Quantitative Relationships Between Yield and Foliar Diseases of Alfalfa

S. C. Broscious, J. K. Pataky, and H. W. Kirby

Former graduate research assistant and assistant professors, respectively, Department of Plant Pathology, University of Illinois at Urbana-Champaign, Urbana 61801.
Present address of first author: American Cyanamid Company, Agricultural Research Center, P.O. Box 400, Princeton, NJ 08540.
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ABSTRACT

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Regression models relating various measures of disease level to yield were developed for the complex of fungi that cause foliar disease on alfalfa (*Medicago sativa*). Different levels of leaf spot epidemics were established in each of 21 trials using natural inoculum, fungicide applications (mancozeb alone or with benomyl), and inoculations with *Phoma medicaginis* and/or *Stemphylium botryosum*. Models to predict yield were fit by ordinary-least-squares regression for each trial using weekly disease severity assessments, defoliation index, or area under disease progress curve (AUDPC) as the independent variable. More than one model adequately described the relationship between disease and yield in each trial. Disease severity 1 wk before harvest (S_1), severity on the day of harvest (S_0), and AUDPC were the variables most consistently related to yield in individual trials. Linear models using each variable to predict the percentage of the maximum yield in the trial explained 52–60% of the variation in the yield data when pooled over 15 of the trials. Models estimated a yield reduction of 2.40, 1.83, and 0.14% for each unit of S_1, S_0, and AUDPC, respectively. The accuracy and precision of the models were validated using data other than those from which the models were developed. Model predictions were relatively accurate within the range of disease levels commonly observed in alfalfa fields. However, at higher levels of disease, all three models underestimated the effects of foliar disease on yield when compared with data from the six additional trials. Mean differences between the model prediction and observed values were +5.0, +2.3, and +2.7% of maximum yield for the S_1, S_0, and AUDPC models, respectively.

Additional key words: crop loss, epidemiology, modeling, yield loss.

Foliar diseases significantly reduce the yield and quality of alfalfa (*Medicago sativa* L.) hay. Yield losses of 10–42% in individual cuttings (8,13,16,17,20,21) and annual losses of 9–27% (22) are caused by fungal leaf spot pathogens. Foliar pathogens cause increased defoliation (8,16,17,20,22) and are associated with decreases in hay digestibility (14), crude protein (6,12,14), and carotene content (22).

Most crop loss estimates for alfalfa leaf spots have compared mean yield from plots with high levels of disease with yields from plots that are relatively disease free. This approach provides an estimate of the damage potential of these foliar diseases, but it is severely limited in application to situations where different levels of disease occur. A more widely applicable approach is to develop a quantitative model that describes the relationship between disease and yield over a continuous range of disease levels. Applications of such models include use with disease survey data to improve estimates of regional crop losses, determination of economically optimal disease-severity level for different control methods, and incorporation of crop loss predictions into comprehensive disease control programs that require economic rationalization of control decisions (9,11).

Several quantitative yield-loss models exist for individual foliar pathogens of alfalfa. Horsfall's (10) model estimates yield reduction by multiplying each percentage incidence of infected leaves by 0.25. Berkenkamp (5) used a similar model to estimate losses caused by individual foliar diseases of forage crops in Canada. However, Berkenkamp's estimates of yield loss were derived by multiplying 0.25% by a disease index for each field. Disease index values ranged from 0.1 to 75.0 and corresponded to a descriptive key that combined disease incidence, severity, vertical progress, and defoliation. Based on observations of natural epidemics caused by *Pseudopeziza medicaginis* (Lib.) Sacc. and inoculations with *Stemphylium botryosum* Wallr., Basu (2) concluded that defoliation was the primary component of yield reduction because the dry weight of alfalfa leaves was not affected by the level of disease severity. He hypothesized that because the ratio of leaf to stem weight was approximately one at early to midbloom, an estimate of yield loss could be obtained by multiplying the proportion of leaf weight (0.5) by the percentage defoliation as measured by counting leaves and leaf scars.

The models used by Horsfall (10) and hypothesized by Basu (2) for predicting yield loss caused by common leaf spot (*P. medicaginis*) of alfalfa have been compared (4). Although values for the independent variables, defoliation and incidence, were moderately correlated, pairwise comparisons of loss estimates from the two models were highly significant ($P \leq 0.01$) for the 3 yr that data were collected. Similar comparisons of arcsine or square-root-transformed yield-loss estimates also were different in all cases.

Neither the precision nor the accuracy of these models has been evaluated. In addition, they attempt to attribute losses to individual pathogens despite the fact that foliar pathogens of alfalfa occur as a complex and cause symptoms that often are not diagnostic for the organisms involved (15). The objectives of this study were to develop and compare yield loss functions for the complex of fungi that cause foliar disease on alfalfa and to evaluate the accuracy and precision of the models using data other than those used to develop them. A preliminary report has been published (7).

