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Use of the Modified Gregory Model to Describe Primary Disease Gradients of Wheat Leaf Rust Produced From Area Sources of Inoculum

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ABSTRACT

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Primary disease gradients of wheat (*Triticum aestivum*) leaf rust (induced by *Puccinia recondita*) were studied around 3.66- × 3.66-m sources of infection in two fields in both Pendleton and Corvallis, OR. The gradients were described well by the modified Gregory model, $y = a(x' + c)^{-b}$, in which y is the number of infections per unit area, 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 of spores, c is a truncation factor that provides for a finite y-intercept when $x' = 0$, and b is a measure of the steepness of the gradient. The model-fitting showed that the modified Gregory model can be used to describe disease gradients away from area sources of inoculum in addition to gradients away from single source plants, for which the model was originally developed. For the Pendleton data, the truncation factor closely approximated the radius of the source when this parameter was estimated by nonlinear regression. For the Corvallis data, estimated values of c for the two fields were considerably less than the radius of the source. The modified Gregory model always provided a better fit to the data than did the Kiyosawa and Shiyomi model, an exponential function often used to describe gradients because it predicts a finite number of infections or propagules at the source.

Additional keywords: epidemiology, spore dispersal.

Dispersal of plant pathogenic propagules is a key factor determining the epidemiology of foliar plant diseases and has important implications for the management of those diseases. Many studies of plant pathogen dispersal have involved introducing an inoculum source into a field, measuring the number of propagules or the resulting infections at different distances from the source, and studying the resulting gradients. The fitting of simple mathematical models to such data can aid in the interpretation of dispersal or disease gradients (5,6), e.g., by comparing estimated gradient slopes for different populations or treatments. In recent years, several comparisons have been made of different models for describing spore or disease gradients (1–3,8,12,14,16).

Most gradient studies fail to provide information about propagule or disease density at the source. Failure to provide such information may be due to the fact that spore or disease density is often very high and, therefore, difficult to measure at the source. In addition, source plants are sometimes treated differently than other plants in the population, e.g., when inoculated, greenhouse-grown transplants are used to initiate a gradient. Thus, data derived from the source plants may be of limited relevance.

Studies of gradients not including the source have provided very useful information about pathogen dispersal and disease spread and are useful for comparing different epidemics. However, such studies do not provide information required for quantifying and modeling certain epidemiological processes. For example, the amount of inoculum exchange among plants influences the effectiveness of using host mixtures for disease control (3,10,11,19). Similarly, the amount of interplot interference in an experimental field trial depends on the amount of inoculum exchange among plots (7,21). If dispersal studies do not measure the proportion of inoculum that is retained at the source, it is not possible to calculate the amount of inoculum exchange. One alternative to actually measuring deposition at the source is to use mathematical models to extrapolate back to the source, a procedure that can be very inaccurate (18). McCartney and Bainbridge (15) developed a mechanistic model to estimate the

amount of inoculum deposited at the source, but the model has not been compared with field data.

A few studies have provided measurements of the number of infections on source plants when working with spore dispersal or primary disease gradients of rust diseases. Roelfs and Martell (23) trapped uredospores of *Puccinia recondita* Rob. ex Desm. in and around groups of 10 wheat culms inoculated with the fungus. Leonard (13) injected oat (*Avena sativa* L.) plants in situ with a suspension of uredeospores of *P. graminis* Pers. and studied primary disease gradients around groups of two or three infected plants. He found that 5–10% of the infections counted in the 1.8- × 1.8-m sampling areas were retained on the originally inoculated plants. Mundt and Leonard (18) used similar methodology in studying primary disease gradients of oat crown rust (induced by *Puccinia coronata* Corda) and common maize (*Zea mays* L.) rust (induced by *Puccinia sorghi* Schw.), but inoculated a single culm at the center of each plot. They found that the gradients could be described well by the modified Gregory model (18),

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

in which y is the number of infections per unit area, 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 sampling unit, c (the truncation factor) is a positive number that allows for prediction of a finite number of infections at the source, and b is a measure of the steepness of the gradient. In fitting this model to primary disease gradient data for oat crown rust and common maize rust using nonlinear regression, Mundt and Leonard (18) found that c approximated the radius of the source plant.

