Analysis of Potato Late Blight Epidemiology by Simulation Modeling

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This work was supported in part by USDA/SEA Cooperative Research Grant 701-15-54.
Accepted for publication 17 November 1980.

ABSTRACT

Bruhn, J. A., and Fry, W. E. 1981. Analysis of potato late blight epidemiology by simulation modeling. Phytopathology 71:612-616.

A mathematical model that simulates effects of environment, fungicide, and host on asexual development of *Phytophthora infestans* on potato foliage was constructed and evaluated. In comparisons involving four potato cultivars during three growing seasons the model accurately mimicked actual epidemic development. The model was then used in conjunction with a weather model and a fungicide model to determine the effect of rate-reducing resistance on the performance of various fixed interval fungicide spray schedules. Simulation results indicated that: intervals between sprays can be extended when resistant potato cultivars are grown; plant disease forecasting systems used to time fungicide applications are likely to be most cost effective for cultivars that are most susceptible; and profit maximization, when used alone, is insufficient for evaluating the effectiveness of potato late blight management policy because plant disease control practices tended to reduce both total costs and their associated variability.

The use of protectant fungicides to control potato late blight is an essential component of potato production in many parts of North America. Chemicals are normally applied on a regular schedule throughout the growing season, but rising costs and increasing governmental regulations are forcing producers to consider more efficient approaches to the use of fungicides. By timing fungicide applications to coincide with environmental conditions that favor disease development, forecasting systems allow for improved fungicide efficiency (1,13,16). The time interval between sprays can be extended when potato cultivars with high levels of rate-reducing resistance are grown (7,8). Further, because the impact of potato late blight on tuber yield declines as the season progresses (14,17,18,25), it may be possible to increase the application intervals in fungicide spray schedules as the crop matures. Current fungicide usage for the control of potato late blight thus seems excessive. A rational and cost-effective policy of fungicide use must consider the effects of environment, host, fungicide, and pathogen on the efficiency of potato late blight management. Analysis with plant disease models may aid these considerations.

Mathematical models have been used to examine a variety of complex pest management systems (12,21,22,29). An accurate model of the potato late blight management system should be useful for evaluating alternative policies of fungicide use. Before a model can be used, the accuracy and reliability of each of its component submodels must be assessed.

The purpose of this report is to present a simulation model describing the development of *Phytophthora infestans* (Mont.) de Bary on potato foliage during one growing season. Model performance is evaluated by comparing actual epidemic development with disease progress predicted by the model. The model is then used with other models to examine the effects of rate-reducing resistance on the performance of various potato late blight management programs. Finally, limitations and applications of the model are discussed.

MATERIALS AND METHODS

Model description. The model operates on a daily time step and simulates the effects of environment, host, and management activities on the asexual development of *P. infestans*. Like other simulation models of plant pathogen development (6,23,24,26–28) the model's state variables represent the major morphological stages of the pathogen, and its state diagram closely parallels the pathogen's life cycle diagram. However, because spore survival is limited to a single day in the model and because changes in both number of lesions and lesion size are described by the model, matrix notation provides a more concise and accurate representation of the model's structure than that afforded by a state diagram and it will be used in describing the model.

The number of lesions on day t in each of 15 age classes and the average surface area of lesions in each age class are represented by the vectors n_t and m_t, respectively, and are described by the following:

$$n_t = P_t n_{t-1}$$

$$m_t = V_t m_{t-1}$$

The 15×15 matrices P_t and V_t describe the day-to-day changes in number of lesions and lesion area per lesion, respectively, associated with each age class. Both P_t and V_t are functions of a number of factors that affect pathogen development (Table 1). The equations used to describe the effects of these factors have been reported elsewhere (2) and are available from the authors upon request.

The matrix P_t can be partitioned into two components. The first component, f'_t, consists of the first row of P_t. Its elements represent the average number of sporangia that have been produced on lesions of each age class, have been dispersed, and have landed on susceptible tissue and are described by:

$$f'_t = z_t m'_t,$$

in which z_t is the average number of successfully dispersed sporangia produced per unit (mm^2) of lesion area and represents an aggregate of the processes of spore production, release, and dispersal and where m'_t is the transpose of the vector m_t. The remaining component of P_t is a 14×15 matrix whose only nonzero elements, p_i, form the off-diagonal elements of P_t and describe the probability of survival from one age class to the next. The first element, p_1, represents the probability that a dispersed sporangium will cause an infection, so that $0 \leqslant p_1 \leqslant 1$. In the model, all successful infections survive 15 days so $p_i = 1$ for $i = 2, 3, \ldots\ldots 14$.

