Ecology and Epidemiology

A Model for the Effects of Metalaxyl on Potato Late Blight Epidemics

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ABSTRACT


Dose-response relationships of Phytophthora infestans to metalaxyl and a description of metalaxyl residue dynamics were incorporated into an existing simulation model of potato late blight. The existing model was modified to include the effects of metalaxyl on lesion establishment, lesion expansion, and sporulation. The model was used to simulate epidemics when metalaxyl was applied under a variety of conditions and compared with observed epidemics in the field. Model predictions for area under the disease progress curve (AUDPC) and final disease level (Ye) were almost always within the range of observed values from the field epidemics. The model predicted well for a variety of metalaxyl treatments, including beginning metalaxyl applications when disease reached levels between 0 and 10%, different doses of metalaxyl, and metalaxyl applied to both susceptible and resistant potato cultivars. Development of a model that realistically simulates the effects of metalaxyl on late blight epidemics may allow the identification of optimal use of metalaxyl for late blight control.

Additional key word: systemic fungicides.

The systemic fungicide metalaxyl is a highly effective fungicide for controlling potato late blight, caused by Phytophthora infestans (Mont.) de Bary, and other diseases caused by Oomycetes. Because of its systemic and eradicant properties, metalaxyl has the potential for dramatically changing late blight management, which previously has relied heavily on protectant fungicides. Ideally, a systemic fungicide, such as metalaxyl, should be integrated into a disease management program to optimize fungicide use. Optimal and efficient use of metalaxyl is also an important goal because of the threat of metalaxyl resistance. Metalaxyl resistance has evolved in P. infestans in Europe and elsewhere (3,5,7,17,26) but has not yet been documented in North America. Management tactics that minimize metalaxyl use while still ensuring adequate disease control are important for reducing the risk of a resistance problem in North America.

One approach to optimizing fungicide use is by simulation modeling. Disease management tactics for the efficient use of protectant fungicides for controlling late blight have been identified (12,31) using a simulation model (2). The key feature in a modeling approach to fungicide optimization is to accurately model the effects of a fungicide on the pathogen life cycle and, consequently, on epidemic development. However, modeling the effects of metalaxyl on late blight epidemics is not as simple as...
modeling protectant fungicides. Unlike protectant fungicides that only inhibit spore germation and infection, metalaxyl affects *P. infestans* by inhibiting lesion establishment, lesion expansion, and sporangium production and viability (1,6). Metalaxyl can also eradicate or inhibit the fungus in lesions that are present when it is applied (1,6,32). Thus, the structure of Bruhn and Fry’s model (2) must be altered substantially to accommodate the modifications necessary to include metalaxyl.

Our primary objective for this research, therefore, was to model the effects of metalaxyl on *P. infestans* and late blight epidemics. In a previous paper, we described an empirical model for the distribution and temporal dynamics of metalaxyl residues in the potato canopy (24). In this paper, the metalaxyl residue model is linked to the late blight simulation model of Bruhn and Fry (2). We begin by briefly describing how we modified Bruhn and Fry’s model to incorporate the effects of metalaxyl on the pathogen. Some of the data for estimating model parameters were available in the literature (1,6). However, we also conducted experiments to provide additional data for the model. The final sections compare the model predictions to observed field epidemics.

**MATERIALS AND METHODS**

**Model description.** The late blight model developed by Bruhn and Fry (2) operates on a daily time-step and simulates the effects of environment, host resistance, and protectant fungicides on the asexual development of *P. infestans*. Model predictions under a variety of conditions compared well to epidemics observed in field experiments (2,12,31). The notation in the following model description is only slightly different from that of the original model (Table 1) (2).

Stages in the life cycle of *P. infestans* are divided into 15 discrete age classes of 1-day duration. The numbers of lesions and the average lesion area for each age class are denoted by vectors *n* and *m*, respectively. New lesions at time *t* are established by multiplying the probability of infection (establishment), *p_1*, times the total number of sporangia that land on susceptible plant tissue. Lesions in age classes 1 to *j*−1 are presymptomatic, where *j* is the length (in days) of the incubation and latent period. Lesions in age classes *j* to 9 expand at a rate *g* (square millimeter per day). Lesions in age classes 9 to 15 produce sporangia at a rate *z* (per square millimeter of lesion area). The rates *g*, *z*, and probability *p_1* are functions of environment and host resistance; *p_1* is also a function of protectant fungicide residues (2).