Fewer data are available from studies of gradients that include measurements of spore or infection density at and away from larger sources of inoculum. Roelfs (22) used impaction spore traps to quantify the number of spores of *P. recondita* and *P. graminis* directly over and at different distances from a 72-m-diameter area of inoculated wheat. Only one field was studied, however. In addition, spores were not trapped within the crop canopy, thus making it difficult to predict the number of spores that would have been deposited on leaves. Kingsolver et al (9) reported on disease gradient data collected from the U.S. Virgin Islands where stem rust severity was studied away from a single, infected wheat field (2.4 ha) in different years. Mundt and Brophy (17) found that the modified Gregory model could be used to describe the gradient for a data set incorporating observations up to 10.5 km from the source when c was assumed to be the distance from the center to the edge of the source field. However, the data of Kingsolver et al (9) were only for the downwind direction from the source, and Mundt and Brophy (17) analyzed data from only 1 yr of the study. Thus, data are needed from replicated trials where gradients are measured in multiple directions from the source.

The objectives of the studies reported in this paper were to determine if the modified Gregory model can be used to describe disease gradients from inoculum sources larger than that of single plants and to determine if the truncation factor of that model approximates the radius of large inoculum sources. An additional objective was to compare the fit of the modified Gregory model with that of the Kiyosawa and Shiyomi model (11), an exponential function that has been used to describe gradients because it predicts a finite number of infections or propagules at the source.

MATERIALS AND METHODS

Field plots. Field trials were conducted at Pendleton and Corvallis, OR. The Pendleton site is east of the Cascade Mountains in the Columbia Basin, with an average annual rainfall of about 450 mm. The Corvallis site is 480 km from Pendleton in the Willamette Valley, where the average annual rainfall is about 1,050 mm. The mean monthly wind movement for April–May 1987 (the time period that included inoculations and evaluations of the gradients) was 3,078 and 2,202 km at Pendleton and Corvallis, respectively (20). The mean monthly rainfall for this same period was 31.2 and 37.6 mm, the mean monthly high temperature was 21.6 and 19.9 C, and the mean monthly low temperature was 5.2 and 6.2 C at Pendleton and Corvallis, respectively (20).

Differences in climate, soil, and indigenous diseases required that different cultivars and cultural practices be used at the two locations. The traditional-height, club-type winter wheat cultivar Moro (C.I. 13740) was planted in Pendleton at the rate of 78 kg/ha on 15 October 1986. Row-spacing was 0.26 m. Rows were oriented east-west in field A and north-south in field B. In Corvallis, the semidwarf, club-type winter wheat cultivar Tyee (C.I. 17773) was planted at the rate of 113 kg/ha on 14 October 1986. Row spacing was 0.18 m, and rows were oriented north-south in both fields. Corvallis fields A and B were approximately 0.6 and 0.4 ha, respectively, and Pendleton fields A and B were approximately 2.4 and 1.0 ha, respectively. Surrounding fields were mostly free of vertical obstructions, except that there were a considerable number of trees nearby Corvallis field B. The two fields at each site were separated by at least 300 m. There was always a minimum of 3 m of wheat, and usually considerably more, surrounding the area that was sampled in each field. Cultural practices customary for commercial wheat production were used at both locations, except that herbicide application was inadvertently omitted at the Corvallis site.

A 3.66- × 3.66-m area was inoculated in each of the fields on 15 and 9 April 1987 in Pendleton and Corvallis, respectively. Inoculations were done with a suspension of uredospores of *P. recondita*, race 2. The spore concentration was 1 mg per milliliter of distilled water and a surfactant (Tween 80) was added at the rate of one drop per 50 ml. The suspension was injected into leaf sheaths with a repeating syringe until the suspension exuded from the whorl. Plants were at the stem-extension stage at the time of inoculation. One tiller was inoculated every 15 cm within each row in the 3.66- × 3.66-m areas. In Pendleton, there were 14 rows within each inoculated area, while at Corvallis the narrower row-spacing provided 20 rows per inoculated area.

