Analysis of Epidemics of Leptosphaerulina Leaf Spots on Alfalfa and White Clover in Time and Space

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ABSTRACT


The spatial and temporal development of leaf spots on alfalfa (Medicago sativa 'Arc') and Ladino-type white clover (Trifolium repens 'Regal') caused by Leptosphaerulina trifoli (Trifoliagr. Petr. 11,12). Losses in quality and yield of forage and reduction in stand are attributed to this pathogen either alone or in disease complexes (1,2,6). Strategies to manage these leaf spot diseases often include the use of resistant cultivars and cultural practices (6,11,12).

In previous studies, we found that isolates of L. trifoli obtained from alfalfa and white clover can infect either of the two legume hosts under controlled-environment conditions (14). Because of the similarity in the ecology and geographic occurrence, nutrient requirements, and weather conditions needed for cultivation of alfalfa and white clover, it is important to understand this cross-infectivity phenomenon as it occurs under field conditions. Management strategies could be improved if sources of inoculum of Leptosphaerulina spp. could be identified and if the characteristics of disease progress and spread from one host to another were described. No information is currently available on the development of disease gradients of Leptosphaerulina spp. over time from a focus of infected alfalfa or white clover.

Spatiotemporal analysis of plant disease epidemics generally follows one of two methodological approaches (3). In the first approach, no distinct disease focus is observable, and the method used is an expansion of time-series analysis that includes spatial pattern as an added dimension (16,17). In this method, a determination can be made with regard to the influence of factors within or external to a location on epidemiological characteristics. In the second approach (9), a distinct, initial focus of disease is present, and the models used are a combination of those used in traditional disease gradient analysis (7,13) and those used to describe curves of disease progress over time (3,22). Jeger (9) developed two-dimensional models to describe disease increase by considering the rate of isopathic movement (change in distance of a given level of disease per unit of time from a focal center). Differential equations from models that describe disease progress (logistic and monomolecular; 22), and spread (power law and exponential; 7,10,13) were combined into pairs of equations based on the characteristics of the pathosystem and possible relationships with rates of isopathic movement.

The goals of this study were 1) to determine if plants of alfalfa or white clover infected with L. trifoli can serve as inoculum sources for healthy alfalfa and white clover plants under field conditions and 2) to characterize the spatial and temporal aspects of disease progress for Leptosphaerulina leaf spots on alfalfa and white clover.

MATERIALS AND METHODS

Field plot establishment. Plots were established in a 0.7-ha field at North Carolina State University Research Unit 1 in Wake County in spring 1987, fall 1987, and spring 1988. Millet (Pennisetum glaucum (L.) R. Br.) was planted in the field in 1986. Residue of each previous crop was incorporated as completely as possible into soil before establishing plots in order to minimize any carryover of inoculum of L. trifoli. For each study, 12 or 16 plots (3.7 × 3.7 m) were established on a 3 × 4 or 4 × 4 grid with 6.1 m between adjacent plots. A central area (0.5 × 0.5 m) was established in the center of each plot to serve as the disease focus.

In spring 1987, oats (Avena sativa L.) were planted as a buffer zone to minimize inoculum exchange between and within plots (among alleys) and also to intercept inoculum of Leptosphaerulina spp. from outside the field. Oats were 60-70 cm tall at the time of plot establishment. Volunteer oats and summer-seeded maize (Zea mays L.) served as buffer crops for the fall 1987 experiment.

Rye (Secale cereale L.) was planted as a buffer crop in October 1987 for the spring 1988 experiment. For each experiment, the
cereal plants in the 0.5- × 0.5-× 0.5-m central area and in 0.3-m-wide strips in eight equally spaced compass directions were cut to a height of 4–6 cm 2–4 days before establishment of alfalfa or clover plants.

