Use of Simulation Models to Develop a Low-Risk Strategy to Suppress Early and Late Blight in Potato Foliage

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ABSTRACT

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Simulation models describing potato early blight development, potato late blight development, and chlorothalonil dynamics were used in analyzing the effect of fungicide applications on disease epidemics. The early blight model previously had received less validation than the other models and therefore was validated by independent sets of data (12 epidemics that had developed in susceptible and moderately resistant cultivars over 2 yr). The early blight model and the previously validated late blight model were used to evaluate the contribution of each fungicide application in a weekly application schedule (common grower practice) relative to overall foliar disease suppression. For early blight, applications beginning at 6–7 wk after planting were the first to make a positive contribution to disease suppression. This timing was consistent regardless of the inoculation date or the susceptibility group of the cultivar. For potato late blight, sprays applied at or just after the date of inoculation were important for suppressing the disease. The date at which sprays can be safely terminated (date of the last application) was similar for both diseases: approximately 3 wk before vine kill. Based on these results, we developed a fungicide use strategy with the goal of reducing the number of sprays while maintaining a low risk of severe disease caused by either pathogen. Our results indicated that using the proposed strategy may save two to four sprays in the northeastern United States without increasing risks from inadequate control.

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

Only two foliar diseases caused by fungi are of significant economic importance in potato (*Solanum tuberosum* L.) production in the northeastern United States: early blight, caused by *Alternaria solani* (Ellis & Martin) Jones & Grout, and late blight, caused by *Phytophthora infestans* (Mont.) de Bary. Even though epidemics are not severe every year, growers must consider the risk of their occurrence and traditionally have used applications of protectant fungicides as an "insurance policy." Initiation of a spray program when plants are 15–20 cm in height has been recommended (10). Plants generally reach this height about early to mid-June depending on the cultivar and the planting date. Sprays then are applied once a week until vine kill (10). Typically, seven to ten sprays of protectant fungicides are applied during a growing season.

Concern about the environment, health risks associated with pesticide use, and the increasing costs of applications have stimulated efforts to reduce the number of fungicide applications. The use of forecast systems to assist in the timing of fungicide applications seemed to be an appropriate solution because fungicides would be applied only when they were needed most (15). Unfortunately, commercial potato growers have not used forecast systems extensively because of the potential risk of mistakes in disease management and the relatively high value of the crop (15). Another factor preventing wide-scale adoption of such a system is that forecasts for one disease typically ignore other diseases; even if the target disease is suppressed adequately, other diseases may not be suppressed as well (12,17).

Previous recommendations have not determined the importance of individual applications of protectant fungicides in a season to overall disease suppression. Krause et al (13), in introducing BLITECAST (forecast system for late blight), emphasized that "all spray applications are *critical* and must be applied to protect against subsequent inoculum buildup." Similarly, other forecast systems (for example, FAST—forecast system for early blight on tomato [16]), as well as the traditional recommendations (10), have not determined the importance of different sprays. The logical conclusion, therefore, is that each spray has the same importance in controlling disease. The implication is that equal risks are involved if a spray is skipped at any point during the entire growing season. In contrast, we hypothesize that the importance of individual sprays changes during the season, and that the risks associated with skipping sprays differ during the season. To test this hypothesis, we have used simulation models that predict disease development and include fungicide dynamics. Such models can be used to investigate questions concerning disease management (8). A model describing the development of early blight was completed recently (18). However, its predictions had not been examined previously with independent sets of data.

This work had two major purposes. The first was to identify the importance of each spray during the season. The second was to develop a low-risk strategy for reducing the number of fungicide applications used in controlling early and late blight in potatoes. Because these goals required validated simulation models and full understanding of these two diseases, a preliminary goal was to validate the early blight model by independent sets of data.

MATERIALS AND METHODS

Validation of the early blight model. Certified seed potatoes were planted on 28 May 1986 (cultivar Norchip) and 1 June 1987 (cultivars Norchip and Katahdin). Norchip is susceptible and Katahdin is moderately resistant to early blight. Seeds consisted of small whole tubers or pieces of tubers, each weighing about 50 g. Fertilizer (175 kg of nitrogen, 175 kg of phosphorus, and 175 kg of potassium/ha) and insecticide (carbofuran, 3.4 kg a.i./ha, in 1986, FMC Corp. Fresno, CA) were applied at planting. Herbicide (linuron 50WP, 1.7 kg a.i./ha, DuPont Chemical Corp.

