# Development of Foliar Diseases of Alfalfa in Relation to Microclimate, Host Growth, and Fertility

K. M. Emery and J. T. English

Graduate research assistant and assistant professor, respectively, Department of Plant Pathology, University of Missouri, Columbia 65211.
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## ABSTRACT

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Foliar diseases of alfalfa in four fertility treatments and an untreated control were evaluated in relation to microclimate, host growth, and soil fertility during 1991 and 1992. Over the course of six growth periods, alfalfa growth and disease development varied significantly. Disease incidence and severity were greater on leaves from the lower half of shoots than on leaves from the upper half. Disease development depended on moisture conditions. The relationship of disease to moisture varied between the upper and lower halves of shoots. Disease on the lower half of shoots was correlated with leaf wetness duration as well as atmospheric moisture conditions; disease on the upper half of shoots was correlated only with cumulative rainfall. Although disease incidence and severity were not correlated significantly with alfalfa growth or soil fertility, growth did influence disease assessments by altering the composition of the pool of leaves sampled. As shoot growth proceeded, newly produced, uninfected leaves reduced the proportion of diseased leaves in the sampling pool, thereby reducing disease incidence and severity.

Foliar epidemics of perennial forage legumes such as alfalfa (*Medicago sativa* L.) have received increased attention recently as epidemiologists have begun to examine the processes leading to their development (6,22,23). Disease development in alfalfa canopies involves complex interactions of numerous fungal and bacterial pathogens and microclimatic conditions. The diverse environmental requirements of these pathogens (8) allows epidemics to occur throughout the growing season.

Recent studies have focused on the influence of infested debris on infection by specific pathogens (6) or on characterizing the patterns of disease development over time (23). Relatively little attention has been given to the relationships of host growth and microclimate to disease development. It has been suggested that canopy development may increase the number of potential infection periods by reducing penetration of sunlight and air movement to lower foliage, thereby increasing periods of high humidity and leaf wetness (8). In addition to influencing microclimate, host growth may influence disease evaluations by altering the composition of the sampling pool of plant tissues over time (16). This possibility has not been investigated with alfalfa.

Canopy structure and its influence on microclimate are difficult to assess in crops such as alfalfa. The structure of the canopy is determined by several growth processes, including increases in the size and number of structural components. The purpose of this research was to evaluate development of foliar epidemics of alfalfa in relation to microclimate and increases in numbers of plant structural components, which might reflect the complexity of the canopy structure. Alfalfa growth also was evaluated in relation to soil fertility, which may affect canopy structure and, thus, indirectly influence microclimate and disease development.

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## MATERIALS AND METHODS

**Establishment of experimental plots.** This research was undertaken as part of a larger study of the influence of soil fertility on insect pests of alfalfa. Growth of alfalfa and development of foliar diseases were evaluated in relation to five fertility treatments. During the spring of 1989, alfalfa seed (Pioneer 5432, Pioneer Seed Company, Johnston, IA) were sown at a rate of 25 kg/ha in a Mexico silt loam soil at the South Farm Agricultural Research Facility of the University of Missouri at Columbia. Fertility treatments, including $K_2SO_4$, KCl, $K_2SO_4$ + $MgSO_4$, and KCl + $MgSO_4$, were designed to achieve specific combinations of potassium, sulfur, and magnesium in the soil at concentrations of 340, 50, and 17 kg/ha, respectively. An untreated control treatment also was evaluated. Of these treatments, KCl is the chemical form of potassium fertilizer most commonly used by alfalfa producers in Missouri. Beginning in 1990, one-third of each treatment was applied during the fall of each year of the experiment, and the remainder was applied during the following spring. The amount of each fertilizer application was based on prior soil-nutrient analyses. Each treatment was applied to four replicate plots (7.3 × 9.75 m) established in a randomized complete-block design.

Plots were managed according to commercial practices. Alfalfa in all plots was cut at approximately the 10%-bloom stage (18). Hay was air-dried in the field for 3–5 days after being cut, baled, and removed. Four harvests were made in 1991 and in 1992 each. After the first harvest during each year, all plots were sprayed with paraquat (Gramoxone Super, ICI Americas Inc., Wilmington, DE) according to the manufacturer's instructions for weed control.

**Assessment of plant growth and disease.** Foliar diseases and plant growth were assessed in all fertility treatments through three of four growth periods during both years of the study. In 1991, growth periods I, II, and III corresponded to the second, third, and fourth harvests, respectively. In 1992, growth periods I, II, and III corresponded to the first, second, and fourth harvests, respectively. Evaluations of growth and disease were made at weekly intervals beginning 7–14 days after the previous harvest. Four evaluations were made during each growth period. On each sampling date in 1991 and 1992, nine and six shoots, respectively, were chosen arbitrarily from each replicate plot and removed to the laboratory for evaluation of growth and disease. Sample shoots were collected from the center of each plot to minimize interplot interference.