Disease assessments. Primary disease gradients were measured by assessing disease after the first generation of spread occurred from the originally inoculated areas (sources), but before the next generation of disease increase. The disease data were taken on 20 May in Pendleton and 14 and 15 May in Corvallis by counting the number of leaf rust pustules on the leaf below the flag leaf (the penultimate leaf) for plants in 17 3.66- × 3.66-m sampling units in each field. One sampling unit was the source, and the other 16 were located in four lines emanating from the source (Fig. 1).

A total of 28 and 40 culms per sampling unit were observed at Pendleton and Corvallis, respectively. These culms were sampled from two intersecting diagonals that formed an X-pattern in each sampling unit (Fig. 1). One culm was randomly chosen from each position where the X crossed a planting row, and a different observer conducted counts for each diagonal of the X. Pustules resulting directly from the injections in the source area were larger and distinguishable from secondary ones; the former were not included in the counts.

Data analysis and model fitting. Pustule counts from the sampled culms were averaged to give the mean number of pustules per leaf for each sampling unit. For each field, the mean pustule count was averaged over the four sampling units that were at the same distance from the source to give the mean pustule count for each distance. Relative pustule counts were then calculated for each distance from the source by dividing the mean pustule count for each distance by the mean number of pustules per leaf for the source. All models were fit to five data points for each field, i.e., the relative pustule count for the source (distance = 0 m, relative pustule count = 1.00) and the relative mean counts for sampling units that were centered at 3.66, 10.97, 18.29, and 25.60 m from the center of the source.

The modified Gregory model (equation 1) was fit to the data by two methods. In both cases, a linearized form of the model was fit to the data by regressing $\log_{10}(y)$ on $\log_{10}(x' + c)$. The first method of fitting the modified Gregory model was to consider c to be an unknown parameter and to simultaneously estimate a, b, and c

using nonlinear regression (PROC NLIN of the Statistical Analysis System [24], using the DUD method). Direct estimates of the model parameters *a*, *b*, and *c* are obtained from the nonlinear regressions. Although linearization of the modified Gregory model is unnecessary for fitting by nonlinear regression, the log-transformation resulted in the data better satisfying the statistical assumption of homogeneity of variance. The second method of fitting the modified Gregory model was to assume that $c = 1.8288$ m (the shortest distance from the center to the edge of the source unit) and to fit the model using simple linear regression. The shortest distance from the center to the edge of the source was considered to be the radius of the source because gradients were always measured perpendicularly to the four sides of the source area. With the linear regressions, the slope of the regression line for each field provided the estimate of *b* and the antilog of the *y*-intercept for each regression provided the estimate of *a*.

The Kiyosawa and Shiyomi model (11), an exponential function, was also fit to the disease gradient data for all four fields. The Kiyosawa and Shiyomi model is

$$y = Ae^{-dx'} \quad (2)$$

in which y and x' are as in equation 1, A is the number of propagules or infections per unit area at the source, e is the base of natural logarithms, and d is a measure of the steepness of the gradient. A linearized form of equation 2 was fit to the data by regressing $\log_e(y)$ on x', using simple linear regression. The slope of the regression line for each field provided the estimate of d and the antilog of the y-intercept provided the estimate of A.

The fit of the mathematical models to the gradient data were evaluated by using coefficients of determination (r^2) and by observing residual patterns.

RESULTS

The mean numbers of pustules per leaf for the source areas were 363.8 (Pendleton field A), 590.7 (Pendleton field B), 464.3 (Corvallis field A), and 318.7 (Corvallis field B). Disease gradients differed with direction from the source for all four fields, but less so for Corvallis field B than for the other three fields (Table 1). Relative pustule numbers averaged over all directions from the source for the two fields at Pendleton were remarkably similar, and the gradients were steeper than at Corvallis. There was a considerable difference in gradients for the two Corvallis fields, with field B having a more shallow gradient than field A (Tables 2 and 3).

