Spread of Maize Chlorotic Dwarf Virus in Maize Fields by its Leafhopper Vector, *Graminella nigrifrons*

L. V. Madden, J. K. Knoke, and Raymond Louie

Associate professor, Department of Plant Pathology; research entomologist, USDA/ARS, Department of Entomology; and research plant pathologist, USDA/ARS, and adjunct professor, Department of Plant Pathology, The Ohio State University, Ohio Agricultural Research and Development Center, Wooster 44691.

Salaries and research support provided by State and Federal funds appropriated to the Ohio Agricultural Research and Development Center, The Ohio State University. Manuscript 152-89.

Accepted for publication 3 October 1989 (submitted for electronic processing).

ABSTRACT

Madden, L. V., Knoke, J. K., and Louie, R. 1990. Spread of maize chlorotic dwarf virus in maize fields by its leafhopper vector, *Graminella nigrifrons*. Phytopathology 80:291-298.

Adult leafhoppers of *Graminella nigrifrons*, given a 2-day acquisition access period to maize chlorotic dwarf virus (MCDV), were released in the center of maize plots planted in early May (1985 and 1986) or in late June to early July (1984–1986). Disease incidence (y) was assessed at least twice after insect release and represented as the proportion of plants infected by MCDV in successive 80-cm wide annuli from the source. Disease gradients were best described by the log-logistic model, i.e., logit of y versus ln(distance) was a straight line. The model indicated that the rate of spread was proportional to y, $1 - y$, and 1/distance. The spread parameter (b), a measure of the gradient steepness and slope of the linearized model, ranged from 1.3 for the early planting in 1985 to 2.0 for the late planting in 1984. In 1984 and the early plantings of 1985 and 1986, there was little change in b over time. In the late plantings, however, b increased (indicating steeper gradients) between 14 and 21 days after release. At ~21 days after release, the distance at which y declined to 0.10 (10%) ranged from 124 to 525 cm. The rates of increase in y over time for the entire plots and at selected distances from the release point were measured using the apparent infection rate (r). There was no discernible effect of distance from the source on r. The r parameter consistently declined over time. Results indicate that MCDV spread can be substantial when viruliferous leafhoppers are introduced into a field of susceptible maize.

Additional keywords: dispersal, quantitative epidemiology, *Zea mays*.

Maize chlorotic dwarf (MCD) is one of the two most economically important virus diseases of maize (*Zea mays* L.) in the United States (7,8). The virus, maize chlorotic dwarf virus (MCDV), type member of the Maize Chlorotic Dwarf Virus Group, is transmitted semipersistently, primarily by the black-faced leafhopper (*Graminella nigrifrons*) (7,25,26). This insect is common in maize fields in the eastern and southern United States. In experimental situations, several other less common leafhopper species have transmitted MCDV (25) but at lower rates than for *G. nigrifrons*.

The geographic distribution of MCDV coincides with the overlap of *G. nigrifrons* and the perennial weed johnsongrass, *Sorghum halepense* L., the overwintering host of the virus. This distribution includes primarily the eastern and southeastern states (8,16). Even in these states, however, MCD incidence generally is high only near patches of johnsongrass or in maize fields infested by this weed. To evaluate maize for resistance to MCDV, genotypes typically are planted adjacent to johnsongrass to help assure high disease incidence in susceptible lines.

One method of quantifying the spread of plant viruses is by determining disease gradients, i.e., disease incidence in relation to distance from a source (3,21,29–31). Despite its economic importance, relatively little research has been conducted on the spread of MCDV from point or area sources in maize fields. Alverson et al (1) described gradients of MCD incidence away from johnsongrass in two commercial maize hybrids in 1 yr. In the more susceptible hybrid, incidence declined from ~0.8 (80%) at the row closest to johnsongrass (0.9 m) to ~0.6 at 30 rows (27 m) from the source. However, source strength (disease incidence in johnsongrass) was not known and disease assessment in maize was done too late in the season to distinguish between primary and secondary virus spread.

The goal of this study was to quantify the spread of MCDV by leafhoppers from a point source. To separate the effect of vectors from the plant source, johnsongrass was not used; instead, viruliferous leafhoppers were released in the center of maize fields. Specific objectives were to: determine the most appropriate model for describing disease gradients; compare gradients among years, plantings within years, and assessment times within plantings; determine disease increase over time in relation to distance from the source; and describe the overall spatio-temporal dynamics of MCD epidemics based on the separate measures of disease increase in time and spread in space.

TABLE 1. Summary of experiments over 3 yr

Year-planting	Planting date	Plot size (plants)	Plots	Leafhopper release date	Number of leafhoppers (per plot)
1984	12 July	60 × 60	1	27 July	2,000
1985-I	8 May	30 × 30	4	5 June	1,000
1985-II	9 July	30 × 30	4	1 August	1,000
1986-I	7 May	20 × 20	3	11 June	810
1986-II	26 June	20 × 20	3	23 July	850

TABLE 2. Four models of Jeger (13)[a] tested for describing gradients of the incidence of maize chlorotic dwarf

Model	$dy/ds =$	$y =$	Linearized equation: Dependent variable (y^*)	Independent variable	Units of slope (b)	Intercept (a)[b]
1	$-b(1-y)$	$1 - A\exp(bs)$	$\ln(1/(1-y))$	s	distance^{-1}	$\ln(1/(1-y_0))$
2	$-by(1-y)$	$1/(1 + A\exp(bs))$	$\ln(y/(1-y))$	s	distance^{-1}	$\ln(y_0/(1-y_0))$
3	$-b(1-y)/s$	$1 - As^b$	$\ln(1/(1-y))$	$\ln(s)$	dimensionless	$\ln(1/(1-y_1))$
4	$-by(1-y)/s$	$1/(1 + As^b)$	$\ln(y/(1-y))$	$\ln(s)$	dimensionless	$\ln(y_1/(1-y_1))$

[a] b represents the spread parameter; A is the constant of integration; a is transformed A in the linarized equation; y is disease incidence (proportion) and s is distance.

[b] y_0 and y_1 represent disease incidence at $s = 0$ and $s = 1$ cm, respectively.