Wilmington, DE) was applied after planting but before plant emergence each year. Insecticides were applied to the foliage during both seasons as needed. Fungicides were applied hydraulically by a tractor-mounted boom. Metalaxyl 2EC (0.28 kg a.i./ha, Ciba-Geigy Corp., Greensboro, NC) was applied each year at the second week of August to all plots to prevent late blight development. Chlorothalonil (the tested fungicide) was applied as Bravo 500 (1.3 kg a.i./ha, Diamond Shamrock Corp., Painesville, OH) in 1986 or as Bravo 720 (0.84 kg a.i./ha, Diamond Shamrock Corp.) in 1987. Plants were hilled only in the 1987 experiment.

Four-row plots (16 plants/row), 0.9 m between rows and 3.7 m long, were planted at about 23-cm spacing within rows. Plots were separated by a fallow area about 4 m wide. Experimental plots were planted in a completely randomized design with four replications per treatment at the Homer C. Thompson Research Farm at Freeville, NY. Treatments consisted of different timings of fungicide (chlorothalonil) applications. Six different schedules were applied in 1986 and four were applied in 1987 (Table 1). Plots were inoculated on 3 July 1986 by sprinkling 500–600 ml of inoculum mixture evenly over each plot. The inoculum mixture consisted of infected dried potato stems and leaves, collected in the previous year after vine kill, mixed with peat and vermiculite. Plants were not artificially inoculated in 1987.

Disease was assessed visually. In 1986 the proportion of defoliation was estimated by dividing each plot into four quadrants, estimating the disease in each quadrant, and calculating the mean. In 1987, disease severity was assessed as follows. First the area of each leaf on four randomly selected stems per plot was estimated nondestructively by measuring the length of each leaf blade and deriving the area of each leaf with a cultivar-specific prediction equation (18). Then the proportion of the leaf area diseased was assessed using standard diagrams. The leaf area per leaf was multiplied by the disease proportion to yield the diseased area per leaf. Whole-stem leaf area and diseased area were obtained by summing the observations and were used to derive whole-stem disease severity. Finally, the mean severity per plot was calculated as the mean of the four stems.

Disease was assessed in both years every 6–10 days, starting in late June and ending in early to mid-September. Every plot was scored independently by two persons to limit subjectivity errors. For some analyses, the area under the disease progress curve (AUDPC) as calculated by Shaner and Finney (20) was used. The duration of the period used for calculating AUDPC was from the date of inoculation (or date of first appearance of disease) until the end of the growing season. AUDPC units are proportion days.

Daily weather data (minimum and maximum temperature, hours of relative humidity greater than 90%, minimal relative humidity, and precipitation) were needed as input for the early blight model. Relative humidity and temperature were monitored from the date of median emergence to vine kill with a hygrothermograph placed in a standard weather shelter located within the plant canopy. Rainfall was recorded from planting date to vine kill with a rain gauge. Median emergence was estimated by linear interpolation of emergence counts over time. It occurred in the 1986 experiment on 17 June (20 days after planting) and in 1987 on 17 June for cultivar Norchip or 19 June for cultivar Katahdin (16 or 18 days after planting, respectively). The initial disease of each simulation was that level of disease first visible in the field. In 1986, initial disease was estimated (by backwards extrapolation of disease progress curve) to be one lesion per plant, 46 days after planting. In 1987, it was one lesion per 10 plants at 56 or 63 days after planting for cultivar Norchip or Katahdin, respectively.

TABLE 1. Schedules of fungicide applications in field experiments in 1986 and 1987 for validating the early blight model[a]

Year	Cultivar	No. of sprays	Date of application
1986	Norchip	0	
		7	7/7 7/17 7/22 7/29 8/5 8/12 8/19 8/26
		2	7/17 7/22
		2	7/22 7/29
		2	7/29 8/5
		2	8/5 8/12
1987	Norchip	0	
		7	7/22 7/28 8/6 8/12 8/19 8/26 9/3
		2	7/22 7/28
		2	8/19 8/26
	Katahdin	0	
		7	7/22 7/28 8/6 8/12 8/19 8/26 9/3

[a]Chlorothalonil was applied formulated as Bravo 500 (1.3 kg a.i./ha) in 1986 and as Bravo 720 (0.84 kg a.i./ha) in 1987.

Simulation experiments. A model that simulates the effects of environment, host growth, cultivar resistance, and fungicide (chlorothalonil) on epidemics of potato early blight (*A. solani*) was developed recently (18). The model operates on a daily time step and is driven by minimum and maximum temperature, minimum relative humidity, hours of relative humidity greater than 90%, and precipitation. Host growth and disease development are simulated in each 15-cm canopy stratum. Plant age and leaf position effects on receptivity, incubation period, and lesion expansion rate are calculated daily for each new cohort of colonies. The version we used was translated from FORTRAN IV (the original version) to the "C" language, occupying about 1,200 programmed lines and 340 variables and parameters.

