An Apple Powdery Mildew Model Based on Plant Growth, Primary Inoculum, and Fungicide Concentration

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ABSTRACT

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In 1981, 1982, and 1983 Rome Beauty apple trees with different amounts of primary mildew were sprayed with various concentrations of the fungicide bitertanol. The number of leaves and proportion of infected leaves per shoot were determined periodically throughout each growing season. Plant growth and disease progress curves were then constructed for each treatment combination of primary mildew and bitertanol concentration. Trees with higher levels of primary mildew were observed to have greater rates of disease progression and higher carrying capacities for disease. Conversely, those trees receiving higher fungicide concentrations had lower rates and carrying capacities. When disease incidence was modeled as a function of shoot growth, the best fit was obtained by substituting a linear function of primary mildew and fungicide concentration for the disease carrying-capacity parameter. Because disease was dependent on the production of young tissue, the end of the epidemics occurred when plant growth approached its carrying capacity. Under these circumstances, the model was reduced from four to three parameters: The final amount of disease was expressed as a function of primary mildew and fungicide concentration. Thus, given estimates of the amount of primary mildew, the concentration of fungicide necessary to manage secondary mildew at a desired level can be predicted. Although this model provides a method for optimizing fungicide use, additional factors need to be considered to augment its accuracy and capability.

The initiation and development of disease on a crop depends on the availability of susceptible tissue, the presence of a virulent pathogen, and the favorableness of environmental conditions. The equivalence theorem states that these three factors, which define the disease triangle, have similar effects on the epidemic process (13,14). Thus, management practices that influence different factors can be readily interchanged without altering the level of disease control. Furthermore, functional relationships between these practices can be derived and used for management purposes. For example, differences in resistance among potato cultivars to *Phytophthora infestans* (Mont.) de Bary have been expressed in terms of fungicide equivalents (5).

Apple powdery mildew epidemics, caused by *Podosphaera leucotricha* (Ell. & Ev.) Salm. can be readily described in terms of the disease triangle. The initial pathogen population, the overwintering primary mildew, is a key determinant in the development of secondary mildew epidemics (2). Also, because young tissue is most susceptible to infection (1), the growth of the plant is crucial to the progression of disease. However, unlike many fungi, which require specific temperatures and wetting periods for infection, the powdery mildews, including *P. leucotricha*, are less dependent on critical environmental conditions (2). Nevertheless, the environment is important in that it is made unfavorable through the application of fungicides.

The objective of this study was to develop a functional relationship between primary mildew and fungicide concentration. We hypothesized that trees or orchards having lower levels of primary (initial) inoculum should require less fungicide to maintain disease at or below some given threshold. And because the production of new leaves is crucial for disease development, plant growth was central to the disease progress model.

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MATERIALS AND METHODS

Field design. In 1981 and 1982 the orchard layout consisted of four rows, each with 12 standard trees of *Malus pumila* Mill. cultivar Rome Beauty on seedling rootstock. In 1983 the design consisted of four rows with 14 standard trees of the same cultivar and rootstock. In all years, trees averaged 5 m high and were spaced 9 × 10.7 m apart. The trees were pruned before the experiment in 1981; however, the 1982 and 1983 trees had not been pruned 1 and 2 yr, respectively, before initiation of the experiment.

Treatment design. During early to full bloom in each year, the total number of shoots and clusters with overwintering (primary) mildew was counted on each tree. The infected shoots and clusters, which were recognized by the mass of white, cottony mycelium covering the leaves and stems, were tagged to avoid counting them twice. The trees were then assigned to groups according to their level of primary mildew (PRIM = number of infected shoots and clusters per tree).

In 1981, six primary mildew groups of seven trees each were established. The range of PRIM within these groups was 0–1, 2–4, 4–6, 6–11, 12–19, and 20–48 infected shoots and clusters per tree. To each of the seven trees in each group, a different concentration (CONC) of the fungicide bitertanol (Mobay Chemical Corp., Kansas City, MO) was randomly assigned; these treatments were 0, 4.69, 9.38, 18.75, 37.5, 75, and 150 mg a.i. L^{-1}. Thus, the treatment design was a 6×7 factorial for a total of 42 trees; the remaining six trees in the block were not used in the experiment.

In 1982, 12 primary mildew groups of four trees each were established. The range of PRIM within these groups was 1–6, 6–7, 7–7, 7–9, 10–10, 11–12, 13–14, 15–15, 16–17, 18–18, 21–22, and 23–36 infected shoots and clusters per tree. To each of the four trees in each group four levels of CONC were randomly assigned: 0, 75, 150, and 225 mg a.i. L^{-1}. Thus, the treatment design was a 12 × 4 factorial for a total of 48 trees.