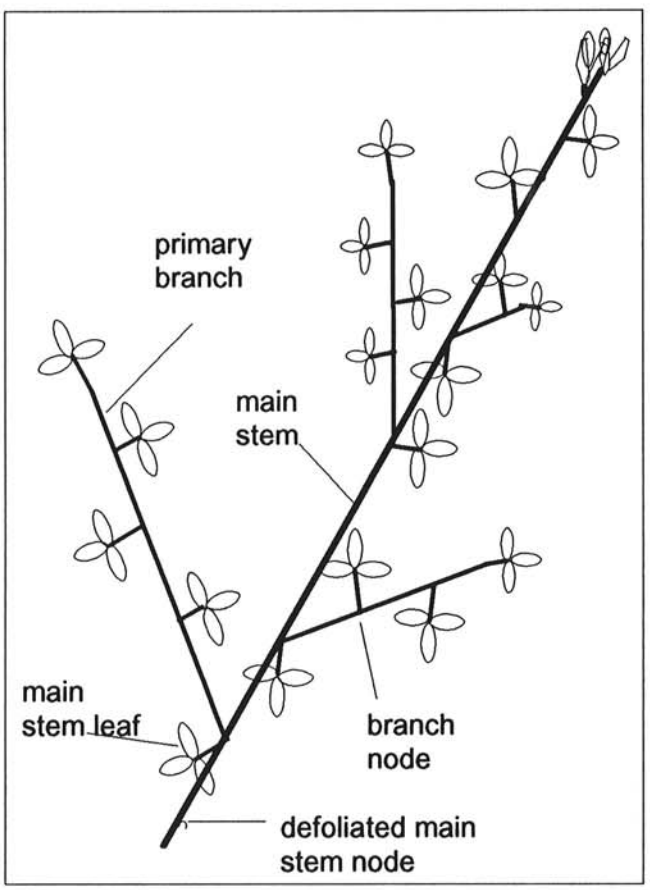


**Fig. 1.** Typical alfalfa shoot after several weeks of growth. With further growth, secondary and tertiary branches are produced from primary branches.

Each shoot was evaluated for length and number of various structural components (Fig. 1), including nodes produced on the main stem and leaves at these nodes (main stem nodes and leaves), as well as nodes produced on branches (branch nodes). A defoliated node was one whose leaf had abscised. Leaf mortality was estimated as the number of defoliated main stem nodes per shoot. Over time the primary branches generally produced secondary and tertiary branches. For the purposes of this research, all nodes produced on primary, secondary, or tertiary branches were considered branch nodes.

Two leaves were selected arbitrarily for disease assessment from the upper and lower half of each sampled shoot. Disease was evaluated as a complex without regard to the causal organism because it was difficult to distinguish between pathogens on the basis of symptoms (1,2,23). Disease incidence was estimated as the proportion of leaves with symptoms, and disease severity was estimated as the proportion of symptomatic leaf area. Severity of disease was estimated by visually comparing sampled leaves to a standardized key (23).

Areas under disease progress curves (AUDPC) were computed relative to the incidence and severity of disease such that:

$$\text{AUDPC} = \sum_{i=1}^{n} [(y_i + y_{i-1})/2](t_i - t_{i-1}),$$

where $i$ = assessment period from 1 to $n$; $y_i$ = disease severity or incidence at period $i$; and $t$ = time in days (3). Estimates of AUDPC were made separately for the upper and lower halves of sampled shoots.

Several times during each growth period symptomatic tissue was collected throughout the field to determine which pathogens were present. Plant material was taken to the laboratory, prepared, and examined according to the methods described by Duthie and Campbell (6).

**Collection of microclimate data.** Microclimate data were recorded hourly within and above the plant canopy with a micrologger (model 21X, Campbell Scientific, Inc., Logan, UT). A shielded sensor for recording above-canopy (ambient) temperature and relative humidity (model 207, Campbell Scientific) was placed 1.5 m above the ground. An identical sensor was placed at ground level in one replicate plot of the KCl treatment, and another was placed in an adjoining control plot. A leaf wetness sensor (model 237, Campbell Scientific) was placed adjacent to each temperature sensor. A tipping bucket rain gauge (model TE525, Campbell Scientific) was used to record rainfall. Vapor pressure and vapor pressure deficit were calculated from average hourly temperature and relative humidity data (20).