MATERIALS AND METHODS

Experiment design and epidemic manipulation. Twenty-one trials were conducted during the second, third, and fourth harvest periods during 1984 and 1985 on a stand of Raidor alfalfa in Clinton County, Illinois, and Vernal alfalfa in Champaign County, Illinois. Both stands were 2 yr old in 1984. Soil types at the locations were Cisne silt loam (fine, montmorillonitic, mesic Mollic Albaqualf) and Proctor silt loam (fine silty, mixed, mesic Typic Arquidoll), respectively. Except for application of foliar fungicides and fungal inoculations, standard alfalfa production practices were followed throughout the study (1). A randomized complete block design and plots measuring 1.5 × 7.6 m were used in all trials, but treatments and numbers of replications varied between trials.

In addition to natural foliar disease epidemics and pathogen complexes, a broader range of epidemics and complexes was established by inoculations and applications of fungicides. Mancozeb (80WP) was applied alone at 2.2 kg/ha or as a tank-mix combination with benomyl (50WP) at 1.1 kg/ha according to various schedules depending on the trial. All fungicide treatments were applied in 327 L of water per hectare using a CO_2-pressurized backpack sprayer with D5-23 nozzles (Spraying Systems Co., Wheaton, IL) on 25.4-cm centers operated at 276 kPa and 1.4 m/sec. Triton B-1956 (Rohm and Haas Co., Philadelphia, PA) was included as a surfactant with all fungicide treatments at 313 μl/L of spray.

Inoculations were made with single-spore isolates of *Phoma medicaginis* Malbr. & Roum. var. *medicaginis* Boerema and *S. botryosum* that were collected in Champaign and Clinton counties, respectively, in 1983. Plots were inoculated with these fungi either alone or in combination. Inoculum was prepared by spreading 1-ml aliquots of a spore suspension of *S. botryosum* on V-8 agar and *P. m.* var. *medicaginis* on oatmeal agar in petri plates measuring 100 × 15 mm. Plates were incubated at 20–22 C for 10–14 days with cultures of *S. botryosum* exposed to 12 hr/day of light. Inoculum concentrate was prepared by grinding fungal cultures with distilled water in a blender. Just before inoculation, a volume of concentrate equivalent to five petri plates of *S. botryosum* and/or two plates of *P. m.* var. *medicaginis* was diluted with tap water to 1 L per plot. Inoculum was applied between 1900 and 2100 hr using a gasoline-powered backpack mist blower (Solo Kleinmotoren GMBH, West Germany) calibrated to dispense 1 L/min.

One of four sets of treatments was used to regulate epidemic development in each trial. The four method A treatments were weekly inoculations with *S. botryosum*, weekly applications of mancozeb and benomyl, inoculations and fungicide sprays, and an untreated control. This method was used in 1984, and treatments were replicated 10 and 5 times in the Raidor and Vernal trials, respectively. Method B, which was also used in 1984, consisted of five treatments replicated eight times on Raidor and five times on Vernal. The five treatments of method B were an untreated control, weekly inoculations with *S. botryosum*, and three schedules of mancozeb and benomyl applications at 10; 10 and 18; or 10, 18, and 26 days before harvest. Method C consisted of five treatments applied weekly in 1985 and replicated eight times on both varieties. The five method C treatments were inoculation with *S. botryosum*, inoculation with *P. m.* var. *medicaginis*, inoculation with both fungi, spraying with a tank-mix of mancozeb and benomyl, or no treatment. Method D consisted of one, two, or three applications of mancozeb made successively at weekly intervals beginning after 10–14 days of crop regrowth and an untreated control. This set of treatments was replicated 10 times on both varieties in 1985 trials. Individual trials will be referred to by a corresponding code defining the variety, year, treatment method, and harvest period: R = Raidor, V = Vernal; 84 = 1984, 85 = 1985; A, B, C, and D = treatment methods; and 2, 3, and 4 = second, third, and fourth harvest periods, respectively. For example, trial V85D3 was the experiment on Vernal in 1985 using treatment method D during the third harvest period.

Measurement of disease and yield. Plots were evaluated for percentage leaf area exhibiting disease symptoms without regard to causal organism using standard area diagrams (Fig. 1). Four disease-severity assessments were made during each trial at about weekly intervals beginning after 10–14 days of crop regrowth. Specific evaluations for disease severity will be referred to by the letter S subscripted with a number indicating the approximate number of weeks before harvest that the evaluation was made. For example, S_3 and S_0 refer to disease severity three and zero (day of harvest) weeks before harvest, respectively. Area under disease progress curve (AUDPC) was calculated using the equation:

$$\text{AUDPC} = \sum_{i=1}^{n-1} [0.5(x_i + x_{i+1})][t_{i+1} - t_i] \qquad (1)$$

where x_i = the percentage of foliar disease severity at the ith evaluation, t_i = time of the ith evaluation in days from the first evaluation date, and n_i = total number of times disease was evaluated. In 1984 trials, 10 stems were randomly selected and sampled at ground level from each plot to obtain an estimate of defoliation. Stem length was measured from the base to the terminal growing point of the main stem. A defoliation index (D) was calculated as the length from the stem base to the first leaf attached directly to main stem expressed as a proportion of the total stem length.

