A Modification of Gregory's Model For Describing Plant Disease Gradients

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ABSTRACT

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Gregory proposed that plant disease gradients can be described by the equation $y = ax^{-b}$ in which y is the number of infections per unit area at distance x from the inoculum source (with $x = 0$ defined as the boundary between source and receptor plants), a is the number of infections per unit area at one unit of distance from the source, and b is a measure of the steepness of the gradient. The model does not predict a finite number of infections at the source and, therefore, has not been used in computerized simulators in which it is necessary to calculate the amount of autoinfection on source plants. We modified Gregory's model to $y = a(x' + c)$ in which a is the number of infections per unit area at $1 - c$ units of distance from the source, x' is the distance from the center of the source to the center of a receptor, and c is a truncation factor that provides for a finite y-intercept when $x' = 0$. The modified model produces curves with shapes similar to those produced by Gregory's original model, but predicts a finite amount of infection on source plants. The modified Gregory model was fit to data from primary disease gradients of oat crown rust, common maize rust, and bean rust by using nonlinear regression. For oat crown rust and maize rust, the model adequately described the gradients and provided reasonable estimates of autoinfection. The model fit the bean rust data less well and overpredicted autoinfection on source plants. Values of c estimated by regression were approximately equal to the radius of the source plant for all three diseases.

Additional key words: *Avena sativa*, corn, epidemiology, *Phaseolus vulgaris, Puccinia coronata, Puccinia sorghi*, spore dispersal, *Uromyces phaseoli, Zea mays*.

Plant disease gradients from a point source of inoculum are often very steep (1,2). Gregory (1) proposed that such gradients can be described by the equation $y = ax^{-b}$ in which y is the number of infections per unit area at distance x from the source of inoculum (with $x = 0$ defined as the boundary between source and receptor plants), a is the number of infections per unit area at one unit of distance from the source, and b is a measure of the steepness of the gradient. The equation can be linearized to give $\log(y) = \log(a) - b[\log(x)]$, which provides a convenient method for calculating the parameters of the equation by linear regression. [Note that the sign of b in this linearized equation is negative, in contrast to the linearized equation given by Gregory (1, p. 191) which contains an error in the log transformation of $y = a/x^{-b}$].

Gregory's model has the disadvantage of predicting an infinite number of infections at the source (4,5). At the source, $x \leqslant 0$ and the equation requires raising zero or a negative number to a negative power, which is mathematically undefined. Prediction of infinity at the source may be of no major concern if one is merely interested in describing and comparing disease gradients. The Gregory model causes difficulties, however, in disease simulators in which it is necessary to quantify the amount of autoinfection (sensu Robinson [11]) (reinfection of a plant by spores produced on that plant). Consequently, Gregory's model was not used in mathematical simulators of epidemic development in multiline cultivars (4,5). MacKenzie (7) used the Gregory model to describe alloinfection (sensu Robinson [11]) (infection by spores produced on other plants) in multiline cultivars, but used a separate term to describe autoinfection.

The purpose of our study was to modify Gregory's model to allow for a gradient curve with a finite y-intercept while maintaining a shape similar to that provided by the original model.

MATERIALS AND METHODS

Description of the model. Gregory's model was modified by adding a constant, c, to give

$$y = a(x' + c)^{-b} \tag{1}$$

in which the parameters are the same as described by Gregory (1) except that a is the number of infections per unit area at $1 - c$ units of distance, x' is the distance from the center of the source to the center of a receptor, and c (the truncation factor) is a positive number that provides for a finite y-intercept when $x' = 0$. The modified Gregory model can be linearized to give

$$\log(y) = \log(a) - b[\log(x' + c)] \tag{2}$$

The effect of the truncation factor in equations 1 and 2 is to shift the curve c units of distance to the left such that the y-axis is intercepted (compare curves A and D in Fig. 1). Of course, merely shifting the curve to the left will not provide an adequate model for describing gradients. If, however, values of a and b are also changed (relative to the those calculated from the original Gregory model), a curve can be obtained that is similar in shape to that provided by the original model but which increases less steeply as the y-axis is approached, and intercepts the y-axis at $x' = 0$ (curve B of Fig. 1).

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Ordinary linear regression can be used to estimate a, b, and c by iteratively choosing values of c and fitting equation 2 to disease or dispersal gradient data by regressing $\log(y)$ on $\log(x' + c)$. In such regressions, a is the antilog of y at $\log(x' + c) = 0$ and the slope of the regression line is $-b$. A less tedious method, however, is to use nonlinear regression to simultaneously estimate the three parameters. With nonlinear regression, either the nonlinear (equation 1) or linear (equation 2) form of the modified Gregory model can be fit to gradient data. In many cases, however, it may be more desirable to fit the linear form of the model to disease or dispersal data because log-transformed values of y often result in better satisfaction of the statistical assumption of homogeneity of variance.

