Development and Evaluation of a General Model for Yield Loss Assessment in Potatoes

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ABSTRACT

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A general model for yield loss assessment in potatoes was developed and parameterized for early blight and late blight. Disease-induced losses for any given crop are predicted relative to a reference crop. Loss is a function of the difference in the relative area under the disease progress curves (RAUDPC) among these crops multiplied by the effect of disease on bulking rate. RAUDPC calculation is based on integrating disease severity over the period of yield accumulation and weighing each chronological day by its relative contribution to host growth. Seventy-one epidemics of early blight or late blight that developed in Freeville, NY, where the model's parameters were estimated, and 53 epidemics from other growing areas or from epidemics having both pathogens simultaneously in Freeville, NY, were used as independent data sets for evaluating the predictions of the model. Differences between predicted and observed losses ranged from −11.2 to 8.1% and were less than 5% in 80% of the cases.

Additional keywords: *Alternaria solani*, *Phytophthora infestans*.

Potato early blight and late blight, caused by *Alternaria solani* (Sorauer) and *Phytophthora infestans* (Mont.) de Bary, respectively, are common in most areas of potato (*Solanum tuberosum* L.) production in the world (3). They are the main causes of yield loss induced by pathogens in this crop if left uncontrolled (16). These diseases may suppress yields dramatically to as little as 70–80% (early blight) or 40–50% (late blight) of the attainable yield (1,2,9,18). In the United States, despite the regular application of fungicides, these diseases have been estimated to cause annual yield losses of 1% (early blight) and 4% (late blight) (5).

Several different approaches have been taken for evaluating yield losses in potatoes. The purpose of most methods has been to find the relationship between disease severity and percent yield reduction. The critical-stage method was developed by Large in England (10). Loss was found to be related to the date at which 75% of the crop green foliage had been destroyed by blight. The earlier this happened, the greater was the loss (10). The critical-injury-threshold model, which was developed by Olofsson in Sweden (15), is an analogous method: Yield loss was inversely proportional to the duration of time the crop was free of disease (10). Multiple regression analyses were used to develop a multiple-point model in which changes in disease progress were used to predict yield losses due to late blight (7) or early blight (25). The multiple-point method considered the incremental contribution of disease to yield loss. The method for estimating late blight losses (7) was modified further to a general form (11) which expresses the predicted percent yield loss as the sum over time of disease increments, weighted by the weekly progress of the epidemic (11). Loss also has been suggested to be proportional to the integral of disease severity over the duration of the epidemic (26). Thus, the area under the disease progress curve (AUDPC) could be used as a predictor of yield reduction. Using a different approach to the above methods, Rotem et al (19) quantified the relationship between the remaining green haulm area and yield. However, variation in meteorological and other undefined factors among seasons resulted in inconsistency in the regression slopes describing the fit between green haulm area and yield in different experiments (19).

Because all of the above methods related yield loss (or yield) to disease (or green haulm area) by means of statistical analysis in specific locations, their implementation in different locations produced inaccurate loss estimations. As an example, the critical-stage method, as developed in England (10), and the critical-injury-threshold method, as developed in Sweden (15), were inappropriate for Canadian conditions (6,7). Both the critical-stage and the multiple-point methods (developed in Canada [7]) gave inconsistent results in Israel (19). Another limitation of these methods is that they concentrate on assessing losses induced by a single pathogen. Thus, they may be inappropriate if many pathogens are active simultaneously.

The major objectives of the work represented here were to develop a general model for yield loss prediction in potatoes and to evaluate the predictions of the model with independent sets of data using potato early blight and potato late blight as experimental systems. In this study, as in all the others mentioned above, no attempt was made to predict yield losses due to tuber infections; only loss of harvested tuber weight was considered.

CONSTRUCTION OF THE MODEL

A conceptual model describing the bulking of potatoes was proposed by Sands et al (22). The temperature dependence of yield accumulation over time was determined by using physiological time (PDAYS) instead of chronological time. According to this model, yield at the end of the season (Y [t/ha]) is defined as a function of maximal bulking rate (B [t/ha/PDAY]) and the length of time for tuber bulking. This period is defined as the duration of the time passed from the date of tuber growth initiation (P_{tg} [PDAYS]) to the date of cessation of bulking (P_{cb} [PDAYS]):

$$Y = (2B/3)(P_{cb} - P_{tg}) \quad (1)$$

Assume that Y, B, P_{tg}, and P_{cb} correspond to a reference (or uninfected) crop and that Y', B', P'_{tg}, and P'_{cb} correspond to

a diseased crop. Yield in diseased crop relative to a reference crop would be as follows:

$$Y'/Y = ((2B'/3)(P'_{cb} - P'_{tg}))/((2B/3)(P_{cb} - P_{tg})) \quad (2)$$

Maximal bulking rate could be affected by disease; thus B' may be smaller than B. The magnitude of the reduction is related to disease intensity and is defined as a function of the difference between the disease intensity of the diseased crop (A') and that of the reference crop (A):

