 1984 and 1985 growing seasons. Diseased leaves were incubated in moist chambers under fluorescent lights for 3 to 5 days at room temperature. Leaves were then observed at 70× magnification for identification of leaf-spotting fungi. In some cases, pathogens were isolated by transferring diseased leaf tissue to acid water agar.

Analysis of disease and host data. The area under the disease progress curve (AUDPC) (1) was estimated for epidemics within each growth cycle. The area under the curve was also calculated and analyzed for plant height, defoliation, and debris. Analysis of variance was performed on the total AUDPC for each year at each location, to compare cultivars. The Waller-Duncan k-ratio t-test ($k = 100$) was used for mean separation of cultivars in cases where the overall F-test for a factor was significant (13).

Correlations between plant height, lower disease severity, upper disease severity, the percentage of total stem length defoliated, and the percentage of ground area covered by debris were calculated. Overall disease severity was not included in these analyses, since it was calculated as the average of the lower and upper disease severities. The correlations were based on the area under the curve for each variable and were calculated for the combined data from Wake 1 and Wake 2 during 1984 and 1985. A hierarchical clustering procedure was then used to group related variables, with the SAS procedure VARCLUS (12) and the centroid option. The procedure forms clusters by splitting clusters until each cluster has only a single eigenvalue greater than 1.

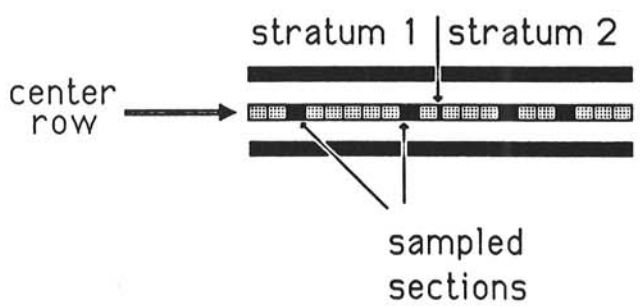

Fig. 1. Subsampling procedure for individual plots at location Wake 1. Two 30-cm sections were sampled from each half (stratum) of the center row of a plot.

Simulated disease progress. A deterministic simulation model was constructed to examine interactions between plant growth, defoliation, debris accumulation, and disease progress for alfalfa leaf spot. The rate of disease increase was varied to determine its effect on these other parameters. The simulator used a daily time step and is summarized in the flow diagram in Figure 2.

Plant growth and disease increase were modeled with a discrete form of the equation

$$dy/dt = rd_0(0,y)d_1(y,1)$$

where y is the proportion of maximum disease severity or plant size, dy/dt is the absolute rate of increase, r is a rate constant, d_0 can be thought of as a driving function, and d_1 as a limiting function (9). Plant growth was modeled with the logistic equation where $d_0 = y$ and $d_1 = (1 - y)$. Disease increase was modeled with the level of disease on defoliated leaves as the driving function ($d_0 = Y_{def}$), since this is considered to be the main source of inoculum. The limiting function was the amount of healthy tissue on an individual leaf ($d_1 = 1 - Y_{leaf}$).

Defoliation occurred when a combination of leaf age and disease

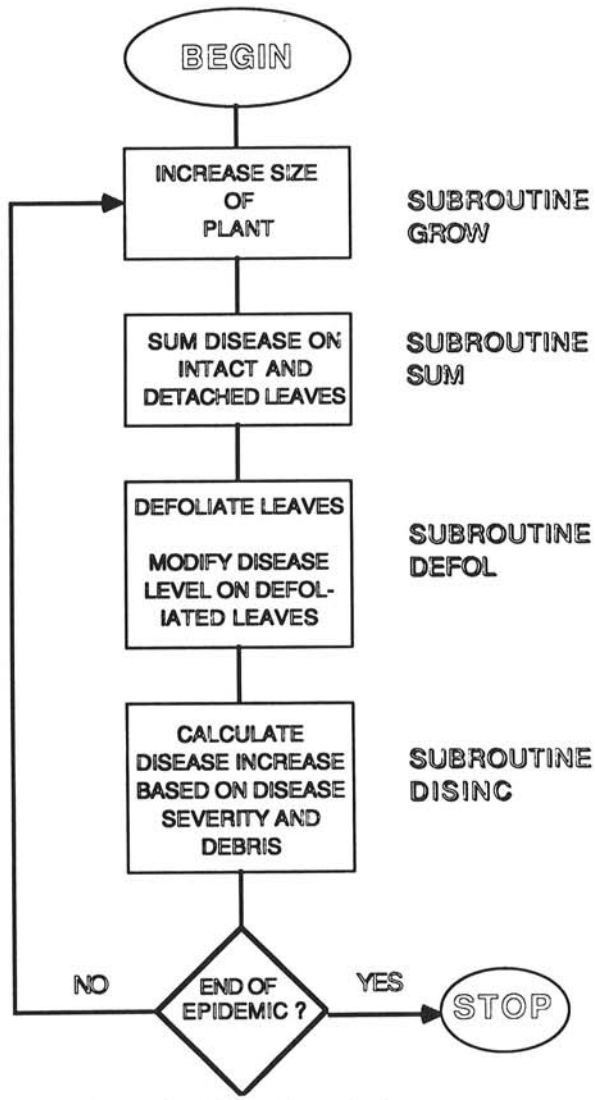

Fig. 2. Flow diagram for alfalfa leaf spot simulator.