The modified Gregory model provided a better fit to the gradient data than did the Kiyosawa and Shiyomi model for all four fields, regardless of the method of fitting the modified Gregory model. The coefficient of determination (r^2) was always higher for the modified Gregory model than for the Kiyosawa and Shiyomi model (Table 3). The r^2 for the fit of the modified Gregory model to data for the two Pendleton fields was identical, to four significant digits, regardless of whether c was considered to be an unknown and estimated by nonlinear regression or if c was assumed to be the radius of the source and the model fit by linear regression. For the Corvallis data, the modified Gregory model provided a higher r^2 when c was estimated than when c was fixed. The modified Gregory model provided a more random pattern in the residuals than did the Kiyosawa and Shiyomi model, with the latter model consistently underpredicting the number of infections at the source and also at the farthest distance from the source. For the Corvallis fields, a more random pattern in the residuals was obtained when c of the modified Gregory model was estimated by nonlinear regression than when c was fixed (Table 2).

For the Pendleton data, both methods of fitting the modified Gregory model resulted in equations that provided accurate estimates of infection at the source. For the Corvallis data, equations derived by considering c to be an unknown variable provided better estimates of infection at the source than did equations derived by assuming c to be fixed. The Kiyosawa and Shiyomi model was particularly poor at predicting infection at the source; the number of pustules predicted at the source was only 38% of that observed with the data set for which the Kiyosawa and Shiyomi model provided the best fit (Table 2).

For the Pendleton site, strong evidence was derived indicating that c is equal to the radius of the source. Estimates of c derived from nonlinear regression were very close to the source radius

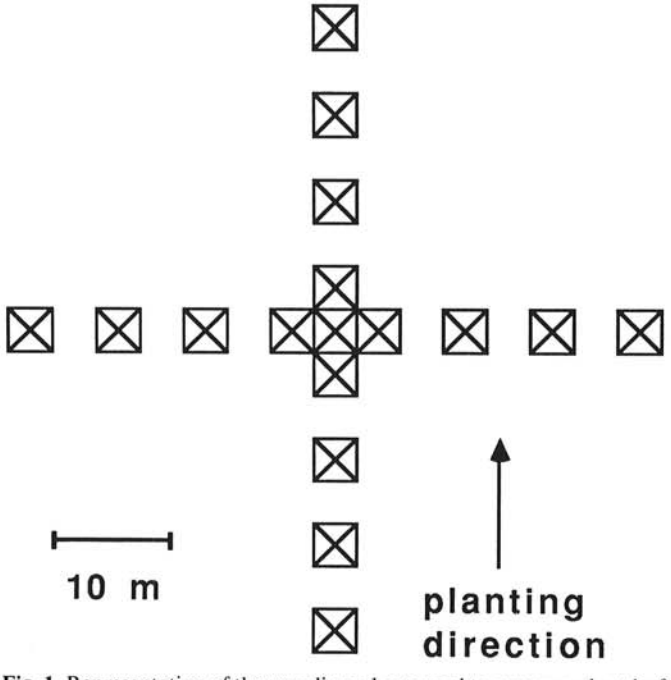

Fig. 1. Representation of the sampling scheme used to measure wheat leaf rust primary disease gradients in the field. Each square represents a 3.66-× 3.66-m sampling unit in a uniform stand of wheat. The X's in each square represent the pattern used to sample individual culms within sampling units. The inoculum source was the sampling unit shown in the center of the figure.

TABLE 1. Observed relative numbers of leaf rust pustules per penultimate wheat leaf in 3.66- × 3.66-m sampling grids at different distances and directions from a 3.66-×3.66-m infection source in two wheat fields at each of Pendleton and Corvallis, OR

Distance[a] (m)	Relative pustules per leaf[b]			
	North	South	East	West
Pendleton field A				
3.66	0.01882	0.05949	0.02081	0.11098
10.97	0.00096	0.00208	0.00296	0.00761
18.29	0.00020	0.00048	0.00049	0.00224
25.60	0.00000	0.00128	0.00012	0.00082
Pendleton field B				
3.66	0.03016	0.06448	0.02642	0.12751
10.97	0.00036	0.00158	0.00268	0.00462
18.29	0.00011	0.00051	0.00024	0.00174
25.60	0.00048	0.00036	0.00024	0.00121
Corvallis field A				
3.66	0.04512	0.14705	0.06024	0.03071
10.97	0.00754	0.02956	0.00930	0.00809
18.29	0.00512	0.01288	0.00732	0.00448
25.60	0.00205	0.00673	0.00542	0.00372
Corvallis field B				
3.66	0.10882	0.19208	0.14823	0.10976
10.97	0.07689	0.10661	0.09999	0.07620
18.29	0.06208	0.08493	0.08064	0.10662
25.60	0.08039	0.09678	0.08360	0.06606

[a] Distance measured from the center of the source area to the center of the sampling unit.