In trials where treatment methods A, B, and C were used, 48–96 leaflets were sampled from all plots for each treatment to determine whether different pathogen complexes had been established. Leaflets were surface sterilized in 70% ethanol followed by 0.525% NaOCl, each for 1 min. After two 60-sec rinses in sterile distilled water, leaflets were aseptically transferred to petri plates containing three layers of moist filter paper. Leaflets were incubated for 5–7 days under 12 hr/day of light at 20–22 C. Adaxial leaf surfaces then were scanned using a dissecting microscope at ×40 for the presence of fungal spores and fruiting structures. The incidence per leaflet of foliar pathogens was recorded. Isolates of the prevalent fungi were collected periodically, single spored, and identified to confirm incidence data.

Trials were harvested about every 35 days, which corresponded to 1/10 to 1/4 bloom stage of crop maturity. A flail-chopper (Carter Manufacturing Co., Inc., Brookston, IN) was used to remove a section measuring 0.9 × 5.8 m from the center of each plot with 5–8 cm of stubble remaining. Subsamples of about 1,000 g were weighed and then dried in an oven at 65 C to determine moisture content. Plot yields then were adjusted for plot size and moisture content and converted to dry matter yield in megagrams per hectare.

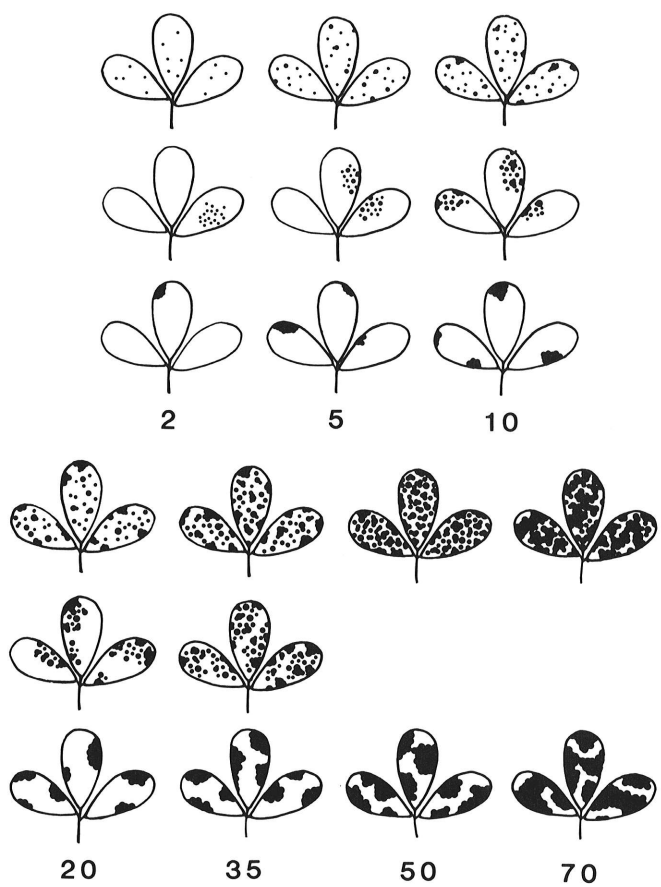

Fig. 1. Standard area diagrams used to evaluate foliar disease severity on alfalfa. Numbers correspond to percentage of leaf area indicated by dark areas of leaves above them.

Model development and validation. Plots and correlations of all possible combinations of weekly disease assessments, D, AUDPC, and yield were examined. For each trial, linear and quadratic models to predict yield were fit with weekly disease-severity assessments, D, or AUDPC as the independent variable using ordinary-least-squares regression. Overall significance of the models was evaluated using the F statistic ($P \leq 0.05$). The improvement from addition of a quadratic term was assessed by examination of the t statistic ($P \leq 0.05$) for the partial regression coefficients. Plots of residuals were examined to determine homogeneity of variance and lack of fit. Coefficients of determination and residuals were used to compare models and to select one "best" model for each trial.

The intercept from the best model for each trial was used to convert yield values to percentage of maximum for the trial. Regression models to predict percentage of maximum yield as a function of disease were fit for each trial. Slope coefficients for percentage of maximum yield models from each trial were tested for homogeneity using analysis of covariance and confidence intervals ($P \leq 0.05$). Data from 15 of the trials (529 observations) were pooled and used to develop an overall model to predict percentage of maximum yield as a function of each disease assessment method. Data from the remaining six trials (205 observations) were used to validate and compare the performance of the models. The six trials used for model validation were selected randomly from subsets of all 21 trials. Subsets were formed according to cutting-variety combinations to represent a range of environments. Data for one trial in each subset (i.e., second cutting trials on Raidor) were included in the model validation. The percentage of estimates within ±10% of the observed yield value and the mean difference between a model estimate and observed yield were calculated for the entire data set and a subset of the data formed based on the range of disease values observed in untreated plots. Trials R85C2 and R85D2 were not used to determine this range because untreated plot values for all predictors were 1.5–2 times greater than those in the next highest trial and therefore were considered to be unrepresentative of the range of disease levels most commonly found in production fields. The range of disease values in the subset of the validation data was $\leq 12\%$ for S_1, $\leq 14\%$ for S_0, and ≤ 220 severity days for AUDPC.