At least 100 8-wk-old seedlings each of alfalfa cultivar Arc and white clover cultivar Regal in 5-cm-diameter pot plants in a commercial potting mix (Metro Mix, W. R. Grace & Co., Cambridge, MA) were inoculated 1 yr. after the experiment with a mixture of isolates of L. trifolii from alfalfa or white clover, respectively. Four isolates of L. trifolii obtained previously from alfalfa and four isolates from white clover were used in spring and fall 1987; however, only two of the isolates from alfalfa and the four isolates from white clover were used in spring 1988 because of a loss of viability of two isolates from alfalfa. The isolates used were designated A86WA4, A86WA1, A85WA1, and A82R62 for alfalfa, and C86WA1, C86WA2, C86WA3, and C85WA1 for clover. (A or C is the host of origin, and Wa or Rw indicate Wake or Rowan County as the place of origin.) The pathogenicity of the isolates on both alfalfa and white clover was confirmed in a detached leaf assay in moist-chamber petri dishes.

Agar plugs of each isolate with actively growing mycelium and immature pseudoblasts were transferred to V-8 juice agar in 9-cm-diameter petri dishes. Cultures were incubated at room temperature (21–24°C) with 12–14 hr day−1 of supplemental illumination from fluorescent lights (GE Fa6T12/CW, 75W) for 7–10 days. To prepare ascospore suspensions, the surface of sporulating cultures was scraped gently with a knife, the mycelial and pseudoblastic agitation was blended in distilled water, and the suspension was filtered through cheesecloth to remove excess mycelial debris. Two to five drops of Tween 20 (polyoxyethylene sorbitan, monolaurate, Sigma Chemical Co., St. Louis, MO) per 100 ml of suspension were added as a surfactant. Inoculation was conducted by atomizing ascospore suspensions (1 × 106 ascospores ml−1) onto plants until runoff. Inoculated plants were incubated in moist chambers in the dark for 72 hr and then exposed to natural light for 7–10 days in the greenhouse to permit symptom development.

Four plants with typical symptoms of Leptosphaerulina leaf spot (mean disease severity 5–10%) were transplanted 4–10 days after inoculation into the central area of each plot to serve as the disease focus. Healthy plants were transplanted at 15.2-cm intervals into the radial arms around the disease focus to serve as the recipient host, with the first plant placed 15.2 cm from the edge of the disease focus. The last plant in each arm was 136.8 cm from the edge of the disease focus. To detect possible interplot interference, as well as external sources of inoculum, potted plants of healthy alfalfa and clover were deployed at intervals around the field and in areas between the plots.

To confirm that symptoms on initially healthy plants were caused by L. trifolii, one or two leaves of alfalfa or white clover were selected arbitrarily in plots from the recipient host plants before the last disease assessment in fall 1987. Leaves were surface-disinfested in 0.26% NaOCl solution for 15–20 sec, placed in a moist-chamber petri dish with 1.5% water agar in the lid, and incubated for 5–7 days with 12 hr day−1 fluorescent light at 22–24°C. After incubation and ejection of ascospores into the water agar, one germinated ascospore was selected arbitrarily from each moist chamber and transferred to V-8 juice agar in a 9-cm-diameter petri dish. Cultures were incubated for 7–10 days, and the growth of isolates was classified as restricted (= colony diameter <4 cm with irregular margins) or expanded (= colony growth covered agar surface) (14).

**Experimental design.** The experiment consisted of a 2 × 2 factorial arrangement of treatments (two recipient hosts and two different diseased-source plants) in a completely randomized design with four replicates in spring 1987 and 1988 and three replicates in fall 1987. Treatment combinations were designated AA, AC, CC, and CA, where A refers to alfalfa and C to clover. The first letter in each treatment combination refers to diseased plants at the focus and the second letter indicates the initially disease-free plants in the radial arms.

**Measurement of disease progress and environment.** Disease severity was assessed visually twice per week on each initially disease-free plant around each disease focus. Assessment began approximately 2–3 wk after transplanting. Disease severity on each of the nine plants per transect (radial arm) for a total of 72 plants per plot was assessed visually with the aid of a rating diagram with logarithmic intervals (20). Potted plants of alfalfa and white clover placed between plots for the detection of background contamination and interplot interference were also examined at each assessment. Temperature, relative humidity (RH), rainfall, leaf-wetness, and wind velocity and direction in the field were monitored by means of electric sensors attached to a data logger (Model CR21, Campbell Scientific, Logan, UT) located in the center of the field. Environmental data were summarized by use of SAS’s PROC SUMMARY (18).