Fit of the model to field data. The modified Gregory model was fit to primary disease gradient data for oat crown rust, common maize rust, and bean rust.

Oat crown rust. Oats (*Avena sativa* L. 'C.I. 7555') were planted on 3 April 1984 at the Peanut Belt Research Station, Lewiston, NC. Prior to planting, seeds were treated with carboxin (75W) at the rate of 0.71 g a.i./kg of seeds. Plots were 6.1 × 6.1 m and were divided into 64 sections by using concrete-reinforcing wire cut into 0.76 × 0.76-m grids. Except for the center four grids, plots were planted by raking back soil in each grid, scattering 200 seeds per grid, and raking the soil back over the seeds. Using concrete-reinforcing wire, we divided the central 1.52 × 1.52 m area (four 0.76 × 0.76-m grids) into 96 0.15 × 0.15-m grids and one 0.30 × 0.30-m section at the center of the plot (Fig. 2). Eight seeds were planted in the center of each 0.15×0.15-m grid. In the center 0.30× 0.30-m section of each plot, 36 holes were made on a 0.05 m equidistant spacing by using wooden dowels connected to a piece of plywood. At least two seeds were planted per hole and these plantings were thinned to one plant per hole on 17 April.

Six plots were planted linearly in an east-west orientation with 6.1 m between plots. The line of plots was located about 6.1 m south of another crown rust experiment that was inoculated simultaneously. Areas between plots and an area ~6-m wide around the experiment were planted to Iowa Multiline E77 using a tractor-drawn grain drill. There was a space of ~0.5 to 1.0 m between the plots and the beginning of the multiline planting. Due to equipment malfunction, the stand in the border strip on the southern edge of the experiment was very sparse. Standard fertilization and weed control practices were used to maintain adequate growth of the crop.

Plots were inoculated on 5 May with *Puccinia coronata* Cda. var. *avenae* Fraser and Ledingham race 264B. The isolate was obtained in 1983 from M. D. Simons and L. J. Michel, Iowa State University, and was maintained on C.I. 7555 and Markton oats in the greenhouse. One plant at the center of each plot was inoculated by injection with a suspension containing 1.0 mg of viable uredospores per milliliter and Tween-20 at two drops per 100 ml in distilled water. Plants were injected in two locations (one below the uppermost and one below the next lower node) until the suspension exuded from the whorl. On 14 May, when pustules resulting from the inoculation were visible but before they erupted, the inoculated plants were removed from three of the plots so that these plots could be used as a check on the background level of infection.

Primary disease gradients were measured in two plots containing artificially inoculated plants on 4 June before the second generation of pustules appeared (disease did not develop sufficiently in the third plot to allow accurate characterization of the gradient). The rather long latent period for oat crown rust may have been due to cool temperatures in May. The total number of pustules on each of the 36 plants in the center of each plot was counted. There were few or no tillers in these center areas, so a plant was considered to consist of a single culm. On the artificially inoculated plant in each plot, new pustules were distinquished from primary ones. Pustules on all culms were counted in 16 of the 0.15× 0.15-m grids and six of the 0.76 × 0.76-m grids (Fig. 2). Each heading culm within these grids was examined individually.

Common maize rust. Maize (*Zea mays* L. 'B37Ht × A632Ht') was planted on 11 May at the Central Crops Research Station, Clayton, NC. Plots were 5.5 × 5.5 m and were divided into 144 sections using concrete-reinforcing wire cut into 0.46 × 0.46 m grids. Two seeds were planted in the center of each grid and plots