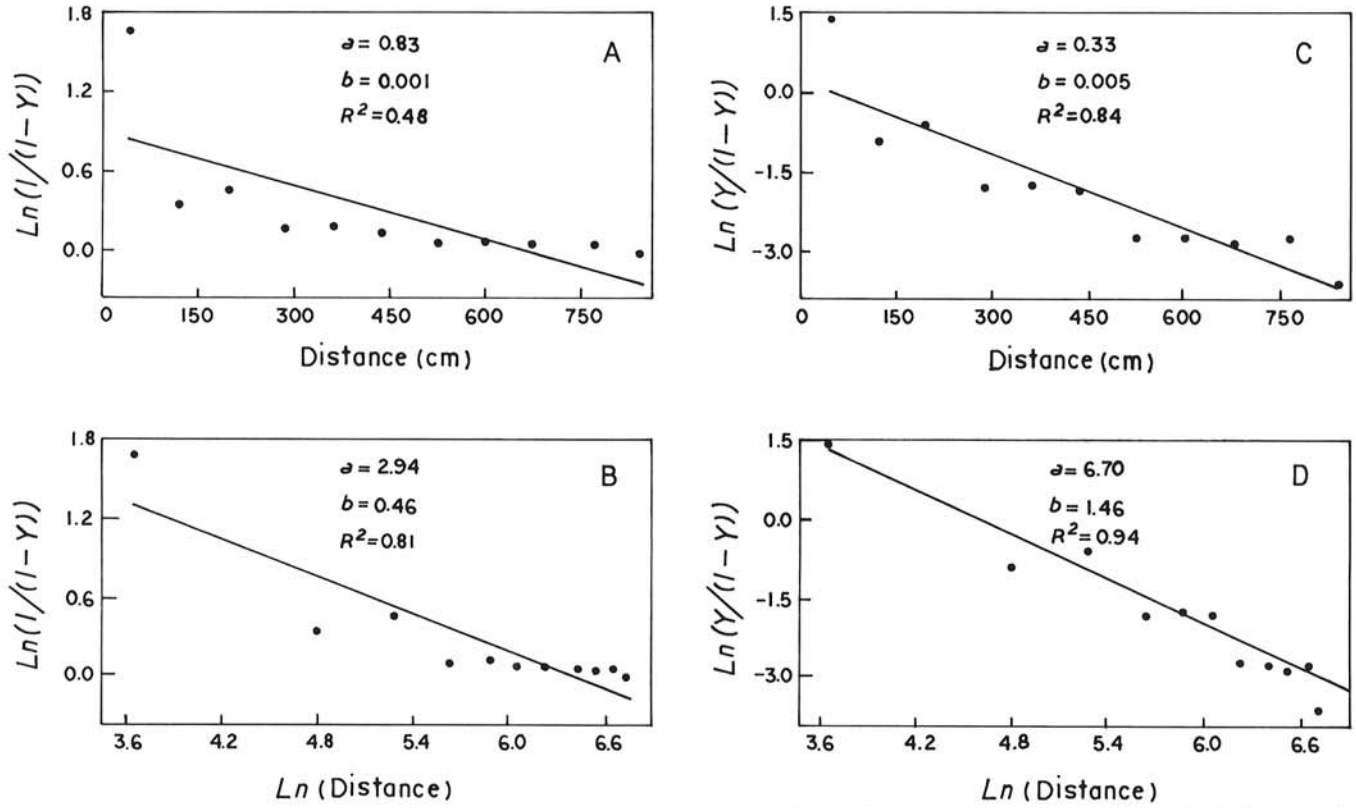

Fig. 1. Observed and predicted transformed incidence (y^*) of maize chlorotic dwarf in relation to distance (s) or $\ln(s)$ from the leafhopper release point, for one replicate plot in 1985. Combination of transformations correspond to Models 1 (**A**), 2 (**C**), 3 (**B**), or 4 (**D**) listed in Table 2. Estimated parameters (a and b) and the coefficient of determination (R^2) are given for each model.

MATERIALS AND METHODS

Field data collection. Maize plots were established at the Ohio Agricultural Research and Development Center near Wooster, OH. In 1985 and 1986, there were two nonoverlapping plantings; in 1984, there was only one planting in July (Table 1). The number of replicate plots also varied from one to four over the 3 yr. Seeds of the hybrid WF9×Oh51A were used for all plantings except the second one of 1986, when inbred Oh28 was used. All plots were hand planted in square plots with 50 cm between plants. Both genotypes are very susceptible to MCDV.

Leafhoppers were reared in the laboratory on oats (*Avena sativa* L.) and maize seedlings that were enclosed in cages (5). (*G. nigrifrons* has only one wing form and all adults can fly.) Newly emerged adults were placed on MCDV-infected seedlings for a 2-day acquisition access period. On the day of vector release, leafhoppers were allowed to fly from the virus acquisition cages into a transfer chamber, then aspirated into 14.0- × 10.2- × 3.8-cm, vented plastic boxes, at no more than 250 leafhoppers/box. Boxes were then transported to field sites and, just before release, vectors were anesthetized with CO_2 and about 12 mg of fluorescent pigment was dusted on the leafhoppers in each box as part of a separate release and recapture study not presented here. In 1984 and 1985, boxes were placed on the soil surface at the appropriate release site and lids removed to permit vectors to escape. In 1986, the dusted leafhoppers were placed for release on prepositioned, gray-colored 23- × 23-cm cards held in place by a centered, 18-cm nail inserted into the soil.

Depending on the year, between 800 and 2,000 MCDV-exposed leafhoppers were placed in the center of each plot (Table 1) when maize plants were in the four-leaf-stage. Because of cooler temperatures, it took longer for plants to reach this stage during the early plantings. In general, leafhoppers were observed to start flying or jumping within minutes after placement in the plots. No individuals were seen walking. Most leafhoppers had left the release point within 4 hr of release.

All plants were assessed visually at least twice for MCD symptoms. Maize chlorotic dwarf symptoms are distinct from other virus diseases of maize (19). The first complete census was determined by observing plants near the release point, starting 1 wk after leafhopper release. All plants were observed when sufficient infected plants near the source were infected for gradients to be determined. Usually, the first assessment time was before much secondary spread could have occurred. The straight-line distance between every plant and the release point was calculated and used in subsequent analysis.

To obtain information on the natural populations of leafhoppers in the test area, a Johnson-Taylor VI-12 insect suction trap (28), modified by adding a blacklight (15), was operated continuously throughout the growing season. In calm air, this trap sampled about 1,000 m^3 of air per hour. Leafhoppers were collected and stored in 70% alcohol, sorted to species, and counted to estimate their population each week. No observations of numbers or behavior of leafhoppers on plants were made.

Data analysis. The number of virus-diseased and disease-free plants in 80-cm wide annuli around the release point were determined for each assessment time. The proportion of MCDV-infected plants (y) then was determined and related to distance from the source (= release point). (Annuli of different widths were evaluated but did not influence the conclusions made [unpublished]). The midpoint of each annulus was used as the distance from the source.