**Data analysis.** The relationships of AUDPC and disease at harvest to microclimate during several time periods within growth periods (Fig. 2) were evaluated by Pearson correlation analyses (17). Microclimate variables for inclusion in correlations were summarized for the 10-day period prior to the second sampling date, the 10- and 16-day periods prior to the third sampling date, and the 10-, 16-, and 21-day periods prior to the fourth and final sampling date of each growth period. Because no significant treatment effects were detected, disease data from fertility treatments were averaged for each growth period to provide six observations for each analysis. Analyses of the relationships of AUDPC and disease at harvest to growth were performed using only growth data at harvest. Microclimate data were not available for the 2-wk interval leading up to the first sampling date of

each growth period in 1991, and therefore, disease data from the first sampling date were not included in calculations of AUDPC.

Selection of microclimate variables for inclusion in correlation analyses was based on knowledge of the environmental requirements of pathogens isolated from alfalfa in 1991 and 1992. Microclimate variables included cumulative hours of leaf wetness, cumulative hours with average relative humidity greater than 90%, cumulative vapor pressure and vapor pressure deficit, and cumulative rainfall.

Effects of fertility on growth and disease were evaluated by analysis of variance (ANOVA) (17). In these analyses, the assumptions of the ANOVA were evaluated by plotting residuals against predicted values and by Bartlett's test for the homogeneity of variance (11). No violations of the assumptions of the ANOVA were detected.

Patterns of alfalfa growth and disease development over time were evaluated by linear orthogonal contrast (4) with the NEWLQC program (University of Missouri Board of Curators, Columbia) to detect significant linear and nonlinear coefficients. These analyses allowed comparisons of fertility treatment effects over time.

## RESULTS

Disease symptoms were observed in all treatment plots throughout all growth periods in 1991 and 1992. Several pathogens were observed, including *Leptosphaerulina trifolii* (Rostr.) Petr., which was isolated frequently during both years, and *Stemphylium botryosum* Wallr., which was isolated throughout 1992. During the final growth period of 1992, infection by the rust fungus *Uromyces striatus* (J. Schröt.) was observed.

There were no significant effects of fertility treatments on plant growth or disease. There also were no significant correlations between alfalfa growth and weather conditions. Variation in growth over time was observed relative to shoot length and number of structural components. Plant growth and disease data from the KCl and control treatments during 1992 are presented to illustrate the range of values and variability observed in patterns of change over growth periods.

Patterns of alfalfa growth were similar among growth periods and fertility treatments, as demonstrated by linear contrasts indicating that all treatments had similar effects. For example, shoot length increased linearly with time in all growth periods of 1991 and 1992 (Table 1, Fig. 3A–C). However, differences in rates of growth resulted in considerable variation among growth periods in shoot length at harvest. The number of main stem nodes per shoot increased linearly with time in all growth periods; however, significant linear increases in the net number of main stem leaves occurred in only three of six growth periods (Table 1). Additionally, a significant quadratic increase in the number of main stem nodes occurred in growth period III of 1991.

Defoliation of main stem nodes frequently resulted in the number of main stem leaves remaining constant (Fig. 3G and I) as the number of main stem nodes increased (Fig. 3D and F). The differences in the number of leaves and nodes reflected the balance of new leaves produced by apical meristems and loss of older leaves after infection and senescence.

The number of nodes produced on branches also increased linearly over time, but by harvest the number of branch nodes was more variable among growth periods than was the number of main stem nodes produced on shoots (Fig. 3J–L). The average number of branch nodes per shoot at harvest in 1992 ranged from 21 in growth period III to 37 in growth period II. Despite this variability, the number of branch nodes increased linearly with time in five of six growth periods (Table 1). The number of main stem and branch nodes produced was not correlated significantly with shoot length.

There was no evidence that patterns of disease development were influenced by alfalfa growth or by the incidence or severity of disease in the preceding growth period. Patterns of disease that increased over time were often similar among growth periods; however, variability in disease levels among shoots was sufficiently large that significant linear or nonlinear trends in disease incidence on leaves from the lower half of shoots were not detected in four of six growth periods (Table 2). In two growth periods, significant linear and nonlinear patterns of increase were detected on leaves from the lower half of shoots. Disease progress during