The structure of V_t is slightly different from that of P_t. A host-dependent latent period of h days is required before lesions have surface area and can produce sporangia. Lesions can then expand for (10−h) days and can produce sporangia for (15−h) days. Thus, the elements of the first row of V_t all equal zero, and the off-diagonal elements, v_i, assume the values:

$$v_i = 0 \qquad i = 1, 2, \ldots, h-1$$

0031-949X/81/06061205/$3.00/0

$$v_i = g_t \qquad i = h, \ldots, 9$$

$$v_i = 1 \qquad i = 10, \ldots, 14$$

in which g_t is the daily rate of lesion area increase per lesion on day t. Finally, the h^{th} diagonal element of V_t equals one for all t and the initial value of the h^{th} element of m_t is defined as 0.8 mm^2 to initiate lesion expansion.

Total host leaf surface area on day t, q_t, was described by a polynomial function of time with coefficients c_i for $i = 1, 2, \ldots 5$, and was derived from Moorby and Milthorpe (19). The impact of foliage blight on tuber yield, d_t, was derived from MacKenzie and Petruzzo (18). By defining w_t and x_t as the cumulative "inactive" lesion area and the proportion of blighted foliage, respectively, on day t, the model can be summarized by the following:

$$n_t = P_t n_{t-1}$$

$$m_t = V_t m_{t-1}$$

$$w_t + w_{t-1} + m_{t-1,\,15}{}^{n_{t-1,15}}$$

$$q_t = \sum_{i=0}^{5} c_i\, t^i$$

$$x_t = (n'_t\, m_t + w_t)/q_t$$

$$d_t = d_{t-1} + (3.3 - .0285t)\,(x_t - x_{t-1}),$$

in which $m_{t-1,15}$ and $n_{t-1,15}$ represent the 15th element of the vectors m_{t-1} and n_{t-1}, respectively.

Model performance. The development of late blight epidemics within 16 m^2 (4×4) plots of the potato cultivars Katahdin, Kennebec, Sebago, and Monona was monitored during 1977, 1978, and 1979 at Freeville, NY. Disease progress information was unavailable for Katahdin and Monona in 1978 and for Kennebec in 1979. Plots were arranged in a randomized complete block design with four replications in 1978 and three replications in 1977 and 1979. Cultural conditions were similar to those previously described (9,10). A sporangial suspension was applied to the foliage of a plant in the center of each plot to initiate epidemic development. Plots were irrigated by sprinklers at dawn and dusk for a total of 0.25 cm of water daily to produce conditions conducive to disease development. Environmental data were collected throughout the growing season with a hygrothermograph recorder and rain gauge. The hygrothermograph was sheltered within a wooden, ventilated structure positioned within the canopy of a potato plot. These weather data, in the form of daily values of average temperature, rainfall, hours of relative humidity exceeding 90%, and average temperature during periods when relative humidity exceeded 90% were used as input to produce the simulated epidemics. Predicted disease progress curves were then compared with actual epidemics that were assessed as described by Fry (9).

Four statistics, the apparent infection rate (r), the area under the disease progress curve (AUDPC), the number of days following inoculation until foliage is 50% blighted (t_{50}) and the level of disease observed at the last assessment date (X_f), were used to characterize and compare the disease progress curves. The statistic, r, was computed as the least squares estimator of the slope of the regression of $\ln(x/[1-x])$ on time for values of x between 0.05 and 0.95 (25). The value of AUDPC was calculated with the equation described by Fry (9).

Model application. Two models were used with the late blight model to examine the effects of rate-reducing resistance on the costs associated with various potato late blight management programs. First, a stochastic weather model (3) was used to generate five seasons of daily weather data that were characteristic of central New York growing conditions. Second, a fungicide model was used to simulate the changes in fungicide residue levels over time and the interaction of fungicide residues with pathogen development. Because sufficient data were unavailable for the development of a complete and accurate fungicide model for any commercially available fungicide, we assume the existence of a fungicide that exhibits the general characteristics of commercial materials (4,5,15,20). Unpublished data for chlorothalonil sprayed on potato foliage to control *P. infestans* were used to estimate model parameters. In the model, the fungicide is applied uniformly over the foliage at a dosage of 2.5 μg a.i./cm^2 of foliage. The fungicide deposit on day t, r_t, declines according to the following:

$$r_t = k r_{t-1} e^{b(\text{rain})},$$

in which k equals 0.90 and b equals −0.56 per centimeter of rain. The dosage response curve for the fungicide is assumed to be linear on a $\log_{10}$ concentration-probit proportion inhibition scale with an intercept of 7.60 and slope of 2.07. Fungicides were applied on various fixed schedules beginning 5 days after the introduction of initial inoculum. To permit computation of cost information, all cultivars were assumed to produce 35,900 kg/ha of tubers in the absence of disease. The price received for potatoes was set at \$0.11/kg and fungicide costs were \$19.77/ha/application.