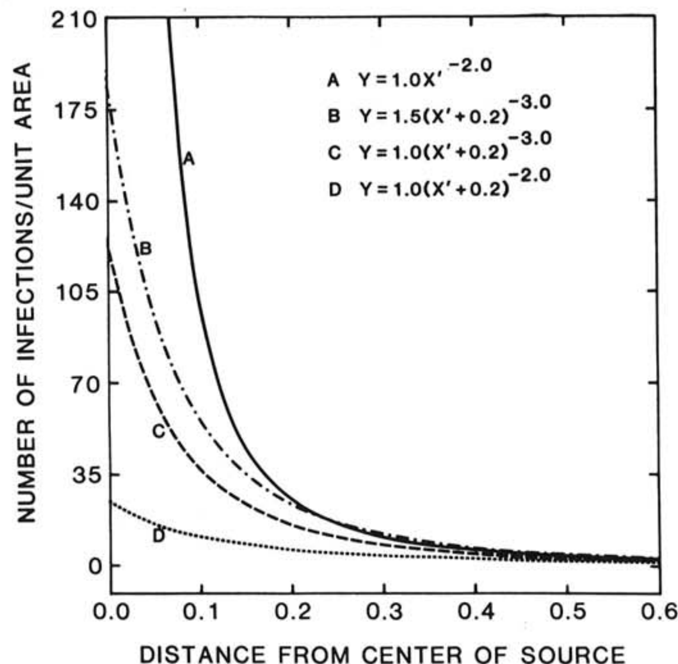

Fig. 1. Gradient curves generated from the original and modified Gregory models. Curve A was calculated from the original Gregory model, but the distance from the center of the source to the center of the receptor (x') was used in the calculations to make the curve comparable with B to D. Curves B to D were calculated from the modified Gregory model with different values of a, b, and c.

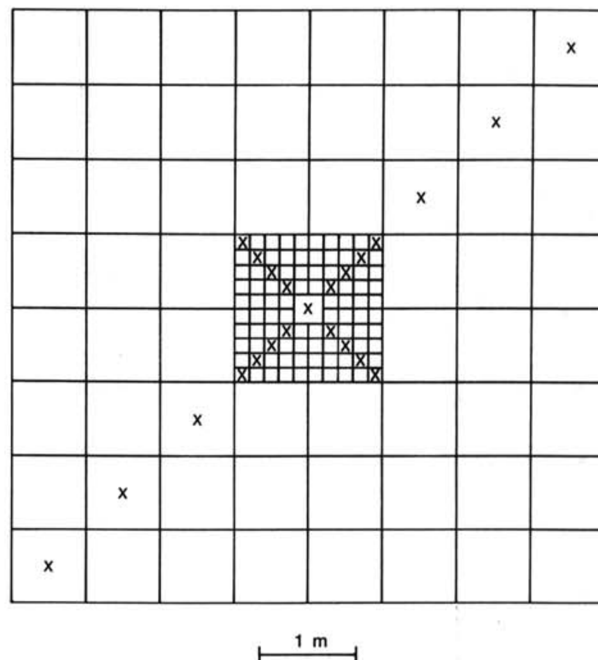

Fig. 2. Representation of planting and sampling designs for obtaining oat crown rust disease gradient data. The entire block represents one field plot. Each of the 60 large squares represents one 0.76×0.76-m planting grid that contained 200 oat seeds. Each of the 96 small squares represent one 0.15× 0.15-m area that contained a hill of eight oat seeds. The square at the center of the figure represents a 0.30×0.30-m area that contained 36 oat plants on a 0.05 × 0.05-m equidistant spacing. Disease was initiated by artificially inoculating a single plant at the center of the 0.30 × 0.30-m planting area. Squares containing an "X" represent planting areas that were sampled to determine the primary disease gradient (see text for details).

were thinned to one plant per grid on 3 June, except for the center four grids which were thinned after disease was established in the plots. Five plots were planted in a line with an east-west orientation, with 4.6 m between plots. The line of plots was located 4.4 m north of another common maize rust experiment that was inoculated simultaneously. Four rows of common maize rust-resistant maize (B37Ht × B14AHt) were planted between the plots and around the perimeter of the experiment, with 1.0 m between rows.

Standard fertilization and weed control practices were used to maintain adequate growth of the crop. Plots were irrigated as necessary to ensure adequate growth of the crop and adequate moisture for rust development.

Plots were inoculated on 2 June with a culture of *Puccinia sorghi* Schw. isolated in 1982 from Clayton, NC, and maintained on field and sweet maize in the greenhouse. Both plants in one of the four central grids of each plot were inoculated twice by injection below the uppermost node with 3 ml of a suspension of uredospores. Tween-20 was added to the suspension at a rate of two drops per 100 ml. The first inoculation was conducted between ~0830 and 1030 hours EDT with a suspension containing 2 mg of viable spores per milliliter of distilled water, and the second was conducted between ~1830 and 2030 hours with a suspension containing 1 mg/ml. The four central grids were thinned to one plant per grid on 9 June when pustules resulting from the artificial inoculation were observed. Both inoculated plants were removed from two of the plots so that these plots could be used as a check on the background level of infection.

Primary disease gradients were measured in the three plots containing artificially inoculated plants on 23 June before there was a second generation of rust increase from the artificially inoculated plants. The total number of new pustules was counted on the artificially inoculated source plant, all eight plants surrounding the source plant, and all plants in lines radiating in eight directions from the centers of the plots to the plot edges.