In 1983, eight primary mildew groups of seven trees each were established. The range of PRIM within these groups was 10–40,

60–100, 110–160, 160–200, 220–250, 260–350, 350–570, and 660–830 infected shoots and clusters per tree. To each of the seven trees within each group seven levels of CONC were randomly assigned: 0, 50, 100, 150, 200, 250, and 300 mg a.i. L^{-1}. Thus, the treatment designed was an 8×7 factorial for a total of 56 trees.

Fungicide treatments were applied to the point of runoff (2,800 L/ha) via a high-pressure handgun at 3,800 kPa. Applications were first made during bloom on 6, 10, and 12 May of 1981, 1982, and 1983, respectively. In each year, the sprays were applied at 14-day intervals for a total of four applications. The trees assigned the 0 mg a.i. L^{-1} treatment were not sprayed.

Disease and plant growth assessment. During each year, 10 vegetative shoots were selected and tagged around the periphery of each tree. During each assessment the total current number of healthy and diseased leaves were counted on each shoot. Disease incidence was expressed as the proportion of infected leaves per shoot: the number of diseased leaves divided by the total number of leaves per shoot. Plant growth was expressed as the total number of leaves per shoot. The 10 recordings for each variable were then averaged to produce one disease and plant growth observation per tree. In any one year, infection was not severe enough to cause leaf drop and, hence, correction for disease removal was not necessary.

Assessments were made at 14-day intervals on the day just before application of the fungicide. In 1981 and 1983, a total of five assessments were made, the last occurring 14 days after the fourth fungicide spray. In 1982 a total of eight observations were made, the first seven observations taken at approximately 7-day intervals and the last observation occurring 16 days after the fourth spray.

RESULTS

Disease/plant growth model. To determine the basic nature of the disease progress curves, the average proportion of diseased leaves, calculated across all PRIM by CONC treatment combinations, was plotted against time (Fig. 1A). Disease appeared to increase initially in a linear fashion, eventually leveling off at some maximum asymptote or carrying capacity. This type of growth pattern could be described by the monomolecular function. The final amount of disease was very similar in 1981 and 1982, but much greater in 1983. This difference may have resulted from the larger amount of overwintering primary mildew observed in 1983.

The production of leaves, observed from onset of the epidemic, followed a growth pattern similar to disease progress (Fig. 1B). Although the 1981 data displayed some curvature during the first half of growth, the 1982 and 1983 curves appeared linear during this phase. Furthermore, in 1981 leaf production ceased at an average of 20.6 leaves per shoot, whereas in 1982 and 1983 only 18.7 and 15.4 leaves per shoot had been produced. This decrease might be attributed to the lack of pruning before experimentation in 1982 and 1983; trees with greater numbers of shoots tended to produce fewer leaves per shoot.

Given that the incidence of apple mildew is dependent on the production of young, susceptible tissue, then the progression of disease can be derived as a function of plant growth (12). The appropriate model, which assumes monomolecular growth for both the disease and the plant, is

$$y = K_1 \quad \{1 - B[(K_2 - (x - x'))/K_2]^{r_g}\} \qquad (1)$$

where x = current numbers of leaves per shoot at some time t, y = proportion of infected leaves per shoot at time t (current number of infected leaves per shoot at time t/x), x' = number of leaves per shoot at onset of the epidemic (first disease assessment), K_1 and K_2 are carrying capacities for disease and the plant, respectively, $r_g = r_1/r_2$ is the intrinsic rate of disease progression (r_1) relative to the intrinsic rate of plant growth (r_2), and when $x = x'$, $B = (K_1 - y)/K_1$, the proportion that disease increases during the epidemic.

The above model was fit to all of the data observed in each year, regardless of fungicide or primary mildew level, using the Gauss–Newton method of nonlinear regression (6,7). Since the K_2 parameter cannot be estimated using this technique (12), it was made a constant in this and all subsequent model fittings: K_2 = maximum $(x - x') + 0.01$. The results of the three regressions, as indicated by their coefficients of determination (r^2), indicate a much better fit in 1981 and 1983 than in 1982 (Table 1). However, this model is only preliminary because it lacks the fungicide concentration and primary mildew variables.