The late blight model (also written in "C") describes the development of *P. infestans* (3), the initial deposition of the fungicide chlorothalonil (5), and its subsequent weathering, redistribution, loss, and efficiency (4). The version we used had been modified and improved by changing some parameters based on recent field results and correcting programming errors (M. A. Doster and W. E. Fry, *unpublished*). Both models were operated on an IBM-PC 640K microcomputer equipped with a "C" compiler (International Business Machines Corp., Armonk, NY).

The length of the season (from the date of planting until the end of the season) used in the simulation experiments was 102 days. Median emergence occurred 18 days after planting. For evaluating the effects of inoculation dates on disease development, inoculation occurred (for both pathogens) in different runs 25, 39, 53, or 67 days (4, 6, 8, or 10 wk) after planting. The initial disease of early or late blight was one lesion per 10 plants. The protectant fungicide chlorothalonil was applied at the rate of 1.34 kg a.i./ha. Four years of meteorological data (1984–1987) recorded at Freeville, NY, were used in all runs. Simulations were done with a susceptible cultivar (for example, Norchip) and a moderately resistant cultivar (for example, Katahdin). In the reference run, a protectant fungicide was applied weekly from the date of inoculation until a week before the end of the season.

The AUDPCs (calculated from the daily simulated severities) of weekly sprayed plots (Aw) and untreated plots (Au) were used to evaluate the control efficiency achieved by applying weekly sprays (Cw). Thus, a higher control efficiency is associated with more effective disease suppression.

$$Cw = 1 - Aw/Au \quad (1)$$

In some sets of runs, sprays were eliminated sequentially from the end of the season. The control efficiency for runs where n sprays were applied (Cn) was calculated using the AUDPC of n sprays (An) and untreated plots (Au) as:

$$Cn = 1 - An/Au \quad (2)$$

The relative contribution of an individual spray may be evaluated by its marginal contribution (= marginal return) in suppressing the epidemic. The marginal return of the i^{th} spray (Mi) was calculated as:

$$Mi = (Cn - Cn-1)/Cw * 100 \quad (3)$$

In other sets of runs, sprays were eliminated sequentially from the beginning of the season. The values of Cn and Mi were calculated as described above.

RESULTS

Validation of the early blight model. The 12 different epidemics in susceptible (Norchip) or moderately resistant (Katahdin) cultivars (Table 1) indicated that model predictions were similar to reality (Fig. 1). Disease developed earlier in 1986 than in 1987 in both predicted and observed epidemics. In both years, disease was delayed by host resistance and protectant fungicide. High correlation between observed and predicted epidemics was obtained in untreated or fully protected plots (Fig. 1). Similar agreement between observed and predicted values for the other six treatments was observed (not shown). The AUDPC was used to evaluate the model's predictions for the entire season of all 12 epidemics simultaneously (Fig. 2). The theoretical line indicating a perfect coincidence between predicted and observed AUDPC had an intercept of 0 and slope of 1. The observed regression equation describing the fit between predicted and observed values has an intercept (−1.56) that was not significantly different from 0 ($P = 0.05$) and a slope (1.06) that was not significantly different from 1 ($P = 0.05$).

Simulation experiments. Simulated epidemics using the weather data from 1984 and 1985 illustrate the effects of inoculation dates and host resistance on the development of early and late blight (Fig. 3). The weather in 1984 was more conducive for early blight than the weather in 1985. Moderate resistance delayed the epidemic and in most cases reduced the rate of disease development relative to susceptible cultivars (Fig. 3). As the crop aged, the time interval between initial inoculation and 1% defoliation decreased. This interval (mean of 1984 and 1985) was 26, 16, 14, or 8.5 days for inoculation occurring 25, 39, 53, or 67 days after planting, respectively, for the susceptible cultivar (Fig. 3). This interval was 34, 25, 18.5, or 12 days for the moderately resistant cultivar (Fig. 3).

For late blight, the intervals between initial inoculation and 1% defoliation ranged from 22 to 28 days and was not affected as the crop aged (Fig. 4). The simulation model does not yet reflect the slight increase in host susceptibility as the plants age. Moderate resistance, however, delayed the onset of epidemics relative to that of the susceptible cultivar (Fig. 4).

As expected, the AUDPC decreased as the inoculation date was delayed and was lower in moderately resistant cultivars relative to susceptible ones, for both early and late blight (Table 2). Weekly applications of fungicide, initiated at the inoculation date and terminating 1 wk before the end of the season, reduced the AUDPC values (Table 2). The control efficiency achieved by fungicides for late blight was generally greater than that for early blight (Fig. 5). The control efficiency for each disease decreased as the epidemic in untreated plots became more severe.