[b] Calculated by dividing the mean number of pustules per leaf in a sampling unit by the mean number of pustules per leaf in the source area of each field.

(Table 3), and this model provided excellent fits to the data when c was set equal to the radius of the source (Tables 2 and 3). Data from Corvallis were very different in this regard. For field A at Corvallis, the value of c derived from nonlinear regression was about half that of the actual radius. The estimate of c for field B was much smaller than the actual radius (Table 3). The modified Gregory model underpredicted infection at the source when c was assumed to be equal to the radius of the source (Table 2), and equations derived with a fixed c provided poorer fits than those derived treating c as an unknown parameter (Tables 2 and 3).

TABLE 2. Observed and predicted relative numbers of leaf rust pustules per penultimate wheat leaf in 3.66- × 3.66-m sampling grids at different distances from a 3.66-×3.66-m infection source in two wheat fields at each of Pendleton and Corvallis, OR

Distance[a] (m)	Relative pustules per leaf			
	OBS[b]	MG1[c]	MG2[d]	KS[e]
Pendleton field A				
0.00	1.00000	1.02849	1.03550	0.25620
3.66	0.05252	1.04481	0.04437	0.09200
10.97	0.00340	0.00393	0.00391	0.01186
18.29	0.00085	0.00107	0.00107	0.00153
25.60	0.00056	0.00044	0.00044	0.00020
Pendleton field B				
0.00	1.00000	1.06615	1.07696	0.24930
3.66	0.06214	0.04288	0.04227	0.08778
10.97	0.00231	0.00350	0.00348	0.01088
18.29	0.00065	0.00092	0.00092	0.00135
25.60	0.00057	0.00037	0.00037	0.00017
Corvallis field A				
0.00	1.00000	1.00359	0.81542	0.29020
3.66	0.07078	0.06749	0.09144	0.14532
10.97	0.01362	0.01511	0.01692	0.03644
18.29	0.00745	0.00719	0.00688	0.00914
25.60	0.00448	0.00437	0.00371	0.00229
Corvallis field B				
0.00	1.00000	1.00002	0.66845	0.37620
3.66	0.13972	0.13423	0.24820	0.28441
10.97	0.08992	0.09805	0.11560	0.16256
18.29	0.08357	0.08472	0.07690	0.09291
25.60	0.08171	0.07695	0.05814	0.05310

[a] Distance measured from the center of the source area to the center of the sampling unit.

[b] Observed relative pustule numbers per leaf. Observed relative pustule numbers were calculated by dividing the mean number of pustules per leaf in a sampling unit by the mean number of pustules per leaf in the source area of each field. Observed relative pustule numbers for nonzero distances were calculated from means of pustule counts for sampling units at four different directions from the source (see Fig. 1).

[c] Predicted relative pustule numbers calculated by fitting the modified Gregory model, $y = a(x' + c)^{-b}$, to the observed data using nonlinear regression and assuming c of the model to be an unknown parameter.

[d] Predicted relative pustule numbers calculated by fitting the modified Gregory model to the observed data and assuming c to be equal to the distance from the center to the edge of the source (1.8288 m).

[e] Predicted relative pustule numbers calculated by fitting the Kiyosawa and Shiyomi model, $y = Ae^{-dx'}$, to the observed data.

DISCUSSION

For the Pendleton data, the modified Gregory model provided a very close fit to primary disease gradients of wheat leaf rust from small sources of inoculum when c was assumed to be equal to the distance from the center to the edge of the source. This result, coupled with observations that the model can describe rust gradients away from both single plants (18) and also from a 2.4-ha field (17), would suggest that c of the modified Gregory model can be considered to be the radius of the source, regardless of source area. For the Corvallis data, however, c of the modified Gregory model did not approximate the source radius, perhaps for reasons that are discussed below.