TABLE 1. Occurrence of leaf-spotting pathogens on alfalfa at two locations in Wake County, North Carolina, during 1984 and 1985

Date	Location	Relative lesion size[a]	Pathogen				
			Leptosphaerulina briosiana	*Stemphylium botryosum*	*Phoma medicaginis*	*Cercospora medicaginis*	*Pseudopeziza medicaginis*
4/84	Wake 1	1, 2	×[b]		×		
	Wake 2	1	×		×		
5/84	Wake 1	1–3	×	×	×		×
	Wake 2	1–3	×		×		
7/84	Wake 1	2, 3	×	×		×	
	Wake 2	2, 3	×			×	
8/84	Wake 1	2, 3	×				
	Wake 2	1, 3	×			×	
9/84	Wake 2	1–3	×			×	
4/85	Wake 1	1, 2	×	×			
	Wake 2	1, 2	×		×		
5/85	Wake 2	1–3	×	×	×		
6/85	Wake 1	1	×				
	Wake 2	1, 2	×	×	×		
8/85	Wake 1	2, 3	×			×	
	Wake 2	1–3	×			×	

[a] Relative lesion size on a scale of 1 to 3: 1 = pepper spot; 2 = slightly enlarged spot (< 1 mm in diameter); 3 = expanded spot (approximately 1 mm or greater in diameter).
[b] × denotes the detection of the pathogen in leaf samples with characteristic leaf spots. None of these pathogens was successfully isolated during 6/84 at Wake 1 or Wake 2 or during 5/85 at Wake 1. No isolations were made during 7/85.

TABLE 2. Mean severity of alfalfa leaf spot on lower and upper halves of the canopy of five alfalfa cultivars at location Wake 1 during each growth cycle in 1984 and 1985

Year	Growth cycle	Portion of canopy[a]	Arc	Cimarron	Pioneer 531	Raidor	WL 318
1984	1	Lower	3.9	5.0	3.5	4.3	5.9
		Upper	0.7	0.9	0.7	0.7	1.4
	2	Lower	9.0	9.2	9.5	7.3	7.4
		Upper	2.4	2.8	2.3	1.9	2.4
	3	Lower	4.5	4.3	4.6	4.1	3.9
		Upper	0.9	0.8	0.8	0.7	0.7
	4	Lower	16.8	13.9	14.3	12.1	12.2
		Upper	2.1	2.1	2.0	1.6	2.0
	5	Lower	2.8	2.3	3.2	1.9	2.6
		Upper	1.2	1.1	1.2	1.0	1.2
	6	Entire[b]	0.6	0.4	0.4	0.4	0.6
1985	1	Lower	2.3	2.4	2.2	2.4	2.3
		Upper	1.2	1.2	1.2	1.2	1.3
	2	Lower	15.3	16.7	15.8	16.2	16.5
		Upper	0.9	0.9	0.8	0.6	0.9
	3	Lower	8.1	8.7	8.6	8.6	8.9
		Upper	0.9	0.9	0.8	0.8	0.9
	4	Lower	3.3	3.9	3.5	3.2	3.0
		Upper	1.3	1.7	1.3	1.2	1.4
	5	Lower	4.5	4.8	6.5	4.4	5.7
		Upper	1.2	1.2	1.1	1.0	1.3
	6	Lower	2.8	2.6	2.9	2.9	2.8
		Upper	1.3	1.2	1.3	1.2	1.2

[a] The value for the entire canopy is the average of the estimates for the lower and upper halves of the canopy. All values are averages of individual measures from each sampling date within a growth period.
[b] Stems were small throughout growth cycle 6 and were thus given only one rating, for the entire canopy.

