Models for the Spread of Plant Disease: Some Experimental Results

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ABSTRACT

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Some predictions of two models of disease spread were examined in field experiments with potato late blight. The logit transformation was used to linearize disease gradients, and the gradient parameter (defined as the rate at which the logit of disease severity declines with distance) was constant after an initial stabilization period. Both rate-limiting resistance and fungicide treatment caused gradients to become significantly flatter. These results are consistent with model predictions. Contrary to predictions, the measured velocity of spread was unaffected by any treatment; some possible reasons for this result are discussed.

In a previous paper (3) we presented an approach to the study of plant disease spread based on the concept that spread occurs as a traveling wave. We defined a new measure of the disease gradient, g, as the slope of the logit of disease severity on distance, and showed that g, the apparent infection rate, r, and the wave velocity, v, are related by the expression $g = r/v$. We also postulated a number of qualitative relationships between these parameters and some factors that determine them—in particular, the latent and infectious periods, the multiplication rate, and the probability function describing spore dispersal.

These concepts and relationships were derived from two related models of disease spread. Our purpose in this paper is to present results of field experiments designed to demonstrate the use of these models and the utility of the parameters g and v. The experiments were carried out with potato late blight.

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MATERIALS AND METHODS

Field plots. All of the plots used in these experiments were 36.6 m long and two rows wide (Fig. 1), and were planted 12 May 1982. Plant spacing was 30.5 cm within rows and 0.9 m between rows. After 14 July, the plots were sprinkler-irrigated each day for 1 hr at 0700 and 1900 hours, at the rate of 25 mm/hr. Plots were isolated from each other with rows of field corn; dimensions of these isolation strips are given in Fig. 1.

The six plots were divided into three treatments of two replicates each. Plots 3, 4, 7, and 8 were planted to the moderately susceptible potato (*Solanum tuberosum* L. 'Katahdin'); plots 7 and 8 were sprayed at weekly intervals (beginning 28 July) with chlorothalonil

(Bravo) at 0.4 kg a.i./ha (treatment F), while plots 3 and 4 were left unsprayed (treatment U). Plants of the moderately resistant potato cultivar Sebago were used in plots 1 and 5 to investigate the effects of rate-limiting resistance (treatment R). (Plots 2 and 6, a mixed planting of cultivars Katahdin and Kennebec, were abandoned due to a severe early blight epidemic that made late blight assessment unreliable). All plots were inoculated at the NW end with an isolate of *Phytophthora infestans* (Mont.) de Bary race 0 (2) on 14 July and again on 21 July.

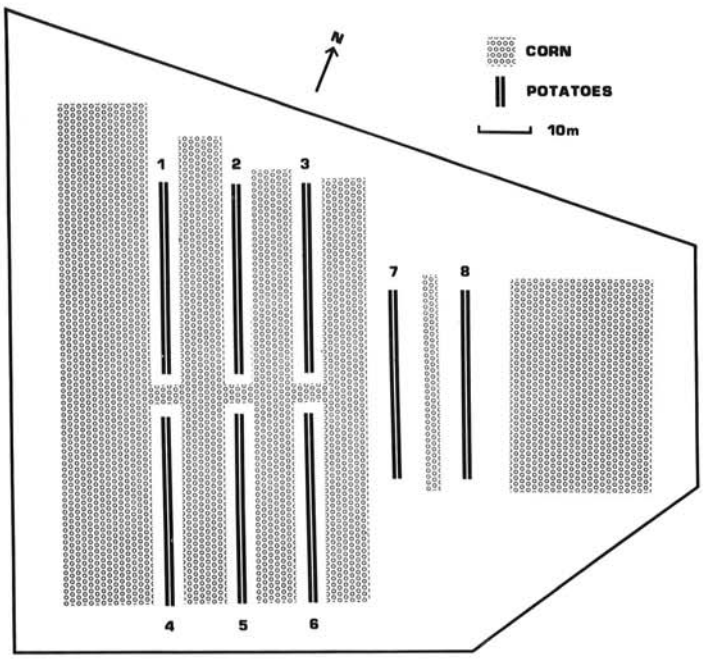

Fig. 1. Plot layout. Scale is approximately 1:1,500.