TABLE 1. Factors influencing the development of *Phytophthora infestans* and operation of a model to simulate potato late blight epidemics

Stage of pathogen development	Factors affecting process
Spore production	Temperature, relative humidity, cultivar
Dispersal	Leaf wetness, rainfall, proportion of susceptible tissue
Germination	Temperature, leaf wetness
Infection	Temperature, leaf wetness, cultivar, fungicide
Lesion expansion	Temperature, cultivar, proportion of susceptible tissue

TABLE 2. Comparison of model predictions with observed epidemic development of late blight on four potato cultivars during 1977

	Sebago		Katahdin		Kennebec		Monona	
Parameter	Observed[a]	Predicted[b]	Observed	Predicted	Observed	Predicted	Observed	Predicted
Apparent infection rate (r)	.172–.186	.183	.175–.204	.161	.196–.227	.211	.339–.386	.375
Area under disease progress curve[c]	7.27–8.21	8.54	10.67–14.49	14.46	6.76–9.87	9.00	21.51–23.14	22.75
Days until 50% blight	37–38	37	28–30	27	34–38	36	20	21
Final level of disease	.65–.75	.68	.81–.86	.88	.75–.83	.85	1.00	1.00

[a] Range observed in three replicated disease progress curves.
[b] Simulation predictions using weather data recorded during observed epidemic development.
[c] Final disease assessment 43 days after inoculation.

RESULTS

Model performance. We wanted to use this model as a tool for evaluating the efficacy of various potato late blight management programs. Therefore, the model should accurately simulate the development of potato late blight epidemics under a variety of environmental conditions. Model performance was evaluated by comparing real disease progress curves in four potato cultivars (which have differing levels of rate-reducing resistance) with disease progress curves generated by the model when using the weather data recorded during each of three seasons. In general, the values of the statistics describing the disease progress curves for the simulated epidemics were very similar to the values obtained in comparable actual epidemics for all 3 yr (Tables 2–4).

The 1977 data illustrate the differences in resistance of the four cultivars. Although simulated epidemics were in close agreement with observed epidemics for all four cultivars in 1977 (Table 2), comparisons using 1977 data do not represent a valid test of model performance because these data also were used to estimate certain parameters describing cultivar resistance.

Disease progress curves were available for plantings of cultivars Sebago and Kennebec in 1978 (Table 3). The rate of epidemic development in 1978 was almost double the rate observed in 1977 for both actual and simulated epidemics. Although the values of r associated with the simulated epidemics were below the range of values for observed epidemics, the differences in r values between simulated and actual epidemics were very small. Because the assessment period was shorter in 1978 (32 days) than in 1977 (42 days), both simulated and observed epidemics were characterized by small values of AUDPC in 1978 relative to 1977. Simulated and actual epidemics were described by very similar values of AUDPC, t_{50} and X_f in 1978.

In 1979 comparisons of epidemic development were performed for the cultivars Sebago, Katahdin, and Monona (Table 4). Simulated and actual disease progress curves were very similar for Monona even though actual epidemic development was based on assessments from a single potato plot. As measured by r, actual epidemic development in 1979 was more rapid than that of 1977, but the differences in r between observed epidemics in those 2 yr were different for each cultivar. These trends also were predicted by the model. Although the model overestimated the value of r for Sebago, the disease progress curves of simulated and actual epidemics were very similar as measured by the other three statistics. Simulated epidemics also were very similar to observed epidemics for the cultivar Katahdin.

The major discrepancy between simulated and actual epidemics was a tendency of the model to predict larger disease levels when $X_t > .50$, which may suggest that the effect of pathogen density on epidemic development has not been completely described. This discrepency was considered unlikely to affect the model's usefulness in management policy evaluation since control programs in which disease levels exceed 50% are unacceptable management alternatives.