TABLE 3. Mean severity of alfalfa leaf spot on lower and upper halves of the canopy of three alfalfa cultivars at location Wake 2 during each growth cycle in 1984 and 1985

Year	Growth cycle	Portion of canopy[a]	Arc	Cimarron	WL 318
1984	1	Lower	15.7	16.4	17.2
		Upper	1.3	1.4	2.0
	2	Lower	12.1	11.4	13.0
		Upper	2.5	2.4	2.3
	3	Lower	7.6	5.0	5.6
		Upper	1.5	1.2	1.3
	4	Lower	3.3	3.5	3.3
		Upper	2.0	1.9	1.9
1985	1	Lower	4.0	3.9	3.1
		Upper	2.4	2.3	1.9
	2	Lower	6.9	7.5	6.4
		Upper	0.9	1.1	1.0
	3	Lower	5.1	3.6	4.2
		Upper	1.1	0.8	1.0
	4	Lower	1.8	2.1	1.6
		Upper	1.2	1.1	1.0

[a] The value for the entire canopy is the average of the estimates for the lower and upper halves of the canopy. All values are averages of individual measures from each sampling date within a growth period.

level reached a threshold value. The simulator used a function of age and Y_{leaf} for comparison with a threshold value. On each day of the epidemic, each leaf was scanned, and the function of leaf age and Y_{leaf} was compared to the threshold. If the value exceeded the threshold, the leaf was removed from the plant and added to the pool of abscised leaves. The contribution of each abscised leaf to disease development through inoculum production was assumed to increase immediately after detachment from the plant and then to decay exponentially on each successive day.

RESULTS

L. briosiana, S. botryosum, and *C. medicaginis* were the most commonly isolated pathogens during 1984 and 1985 (Table 1). *L. briosiana* was present throughout the entire growing season, and *S. botryosum* was present during many months of the season. *C. medicaginis* was present during the latter half of the growing season, and *Phoma medicaginis* was observed during several months in the early portion of each season. *Pseudopeziza medicaginis* was observed in only one sample.

Overall disease severity at Wake 1 ranged from 0.4% for Cimarron, Pioneer 531, and Raidor during the sixth growth cycle of 1984 to 9.5% for Arc in the fourth growth cycle of 1984 (Table 2). Overall disease severity at Wake 2 ranged from 1.3% for WL 318 during the fourth growth cycle of 1985 to 9.6% for WL 318 during the first growth cycle of 1984 (Table 3). Ranges at Wake 1 were 0.4 to 9.5% in 1984 and 1.7 to 8.8% in 1985 (Table 2); ranges at Wake 2 were 2.6 to 9.6% in 1984 and 1.3 to 4.3% in 1985 (Table 3). Disease was consistently more severe in the lower half of the canopy than in the upper half, and this effect was more pronounced when disease severity was high.

Many of the correlations between measured or calculated variables were significant, but most were relatively low (Table 4).

The highest correlations were between percent debris coverage and the percentage of total stem length defoliated ($r = 0.77, p = 0.0001$), between percent debris coverage and height ($r = 0.65, p = 0.0001$), and between height and the percentage of total stem length defoliated ($r = 0.49, p = 0.0001$). The only negative correlation was between lower disease severity and the percentage of total stem length defoliated ($r = -0.22, p = 0.0001$). The correlation between upper disease severity and the percentage of total stem length defoliated was positive ($r = 0.30, p = 0.0001$).

The variables were grouped into three clusters by the cluster analysis. The first cluster contained canopy height, the percentage of total stem length defoliated, and percent debris coverage. Lower and upper disease severities were in separate clusters, making up the second and third clusters.

The cultivars generally did not differ with respect to disease severity, the percentage of total stem length defoliated, and debris coverage; however, differences in canopy height were generally significant (Tables 5 and 6). Cimarron had the greatest canopy height over all growth cycles in 1985 at Wake 1 (Table 7) and in 1984 and 1985 at Wake 2 (Table 8).

Cultivars at Wake 1 in 1985 differed in AUDPC ($p = 0.04$), and AUDPC was significant in five of the 20 individual growth cycles (Tables 5 and 6). During the two growth cycles in which AUDPC was significant at Wake 1, Raidor had the lowest amount of disease (Table 9). The cultivar Arc was in the highest grouping in four of the five growth cycles in which AUDPC was significant, and Pioneer 531, which was present only at the Wake 1 location, had the greatest AUDPC for the two growth cycles in which AUDPC was significant at that location.

The percentage of total stem length defoliated was almost never significantly different among cultivars; however, the amount of debris usually differed significantly among cultivars ($p = 0.10$). Plants of the cultivar Arc consistently had the lowest amount of leaf debris on the soil surface (Tables 7 and 8), and Raidor was second lowest for both years at Wake 1 (Table 7). Plants of Arc were shorter than most plants of the other cultivars, and plants of Raidor generally had lower disease severity, which may account for the low amount of debris associated with these cultivars.