$$B - B' = \alpha(A' - A). \quad (3)$$

The coefficient α represents the reduction in maximal bulking rate (B) induced by an increase of one unit in disease intensity differential. Disease may cause bulking to cease sooner; therefore, $P'_{cb} < P_{cb}$. In a diseased crop, the date of cessation of bulking is a function of disease intensity. Therefore, we assumed that the date of bulking cessation in a diseased crop (P'_{cb}) is when disease reaches *dmax* ($P_{dmax} = P'_{cb}$). Rearranging equations 2 and 3 and expressing yield losses as the percent reduction in yield relative to a reference crop results in a general model for predicting yield losses (YL[%]) in potatoes:

$$YL(\%) = 100 - (1 - \alpha(A' - A)/B))((P_{dmax} - P'_{tg})/(P_{cb} - P_{tg}))100. \quad (4)$$

Yield losses of a particular crop relative to the attainable yield may be estimated by assigning disease intensity of the reference crop (A) to 0.0.

MATERIALS AND METHODS

Quantifying the effect of early blight and late blight on yield accumulation. Parameters of the model were estimated using a series of treatments in which the effects of early blight and late blight on yield accumulation were investigated. The experiments were conducted in 1988 at the Homer C. Thompson Research Farm at Freeville, NY. Certified potato seed pieces were planted mechanically on 12 May (cultivar Norchip) or 17 May (cultivar Katahdin). Norchip is an early cultivar susceptible to early blight. Katahdin is a mid-late cultivar moderately susceptible to late blight. Seed pieces consisted of small whole tubers or pieces of tubers, each weighing about 50 g. Four-row plots, 0.9 m between rows and 3 m long, were planted at about 23-cm spacing within a row. Plots were separated from each other in all directions by a fallow area of about 4 m. Experiments were planted according to a randomized complete block design with three replicates per treatment. Fertilizer (175 kg N, 175 kg P, and 175 kg K/ha) was applied at planting. Herbicide (Linuron 50WP, Du Pont Chemicals Corp., Wilmington, DE) was applied at 1.7 kg a.i./ha after planting but before plant emergence. Insecticides were applied in each experiment every 10–14 days, starting on 6 June. Plants were hilled during the last week of June.

Some of the late blight experimental plots were inoculated by applying 1 drop (approximately 0.05 ml/drop) of a mixture of isolates of race 0 of *P. infestans* (isolates 163, 182, and 183; 20,000 sporangia/ml) on 12 and 13 July to each of four plants in the outer rows of each plot. Plots were not artificially inoculated with *A. solani* in the early blight experiment because soil at the test site already was heavily infested.

Treatments consisted of different application schedules of fungicide to obtain varying epidemics. Chlorothalonil (Bravo 720, Diamond Shamrock Corp., Painesville, OH), a broad-spectrum protectant fungicide (0.84 kg a.i./ha), and terbutrazole (Folicur 1.2EC, Mobay Corp., Kansas City, MO), an experimental systemic fungicide selective for *A. solani* (0.20 kg a.i/ha, in the early blight experiment only), were applied in water at 470 L/ha and 860 KPa with a tractor-mounted boom sprayer. Timing of applications in the different treatments were as follows. In the early blight experiment, there were: A) six biweekly applications of Folicur initiated on 16 June; B) six weekly applications of Bravo initiated on 30 June; C) 12 weekly applications of Bravo initiated on 16 June; D) six weekly applications of Bravo initiated on 16 June; and E) untreated. In the late blight experiment, there were uninoculated plots: A) nine weekly applications of Bravo initiated on 14 July; and inoculated plots: B) nine weekly applications of Bravo initiated on 14 July; C) five weekly applications of Bravo initiated on 14 July; D) seven weekly applications of Bravo initiated on 28 July; and E) untreated.

Disease was assessed visually. In the late blight experiment, disease severity and defoliation in the two middle rows of each plot was estimated every 3–5 days using a modification of a blight

TABLE 1. Physiological age (PDAYS) accumulated since emergence until the initiation of tuber growth (P_{tg}) and cessation of bulking (P_{cb}) for different maturity classes of cultivars[a]

Maturity class of cultivar	P_{tg}	P_{cb}
Very early	200	500
Early	200	550
Mid-season	200	600
Mid-late	200	630
Late	200	680

[a] After Johnson et al (8) and Sands et al (22).

TABLE 2. Description of the independent data sets originated in Freeville, NY, on which the loss model was evaluated

Disease	Year	Cultivar	Number of epidemics	Source of data
Early blight	1984	Norchip, Kennebec, Rosa	9	Pelletier (17)
	1985	Kennebec, Rosa	6	Pelletier (17)
	1987	Norchip, Katahdin	6	Shtienberg et al (24)
	1988	Norchip	12	Shtienberg and Fry (*unpublished*)
Late blight	1978	Hudson	9	Fry et al (2)
	1980	Hudson	5	Fry et al (1)
	1981	Hudson, Rosa, Sebago	16	Fry et al (1)
	1983	Hudson, Rosa	8	Fry (*unpublished*)

TABLE 3. Description of the independent data sets from sites other than Freeville, NY, or having more than one pathogen (in Freeville, NY), on which the loss model was evaluated