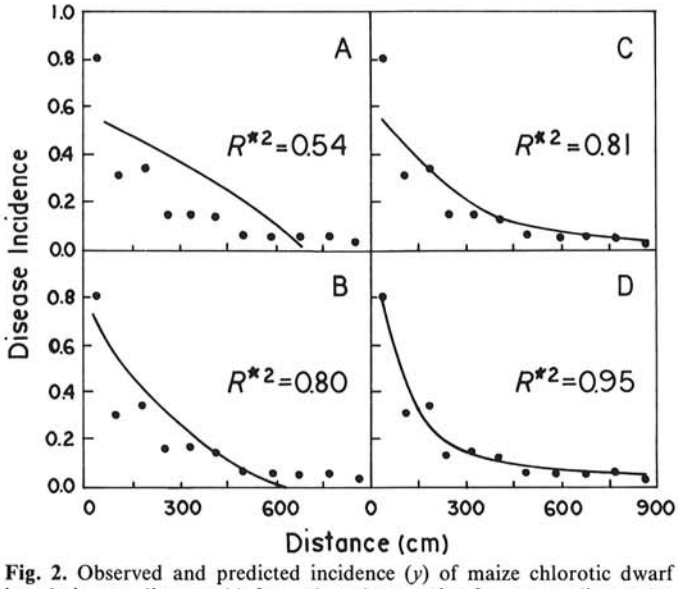

Fig. 2. Observed and predicted incidence (y) of maize chlorotic dwarf in relation to distance (s) from the release point for one replicate plot in 1985. Solid line corresponds to predicted y for Models 1 (**A**), 2 (**C**), 3 (**B**), or 4 (**D**) listed in Table 2. The coefficient of determination for the agreement between observed and predicted y (not transformed y [y^*]) (R^{*2}) is presented.

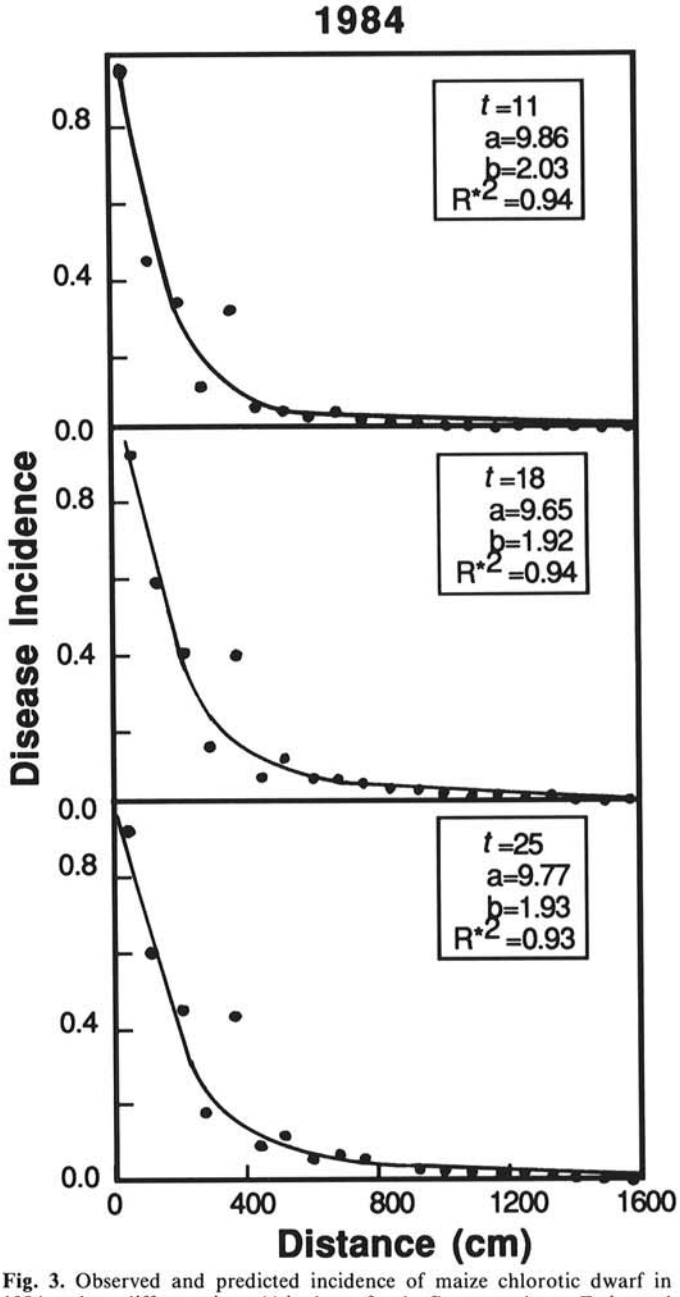

Fig. 3. Observed and predicted incidence of maize chlorotic dwarf in 1984 at three different times (t) in days after leafhopper release. Estimated parameters (a and b) for Model 4 (Table 2) and the coefficient of determination (R^{*2}) are also given.

Linear forms of the four disease spread models of Jeger (13; Table 2) were fitted to the gradient data for each replicate, assessment time, planting, and year using least squares (27). Each model incorporates a correction factor for maximum disease level $(1 - y)$. The models differ in the dependence of the absolute rate of change in y over distance (dy/ds) on disease level (y), or distance (s). In the first two models, dy/ds does not depend on s. Model 1 also indicates that dy/ds does not depend directly on y. Model 2 indicates that dy/ds depends on y as well as $1 - y$ at any given distance. The last two models, expansions of 1 and 2, respectively, incorporate an inverse spatial dependence, corresponding to a dilution of viruliferous leafhoppers with increasing distance from the release point. These four models can be referred to as the monomolecular, logistic, log-monomolecular, and log-logistic, respectively. In these models, b, the spread parameter, is a measure of the steepness of the gradient; A represents predicted transformed y at either 0 cm (Models 1 and 2), or 1 cm (Models 3 and 4) from the source; a is a transformation of A in the linear form of the model (Table 2) (3,13).

Goodness of fit of each model was evaluated with: a graphic appraisal of the residuals (observed minus predicted dependent variable); coefficient of determination (R^2) for the agreement between the observed and predicted *transformed* disease incidence (e.g., $y^* = \ln[y/(1 - y)]$ for Model 2); and the coefficient of determination for the agreement between the observed and predicted disease incidence (determined after first *back-transforming* predicted y^*-values) (R^{*2}) (20).

For the entire plots and at each distance from the point source, the apparent infection rate (r) for increase in disease over time was calculated between successive assessment times. Except for the second planting of 1986, there were not enough time periods to use regression of disease incidence on time (t) and assess the appropriateness of the logistic model. Distances of equal disease incidence, *isopaths* (e.g., distance at which $y = 0.10$ [$= s_{0.10}$]), were determined for each assessment time by rearranging the appropriate spread model (Table 2). The rate of change of selected isopaths (e.g., $s_{0.10}$) between times was then determined, using techniques appropriate for the selected model (13,22) (see below). Agreement between observed isopath rates and those determined from the appropriate gradient model then was evaluated.