Bean rust. Snap beans (*Phaseolus vulgaris* L. 'Bush Blue Lake 47') were planted on 21 May 1984 at the Peanut Belt Research Station, Lewiston, NC. Standard fertilization, weed control, and insect control practices were used to maintain adequate growth of the crop.

Plots were 3.6 × 3.6 m and were divided into 576 sections by using concrete-reinforcing wire with 0.15 × 0.15-m grids. At least two seeds were planted in the center of each grid, and the plots were thinned to one plant per grid on 7 June. Six plots were planted in a 2 × 3 matrix with 3.6 m between adjacent plots. Plots were located 3.6 m south of another bean rust experiment that was inoculated simultaneously. Four rows of soybeans (*Glycine max* L. 'Lee') on a 0.9-m spacing were planted between adjacent plots and at least four rows were planted on each side of the experiment.

The plots were inoculated between 1900 and 2100 hours EDT on 13 June with *Uromyces phaseoli* (Reben) Wint. collection 16. The isolate of *U. phaseoli* was obtained in 1982 from J. R. Stavely, Applied Plant Pathology Laboratory, Beltsville, MD, and was maintained on Bush Blue Lake 47 snapbeans in the greenhouse. A single trifolioliate leaf was inoculated on a plant near the center of each plot with a suspension containing 0.5 mg of uredospores per milliliter of distilled water to which two drops of Tween-20 per 100 ml had been added. The suspension was applied to the point of runoff with a gas-propellant, thin-layer chromatography sprayer. The inoculated plants were immediately covered with moistened plastic bags, which were removed between 0830 and 0900 hours EDT the following day. On 20 June, the inoculated leaves were trimmed, leaving only one infected leaflet per plot.

Primary disease gradients were measured on 2 July before there was a second generation of increase from the artificially inoculated plants. In only two plots did disease develop sufficiently to allow gradients to be characterized. In one of these plots, the number of new pustules was counted on the source plant and on plants in eight lines radiating from the source plant to the plot edges. In the other plot, new pustules were counted on the source plant and on plants in four lines radiating at right angles to each other from the source plant to the plot edges; there were too many missing plants in the other four lines to obtain accurate gradient data.

Data analysis. With all three diseases, sampling units were the plant(s) contained within a planting grid and the number of pustules counted within a sampling unit was considered to be a mean that was located at the center of the sampling unit. With oat crown rust, the sampling units consisted of single culms at the very center of the plots, groups of 3–10 (mean, 6.7) culms in the 0.15 × 0.15-m grids, and groups of 134–201 (mean, 173.8) culms in the 0.76 × 0.76-m grids. For oats, data were expressed as the mean number of pustules per culm for each sampling unit. In the maize and bean experiments, the sampling units were always single plants and data were expressed as the number of pustules per plant. For oat crown rust and common maize rust, pustule counts were averaged over all sampling units at the same distances from the source and over all plots. For bean rust, means of observations from the same distances were analyzed separately for each of the two plots because a different sampling scheme was used in each, and because the amount of disease differed greatly between them. For all three diseases, models were fit to the means. Because source plants were not exactly at plot centers, there were missing plants, and because there was a different number of plots for each of the diseases, the number of observations per mean ranged from 1 to 16 for oat crown rust, from 3 to 16 for maize rust, and from 1 to 4 for each of the two bean rust plots. Means from sampling units at plot edges were not used because there seemed to be an edge effect on pustule counts. With oat crown rust and bean rust, zero observations were replaced with the smallest nonzero observation in the data sets (0.01 for oat crown rust and 1 for bean rust) to permit logarithmic transformation of pustule counts. There were no means of zero for the maize rust data.

The linear form of the modified Gregory model (equation 2) was fit to disease gradient data by using non-linear regression [PROC NLIN, using the derivative-free option (12)]. Base-10 logarithms were used in the model. The coefficient of determination was calculated as $R^2 = 1 -$ residual sums of squares/total corrected sums of squares.

RESULTS

Background levels of disease were low and did not interfere with characterization of gradients from artificially inoculated plants. Very few crown rust pustules occurred at edges of inoculated oat plots, so it was deemed unnecessary to make detailed counts of infection in check plots from which the inoculated plants had been removed before spore dispersal occurred. In the two check plots of maize, mean numbers of rust pustules per plant for 10 plants sampled in an X-pattern were 5.6 and 4.9. With bean rust, a plot in which the artificial inoculation had been unsuccessful was chosen for evaluation of the background level of disease. No pustules were found in this check plot except on leaves adjacent to a few large gaps in the crop canopy.