Disease	Location	Cultivar	No of experiments	No. of epidemics	Source of data
Early blight	Hancock, WI	Russet-Burbank	4	22	Pscheidt and Stevenson (18)
Late blight	Israel	Up-to-Date	3	15	Rotem et al (19)
Early and late blight	Freeville, NY	Norchip, Hudson, Rosa	1	16	Shtienberg and Fry (*unpublished*)

assessment key published by the British Mycological Society (2). In the early blight experiment, disease assessments were made on four randomly selected main stems per plot every 6–8 days. Percent disease and defoliation per plot was calculated as the mean of the percent defoliation of each of the four stems. Assessments started in each experiment after symptoms first became apparent (mid-July) and continued until the last harvest date (mid-September).

For some analyses, the relative area under the disease progress curve (RAUDPC) and the AUDPC were calculated. Defoliation ratings (X_i) of n assessment dates, using a PDAYS time scale (P_i, $i = 1...n$), were used for RAUDPC calculations as follows:

$$RAUDPC = \left[\sum_{i=1}^{n} ((X_{i+1} + X_i)/2)\,(P_{i+1} - P_i)\right]/(P - P_1). \quad (5)$$

If not identified otherwise, RAUDPC calculation was based on disease assessments conducted in the period of yield accumulation. AUDPC was calculated using a chronological time scale (t_i, $i = 1...n$) as:

$$AUDPC = \sum_{i=1}^{n} (X_{i+1} + X_i)/2)\,(t_{i+1} - t_i). \quad (6)$$

For yield evaluation, four plants having three main stems each were harvested periodically from each replicate in the early blight experiment. Harvesting started on 27 June and continued every 2 wk until mid-September. After each plot was harvested, the remaining plants in the plot were left unsprayed. In the late blight experiment, the two middle rows of plots from each replicate were harvested starting 18 July. The remaining plants in the plot were mechanically vine killed.

Data observed in the field were used for estimating the maximal bulking rate (B, equation 4). Yield accumulation over time was described by fitting a Gompertz function to data from each of the treatments. This particular function was chosen because it is an asymptotic function which is simpler than Richards' function and because the curve is not necessarily symmetric around the inflection point (as is the logistic function). The linear rearrangement of the function was used for curve fitting. Yield records of the last harvest date were used as an estimation of asymptotes for the Gompertz function because we did not have enough data points to estimate this parameter statistically. The first derivative of the function quantifies changes in the rate of yield accumulation over time and was used to estimate the maximal bulking rate (B).

Estimating parameters of the model. Parameters of the model described in equation 4 were estimated as follows. Values of B and α were determined using the results of yield accumulation experiments (see Results section). P_{tg} and P_{cb} values for several maturity classes of cultivars were estimated by Sands et al (22) and Johnson et al (8) (Table 1). P_{dmax} was defined arbitrarily as the date of 95% defoliation. This definition had a very small influence on the precision of loss predictions because in 98% of the epidemics disease did not reach this level at P_{cb}. P'_{tg} was defined as not different from P_{tg} because early blight will not develop so early in the season (accumulation of 200 PDAYS from emergence) and late blight is extremely unlikely to occur early enough to affect the date of tuber growth initiation. Disease intensity (A, A') was calculated as RAUDPC, as defined in equation 5.

Evaluating the predictions of the model with independent sets of data. Two types of data sets were used for evaluating the predictions of the model. In the first, experiments conducted in Freeville, NY, were used. This was the location where model parameters were estimated in 1988. However, evaluation data sets consisted of different years (1978–1987) or treatments (in 1988) than those used for constructing the model. Epidemics in these experiments were manipulated by varying the type of fungicide or the spray schedule in different treatments. The data represent means of treatment effects. Temperature was recorded in each experiment by a hydrothermograph installed in a weather station resting about 25 cm above the ground. Experimental procedures differed slightly among years and experiments, but in general they were similar to those described above. The data represent 71 epidemics and corresponding tuber yields (33 for early blight and 38 for late blight) over nine experiments and six cultivars (Table 2). Norchip is an early maturing cultivar; Hudson, Kennebec, Katahdin, and Sebago are mid-season cultivars; and Rosa is a mid-late cultivar.

The second type of data set used for evaluating the model was based on experiments conducted in other areas having considerably different climates than that of Freeville, NY. However, an experiment conducted in Freeville, NY, also was included in this group because early and late blight developed simultaneously in the experimental plots. These data represent 53 epidemics and corresponding tuber yields from eight experiments and five cultivars (Table 3). Up-to-Date is a mid-season cultivar, and Russet-Burbank is a late cultivar.