Modifications have been made to *g*, *z*, and *p_1* as defined by Bruhn and Fry (2) to reflect the effects of metalaxyl on each of these parameters. The effects of metalaxyl on lesion establishment are modeled by modifying the parameter *p_1*. Metalaxyl affects lesion establishment only after infection, when the fungus has penetrated into the leaf tissue (1,33). Assuming that the effects of metalaxyl on establishment are independent of infection, the probability of infection and lesion establishment when metalaxyl is present is

\[ p_1' = p_1 \cdot E[h(X_i)] \]  

(1)

in which the prime denotes the presence of metalaxyl, and *E[h(X_i)]* is the expected probability of lesion establishment as a function of metalaxyl residues, *X_i* in the leaf at time *t*. *X_i* is assumed to be a random variable that is lognormally distributed (24); therefore, *h(X_i)* is also a random variable.

Lesion expansion is also affected by metalaxyl and is modeled by modifying the lesion expansion rate, *g*. Because the effects of metalaxyl on lesion expansion vary with lesion age at the time metalaxyl is applied (1,6), *g* varies among the lesion cohorts in relation to the time of application (all lesions in the same age class are a cohort).

\[ g' = g \cdot f(s) \]  

(2)

in which *g'*, is the lesion expansion rate in the presence of metalaxyl for the *r*th cohort at time *t*, and *f(s)* is a function that describes the reduction in expansion rate for each cohort. Lesion expansion rate is only weakly dependent on the dose of metalaxyl applied (6), and, therefore, *f(s)* is assumed to be independent of dose.

The effects of metalaxyl on sporulation rate, *z*, depend on dose applied and the lesion age at time of application,

\[ z' = z \cdot k(s,d) \]  

(3)

in which *z'* is the sporulation rate for the *r*th cohort at time *t* as a function of the dose of metalaxyl applied, *d*, *k(s,d)* is a function that describes the reduction in sporulation rate as a function of *s* and *d*. Values for this function were determined from previously published data (1).

**Experiments to estimate model parameters.** Lesion establishment as a function of metalaxyl residues in potato leaves was determined using the “most probable number” (MPN) technique (27). This technique was originally used for estimating microbial population densities by determining the presence or absence of colony-forming units in serial dilutions of a sample (4). Plender et al (27) modified this technique in order to estimate the density of infective propagules of *Aphanomyces eutiches* in soil. They scored for infected plants in different dilutions of infested soil. In this study, we estimated the number of infective sporangia at each metalaxyl concentration by scoring for lesion establishment in dilutions of known total numbers of sporangia.

The experiments were done using a floating leaf disk assay (3) with metalaxyl concentrations from 0 to 0.20 mg a.i./ml (25 WP formulation of metalaxyl was obtained from Ciba-Geigy Corp., Greensboro, NC). Leaf disks (22.5-mm diameter) were cut from 6-wk-old, greenhouse-grown potato plants (cultivar Norchip) and

| TABLE 1. List of symbols used in the simulation model for the effects of metalaxyl on potato late blight epidemics.
<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
</tr>
</thead>
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<tr>
<td><em>t</em></td>
<td>Time (days) since inoculation</td>
</tr>
<tr>
<td><em>n</em></td>
<td>Vector for number of lesions in age classes 1−15 on day <em>t</em></td>
</tr>
<tr>
<td><em>n_1</em></td>
<td>Number of new lesions (age class 1) that established on day <em>t</em></td>
</tr>
<tr>
<td><em>m</em></td>
<td>Vector for average lesion area in age classes 1−15 on day <em>t</em></td>
</tr>
<tr>
<td><em>p_1</em></td>
<td>Probability of infection and establishment on day <em>t</em> (independent of metalaxyl)</td>
</tr>
<tr>
<td><em>s</em></td>
<td>Length (in days) of the incubation and latent period</td>
</tr>
<tr>
<td><em>g_1</em></td>
<td>Lesion expansion rate (mm day−1)</td>
</tr>
<tr>
<td><em>z_1</em></td>
<td>Sporulation rate (mm day−1)</td>
</tr>
<tr>
<td><em>f_1</em></td>
<td>Probability of infection and establishment on day <em>t</em> as a function of metalaxyl</td>
</tr>
<tr>
<td><em>X_i</em></td>
<td>Metalaxyl residues (mg cm−2 of leaf tissue)</td>
</tr>
<tr>
<td><em>X'_i</em></td>
<td>Metalaxyl residues (mg cm−2 of leaf tissue)</td>
</tr>
<tr>
<td><em>h(X)_i</em></td>
<td>Proportion of successful infections that establish new lesions, as a function of metalaxyl residues</td>
</tr>
</tbody>
</table>