For the years with replicate plots (1985 and 1986), means and standard errors were calculated for disease incidence, and for estimated parameters of the appropriate spatial (b and a) and temporal (r) models. In 1984, standard errors for b and a were taken directly from the regression analysis because there were no replicate plots.

RESULTS

Selection of gradient model. In >90% of the 46 observed gradients, the log-logistic model (Model 4) provided the best fit. Residuals generally had a random scatter and R^2 and R^{*2} were substantially higher for this model than the others. This is demonstrated in Figures 1 and 2 for one plot during the second planting of 1985. Model 4 clearly provided the best fit to the data whether in the transformed (Fig. 1) or untransformed form (Fig. 2). Model 1 gave the poorest fit and Models 2 and 3 were intermediate. In all subsequent gradient analysis, only the results from Model 4 are presented.

Disease gradients in the 3 yr. The first year of this study (1984), with the largest number of released leafhoppers, was characterized by the highest estimated a and b parameters (Fig. 3, Table 3). As y increased over time, there was little discernible change in the gradient. For the first planting of 1985, there was virtually no change in mean a or b values between the two assessment times, when average y increased from ~0.10 to 0.14 (Table 3). Data from a representative replicate plot are given in Figure 4. For the second planting, in which the first assessment was 7 days earlier than the first planting, there was an increase in a and b (i.e., a steeper gradient) as mean y increased from ~0.07 to 0.13. The gradient at 21 days was very similar for the two plantings.

The first planting of 1986 was characterized by lower disease levels (y) and a values than in 1985 (Fig. 5, Table 3). The mean a values increased over time from 3.8 to 5.2; the spread parameter (b) showed only a slight tendency to increase between 14 and 28 days after leafhopper release. Disease level was substantially higher for the second planting of 1986, as compared with the first (Table 3), although the mean spread parameter at day 14 was virtually identical for the two plantings. In contrast to the first planting, however, there was a clear increase in a and b between the first two assessment times during the second planting (Fig. 6, Table 3). After day 19, there was a general decline in both estimated parameters.

TABLE 3. Mean estimated parameters of the log-logistic model[a] fitted to gradient data of maize chlorotic dwarf incidence, disease incidence (y), and estimated distance at which $y = 0.10$ occurs ($s_{0.10}$)

Year - planting	Days[b]	a	b	y	$s_{0.10}$ (cm)[c]
1984	11	9.86 [0.88][d]	2.03 [0.14]	0.032	380.3
	18	9.65 [0.74]	1.92 [0.11]	0.045	479.1
	25	9.77 [0.76]	1.93 [0.12]	0.050	493.8
1985-I	21	7.98 (0.80)[e]	1.67 (0.12)	0.097 (0.009)	417.9 (17.0)
	28	7.76 (0.62)	1.59 (0.10)	0.142 (0.011)	559.1 (27.0)
1985-II	14	5.48 (1.04)	1.33 (0.16)	0.070 (0.012)	334.2 (37.2)
	21	7.16 (0.88)	1.50 (0.14)	0.130 (0.010)	525.0 (37.2)
1986-I	14	3.77 (0.38)	1.36 (0.02)	0.012 (0.0003)	83.3 (18.1)
	21	4.28 (0.08)	1.35 (0.003)	0.029 (0.005)	123.5 (5.4)
	28	5.20 (0.57)	1.43 (0.10)	0.050 (0.004)	178.2 (9.3)
1986-II	14	4.87 (0.69)	1.37 (0.14)	0.047 (0.003)	178.4 (11.0)
	19	8.14 (0.24)	1.85 (0.06)	0.099 (0.015)	273.3 (25.0)
	23	7.67 (0.33)	1.73 (0.09)	0.114 (0.016)	313.0 (43.4)
	28	7.35 (0.38)	1.64 (0.10)	0.134 (0.024)	357.7 (59.5)
	35	7.13 (0.18)	1.57 (0.07)	0.147 (0.029)	395.9 (74.0)
	42	6.97 (0.22)	1.51 (0.08)	0.173 (0.033)	472.8 (106)

[a] Model 4 of Table 2; a represents predicted logit of y at $s = 1$ cm [$\ln(y_1/(1 - y_1))$]; b is the spread parameter representing the change in logit of y over $\ln(s)$.
[b] Time since leafhopper release.
[c] Distance calculated for each plot and then averaged.
[d] Standard error for 1984 directly from regression analysis.
[e] Standard error for 1985 and 1986 based on values from each plot ($n = 4$ in 1985 and $n = 3$ in 1986).

The distance at which $y = 0.10$ ($s_{0.10}$) also was used for comparing MCDV spread between plantings and among years (Table 3). (This level of y was chosen because it was observed at some distance for all assessments. A higher level, e.g., $y = 0.50$, was not always obtained.) For instance, at day 21 for the two plantings of 1985, mean $s_{0.10}$ was 418 and 525 cm, respectively. The slightly larger b for the first planting (1.67 vs. 1.50) resulted in a difference in $s_{0.10}$ of over 100 cm. The lower mean y and a in the first planting of 1986 was reflected by $s_{0.10}$ only reaching 178 cm by day 28. The second planting of 1986, by contrast, had an $s_{0.10}$ of 178 by the first assessment date. By day 28 of the second planting of 1986, $s_{0.10}$ had not yet reached a level observed for $t = 21$–28 days in 1984 or 1985.

Disease progress over time. When the apparent infection rate, r, was calculated for the increase in disease incidence for entire plots, the mean estimated parameter ranged from 0.02 to 0.16 per day (Table 4). When there were more than two assessment times (1984 and 1986), a decline in r over time was observed. This was also found when either the Gompertz or monomolecular model was used instead of the logistic (Madden, *unpublished*). The highest r values corresponded to the early period of both plantings of 1986. The rate parameter also was calculated for the different annuli around the point source; results for three distances are given in Table 4. There was no systematic change in the estimated infection rates over distance from the source.

Rate of isopath movement. When disease over distance and disease over time are described by the log-logistic (Model 4) and logistic models, respectively, the rate of isopath movement ($\partial s/\partial t$) is given by $(r/b)s$ (3,13). That is, the rate is directly proportional to s; the rate is greater at greater distances from the source. Because two variables (t and s) are considered, partial derivatives are used instead of ordinary derivatives (e.g., $\partial y/\partial s$ instead of dy/ds). The composite parameter r/b was used to measure the rate of movement. Because b sometimes varied over time, mean b between successive times was used in the calculations. The composite parameter was calculated for each replicate plot and then the mean determined (Table 4).

