Coupling a Disease Progress Model for Early Blight to a Model of Potato Growth

K. B. Johnson and P. S. Teng

Former research associate/lecturer and former professor, respectively, Department of Plant Pathology, University of Minnesota, St. Paul 55108.
Present address of first author: Dept. of Botany and Plant Pathology, Oregon State University, Corvallis 97331-2902. Present address of second author: Dept. of Plant Pathology, International Rice Research Institute, Los Banos, The Philippines.
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ABSTRACT

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A disease progress model for early blight disease, caused by *Alternaria solani*, was coupled to a dynamic potato growth model that accumulates and partitions dry matter into leaves, stems, roots, and tubers. The disease progress model was a modified logistic with terms for new infection, lesion expansion, and a variable incubation period. In the crop model, new infection and lesion expansion reduced green leaf tissue, and cohorts of leaf tissue were prematurely senesced after a maximum proportional lesion severity was attained. In field experiments, incubation period was found to depend on the age of leaf tissue, and the proportional lesion area that induced leaf senescence was dependent on the age of the crop. Observed relative lesion expansion rates (day^{-1}) were independent of leaf or crop age. Values for the infection rate parameter were determined by fitting the model to periodically sampled crop biomass and disease severity data from inoculated field plots of Russet Burbank potato. Compared to controls, both observed and simulated epidemics did not reduce tuber yield until late in the season. In simulation analyses, reductions in tuber yield were most sensitive to changes in the rate of lesion expansion followed by the time of disease onset. When early blight epidemics were initiated early in the season, the incubation period and the rate of leaf area expansion limited the rate of disease progress until near midseason. Simulated epidemics with onset times in the last quarter of the season had little effect on tuber yield.

Additional keywords: epidemiology, pest management, simulation modeling, *Solanum tuberosum*, yield loss.

The effects of plant disease epidemics on crop productivity generally have been studied in one of two ways: either by correlating reductions in yield to a direct measure of disease or pathogen intensity (29) or by imposing treatments that vary important epidemiological parameters (e.g., onset time or infection rate) and then comparing the yield differences (8,19). Both methods, although principally concerned with the fate of the crop, consider the host to be a relatively static entity. Recent reviewers (22,27) have suggested that incorporation of host growth dynamics into epidemiological investigations leads to a better understanding of

how disease affects growth and yield, as well as examination of how host factors may influence or regulate disease progress.

Crop simulation models have now been developed for many agronomic plant species (32). These models describe crop growth and partitioning of photosynthetic assimilates based on environmental inputs such as solar radiation, temperature, and water availability. Most crop models predict biomass and yield per unit area by integrating important growth, development, and competition processes at the whole plant, organ, and tissue levels of biological organization. The mechanistic basis of these models allows for dynamic feedback after stresses are imposed, and this feedback, in turn, allows for the explanation of phenomena at the population (crop) level of biological hierarchy (22,30).

Approaches to coupling disease stresses to crop models have been discussed primarily from the physiological point of view (3,22,23). These approaches vary from the relatively simple idea that disease effects on growth can be understood in terms of effects on radiation interception and radiation use efficiency (11) to more detailed handling of disease effects on specific processes (e.g., increased respiration, decreased photosynthesis, altered tissue partitioning ratios, stand reduction, and accelerated senescence) (3,23). The epidemiological considerations of combining disease dynamics with crop growth models has received much less attention. Berger and Jones (2) recently developed a disease progress model that includes a term for dynamic host growth feedback; this model also expands on the analytical disease progress models proposed by Vanderplank (31) by including terms for a variable latent/incubation period and lesion expansion. Other potentially important epidemiological considerations include asymptotes (i.e., upper limits) of disease severity, the distribution and spread of disease within canopies, the canopy environment, and the effects of host age on the following variables: the distribution of susceptible tissue; the durations of incubation, latent, and spore production periods; and the rates of infection, lesion expansion, and production of secondary inoculum (27).

The objective of this study was to initiate research to couple an epidemic model for early blight to a model of potato growth. Early blight, caused by *Alternaria solani* Sorauer, is a problem in most potato production areas of North America (5,8,9,19,28). This disease has been described as one occurring primarily on older, less productive leaves, resulting in premature senescence (20,24) and reduced leaf area duration. Frequent applications of protectant fungicides are generally required to control this disease (5,8,19,28); however, economic benefits from fungicidal control are not always attained (5). Several studies have suggested that, because potato plants are partially resistant to early blight when they are young, repeated fungicide applications should not begin until near midseason (8,19,28).

The models chosen for this research were the disease progress model developed by Berger and Jones (2) and the potato growth model developed by Johnson et al (14). This crop model has a simple structure and has been validated for growth response within several production systems and for response to defoliation (13). The primary purpose for coupling the models was to examine the interrelationships between the dynamics of early blight and its effect on potato yield. Consequently, for the present study, the direct effects of environment on early blight infection processes are not considered.

THE MODEL

Potato growth model. Using inputs of solar radiation, temperature, and soil water potential, the growth model accumulates and competitively partitions dry matter on a daily basis into leaves, stems, roots, and tubers (13,14). Growth response to temperature was modeled with the physiological P-Day function of Sands et al (25). Cumulative totals of P-Day units are termed herein as physiologic age (P_A). Details of the model's structure and its response to other disease and insect stresses have been published (12,15).

Age structure of leaf tissue was modeled by storing and aging daily additions to the crop as separate similarly aged cohorts within a boxcar train array (12,14). A cohort was senesced after its age exceeded a maximum P_A (12).

Disease progress model. The early blight disease progress model was a modified logistic equation with added functions for variable incubation period and a term for lesion expansion (2). Rate of increase of disease within a cohort of leaf tissue was calculated by:

$$dI(a)/dt = \{k_r \cdot Y \cdot [(L(a) - I(a))/L]\} + \{k_{lx} \cdot Y(a) \cdot [(L(a) - Y(a))/L(a)]\}$$

where the first line of the equation represents new infections, and the second line represents expansion of existing lesions. I is the amount of infected leaf tissue (g), Y is the amount of diseased leaf tissue beyond the incubation period and considered infectious (g), and L is the total grams of leaf tissue, healthy or otherwise. The subscript, a, indicates an individual cohort, and its absence after a variable means that the value for the variable was summed over all cohorts. Parameters k_r and k_{lx} are constants (day^{-1}) for the relative rates of new infection and lesion expansion, respectively, and must be experimentally determined. Note that the total amount of infectious leaf tissue contributed to the epidemic within any one cohort.