**Data analysis and model evaluation.** Area under the disease progress curve (AUDPC) (19) was computed to characterize disease progress in time among the four treatments. Units for AUDPC are percent-days (an observed percentage of disease that occurred over a number of days). Volume under the disease progress curve (VUDPC) in time and space was calculated by an extension of the method for calculating AUDPC such that:

\[
VUDPC = \sum_{i=1}^{n} \left( \frac{y_t + y_{t+1}}{2} \right) (d_t - d_{t-1})
\]

where \( i \) is the time dimension, \( j \) is the distance dimension, \( y \) is disease severity, \( t \) is time, \( d \) is distance, and \( n \) is the number of observed times and distances. Units of VUDPC are percent-days-centimeters (an observed percentage of disease occurring over a number of days at a number of distances measured in centimeters from the disease focus). Disease severity data were averaged by treatment, distance, time, and plot for computation of AUDPC and VUDPC. Analysis of variance for VUDPC was performed by use of SAS’s PROC ANOVA (18). Three-dimensional plots were generated from the mean disease severity values across replications at each distance and time of assessment for each treatment combination (18).

The eight models of disease increase in time and space proposed by Jeger (9) were evaluated for goodness-of-fit to the data by means of nonlinear regression (PROC NLIN; 18) and the Marquardt compromise for estimation of parameters. A new model was also developed and evaluated by means of nonlinear regression analysis to take the dimensions of time and space and the shape of the disease progress curves into account. The functional, integrated form of the model was:

\[
y = a(1 - \exp(-bt))/\left(1 + \exp(c(d-e))\right)
\]

where \( y \) is the disease severity (proportion) at \( d \) units (distance in centimeters) from the infection focus at time of assessment \( t \), \( a \) is a scaling parameter with \( a/2 \) equivalent to the maximum disease severity at the focus edge, \( b \) represents the rate of disease increase over time (days), and \( c \) is the slope of the disease gradient.

**RESULTS**

**Disease development.** Characteristic pepper spot or Leptosphaerulina leaf spot symptoms were observed on initially disease-free plants in each plot within 14–21 days after placement of the focal source plants. No disease was observed at any time on the initially healthy plants of alfalfa or white clover placed around the field or between plots to detect possible interplot interference. Colonies of L. trifolii obtained from leaflets of alfalfa or white clover in fall 1987 were consistently (12 of 12 cases) of the expanded type when the host at the disease focus was alfalfa. In eight of 11 cases, colonies were of the restricted type, whether obtained from alfalfa or clover leaflets, when white clover was the host at the disease focus. In the other three cases, isolates from clover leaflets infected from a clover plant at the disease focus were of the expanded colony type.
Maximum disease severity did not exceed 3% in the spring 1987 experiment. Total rainfall during the experiment (from day of year [DOY] 140–181) was only 5.3 cm, mean RH was 69.7%, mean leaf wetness duration was 6.2 hr day⁻¹, and mean maximum and minimum daily temperatures were 30.3 and 18.8 C, respectively. Wind was generally from the south-southeast (mean = 201°) at a mean speed of 0.77 m s⁻¹; however, no differences in disease severity were observed between plants in radial arms downwind vs. upwind from the disease focus. Disease developed primarily around infection foci in treatments AA and CA (Fig. 1) with a maximum AUDPC of 34.8 and 30.4 %-days, respectively, at a distance of 15.2 cm from the edge of the focus. Very little disease was present on plants in treatments CC and AC with maximum AUDPC of <2.4 %-days during the 28-day observation period. VUDPC values (Fig. 2) ranged from 81.9 to 1,512.7 %-days-cm for treatments CC and AA, respectively. VUDPC was greater (P = 0.05) in treatment AA than in treatment CA and was greater (P = 0.05) in treatments AA and CA than in treatments AC and CC.