*The notation is only slightly different from that of Bruhn and Fry (2) from which this model was derived. Symbols are listed in the order in which they appear in the model description in the text.*
The regression equation was used for extrapolation to estimate residues in the lesion establishment experiment. Direct measurements of metalaxyl was not possible below 0.2 μg/ml because of the detection limit of the bioassay used (23, 24).

Lesion expansion in response to metalaxyl was determined using detached leaflets from 6-wk-old, greenhouse-grown potato plants (cultivar Norchip). Each leaflet was inoculated on the abaxial leaf surface with a 50-μl droplet of a sporangial suspension containing 400 sporangia (8 × 10^3 sporangia per milliliter). Leaflets were incubated at 18°C in low light (750 erg/cm^2/sec) in inverted water agar petri dishes such that the leaf did not touch the agar. Metalaxyl was applied by dipping inoculated leaflets for 5 sec in suspensions of metalaxyl (25 WP formulation) with concentrations of 0, 1, 5, 10, 15, and 25 μg a.i./ml. Four leaflets were treated with each concentration each day from 1 to 5 days after inoculation. Lesion diameters were measured at least twice on each leaflet after metalaxyl application. Lesion expansion rate (millimeter per day increase in diameter) was determined for each leaflet and analyzed using a two-way analysis of variance for metalaxyl dose and days after inoculation. Data for modeling the effects of metalaxyl on lesion expansion when establishment occurred after metalaxyl was applied were obtained from Bruck et al (1).

Conversions were also made from metalaxyl concentrations (micrograms per milliliter), in which leaflets were dipped, to residues in nanograms per square centimeter. Previous conversions were for leaf disks floated on metalaxyl for 24 hr. Metalaxyl residues were determined by bioassay (23) for leaflets dipped in 1, 5, 10, and 25 μg/ml. Each leaflet was rinsed for 5 sec, allowed to dry for 1 hr, and then rinsed in distilled water to remove some of the external residues. Residues can be quantified more accurately for intermediate levels of residue (23), such as those encountered after washing off some of the external residues, than for high levels found just after dipping leaflets in metalaxyl. Ten leaflets were assayed for each concentration.

**Model performance.** Late blight epidemics were monitored in 4 × 4-m field plots under a variety of metalaxyl application regimes in 1977, 1978, and 1979. Data from 1977 and 1978 have been published previously (13–15). Field experiments in 1979 were conducted under very similar cultural and experimental conditions as in 1977 and 1978 but have not been reported previously. Plots of the susceptible potato cultivar Hudson were arranged in a randomized complete block design with three blocks in all 3 yr. Hudson has similar susceptibility to late blight as the other cultivar Seago, which has rate-reducing resistance, was used for some experiments in 1979. When plants had reached nearly full size, in late July, a plant in the center of each plot was inoculated by spraying a sporangial suspension of *Phytophthora infestans* onto its foliage. In 1978, additional inoculum was provided by placing a potted plant with 10–20 lesions in the center of each plot (13). Plots were irrigated by overhead sprinkler irrigation at dawn and dusk for an approximate total of 0.25 cm of water per day. Environmental data were collected with a hygrothermograph and rain gauge located in the same field. Disease assessments were made every 3–7 days as described by Fry (10) through the first week of September. Model predictions of disease progress were compared to actual epidemics by comparing the area under the disease progress curve (AUDPC) (29) and the final proportion of disease (17).