The marginal return of specific spray applications for either early blight or late blight suppression was influenced by the date of inoculation but not by the resistance of the cultivar (Figs. 6 and 7). For early blight, when the date of initial inoculation was early (4 or 6 wk after planting), the marginal return of early and late sprays was low, whereas the marginal return of the middle sprays was high. Some of the very early sprays had a negative contribution to disease suppression, indicating that applying those sprays resulted in a more severe epidemic than that which developed in an untreated crop (Fig. 6). When inoculations occurred mid-season (8 or 10 wk after planting), sprays applied before this date had no effect or just a small effect in suppressing the epidemic. At this point in the season, the most important spray was the one applied at the inoculation date. The marginal return of subsequent sprays diminished successively (Fig 6).

For late blight, the magnitude of the marginal return of each fungicide application was affected by the length of the interval between the date of inoculation and the end of the growing season. Sprays applied before the date of inoculation as well as those applied late in the season were less important than those applied at or immediately after the date of inoculation (Fig. 7). Similar results to those presented in Figures 6 and 7 were obtained in simulation experiments in which sprays were eliminated sequentially from the beginning of the season (results not shown).

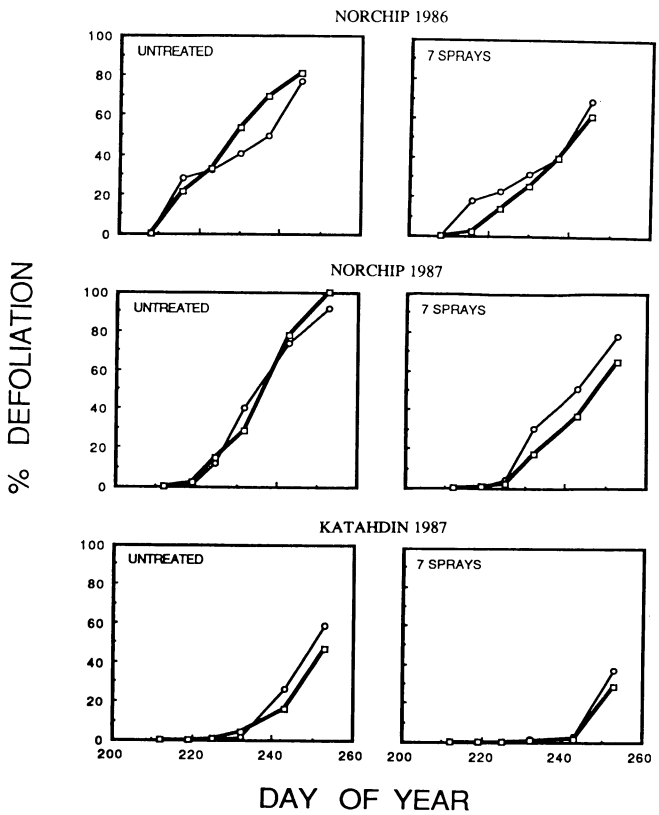

Fig. 1. Early blight progress curves as predicted by the simulation model (squares) or observed in field experiments (circles) in two cultivars, growing seasons, and fungicide treatments.

DISCUSSION

Predictions of the early blight model compared favorably with the field observations (Fig. 1). Such visual comparisons can be

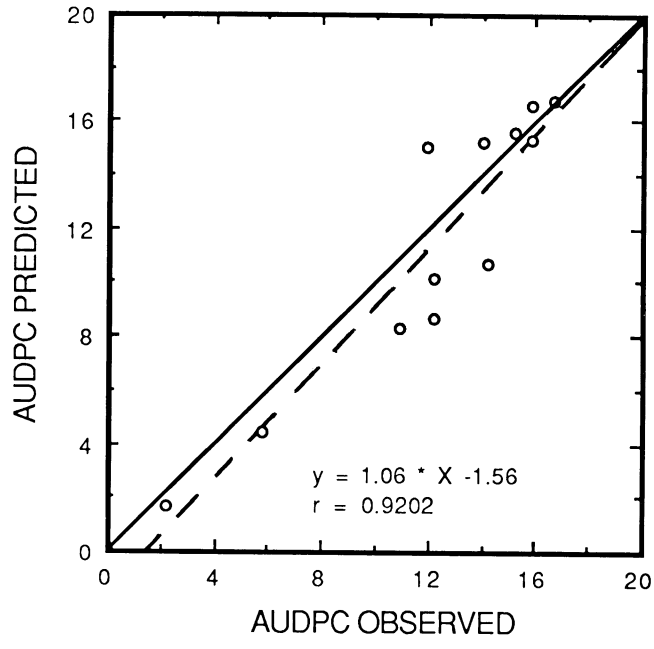

Fig. 2. Model predictions of the area under the disease progress curves (AUDPC) for potato early blight compared with actual values observed in field experiments. The dashed line indicates the regression equation describing the fit between observed and predicted values. The solid line indicates the relationship for perfect coincidence between observed and predicted values.