TABLE 4. Correlations between canopy height, lower and upper disease severity and percent debris coverage, for alfalfa cultivars at locations Wake 1 and Wake 2 during 1984 and 1985[a]

	Lower disease severity	Upper disease severity	% Stem length defoliated	Percent debris coverage
Canopy height	−0.07	0.14**[b]	0.49**	0.65**
Lower disease severity		0.08	−0.22**	0.01
Upper disease severity			0.30**	0.24**
% Stem length defoliated				0.77**

[a] The correlations are based on the area under the progress curve for the indicated variable; the curves were constructed on the basis of 420 observations during 20 growth cycles (six growth cycles per year at Wake 1 and four growth cycles per year at Wake 2).
[b] ** = Correlation coefficient significant at $p = 0.01$.

TABLE 5. Significance level of *F*-test for cultivar effect for alfalfa leaf spot data at location Wake 1[a]

Year	Growth cycle	Canopy height	Disease severity: Entire canopy	Disease severity: Lower canopy	Disease severity: Upper canopy	Percent defoliation	Debris coverage
1984	1	0.28	0.22	0.19	0.25	—[b]	0.75
	2	0.24	0.17	0.46	0.12	0.39	0.48
	3	0.75	0.11	0.09	0.15	0.46	0.43
	4	0.26	0.25	0.56	0.22	0.74	0.15
	5	0.0003	0.01	0.17	0.01	0.79	0.006
	6	0.03	0.35	—	—	—	0.77
	Mean	0.17	0.35	0.23	0.33	0.82	0.09
1985	1	0.002	0.46	0.45	0.64	0.15	0.78
	2	0.20	0.45	0.14	0.38	0.05	0.54
	3	0.11	0.64	0.72	0.56	—	0.15
	4	0.06	0.43	—	0.55	0.91	0.009
	5	0.03	0.009	0.17	0.003	0.37	0.01
	6	0.15	0.22	0.24	0.29	0.50	0.13
	Mean	0.05	0.19	0.09	0.15	0.63	0.02

[a] All tests were based on the area under the progress curve for the indicated variable. Significance levels are given for each growth cycle within a year and for the entire season.
[b] Dashes indicate that no measurement was available during the specified growth cycle.

TABLE 6. Significance levels of *F*-test for cultivar effect for alfalfa leaf spot data at location Wake 2[a]

Year	Growth cycle	Canopy height	Disease severity: Entire canopy	Disease severity: Lower canopy	Disease severity: Upper canopy	Percent defoliation	Debris coverage
1984	1	0.0001	0.61	0.03	0.74	—[b]	0.12
	2	0.02	0.25	0.49	0.24	0.09	0.10
	3	0.006	0.03	0.03	0.04	0.22	0.88
	4	0.005	0.88	0.85	0.87	0.22	0.08
	Mean	0.008	0.60	0.20	0.69	0.81	0.19
1985	1	0.03	0.45	0.05	0.13	0.19	0.07
	2	0.28	0.23	0.16	0.21	0.46	0.59
	3	0.04	0.006	0.007	0.01	0.43	0.005
	4	0.03	0.29	0.80	0.15	0.54	0.06
	Mean	0.04	0.004	0.68	0.04	0.44	0.04

[a] All tests were based on the area under the progress curve for the indicated variable. Significance levels are given for each growth cycle within a year and for the entire season.
[b] No measurement was available during this growth cycle.

TABLE 7. Rankings of alfalfa cultivars by measured variables having a significant *F*-test ($p = 0.10$) at location Wake 1 during 1984 and 1985[a]

Variable	Year	Highest[b] → Lowest				
Canopy height	1985	Cimarron	WL 318	Raidor	Arc	Pioneer 531
		A	A	A	A	
			B	B	B	B
Lower canopy severity	1985	Cimarron	Arc	WL 318	Pioneer 531	Raidor
		A	A	A	A	
				B	B	B
Debris coverage	1984	WL 318	Pioneer 531	Cimarron	Raidor	Arc
		A	A	A	A	B
			B	B	B	
	1985	WL 318	Cimarron	Pioneer 531	Raidor	Arc
		A	A	A	C	C
			B	B	B	

[a] All tests were based on the area under the disease progress curve for the indicated variable over an entire growth season.
[b] Mean separations (indicated by letters) are based on the Waller-Duncan *k*-ratio *t*-test (k = 100) for the total area when the analysis of variance *F*-test was significant at the 0.10 probability level.

The general shapes of the curves for plant growth, disease progress, defoliation, and debris coverage were similar among the cultivars at a given location during the same growth cycle (Fig. 3). The shapes of the curves were also similar when the two years at a single location were compared. Representative disease progress curves are presented for the cultivar Raidor at Wake 1 in 1984 and 1985 (Fig. 4) and for Arc at Wake 1 and Wake 2 in 1984 and 1985 (Figs. 5 and 6). The arrows on graph A of Figures 4–6 indicate the approximate time at which the plants entered the reproductive growth phase (5).