With both oat crown rust and common maize rust, the linear form of the modified Gregory model fit the disease gradient data well and provided reasonable estimates for the number of new infections on source plants (Fig. 3A and B). The smaller number of observed than of predicted pustules on source plants for oat crown rust probably resulted from the source plant in one of the two plots being stunted (perhaps due to the inoculation procedure) and bearing not many more pustules than adjacent plants. Plots of residuals (observed minus predicted numbers of pustules) versus distance for both oat crown rust and common maize rust data showed nonsystematic variation from the model, except for a tendency for the residuals from the oat crown rust data to be smaller for points near the inoculum sources than for those farther away.

With bean rust, the modified Gregory model overpredicted the number of new pustules on the source plant for both plots (Figs. 3C and D). For data of Fig. 3C, the observed and predicted numbers of new pustules were 3,451 and 7,169, respectively. The corresponding values for data of Fig. 3D were 1,121 and 1,755. For one of the plots, the model also tended to underpredict the number of pustules

on plants less than 1 m from the source (Fig. 3C). For the other plot (Fig. 3D), there was no obvious systematic pattern in the residuals.

Curves calculated from the modified Gregory model had a shape similar to those calculated from the original model, as shown for oat crown rust in the comparison of Fig. 3A with Fig. 4. Similar results were obtained for common maize rust and bean rust, with calculated values of a being 40.031, 15.988, and 16.666, and calculated values of b being 2.015, 2.896, and 1.903 for data of Figs. 3B, C, and D, respectively.

We obtained evidence which suggested that the truncation factor is proportional to the size of the inoculum source. In fitting the linear form of the modified Gregory model to our field data, we obtained c-values of 0.0378, 0.248, and 0.219 m for oat crown rust, common maize rust, and bean rust (mean of the two plots), respectively. Assuming source plants to be completely contained within planting grids, we calculated radii of source plants to be 0.0307, 0.276, and 0.184 for oats, maize, and beans, respectively.

DISCUSSION

Model performance. For oat crown rust and common maize rust, the modified Gregory model adequately described disease gradients, provided reasonable estimates of autoinfection on source plants, and produced a curve with the same basic shape as that from the original model. The coefficients of determination for oat crown rust and common maize rust were reasonably high and the model fit equally well over the range of distances evaluated. When the modified Gregory model was fit to the bean rust data, coefficients of determination were lower and predictions of autoinfection on source plants were less accurate than those for oat crown rust and common maize rust. The lower coefficient of determination for bean rust was partly due to variability in plant stand caused by damping-off in the plots. Overprediction of autoinfection on source plants for bean rust may be due to a difference in canopy structure between beans and the two grasses that could cause the gradient to decrease less steeply near the source with bean rust. For example, bean plants may overlap each other more than oat or maize plants do, and this might obscure the actual relationship between the number of infections per plant and distance from the source.

We have found nonlinear regression to be a fast and inexpensive procedure for fitting the modified Gregory model to disease gradient data. We would strongly suggest fitting the linear form of the modified Gregory model to disease or dispersal gradient data