It is logical that c of the modified Gregory model should approximate the radius of the source. The model predicts the number of infections in a unit area at the center of the source when $x' = 0$, i.e., $y = ac^{-b}$ when $x' = 0$. If c is the radius of the source, then c also defines the distance from source center for which a circle would circumscribe the source. Thus, if c is the radius of the source, the modified Gregory model should predict the number of spores or infections in the source area when $x' = 0$. The exponent b of the modified Gregory model would then describe the number of times this concentration (the number of spores or infections/source area) becomes diluted with distance (x') from the source as the spores are carried in a turbulent, diffusing air mass away from the source.

In contrast to the Pendleton site, the modified Gregory model did not provide a very good fit to the Corvallis data when c was fixed, as evidenced by lower coefficients of determination, poorer predictions of infection at the source, and nonrandomness of residuals. Treating c as an unknown parameter, on the other hand, provided an excellent fit of the model to the Corvallis data. This would be expected, since simultaneously altering three parameters provides much flexibility in altering the shape of the gradients. However, the model parameters lose much of their biological meaning, and it is more difficult to make comparisons among epidemics when all three parameters are varied. For example, values of b among plots cannot be directly compared when equations also have different values of c (18).

The modified Gregory model provided a superior fit to the data relative to the Kiyosawa and Shiyomi model, regardless of the method used to fit the former model. This confirms other studies in which the original Gregory model (5,6) provided a better fit to spore dispersal or disease gradient data than did the Kiyosawa and Shiyomi model (1,4,8,14). Roelfs and Martell (23) suggested that the decline in numbers of uredeospores of *P. graminis* with distance from the source could be accounted for solely by diffusion. My data are consistent with this observation, because the Gregory model would describe a gradient if the decrease in spore numbers with distance is due primarily to diffusion, while the Kiyosawa and Shiyomi model would be expected to fit the gradient if the decrease in spore numbers with distance were due primarily to removal of spores by deposition on foliage (15).

Fitt et al (2) recently analyzed 325 data sets consisting of disease, spore deposition, pollen deposition, and liquid droplet deposition gradients. Overall, they concluded that neither the Gregory nor the Kiyosawa and Shiyomi model provided a superior fit to the data

TABLE 3. Model parameters and coefficients of determination for fit of the modified Gregory and the Kiyosawa and Shiyomi models to primary disease gradient data for wheat leaf rust

Field	MG1[a]				MG2[b]				KS[c]		
	a	b	c	r^2	a	b	c	r^2	A	d	r^2
Pendleton A	6.2139	2.8863	1.8649	0.9961	5.8460	2.8673	1.8288	0.9961	0.2562	0.2800	0.8699
Pendleton B	6.9869	2.9755	1.8810	0.9849	6.3805	2.9472	1.8288	0.9849	0.2493	0.2854	0.8461
Corvallis A	0.6565	1.5318	0.7580	0.9992	2.7134	1.9916	1.8288	0.9898	0.2902	0.1891	0.8173
Corvallis B	0.1946	0.2861	0.0033	0.9972	1.1521	0.9018	1.8288	0.8517	0.3762	0.0765	0.5609

[a] Model parameters and coefficient of determination (r^2) obtained when the modified Gregory model, $y = a(x' + c)^{-b}$, was fit to the observed data of Table 2 using nonlinear regression and c was assumed to be an unknown parameter.

[b] Model parameters and coefficient of determination obtained when the modified Gregory model was fit to the observed data of Table 2 using linear regression and c was assumed to be equal to the distance from the center to the edge of the source (1.8288 m).

[c] Model parameters and coefficient of determination obtained when the Kiyosawa and Shiyomi model, $y = Ae^{-dx'}$, was fit to the observed data of Table 2.

sets. However, there was a tendency for the Gregory model to provide a better fit to gradient data for pathogens with spores less than 15 μm in diameter and for the Kiyosawa and Shiyomi model to provide a better fit for splash-dispersed pathogens. These trends are logical, since small spores have a low impaction efficiency (6) and could allow diffusion to dominate the dispersal process. The large droplets involved in splash dispersal, on the other hand, would be expected to have a high impaction efficiency and to cause deposition to be important in determining the gradient. As Aylor (1) recently noted, however, diffusion can dominate the dispersal process even when a gradient is fit well by the Kiyosawa and Shiyomi model.