The lowest r/b values occurred between days 18 and 25 in 1984 (Table 4) and after day 21 in 1986-II (0.01–0.02/day). The largest rates were at the beginning of both plantings of 1986 (0.09–0.10/day). The second planting of 1985 had a substantially larger rate than the first. Because r declined over time (1984 and 1986), r/b did as well.

When the presumed rate of isopath movement is equal to $(r/b)s$, $\ln(s)$ is a linear function of time (22), with a slope of $g = r/b$. Therefore, an estimate of g was determined by:

$$g = [\ln(s_{0.10})_2 - \ln(s_{0.10})_1]/[t_2 - t_1]$$

in which the 1 and 2 subscripts refer to two successive times. In general, there was a fairly close agreement between g and the theoretical r/b (Table 4). For instance, r/b and g equaled 0.03 per day at the beginning of the 1984 epidemic. The greatest discrepancy of the means was for the beginning of the first planting of 1986. A paired t test indicated that the mean difference in the two parameters ($r/b - g$) for all years and plantings was not different from 0 ($t = 0.98$, $P = 0.34$).

Leafhopper trapping. The years and plantings within years varied considerably in number of adults of *G. nigrifrons* caught in the suction trap (Table 5). None of the trapped leafhoppers were marked with dye. During the week immediately after leafhopper release, numbers trapped ranged from 0 to 1,726. Although it was anticipated that the first planting would have fewer leafhoppers than the second, this was only apparent in 1985.

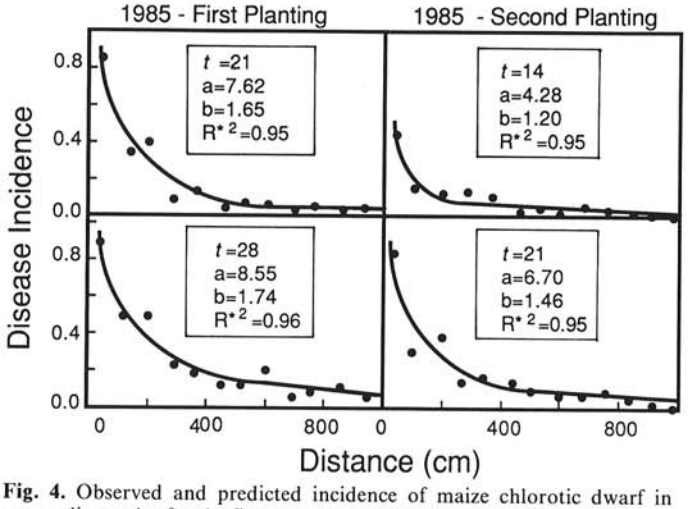

Fig. 4. Observed and predicted incidence of maize chlorotic dwarf in one replicate plot for the first and second planting of 1985 at two different times (t) in days after leafhopper release. Estimated parameters (a and b) for Model 4 (Table 2) and the coefficient of determination (R^{*2}) are also given.

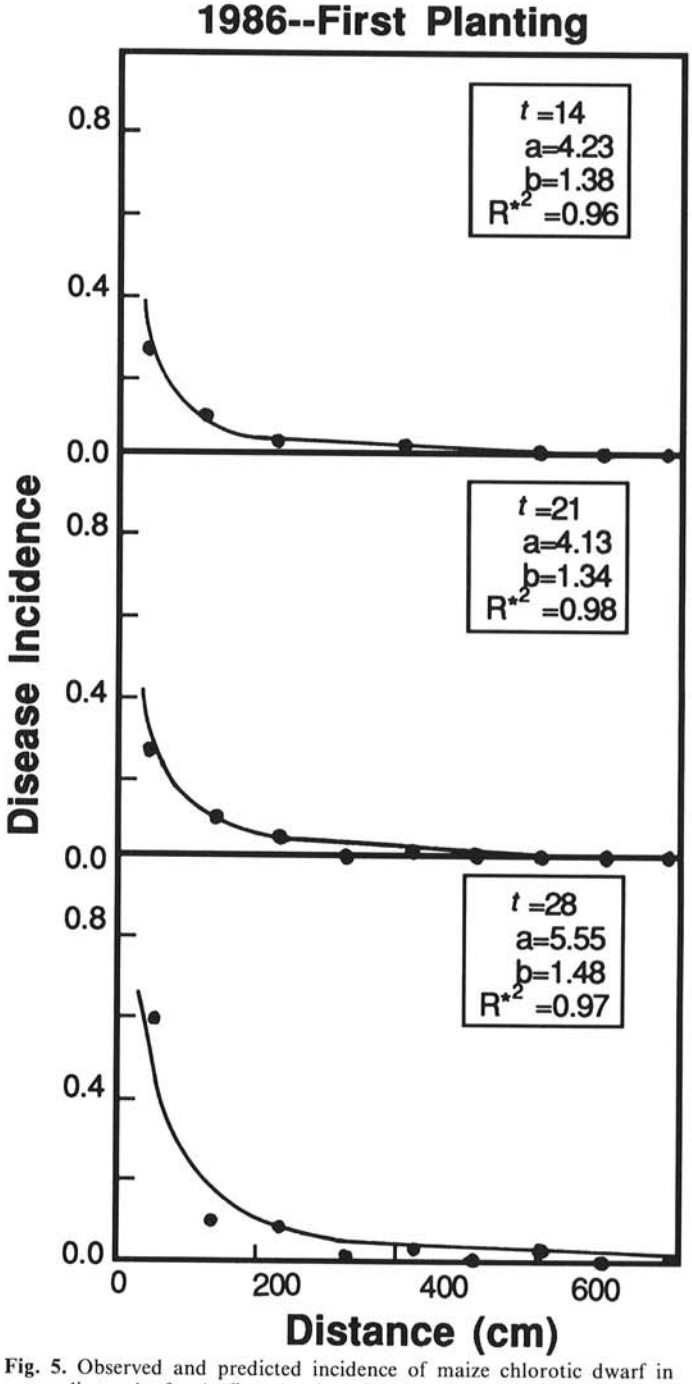

Fig. 5. Observed and predicted incidence of maize chlorotic dwarf in one replicate plot for the first planting of 1986 at three different assessment times (t) in days after leafhopper release. Estimated parameters (a and b) for Model 4 (Table 2) and the coefficient of determination (R^{*2}) are also given.