Model application. Mathematical models can provide new insight into the behavior of complex plant disease management systems because a more complete array of management alternatives can be considered than is usually practical with field experiments. We used the potato late blight model in combination with the fungicide and weather models to examine the effect of rate-reducing resistance on the performance of various fixed-interval fungicide schedules for control of *P. infestans*. The efficacy of a protectant fungicide applied on 5-, 7-, 10-, 14-, 21-, and 28-day schedules over five seasons on late blight development on plants of potato cultivars Monona, Katahdin, and Kennebec was considered (Table 5). The average disease management costs per hectare and the associated standard deviation were used to assess the effectiveness of each management program. Management costs were computed as the total of fungicide costs and lost revenue associated with pest damage.

The level of resistance in the three cultivars was never sufficient to eliminate the need for protectant fungicide at regular intervals. However, longer intervals between sprays were possible for cultivars with relatively high levels of resistance than for those of low resistance. A 10-day schedule minimized average costs for Monona, the most susceptible cultivar, while fungicide application every 14 days was the most cost-effective alternative for Katahdin and Kennebec (moderately susceptible and moderately resistant, respectively).

The data also showed that minimization of expected costs was not a sufficient criterion for assessing the performance of potato late blight management policy because variability in costs was affected by both the intensity of fungicide use and the level of resistance. For example, although the 14-day fungicide application schedule minimized expected costs for cultivar Katahdin, only $13.44 per hectare per season was saved in comparison to the 7-day schedule while the standard deviation of costs for the 14-day schedule was substantially higher than that of the 7-day schedule, $51.89 and $5.07 per hectare per season for the 14- and 7-day schedules, respectively. The acceptable tradeoffs between increased profits and reduced variability will differ among individuals (11), but it is likely that many producers averse to taking risks would consider a 7-day fungicide schedule to be optimal for Katahdin.

TABLE 3. Comparison of model predictions with observed epidemic development of late blight on two potato cultivars during 1978

Parameter	Sebago		Kennebec	
	Observed[a]	Predicted[b]	Observed	Predicted
Apparent infection rate	.338–.361	.318	.394–.395	.349
Area under disease progress curve[c]	3.19–6.39	5.27	2.53–4.57	4.66
Days until 50% blight	27–32	32	29–32	29
Final level of disease	.54–.76	.79	.52–.78	.80

[a] Range observed in four replicated plots.
[b] Simulated disease progress curves using weather data recorded during observed epidemic development.
[c] Final disease assessment 33 days after inoculation.

TABLE 4. Comparison of model predictions with observed epidemic development of late blight on three potato cultivars during 1979

Parameter	Sebago		Katahdin		Monona	
	Observed[a]	Predicted[b]	Observed	Predicted	Observed[c]	Predicted
Apparent infection rate	.180–.197	.267	.332–.361	.351	.412	.445
Area under disease[d] progress curve	16.66–18.25	16.04	21.69–23.66	21.61	27.75	26.29
Days until 50% blight	32–34	35	28–30	31	26	26
Final level of disease	.91–.95	.98	1.00	1.00	1.00	1.00

[a] Range observed in three disease progress curves.
[b] Simulation predictions using weather data recorded during epidemic development.
[c] Nonreplicated disease progress curve was used.
[d] Final disease assessment 52 days after inoculation.

TABLE 5. The effect of rate-reducing resistance on the cost effectiveness of various fungicide schedules for controlling *Phytophthora infestans* on three potato cultivars: simulation results

Fungicide schedule	Applications per season	Monona		Katahdin		Kennebec	
		Average[a] costs	± s.d.[b]	Average costs	± s.d.	Average costs	± s.d.
5-day	11	228.81	6.02	224.56	1.58	243.64	1.58
7-day	8	178.16	6.42	171.59	5.07	162.10	1.98
10-day	6	169.51	89.20	167.63	47.59	130.54	11.47
14-day	5	196.20	137.14	158.14	51.89	105.17	15.34
21-day	3	320.24	333.83	287.03	280.71	144.70	141.27
28-day	3	585.13	354.59	389.82	331.71	206.38	249.87
No fungicide	0	1681.86	33.61	1330.78	44.28	1059.56	73.93

[a] Average cost (dollars per hectare per season) over five growing seasons. Computations assume: average tuber yield without late blight = 35,900 kg/ha; price of potatoes = $0.11/kg; and fungicide costs = $19.77 per hectare per application.
[b] Standard deviation of costs over five growing seasons.