During the fall of 1987, mean maximum disease severity was 3.8, 2.2, 1.6, and 2.1% for treatments AA, CA, AC, and CC, respectively (Fig. 3). Total rainfall for the 38 days of disease assessment (DOY 266–324) was 8.1 cm, mean RH was 79.0%, mean leaf wetness duration was 9.5 hr per day, and mean maximum and minimum daily temperatures were 21.0 and 7.4 C, respectively. Wind was generally from the southeast (mean 148.7°) at a mean speed of 0.79 m s⁻¹; no differences in disease gradients attributable to wind direction were observed. Maximum AUDPC (at 15.2 cm from the edge of the disease focus) was 98.0, 54.2, 54.8, and 44.1 %-days (Fig. 1) for plants of treatments AA, CA, AC, and CC, respectively. VUDPC values ranged from 1,257.2 to 3,779.5 %-days-cm in treatments CC and AA, respectively (Fig. 2). VUDPC was greater (P = 0.05) in treatment AA than in treatment CA and was greater (P = 0.05) in treatments AA and CA than in treatments AC and CC.

In the spring of 1988, mean maximum disease severity ranged from 1.1% on plants of white clover in the CC treatment to 2.8% on plants of alfalfa in the AA treatment (Fig. 4). Total rainfall for the period DOY 116–165 was 14.2 cm, mean RH was 78.0%, mean leaf wetness was 10.6 hr day⁻¹, and mean maximum and minimum daily temperatures were 26.3 and 11.9 C, respectively. Wind was generally from the south (mean 178.1°) at a mean speed of 0.54 m s⁻¹; no differences in disease gradients attributable to wind direction were observed. Maximum AUDPC (at 15.2 cm from the edge of the disease focus) was 75.3, 59.0, 40.6, and 44.1 %-days for plants of treatments AA, CA, AC, and CC, respectively (Fig. 1). VUDPC values ranged from 1,184 to 5,011.8 %-days-cm in treatments AC and AA, respectively (Fig. 2). VUDPC was greater (P = 0.05) in treatments AA and CA than in treatments AC and CC.

**Model evaluation.** Values of the coefficient of determination, plots of standardized residuals vs. predicted values, and plots of predicted values vs. actual values were evaluated to subjectively assess appropriateness of models for describing the data. Based
on these criteria, among the eight models proposed by Jeger (9) to describe disease progress in time and space, only the monomolecular/negative exponential could be considered as a candidate to describe the epidemics of Leptosphaerulina leaf spot. The monomolecular/negative exponential model can be written as:

\[ y = a \exp(-cd)(1 - \exp(-b\theta)) \]  

(3)

with variables and parameters as defined previously except \( a \) is an estimate of maximum disease severity. Estimates of the maximum disease severity \( (\alpha) \) were consistently greater than the actual values with this model for treatments AA and CA in spring 1987 with estimated \( R^2 \) values of 51 and 58%, respectively (Table 1). For fall 1987, \( R^2 \) values were greater than for spring 1987 (Table 1) and estimates of \( a \) were somewhat high but realistic. For disease progress in spring 1988, \( R^2 \) values could be calculated only for three of four treatments and values of \( a \) were again realistic. Most of the residual plots for the monomolecular/negative exponential model were judged to be acceptable.

The monomolecular/negative logistic model (equation 2) better described the disease progress data from all treatments and experiments than did the monomolecular/negative exponential model. \( R^2 \) values were greater and standard errors of the estimates for \( a, b, \) and \( c \) were lower in most cases for the monomolecular/negative logistic than for the other model (Table 1). All residual plots were judged to be acceptable. Values of \( a/2 \) were similar to actual observed values of maximum disease severity. The estimates of \( b \) and \( c \) were not correlated for either the monomolecular/negative exponential or the monomolecular/negative logistic model. No distinct differences in degree of correlation among estimates of values of \( a \) vs. \( b \) or \( a \) vs. \( c \) could be discerned between the two models (Table 1).

**DISCUSSION**

Leptosphaerulina leaf spots are prevalent in the spring and fall on white clover and alfalfa, but disease severity rarely exceeds 10-12% and usually is 5-6% or less (2,20,21,23) in North Carolina. Mean disease severity in our experiments ranged from less than 1% to nearly 4%. Lower amounts of rainfall, relative humidity, and leaf wetness duration and higher mean maximum and minimum daily temperatures of 30.3 and 18.8 C, respectively, probably contributed to the lower disease levels observed in spring 1987 compared to the levels in fall 1987 and spring 1988. Occurrence of Leptosphaerulina leaf spots of alfalfa decreased in previous field studies when mean daily maximum temperatures approached 30 C (23).