In 1977 and in some plots in 1978, metalaxyl was applied before any disease was observed in the plots. In 1977 and 1978, metalaxyl applications began at about 0.5, 2–5, and 10% disease by beginning applications at different times after inoculation. Treatments in 1979 also varied in dose and spray intervals. Only one field experiment in 1977 was conducted in which metalaxyl was applied in a mixture with a protectant fungicide; the protectant fungicide chlorothalonil was used at half the normal field rate when mixed with metalaxyl (15).

Model simulations were done to correspond to field conditions by using recorded weather data and actual fungicide application dates (Table 3). Simulations were also conducted with a daily influx of inoculum to reflect field conditions in which there was inoculum blowing in from other experiments in the same field. The

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**Table 2. Lesion establishment in potato leaf disks by *Phytophthora infestans* as a function of metalaxyl concentration.**

<table>
<thead>
<tr>
<th>Sporangia applied per leaf disk (no.)</th>
<th>Metalaxyl(^#) (μg/ml)</th>
<th>1,000</th>
<th>400</th>
<th>200</th>
<th>100</th>
<th>50</th>
<th>20</th>
<th>10</th>
<th>5</th>
<th>MPN(^#) % MPN(^#)</th>
</tr>
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<tr>
<td>1</td>
<td></td>
<td>0.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
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</tr>
<tr>
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<td>11.41</td>
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<td></td>
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<tr>
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<td>0.00</td>
<td></td>
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<td></td>
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<td>0.005</td>
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<td>0.00</td>
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<td></td>
<td></td>
<td>23.06</td>
</tr>
<tr>
<td>0.01</td>
<td></td>
<td>0.00</td>
<td></td>
<td></td>
<td></td>
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<td>23.06</td>
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<td>0.10</td>
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<td>0.00</td>
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<td></td>
<td>23.06</td>
</tr>
</tbody>
</table>

\(^\#\) The effect of metalaxyl is estimated using the most probable number technique (MPN). This technique estimates the number of infective sporangia (from maximum numbers of 400 and 1,000 in experiments 1 and 2, respectively) that established lesions at each metalaxyl concentration. Different dilutions of sporangia were used for different metalaxyl concentrations to ensure that some leaf disks, but not all, would show symptoms at each concentration.

\(^\#\) Metalaxyl concentrations (μg/ml) on which leaf disks (22.5 mm diameter) were floated for 24 hr before inoculation. Disks were removed from metalaxyl 24 hr after inoculation, rinsed, and floated on distilled water for an additional 5 or 6 days.

\(^\#\) MPN was calculated using a FORTRAN program obtained from D. Rouse, Department of Plant Pathology, University of Wisconsin, Madison. MPN calculations were based on a maximum of 400 in experiment 1 and 1,000 in experiment 2.

\(^\#\) MPN was calculated by dividing MPN by the maximum number of sporangia in each experiment (400 or 1,000) and multiplied by 100 to get percentages. % MPN is the estimated percentage of the sporangia that can infect and establish lesions at each metalaxyl concentration; this is analogous to infection efficiency (18).

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daily influx varied according to the favorability of the weather for late blight. Disease favorability was expressed as “blight units” (BU), which was defined by Fry et al. (12). BU range from 0, in unfavorable conditions, to 6, in highly favorable conditions for disease development. Exogenous inoculum was arbitrarily simulated by an influx of \(10^6\) sporangia per day (9). Field epidemic data were independent of the data used to estimate or calibrate model parameters.

**RESULTS**

**Experimental results and parameter estimation.** Lesion establishment in response to metalaxyl was quantified using the MPN technique (Table 2). Estimates of the percentage of sporangia applied that established lesions (%MPN) when no metalaxyl was present are 1.75 and 2.55% for experiments 1 and 2, respectively (Table 2). These estimates are similar to those for infection efficiency on the cultivar, Norchip, using a different technique (18). Estimates for %MPN on metalaxyl-treated leaf disks were divided by the %MPN without metalaxyl in each experiment to get estimates of the proportion of establishment as a function of metalaxyl concentration, \(h(x)\). These proportions are shown plotted against metalaxyl concentrations that were converted to leaf residues (nanograms per square centimeter) in Figure 1. The relationship shown in Figure 1 can be linearized using a log-log transformation, resulting in the regression model

\[
\ln h(X) = \alpha + \beta \ln X_i + \epsilon_i
\]

where \(\alpha\) stands for the natural logarithm, and the \(\epsilon_i\)'s are independent, identically distributed random variables that are assumed to be normal with mean 0, and variance \(\sigma^2\).