The increase in plant height was fairly constant and almost linear for the first four or five growth cycles of each year (Figs. 4–6); in the last growth cycle, the rate of increase in plant height was generally greatest during the early part of an epidemic and then decreased toward the end. The total stem length defoliated usually paralleled increases in plant height (Figs. 4–6).

The level of disease was low during the first growth period of each year (Figs. 4–6). Debris coverage was between 5 and 10% for most of the first epidemic; however, it increased and then decreased near the end of the epidemic. During epidemics 2 through 5 at Wake 1 and epidemics 2 and 3 at Wake 2, disease severity usually increased rapidly during the first few weeks and often decreased before harvest. Debris coverage followed a more erratic pattern, often starting high, decreasing toward the middle of the epidemic, and increasing at the end. Debris coverage sometimes followed a pattern opposite to that of disease severity.

The leaf spot simulator was utilized to predict the effect of constant and changing r values on the pattern of disease progress. All other parameters were held constant. The effect of low versus high r values is illustrated in Figure 7. Disease severity exhibited a general increase over the period of the epidemic when r was low (Fig. 7A and B) but decreased at the end of epidemics when r was high (Fig. 7C and D). This decrease was due to the defoliation of leaves with higher disease levels. The simulation in which r steadily increased (Fig. 7E) resulted in lower final disease severity and less defoliation than the simulation with steadily decreasing disease rate (Fig. 7F). The simulated disease progress curves given in Figure 7G and H resulted from alternating periods of 5 days at a high rate and 5 days at a low rate, starting with the low rate in Figure 7G and starting with the high rate in Figure 7H. The alternating regimes gave similar results, with both having lower final disease severity and defoliation than the regimes with steadily increasing or decreasing disease rates (Fig. 7E and F).

DISCUSSION

The major purpose of the present research was to quantify and examine the progress of alfalfa leaf spot, a disease that has only

TABLE 8. Rankings of alfalfa cultivars by measured variables having a significant F-test ($p = 0.10$), at location Wake 2 during 1984 and 1985[a]

Variable	Year	Highest[b] →		Lowest
Canopy height	1984	Cimarron	WL 318	Arc
		A	B	B
	1985	Cimarron	WL 318	Arc
		A	A	
			B	B
Entire canopy severity	1985	Arc	Cimarron	WL 318
		A	A	
			B	B
Upper canopy severity	1985	Arc	Cimarron	WL 318
		A	A	
			B	B
Debris coverage	1985	Cimarron	WL 318	Arc
		A	A	
			B	B

[a] All test were based on the area under the progress curve for the indicated variable over an entire growth season.

[b] Mean separations (indicated by letters) are based on the Wallter-Duncan k-ratio t-test (k = 100) for the total area when the analysis of variance F-test was significant at the 0.10 probability level.

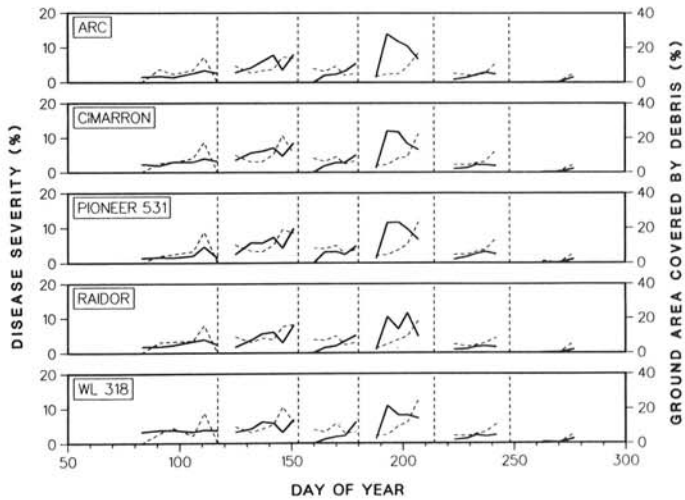

Fig. 3. Disease severity (solid line; vertical scale on left side) and leaf debris coverage on the ground next to the row sections rated (dashed line; vertical scale on right side), plotted against days of the year for five alfalfa cultivars at location Wake 1 during the 1984 season. The dashed vertical lines represent harvest dates.

TABLE 9. Ratings of alfalfa cultivars for growth cycles in which the area under the disease progress curve was significantly different among cultivars at locations Wake 1 and Wake 2 in 1984 and 1985

				Area under disease progress curve				
Location	Year	Growth cycle	Prob[a]	Highest[b] →				Lowest
Wake 1	1984	5	0.01	Pioneer 531	Arc	WL 318	Cimarron	Raidor
				A	A	C	C	C
					B	B	B	
	1985	5	0.009	Pioneer 531	WL 318	Cimarron	Arc	Raidor
				A	A		C	C
					B	B	B	
Wake 2	1984	3	0.03	Arc		WL 318		Cimarron
				A		A		
						B		B
	1985	1	0.05	Arc		Cimarron		WL 318
				A		A/B		A/B
		3	0.006	Arc		WL 318		Cimarron
				A		B		B

[a] Probability of a greater F-value from analysis of variance.