In each experiment, the treatment having the lowest RAUDPC values was chosen as the reference treatment. Predicted yield loss for each treatment (YL) was calculated according to equation 4, relative to this reference treatment. Observed losses were calculated as percent yield reduction for a treatment relative to the yield of the reference treatment. Predicted and observed yield

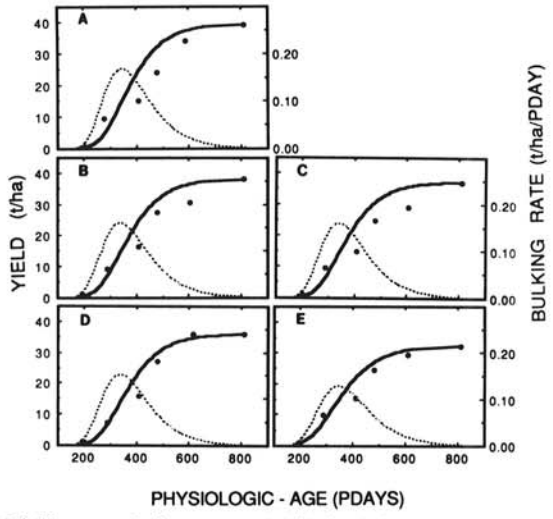

Fig. 1. Yield accumulation curves (solid lines) for several epidemics of early blight. Parameter estimates and regression statistics for a Gompertz function are presented in Table 4. Bulking rate curves (dashed lines) are the first derivative of the fitted Gompertz function.

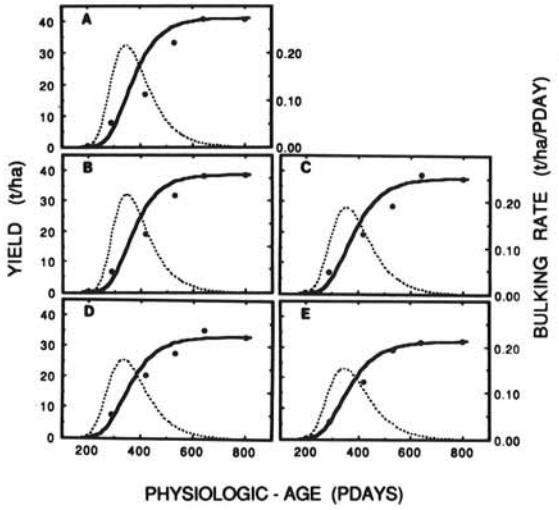

Fig. 2. Yield accumulation curves (solid lines) for several epidemics of late blight. Parameter estimates and regression statistics for a Gompertz function are presented in Table 4. Bulking rate curves (dashed lines) are the first derivitive of the fitted Gompertz function.

TABLE 4. Parameter estimates and regression statistics of the Gompertz equation that were fitted to the yield accumulation curves[a]

Disease	Experimental treatment[b]	Parameters of the equation			
		a	*b*	*c*	r^2
Early blight	A	39.3	57.4	0.012	0.90
	B	38.1	49.4	0.012	0.90
	C	37.1	52.4	0.012	0.87
	D	35.9	50.4	0.012	0.96
	E	32.2	35.1	0.011	0.96
Late blight	A	40.9	144.0	0.014	0.93
	B	38.3	194.4	0.015	0.95
	C	37.5	122.7	0.014	0.95
	D	34.0	104.5	0.014	0.96
	E	31.7	86.5	0.013	0.99

[a] $Y = a \exp(-be - c\,\text{PDAYS})$. Curves are presented in Figures 1 and 2.
[b] See text for details about the timing of fungicide applications in each experimental treatment.

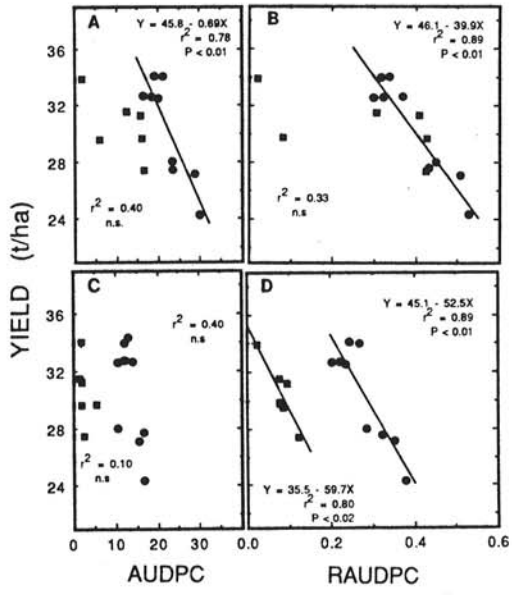

Fig. 3. Effects of early blight on potato yield in the 1984 (circles, after reference 17) and 1987 (squares, after reference 24) growing seasons. Disease intensity is expressed as the area under the disease progress curve (AUDPC) based on chronological time **(A, C)** or as the relative area under the disease progress curve (RAUDPC) based on physiological time (PDAYS) **(B, D)**. Disease ratings were accumulated throughout the entire season **(A, B)** or during the period of tuber yield accumulation **(C, D)**. Regression lines are presented only for those cases in which there was a significant ($P < 0.05$) relationship within each year.

losses were plotted against each other and compared using regression analysis.