In 1985, no individuals of *G. nigrifrons* were trapped during the first planting. The trap was working correctly because other (nonvector) leafhopper species were collected (data not shown). Apparently, the population level was below the detection limit during this planting. At the other extreme, the greatest number of individuals were collected in 1984. During the 21 days after leafhopper release, 6,779 adults were collected in 1984, compared with 2,000 for the second planting of 1985.

DISCUSSION

As stated by Thresh (29), "there are great differences between [virus] diseases in the amount and distance of spread, but many gradients have the same curvilinear shape." Since 1949 (11), it has been common to describe these curvilinear virus disease gradients using either the exponential ($y = A\exp[-bs]$) or power ($y = As^{-b}$) model (3,6,9,12,17,20,21,29,31). Neither model has been found to be superior for the virus diseases studied (3,6,20). However, these models do not consdier the limitation to disease incidence, i.e., a maximum disease level of 1. Gregory and Read (11) and Gregory (10) presented an empirical correction for this by replacing y with the multiple-infection transformation $y' = \ln(1/(1-y))$. The logarithm of y' then is taken to obtain a linear model [e.g., $y'^* = \ln(\ln(1/(1-y)))$]. This transformation has not always been used when analyzing gradients of disease caused by viruses or other pathogens. Jeger (13) expanded our understanding of disease gradients compared with the older empirical work and improved the means of describing these gradients by incorporating the proportion of healthy plants $(1-y)$ directly into the equations for the rate of change in y over s (i.e., dy/ds). He described four models that can account for dependence (or independence) of dy/ds on y or s, and discussed an epidemic scenario for each model.

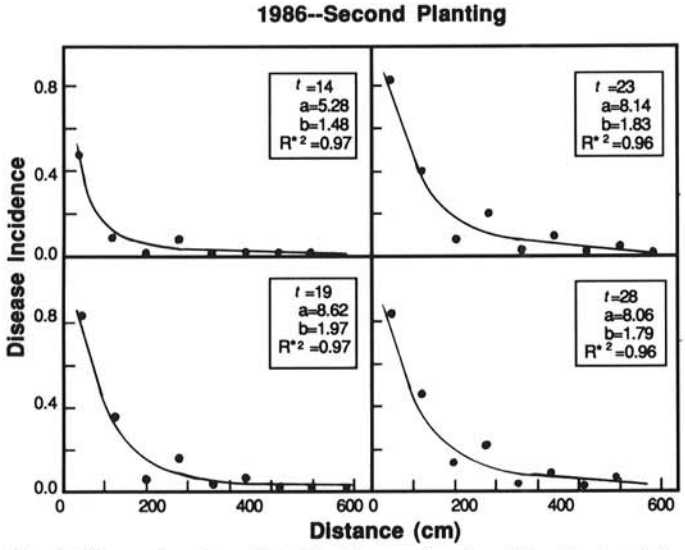

Fig. 6. Observed and predicted incidence of maize chlorotic dwarf in one replicate plot for the second planting of 1986 at the first four assessment times (t) in days after leafhopper release. Estimated parameters (a and b) for Model 4 (Table 2) and the coefficient of determination (R^{*2}) are also given.

As indicated by the gradients of disease incidence in our study, there was consistent spread of MCDV in susceptible maize genotypes by released viruliferous leafhoppers. The gradients were well-described by the log-logistic model of Jeger (13) (Model 4) over each of 3 yr and two planting dates within years (Figs. 1–6). According to Jeger (13), this model is appropriate for describing polycyclic (compound interest) epidemics when there is a relatively large number of propagules (or viruliferous insects or diseased plants) at the source compared with the rest of the plot. Our results are consistent with this premise. Relatively large numbers of viruliferous leafhoppers were introduced into the center of field plots of healthy plants. After flights, landings, and feeding by the leafhoppers, a small proportion of all the plants, and a higher proportion near the release point, became infected. These diseased plants became the source for subsequent MCDV spread. The released leafhoppers, those in the natural population, or both, were responsible for secondary spread from diseased plants. Leafhoppers in the natural population, however, probably were not viruliferous before entering the experimental plots because no MCDV-infected plants were found in nearby plots of susceptible maize genotypes in any year of this study unless viruliferous insects were introduced (J. K. Knoke and R. Louie, *unpublished*). Considerably more work still is needed to determine the effect of vector movement, population dynamics, and transmission efficiency on the spread of MCDV and other viruses.

Berger and Luke (2) and Jeger et al (14) found the log-logistic model (Model 4, Table 2) to be appropriate for representing disease gradients of oat crown rust and Septoria nodorum blotch on wheat, respectively. For the latter disease, this model was appropriate only for disease incidence. Severity was better described by the logistic equation (Model 2; Table 2). With the logistic model, dy/ds is not directly related to distance from the point source. As hypothesized by Jeger et al (14), "outward spread of incidence . . . is dependent on dispersal from plot centers,

TABLE 4. Mean apparent infection rates (day^{-1}) for entire plots and at three selected distances from plot center and rate of isopath movement between successive times

		Apparent infection rate				Rate of isopath movement[a]	
			Distance from plot center (cm)				
Year - planting	Days	Total plot	160	320	480	r/b	$\Delta\ln(s_{0.10})/\Delta t$
1984	11–18	0.05	0.08	0.04	0.05	0.03	0.03
	18–25	0.02	0.00	0.02	0.02	0.01	0.01
1985-I	21–28	0.06 (0.006)[b]	0.10 (0.010)	0.06 (0.013)	0.09 (0.025)	0.04 (0.006)	0.04 (0.003)
1985-II	14–21	0.10 (0.015)	0.12 (0.023)	0.11 (0.033)	0.12 (0.060)	0.07 (0.012)	0.07 (0.015)
1986-I	14–21	0.13 (0.087)	0.15 (0.150)	0.08 (0.080)	0.12 (0.117)	0.09 (0.053)	0.06 (0.040)
	21–28	0.08 (0.015)	0.07 (0.035)	0.04 (0.042)	0.04 (0.040)	0.06 (0.010)	0.05 (0.012)
1986-II	14–19	0.16 (0.020)	0.20 (0.042)	0.10 (0.051)	0.14 (0.072)	0.10 (0.019)	0.07 (0.022)
	19–23	0.04 (0.007)	0.02 (0.017)	0.08 (0.048)	0.13 (0.082)	0.02 (0.003)	0.04 (0.009)
	23–28	0.03 (0.008)	0.03 (0.014)	0.06 (0.016)	0.06 (0.031)	0.02 (0.006)	0.02 (0.010)
	28–35	0.02 (0.008)	0.02 (0.016)	0.02 (0.012)	0.03 (0.020)	0.01 (0.004)	0.01 (0.006)
	35–42	0.03 (0.003)	0.03 (0.001)	0.02 (0.010)	0.03 (0.015)	0.02 (0.002)	0.02 (0.005)

[a] Two methods were used. r/b: The apparent infection rate (r) was simply divided by the spread parameter (b). $\Delta\ln(s_{0.10})/\Delta t$: The change in natural logarithm of distance at which $y = 0.1$ occurs was divided by the change in time ($= g$). These statistics were calculated for each plot and then averaged.