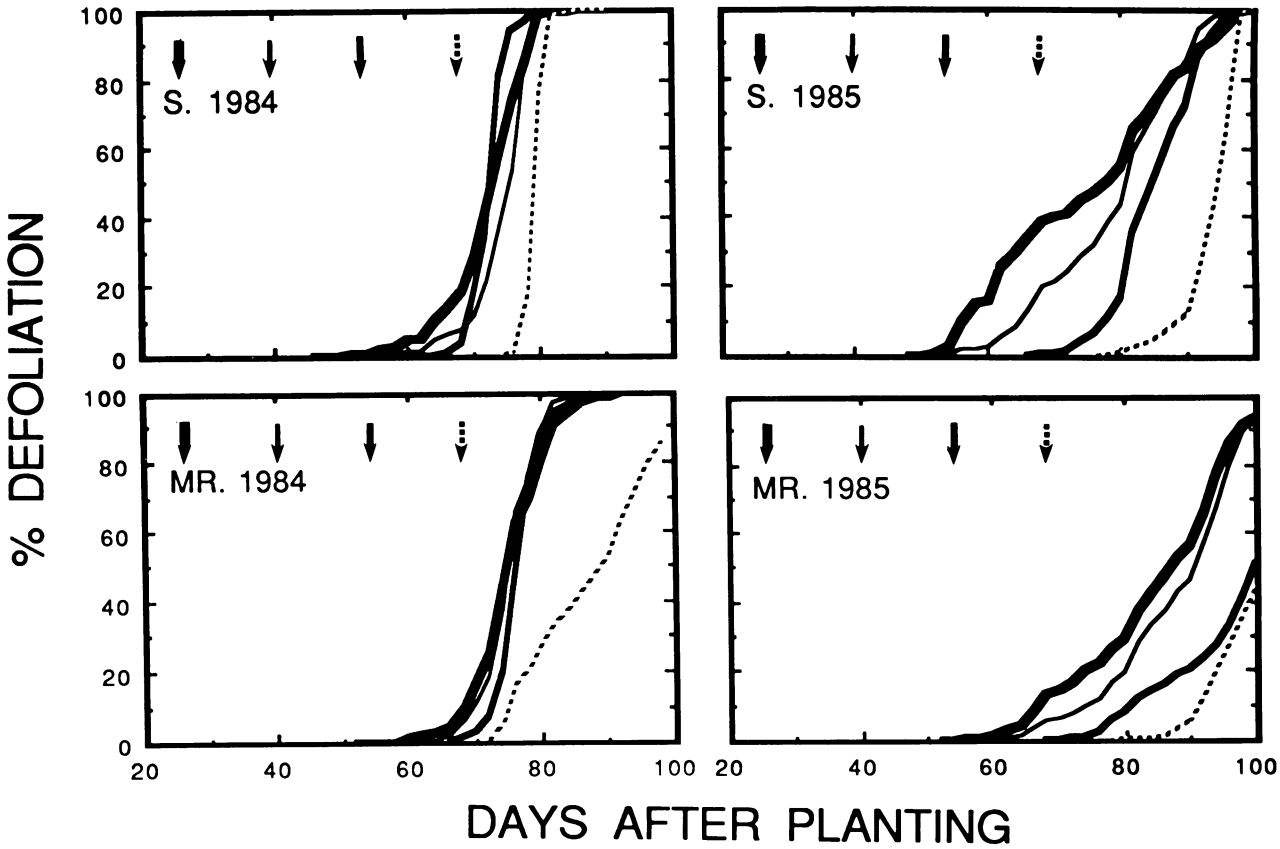

Fig. 3. Computer simulations of early blight epidemics in susceptible (S) and moderately resistant (MR) cultivars in two growing seasons using four different inoculation dates. Inoculation dates are indicated by arrows: from left to right, 25, 39, 53, 67 days after planting.