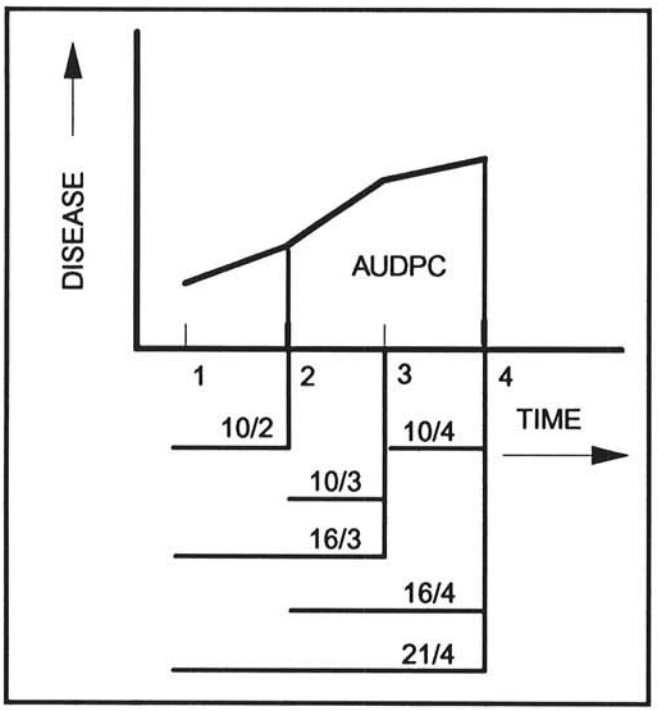


**Fig. 2.** Schematic diagram of disease progress and the time frames within a growth period for which microclimate variables were summarized. For example, 10/4 indicates that a variable was summarized for the 10-day period prior to the fourth sampling date. All growth periods had four sampling dates; harvest occurred on sampling date four.

TABLE 1. Temporal patterns of alfalfa growth during 1991 and 1992 in plots that received different fertility treatments[a]

| Growth period[b] | Linear coefficient[c] | Shoot length (cm) | No. of main stem nodes[d] | No. of main stem leaves | No. of branch nodes |
|---|---|---|---|---|---|
| 1991 | | | | | |
| I | Linear | *[e] | * | * | * |
| | Quadratic | ns | ns | ns | ns |
| II | Linear | * | * | ns | ns |
| | Quadratic | ns | ns | ns | ns |
| III | Linear | * | * | * | * |
| | Quadratic | ns | ns | * | ns |
| 1992 | | | | | |
| I | Linear | * | * | ns | * |
| | Quadratic | ns | ns | ns | ns |
| II | Linear | * | * | * | * |
| | Quadratic | ns | ns | ns | ns |
| III | Linear | * | * | ns | * |
| | Quadratic | ns | ns | ns | ns |

[a] Fertility treatments included KCl, $K_2SO_4$, KCl + $MgSO_4$, $K_2SO_4$ + $MgSO_4$, and untreated control.

[b] Growth periods I, II, and III correspond to days of the year 164–182, 204–225, and 241–262, respectively, in 1991 and 115–136, 169–189, and 238–262, respectively, in 1992.

[c] Coefficients representing alfalfa growth as evaluated by orthogonal contrasts.

[d] Main stem and branch nodes included both those that had leaves and those that were defoliated.

[e] * indicates that the coefficient was significant ($P = 0.05$) for all fertility treatments; ns indicates that coefficient was not significant for any fertility treatment. All coefficients were positive.

these growth periods typically was characterized by initial disease levels of less than 30% on the first sampling date followed by increases to over 80% by the final sampling date.

The range of temporal patterns of change in disease incidence on leaves from the lower half of shoots (Fig. 4A–C) was similar to that observed on leaves from the upper half of shoots (Fig. 4D–F). However, in contrast to leaves on the lower half of shoots, significant linear and nonlinear trends in incidence on the upper half of shoots were detected in five of six growth periods (Table 2). For example, incidence decreased linearly in growth period I of 1992 (Fig. 4D) but increased linearly in growth periods II and III of that year (Fig. 4E and F).

Disease severity generally was low throughout 1991 and 1992, and there were few significant linear or nonlinear trends over time detected on leaves from upper or lower halves of shoots (Table 2). Disease severity was consistently higher on leaves from the lower half of shoots (Fig. 4G–I) than on leaves from the upper half of shoots (Fig. 4J–L). For example, during growth period II in 1992, disease severity increased linearly on the lower portion of shoots to a maximum of 5% at harvest on day 189 (Fig. 4H). During the same period, severity on the upper portion of shoots increased linearly to a maximum of 2% (Fig. 4K).

Microclimate differed above and within the plant canopy and among growth periods. Differences were most evident with respect to moisture conditions (Table 3). Average relative humidity tended to be higher within the canopy than above it, and differences increased as alfalfa shoots grew up around the sensors. For example, differences between average ambient and canopy relative humidities ranged from 3% during growth period I in 1991 to 16% during growth period I in 1992. Similarly, durations of leaf wetness in the canopy were always greater than those above it. Differences in cumulative duration of canopy and ambient leaf wetness ranged from as little as 1 h during growth period II in 1992 to as much as 37 h during growth period III in 1991. In contrast to moisture conditions, mean daily temperatures above and within the canopy generally differed by only 1 C.

Disease development over growth periods varied in relation to microclimatic conditions. As indicated by correlation analyses, disease at harvest in all six growth periods often was related significantly to canopy and ambient moisture conditions within respective periods. However, only relationships between disease development and canopy microclimate are presented.