[b] Standard error ($n = 4$ in 1985 and $n = 3$ in 1986).

but intensification of disease within this area is dependent on more limited and local dispersal, perhaps on the same leaf." The latter part of this hypothesis could not be tested with MCD because the meaning of severity for virus diseases is very different from that of fungal diseases of aerial plant parts.

Disease gradients were fairly stable over the 3 yr as measured by the log-logistic model. The first year of the study (1984) was the most unusual of the three by having the largest a and b values. However, twice as many leafhoppers were released in this year and the plots were twice as large. Considerably more leafhoppers were trapped during this year also. There was, however, very little change in the two parameters over the three assessment times as y (for the entire plot) increased from 0.03 to 0.05. With regard to the stability of parameters, 1984 results were similar to the first planting of 1985 and 1986. In 1985, there was only a slight or no change in the spread parameter b and scale parameter a. In 1986, a increased over time, as y increased from ~0.01 to 0.05, but gradient steepness was fairly stable during this planting.

Epidemics of the second planting of 1986, and also 1985 to a lesser extent, differed from the first-planting epidemics in more than one way. The b parameter increased substantially between days 14 and 19 of 1986, and then exhibited a fairly slow decline over time, indicating that the steepness of the gradient first increased and then slowly decreased. The a parameter also increased substantially between the first and second times, when y increased from ~0.05 to 0.10, and then exhibited a very slow decrease over time in 1986-II.

A slight reduction in parameter a should not be interpreted simply as a decline in predicted y. This parameter represents predicted logit of y at 1 cm (i.e., when $\ln(s) = 0$) (Table 2). However, the first distance is $s = 40$ cm (encompassing plants up to 80 cm from the release point). When b decreases, a may decrease somewhat, but predicted y may be the same at any observed distance. For instance, in the second planting of 1986, mean a equaled 8.14 and 7.67 for days 19 and 23, respectively. With the corresponding b values (1.85 and 1.73), predicted logit of y (y^*) at 40 cm equaled ~1.3 at both times. This corresponded to predicted y of 0.79 ($= 1/(1 + \exp[-1.3])$) at 40 cm.

The observed spread of MCD was more restricted than for several other virus diseases (17,29,31). With many viruses, including those transmitted semipersistently by aphids, it is not uncommon for $s_{0.10}$ to exceed 10 m from the source and for y (for the entire plot) to exceed 0.25. Results for MCDV can be compared to a published disease gradient for the only other disease caused by viruses that are semipersistently transmitted by leafhoppers, rice tungro (18). The virus source consisted of adults of *Nephotettix virescens* released in the center of the plot. Disease incidence of rice tungro was considerably higher than for MCD, but source strength also was considerably higher. Viruliferous leafhoppers were released twice a week for 4 wk instead of once at the beginning of the epidemics as in our experiments. We determined that the log-logistic model was appropriate for the rice tungro data (18). The estimated a and b parameters were 7.4 and 1.3, respectively (unpublished), very similar to the MCD results presented here, indicating that the change in disease incidence over distance from the source was quite similar for the two semipersistent virus diseases. Differences in disease incidence but similarities in gradient characteristics demonstrate the importance of considering more than one variable when attempting to compare disease spread. More details on assessing and interpreting virus disease gradients are given by Thresh (29–31).

TABLE 5. Number of adults of *Graminella nigrifrons* collected per week in a suction trap near the maize field plots

Year - planting	Days after release				
	0[a]	7	14	21	28
1984	429	1,726	1,806	3,247	519
1985-I	0	0	0	0	0
1985-II	221	76	1,640	284	314
1986-I	864	52	156	0	357
1986-II	889	164	156	538	188

[a] Day 0 represents number of leafhoppers in the trap during the 7 days before release.

The rate of disease progress over time for MCD, as measured by r, was generally invariant to distance from the release point. This made it possible to calculate rates of isopath movement (or velocities of spread) (3,13,22) between successive assessment times. These velocities have been calculated by other researchers to characterize spread of several diseases (review in 22). When disease gradients depend on distance, i.e., dy/ds is inversely related to s, velocity of isopath movement depends on s. One cannot summarize this velocity independent of distance. Both the composite parameter r/b and the slope of $\ln(s)$ versus t give the velocity of spread relative to s. One can calculate the velocity at any distance simply by multiplying r/b by a chosen distance. For instance, at $s = 375$ cm (half the distance from the center to the plot edge for 1985), the velocity ranged from 3 to 37 cm/day with a median value of ~15 cm/day. This was similar to velocities found for several diseases including those with wind-dispersed and rain-splash dispersed spores (2,13,22,32,33). However, the velocities were slower than those of potato late blight (24) and considerably faster than for tomato ringspot nepovirus on raspberry (4). The velocities were also slower than that observed for the movement of rubidium-labeled adults of *G. nigrifrons* during one season in Georgia (1). If the rate reported by Alverson et al (1) is typical, simple movement and very short feedings by leafhoppers may not be good predictors of virus spread. Disease incidence, unfortunately, was only reported for a single assessment time in the study by Alverson et al (1). Alternatively, leafhopper (and virus) spread may be much faster in the southern United States than in Ohio.

Contrary to the predictions from some theoretical models (32,33), MCDV did not proceed as a "wave," i.e., the rate of isopath movement depended on distance from the source and also declined over time instead of staying constant. With the log-logistic spread model and the logistic temporal model, one does not expect an isopath rate independent of distance (13,23), but, rather, a rate that increases with distance. Variation in b or r, however, can produce a temporally changing isopath rate (3). Even though b changed somewhat in these epidemics, the decline in r over time was primarily responsible for this reduction in isopath rate of movement.