Residual mean squares for each of the models were calculated for both the model construction and model validation data sets. Using these values, models were compared within each data set using the F statistic ($P \leq 0.025$).

RESULTS

Disease symptoms were not observed in any trial until about 2 wk before harvest except in R85C2 and R85D2, where disease severity was approximately 7% after 9 days of regrowth. Disease severity in untreated plots increased with time. Means over trials for S_2, S_1, S_0, and AUDPC were 5.3%, 7.8%, 9.8%, and 146.8 severity days, respectively. In individual trials, means for untreated checks ranged from 4.2 to 19.5%, 4.8 to 20.2%, and 56.0 to 438.1 severity days for S_1, S_0, and AUDPC, respectively. Values for individual plots over all trials ranged from 0.0 to 28.0% for S_0, 0.0 to 22.0% for S_1, 0.0 to 20.0% for S_2, and 0.0 to 506.0 severity days for AUDPC. The minimum mean S_0 for a treatment in each trial was less than 3% except for R85C2, R85D2, V85D3, and V85D4, which had values of 8.9, 7.8, 6.3, and 5.4%, respectively.

Yields from individual plots in the model development data set ranged from 1.7 to 6.3 Mg/ha, and those in the model validation data set ranged from 1.5 to 5.6 Mg/ha. Mean yield from untreated control plots in individual trials for the model development and model validation data sets ranged from 2.1 to 4.6 Mg/ha and 1.8 to 4.6 Mg/ha, respectively. Mean maximum yield for any treatment in an individual trial ranged from 2.0 to 5.7 Mg/ha.

In addition to the variation in epidemic development, the relative proportions of pathogens involved also varied between treatments and trials, as illustrated by the incidence data in trials V84B3 and V85C2 (Table 1). The fungi in control plots varied between trials, and inoculations with a particular fungus usually increased the incidence of that fungus relative to the control. Increasing the number of fungicide applications generally decreased the incidence of all fungi. *P. m.* var. *medicaginis* and *S. botryosum* were the pathogens observed most frequently throughout all trials, but *Colletotrichum* and *Leptosphaerulina* spp. also were important components in the pathogen complexes.

Disease severity and AUDPC were correlated significantly ($P \leq 0.05$) over all trials and within trials except for S_2 with S_1 and S_0 in trial V84B3. Simple correlations between D and the other disease measures were significant except at R84B4. Consequently, multiple-point models to predict yield were not evaluated to avoid problems associated with collinearity of predictors.

All possible models using S_2, S_1, S_0, D, or AUDPC as predictors in linear and quadratic functions were evaluated to predict actual yield in individual trials. Models with S_2 or D as predictors did not explain the variation in yield consistently across trials and thus were dropped from further consideration. In most trials, S_1, S_0, and AUDPC models adequately described the relationship between disease and yield. Slope coefficients for linear models to predict actual yield in individual trials ranged from −0.037 to −0.190 for S_1, −0.028 to −0.117 for S_0, and −0.0025 to −0.0089 for AUDPC. Addition of a quadratic term significantly improved models with S_1 or AUDPC as the predictor in four trials and S_0 in six trials.

When yield data were converted to percentage of maximum and analyzed over trials using analysis of covariance, predictor × trial interactions were significant for all three predictors. Slope coefficients for linear models to predict percentage of maximum yield in individual trials ranged from −0.90 to −4.15 for S_1, −0.68

TABLE 1. Percentage incidence per leaflet of fungal pathogens observed on microscopic examination of leaflets from different treatments in trials on third cutting of Raidor alfalfa in 1984 and second cutting of Vernal alfalfa in 1985

Trial and treatments	*Colletotrichum* spp.	*Leptosphaerulina* spp.	*Phoma medicaginis* var. *medicaginis*	*Stemphylium botryosum*
Raidor third cut, 1984[a]				
Stemphylium inoculation	20	27	88	98
One fungicide spray	7	35	62	48
Two fungicide sprays	2	10	30	17
Three fungicide sprays	0	2	2	13
Untreated check	5	18	88	45
Vernal second cut, 1985[b]				
Stemphylium + *Phoma* inoculation	25	5	92	91
Phoma inoculation	29	15	72	89
Stemphylium inoculation	27	19	96	61
Fungicide sprays	0	0	7	3
Untreated check	31	11	63	40

[a] Treatments: weekly inoculation with *S. botryosum* and three schedules of mancozeb (80WP; 2.2 kg/ha) with benomyl (50WP; 1.1 kg/ha) applications at 10 days; 10 and 18 days; or 10, 18, and 26 days before harvest.