Disease assessment. Each plot was marked at 0.9-m intervals, and disease severity was assessed in each of the resulting 40 "cells" individually. A modified visual assessment key for late blight was used (1). Observations were made at 2- to 4-day intervals between 3 and 29 August.

Data analysis. All disease severity data were transformed to logits for analysis, except that severity measurements less than 1% or greater than 99% were not used. In these ranges, visual estimates of severity were considered unreliable. The apparent infection rate r and the gradient parameter g were estimated by regressing the logit of disease severity on time or distance, respectively, as previously described (3). Several estimates of r and g were selected, using criteria described in the Results section, and pooled (4) to give final estimates for each plot. In addition, the coefficients of the regression on distance were used to estimate s_{50}, the distance from the point of inoculation at which disease severity was 50%. The velocity of spread v was then found by regressing s_{50} on the time of observation. (A simpler and mathematically equivalent procedure is to divide the estimate of r by the estimate of g; this was used as a check on calculations).

Treatment effects on r, g, and v were analyzed with a one-way analysis of variance. When the F-statistic was significant at $P = 0.95$, pairwise differences were examined with the LSD test, also at $P = 0.95$.

RESULTS

The disease gradient in all plots was initially quite steep, and rapidly became flatter over time (Figs. 2 and 3). Within 7–10 days of the first observations, this flattening ceased, and the gradients were stable thereafter. In several plots the gradient over the first 4–5 cells (3–4 m) from the site of inoculation remained steep throughout the epidemic (Fig. 4). Data from these cells were not used for parameter

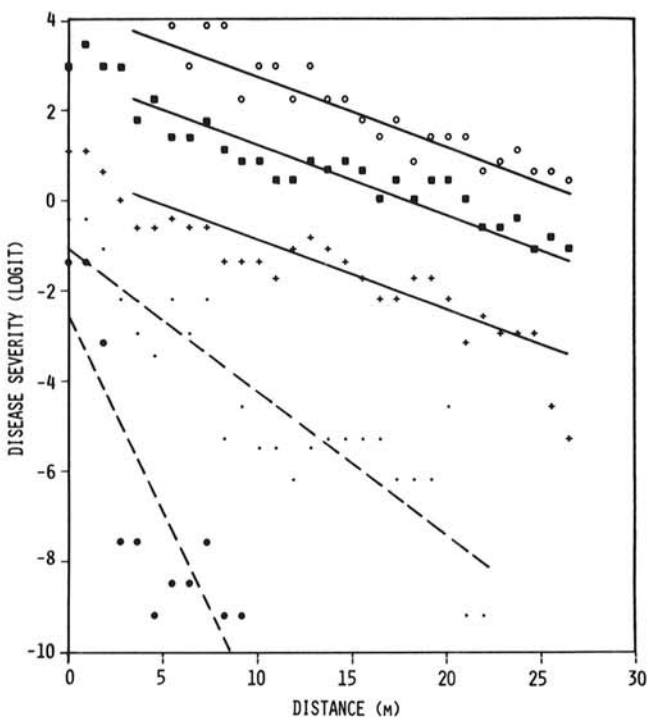

Fig. 2. Potato late blight severity as a function of distance from the point of inoculation, for plot 4 (unsprayed Katahdin). Observation dates are: large dots, 3 August; small dots, 6 August; crosses, 9 August; squares, 13 August; open circles, 17 August. Solid lines are the regression of the logit of disease severity on distance over the range 4.5–26.5 m, with slopes pooled (4) for 9 August to 17 August (data for 11 August and 15 August were included in the analysis, but for clarity are not shown in the figure). Dotted lines are unpooled regressions for 3 and 6 August.