Generally, levels of Leptosphaerulina leaf spot may have been lower in our 1987 and 1988 field experiments than in actual fields of white clover or alfalfa because of the physical arrangement of plants in rows only one plant wide around the central disease focus. The lack of adjacent clover or alfalfa plants on two sides may have resulted in an altered microenvironment with reduced leaf wetness and, perhaps, even slightly higher ambient temperatures. Although no specific attempt was made to characterize the microenvironment in a field of alfalfa or a clover-grass pasture in this study, the possible alterations could tend to reduce disease severity in our experiments compared to that in actual fields with alfalfa or white clover.

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Fig. 3. Three-dimensional curves representing mean disease severity (%) at each distance (cm) from the infection source on each day of disease assessment in an experiment with four treatment combinations arranged in a completely randomized design. During fall 1987, day 1 corresponds to day of year (DOY) 266; plant placement occurred on DOY 266. Treatments were: A, infected alfalfa source and healthy recipient alfalfa plants (AA); B, infected white clover source and healthy white clover host (CC); C, infected alfalfa source, healthy white clover hosts (AC); and D, infected white clover source and recipient alfalfa host (CA).

1344 PHYTOPATHOLOGY
Plants of alfalfa and white clover infected with *L. trifolii* served as an inoculum source for initially disease-free plants of both alfalfa and white clover in the field. This effectiveness of both diseased alfalfa and white clover in providing inoculum to both hosts confirms our previous results from controlled-environment studies (14) and the results of others (5,8). Cultural morphology of isolates *L. trifolii* recovered from alfalfa or white clover generally was similar to that of the isolates used to inoculate plants at the disease focus. Three isolates recovered from leaflets of white clover when white clover was the host at the disease focus, however, had the expanded colony type. These observations confirm our observations (14, unpublished data) and those of others (5) that isolates of *L. trifolii* from alfalfa generally have an expanded colony type while isolates from white clover have a restricted colony type. Although the sample size is too small to allow comment on the significance of this observation, colony morphology among isolates of *L. trifolii* from different legume hosts should be investigated further to determine if colony type has any relationship to disease components, such as a latent or infectious period, and to ascertain whether true biotypes of *L. trifolii* exist in nature.

The efficacy of alfalfa and clover plants as recipient hosts and inoculum sources apparently differs. Based upon rankings of VUDPC for the four treatments, alfalfa was a better recipient host for *L. trifolii* than was white clover, regardless of whether alfalfa or white clover was the host at the disease focus. Differences in efficacy of alfalfa or white clover as source or recipient hosts may relate to differences in canopy architecture between the two plants. Alfalfa plants are generally more upright and taller than plants of white clover and may serve to intercept ambient inoculum of *L. trifolii* better than the shorter, more prostrate plants of white clover. Alternatively, alfalfa plants of the cultivar Arc may be more susceptible to the disease than white clover plants of the cultivar Regal.

Alfalfa was also apparently a better source of inoculum for development of disease on alfalfa than was clover. The same was not true, however, when white clover was the recipient host. Because alfalfa source plants were inoculated only with a mixture of alfalfa isolates of *L. trifolii* and white clover source plants were inoculated only with a mixture of white clover isolates of *L. trifolii*, the greater efficacy of alfalfa as a source of inoculum when alfalfa was the recipient host may relate to the relatively greater sporulation by alfalfa isolates on detached leaves than that of clover isolates (14).

The spatiotemporal modeling efforts provided an opportunity to characterize epidemics of *Leptosphaerulina* leaf spots more fully than would be possible with temporal or spatial analysis alone. Among the models proposed by Jeger (9), the monomolecular/negative exponential model provided the best overall description of the leaf spot epidemics. In the original development of the models as provided by Jeger (9), this would indicate that disease increase in time was monoeicotic. With regard to distance, the interpretation would be that the rate of isopathic movement was constant or, in other words, that the rate of isopathic movement was independent of the amount of disease, i.e., the rate was the same at all levels of disease. Such interpretations may not always be appropriate (15), but they do provide an opportunity to develop working hypotheses. Because of the relatively long latent period of *L. trifolii* (at least 14–18 days under conducive, controlled-environment conditions) (14), the general lack of leaf senescence and defoliation (which are needed for sporulation of *L. trifolii*) that was observed during our studies.