LITERATURE CITED

1. Alverson, D. R., All, J. N., and Kuhn, C. W. 1980. Simulated intrafield dispersal of maize chlorotic dwarf virus by *Graminella nigrifrons* with a rubidium marker. Phytopathology 70:734-737.
2. Berger, R. D., and Luke, H. H. 1979. Spatial and temporal spread of oat crown rust. Phytopathology 69:1199-1201.
3. Campbell, C. L., and Madden, L. V. 1990. Introduction to Plant Disease Epidemiology. Wiley-Interscience, New York. 532 pp.
4. Converse, R. H., and Stace-Smith, R. 1971. Rate of spread and effect of tomato ringspot virus on red raspberry in the field. Phytopathology 61:1104-1106.
5. D'Arcy, C. J., and Nault, L. R. 1982. Insect transmission of plant viruses, mycoplasmalike and rickettsialike organisms. Plant Dis. 66:99-104.
6. Fitt, B. D. L., Gregory, P. H., Todd, A. D., McCartney, H. A., and Macdonald, O. C. 1987. Spore dispersal and plant disease gradients: A comparison between two empirical models. J. Phytopathol. 118:227-242.
7. Gingery, R. E. 1988. Maize chlorotic dwarf and related virus. Pages 259-272 in: The Plant Viruses, Vol. 3. R. Koenig, ed. Plenum Publishing Corp., New York.
8. Gordon, D. T., Bradfute, O. E., Gingery, R. E., Knoke, J. K., Louie, R., Nault, L. R., and Scott, G. E. 1981. Introduction: History, geographical distribution, pathogen characteristics, and economic importance. Pages 1-12 in: Virus and Viruslike Diseases of Maize in the United States. D. T. Gordon, J. K. Knoke, and G. E. Scott, eds. South. Coop. Series Bull. 247. 218 pp.
9. Gray, S. M., and Lampert, E. P. 1988. Relationship between inoculum density and vector phenology on the incidence of potato virus Y

in tobacco. Ann. Appl. Biol. 112:303-312.
10. Gregory, P. H. 1973. The Microbiology of the Atmosphere. 2nd ed., Leonard Hill, London.
11. Gregory, P. H., and Read, D. R. 1949. The spatial distribution of insect-borne plant-virus diseases. Ann. Appl. Biol. 36:475-482.
12. Irwin, M. E., and Goodman, R. M. 1981. Ecology and control of soybean mosaic virus. Pages 181-220 in: Plant Diseases and Their Vectors: Ecology and Epidemiology. K. Maramorosch and K. F. Harris, eds. Academic Press, New York. 360 pp.
13. Jeger, M. J. 1983. Analysing epidemics in time and space. Plant Pathol. 32:5-11.
14. Jeger, M. J., Jones, D. G., and Griffiths, E. 1983. Disease spread of non-specialised fungal pathogens from inoculated point sources in intraspecific mixed stands of cereal cultivars. Ann. Appl. Biol. 102:237-244.
15. Knoke, J. K., Anderson, R. J., and Louie, R. 1977. Virus disease epiphytology: Disease field tests for disease resistance in maize. Pages 116-121 in: L. E. Williams, D. T. Gordon, and L. R. Nault, eds. Proc. Int. Maize Virus Dis. Colloq. Workshop, 16-19 Aug., 1976. Ohio Agric. Res. Dev. Cent., Wooster. 145 pp.
16. Knoke, J. K., and Louie, R. 1981. Epiphytology of maize virus diseases. Pages 92-102 in: Virus and Viruslike Diseases of Maize in the United States. D. T. Gordon, J. K. Knoke, and G. E. Scott, eds. South. Coop. Ser. Bull. 247. 218 pp.
17. Knoke, J. K., Louie, R., Madden, L. V., and Gordon, D. T. 1983. Spread of maize dwarf mosaic virus from johnsongrass to corn. Plant Dis. 67:367-370.
18. Kondaiah, A., Rao, A. V., and Srinivasan, T. E. 1976. Factors favoring spread of rice "tungro" disease under field conditions. Plant Dis. Rep. 60:803-806.
19. Louie, R., and Knoke, J. K. 1981. Symptoms and disease diagnosis. Pages 13-18 in: Virus and Viruslike Diseases of Maize in the United States. D. T. Gordon, J. K. Knoke, and G. E. Scott, eds. South. Coop. Ser. Bull. 247. 218 pp.
20. Madden, L. V., and Campbell, C. L. 1986. Descriptions of virus disease epidemics in time and space. Pages 273-293 in: Plant Virus Epidemics. G. D. McLean, R. G. Garrett, and W. G. Ruesink, eds. Academic Press, Sydney, Australia. 550 pp.
21. Madden, L. V., Knoke, J. K., and Louie, R. 1986. Effect of source strength, distance and direction on the spread of maize dwarf mosaic virus. J. Phytopathol. 117:92-96.
22. Minogue, K. P. 1986. Disease gradients and the spread of disease. Pages 285-310 in: Plant Disease Epidemiology; Population Dynamics and Management. K. J. Leonard and W. E. Fry, eds. Macmillan Publishing Co., New York. 327 pp.
23. Minogue, K. P. 1989. Diffusion and spatial probability models for disease spread. Pages 127-143 in: Spatial Components of Plant Disease Epidemics. M. J. Jeger, ed. Prentice Hall, Englewood, NJ.
24. Minogue, K. P., and Fry, W. E. 1983. Models for the spread of disease: Some experimental results. Phytopathology 73:1173-1176.
25. Nault, L. R., and Madden, L. V. 1988. Phylogenetic relatedness of maize chlorotic dwarf virus leafhopper vectors. Phytopathology 78:1683-1687.
26. Nault, L. R., Styer, W. E., Knoke, J. K., and Pitre, H. N. 1973. Semipersistent transmission of leafhopper-borne maize chlorotic dwarf virus. J. Econ. Entomol. 66:1271-1273.
27. Neter, J., Wasserman, W., and Kutner, M. H. 1983. Applied Linear Regression Models. Richard D. Irwin, Inc., Homewood, IL.
28. Taylor, L. R. 1951. An improved suction trap for insects. Ann. Appl. Biol. 38:582-591.
29. Thresh, J. M. 1976. Gradients of plant virus diseases. Ann. Appl. Biol. 82:381-406.
30. Thresh, J. M. 1980. An ecological approach to the epidemiology of plant virus diseases. Pages 57-70 in: Comparative Epidemiology. J. Palti and J. Kranz, eds. PUDOC, Wageningen, The Netherlands. 122 pp.
31. Thresh, J. M. 1986. Plant virus dispersal. Pages 51-106 in: The Movement and Dispersal of Agriculturally Important Biotic Agents. D. R. MacKenzie, C. S. Barfield, G. G. Kennedy, and R. D. Berger, eds. Calitor's Publishing Division, Baton Rouge, LA.
32. van den Bosch, F., Frinking, H. D., Metz, J. A., and Zadoks, J. C. 1988. Focus expansion in plant disease. III: Two experimental examples. Phytopathology 78:919-925.
33. van den Bosch, F., Zadoks, J. C., and Metz, J. A. 1988. Focus expansion in plant disease. I: The constant rate of focus expansion. Phytopathology 78:54-58.