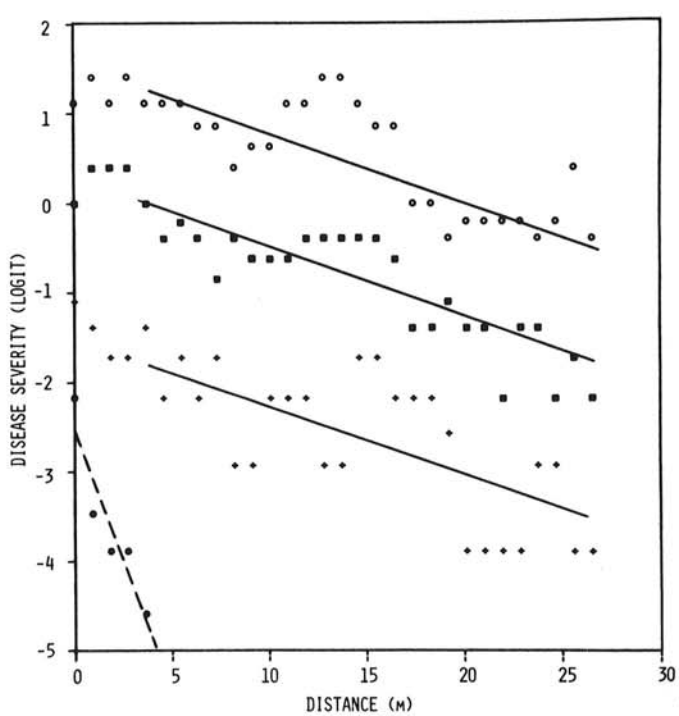

Fig. 3. Potato late blight severity as a function of distance from the point of inoculation, for plot 8 (fungicide-sprayed Katahdin). Observation dates are: large dots, 11 August; crosses, 16 August; squares, 20 August; open circles, 26 August. Solid lines are the regression of the logit of disease severity on distance over the range 4.5–26.5 m, with slopes pooled (4) for 16 to 29 August (data for 23 and 29 August were included in the analysis, but for clarity are not shown in the figure). Dotted line is the unpooled regression for 11 August.

estimation, since g and v are poorly defined close to the point of inoculation. This is because the traveling wave, which we have used to characterize disease spread, is a steady state phenomenon that requires time and space to develop, and it is not apparent in the immediate vicinity of a point inoculation. Also, apparently because of the cross-contamination or environmental heterogeneity, several plots showed an increase in disease at the end farthest from the point of inoculation (the SE end). To minimize the effects of this artifact, data from the last 10 cells (9.2 m) of each plot were discarded.

With these exceptions, the disease gradients were approximately linear on the logit scale. This linearity reflects only the average gradient: on a finer scale, the focal nature of disease spread produced noticeable fluctuations about the mean gradient. After the gradients had stabilized, the apparent infection rate in all plots showed no trend with distance (Fig. 5). This is consistent with the prediction that disease spreads as a traveling wave. The rate of spread of the wave was approximately constant over the limited time of observation (Fig. 6).

The onset of a stable gradient and a constant velocity was taken as an indication that the epidemic had reached an approximately steady state, and was used as the criterion for selecting values of g to be pooled for the final estimates. Local values of r were calculated for the same time period, and were pooled over the same range of locations that had been used in calculating g.

The estimated values of g, r, and v for each treatment are shown in Table 1. The apparent infection rate r, as expected, was significantly lower for plots of sprayed (F) or resistant (R) potatoes than for the unsprayed plots of susceptible potatoes (U). The same relationships were true for g. Surprisingly, the velocity of spread did not differ significantly among treatments.