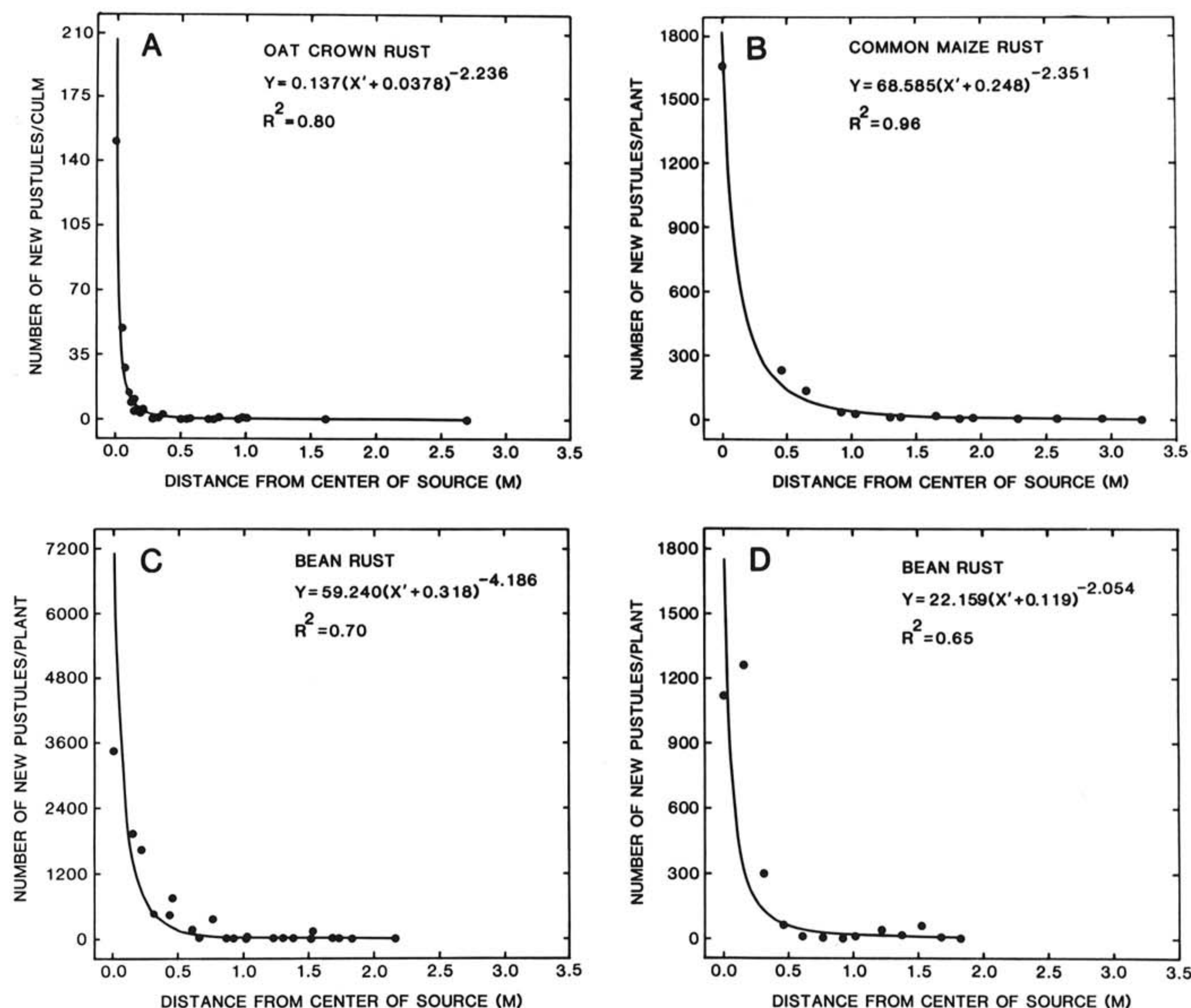

Fig. 3. Primary disease gradient data for three rust diseases from field plots with single infected source plants. Each point is a mean of all observations for sampling units at the same distance from the source (see text for details). Curves were derived by fitting the linear form of the modified Gregory model, $\log(y) = \log(a) - b[\log(x' + c)]$ to disease gradient data. Data represented in A and B are means over two and three plots, respectively, those in C and D are data from single plots.

when it will better satisfy the assumption of homogeneity of variance. When we fit the nonlinear form of the model to our oat crown rust and common maize rust data, the resulting equations overpredicted at the farther distances from the source because a disproportionate weight was given to the very large variances of observations close to the source. This is because plants close to the source will have a larger number of infections and, therefore, greater potential variation. In some cases (e.g., with our bean rust data in Fig. 3C) it may be desirable to use the nonlinear model to get a closer fit near the source if this is the area that is of most interest. Gregory (1, page 191; 2, page 249) suggested fitting his model by using weighted linear regression "... giving less weight to low counts, which are usually unreliable and confounded with background contamination."

An advantage of Gregory's model is that the slope, b, can be used to compare gradients among pathogens or experimental treatments. With the modified model, however, the calculated value of b depends on the value of the truncation factor, c. One way to compare gradient steepnesses with the modified Gregory model is to calculate the distance from the source at which the number of infections is decreased by some given proportion relative to the number of infections on the source. For example, we calculated that the distance at which the predicted number of pustules was reduced by 90% (relative to the predicted number of pustules on the source plants) was 0.07 and 0.41 m for oat crown rust and maize rust, respectively.

For all three diseases, values of c derived by regression were approximately equal to the radius of the source plant. This result suggests that c may have a biological or physical meaning. A precise mathematical or biological definition for c remains elusive, however, and we have obtained evidence which suggests that c is proportional (but not necessarily equal) to the size of the sampling unit that contains the source rather than the size of the source per se (*unpublished*).