[b] Mean separations (indicated by letters) are based on the Waller-Duncan k-ratio t-test (k = 100) for the total area when the analysis of variance F-test was significant at the 0.10 probability level.

been described qualitatively. Host growth, disease development, and defoliation are all important factors in this pathosystem, and any interpretations concerning the alfalfa leaf spot system must take each of these factors into account. This study is among only a few studies to examine the role of host growth in disease progression (11). Height was used as the sole quantifier for plant growth. This is admittedly a relatively crude measure of host growth; however, we believe it is indicative of the relative growth rate of alfalfa. Since the growth of alfalfa appears to play a significant role in leaf spot epidemics, more sophisticated measures of host growth, such as leaf area, may need to be examined in future studies.

Cultivars of alfalfa were selected to give as great a range of disease resistance as possible (14). Differences observed among various cultivars were not large but are representative of the current status of resistance to leaf spot diseases in alfalfa. Thus, the

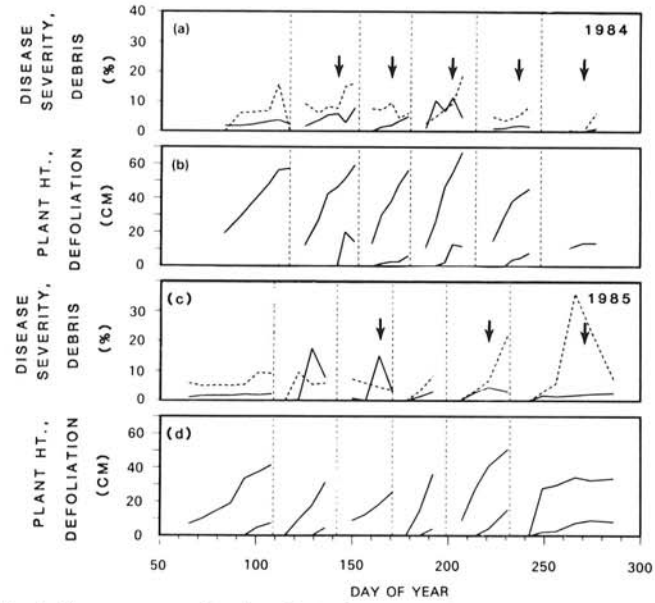

Fig. 4. Disease progression data for leaf spot on alfalfa cultivar Raidor at location Wake 1 in 1984 and 1985. **A and C,** Disease severity (solid line) and percent leaf debris coverage on the ground next to the row sections rated (dashed line). The vertical arrows indicate the first rating date for which the plant growth stage was 2.3 or greater. **B and D,** Plant height (upper line) and length of defoliation up the stem (lower line); where only one line is present, defoliation was near zero for the entire growth cycle. The dashed vertical lines represent harvest dates.

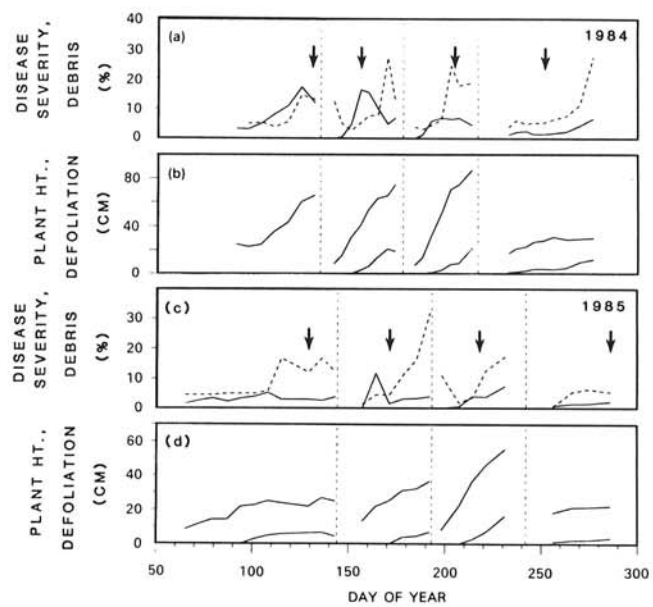

Fig. 6. Disease progression data for leaf spot on alfalfa cultivar Arc at location Wake 2 in 1984 and 1985. **A and C,** Disease severity (solid line) and percent leaf debris coverage on the ground next to the row sections rated (dashed line). The vertical arrows indicate the first rating date for which the plant growth stage was 2.3 or greater. **B and D,** Plant height (upper line) and length of defoliation up the stem (lower line); where only one line is present, defoliation was near zero for the entire growth cycle. The dashed vertical lines represent harvest dates.