Integration of primary mildew and fungicide concentration. The level of primary mildew and of fungicide concentration had similar but opposing effects on disease progression. Larger amounts of initial inoculum or lower concentrations of bitertanol allowed greater rates of disease increase and, ultimately, a higher K_1, the carrying capacity of the environment for disease (Fig. 2). Thus, both r_g and K_1 could be expressed as some function of primary mildew (PRIM) and of bitertanol concentration (CONC): $r_g = f(\text{PRIM,CONC})$ and $K_1 = f(\text{PRIM,CONC})$. The linear

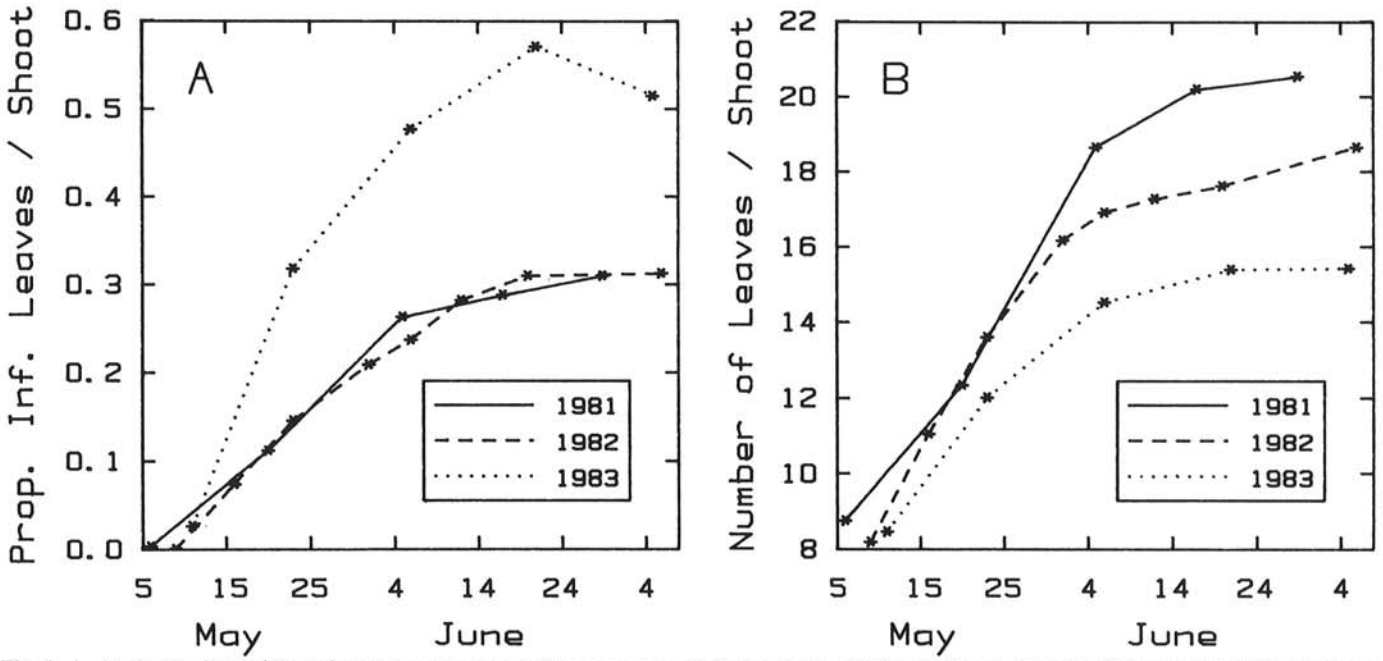

Fig. 1. A, Apple powdery mildew disease progress, and **B,** Shoot growth. Each mean was calculated over all primary mildew by fungicide treatment combinations; thus, each point is an average of 42, 48, and 56 trees in 1981, 1982, and 1983, respectively.

representation of these functions would be:

$$K_1 = g_0 + g_1 \text{PRIM} - g_2 \text{CONC} \quad (2)$$

$$r_g = g_3 + g_4 \text{PRIM} - g_5 \text{CONC} . \quad (3)$$

The g_1PRIM and g_4PRIM terms are added to the constants g_0 and g_3, respectively, because an increase in PRIM is hypothesized as causing an increase in either or both K_1 and r_g. Similarly, the CONC terms are subtracted because an increase in CONC should cause a reduction in either or both parameters.

Equations 2 and 3 were substituted into equation 1 to examine if the PRIM and CONC factors could describe additional variation in the data. The three possible models were:

$$y = (g_0 + g_1 \text{PRIM} - g_2 \text{CONC}) \{1 - B[(K_2 - (x - x'))/K_2]^{g_3 + g_4 \text{PRIM} - g_5 \text{CONC}}\} \quad (4)$$

$$y = K_1 \{1 - B[(K_2 - (x - x'))/K_2]^{g_3 + g_4 \text{PRIM} - g_5 \text{CONC}}\} \quad (5)$$

TABLE 1. Results of nonlinear regression performed on a composite disease/plant growth model[a] that assumes monomolecular growth of disease incidence and leaf production