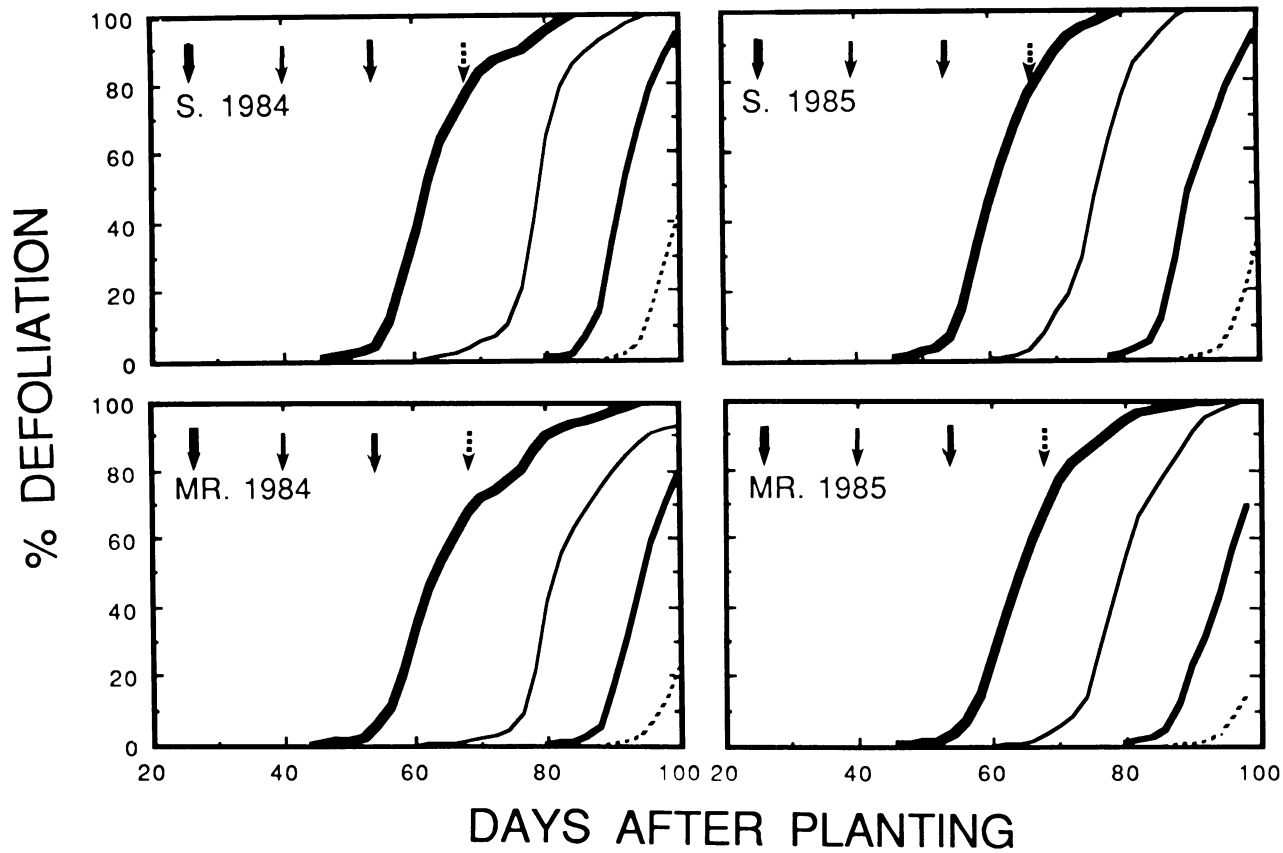

Fig. 4. Computer simulations of late blight epidemics in susceptible (S) and moderately resistant (MR) cultivars in two growing seasons using four different inoculation dates. Inoculation dates are indicated by arrows: from left to right, 25, 39, 53, or 67 days after planting.

more critical than some statistical tests (21). In addition, while relating the observed and predicted severities (expressed as AUDPC), we found that they were not significantly different (the intercept was not different from 0 and the slope was not different from 1, $P = 0.05$). The AUDPC was used for statistical comparison because of problems inherent with the use of final disease level or apparent infection rate in field experiments (9).

TABLE 2. Effects of early blight or late blight, host resistance, inoculation date, and protectant fungicide on the area under the disease progress curve (AUDPC) in simulation experiments[a]

Inoculation date[b]	Fungicide treatment[c]	Early blight		Late blight	
		S[d]	MR[d]	S	MR
25	Untreated	30.3	23.3	40.0	36.6
		(2.5)	(3.4)	(0.3)	(0.1)
	Sprayed	23.3	10.3	23.0	15.7
		(2.9)	(2.8)	(1.1)	(1.0)
39	Untreated	27.9	19.5	26.6	22.9
		(2.1)	(4.2)	(1.3)	(1.6)
	Sprayed	20.8	8.7	9.9	4.6
		(2.8)	(2.5)	(0.5)	(0.4)
53	Untreated	23.8	13.6	12.2	9.0
		(3.3)	(3.3)	(0.9)	(0.9)
	Sprayed	13.9	5.8	1.3	0.5
		(3.4)	(3.0)	(0.5)	(0.2)
67	Untreated	7.4	2.5	1.5	0.8
		(2.2)	(0.4)	(0.5)	(0.3)
	Sprayed	1.1	0.1	0.05	0.02
		(0.1)	(0.07)	(0.02)	(0.01)

[a] AUDPC was calculated from the day of inoculation until the end of the growing season. Results are the mean of four growing seasons. Numbers in brackets are the standard error.
[b] Days after planting.
[c] Fungicide (chlorothalonil at the rate of 1.34 kg a.i./ha) applied weekly starting on the day of inoculation and terminating 1 wk before the end of the growing season.
[d] S = susceptible cultivar; MR = moderately resistant cultivar.