[b] Treatments applied weekly: inoculation with *S. botryosum*, *P. m.* var. *medicaginis*, combination of the two fungi, or three applications of mancozeb (80WP; 2.2 kg/ha) with benomyl (50WP; 1.1 kg/ha).

to −2.51 for S_0, and −0.073 to −0.183 for AUDPC. Pairwise comparisons showed that 17, 18, and 19 of the 21 individual trial slope coefficients for S_1, S_0, and AUDPC, respectively, did not differ. Hence, significant predictor × trial interactions were due to results from 4, 3, and 2 of 21 trials for S_1, S_0, and AUDPC, respectively.

Pooled data from 15 trials were used to develop linear models to predict percentage of maximum yield using S_1 or S_0 as the independent variable (Table 2; Fig. 2A and B). Models estimated yield reductions of 2.40 and 1.83% for each percentage of disease severity observed 1 wk before harvest and on the day of harvest, respectively. Addition of a quadratic term did not improve either model significantly. The proportions of variation in yield explained by the S_1 and S_0 models were 0.56 and 0.60, respectively. A similar model was constructed using AUDPC as the predictor (Table 2; Fig. 2C). Data from trials R85C2 and R85D2 were excluded from the model based on the poor distribution observed in plots of residual vs. predicted values. A linear model for data pooled over the other 13 trials estimated a yield decrease of 0.14% for each severity-day unit of AUDPC accumulated throughout the regrowth period. The model explained 52% of the variation in yield and was not significantly improved by addition of a quadratic term.

Estimates from all models tended to be greater than actual percentage of maximum yield for the 205 observations from the six trials not used to construct the models (Fig. 3). Mean differences between predicted and observed values were +5.0, +2.3, and +2.7% for the S_1, S_0, and AUDPC models, respectively. Model estimates were more accurate at values of the predictor variable below the maximum level commonly observed in untreated control plots. Mean differences between predicted and observed values were +3.8% when S_1 was below 13% and +1.4% when S_0 and AUDPC were below 15% and 221, respectively. All three models predicted values within ±10% of the observed value in at least 75% of the estimates for the full validation data set.

Residual mean square from the model development data set for the AUDPC model was 64.6. This variance was significantly less than that for the other two models, 95.9 for S_1 and 85.8 for S_0, which were not different from each other. Comparing the residual mean square from the validation data set, the S_1 and AUDPC models were homogeneous with values of 95.2 and 90.9, respectively. Both of these values were different from that of the S_0 model, which was 60.8.

DISCUSSION

The models developed in this investigation demonstrate a significant negative relationship between various measures of foliar disease caused by a complex of foliar pathogens and alfalfa hay yields. Mean yield loss rates were 2.4 and 1.8% of maximum yield per 1% S_1 and S_0, respectively. Maximum yield was reduced 0.14% for each severity-day unit of AUDPC accumulated during the regrowth period. The relationships were stable over both varieties despite considerable variation in regrowth-period environment, disease level, and pathogen composition of the