Year	r^2[b]	df error	Parameter	Estimate	Standard error	Relative variation[c] (%)	95% Confidence interval Lower	95% Confidence interval Upper
1981	0.74	206[d]	K_1	0.3030	0.0101	3	0.2831	0.3228
			B	1.0109	0.0343	3	0.9432	1.0786
			r_g	1.9531	0.2599	13	1.4406	2.4655
1982	0.51	381	K_1	0.4762	0.0518	11	0.3744	0.5781
			B	1.0056	0.0271	3	0.9524	1.0588
			r_g	0.8738	0.1625	19	0.5542	1.1934
1983	0.89	276[d]	K_1	0.5933	0.0144	2	0.5650	0.6215
			B	0.9609	0.0155	2	0.9304	0.9914
			r_g	1.8032	0.1301	7	1.5471	2.0592

[a] $y = K_1 \{1 - B[(K_2 - (x - x'))/K_2]^{r_g}\}$.
[b] r^2 = 1 − (residual sum of squares/corrected total sum of squares); all f-values were highly significant, $P < 0.0001$.
[c] (Standard error of estimate/estimate) × 100.
[d] One observation, an outlier, was omitted from the data set.

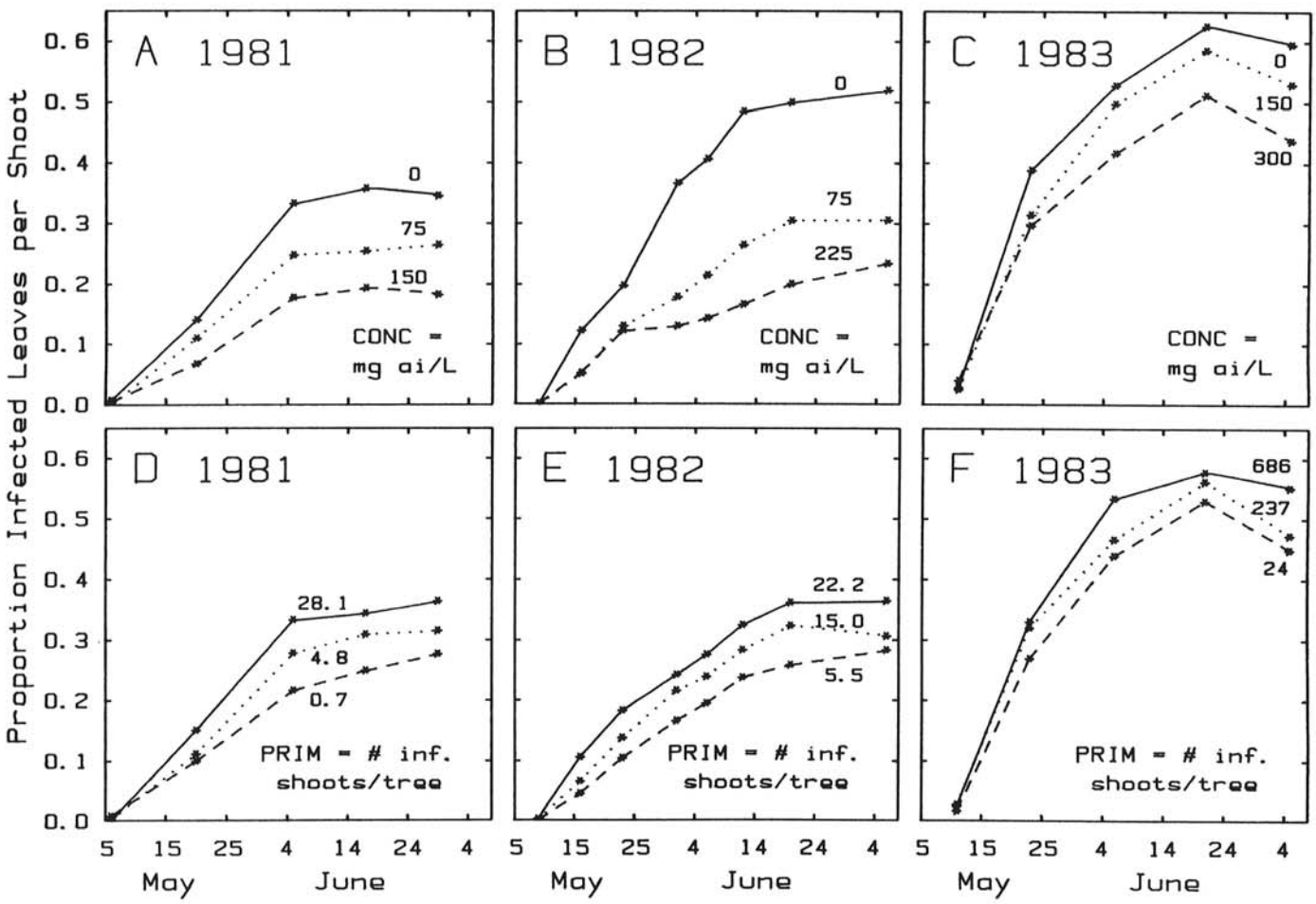

Fig. 2. The progression of apple powdery mildew for selected levels of the bitertanol concentration **(A, B, C)** and primary mildew **(D, E, F)** factors. Each point for a CONC level is an average over all PRIM levels; thus, $n = 6$, 12, and 8 trees/point in 1981, 1982, and 1983, respectively. The lowest, highest, and an intermediate level were chosen for display; all other intermediate levels not shown had progress curves of intermediate severity.

TABLE 2. Comparison of nonlinear regression estimates for three composite disease/plant growth models that incorporate a linear function of primary mildew and fungicide concentration for the rate and/or carrying capacity parameters

Year	r^2[a]	df error	Parameter	Estimate	Standard error	Relative variation[b] (%)	95% Confidence interval	
							Lower	Upper
			Rate and carrying capacity as functions of PRIM and CONC[c]					
1981	0.89	202[d]	g_0	0.3125	0.0112	4	0.2904	0.3346
			g_1	0.0066	0.0009	14	0.0048	0.0085
			g_2	0.0012	0.0001	9	0.0010	0.0014
			B	0.9947	0.0196	2	0.9561	1.0333
			g_3	1.7963	0.1819	10	1.4375	2.1550