RESULTS

Quantifying the effects of early blight and late blight on yield accumulation. Varying schedules of fungicide application resulted in diverse epidemics in the experiments conducted in 1988. Early and late blight disease progress curves for the different treatments are not presented in this report. Yield accumulations over time followed a sigmoid pattern (Figs. 1 and 2), and fitting the Gompertz function to the data resulted in highly significant r^2 values (Table 4). Tuber growth was initiated in both cultivars when approximately 200 PDAYS were accumulated from emergence. Yield reached its asymptote around 550–600 and 550–650 PDAYS for cultivars Norchip (Fig. 1) and Katahdin (Fig. 2), respectively. Observed ranges showed reasonable agreement with the values reported by Sands et al (22) and Johnson et al (8) for these maturities (Table 1). Similar agreement was observed in tuber growth initiation dates for cultivars Norchip, Kennebec, and Rosa in 1984 and 1985 and in cessation of bulking for these three cultivars in 1984 (Pelletier and Fry, *unpublished*).

Yield was reduced in the different treatments by 3.0–18.0% in the early blight experiment and by 6.3–22.4% in the late blight experiment, relative to the highest yielding treatment of each experiment (parameter *a*, Table 4). Date of observed maximal bulking growth rate was not affected by either disease (Figs. 1 and 2). The maximum rates in the different treatments ranged from 0.129 to 0.169 t/ha/PDAY for Norchip (Fig. 1) and 0.154 to 0.216 t/ha/PDAY for Katahdin (Fig. 2). These values are within the ranges reported by Sands et al (22).

One of the decisions we had to make while developing the yield loss model was to choose the time scale to be used for disease intensity calculations. For the purpose of illustration, the relationship between yield and disease intensity accumulated over different time scales is presented in Figure 3. Yield was related significantly to AUDPC values (based on chronological time scale) in one case. However, a better relationship was observed when RAUDPC (based on PDAYS time scale accumulated over the tuber bulking period) was used (Fig. 3). Therefore, RAUDPC was chosen to present disease intensity in our model.

Observed values of maximal bulking rate were linearly related to the RAUDPC in each one of the 1988 experiments (Fig. 4). The slope of each curve represents the reduction of maximal bulking rate corresponding to each differential unit of RAUDPC. We defined this parameter as α (equation 3). Because the slopes calculated for cultivars Norchip and Katahdin did not differ significantly according to *t*-test ($P = 0.05$, Fig. 4), the mean value (0.252 t/ha/PDAY/RAUDPC unit) was used in further analysis as an overall estimation of α. The intercept of each curve (at RAUDPC = 0.0) represented the maximal bulking rate in the absence of disease for this cultivar under the growing conditions of the experiment. This parameter was defined as *B* (equation 1). These values were 0.184 and 0.257 t/ha/PDAY for Norchip and Katahdin, respectively. The maximal bulking rate is dependent on the maturity characteristics of the cultivar (22). We arbitrarily assumed a linear relationship between the maturity class of cultivars and their maximal bulking rate and calculated a value for each maturity class of cultivars by linear interpolation from the observed values. Calculated *B* values for the corresponding groups were 0.138, 0.184, 0.229, 0.257, and 0.302 t/ha/PDAY for very early, early, mid-season, mid-late, and late cultivars.

Evaluating the predictions of the model with independent sets of data. Predictions of percent yield loss for the 71 epidemics of early and late blight in Freeville, NY (minus the nine reference treatments) were plotted against observed values (Fig. 5). By using a standard deviation of 4 as the threshold for selecting outliers (14), three data points were considered as outliers and excluded from the data set.

The theoretical line indicating a perfect coincidence between predicted and observed losses has an intercept of 0 and slope of 1. The observed regression equation describing the fit between predicted and observed values had an intercept (0.12%) that was not significantly different from 0 ($P = 0.05$) and a slope (0.97)

that was not significantly different from 1 ($P = 0.05$). Standardized residuals were distributed randomly around 0 (Fig. 6), indicating that the predictions of the model were independent of the values of observed yield losses. Most of the predictions (72%) were within the range of 1 standard deviation, 22% were within 1–2 standard deviations, and only 6% were within 2–3 standard deviations of the observed value (Fig. 6).

For comparison, models proposed by Teng and Bissonnette (25) for early blight and by Latin et al (11) for late blight were used for predicting yield losses for the same data set. Predictions of the early blight model (developed in Minnesota) consistently deviated from observed, whereas the predictions of the late blight model (developed in Pennsylvania) were closer. However, the accuracy of predictions obtained by the Pennsylvania model was slightly less than that of the model proposed in this study (Fig. 5), as indicated by a lower r^2 value (0.87 versus 0.94) and a higher standard deviation value (9.57 versus 3.74).