Disease incidence and severity at harvest on leaves from the upper half of shoots were correlated significantly ($P = 0.10$) only

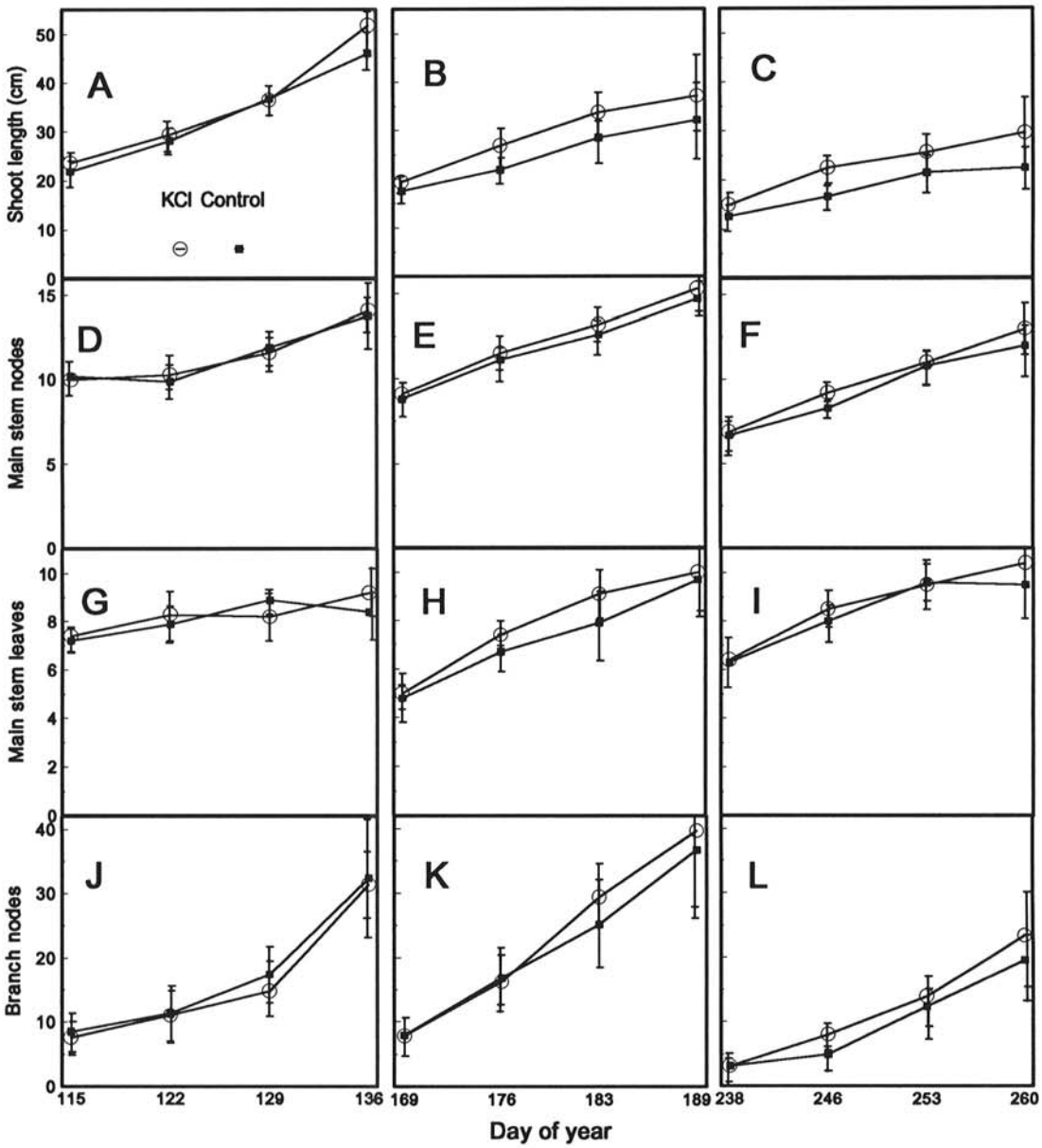


**Fig. 3.** Alfalfa growth during the first (**A, D, G, and J**), second (**B, E, H, and K**), and third (**C, F, I, and L**) growth periods of 1992.

with rainfall during the middle of the growth period and not with any measure of atmospheric moisture over the time periods considered (Table 4). In contrast, disease incidence at harvest on leaves from the lower half of shoots was not correlated with any moisture variable. Disease severity on these leaves was correlated with several measures of atmospheric moisture, including a negative correlation with vapor pressure deficit (Table 4).

There were several significant correlations between AUDPC associated with incidence and severity on leaves from the upper half of shoots and moisture conditions. These correlations tended to be significant over the middle and end of growth periods (Table 4). Unlike disease at harvest, there were no correlations between AUDPC and rainfall; however, there were significant correlations between AUDPC and cumulative duration of leaf wetness. There also were positive correlations between these AUDPC and vapor pressure deficit (Table 4).