			g_4	−0.0214	0.0068	32	−0.0348	−0.0080
			g_5	−0.0002	0.0015	780	−0.0032	0.0028
1982	0.84	377	g_0	0.4292	0.0269	6	0.3763	0.4820
			g_1	0.0161	0.0021	13	0.0120	0.0202
			g_2	0.0016	0.0002	10	0.0012	0.0019
			B	1.0081	0.0150	1	0.9786	1.0375
			g_3	1.2838	0.1260	10	1.0360	1.5317
			g_4	−0.0154	0.0039	25	−0.0231	−0.0077
			g_5	0.0015	0.0005	34	0.0005	0.0025
1983	0.93	272[d]	g_0	0.6117	0.0220	4	0.5683	0.6551
			g_1	0.0030	0.0001	17	0.0002	0.0004
			g_2	0.0005	0.0001	16	0.0004	0.0007
			B	0.9599	0.0116	1	0.9370	0.9829
			g_3	1.9577	0.1667	8	1.6296	2.2859
			g_4	−0.0010	0.0002	24	−0.0015	−0.0006
			g_5	−0.0003	0.0006	214	−0.0015	0.0009
			Rate as function of PRIM and CONC[e]					
1981	0.86	204[d]	K_1	0.3668	0.0140	4	0.3391	0.3945
			B	0.9710	0.0194	2	0.9327	1.0094
			g_3	1.0956	0.1264	12	0.8463	1.3450
			g_4	0.0323	0.0061	19	0.0203	0.0444
			g_5	0.0065	0.0008	13	0.0049	0.0082
1982	0.80	379	K_1	0.6351	0.0355	6	0.5653	0.7049
			B	0.9937	0.0119	1	0.9704	1.0171
			g_3	0.7076	0.0714	10	0.5672	0.8481
			g_4	0.0176	0.0024	14	0.0128	0.0224
			g_5	0.0030	0.0003	11	0.0024	0.0037
1983	0.92	274[d]	K_1	0.6212	0.0128	2	0.5960	0.6463
			B	0.9755	0.0121	1	0.9338	0.9815
			g_3	1.8626	0.1328	7	1.6010	2.1242
			g_4	0.0010	0.0002	19	0.0006	0.0014
			g_5	0.0033	0.0004	12	0.0025	0.0041
			Carrying capacity as a function of PRIM and CONC[f]					
1981	0.89	204[d]	g_0	0.3333	0.0102	3	0.3131	0.3535
			g_1	0.0043	0.0005	11	0.0033	0.0053
			g_2	0.0013	0.0001	7	0.0011	0.0014
			B	0.9959	0.0203	2	0.9558	1.0360
			r_g	1.5610	0.1464	9	1.2723	1.8496
1982	0.83	379	g_0	0.4944	0.0254	5	0.4445	0.5443
			g_1	0.0087	0.0010	11	0.0068	0.0107
			g_2	0.0018	0.0001	6	0.0016	0.0020
			B	1.0209	0.0181	2	0.9853	1.0564
			r_g	1.1308	0.1063	9	0.9217	1.3399
1983	0.93	274[d]	g_0	0.6415	0.0156	2	0.6108	0.6723
			g_1	0.0001	0.00002	15	0.0001	0.0002
			g_2	0.0005	0.00005	9	0.0004	0.0006
			B	0.9609	0.0120	1	0.9372	0.9856
			r_g	1.7282	0.0992	6	1.5330	1.9235