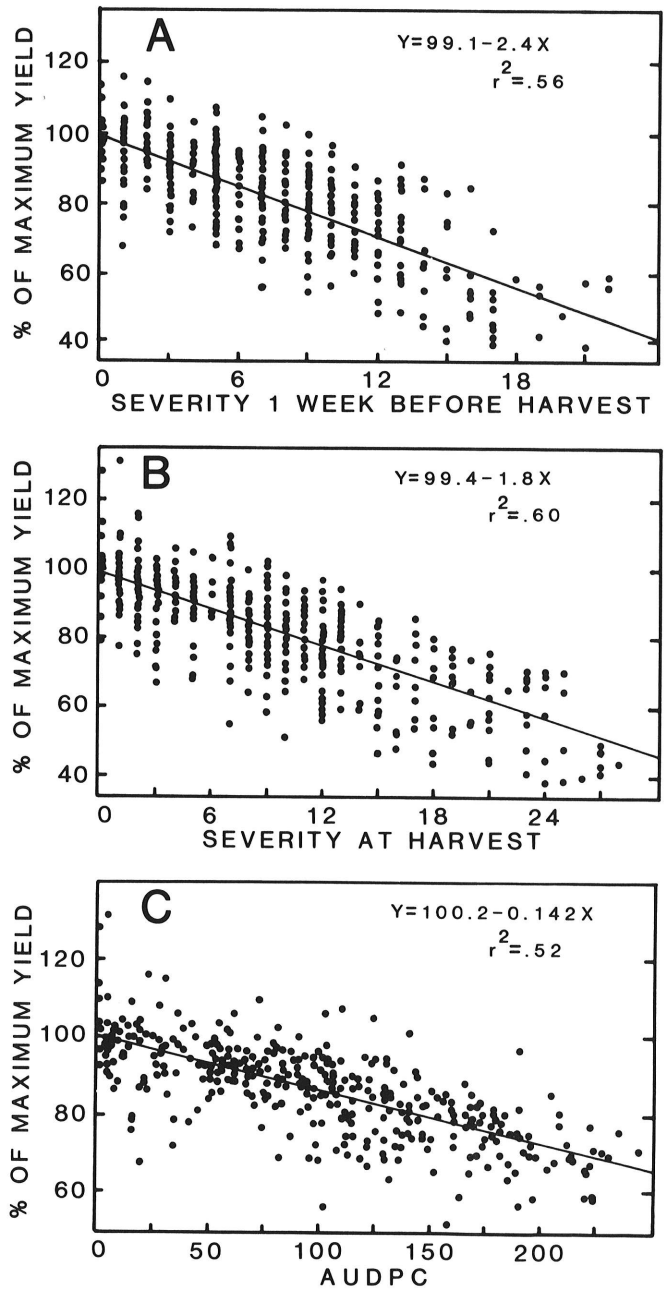

Fig. 2. Regression models to predict percentage of maximum yield as function of **(A)** foliar disease severity 1 wk before harvest (S_1), **(B)** disease severity at harvest (S_0), and **(C)** area under disease progress curve (AUDPC). Plotted data for S_1 and S_0 are 529 observations from 15 trials and for AUDPC are 452 observations from 13 trials on Raidor or Vernal alfalfa in 1984 or 1985 used to develop the regression model.

TABLE 2. Regression models to predict percentage of maximum yield using foliar disease severity 1 wk before harvest (S_1), severity on day of harvest (S_0), and area under disease progress curve (AUDPC)

Independent variable	n[a]	Intercept	Standard error of intercept	Partial regression coefficient	Standard error of regression coefficient	r^2	Standard error of dependent variable
S_1	529	99.08	0.751	−2.40	0.093	0.56	9.79
S_0	529	99.37	0.700	−1.83	0.064	0.60	9.26
AUDPC[b]	452	100.22	0.724	−0.14	0.006	0.52	8.04

[a] Number of observations used in regression model development.

[b] AUDPC $= \sum_{i=1}^{n-1} [0.5(x_i + x_{i+1})][t_{i+1} - t_i]$ where x_i = percentage of foliar disease severity at ith evaluation, t_i = time of ith evaluation in days from the first evaluation date, and n = total number of times disease was evaluated.

disease complex as evidenced by the homogeneity of regression coefficients from individual trial models. The generally good agreement between model estimates and actual yields when models were applied to data that were not used to construct them further validates the models. Such stability implies that these models also may estimate yield reasonably well in other regions. Further validation using methods similar to those in this study should be done in other regions to test this hypothesis before the models can be widely adopted.

The high degree of stability associated with our models also demonstrates that the disease-yield relationship can be successfully modeled for a complex of alfalfa foliar pathogens. This is in contrast to previous yield loss models (2,5,10), which estimate reductions attributed to individual pathogens. Attempts to incorporate a semiquantitative measure of the incidence of specific foliar pathogens did not result in significant model improvement in this study. Based on this and the observation that foliar pathogens generally occur as a complex with highly variable symptom development, we feel justified in recommending that foliar disease severity be assessed in total without attributing amounts to specific pathogens when estimating yield reduction. Techniques such as observation of fungal fruiting structures should be used when estimates of pathogen prevalence are required. Improvements on our semiquantitative technique may enhance the accuracy of future alfalfa yield loss models.