In contrast to AUDPC associated with disease on the upper half of shoots, there were fewer significant correlations between AUDPC for disease incidence and severity on leaves from the lower half of shoots and moisture variables. Generally, these correlations were with moisture conditions during the early to middle part of growth periods (Table 4). The AUDPC associated with disease severity on these leaves was correlated only with vapor pressure deficit. A similar negative correlation existed with the AUDPC for incidence on these leaves.

## DISCUSSION

Although environment has long been known to influence the development of foliar diseases of alfalfa (5,7,10,12,21), this is the first study to evaluate foliar epidemics of alfalfa in relation to canopy microclimate. Previous researchers evaluated epidemics in relation to general weather conditions (6), but analyses of specific relationships between canopy microclimate variables and disease were not made.

The relationships of disease to moisture conditions generally reflected the importance of moisture in the infection process. The appearance of disease symptoms lagged behind the occurrence of specific moisture conditions. Pathogen biology may account for this lag in disease development. For example, incubation periods of two or more days have been reported for *S. botryosum* or *L. trifolii* under ideal conditions (13,14) of extended periods of relative humidity greater than 90%. These conditions occurred infrequently in the field during 1991 and 1992, and consequently,

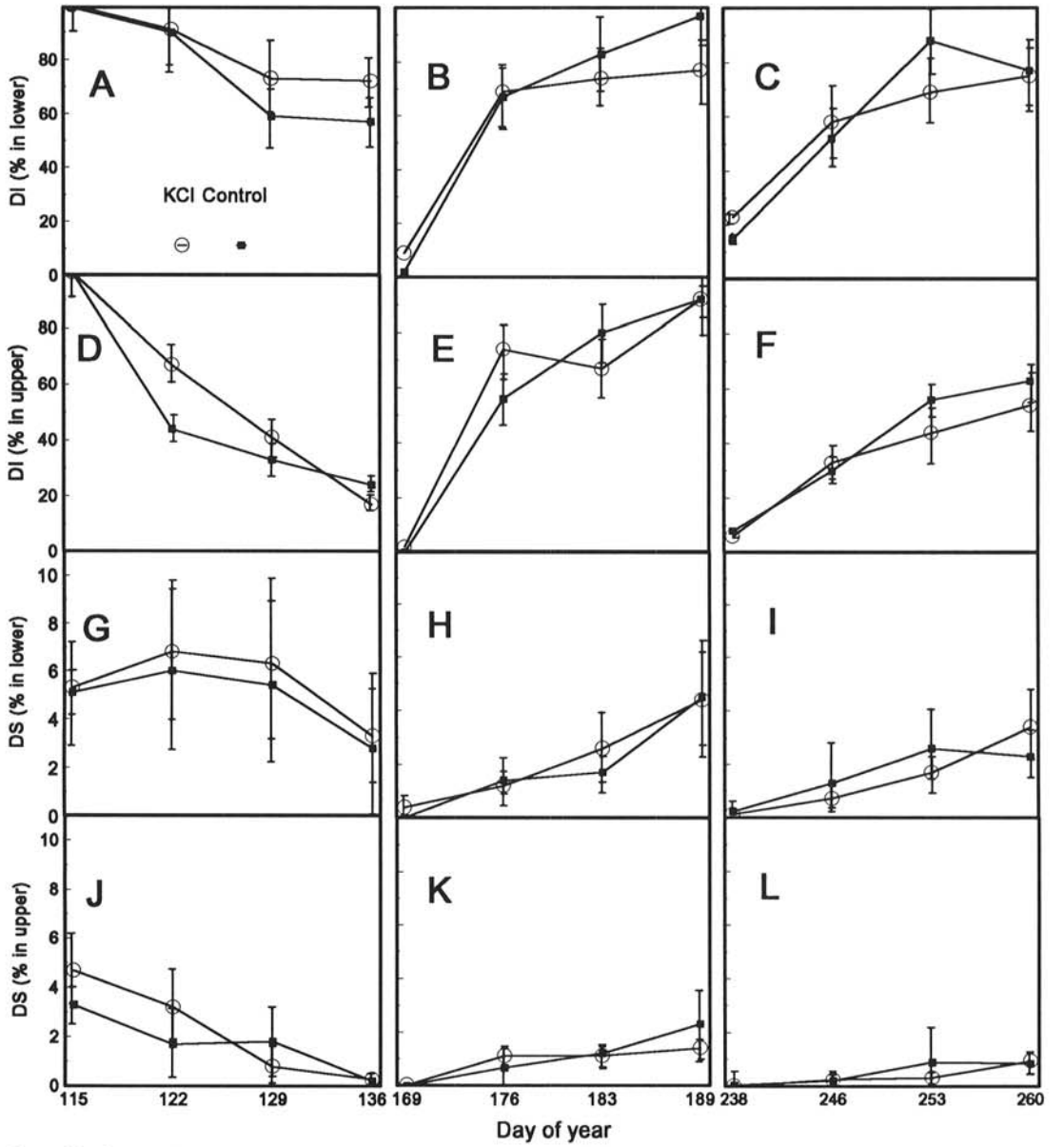


**Fig. 4.** Progression of incidence (DI) and severity (DS) of foliar diseases of alfalfa on leaves from the lower and upper halves of shoots during the first (**A, D, G, and J**), second (**B, E, H, and K**), and third (**C, F, I, and L**) growth periods of 1992.

longer periods of time often may have been required for symptoms to develop.

Under favorable conditions, secondary inoculum may have been produced on tissues infected early in a growth period. The latent periods of *L. trifolii* and *S. botryosum*, for example, range from 14 to 15 days and 5 to 8 days, respectively (14,19). Thus, lesions produced by these pathogens could have been infectious by the second or third week of a growth period.

The importance to disease development of secondary inoculum produced within a growth period compared to that produced on debris from previous growth periods is uncertain. Duthie and Campbell (6) reported that removal of debris from alfalfa plots seldom reduced disease incidence significantly and suggested that inoculum produced in lesions on current growth may be more important in disease development. However, they also speculated that weather conditions may overwhelm any effects of inoculum availability.

This is the only study that has evaluated the development of foliar disease in relation to host growth as measured by increases in the number rather than size of structural components. Previous studies on the relationship of disease to host growth used canopy height as the only measure of alfalfa growth (6,23). There was no evidence of a relationship between the number of structural components and disease development. The evaluation of growth relative to number rather than size of structural components may account for this lack of correlation. A more thorough evaluation of canopy structure incorporating changes in the size of leaves as well as three-dimensional shoot architecture may reflect the relationship between growth and disease more accurately. Increases in leaf size could influence disease levels by altering microclimate and increasing the availability of susceptible leaf tissue.