[a] $r^2 = 1 -$ (residual sum of squares/corrected total sum of squares); all f-values were highly significant, $P < 0.0001$.
[b] (Standard error of estimate/estimate) × 100.
[c] See equation 4.
[d] One observation, an outlier, was omitted from the data set.
[e] See equation 5.
[f] See equation 6.

$$y = (g_0 + g_1\text{PRIM} - g_2\text{CONC}) \left\{1 - B\left[(K_2 - (x - x')/ K_2\right]^{'g}\right\} \quad (6)$$

The coefficients of determination (r^2) for fitting equation 4 to the data increased by 0.15, 0.33, and 0.04 over the preliminary model (Eq. 1) for years 1981, 1982, and 1983, respectively (Tables 1 and 2). However, the standard errors for the g_4 and g_5 parameters were quite high, ranging from 24 to 780% of the parameters' estimated value. Furthermore, the confidence interval for the g_5 parameter in 1981 and 1983 contained zero, indicating that the CONC factor in the exponent had little or no influence.

Equations 5 and 6 described almost as much variation as equation 4, but with fewer parameters (Table 2). The r^2 values for the latter model, however, were slightly higher than those of the former model, and the standard errors for their parameters were relatively smaller. Thus, the model incorporating a linear function of PRIM and CONC for K_1, equation 6, was considered the best of the three possibilities examined.

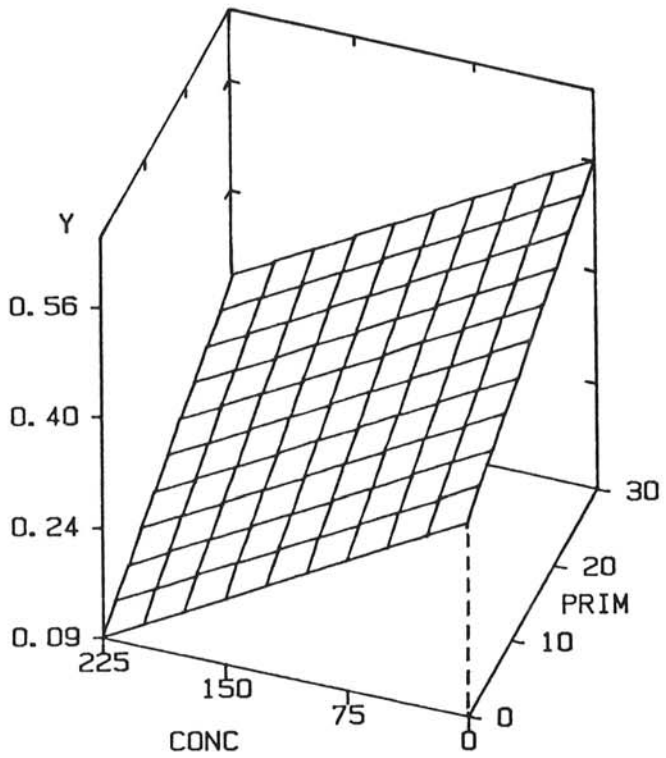

Fig. 3. Response plane depicting the effect of primary mildew (PRIM) and bitertanol concentration (CONC) on the incidence of apple powdery mildew (y) at the end of the growing season/epidemic. The linear function describing the plane, $y = 0.3639 + 0.0064\text{PRIM} - 0.0012\text{CONC}$ ($r^2 = .80$), was derived from a nonlinear model (see text, equation 6), which assumes monomolecular disease progression and plant growth. The nonlinear model was fit to the combined 1981 and 1982 data set, $n = 593$ observations; one observation, an outlier, was omitted. y = Proportion of infected leaves per shoot, PRIM = number of primary mildewed shoots and clusters per tree, and CONC = mg a.i. bitertanol per liter of spray.

Application. The differences between corresponding parameter estimates among the years underscore the difficulty in applying the chosen model (Eq. 6) for management purposes (Table 2). In most cases, a parameter estimate for the model in one year does not fall within the confidence interval for that same parameter in either or both of the other two years. This was particularly true for the g_1 and g_2 parameters in 1983, which were substantially lower in value than in 1981 and 1982. For example, the g_1 value of 0.0087 in 1982 is 87 times the size of its value of 0.0001 in 1983. These lower parameter estimates are most likely due to the dramatically larger amounts of primary mildew and, subsequently, less fungicide control in 1983. The minor effects exerted by PRIM and CONC on the epidemic in 1983 are evident in the smaller degree of separation of the disease progress curves (Fig. 2). Nevertheless, the 1981 and 1982 data sets were considered similar enough to each other and to what might be called a typical situation to warrant fitting the model to their combined data set (Table 3). The resulting model described 80% of the variation in the data.