Because of the problem of scaling for functions that decrease rapidly with distance, graphical representations of disease gradients or propagule dispersal gradients can often be misleading. For example, examination of a plot of the relative number of pustules per tiller versus distance from the source for the data of this paper (Fig. 2) would suggest that the gradient for Corvallis Field A is very similar to that of the two Pendleton fields. However, Figure 2 does not reflect the fact that there was an eightfold difference in relative pustule numbers at 25.6 m from the source for Corvallis field A versus the Pendleton fields (Table 2).

The planting rows for the two fields at Pendleton were oriented in opposite directions relative to the predominant winds. Nevertheless, disease gradients for these two fields were extremely similar. This observation suggests that row orientation may not be an important factor influencing disease gradients of cereal rusts.

The difference in steepness of gradient between the Pendleton and Corvallis locations could have been due to the windier conditions at Pendleton. Because the diffusion of spores in air increases with wind speed (6), a volume of air containing uredospores would diffuse more per unit of distance traveled under windier conditions, resulting in a steeper gradient.

The difference of gradient steepness between the Pendleton and Corvallis sites and between the two fields at Corvallis could also have been influenced by background infection, which would be expected to decrease the slope of a disease gradient (5,6). Pockets of unusually high pustule concentration were observed in field B at Corvallis (the field with the shallowest gradient), indicating that there may have been overwintering of leaf rust in this field. Additional evidence of background infection in Corvallis field B is that the disease gradient was less directional than for the other field at Corvallis (Table 1). Evidence of infection from natural sources was not observed in Corvallis before the time that disease spread from the artificial inoculations, but natural infections could have been present at concentrations not detectable by casual observations. Evidence of infection from natural sources was not observed in field A at Corvallis, but the possibility that such infection occurred and decreased the slope of the observed gradient cannot be ruled out. Background infection could also have caused values of c estimated by nonlinear regression to be less than the radius of the source.

An additional explanation for the difference between Corvallis field B and the other fields is that the trees growing nearby this field influenced the gradients. These trees may have caused increased turbulence, resulting in the pockets of high pustule density and the less directional gradient that was observed in this field.

Data reported in this paper show that the modified Gregory model can effectively be used to describe disease gradients away from area sources of inoculum in addition to gradients away from single plants, for which the model was originally developed. Thus, the modified Gregory model can be used to study processes such as interplot interference and the effects of deploying host resistance genes in different spatial patterns.

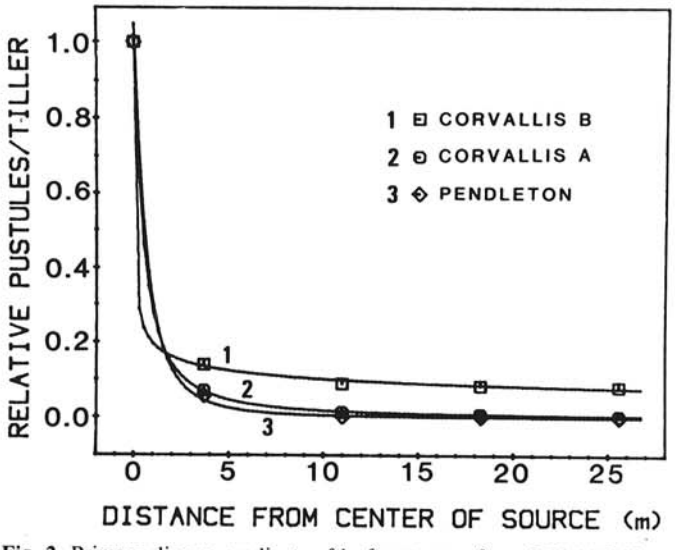

Fig. 2. Primary disease gradients of leaf rust away from 3.66- × 3.66-m sources of inoculum. Symbols represent observed values of relative pustule numbers, which were calculated by dividing pustule numbers at each distance from the source by the number of pustules at the source for each field. Curves show the fit of the modified Gregory model $y = a(x' + c)^{-b}$ to the observed data when c of that model was estimated by nonlinear regression. The model was fit to the combined data from the two fields at Pendleton because the gradients in these two fields were nearly identical.

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