Although disease could not be related significantly to alfalfa growth in these experiments, growth did influence assessments of disease on leaves from the upper and lower halves of shoots. For example, the incidence of disease on leaves from both halves of shoots frequently increased only gradually during the latter half of growth periods and sometimes even decreased after increasing rapidly during the first half. These patterns of change in incidence can be attributed, in part, to the addition by growth of uninfected leaves that contributed to an overall reduction in the proportion of diseased leaves in the sampling pool of either the upper or lower half of shoots.

An additional explanation for constant or declining disease incidence over time involves the removal of diseased leaves through abscission. Thal and Campbell (22) reported decreases in disease severity near the end of growth periods and suggested such decreases were partly due to increased defoliation over time. Although defoliation of main stem nodes did occur over time in this experiment, it did not appear to have a significant effect on disease assessments. The proportion of nodes that were defoliated remained small, perhaps because disease levels were quite low.

Through two growing seasons, a significant effect of soil-fertility treatment on alfalfa growth or the incidence or severity of disease was not detected. Research with other host-pathogen systems generally has indicated that increasing the amount of potassium available to plants increases their resistance to disease (9,15). Very little information is available, however, regarding specific relationships of potassium or other nutrients to the development of foliar diseases of alfalfa. It is possible that soil nutrient levels in the control plots did not decline sufficiently over the 2-yr period to observe any impact of low soil nutrient status on disease. Additionally, the high degree of variability in data of this type could have obscured treatment effects.

Our research emphasizes the complexity of the host-parasite interactions that take place in plant canopies, including changes in the leaf sampling pool over time and the influence of the canopy on microclimate. Further elucidation of these interactions will be attained with appropriate quantification of specific microclimatic factors within clearly defined regions of the plant canopy and with appropriate quantification of plant growth in terms of both numbers and sizes of plant structural components.

TABLE 2. Temporal changes in incidence and severity of foliar diseases of alfalfa during 1991 and 1992 in plots that received different fertility treatments[a]

| Growth period[b] | Linear coefficient[c] | Disease incidence | | Disease severity | |
|---|---|---|---|---|---|
| | | Lower leaves[d] | Upper leaves | Lower leaves | Upper leaves |
| 1991 | | | | | |
| I | Linear | *[e] | ns | ns | ns |
| | Quadratic | * | * | ns | ns |
| II | Linear | ns | ns | ns | ns |
| | Quadratic | ns | ns | ns | ns |
| III | Linear | ns | * | ns | ns |
| | Quadratic | ns | * | ns | ns |
| 1992 | | | | | |
| I | Linear | ns | *[f] | ns | ns |
| | Quadratic | ns | ns | ns | ns |
| II | Linear | * | * | * | * |
| | Quadratic | * | ns | ns | ns |
| III | Linear | ns | * | * | ns |
| | Quadratic | ns | ns | ns | ns |