Incubation period was modeled using a time-varying second-order distributed delay in a manner analogous to that described by Berger and Jones for latent period (2). The time-varying delay accounted for effects of leaf tissue P_A on incubation period.

As an epidemic progressed, cohorts of leaf tissue were lost through natural attrition and disease-induced senescence. Senesced diseased tissue, however, remained in the pool of infectious tissue, Y. This was done to account for two processes: spore production by *A. solani* on dead tissues (1, 20) and survival of *A. solani* spores, which can retain infectivity for relatively long periods (up to 8 wk [21]). Infected leaf tissue associated with senesced cohorts did not continue to expand.

Simulated disease progress was initiated at a specified onset time by introducing new infections to all cohorts present in the leaf tissue array. The amount of disease introduced to a cohort was proportional to its age. For example, if the oldest cohort began with 0.5% disease, a cohort half as old received 0.25% disease. The logic of this pyramidal initialization is simply based on the length of time a cohort had to trap spores. Overall disease at onset was about half that introduced to the oldest cohort of leaf tissue.

Modeling disease effects on growth. Early blight affected modeled crop growth by reducing the amount of healthy leaf tissue in the crop through new infection and expansion of existing lesions and by causing premature senescence of cohorts of leaf tissue after the proportion of disease within a cohort exceeded a maximum severity. This approach to modeling disease effects does not directly reduce the efficiency of the remaining healthy tissues. However, in the model used here (14), as well as in another potato growth model (16), 50% of leaf dry matter is remobilized back into the pool of nonpartitioned assimilates as it is senesced. In modeling early blight, this remobilization function was modified in two ways: either by remobilizing dry matter only from senescing noninfected leaf tissue or by completely disabling it if senescence was brought on by disease rather than maturity.

MATERIALS AND METHODS

Crop biomass and disease progress experiments. Field plots of Russet Burbank potato were established in a Waukegan silt loam soil located on the University of Minnesota Rosemount Agricultural Experiment Station to collect data on early blight disease progress and changes in crop biomass over time. Planting dates were 22 May 1985 and 17 May 1986. Whole B-sized potato seed pieces were placed in rows 1 m wide and 25–30 cm apart. Cultural practices (tillage, fertilization, herbicide use, and insect control) have been described (13). Plot dimensions were six rows wide × 12.2 or 15.2 m long. Within each plot, rows 1, 4, and 6 were defined as borders. Rows 2 and 3 were used for sequential

samples of crop biomass, and row 5 was used to measure final tuber yield.

Experimental design was a randomized block with four replications. Treatments were an inoculation with *A. solani* and a fungicide-protected control. The fungicide was chlorothalonil (0.28 kg a.i./ha), applied in 610 L/ha with a drop nozzle sprayer on a 7- to 10-day schedule beginning in mid-July (crop P_A = 300) of both years.

Early blight was established by inoculating plots with a culture suspension of *A. solani* near full bloom (24 July 1985 [P_A = 305] and 19 July 1986 [P_A = 304]). Inoculum was prepared by growing two isolates of *A. solani* at room temperature on one quarter strength Difco potato-dextrose agar plus streptomycin (200 mg/L) in 10-cm petri plates for 10–12 days. After this time, the nonsporulating cultures were pureed with water in a blender for 30 sec, the isolates were mixed, and the concentration was adjusted to four plates per liter. The culture suspension was applied with a hand-held sprayer to each plot of the inoculated, nonsprayed treatment at a rate of 2.5 L per plot. Inoculations were made near dusk on days when rain showers had occurred during the afternoon.

The percent leaf area with early blight lesions was estimated in each plot at 12, 27, 42, 51, and 62 days after inoculation in 1985 and at 8, 17, 30, 45, and 53 days after inoculation in 1986. The assessments were made on three plants, each located in a different area of the plot, by first estimating the proportion of leaves showing early blight lesions and then estimating the average severity of lesions on those leaves with the aid of standard area diagrams (6). In 1986, the vertical distribution of early blight was also estimated on five individual plant stems within each plot 17, 30, and 45 days after inoculation. For each stem, the average percent lesion area was recorded for groups of three consecutive leaves descending from the top of a stem.

Plants used to determine crop biomass were harvested every 2 wk throughout the season. Final harvests were made on 24 September in 1985 and 16 September in 1986. On each harvest date, four or five plants (hills) were sampled from each plot and separated into green leaf tissue, stems, and tubers. On the last two sampling dates in 1986, tubers were harvested from a second set of plants to improve the precision of the yield estimate. When plants were processed, roots and dead and chlorotic leaves were discarded. Fresh weights of tubers were recorded immediately and dry weights of the other tissues were recorded after desiccation at 60 C for 1–2 wk. Leaf dry weight was transformed to leaf area index (LAI) and tuber dry weight was estimated from the fresh weight values with procedures previously described (13). The estimates of percent lesion area over time were used to adjust the green leaf tissue biomass to the amount that was actually healthy.

Epidemiological components. Observations on the relative rate of lesion expansion and on the proportional lesion severity of early blight that induced premature leaf senescence were made in the crop biomass field plots in 1986. In 1987, these same two components, as well as incubation period, were monitored within small plots planted on three different dates (discussed below). Several observations were made on each component during a season to evaluate potential influences of individual leaf and cumulative crop P_A. The P_A for individual leaves was determined within each planting by weekly tagging the day's date on the top leaf of 15–20 plants and, at the same time, noting the position on the stem of previously tagged leaves.

Relative rates of lesion expansion (day^{-1}) were determined by measuring the change in the area of lesions individually marked with adhesive stickers over intervals of 5–7 days. Areas were approximated by measuring the diameter of a lesion in two perpendicular directions and using the average to compute a circular area. A relative rate of expansion was calculated for each lesion by taking the natural log of the ratio of final lesion area to initial lesion area and then dividing this value by the number of days in the interval. During 1986, 27–38 lesions were measured over 4- to 9-day intervals in August and early September (crop P_A range from 425 to 727). Similarly, in 1987, the changes in diameter of 15–17 lesions were recorded over an interval from 27 August to 1 September in both the intermediate and late plantings of the small plots (crop P_As of 500 and 670, respectively).