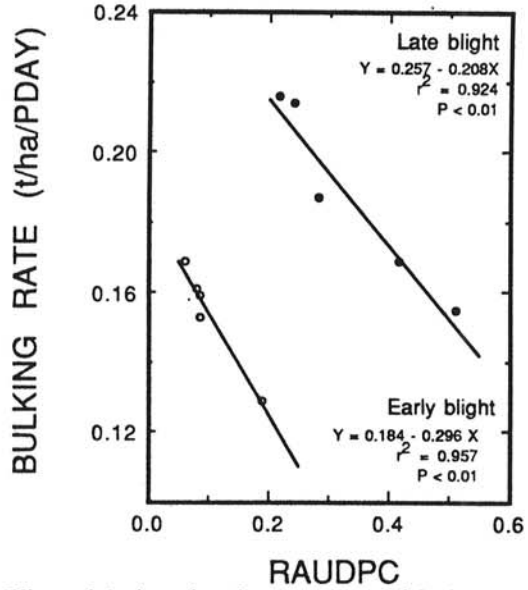

Fig. 4. Effects of the intensity of early and late blight (expressed as the relative area under the disease progress curve, RAUDPC) on maximal observed bulking rate as recorded in Freeville, NY, in 1988. Open circles = cultivar Norchip; closed circles = cultivar Katahdin.

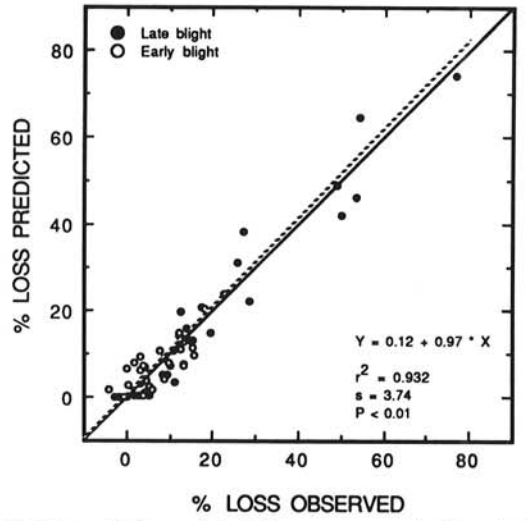

Fig. 5. Model predictions of yield losses compared with actual values observed in field experiments conducted in Freeville, NY. The dashed line indicates the regression equation describing the fit between observed and predicted values. The solid line indicates the relationship of a perfect coincidence between observed and predicted values. See Table 2 for more information about the source of the data.

Model predictions of yield losses in field experiments conducted in locations other than Freeville, NY, or having more than one pathogen (in Freeville, NY) were not as precise as those predicted for experiments conducted in Freeville, NY, with one pathogen. Nevertheless, predictions for yield loss in epidemics involving both pathogens, or occurring in locations other than Freeville, NY, were reasonably coincident with observed losses (Fig. 7). The regression describing the fit between predicted and observed values had an intercept (0.41%) that was not significantly different from 0 ($P = 0.05$) and a slope (0.87) that was not significantly different from 1 ($P = 0.05$). Standardized residuals were distributed randomly around 0, among which 65% were within the range of 1 standard deviation, 25% were within 1–2 standard deviations, and 10% were within 2–3 standard deviations of the observed (Fig. 8).

DISCUSSION

The requirements of a method for predicting yield losses were listed and discussed by James et al (7): Loss predictions should be within 5% of the actual loss, and the method should estimate the loss from any given disease progress curve and allow comparison of losses from any given pair of progress curves (7). If a general method is to be developed, its ability to predict losses accurately in different cultivars, climatic conditions, and growing areas should be included as an additional requirement.

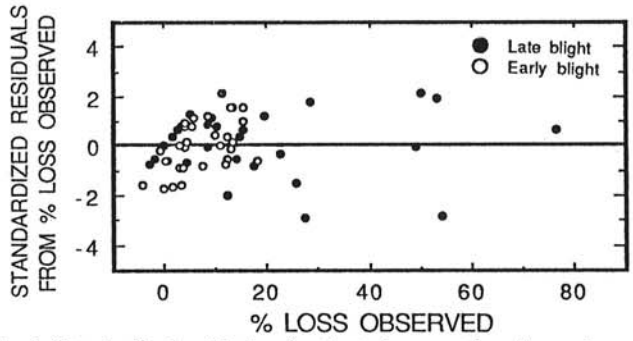

Fig. 6. Standardized residuals of estimated percent loss from observed percent loss for epidemics of early and late blight in Freeville, NY. Standardized residuals were calculated as: (Observed − Predicted)/σ (after reference 14), where σ is the standard deviation about the regression line. See Table 2 for more information about the source of the data.

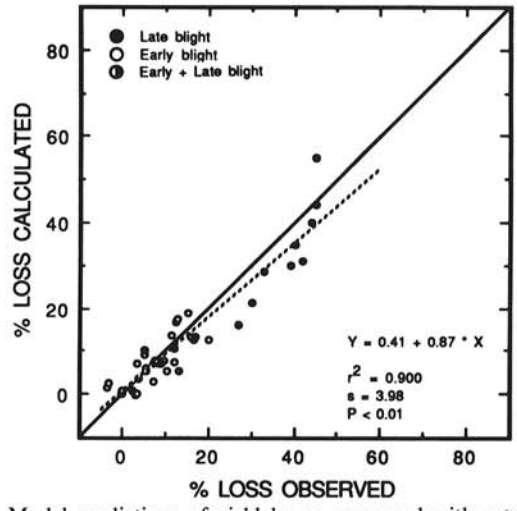

Fig. 7. Model predictions of yield losses compared with actual values observed in field experiments conducted in locations other than Freeville, NY, or having more than one pathogen (in Freeville, NY). The dashed line indicates the regression equation describing the fit between observed and predicted values. The solid line indicates the relationship of a perfect coincidence between observed and predicted values. See Table 3 for more information about the source of the data.