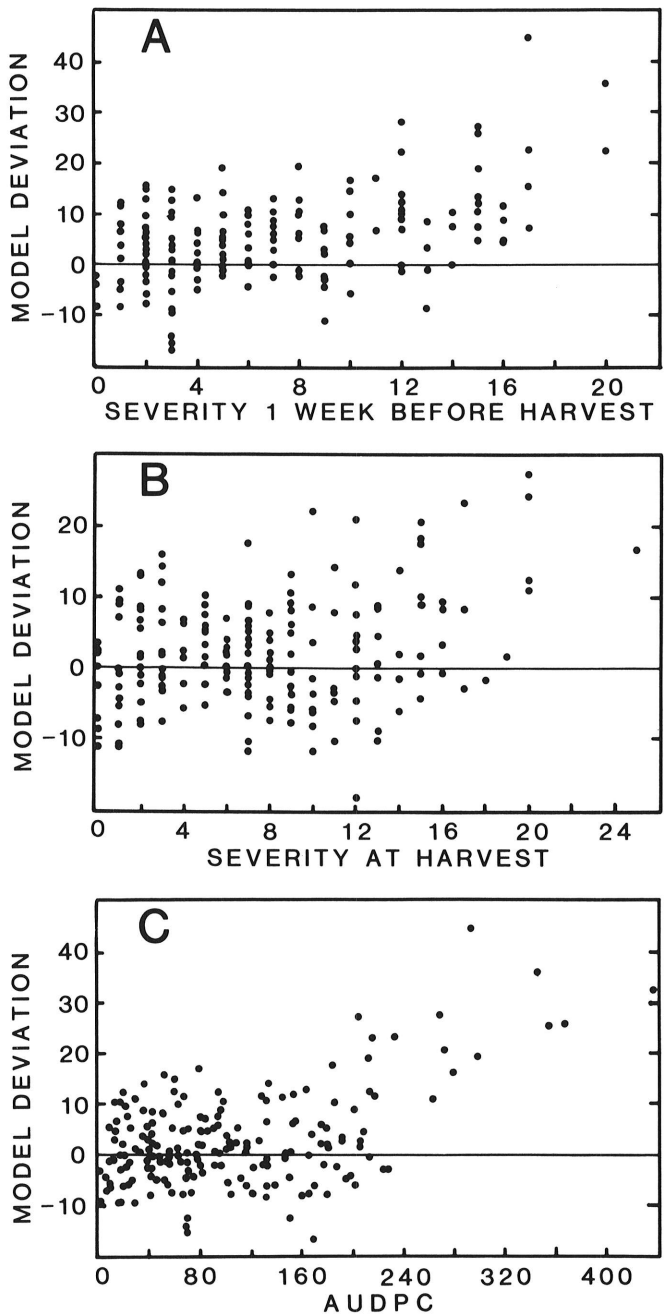

Fig. 3. Difference between percentage of maximum yield regression model prediction and observed percentage of maximum yield (model deviation) plotted as function of **(A)** disease severity 1 wk before harvest (S_1), **(B)** disease severity at harvest (S_0), and **(C)** area under disease progress curve (AUDPC). Predicted and observed percentages of maximum yield values were determined using following models: $Y = 99.1 - 2.4S_1$; $Y = 99.4 - 1.8S_0$; $Y = 100.2 - 0.142$ AUDPC and 205 observation data set not used to develop models.

Both the critical-point and AUDPC models that were developed in this study adequately described the relationship between foliar disease and alfalfa yield. James (11) has generalized that the type of yield loss model most suited to a particular pathosystem is related to the duration of time during which yield is primarily determined and to the onset and length of the epidemic. Since alfalfa yield accumulation and epidemic development occur throughout the entire regrowth period, AUDPC models might be expected to describe the disease-yield relationship better than critical-point models. The equivalency of the two models in this study may be related to the high degree of consistency observed in timing of disease onset and the shape of typical disease progress curves. Most curves were about linear, with severity increasing through the entire regrowth period or reaching a plateau over the last week before harvest. As a result, late-season disease severity values would be a function primarily of the rate of epidemic development and therefore a relative reflection of disease effects over the entire epidemic. The consistency in time of onset and significant correlations between S_1, S_0, and AUDPC values observed throughout this study also support this idea. In trials where onset of disease occurred earlier than usual (R85C2, R85D2), data did not conform to the AUDPC model for the other 13 trials, thus illustrating the inability of AUDPC models to weight disease severity relative to time of occurrence and indicating that application of the AUDPC model developed in this study should be limited to epidemics where onset occurs after at least 14 days of regrowth. No lack of fit was observed when S_1 and S_0 models were constructed over locations; thus, our critical-point models are more robust in terms of tolerating variation in the time of disease onset.

The two critical-point models developed in this study are likely to have wider application and be more readily accepted than the AUDPC model since the latter requires that several disease evaluations be made for each cutting. When used within the range of commonly observed values for their respective predictors, all models were equally precise and therefore no significant benefit would result from making the additional evaluations and calculations necessary to use the AUDPC model.

One important use of the critical-point models would be in obtaining more accurate estimates of losses caused by alfalfa leaf spots on a regional basis. Previous regional estimates have been made for individual pathogens using methods that have not been verified for precision or accuracy (5). The development of regionally validated loss models based on quantitative disease-assessment methods similar to those presented in this study, and their use in conjunction with other research results concerning spatial distributions (19) and survey sampling methods (3) for alfalfa leaf spots, would substantially improve the accuracy and precision of regional loss estimates.

All the models developed in this study involve disease assessments made late in the regrowth period. Since fungicide application for control of alfalfa leaf spots is most effective and economical when done about 10–14 days after regrowth begins (S. C. Broscious, *unpublished data*), it is unlikely that any of the models could be incorporated into an action-threshold decision-directed disease control program. However, the models would have application in determining the economic threshold (sensu Headley [9]) for any number of control methods. Without models

describing the quantitative relations between disease and yield, determination of this economic optimum is difficult. The concept of economically optimizing agricultural production instead of maximizing it has gained increased understanding and acceptance. As this awareness increases, the need for and utilization of validated crop loss models and control cost functions also will increase.