[a] Fertility treatments included KCl, $K_2SO_4$, KCl + $MgSO_4$, $K_2SO_4$ + $MgSO_4$, and untreated control.
[b] Growth periods I, II, and III correspond to days of the year 164–182, 204–225, and 241–262, respectively, in 1991 and 115–136, 169–189, and 238–262, respectively, in 1992.
[c] Coefficients representing temporal changes in disease in relation to fertility treatments as evaluated by orthogonal contrasts.
[d] Upper leaves were selected from the upper half of alfalfa shoots. Lower leaves were selected from the lower half of alfalfa shoots.
[e] * indicates that the coefficient was significant ($P = 0.05$) for all treatments; ns indicates that the coefficient was not significant for any treatment.
[f] The coefficient was negative, all others were positive.

TABLE 3. Weather summaries for periods of alfalfa growth during 1991 and 1992

| Growth period[a] | Ambient air temperature[b] (C) | Relative humidity (%)[c] | | Leaf wetness (h)[d] | | Rainfall[d] (mm) |
|---|---|---|---|---|---|---|
| | | Ambient | Canopy | Ambient | Canopy | |
| 1991 | | | | | | |
| I | 25.3 | 80 | 83 | 98 | 131 | 35.8 |
| II | 24.1 | 76 | 80 | 135 | 143 | 21.1 |
| III | 22.8 | 85 | 91 | 185 | 218 | 0 |
| 1992 | | | | | | |
| I | 15.2 | 66 | 82 | 166 | 178 | 21.0 |
| II | 23.2 | 72 | 78 | 160 | 161 | 40.6 |
| III | 21.4 | 69 | 81 | 148 | 157 | 33.5 |

[a] Growth periods I, II, and III correspond to days of the year 164–182, 204–225, and 241–262, respectively, in 1991 and 115–136, 169–189, and 238–260, respectively, in 1992.
[b] Mean daily.
[c] Mean.
[d] Cumulative.

TABLE 4. Correlation of disease with canopy microclimate variables during 1991 and 1992

| Disease variable[a] | Vapor pressure[b] (kPa) | Vapor pressure deficit (kPa)[b] | Leaf wetness duration (h)[b] | Rainfall[b] (mm) | Relative humidity >90% (h)[b] |
|---|---|---|---|---|---|
| $DI_{lower}$ | ns | ns | ns | ns | ns |
| $DI_{upper}$ | ns | ns | ns | 0.8[c]<br>0.05[d]<br>10/3[e] | ns |
| $DS_{lower}$ | ns | −0.76<br>0.08<br>10/4 | 0.84<br>0.04<br>16/3 | ns | 0.75<br>0.09<br>10/2 |
| $DS_{upper}$ | ns | ns | ns | 0.93<br>0.006<br>10/3 | ns |
| AUDPC[f] | | | | | |
| $DI_{lower}$ | ns | −0.8<br>0.07<br>16/3 | 0.82<br>0.05<br>10/2 | ns | 0.93<br>0.007<br>10/2 |
| $DI_{upper}$ | 0.95<br>0.004<br>16/4 | 0.88<br>0.02<br>10/3 | 0.93<br>0.0074<br>10/3 | ns | 0.93<br>0.007<br>10/3 |
| $DS_{lower}$ | ns | −0.78<br>0.06<br>16/3 | ns | ns | ns |
| $DS_{upper}$ | 0.96<br>0.0023<br>16/4 | 0.84<br>0.036<br>21/4 | 0.99<br>0.0002<br>10/3 | ns | 0.95<br>0.003<br>21/4 |

[a] Disease incidence (DI) and severity (DS) at harvest. $DI_{lower}$ and $DI_{upper}$ are incidences of disease on leaves from lower and upper halves of shoots, respectively; $DS_{lower}$ and $DS_{upper}$ are severities of disease on leaves from lower and upper halves of shoots, respectively.
[b] Cumulative. ns = not significant.
[c] Correlation coefficient.
[d] Significance level.
[e] The number of days (first number) prior to the weekly sampling day within a growth period (second number) over which the weather variable was summarized. For example, 10/4 indicates that a variable was summarized during the 10-day period prior to the fourth weekly sampling date. Harvest occurred after sampling date four.
[f] AUDPC associated with $DI_{lower}$ and $DI_{upper}$ are the areas under the disease incidence progress curves for leaves from the lower and upper halves of shoots, respectively; AUDPC associated with $DS_{lower}$ and $DS_{upper}$ are the areas under the disease severity progress curves for leaves from the lower and upper halves of shoots, respectively.

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