Estimates of proportional lesion severities that induced premature leaf senescence were made by periodically sampling the oldest leaves remaining on stems in the inoculated treatment, but only after disease-induced defoliation was visually apparent within the plots when compared to the controls. Over both years, 8–13 leaves were sampled at crop P_As of 480, 560, 569, 650, 667, 734, and 750. The total area of sampled leaves was measured with a leaf area meter (Licor, Inc., Lincoln, NE). Lesion area was similarly measured after tracing and filling lesion outlines on clear transparent film (3M, Inc., St. Paul, MN) with black felt-tip pens. As controls, cardboard squares and blackened transparencies of the same size were analyzed with the area meter to assure the accuracy of the technique. Proportions were attained by dividing lesion area into leaf area (Fig. 1).

Six 3- × 3-m plots were used to study incubation period in 1987. Three plots were planted on each of the following dates: 24 April, 12 May, and 6 June. These plots were given the same cultural management as described previously. Sets comprising one plot from each planting date were inoculated with *A. solani* on 21 July and 10 August. Inoculations were done at dusk on four plants in each plot using pureed cultures as described above. Before inoculation, the plots were irrigated to assure high humidity; after inoculation, the plants were covered with plastic bags for about 12 hr. The position of each leaf on the stem and the number of visible lesions (about 0.5 mm^2) per leaf were recorded on two stems of each plant 3, 6, 9, 12, and 15 days after inoculation. The incubation period of the disease on a particular leaf was estimated by interpolation of the lesion numbers to the day after inoculation when 50% of the total number of lesions appeared.

Environmental monitoring. Minimum and maximum temperature, solar radiation, and rainfall were monitored and recorded daily with a portable weather station equipped with a CR-21 micrologger (Campbell Scientific, Logan, UT) located in or near the experimental sites. Soil moisture content was measured gravimetrically twice a week in each replication at depths of 0–15 and 16–30 cm below the hill (13). Daily increments in P_A were calculated from the temperature data.

Data analysis. The effects of crop and leaf P_A on the epidemiologic components were analyzed with regression analysis. For

Fig. 1. Depiction of a potato leaf showing lesions colonized by *Alternaria solani*. The lesion area in this figure is 24%, a degree of lesion coverage at which disease-induced senescence of leaves commonly occurred.

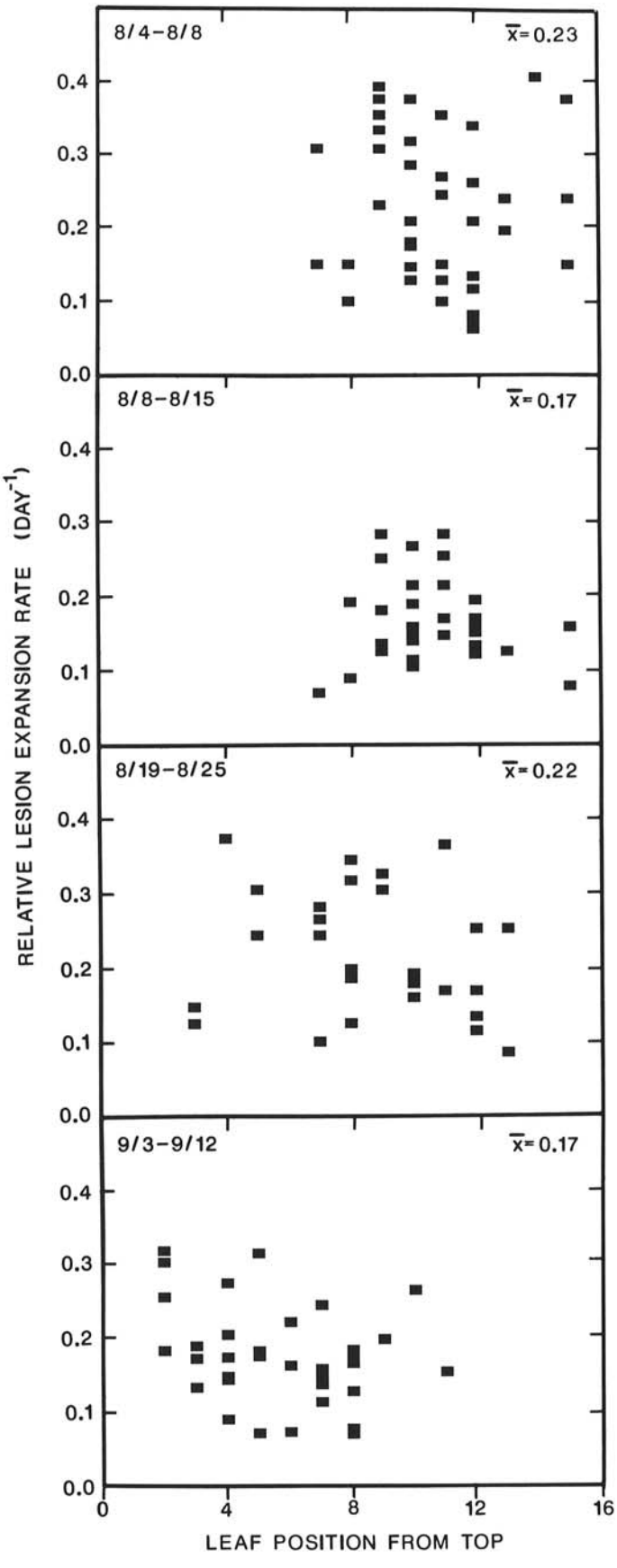

Fig. 2. Relative rate of early blight lesion expansion by position of a leaf from the top of Russet Burbank potato plants during four intervals in the 1986 season. Relative rates were obtained by dividing the change in the natural logarithm of lesion area into the number of days in the calendar interval given in each graph.

a given crop P_A, ages of individual leaves were estimated from the mean P_A of leaves holding the same position on tagged plants for that planting date. Multiple regression models with crop P_A and leaf P_A as independent variables were constructed to evaluate age effects on the relative lesion expansion rate and incubation period data. The data collected on the proportional lesion severity that induced leaf senescence was only regressed on crop P_A. Results of these analyses provided either parameter values or age-dependent functional equations to use within the disease progress model.