Predicting autoinfection. If predicting autoinfection on source plants is critical to a study, it would be best to measure autoinfection rather than estimate it by extrapolating a model to the y-axis. Autoinfection on source plants has rarely been measured, however, and it may sometimes be necessary to estimate this parameter by extrapolation. When we fit the modified Gregory model to our field data but removed observations from source plants, we calculated values of a, b, and c that yield predictions of inaccurate or even infinite amounts of autoinfection. When, however, we let c equal the radius of the source plant, we derived equations that provide biologically reasonable estimates of autoinfection for the source, in most cases. When we let c equal the radius of the source, the estimated numbers of new infections on source plants was 313, 1,823, 21,830, and 1,773 for oat crown rust, common maize rust, and the two bean rust plots, respectively, as compared with the corresponding measured values of 150, 1,658, 3,451, and 1,121. Therefore, in absence of biological data for autoinfection, a reasonable procedure would be to let c equal the radius of the source and estimate a and b by regression. A procedural advantage to assigning rather than estimating a value for c is that the model can then be fit with ordinary linear regression by regressing $\log(y)$ on $\log(x' + c)$.

There are other gradient models that also have a finite y-intercept and, therefore, allow prediction of autoinfection. Kampmeijer and Zadoks (4) used a model based on equations of Pasquill (9) to describe dispersal in a computerized simulation model. This dispersal model describes a Gaussian distribution, however, and gives a curve shape very dissimilar from those based on disease gradient data that we collected in the field and have found in the literature. Kiyosawa and Shiyomi (5) used an exponential function to describe spore dispersal in multiline cultivars. Kiyosawa and Shiyomi's model has been used to study interplot interference (10) and has been incorporated into equations that describe simultaneously the spatial and temporal increase of disease (3). We have found that Kiyosawa and Shiyomi's model often underpredicts the number of infections near the source with rust diseases. Paysour and Fry (10) found that Kiyosawa and Shiyomi's model described disease gradients well for potato (*Solanum tuberosum* L.) late blight [induced by *Phytophthora infestans* (Mont.) de Bary]. The data they reported, however, showed no observations closer than 1 m from the source. Lambert et al (6) described a gradient model that allows for the curve to assume different shapes, including the shapes of the Gregory, Kampmeijer and Zadoks, and Kiyosawa and Shiyomi models. In limited studies, we have found that it is difficult to fit this model to disease gradient data by using a desk calculator and that PROC NLIN (12), a nonlinear regression program, often fails to converge on a realistic solution.

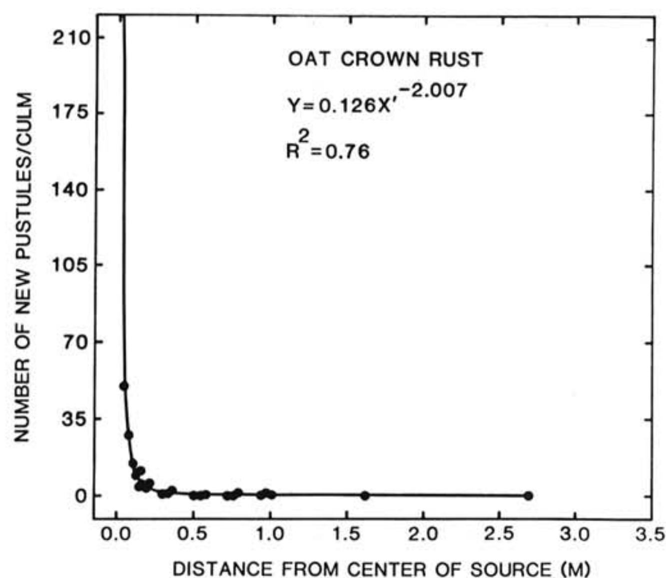

Fig. 4. The model $\log(y) = \log(a) - b\,[\log(x')]$ fit to oat crown rust dispersal data; this model is the same as the linear form of the original Gregory model except that the distance from the center of the source to the center of the receptor (x') was used so that the curve would be comparable with that of Fig. 3A. Data are the same as in Fig. 3A except that the observation at $x' = 0$ was not used in the analysis.