TABLE 1. Estimated values of the gradient parameter (g), the apparent infection rate (r), and the velocity of spread (v) for susceptible unsprayed (U), resistant (R), and fungicide-treated (F) plots of potatoes infected with *Phytophthora infestans*. Each value is the mean of two replicates. In each column, values followed by different letters are significantly different at $P = 0.95$ by Fisher's LSD test

Treatment	g (m^{-1})	r (day^{-1})	v (m/day)
U	0.156 a	0.465 a	2.98 a
R	0.037 b	0.163 b	4.44 a
F	0.065 b	0.235 b	3.62 a

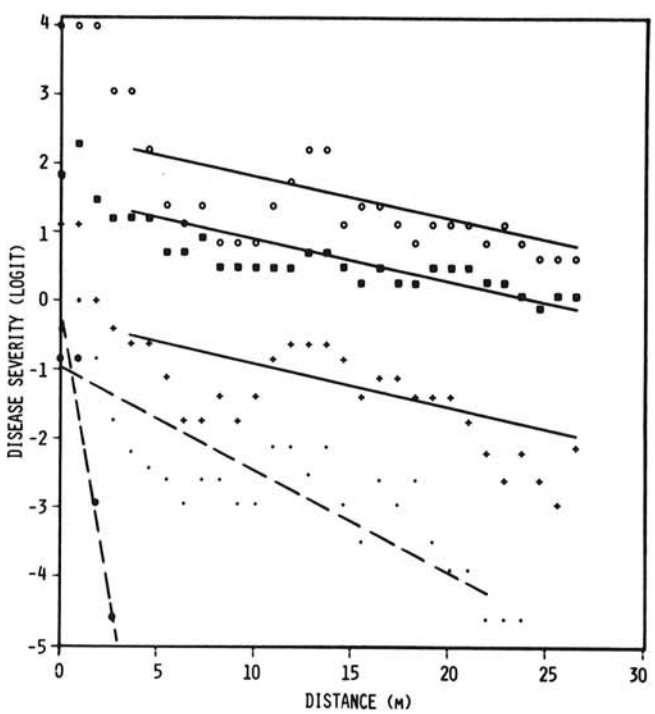

Fig. 4. Potato late blight severity as a function of distance from the point of inoculation for plot 5 (Sebago). Observation dates are: large dots, 6 August; small dots, 11 August; crosses, 14 August; squares, 20 August; open circles, 29 August. Solid lines are the regression of the logit of disease severity on distance over the range 4.5 to 26.5 m, with slopes pooled (4) for 14 to 29 August (data for 17, 23, and 26 August were included in the analysis, but for clarity are not shown in the figure). Dotted lines are unpooled regressions for 6 and 11 August.

DISCUSSION

The results of these experiments are for the most part consistent with the predictions of our models of disease spread. The logit transformation provided a useful linearization of the disease gradient, which allowed meaningful estimates to be calculated for the gradient parameter g and the velocity of spread v. As predicted by the traveling wave concept, g and v were constant over time and r

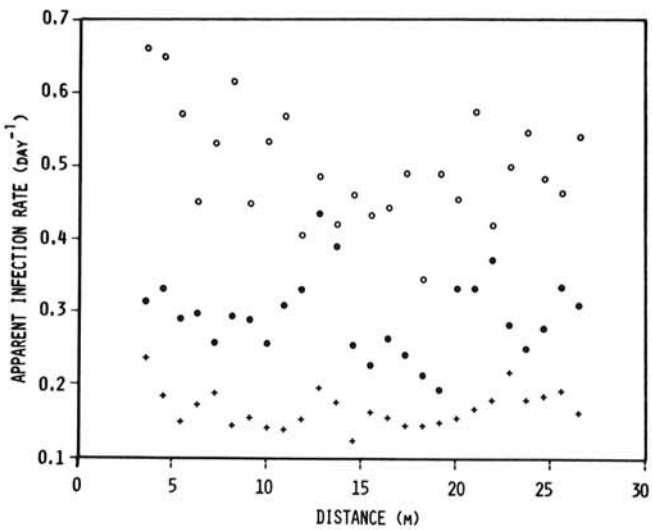

Fig. 5. Local apparent infection rate at various distances from the point of inoculation. Open circles, plot 4 (unsprayed Katahdin); dots, plot 8 (fungicide-treated Katahdin); crosses, plot 5 (Sebago). Time periods over which infection rates were calculated are: plot 4—9 to 17 August; plot 8—16 to 29 August; plot 5—14 to 29 August.