Estimates for the infection rate parameter, k_r (day^{-1}), were determined by iteratively executing the plant growth and disease progress models with measured 1985 and 1986 environmental data (13), the estimates for the other epidemiological components discussed above, a crop P_A at disease onset of 300, an initial disease proportion of 0.0025 (arbitrary), and validated partitioning parameters previously determined for the Russet Burbank cultivar (13,14). Selection of a value for k_r was based on comparison of simulated disease progress with the epidemics observed in the field. Criteria for selection included correspondence of simulated

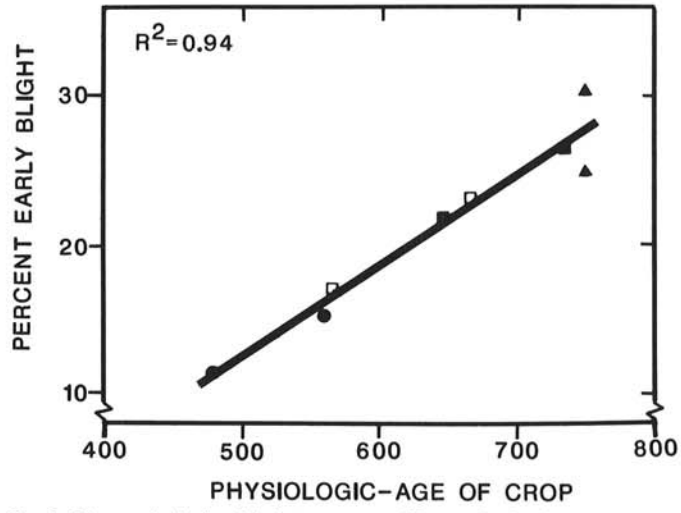

Fig. 3. Observed relationship between age of Russet Burbank potato crops and proportional severity of lower leaves infected with early blight just before disease-induced senescence occurred. Data are from one crop grown in 1985 (open square) and from crops planted on different dates in 1986 (solid triangle = 24 April, solid square = 12 May, and solid circle = 6 June).

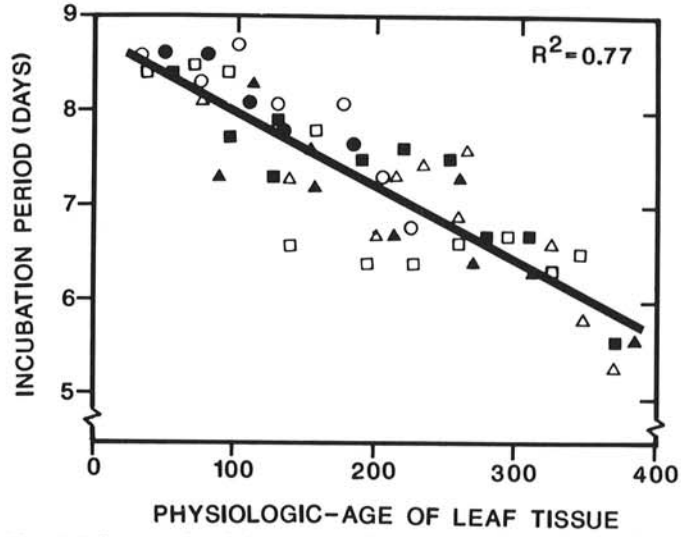

Fig. 4. Influence of leaf-tissue age on incubation period of early blight disease. Data are from three crops of Russet Burbank potato planted on different dates (triangle = 24 April, square = 12 May, and circle = 6 June) and inoculated two times during the 1987 season (open marker = 21 July, solid marker = 10 August).