The amount of disease at the end of an epidemic, like the infection rate or the area under the disease progress curve, can be used as a measure of the overall disease severity for the season. For apple powdery mildew, disease progression ceases when shoot growth stops, since only young tissue is susceptible to infection. Mathematically, the epidemic approaches termination as $x - x'$ approaches K_2. The $[K_2 - (x - x')]/ K_2$ term in equation 6 (Table 3) becomes zero or very small, thereby reducing the equation to

$$y = 0.3639 + 0.0064\ \text{PRIM} - 0.0012\ \text{CONC}. \quad (7)$$

This function describes a plane that depicts a linear increase in the proportion of diseased tissue with either or both an increase in primary mildew or decrease in bitertanol concentration (Fig. 3). Furthermore, equation 7 can be altered to define a functional relationship between primary mildew and fungicide concentration. Given estimates of the amount of primary mildew at the beginning of the epidemic, the concentration of bitertanol necessary to manage secondary mildew at a desired level can be predicted (Fig. 4).

DISCUSSION

Epidemics caused by *P. leucotricha* are characterized by the occurrence of many secondary infection cycles. Although such polycyclic processes should theoretically produce sigmoidal disease progress, disease in this study was observed to increase in a monomolecular fashion. This conclusion, however, was based solely on the observation of plotted data; little or no curvature was apparent during the early stages of the epidemic. Since there was no replication for any one PRIM by CONC treatment combination, the statistical fitting of various growth functions was not attempted. Past research has indicated that the Richards or Gompertz function may be appropriate models (9). Experimentation specifically designed to determine the appropriate plant and pathogen growth models is needed. From this data it should be possible to calculate and compare the parameters estimated from the separate fits with those from the combined fit, e.g., r_g from r_1 and r_2.

TABLE 3. Results of nonlinear regression on the combined 1981 and 1982 data sets for a composite disease/plant growth model[a] which incorporates a linear function of primary mildew and fungicide concentration for the carrying capacity (K_1) parameter

r^2[b]	df error	Parameter	Estimate	Standard error	Relative variation[c] (%)	95% Confidence interval	
						Lower	Upper
0.80	588[d]	g_0	0.3639	0.0111	3	0.3420	0.3857
		g_1	0.0064	0.0005	8	0.0054	0.0075
		g_2	0.0012	0.00005	5	0.0011	0.0013
		B	1.0182	0.0182	2	0.9825	1.0538
		r_g	1.6511	0.1275	8	1.4007	1.9016

[a] See text, equation 6.
[b] r^2 = 1 − (residual sum of squares/corrected total sum of squares); the f-value was highly significant, $P < 0.0001$.
[c] (Standard error of estimate/estimate) × 100.
[d] One observation, an outlier, was omitted from the data set.

Because observations on leaf production were not made before onset of the epidemic, the nature of the early stage of the plant growth curve was unknown. Indeed, plant growth may well have been logistic, but only the linear middle section and asymptotic ending were recorded. A plot of such data would therefore resemble a curve produced by the monomolecular function. Furthermore, if the infection of new leaves was solely dependent on the production of those leaves, then the disease progress curve would resemble the plant growth curve; that is, in this case, it would appear monomolecular. This dependence of disease on plant growth seems tenable since it was likely that neither environmental conditions nor inoculum availability were limiting. In addition, all the r_g estimates were greater than unity, indicating that the intrinsic rate of leaf infection, r_1, was greater than the intrinsic rate of leaf production, r_2. Hence, leaves became infected as soon as they were produced. The dependence of disease on plant growth was also suggested as a cause for logistic growth of tobacco black shank, a soilborne disease that theoretically should increase in a monomolecular fashion (4). If tobacco roots grow in a logistic manner, then disease progression may also occur logistically as host roots grow out into infested soil and encounter increasing amounts of inoculum.

In modeling the progression of apple mildew, plant growth was viewed as the impetus for the epidemic. However, primary mildew and fungicide concentration were conceptualized as opposing factors, which together determine the final severity or carrying capacity of the environment for disease. Similarly, Jeger (10) noted that "the protective properties of fungicides were transplanted, epidemiologically, into lowered asymptotes of disease." Thus the model presented above provides one possible approach to disease management. For example, if a grower had on the average 15 primary infected shoots per tree, then he would need to spray at 217 mg a.i. L^{-1} at 2-wk intervals to end the season at 20% disease (Fig. 4). Or alternatively, he could prune out 10 of the 15 shoots, thus allowing a spray concentration reduction to 163 mg a.i. L^{-1}. If higher secondary mildew levels were acceptable, then either less fungicide would need to be applied or more primary mildew could be tolerated. Although this approach allows for optimization of management strategies, a variety of factors that affect the model's accuracy and capability need further consideration.