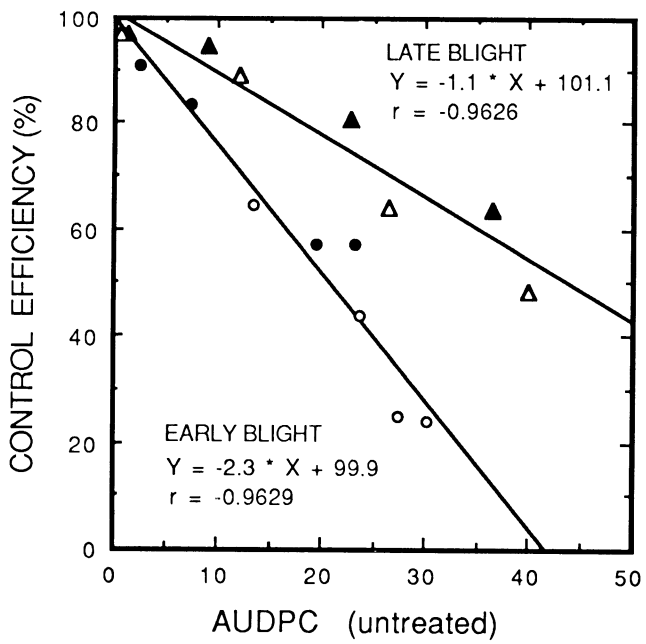

Fig. 5. Effect of potential disease (expressed as area under the disease progress curve [AUDPC] in untreated plots) on the control efficiency achieved by weekly applications of a protectant fungicide. The spray program was initiated at the date of inoculation and terminated 1 wk before the end of the growing season. Simulations were conducted by using the early blight (circles) or the late blight (triangles) models. Points are results of four inoculation dates involving a susceptible (open symbols) or a moderately resistant (solid symbols) cultivar. Each data point is a mean of simulated epidemics of four growing seasons.

Although the early blight model is not an exact replica of the real system, we found that it predicted epidemic development sufficiently well to be used in our investigation of disease management strategies. The simulations for early blight (Fig. 3) correctly indicated that rapid epidemics developed only after the plants became susceptible (7,11,12,18). Regardless of the date of inoculation in our simulation experiments, applications made

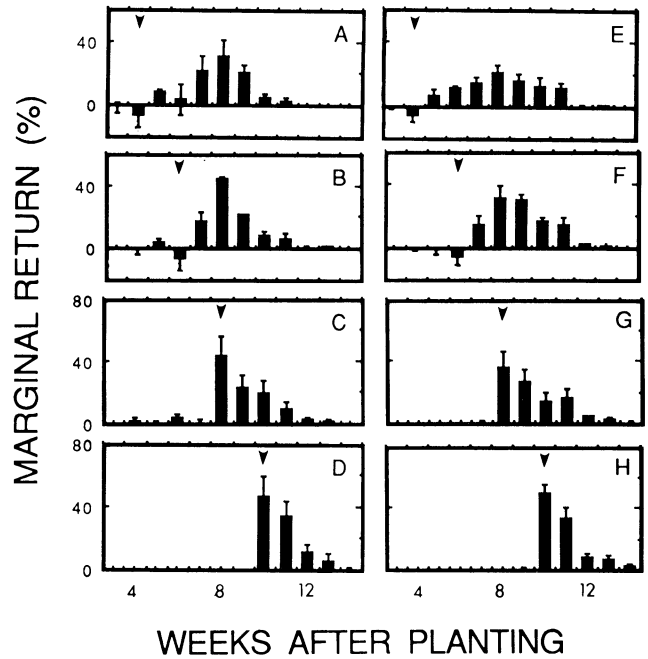

Fig. 6. Marginal return values of individual sprays in suppressing early blight epidemics as influenced by four different inoculation dates (indicated by arrows). **A-D,** Susceptible cultivars. **E-H,** Moderately resistant cultivars. Results represent sprays applied at that time and are means of simulated epidemics of four growing seasons. Bars indicate the standard error.