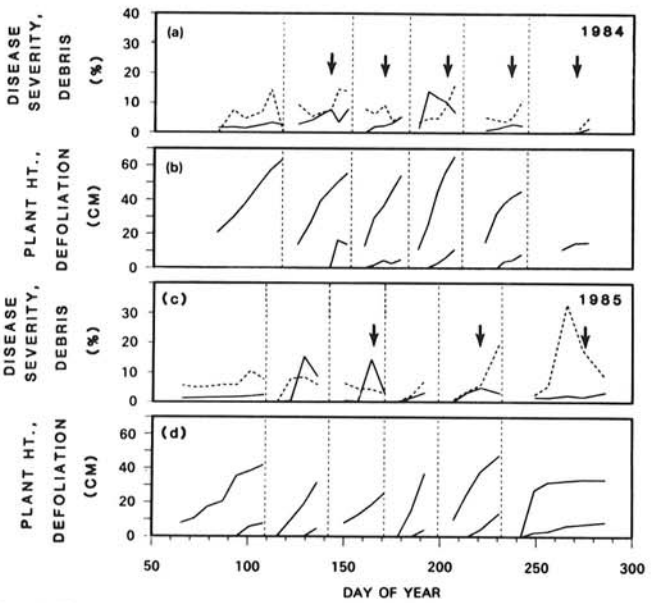

Fig. 5. Disease progression data for leaf spot on alfalfa cultivar Arc at location Wake 1 in 1984 and 1985. **A and C,** Disease severity (solid line) and percent leaf debris coverage on the ground next to the row sections rated (dashed line). The vertical arrows indicate the first rating date for which the plant growth stage was 2.3 or greater. **B and D,** Plant height (upper line) and length of defoliation up the stem (lower line); where only one line is present, defoliation was near zero for the entire growth cycle. The dashed vertical lines represent harvest dates.

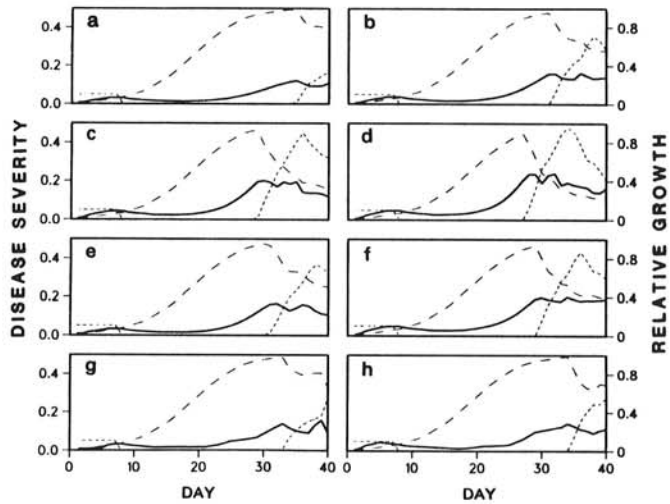

Fig. 7. Simulated disease progress curves for alfalfa leaf spot: disease severity (solid line; vertical scale on left side); leaves on plant, expressed as a proportion of the asymptote (dashed line with long dashes; vertical scale on right side); and contribution of debris to disease increase (dashed line with short dashes; vertical scale on left side gives proportion of maximum possible contribution). **A–D,** Constant rates of disease increase, from low to high (r, the constant representing the rate of disease increase in the simulation, was 0.5, 0.6, 0.7, and 0.8, respectively). **E and F,** Increasing and decreasing disease rates, respectively, over the length of the epidemic (r values ranged from 0.5 to 0.8). **G and H,** Alternating 5-day periods of high disease rate ($r = 0.9$) and low disease rate ($r = 0.2$), beginning with the low rate (G) and beginning with the high rate (H).

lack of difference emphasizes the need for continued efforts in breeding alfalfa to increase levels of leaf spot resistance.

A common phenomenon in the disease progress curves was a decrease, or at least a leveling off, of curves just prior to harvest. This may be partially due to the abscission of leaves with high levels of leaf spot. The alfalfa leaf spot disease simulator included variables and models to account for plant growth, disease increase (based on disease severity, debris buildup, and decreased susceptibility of older leaves), and defoliation due to age and leaf spot. Disease progress curves produced by the simulator showed a leveling off or slight decrease in disease at the end of an epidemic, but the decreases were generally not as large as those observed in the field data. Even in cases where the r value was reduced toward the end of an epidemic, the simulator indicated only a slight decrease in overall disease severity. Only in cases of severe defoliation did the simulator indicate a sharp decrease in disease at the end of an epidemic. Such severe levels of defoliation were never seen in the field. It appears likely, therefore, that defoliation plays a part in this decrease, but other factors, such as changes in crop physiology and microclimate, may be involved.