The model described in this report closely fits these requirements. Losses predicted by the model presented were not significantly different from observed values for epidemics in dissimilar environments (Figs. 5–8). We evaluated the precision of predictions in absolute values (that is, deviation in percent loss predicted and the observed) and not in relative values (that is, percent deviation) because this is more meaningful biologically and economically (7). In 80% of the cases, prediction of losses was within 5% of the observed when independent data sets were analyzed (Figs. 5 and 7). James' multiple regression model (7) predicted late blight losses within 5% of the observed in 90% of the cases. However, these were calculated by analyzing the data set that was used for the construction of the very same regression model. Our loss model, unlike previous empirical ones, is considered a general model because it has provided accurate loss predictions for data sets originated under diverse agricultural practices and environmental conditions, for single pathogens as well as for two pathogens (Figs. 5 and 7). We conclude, therefore, that our model successfully fulfills the requirements of a general loss model.

Integration of early mild epidemics or late severe ones over time may result in the same AUDPC values. Hence, effects of such diverse epidemic profiles on yield may vary, leading James (4) to conclude that AUDPC is an inappropriate predictor of yield losses. The results demonstrated in Figure 3A and C support this conclusion. Nevertheless, in a survey of papers from 1948 to 1982 dealing with empirical modeling of crop losses as a function of disease, about 30% of the authors used AUDPC (alone or in combination with other parameters) as a predictor of yield losses with adequate to excellent success (12). This means that, at least for some host-pathogen interactions, AUDPC may be used for accurate loss estimations. The dynamics and duration of yield accumulation, which vary among crops, may result in inconsistent success of AUDPC as a loss predictor in different studies (23). We hypothesized that consideration of yield dynamics in the loss model should improve the precision of loss predictions. Potato tuberization starts about one-third to one-half way through the growing season and continues until its end. About 90% of the dry matter accumulated by the tubers is from photosynthesis that occurs after tuber initiation (13). Any change in disease during this period may affect the attainable yield (7). The effect of environment on the rate of potato growth and yield accumulation may be considered by employing a physiological time scale (8,22). Integrating disease severity over the period of yield accumulation and weighing each chronological day by its relative contribution to host growth should improve the precision of yield loss predictions. This study demonstrates that RAUDPC is indeed an adequate predictor of yield (Fig. 3D) and yield loss (Figs. 5 and 7) in potatoes.

In most empirical models for predicting yield loss, including the model presented in this study, disease severity and yield loss are expressed as percent values (4). However, because plant size and yield may vary in different seasons and locations, different amounts of healthy foliage may be present with the same percentage of defoliation (20). Other authors, therefore, have proposed healthy haulm area as a preferable parameter for yield loss assessment (19,20,27). Plant growth simulators that dynamically relate plant growth and environmental measures, coupled with pest population submodels, have been suggested as the best method to improve analysis of pest-induced losses (8,21). The development of such an amalgamated simulation model is complicated, expensive, and time consuming. Its evaluation procedures and implementation may be even more intricate than the development process. Loss estimates from such combined models are expected to be more widely applicable than those from empirical models because they are biologically mechanistic (21). However, the simplicity of the empirical model presented in this study, the acceptable accuracy of its predictions, and its generality make this model attractive.

The empirical model presented in this study was parameterized for estimating losses induced by early blight and/or late blight. However, it may be used for any other foliar pathogens or pests of the potato crop. Because the α parameter might be pathogen dependent, it must be estimated empirically for each pest, or combination of pests, separately.

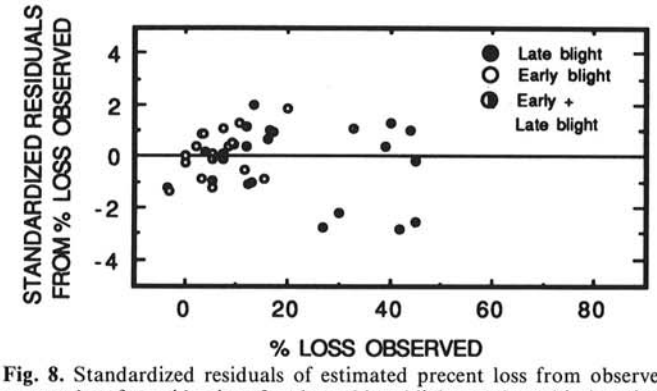

Fig. 8. Standardized residuals of estimated precent loss from observed percent loss for epidemics of early and late blight conducted in locations other than Freeville, NY, or having more than one pathogen (in Freeville, NY). Standardized residuals were calculated as: (Observed − Predicted)/σ (after reference 14), where σ is the standard deviation about the regression line. See Table 3 for more information about the source of the data.