![Diagram](image)

**Fig. 4.** Three-dimensional curves representing mean disease severity (%) at each distance (cm) from the infection source on each day of disease assessment in an experiment with four treatment combinations arranged in a completely randomized design. During spring 1988, day 1 corresponds to day of year (DOY) 130; plant placement occurred on DOY 116. Treatments were: A, infected alfalfa source and healthy recipient alfalfa plants (AA); B, infected white clover source and healthy white clover hosts (CC); C, infected alfalfa source, healthy white clover hosts (AC); and D, infected white clover source and recipient alfalfa host (CA).
TABLE 1. Maximum mean disease severity, estimates of parameters (a, b, and c), standard error of the estimate, and correlation between parameter estimates for two models used to describe increase of Leptospaeraulina leaf spot in space and time in four treatment combinations in a gradient study with alfalfa and white clover during three seasons

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Parameter estimate</th>
<th>Standard error of the estimate</th>
<th>Correlation between parameter estimates</th>
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<tr>
<td></td>
<td>y-max (%)</td>
<td>a</td>
<td>b</td>
</tr>
<tr>
<td>Monomolecular/negative exponential</td>
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<tr>
<td>AA</td>
<td>2.8</td>
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<td>CA</td>
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<tr>
<td>CC</td>
<td>2.1</td>
<td>3.428</td>
<td>0.057</td>
</tr>
<tr>
<td>Spring 1987</td>
<td></td>
<td></td>
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<tr>
<td>AA</td>
<td>2.8</td>
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1. Treatment combinations: A = alfalfa, C = white clover; first letter designates infected host, second letter designates recipient host plant.
2. Maximum mean disease severity.
3. $R^2$ = coefficient of determination.
4. The monomolecular/negative exponential is $y = a \exp(-cd)x(1 - \exp(-bx))$, and the logistic is $y = a(1 - \exp(-bx))(1 + \exp(cx))$ where $y$ is the percent of disease, $a$ is a scaling parameter related to maximum disease severity, $b$ is the rate of disease increase over time, and $c$ is the slope of the gradient over distance.

and the lack of observed flattening of the disease gradients over time (Figs. 3 and 4), it is plausible that our observations of disease occurred primarily during a monocyte of the epidemic.

The interpretation of the rate of isopack movement and characteristics of the gradient for Leptospaeraulina leaf spot on alfalfa and white clover is complicated by the difficulties in inferring the biology of the pathosystem from the model (3,15) and the result that the newly proposed monomolecular/negative logistic model better described the epidemics than did Jeger's monomolecular/negative exponential model. The appropriateness of the negative logistic model for distance suggests that a possible plateau or shoulder occurred in level of disease severity close to the disease focus. The presence of such in the initial flattening or shoulder in the disease curve over distance is somewhat evident for treatments AC and CA in fall 1987 (Fig. 3C and D) and in treatments AA and CA in spring 1988 (Fig. 4). Biologically, this may occur because of the operation of two mechanisms or modes of dispersal for ascospores of L. trifoli. The plateau in disease level near the focus may represent the result of dispersal primarily because of the forcible ejection of ascospores from the pathosystem (4), i.e., ascospores are ejected and propelled a limited distance after which they land and initiate disease. The second mechanism would then be a combination of forcible ejection and dispersal by wind. Only a limited portion of the ascospores ejected might escape the canopy and be propelled to a height sufficient to allow entrainment into moving air currents above the canopy. This second mechanism would then account for the dispersal of spores over longer distances and would result in the typical, concave curve of disease severity along a dispersal gradient. Also, saturation of available infection sites near the focus could account for a portion of the observed plateau in the disease gradient. Studies are needed on the ballistics of ascospore ejection from pseudotheca of L. trifoli and on the dispersal dynamics of the ascospores to further explain the observed plateau in the dispersal gradient near the disease focus.

LITERATURE CITED