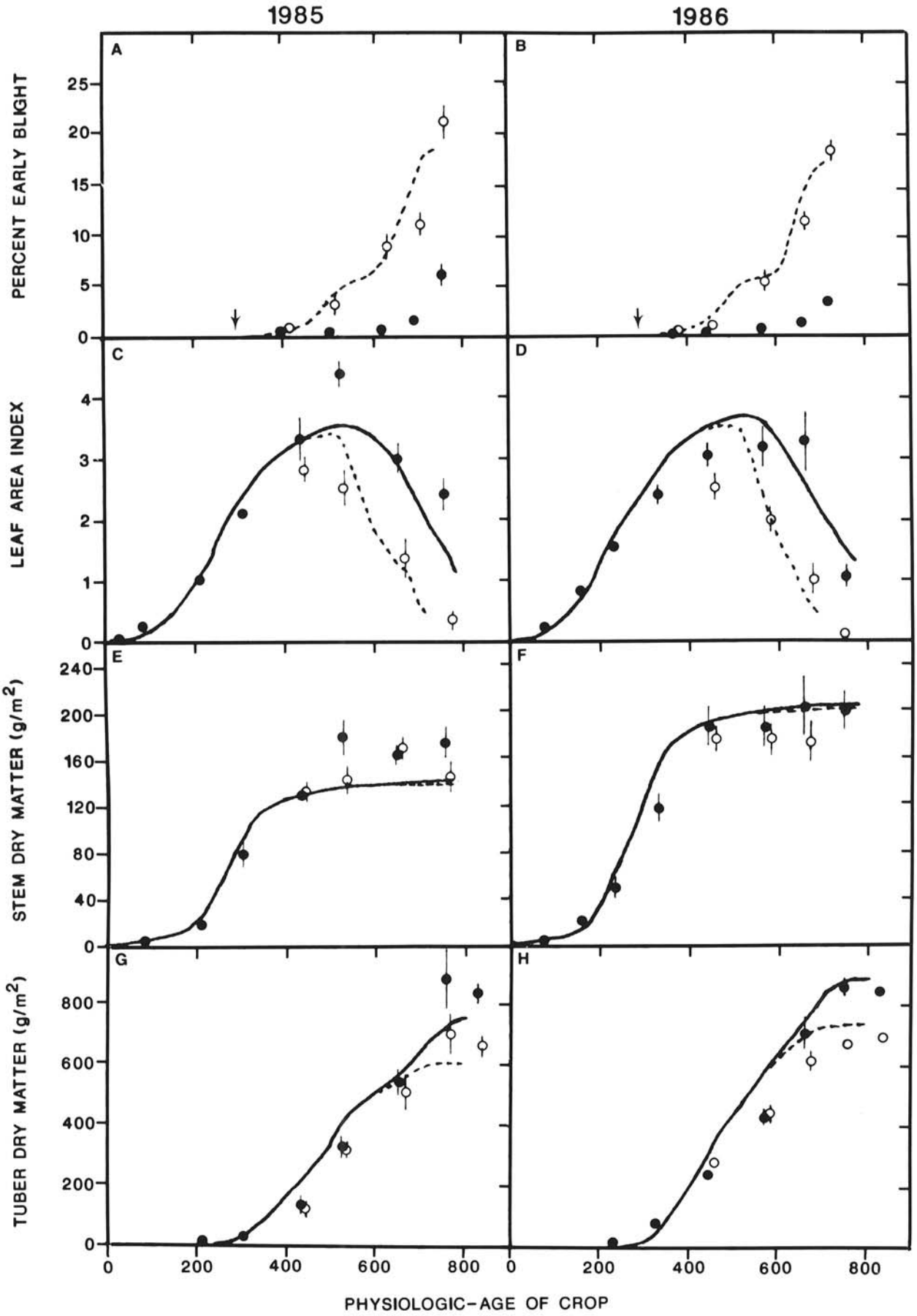

Fig. 5. Simulated (lines) and observed (points) severity of early blight (**A, B**), leaf area index (**C, D**), stem dry weight (**E, F**), and tuber dry weight (**G, H**) from infested (open circles and dashed lines) and control (solid circles and lines) crops of Russet Burbank potatoes grown in 1985 and 1986. Inoculations are indicated by the arrows in **A** and **B**.

disease percentages with observed values, correlation of the modeled difference in percent disease between early blight infected and nondiseased control crops to the differences observed in the field, and evaluation of temporal development and attrition of simulated and observed LAI in graphical arrays. For the 1986 data, after the value was estimated for k_r through this process, the vertical distribution of early blight within the modeled age-structured array of leaf tissue was compared to the observed vertical distribution of percent disease in the field. Simulation runs were stopped when crop P_A exceeded 780 or when LAI had declined to less than 0.50.

Two sample t tests ($P = 0.05$) were used to evaluate differences between the fungicide-sprayed control and the inoculated blight treatments. Standard errors were computed for estimated parameters and regression coefficients.

Simulation analyses. Sensitivity of simulated yield to changes in the values of the epidemiological components was evaluated by executing the model with each component changed by plus or minus 20%, holding other components at their initial settings. For epidemiological components that were found to be functions of crop or leaf age, this resulted in an overall 20% increase or decrease across all ages. Components evaluated were: initial proportion of disease, onset P_A, k_r, k_{lx}, incubation period, and proportional severity that induced senescence.

Further simulation analyses were done to evaluate the effect of disease onset time and rate of new infection on epidemic development and loss of LAI and tuber yield.

First, four epidemics were begun at cumulative crop P_As of 100, 200, 300, and 400. Disease progress and the temporal development and attrition of leaf area were then compared in graphical arrays. Because the amount of leaf area present in the crop varied with each onset, epidemics were either begun with the same initial proportion of disease (0.25%) or with the same amount of infected leaf area (0.20 g/m^2 [about 50 cm^2/m^2]). For the latter, 0.20 g/m^2 amounted to initial disease percentages of 2.5, 0.5, 0.25, and 0.20 for the crop P_As of 100, 200, 300, and 400, respectively. The value used for k_r was 0.015/day.

In the second analysis, epidemics were initiated at cumulative crop P_As of 100, 200, 300, 400, 500, and 600 with an infection rate value of either $k_r = 0.015$ or 0.000/day and an initial proportion of disease of either 0.0025 or 0.03. Setting k_r at 0.000/day after initiating an epidemic allowed for the evaluation of yield-loss sensitivity based on only the lesion expansion component of the disease progress model. Percent yield of each diseased crop was computed by dividing final tuber yield by that of the nondiseased control.

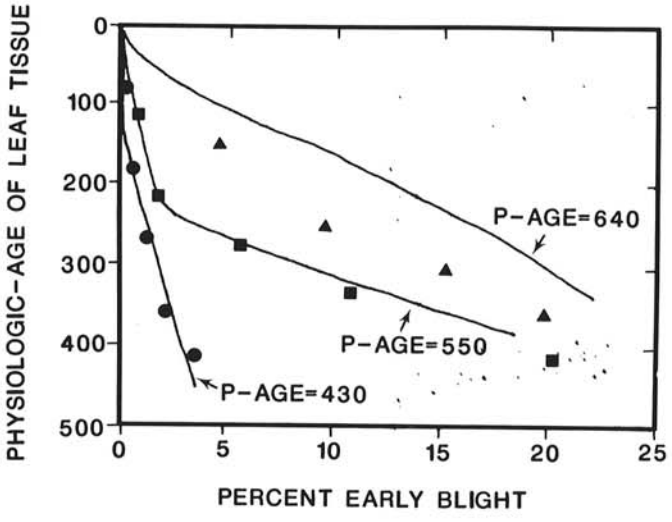

Fig. 6. Observed (points) and simulated (lines) vertical distributions of early blight disease at crop physiologic ages (P_A) of 430 (circles), 550 (squares), and 640 (triangles) on Russet Burbank potato in 1986.

RESULTS

Epidemiological components. Relative rate of lesion expansion averaged 0.198 ± 0.016/day over four intervals in August and September 1986 (Fig. 2). In 1987, the relative rate of lesion expansion was slightly slower, averaging 0.187 ± 0.023/day. Regressions of relative lesion expansion rate on either leaf or crop P_A were not significant ($P > 0.05$). Based on these results, a value of $k_{lx} = 0.20$/day was used in the model simulations discussed below.

In both years, defoliation induced by early blight was visually apparent in the crop biomass plots about 20–25 days after inoculation (crop P_A = 450–500). Within these plots, the proportional lesion area on older leaves about to senesce was significantly influenced ($P < 0.01$) by the age of the crop (Fig. 3). Values for the regression coefficient, b, and the y intercept were 0.061 ± 0.006 and −18.3 ± 1.6, respectively. Mean proportional lesion area varied by sampling date from 11.3% (crop $P_A = 480$) to 30.3% (crop $P_A = 750$).