Validation studies are a critical phase in model development that generally has been neglected in previous yield-loss modeling efforts (18). Model evaluation using data not involved with its development provides a more realistic estimate of the true or expected accuracy and performance of the model than judgments based on the standard error of the dependent variable, coefficient of determination, or other parameter estimates associated with the data from which the model was derived. The data set × model interactions for unexplained variation in the yield data observed in this study demonstrate that the criteria commonly used to assess the reliability of yield loss models developed using regression should not be relied on as the only means of doing so.

LITERATURE CITED

1. Barnes, D. K., and Sheaffer, C. C. 1985. Alfalfa. Pages 89-97 in: Forages. M. E. Heath, R. F. Barnes, and D. S. Metcalfe, eds. Iowa State University Press, Ames. 643 pp.
2. Basu, P. K. 1976. Measuring severity of common and Stemphylium leaf spots of alfalfa for loss assessment. Plant Dis. Rep. 60:1037-1040.
3. Basu, P. K., Lin, C. S., and Binns, M. R. 1977. A comparison of sampling methods for surveying alfalfa foliage diseases. Can. J. Plant Sci. 57:1091-1097.
4. Basu, P. K., and Poushinsky, G. 1978. A comparison of two methods for estimating common leaf spot severity and yield loss of alfalfa. Plant Dis. Rep. 62:1002-1005.
5. Berkenkamp, B. 1971. Losses from foliage diseases of forage crops in central and northern Alberta in 1970. Can. Plant Dis. Surv. 51:96-100.
6. Brigham, R. D. 1959. Effect of Cercospora disease on forage quality of alfalfa. Agron. J. 51:365.
7. Broscious, S. C., and Kirby, H. W. 1985. Quantitative relationships between alfalfa leaf spot diseases and yield. (Abstr.) Phytopathology 75:1299.
8. Hart, R. I. K., and Close, R. C. 1976. Control of leaf diseases of lucerne with benomyl. Pages 42-45 in: Proc. N.Z. Weed Pest Control Conf., 29th.
9. Headley, J. C. 1972. Defining the economic threshold. Pages 100-108 in: Pest Control Strategies for the Future. R. L. Metcalf, E. N. Castle, B. E. Day, R. Hansberry, W. J. Hays, A. Kelman, E. F. Knipling, L. D. Newsom, F. W. Slife, and C. M. Williams, eds. Natl. Acad. Sci., Washington, D.C. 376 pp.
10. Horsfall, J. G. 1930. A study of meadow-crop diseases in New York. N.Y. Agric. Exp. Stn. Ithaca Mem. 130. 139 pp.
11. James, W. C. 1974. Assessment of plant diseases and losses. Annu. Rev. Phytopathol. 12:27-48.
12. Mainer, A., and Leath, K. T. 1978. Foliar diseases alter carbohydrate and protein levels in leaves of alfalfa and orchardgrass. Phytopathology 68:1252-1255.
13. Morgan, W. C., and Parbery, D. G. 1977. Effects of Pseudopeziza leaf spot disease on growth and yield in lucerne. Aust. J. Agric. Res. 28:1029-1040.
14. Morgan, W. C., and Parbery, D. G. 1980. Depressed fodder quality and increased oestrogenic activity of lucerne infected with *Pseudopeziza medicaginis*. Aust. J. Agric. Res. 31:1103-1110.
15. Renfro, B. L., and Kernkamp, M. F. 1963. Fungi isolated from black stem of alfalfa and the influence of temperature on lesion formation and disease severity. Phytopathology 53:774-777.
16. Summers, C. G., and McClellan, W. D. 1975. Interaction between Egyptian alfalfa weevil feeding and foliar disease: Impact on yield and quality in alfalfa. J. Econ. Entomol. 68:487-490.
17. Summers, C. G., and McClellan, W. D. 1975. Effect of common leaf spot on yield and quality of alfalfa in the San Joaquin valley of California. Plant Dis. Rep. 59:504-506.
18. Teng, P. S. 1980. Modelling systems of disease and yield loss in cereals. Agric. Syst. 6:131-154.
19. Thal, W. M., and Campbell, C. L. 1986. Spatial pattern analysis of disease severity data for alfalfa leaf spot caused primarily by *Leptosphaerulina briosiana*. Phytopathology 76:190-194.
20. Wilcoxson, R. D., and Bielenberg, O. 1972. Leaf disease control and yield increase in alfalfa with fungicides. Plant Dis. Rep. 56:286-289.
21. Wilcoxson, R. D., Bielenberg, O., and Bissonnette, H. L. 1973. Yield of alfalfa hay increased by control of foliar diseases. Plant Dis. Rep. 57:353-354.
22. Willis, W. G., Stuteville, D. L., and Sorensen, E. L. 1969. Effects of leaf and stem diseases on yield and quality of alfalfa forage. Crop Sci. 9:637-640.