Quantitative Relationships Between Yield and Foliar Diseases of Alfalfa

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ABSTRACT


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 was developed 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 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 Si, So, 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 < 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 x 7.6 m were used in all trials, but treatments and numbers of replications varied between trials.

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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 CO2-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 O. 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 O. 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 O. 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 O. 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 O. 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 O. 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, S0 and S0 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:

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

where \( x_i \) = the percentage of foliar disease severity at the \( i \)th evaluation, \( t_i \) = time of the \( i \)th evaluation in days from the first evaluation date, and \( n \) = 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.](image-url)
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 ≤12% for $S_1$, <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 $S_1$, $S_0$, 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 2.1 to 4.6 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 ($P < 0.05$) over all trials and within trials except for $S_1$ with $S_0$ and $S_2$ 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 $S_1$, $S_0$, $D$, or AUDPC as predictors in linear and quadratic functions were evaluated to predict actual yield in individual trials. Models with $S_1$ 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 $\times$ 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 $S_1$, $-0.68$ to $-4.15$ for $S_0$, and $-0.117$ to $-0.190$ for AUDPC. When yield was evaluated over all trials, the best models to predict percentage of maximum yield were AUDPC, $S_1$, and $S_0$. Slope coefficients for linear models to predict percentage of maximum yield in individual trials ranged from $-0.190$ to $-4.15$ for $S_1$, $-0.68$ to $-4.15$ for $S_0$, and $-0.117$ to $-0.190$ for AUDPC.

### 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

<table>
<thead>
<tr>
<th>Trial and treatments</th>
<th>Colletotrichum spp.</th>
<th>Leptosphaerulina spp.</th>
<th>Phoma medicaginis var. medicaginis</th>
<th>Stemphylium botryosum</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Raidor third cut, 1984</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$S. botryosum$ inoculation</td>
<td>20</td>
<td>27</td>
<td>88</td>
<td>98</td>
</tr>
<tr>
<td>One fungicide spray</td>
<td>7</td>
<td>35</td>
<td>62</td>
<td>48</td>
</tr>
<tr>
<td>Two fungicide sprays</td>
<td>2</td>
<td>10</td>
<td>30</td>
<td>17</td>
</tr>
<tr>
<td>Three fungicide sprays</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>13</td>
</tr>
<tr>
<td>Untreated check</td>
<td>5</td>
<td>18</td>
<td>88</td>
<td>45</td>
</tr>
<tr>
<td><strong>Vernal second cut, 1985</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$S. botryosum + Phoma$ inoculation</td>
<td>25</td>
<td>5</td>
<td>92</td>
<td>91</td>
</tr>
<tr>
<td>$Phoma$ inoculation</td>
<td>29</td>
<td>15</td>
<td>72</td>
<td>89</td>
</tr>
<tr>
<td>$S. botryosum$ inoculation</td>
<td>27</td>
<td>19</td>
<td>96</td>
<td>61</td>
</tr>
<tr>
<td>Fungicide sprays</td>
<td>0</td>
<td>0</td>
<td>7</td>
<td>3</td>
</tr>
<tr>
<td>Untreated check</td>
<td>31</td>
<td>11</td>
<td>63</td>
<td>40</td>
</tr>
</tbody>
</table>

*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.

*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 $S_i$, $S_0$, and AUDPC, respectively, did not differ. Hence, significant predictor × trial interactions were due to results from 4, 3, and 2 of 21 trials for $S_i$, $S_0$, and AUDPC, respectively.

Pooled data from 15 trials were used to develop linear models to predict percentage of maximum yield using $S_i$ 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 $S_i$ 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 $S_i$, $S_0$, 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 $S_i$ was below 13% and +1.4% when $S_0$ and AUDPC were below 15% and 221, respectively. All three models predicted percentage of maximum yield for the 205 observations from the six trials with a mean difference of zero.

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 $S_i$ and 85.8 for $S_0$, which were not different from each other. Comparing the residual mean square from the validation data set, the $S_i$ 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% $S_i$ 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 alfalfa in 1984 or 1985 used to develop the regression model.

![Fig. 2. Regression models to predict percentage of maximum yield as function of (A) foliar disease severity 1 wk before harvest ($S_i$), (B) disease severity at harvest ($S_0$), and (C) area under disease progress curve (AUDPC). Plotted data for $S_i$ 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.](image)

| TABLE 2. Regression models to predict percentage of maximum yield using foliar disease severity 1 wk before harvest ($S_i$), severity on day of harvest ($S_0$), and area under disease progress curve (AUDPC). |
|-----------------|---------|---------------|----------------|----------------|----------------|
| Independent variable | $n^*$ | Intercept | Standard error of intercept | Partial regression coefficient | Standard error of regression coefficient | $r^2$ | Standard error of dependent variable |
| $S_i$ | 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 | 452 | 100.22 | 0.724 | -0.14 | 0.006 | 0.52 | 8.04 |

*a* Number of observations used in regression model development.

*b* $\text{AUDPC} = \sum_{i=1}^{n-1} [0.5(x_i + x_{i+1})](t_{i+1} - t_i)$ where $x_i =$ percentage of foliar disease severity at $i$th evaluation, $t_i =$ time of $i$th evaluation in days from the first evaluation date, and $n =$ total number of times disease was evaluated.
Validation using methods similar to those in this study should generally occur as a complex with highly variable symptom validation. 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.

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 S1, S0, 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 S1 and S0 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. Brosious, 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

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 (S1), (B) disease severity at harvest (S0), and (C) area under disease progress curve (AUDPC). Predicted and observed percentages of maximum yield values were determined using following models: Y = 99.1 - 2.4S1; Y = 99.4 - 1.8S0; Y = 100.2 - 0.142 AUDPC and 205 observation data set not used to develop 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 X 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