Similarly, by adopting a 7-day schedule instead of a 10-day schedule for Monona, a producer would pay only $8.65 per hectare per season for a 10-fold reduction in the standard deviation of costs (s.d. = $ 6.42 and $89.20 per hectare per season for 7- and 10-day schedules, respectively). A 14-day schedule was, however, clearly superior to a 7-day schedule for the resistant cultivar Kennebec since savings are high, $56.83 per hectare per season, in relation to variability in costs (s.d. = $1.98 and $15.34 for 7- and 14-day schedules, respectively.)

In general, the fungicide policy that minimized expected costs over all five seasons was not likely to be optimal for any given season. For Monona (Table 6) a 7-day schedule was most cost effective in only one of the five seasons while 10- and 14-day schedules each minimized costs in two of the years. If the time interval between fungicide applications had been adjusted to compensate for the environmental conditions encountered in each season, costs would be reduced $37.16 per hectare per season for Monona. Using the fungicide policy that minimizes costs for each season also reduced costs for Katahdin and Kennebec but the magnitude of the savings declined with the level of resistance associated with each cultivar; reduced costs equaled $30.25 and $17.40 per hectare per season for Katahdin and Kennebec, respectively. These results imply that if fungicide is applied only when environmental conditions are conducive for disease development the efficiency of fungicide use will increase. Moreover, the relative benefits derived from the use of such a forecasting system are likely to be greatest for the most susceptible cultivars.

TABLE 6. Plant disease management costs associated with the application of fungicides for control of *Phytophthora infestans* on 7-, 10-, and 14-day schedules on plantings of potato cultivar Monona during each of five growing seasons—simulation results

Season	Fungicide schedule		
	7-day	10-day	14-day
1	173.46[a]	118.11	106.25*
2	185.32	145.79*	153.70
3	185.32*[b]	384.99	410.68
4	173.46	137.88	106.25*
5	173.46	118.11*	126.02

[a] Potato late blight management costs (dollars per hectare per season). Cost computations assume: average tuber yield without late blight = 35,900 kg/ha; price of potatoes = $0.11/kg; and fungicide costs = $19.77 per hectare per application.
[b] * Fungicide policy that minimizes costs in the current year.

DISCUSSION

The model presented here provides an accurate representation of the development of *P. infestans* on potato foliage over a range of environmental and host conditions. The model was developed for use as a tool to evaluate the effectiveness of potato late blight control policy and was used with models of other components of the management system to show that fungicide use can be reduced by using cultivars with increased levels of rate-reducing resistance. The simulation results also showed that the benefits derived from a forecasting system are greatest when cultivar resistance is lowest. The model affirmed that plant disease control practices reduce costs and stabilize profits. Simulations suggested that profit maximization, when used alone, may not be a sufficient criterion for evaluating the effectiveness of plant disease management policy because variability in policy effectiveness, as measured by costs, is reduced when resistant cultivars are grown or when fungicide is used intensively.

By definition, model construction requires that certain features of the real system be simplified or omitted from consideration. These assumptions aid in the analysis but place restrictions on the reliability of model performance and limit the type of problems that can be considered with the model. Because this model does not describe the overwintering of the pathogen or the production of initial inoculum, its use as an "on-line" pest management tool for predicting disease incidence and yield loss in specific fields is limited. An "on-line" application of the model is further restricted because the spatial dynamics of epidemic development are not considered. These restrictions do not, however, limit the usefulness of the model for evaluating alternative plant disease management programs.

The mathematical structure of the model could have been made more flexible by making the latent period and lesion survival more responsive to environmental conditions. Such a modification would allow the model to operate on a physiological time basis. Although the concept of physiological time has proven useful for simplifying the analysis of pest management systems (21,22), its application to *P. infestans* is difficult because the rate of pathogen development is dependent upon a number of environmental factors. Since the current model structure appears to provide an accurate representation of the development of *P. infestans* on potatoes, physiological time was not used.

The potato late blight model accurately simulated pathogen development and was used with a very simple fungicide model to qualitatively analyze the effectiveness of various fungicide spray schedules. The results of this analysis suggested approaches to developing fungicide programs that will control *P. infestans* more efficiently. Because certain features of fungicide deposition and loss were oversimplified in the fungicide model, the quantitative accuracy of our analysis is uncertain. Thus, our results should not be used for making specific management recommendations. Models to describe the temporal dynamics of fungicide residue levels and the infection of potato tubers by *P. infestans* are being developed. These models, when used with the pathogen model, should allow for a thorough analysis of the potato late blight management system.

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