Implicit in this log-log regression model is that the random variable \(h(X) / h(X)\) (given \(X\)) is distributed lognormal (\(\alpha + \beta \ln X, \sigma^2\)). Therefore,

\[
E[h(X) / X] = \exp(\alpha + \beta \ln X + \sigma^2/2)
\]

the expected value for a lognormal random variable (25). As \(X\) gets smaller, \(E[h(X) / X]\) becomes greater than 1.0 (because \(\beta\) is less than 0). Because we are modeling the proportion of lesion establishment as a function of metalaxyl residues, and therefore want to keep \(E[h(X) / X]\) from becoming greater than 1.0, the expectation is changed to

\[
E[h(X) / X] = \begin{cases} 
  x^\theta \cdot \exp(\alpha + \sigma^2/2) & \text{for } x_0 < x < \infty \\
  1.0 & \text{for } 0 < x \leq x_0
\end{cases}
\]

where \(x_0\) is the threshold level of metalaxyl residues at which \(E[h(X) / X] = 1.0\). Then, by iterated expectation (25),

\[
E[h(X)] = E[E[h(X) / X]]
\]

\[
= \int_0^\infty f_x(x) dx + \int_{x_0}^\infty x \cdot \theta \cdot \exp(\alpha + \sigma^2/2) f_x(x) dx
\]

where \(f_x(x)\) is the lognormal probability density function for metalaxyl residues.

Parameter estimation for \(\alpha\), \(\beta\), and \(\sigma^2\) was done using linear regression of the natural logarithm of \(h(x)\) on the natural logarithm of \(x\). The prediction equation is

\[
\ln h(x) = -2.1476 - 1.1988 \cdot \ln x
\]

\((R^2 = 0.85)\) and the estimate for \(\sigma^2\) is 0.572. For these parameter values

<table>
<thead>
<tr>
<th>Year</th>
<th>Dose</th>
<th>Timing</th>
<th>(Y_i)</th>
<th>Model</th>
<th>Observed</th>
<th>(Range)</th>
<th>(Y_i)</th>
<th>Model</th>
<th>Observed</th>
<th>(Range)</th>
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<td>1977</td>
<td>0.28</td>
<td>14</td>
<td>0</td>
<td>0.130</td>
<td>0.146</td>
<td>(0.00-0.40)</td>
<td>1.1</td>
<td>2.5</td>
<td>(0-6.5)</td>
<td></td>
</tr>
<tr>
<td>0.14</td>
<td>14</td>
<td>0</td>
<td>0.223</td>
<td>0.256</td>
<td>(0.02-0.49)</td>
<td>2.4</td>
<td>4.5</td>
<td>(0.5-13.5)</td>
<td></td>
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<tr>
<td>1978</td>
<td>0.22</td>
<td>14</td>
<td>0</td>
<td>0.383</td>
<td>0.272</td>
<td>(0.11-0.48)</td>
<td>3.9</td>
<td>10.7</td>
<td>(4-20)</td>
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</tr>
<tr>
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<td>21</td>
<td>0</td>
<td>0.210</td>
<td>1.685</td>
<td>(1.13-2.34)</td>
<td>1.8</td>
<td>47.8</td>
<td>(35-68)</td>
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<tr>
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<td>14</td>
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<td>3.477</td>
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<td>22.7</td>
<td>38.3</td>
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<td>6.872</td>
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<td>41.1</td>
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<td>Hudson</td>
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<td>7, 14</td>
<td>10</td>
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<td>9.393</td>
<td>(7.39-13.34)</td>
<td>17.4</td>
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<td>25.3</td>
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<td>3.311</td>
<td>(2.27-4.12)</td>
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<td>39.2</td>
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<td>60.7</td>
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<td>28.7</td>
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1. Data from 1977 and 1978 have been published previously (13-15).
2. Area under disease progress curve (29).
3. Final percent disease.
4. Metalaxyl doses (a.i. kg/ha).
5. Intervals between spray applications (days).
6. Initial disease level (%) when first fungicide application made.
7. Mean observed values, \(n = 3\) for all treatments.
8. Range of observed values, \(n = 3\) for all treatments.
9. Mixture of 0.14 kg/ha of metalaxyl and 0.63 kg/ha of chlorothalonil.
10. Potato cultivars Hudson and Sebago were used in 1979. Only Hudson was used in 1977 and 1978.
11. Interval between first and second metalaxyl applications was 7 days; subsequent intervals were 14 days.
estimates, the threshold metalaxyl residue level, \( x_0 \), is 0.212 ng/cm².