Regression of incubation period on the P_A of individual leaves was highly significant ($P < 0.01$) (Fig. 4), but crop age did not

TABLE 1. Sensitivity analysis[a] of factors governing the increase of early blight infected leaf tissue within the potato growth simulation model

Factor	Initial value	20% change (+ or −)	Resulting yield loss compared to the healthy control[b] (%)	Percent change in yield loss compared to the diseased control[c]
Initial percent disease at onset	0.25%	−	16	−16
		+	20	5
Onset physiologic age (P_A)	300	−	25	32
		+	13	−32
k_r	0.015	−	17	−11
		+	20	5
k_{lx}	0.20	−	10	−47
		+	26	37
Incubation period	$IP = -0.0078 \times P_A + 8.8$	−	21	10
		+	16	−16
Proportional disease severity that induced leaf senescence	$EB_{max} = 0.061 \times$ crop $P_A - 18.3$	−	21	10
		+	17	−11

[a]Each factor tested was increased or decreased by 20% holding the other factors constant. Environmental data from 1986 were used in the simulations.
[b]Yield loss in the diseased control compared to the healthy control was 19%.
[c]Amount in the preceding column, divided by 19%, from which 100.0 was subtracted.

significantly ($P > 0.05$) account for additional variation. On older leaves ($P_A > 350$), lesions began to appear 3 days after inoculation, with the mean number of new lesions appearing about 5.5 days after inoculation. Mean incubation period on very young leaves ($P_A < 100$) was 8.5 days. The functional relationship in Figure 4 ($b = -0.0078 \pm 0.0006$; y intercept $= 8.8 \pm 0.4$) determined the mean incubation period of new infections entered into the distributed delay of the disease progress model.

Disease progress and host response. In plots inoculated with *A. solani*, early blight lesions were detected 12 ($P_A = 411$) and 17 ($P_A = 442$) days after inoculation in 1985 and 1986, respectively (Fig. 5A and B). In both seasons, percent lesion area remained below 5% at crop P_As less than 550 and then rapidly increased from about 5 to 20% over the crop P_A interval from 600 to 750. In fungicide-treated control plots, the leaf area with lesions of early blight was always less than 1.5% except for very late in the season ($P_A > 700$) (Fig. 5A and B).

Compared to the fungicide-treated controls, observed LAI was significantly reduced ($P < 0.05$) in inoculated plots by mid-August of both years (crop $P_A = 550$–600)(Fig. 5C and D). As the potato crops approached maturity, LAI declined more rapidly in the inoculated plots than in the fungicide-protected plots. Early blight did not significantly affect observed stem dry weight on any sampling date in either year (Fig. 5E and F). Tuber dry weights were significantly reduced ($P < 0.05$) in the inoculated plots but not until the crop was very close to maturity (Fig. 5G and H). Loss of tuber dry matter at maturity averaged 159 ± 26 g/m^2 (21%) in 1985 and 147 ± 29 g/m^2 (18%) in 1986.

Values for k_r that best simulated disease progress and LAI response were 0.014 and 0.015/day for 1985 and 1986, respectively. Simulated disease progress was similar to field observations in that disease development immediately after onset was initially slow and then increased rapidly after the cumulative P_A of 500 (Fig. 5A and B). Loss of leaf tissue to disease-induced defoliation made the apparent rate of disease progress variable over time. Correlations of the differences in percent lesion area observed between treatments on each date in the field with the differences between the early blight-infected crops and the nondiseased control crops obtained with the model were 0.91 and 0.96 for 1985 and 1986, respectively. Comparison of the simulated vertical distributions of percent lesion area within the model's age-structured array of leaf tissue cohorts to the observed distributions from 1986 showed similar patterns of disease development (Fig. 6).

Modeled LAI declined more rapidly over time in response to early blight than that of the simulated control (Fig. 5C and D). This response resembled the observations made on field-grown plants with the exception that modeled losses were slightly delayed (about 75 P_A units). Simulated early blight epidemics only slightly affected stem dry weights very late in the season (Fig. 5E and F).

Effects of simulated early blight on tuber dry accumulation most closely resembled the observed pattern of tuber dry matter loss if the leaf dry matter remobilization function (discussed above) was completely disabled when senescence was brought on by disease (Fig. 5G and H). Reduction of tuber dry weight caused by early blight began to occur at a cumulative crop P_A of about 600, and final tuber yields were reduced 147 g/m^2 (20%) and 170 g/m^2 (19%) in simulations using the environmental data of 1985 and 1986, respectively. When the leaf dry matter remobilization function was not disabled, yield reductions were unrealistically delayed and diminished in magnitude to a yield-loss range of 14–16%.

Simulation experiments. In the sensitivity analyses, final tuber yield was most sensitive to changes of plus or minus 20% in