Given the differences between levels of primary mildew and concentrations of fungicide on adjacent trees, interplot interference was probably the most important single factor altering the model's accuracy. When no initial inoculum is present (PRIM = 0), and when no fungicide is applied (CONC = 0), equation 7 still predicts 36% of the leaves to be infected. This bias was attributed to the exchange of primary and secondary inoculum among trees. Since the model overestimates the amount of disease, it consequently overestimates the fungicide concentration necessary to manage the epidemic for a given level of primary mildew. Observations on larger experimental units (e.g., several trees) may reduce the degree of interference. Although it would be difficult to obtain a block of trees with each tree having the same primary mildew level, perhaps an average of the levels could be used instead; this may also be more similar to a commercial setting. The amount of primary mildew could then be expressed as the number of infected shoots per volume of foliage per unit area of orchard, thus allowing for differences in tree size as well as planting density.

The application of any model ultimately depends on its ability to handle a varied set of conditions under which growers operate. The capability of the proposed model is no doubt limited to the cultivar Rome Beauty and the fungicide bitertanol. However, given information on the growth and disease resistance characteristics of other cultivars, and on the efficacy of other fungicides, the model parameters could be adjusted to reflect any differences. Recent studies have shown that apple cultivars differ in partial resistance to mildew through differences in various components of the disease monocycle such as latent period or sporulation (8,11). Furthermore, these studies have shown that fungicides differ in their epidemiological modes of action by also differentially affecting these same components. Consequently, the proper matching of a fungicide with a given cultivar could conceivably

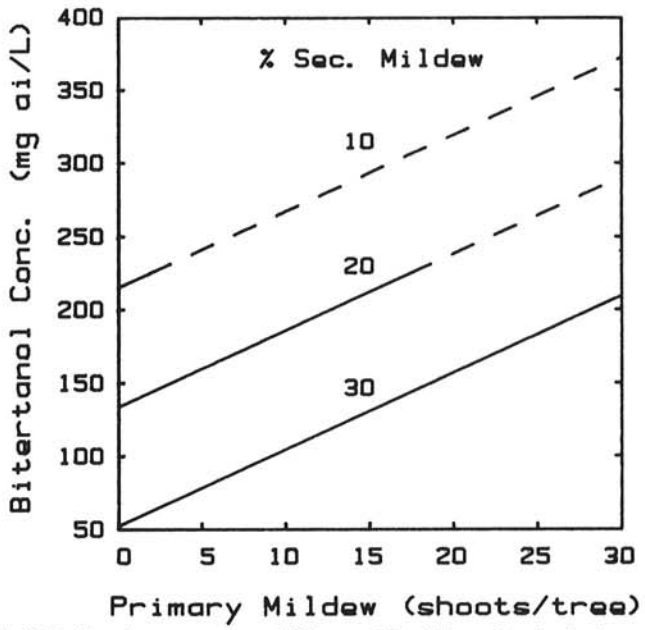

Fig. 4. Predicted concentrations of bitertanol for different levels of primary mildew necessary to maintain the percent of secondary mildew (percentage of leaves infected per shoot) at a given level by the end of the epidemic. The function which describes the contours, CONC = 303.25 − Y/0.0012 + 5.3333PRIM, $r^2 = .80$, is an algebraic alteration of the function describing the response plane in Fig. 3. The dashed lines are extrapolations of the model since the highest bitertanol concentration in the 1981/1982 data set was 225 mg a.i. L^{-1}.

provide enhanced disease control. Research has also been recently conducted to determine the economic injury level from the relationship between disease incidence and crop loss (3). This type of information could be integrated within the context of the model presented in this paper. Given a particular cultivar and assessed level of primary mildew, the correct type and amount of fungicide could be applied to keep secondary mildew below the economic injury level.

In addition to data on cultivar and fungicide differences, the distribution of primary mildew in orchards needs to be examined so that an adequate sampling plan can be formulated. An estimate of the amount of primary mildew at the beginning of the growing season is a prerequisite for model application. Research is also needed to examine the impact of pruning out primary mildew on the epidemic. Although it may not be economically feasible to remove all the primary mildewed shoots, a roguing of only those most easily reached may have enough impact. However, by the time the infected shoots are visible, much initial inoculum may have already been dispersed.

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