LITERATURE CITED

1. Fry, W. E., Apple, A. E., and Bruhn, J. A. 1983. Evaluation of potato late blight forecast modified to incorporate host resistance and fungicide weathering. Phytopathology 73:1054-1059.
2. Fry, W. E., Bruck, R. I., and Mundt, C. C. 1979. Retardation of potato late blight epidemics by fungicides with eradicant and protected properities. Plant Dis. Rep. 63:970-974.
3. Hooker, W. J., ed. 1981. Compendium of Potato Diseases. American Phytopathological Society, St. Paul, MN. 125 pp.
4. James, W. C. 1974. Assessment of plant diseases and losses. Annu. Rev. Phytopathol. 12:27-48.
5. James, W. C. 1981. Estimated losses of crops from plant pathogens. Pages 79-94 in: Handbook of Pest Management in Agriculture. D. Pidmentel, ed. CRC Press, Boca Raton, FL. 579 pp.
6. James, W. C., Callbeck, L. C., Hodgron, W. A., and Shih, C. S. 1971. Evaluation of a method used to estimate loss in yield of potatoes caused by late blight. Phytopathology 61:1471-1476.
7. James, W. C., Shih, C. S., Hodgson, W. A., and Callbeck, L. C. 1972. The quantitative relationship between late blight of potato and loss in tuber yield. Phytopathology 62:92-96.
8. Johnson, K. B., Johnson, S. B., and Teng, P. S. 1986. Development of a simple potato growth model for use in crop pest management. Agric. Syst. 19:189-209.
9. Johnson, K. B., Radcliffe, E. B., and Teng, P. S. 1986. Effects of interacting populations of *Alternaria solani*, *Verticillium dahliae*, and the potato leafhopper (*Empoasca fabae*) on potato yield. Phytopathology 76:1046-1052.
10. Large, E. C. 1952. The interpretation of progress curves for potato late blight and other plant diseases. Plant Pathol. 1:109-117.
11. Latin, R. X., Mackenzie, D. R., and Cole, H., Jr. 1978. Crop loss prediction tables for potato late blight epidemics. Am. Potato J. 55:541-548.
12. Madden, L. V. 1983. Measuring and modeling crop losses at the field level. Phytopathology 73:1591-1596.
13. Moorby, J. 1982. The physiology of growth and tuber yield. Pages 152-194 in: The Potato Crop. The Scientific Bases for Improvement. P. M. Harris, ed. Chapman and Hall, London. 730 pp.
14. Neter, J., Wasserman, W., and Kutner, M. H. 1985. Applied Linear Statistical Models. Richard D. Irwin, Homewood, IL. 1127 pp.
15. Olofsson, B. 1968. Determination of the critical injury threshold for potato late blight (*Phytophthora infestans*). Waxtskyddsanst Stokholm 14:85-93.
16. Palm, E. W. 1981. Estimating crop losses without the use of fungicides and nematocides and without nonchemical control. Pages 139-158 in: Handbook of Pest Management in Agriculture. D. Pimentel, ed. CRC Press, Boca Raton, FL. 579 pp.
17. Pelletier, J. R. 1987. Computer simulation of cultivar resistance and fungicide effects on epidemics of potato early blight. Ph.D. thesis. Cornell University, Ithaca, NY. 127 pp.
18. Pscheidt, J. W., and Stevenson, W. R. 1988. The critical period for control of early blight (*Alternaria solani*) of potato. Am. Potato J.

65:425-438.
19. Rotem, J., Bashi, E., and Kranz, J. 1983. Studies of crop loss in potato blight caused by *Phytophthora infestans*. Plant Dis. 32:117-122.
20. Rotem, J., Kranz, J., and Bashi, E. 1983. Measurement of healthy and diseased haulm area for assessing late blight epidemics in potatoes. Plant Pathol. 32:109-115.
21. Rouse, D. I. 1983. Plant growth models and plant disease epidemiology. Pages 387-398 in: Challenging Problems in Plant Health. T. Kommedahl and P. H. Williams, eds. American Phytopathological Society, St. Paul, MN. 538 pp.
22. Sands, P. J., Hackett, C., and Nix, H. A. 1979. A model of the development and bulking of potatoes (*Solanum tuberosum* L.). I. Derivation from well managed field crops. Field Crops Res. 2:309-331.
23. Schneider, R. W., Williams, R. J., and Sinclair, J. B. 1976. Cercospora leaf spot of cowpea: Models of estimating yield loss. Phytopathology 66:384-388.
24. Shtienberg, D., Doster, M. A., Pelletier, J. R., and Fry, W. E. 1989. Use of simulation models to develop a low-risk strategy to suppress early and late blight in potato foliage. Phytopathology 79:590-595.
25. Teng, P. S., and Bissonnette, H. L. 1985. Developing equations to estimate potato yield loss caused by early blight in Minnesota. Am. Potato J. 62:607-618.
26. Vanderplank, J. E. 1963. Plant Diseases: Epidemics and Control. Academic Press, New York. 349 pp.
27. Waggoner, P. E., and Berger, R. D. 1987. Defoliation, disease and growth. Phytopathology 72:393-398.