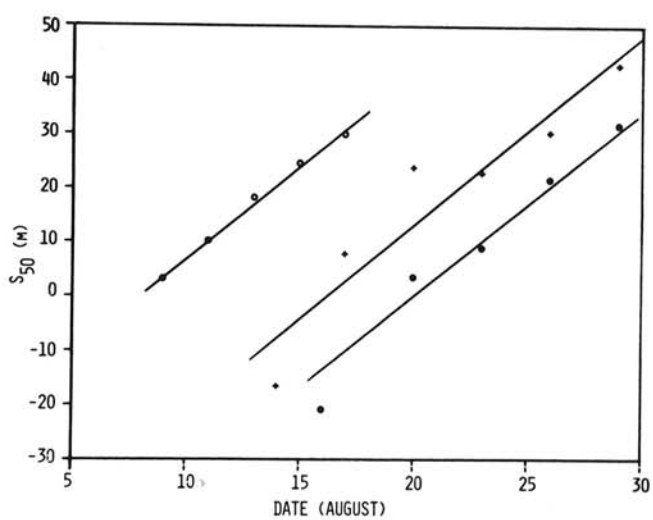

Fig. 6. Estimated value of s_{50}, the distance from the point of inoculation at which disease severity equals 50%, as a function of the date of observation for plot 4, unsprayed Katahdin (open circles); plot 5, Sebago (crosses); and plot 8, fungicide-treated Katahdin (dots). Lines are the regression of s_{50} on time; the slopes of these lines are used as estimates of v, the velocity of spread.

was constant over distance; of course, this constancy is to some extent dependent on the limited time and space scales within which the experiments were carried out. The flattening of the gradient that occurred in sprayed and resistant plots was also expected on the basis of our models.

Because these treatments decreased g to approximately the same extent that they decreased r, the velocity v (which equals r/g) remained essentially constant for all treatments. This was unexpected and remains puzzling; we had predicted that v would respond in much the same manner as r. It may be, because of the very flat gradients encountered, that our plots were too short to accurately estimate v; further work, both theoretical and experimental, seems necessary to resolve this point.

The magnitude of the estimates of v, averaging 3.7 m/day, is quite small, especially when disease spread throughout a large region is considered. This appears to be an effect of plot size. Dispersal by a variety of methods produces patterns of disease on a variety of scales, and what appears as a gradient on one scale may be only a local fluctuation of disease severity when the scale of observation is increased. For *P. infestans* and many other fungi, the importance of wind as a dispersal agent increases relative to rain at larger scales. As a result the gradient can be expected to appear flatter, and the velocity faster, as the scale of observation increases. Such behavior has been observed in our stochastic model of disease spread. The scale of our experiments is appropriate to spread around a single focus; for such spread a velocity of 3.7 m/day seems reasonable.

The rapid flattening of the gradient that occurs in the initial stages of the epidemic implies that, until a steady state is reached, the apparent infection rate increases with distance from the focal center. The following mechanism is proposed to explain this phenomenon. When the gradient is steeper than its steady state value, regions of high and low disease severity are relatively close to each other. As a result of dispersal, regions of high severity will, under such conditions, show a net loss of sporangia, while regions of low severity will show a net gain. The apparent infection rate will then be higher where disease severity is low. The effect of this imbalance is to flatten the gradient, and this flattening will continue until the distribution of disease is such that losses and gains of sporangia are balanced at every point. The gradient will then remain stable.

A similar mechanism may be involved when the gradient flattens in response to a decrease in the multiplication rate. We have observed in our simulation results that such flattening occurs only when latent and infectious periods are included in the model (*unpublished*): if the latent period is zero and the infectious period is infinite, then the gradient shows no response to changes in the multiplication rate. This seems to imply that the observed flattening is somehow related to the age distribution of lesions, which is in general different in a slowly developing epidemic (small r) than in a rapidly developing one. The details of this relationship remain elusive; once again the need for further research is clear. Relationships such as this, however, demonstrate how intimately spatial and temporal factors interact, and how important it is to consider factors of both kinds together if we are to understand the nature of disease spread.

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