Lesion expansion rates (millimeter per day increase in diameter) are inhibited when metalaxyl is applied after lesion establishment. No lesion expansion was observed when leaves were treated with metalaxyl 1 day after inoculation. Inhibition of rates decreases linearly as the age of the lesion at the time of application increases (Fig. 2). Two-way analysis of variance showed that lesion expansion rates are not affected by metalaxyl doses from 1 to 25 \( \mu g/ml \) (\( F_{1,12} = 1.32, P > 0.25 \)).

Metalaxyl residues (nanograms per square centimeter) from leaflets dipped in 1–25 \( \mu g/a.i./ml \) of metalaxyl in the laboratory represent the range of residues likely to be found in the field 1 h after spraying under normal field conditions (24, unpublished). Leaflets dipped in 25 \( \mu g/a.i./ml \) had a mean residue of 77.2 ng/cm² (SD = 32.2, \( n = 8 \)). The median residue from leaflets collected in the field 1 h after spraying with 20.0 \( kg/a.i./ha \) of metalaxyl (214 \( \mu g/a.i./ml \)) was only 23.1 ng/cm² (SD = 6.8, \( n = 8 \)) after being rinsed in distilled water (24, unpublished). Residues from the same field plots 1 h after spraying had a minimum of 3.0 ng/cm² (SD = 3.32, \( n = 8 \), unpublished), while the residues on leaflets dipped in 1 \( \mu g/a.i./ml \) of metalaxyl had a mean of 1.9 ng/cm² (SD = 1.67, \( n = 10 \)). Therefore, the range of concentrations used in the laboratory includes the range of residues encountered when metalaxyl is applied under normal field conditions.

**Model performance.** Model predictions were compared to field epidemics observed in 1977, 1978, and 1979. Predictions of disease progress curves were similar to those observed in field plots in all 3 yr (Fig. 3). When metalaxyl was applied in a protectant manner (application began before disease was observed), predicted disease levels gradually increased to only 1% in 1977 (Fig. 3A) and to 5% in 1978 (Fig. 3B). When metalaxyl was applied in an eradicant manner (application began after some disease was observed), epidemic development slowed within 5–7 days after the first application (Fig. 3B–D). Model predictions for AUDPC were within the range of observed values from the field in 13 out of 17 cases (Table 3). Final disease, \( Y_F \), was not predicted as well as AUDPC: \( Y_F \) was within the range of observed values in only 9 of 17 epidemics (Table 3). Observed disease sometimes increased noticeably at the end of the season (15), resulting in five of the 17 predictions underestimating \( Y_F \). Model predictions for the effects of metalaxyl on both the susceptible and resistant cultivars used in 1979 are similar to observed epidemics (Fig. 3C and D). The model also predicted the effects of various metalaxyl doses well and the effects of metalaxyl when the first sprays were made at different levels of disease (Table 3).

**DISCUSSION**

The model presented here realistically described the effects of the systemic fungicide metalaxyl on late blight epidemics on potatoes under a variety of conditions. AUDPC is the most useful measure of disease for evaluating model predictions relative to field observations because it incorporates disease levels for the entire epidemic (29). The model accurately predicted disease progress when the first metalaxyl application was made either before or after disease was observed in the field. Comparing model predictions for eradicant use of metalaxyl against field observations is a rigorous test of the model because the eradicant components of the model must function at the same time as the components that inhibit new lesion establishment. Model predictions for disease progress when metalaxyl is used in a protectant manner are also generally within the range of observed values. Disease at the end of epidemics may have been underestimated because the model did not adequately account for multitreatment interference or the increase in susceptibility to late blight as plants age (11).