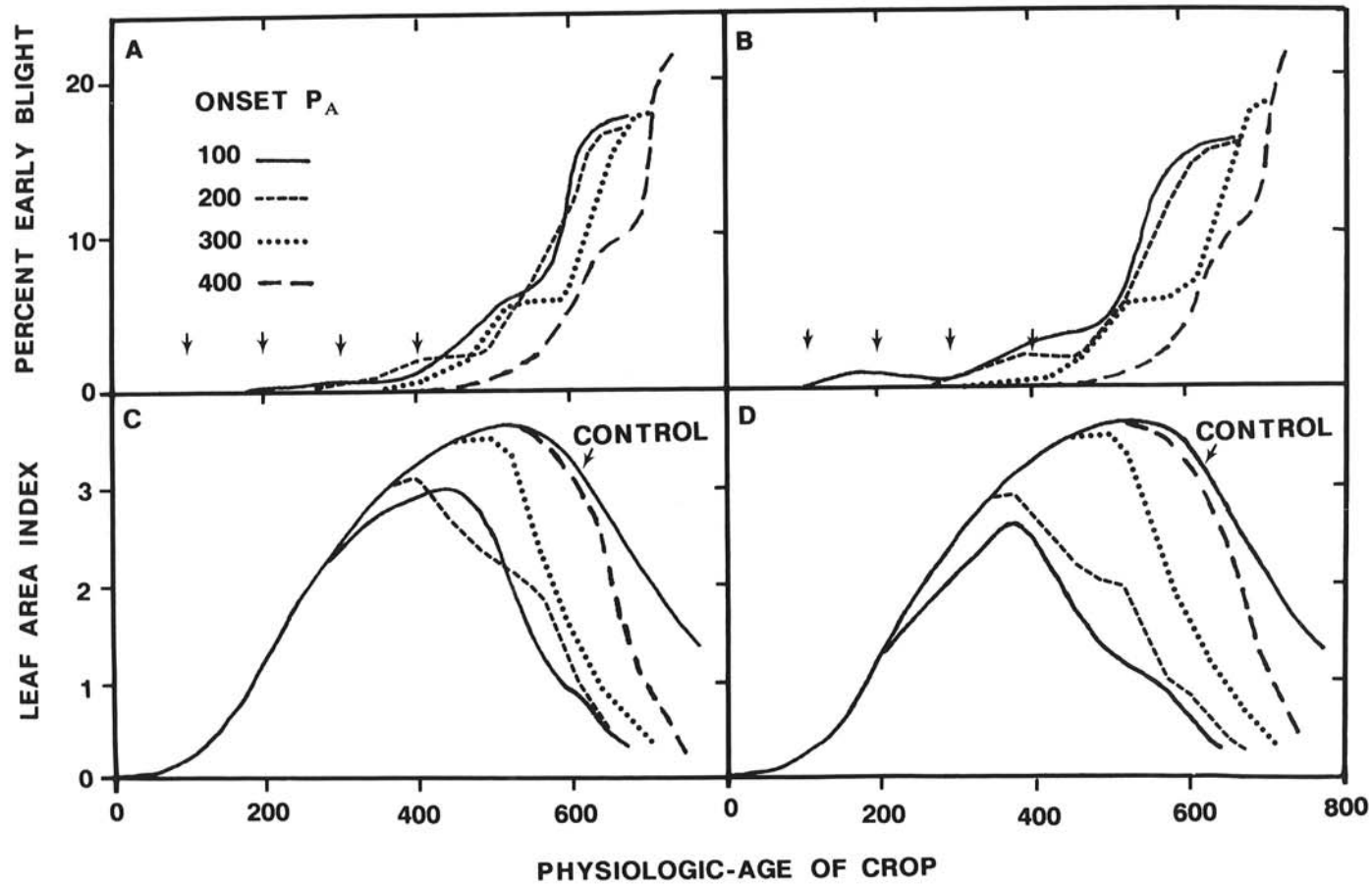

Fig. 7. Simulated early blight disease progress and leaf area index curves resulting from epidemics initiated at the cumulative crop P_A's of 100, 200, 300, and 400 on Russet Burbank potato. **A, C**, Epidemics initiated with a constant proportion of disease (2.5%) relative to the amount of leaf tissue present at each onset P_A. **B, D**, Epidemics initiated with a constant amount of diseased leaf tissue (0.20 g/m^2 [about 50 cm^2/m^2]) at each onset P_A.

the rate of lesion expansion (k_{lx}), followed by the crop P_A at disease onset (Table 1). Tuber yield was relatively insensitive to changes in the initial proportion of disease (k_r), the incubation period, and the proportion of disease that induced leaf senescence.

Simulated epidemics that were begun at different crop P_As showed similarity in that, regardless of the amount of infected leaf tissue used to initiate disease progress, the proportion of nondefoliated leaf area affected by early blight remained below 5% until a cumulative crop P_A of about 500, after which time diseased proportions increased rapidly (Fig. 7A and B). Leaf area index curves for the various disease onset conditions showed greater differences between epidemics than did the curves for percent leaf area infected with disease (Fig. 7A–D). Epidemics begun with a constant proportion of disease had smaller differences between the disease progress and LAI curves than did epidemics initiated with a constant amount of infected leaf tissue.

Effect of disease onset P_A on final yield response was differentially influenced by the values chosen for the initial proportion of disease and the infection rate (Fig. 8). When the value $k_r = 0.015$ was used, early disease onset ($P_A \leq 200$) most greatly reduced yield. In contrast, when secondary infection was inhibited and only lesion expansion was allowed to proceed ($k_r = 0.00$), yield reductions were greatest for epidemics initiated at $P_A = 300$ when the initial percentage of disease was 0.25% and at $P_A = 400$ when the initial percentage of disease was 3.0%.

DISCUSSION

Temporal responses of simulated disease progress (Fig. 5A and B), leaf area index (Fig. 5C and D), and the vertical distribution of disease (Fig. 6) each indicate that the two models used in this study reasonably described the dynamic interaction between early blight disease progress and potato crop growth. In the field, reductions of tuber yield caused by early blight were 18 and 21% in two crops with uncontrolled epidemics. Simulation of these crops and the epidemics resulted in yield losses of 19 and 20%, demonstrating that the approach taken to account for early blight effects on net dry matter accumulation was realistic. Modeling this disease as one that primarily affects leaf area duration and defoliation, with some effect on efficiency of carbohydrate production within diseased leaves, also corresponds well with studies done with ^{14}C labeled assimilates in tomatoes infected with early blight (4).

The finding that the age of host tissue affects the incubation period of early blight concurs with the work of Pelletier and Fry (18), in which this parameter was studied quantitatively with respect to host resistance. They also demonstrated a positive correlation between leaf age and absolute rates of lesion expansion, in contrast to the observations made here, in which the relative rate of lesion expansion was unaffected by the position of the leaf in the canopy (Fig. 2). One explanation for the difference is that lesion size is positively correlated with leaf age ($r = 0.37$ in this study), and large lesions, given a constant relative rate, expand more rapidly than small lesions when measured on an absolute scale. Pelletier and Fry (18) also found that production of *A. solani* spores was linearly correlated with visible lesion area. This result supports the linear form of the equation ($k_r \cdot Y$) that determines new infections within the disease progress model.

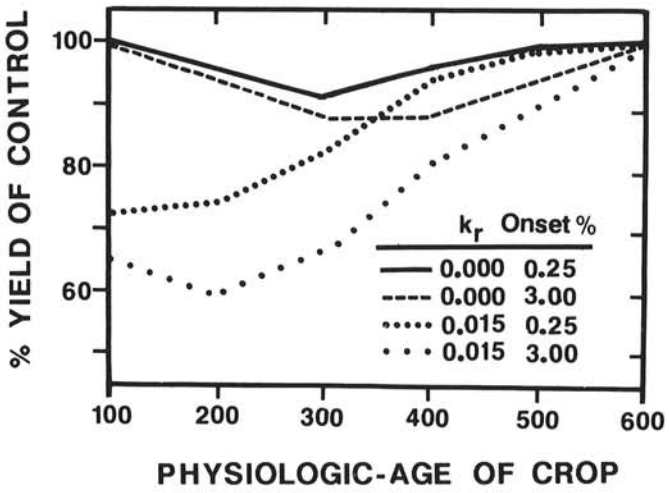

Fig. 8. Effect of crop age at epidemic onset on simulated final tuber yield of Russet Burbank potato. Two infection rate conditions were evaluated: epidemics initiated and then allowed to proceed with an infection rate parameter value of $k_r = 0.015$/day and a lesion expansion rate of $k_{lx} = 0.20$ and epidemics that were initiated but in which only lesion expansion was allowed to proceed ($k_r = 0.000$/day). Two different percentages of disease were used to initiate epidemics, 0.25% and 3.0%.