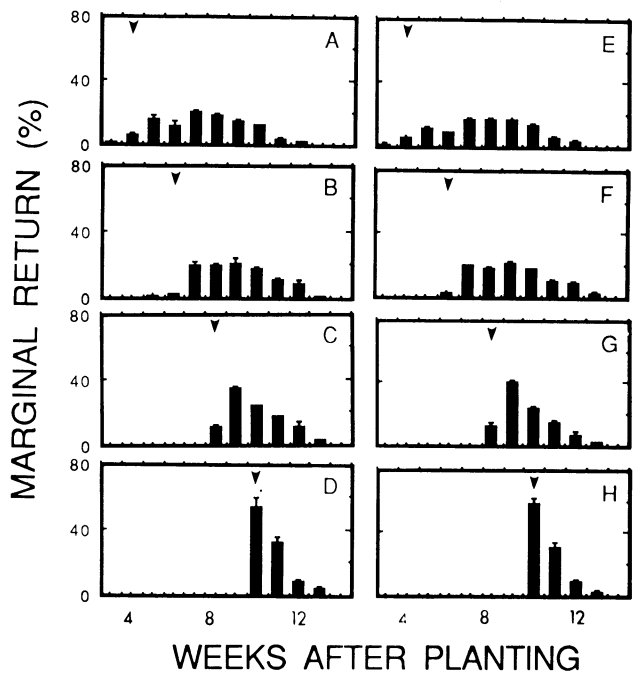

Fig. 7. Marginal return values of individual sprays in suppressing late blight epidemics as influenced by four different inoculation dates (indicated by arrows). **A-D,** Susceptible cultivars. **E-H,** Moderately resistant cultivars. Results represent sprays applied at that time and are means of simulated epidemics of four growing seasons. Bars indicate the standard error.

early in the season were not important in early blight suppression (Fig. 6). In fact, we were surprised to find that early sprays resulted in negative marginal return values, indicating that they intensified disease. However, this simulation result is consistent with experimental results. Nutter and MacHardy (17) found higher disease in field plots that were treated early than in plots that were not treated at all. Some attempts have been made to explain this phenomenon theoretically (1,17), but resolution of the issue awaits experimental evaluation.

We concluded that the first spray for early blight suppression in the northeastern United States should be applied not earlier than 6–7 wk after planting. This initiation date should provide adequate suppression of the disease and may save one to two sprays (compared with standard practice) without increasing risks. Because the marginal return of fungicides was similar in cultivars susceptible to early blight and those moderately resistant to early blight (Fig. 6), we believe that sprays should be initiated at the same time of the season for both types of cultivars. Other researchers working with field experiments also concluded that the first spray against early blight should not be applied early in the season. However, the exact date after planting for initiating control has a wide range: 48 days in New Hampshire (17), 56 days in Colorado (12), 69 days in south central Idaho (6), about 70 days in Wisconsin (19), and 81 days after planting in eastern Idaho (6). Thus, the optimal date for initiating control should be found experimentally wherever needed.

The situation for the initial late blight sprays is different from the situation for early blight. The precise date of late blight inoculation influenced the importance of sprays (Fig. 7). Therefore, because the natural occurrence of late blight is more sporadic than that of early blight, initiation of sprays should be recommended separately for each situation. Dates of initial infection may be predicted by a forecast system, perhaps integrated with a scouting program.

For both diseases and resistance groups, the importance of sprays diminished toward the end of the growing season. Sprays applied 7–14 days before the end of the growing season had only a small contribution to the overall disease suppression (Figs. 6 and 7). Thus, we concluded that applying the last spray approximately 3 wk before vine kill would not affect the control efficiency substantially and would save another two sprays.

Based on the above analysis, we propose the following strategy to control early blight and/or late blight in potatoes. Sprays should be initiated when late blight is predicted according to a forecast system (that is, BLITECAST [13]) or 6–7 wk after planting (whichever comes first). Subsequent spray intervals would be determined according to the late blight resistance group of the cultivar (10) during the first 6–7 wk after planting, and in a weekly schedule after 7 wk. The last spray should be applied approximately 3 wk before vine kill. The use of this strategy should save up to two to four sprays without substantially increasing disease in the northeastern United States.

An important factor in late blight management but not yet included in our proposed strategy is a consideration of tuber infections. Sporangia washed from foliar lesions are the inoculum for these infections (14). Because high concentrations of chlorothalonil suppressed the viability of sporangia produced from treated foliage in greenhouse studies (2), early termination of a spraying program may increase the number of sporangia even if the proportion of tissue affected is not measurably affected. Adoption of our proposed strategy awaits evaluation of the impact of potential tuber infections.

This proposed strategy is different in principle from other disease management strategies for potatoes. Previous forecasts attempted to reduce the number of sprays for diseases individually without attempting to characterize risk (13,19). The traditional schedule (weekly) aimed to minimize the risks without considering the number of applications (10). The main goal of the strategy proposed here is to reduce the number of sprays while considering both diseases and without increasing risks. The criteria used to develop the strategy proposed in this work are applicable in any host-parasite system. We believe that they are most suitable to intensive crops, where risks associated with inadequate control may be more influential than the benefits of saving the costs of a few sprays.

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