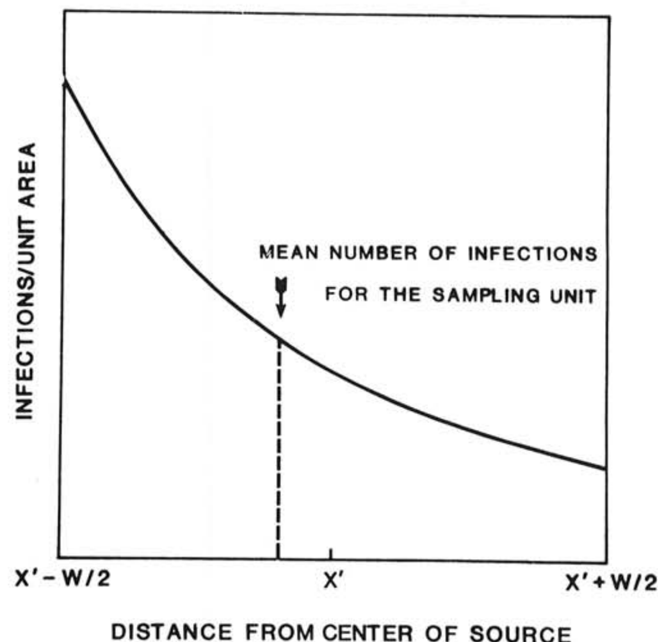

Fig. 5. Diagrammatic representation of a disease gradient over a sampling unit showing the location of the mean infection density. The center of the sampling unit is represented by x'; W = the width of the sampling unit. The diagram was derived by using the disease gradient equation from Fig. 3B and assuming a sampling unit of 0.5 m in diameter with a center located 0.5 m from the center of the source.

McCartney and Bainbridge (8) recently described a method for mathematically estimating autoinfection from gradients of integrated spore deposition. Their method requires calculating or estimating several physical and biological parameters, however, and we have not attempted to use their method with our gradient data.

Use of the model in computerized simulations. One use for the modified Gregory model is to describe spore dispersal in computerized simulators that model the spatial distribution of disease. In the modified Gregory model, the constant a is proportional to the source strength. In simulating an epidemic, however, each infected plant becomes a source, with the source strength varying among plants and over time. Therefore, the dispersal equation must be modified to describe probability density, i.e., the relative proportion of total inoculum from the source that is deposited in an area at any distance from the source.

Others have arrived at an expression of probability density by calculating or approximating the integral of a gradient equation and setting the area under the volume to one (4,10). We attempted the same procedure with our model and found the integral to be an innaccurate descriptor of disease gradient data collected from the field. We believe that this inadequacy of the integral is due to the manner in which the gradient data were obtained and is probably relevant to most or all gradient models. We collected disease gradient data as the number of pustules per sampling unit (usually per plant) and recorded their positions as single locations at the centers of sampling units. In reality, the mean pustule density does not occur at the center of the sampling unit, but at some distance closer to the source. This is because leaves on the side of a plant that is nearer to the source are likely to have more pustules than the leaves on the opposite side, and because the disease gradient is nonlinear (Fig. 5). Thus, data used in calculating or approximating the integral are inaccurately placed in relation to distance from the source. This inaccuracy could be corrected by estimating the real location of the means or by finding a mathematical conversion between equations based on assumed versus actual locations. In our computerized simulation studies of epidemic development in host mixtures, however, we have chosen to use a method that allows for calculation of mean relative deposition into subunits of the host population without the need for integrating the equation (*unpublished*).

LITERATURE CITED

1. Gregory, P. H. 1968. Interpreting plant disease dispersal gradients. Annu. Rev. Phytopathol. 6:189-212.
2. Gregory, P. H. 1973. The Microbiology of the Atmosphere. John Wiley and Sons, New York. 377 pp.
3. Jeger, M. J. 1983. Analysing epidemics in time and space. Plant Pathol. 32:5-11.
4. Kampmeijer, P., and Zadoks, J. C. 1977. EPIMUL, a Simulator of Foci and Epidemics in Mixtures of Resistant and Susceptible Plants, Mosaics and Multilines. PUDOC (Centre for Agricultural Publishing and Documentation), Wageningen, Netherlands. 50 pp.
5. Kiyosawa, S., and Shiyomi, M. 1972. A theoretical evaluation of mixing resistant variety with susceptible variety for controlling plant diseases. Ann. Phytopathol. Soc. Jpn. 38:41-51.
6. Lambert, D. H., Villareal, R. L., and MacKenzie, D. R. 1980. A general model for gradient analysis. Phytopathol. Z. 98:150-154.
7. MacKenzie, D. R. 1979. The multiline approach to the control of some cereal diseases. Pages 199-216 in: Proceedings of the Rice Blast Workshop. Int. Rice Res. Inst., Los Baños, Philippines. 222 pp.
8. McCartney, H. A., and Bainbridge, A. 1984. Deposition gradients near to a point source in a barley crop. Phytopathol. Z. 109:219-236.
9. Pasquill, F. 1962. Atmospheric Diffusion: The Dispersion of Windborne Material From Industrial and Other Sources. Van Nostrand, London. 297 pp.
10. Paysour, R. E., and Fry, W. E. 1983. Interplot interference: A model for planning field experiments with aerially disseminated pathogens. Phytopathology 73:1014-1020.
11. Robinson, R. A. 1973. Plant Pathosystems. Springer-Verlag, New York. 184 pp.
12. SAS Institute, Inc. 1982. SAS User's Guide: Statistics. SAS Institute, Inc., Cary, NC. 584 pp.