The dependence of the proportional lesion area that induced leaf senescence on cumulative crop P_A (Fig. 3) was probably related to changes in the physiological condition of leaves that were defoliated at different stages of the epidemic. The lowest proportions of disease that induced senescence occurred near midseason, corresponding to the period of maximum LAI. Leaves defoliated by early blight at this stage of growth were typically low in the canopy and very heavily shaded. Rowell (24) found that low light intensities reduced host resistance and hastened senescence of tomato leaves infected with early blight. After midseason, the relative tolerance of leaves to early blight increased linearly (Fig. 3) as LAI declined linearly (Fig. 5C and D) over time; lower leaves defoliated by early blight late in the season were generally exposed to direct sunlight. A second factor that may have influenced how much disease individual leaves could tolerate before senescing was that the average age of leaves defoliated by early blight shifted slightly as the epidemic progressed. Primarily, older leaves nearing their maximum P_A were attacked early in the epidemic; however, as the disease progressed, a greater number of younger leaves were infected and these leaves may have withstood a higher proportional lesion area before senescence was induced.

In the sensitivity analysis, proportional changes in some components of an epidemic influenced the amount of disease and corresponding yield loss more than others. Lesion expansion was the most sensitive variable, indicating that, although new infections must occur, most of the increase of disease and the damage to the crop was through lesions that already existed. As discussed below, the present structure of the disease progress model may overstate the importance of lesion expansion by not accounting for incidence/severity relationships among similarly aged potato leaflets. Nonetheless, the magnitude of tuber yield losses owing to relatively smaller changes in lesion expansion indicates that this variable may be particularly useful as a selection criteria when breeding for resistance against this disease.

Knowledge that young potato plants are partially resistant to early blight has been used as the justification for at least three different methods of timing the first fungicide application that a crop receives (7,8,19,28). The data in Figure 4, which illustrate an effect of leaf age on incubation period, supports the observation that young plants are indeed more resistant, and Figure 7 demonstrates that early blight epidemics proceed very slowly in young crops. However, the slow rate of disease progress at early stages of crop growth was also affected by a differential rate of new leaf production at different stages of growth. Young plants (crop P_A range 170–340) inoculated with *A. solani* produced seven to eight new leaves by 9 days after inoculation, whereas older plants (crop P_A 570–700) only produced one to two. Observed incubation periods on leaves produced near midseason were not significantly different from similarly aged leaves produced at an earlier stage of growth (Fig. 4); however, simulated epidemics with an onset $P_A = 100$ actually had negative rates of disease progress for a period of time coinciding with a period of rapid expansion of LAI (Fig. 7).

Results of simulations in which epidemics were initiated at different cumulative crop P_As but only lesion expansion was allowed to progress also shows the importance of a declining rate of new foliage production to a crop's sensitivity to damage. Foliage

produced at midseason contributes more photosynthate to developing tubers and to extending leaf area duration because it is not as rapidly replaced. The modeled period of greatest yield-loss sensitivity to early blight without new infection fell between crop P_As 200 and 500 (Fig. 8). The lower limit of this range corresponds to one previous recommendation (28) that fungicide applications to control early blight should not begin until 6–7 wk after planting (approximate crop P_A = 200–250 under Minnesota conditions) but occurred earlier than another recommendation that fungicide application begin at crop P_As of 300–350 (8–10 wk after planting) (19). The upper limit of yield-loss sensitivity (P_A = 600) supports the suggestion (28) that fungicide spraying could be stopped 3 wk before vine kill in production areas where tuber infection by *A. solani* is not a problem.

Many growers in Minnesota produce potatoes for fresh market. These growers often kill vines and harvest their crop before natural maturity to obtain a better price. The results of this study show that, even when early blight epidemics began during the period of high yield-loss potential, actual reductions in tuber yield did not occur until LAI had declined substantially late in the season (Fig. 5G and H). Based on yield considerations alone, growers who artificially defoliate and harvest their crop before maturity may not receive any benefit from control of this disease (5).

Epidemiologically, the model proposed by Berger and Jones (2) served as a reasonable first approximation to the complex process of coupling a model of disease progress to a model describing crop growth. Two improvements could be made to the disease progress model, however, to more realistically describe the crop and disease interaction. The first concerns whether the infection rate parameter, k_r, would be more appropriately modeled as a variable rather than as a constant. Pelletier and Fry (17) reported that receptivity of potato foliage increases as a crop grows older. In contrast, offsetting this observation is a theoretical concept that a variable like k_r, which is defined similarly to Vanderplank's (31) basic infection rate (R_c), declines with increase of infection (10,31). A second concern is the indeterminate nature of the lesion expansion rate parameter, k_{lx}, and its effect on realistically modeling crop productivity losses. In an actual potato crop, leaves and leaflets are discrete entities, which places limits on the amount of leaf tissue that can be destroyed by any one expanding lesion. Within the model, however, lesion expansion can eliminate a whole age-class after initial infection. The importance of this problem is probably greatest early in an epidemic at low severity when the proportional incidence of disease on leaflets is significantly less than unity. A solution would be to subdivide the array of leaf tissue into discrete units within age-classes and then use incidence-severity relationships (26) to selectively introduce new infection into these units. This modification might clear up inconsistencies between observed and modeled results such as the initial delay of defoliation in the simulated epidemics as compared to the actual epidemics (Fig. 5C and D) and the significant increase in yield-loss sensitivity to early blight occurring at an earlier crop P_A than was shown in other published observations (8,19).

Coupled models of crop growth and epidemic progress should prove to be useful tools for both theoretical and practical challenges facing crop protection specialists. One of these challenges is to obtain a better understanding of how host growth regulates epidemic progress. An example is the disease progress curves in Figure 7 that converge after being initiated at widely spaced intervals. In this regard, two important questions need further research. To what degree does the rate of leaf area expansion and attrition influence this convergence? Do important interactions occur between the rate of new infection and the rate of production and duration of crop foliage? On the practical side, the same converging epidemics in Figure 7 give strength to the idea that there is a strategic interval in crop development where management practices that interrupt infection cycles are most needed to maximize productivity. Because many crops and diseases have developmental patterns similar to those of early blight of potatoes, using the coupled models to further develop the theoretical basis for this idea of strategic need would probably provide benefits that extend to a wide range of pathosystems.

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