The difficulties of assessing disease in metalaxyl-treated plots in the field may contribute to some of the apparent discrepancies between predicted and observed disease levels. After metalaxyl applications, late blight lesions dry up and lose their characteristic appearance. In this condition, they can easily be confused with insect damage or early blight lesions caused by *Alternaria solani*. These difficulties are compounded by the difficulty of accurately assessing very low levels of disease, resulting in some erratic observed disease progress curves (Fig. 3B and D).

We have assumed that there is no host plant growth. This is a reasonable assumption for the field conditions simulated in this study because plants had grown to nearly full size before inoculation and fungicide application. However, potato growers in the northeastern United States usually apply fungicides long before plants are fully grown (16). The redistribution of metalaxyl residues to developing tissues would need to be included in a model that incorporated host growth.

This model was developed as a tool for identifying optimal uses of metalaxyl for controlling potato late blight. An important consideration is how to prevent or delay the buildup of metalaxyl resistance in *P. infestans*. Theoretical models (8,19,20,22,30) and experimental evidence from greenhouse (28,34) and field studies (21) predict that mixtures of metalaxyl with protectant fungicides will delay the buildup of resistance. However, only one controlled field study has been done for metalaxyl resistance in the potato late blight system (7). By using a realistic simulation model we should be able to evaluate numerous different strategies for metalaxyl resistance management under a variety of environmental conditions. An important factor in identifying appropriate resistance management strategies is the simultaneous goal of managing disease at economic levels. A mathematical model such as this one could be an essential component for optimizing fungicide use given these potentially conflicting goals.

![Fig. 1](image-url)  
**Fig. 1.** The effects of metalaxyl on lesion establishment by *Phytophthora infestans* on potato leaf tissue. Proportion lesion establishment is relative to the control when no metalaxyl is present. Metalaxyl residue concentrations are expressed as nanograms per square centimeter of leaf. Data plotted in this figure are also presented in Table 2. The equation for the fitted curve is \( h(x) = e^{2.15 \cdot x^{1.12}} \), where \( x \) is metalaxyl residue.

![Fig. 2](image-url)  
**Fig. 2.** The percent inhibition of lesion expansion by *Phytophthora infestans* in potato leaflets by metalaxyl as a function of lesion age (days) when metalaxyl was applied. Leaflets were dipped in metalaxyl suspensions with concentrations of 1, 5, 10, 15, and 25 \( \mu g/ml \). Because there was no significant effect of concentration, data plotted in this figure are means from four leaflets per concentration, with all concentrations pooled (\( n = 20 \)). Error bars represent one standard error. The equation for the regression line is \( Y = 120.7 - 19.2x \), where \( x \) is lesion age.
DAYS AFTER INOCULATION

Fig. 3. Comparisons of observed and predicted disease progress curves for late blight epidemics, caused by Phytophthora infestans on potatoes, when metalaxyl is applied. Observed disease progress data (solid symbols) are means of three replicate plots. Predicted curves (open symbols) were simulated using actual weather and fungicide application data as input for the model described in the text. Metalaxyl was applied at 14-day intervals after the first application. The susceptible potato cultivar Hudson was used in all 3 yr; the resistant cultivar Sebago was used in 1979 only. Observed disease progress curves from unsprayed plots are shown for scale. A, Epidemics in 1977 (15) in which metalaxyl (0.28 kg/ha) was applied in a protectant manner. The first application was on the same day as inoculation (day 0). B, Epidemics in 1978 (14) in which metalaxyl (0.22 kg/ha) was applied in both a protectant and eradicant manner. The first applications were on day 0 for the protectant treatment (squares) and day 18 (see arrow) for the eradicant treatment (triangles) when disease was approximately 2–5%. C, Epidemics on the potato cultivar Hudson in 1979. Metalaxyl was first applied on day 12 (see arrow), when disease reached approximately 2–5%, at the rates of 0.28 kg/ha (squares) and 0.14 kg/ha (triangles). D, Epidemics on the resistant potato cultivar Sebago in 1979. Metalaxyl was first applied on day 18 (see arrow), when disease reached approximately 2–5%, at the rates of 0.28 kg/ha (squares) and 0.14 kg/ha (triangles).

LITERATURE CITED

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