To account for the observed decreases in disease severity, it may be necessary to investigate changes in the physiology of alfalfa and in the crop microenvironment. The physiology of the alfalfa plant changes from the beginning to the end of a growth cycle, as the plants change from the vegetative to the reproductive phase. This change may correspond to a decrease in susceptibility to one or more of the pathogens in this disease complex. The simulator accounted for a decrease in the susceptibility of leaves with age but not for decreased susceptibility of new leaves produced on an older plant. The arrows on graph A of Figures 4–6 indicate the point at which plants reached growth stage 2.3, the late prebud stage (5). This often corresponded to the time when disease began to decrease.

The level of disease severity detected is influenced by the time within the alfalfa growth cycle when an observation is made. The disease severity level can change significantly shortly before harvest. This could result in changes in rankings if the timing of this decrease differed among cultivars.

Defoliation was evaluated in two ways: the height to the first intact node and the amount of debris on the ground. The first factor is, by nature, partly a function of the height of the plant, as can be seen by the graphs of height and defoliation length versus time. Estimates of the amount of debris may give a better idea of leaf loss. There were high levels of debris early in a growing period, and they may have contributed to the rapid increase in disease at the beginning of some growth cycles. Debris deteriorated rapidly, as can be seen from fluctuations in the amount of debris observed during the growing season. A confounding factor is that debris may at times consist of a single layer of leaves and may at other times be several layers thick. Thus, an additional quantifier of debris, besides the percentage of the soil surface covered, may be needed.

The present study concentrated on the overall characteristics of the disease progress of leaf spots on alfalfa. The environment was generally ignored, except for large-scale trends that occur over the growth cycles in a season and were seen from one year to the next. Studies that look more closely at the important environmental parameters (rainfall and temperature) would be useful if predictive models are to be developed. The effect of environment, not only on disease but also on debris and defoliation, should be investigated.

LITERATURE CITED

1. Fry, W. E. 1978. Quantification of general resistance of potato cultivars and fungicide effects for integrated control of potato late blight. Phytopathology 68:1650-1655.
2. Graham, J. H., Frosheiser, F. I., Stuteville, D. L., and Erwin, D. C. 1979. A Compendium of Alfalfa Diseases. American Phytopathological Society, St. Paul, MN. 65 pp.
3. Hanson, C. H. 1972. Alfalfa Science and Technology. American Society of Agronomy, Madison, WI. 812 pp.
4. Horsfall, J. G., and Barratt, R. W. 1945. An improved grading system for measuring plant diseases. (Abstr.) Phytopathology 35:655.
5. James, W. C. 1971. A Manual of Assessment Keys for Plant Diseases. Can. Dep. Agric. Publ. 1458.
6. Leath, K. T. 1981. Pest management systems for alfalfa diseases. Pages 293-301. In: Handbook of Pest Management in Agriculture, Vol. III. D. Pimental, ed. CRC Press, Boca Raton, FL.
7. Leath, K. T., and Hill, R. R., Jr. 1974. *Leptosphaerulina briosiana* on alfalfa: Relation of lesion size to leaf age and light intensity. Phytopathology 64:243-245.
8. Luttrell, E. S. 1983. Fungi. Pages 258-266 in: Challenging Problems in Plant Health. T. Kommedahl and P. H. Williams, eds. American Phytopathological Society, St. Paul, MN.
9. Madden, L. V. 1980. Quantification of disease progression. Prot. Ecol. 2:159-176.
10. McDonald, W. C. 1958. The *Pseudoplea-Stemphylium* complex on alfalfa. Phytopathology 48:365-369.
11. Rouse, D. I. 1983. Plant growth models and plant disease epidemiology. Pages 387-398 in: Challenging Problems in Plant Health. T. Kommedahl and P. H. Williams, eds. American Phytopathological Society, St. Paul, MN.
12. SAS Institute Inc. 1985. SAS User's Guide: Statistics, Version 5 Edition. SAS Institute Inc., Cary, NC. 956 pp.
13. Steel, R. G. D., and Torrie, J. H. 1980. Principles and Procedures of Statistics, 2nd ed. McGraw-Hill, New York. 633 pp.
14. Thal, W. M., and Campbell, C. L. 1987. Assessment of resistance to leaf spot diseases among alfalfa cultivars in North Carolina fields. Phytopathology 77:964-968.
15. van Nuffelen, M., and Berger, R. D. 1983. Rust of alfalfa as a model pathosystem to examine sequential epidemics. (Abstr.) Phytopathology 73:804.
16. Von Chong, K. 1987. Epidemiology of alfalfa leafspot diseases: Pathogen occurrence and the relationship among environmental factors, inoculum density, and disease progress. Ph.D. thesis, North Carolina State University, Raleigh. 93 pp.