Coupling Disease-Progress-Curve and Time-of-Infection Functions for Predicting Yield Loss of Crops

L. V. Madden, G. Hughes, and M. E. Irwin

First author: Department of Plant Pathology, Ohio State University, Wooster 44691-4096; second author: Institute of Ecology and Resource Management, University of Edinburgh, West Mains Road, EH9 3JG, Scotland; and third author: Department of Natural Resources and Environmental Sciences, University of Illinois, Urbana 61801.
Accepted for publication 27 April 2000.

ABSTRACT

Madden, L. V., Hughes, G., and Irwin, M. E. 2000. Coupling disease-progress-curve and time-of-infection functions for predicting yield loss of crops. *Phytopathology* 90:788-800.

A general approach was developed to predict the yield loss of crops in relation to infection by systemic diseases. The approach was based on two premises: (i) disease incidence in a population of plants over time can be described by a nonlinear disease progress model, such as the logistic or monomolecular; and (ii) yield of a plant is a function of time of infection (t) that can be represented by the (negative) exponential or similar model ($\zeta(t)$). Yield loss of a population of plants on a proportional scale (L) can be written as the product of the proportion of the plant population newly infected during a very short time interval ($X'(t)dt$) and $\zeta(t)$, integrated over the time duration of the epidemic. L in the model can be expressed in relation to directly interpretable parameters: maximum per-plant yield loss (α , typically occurring at $t = 0$); the decline in per-plant loss as time of infection is delayed (γ ; units of time^{-1});

and the parameters that characterize disease progress over time, namely, initial disease incidence (X_0), rate of disease increase (r ; units of time^{-1}), and maximum (or asymptotic) value of disease incidence (K). Based on the model formulation, L ranges from αX_0 to αK and increases with increasing X_0 , r , K , α , and γ^{-1} . The exact effects of these parameters on L were determined with numerical solutions of the model. The model was expanded to predict L when there was spatial heterogeneity in disease incidence among sites within a field and when maximum per-plant yield loss occurred at a time other than the beginning of the epidemic ($t > 0$). However, the latter two situations had a major impact on L only at high values of r . The modeling approach was demonstrated by analyzing data on soybean yield loss in relation to infection by *Soybean mosaic virus*, a member of the genus *Potyvirus*. Based on model solutions, strategies to reduce or minimize yield losses from a given disease can be evaluated.

Additional keywords: crop loss assessment, disease management strategies, quantitative epidemiology.

There has long been an interest in determining the impact of plant diseases on crop yield and yield loss (28,32), and there are many published models for relating disease incidence or severity (5) to crop yield (37). These models are based on empirical descriptions of the disease-yield relationship from population (field) studies or investigations of crop physiology (8,31,37). Recent research has focused on the extent to which plant diseases affect crop yield through changes in radiation use efficiency and radiation interception (RI) integrated over time (2,31).

A key result of many studies on crop yield in relation to disease is that time of plant infection (i.e., plant age when the plant becomes infected or diseased) has a major effect on the resulting yield (28,32), with decreasing impact of disease as time of infection is delayed. This is the basis for use of area under disease progress curves (AUDPC; 5,19) and other weighted summations of disease intensity over time (37) for predicting yield. The integration of RI over time (2,31) also exemplifies how time of infection is used (indirectly in this case) to predict yield response. The relationship between time of infection and yield may be due to increasing resistance as plants age—leading to a lower rate of development of disease in plants infected late rather than early in the growing season—or to decreasing physiological effects of disease on crop growth and yield (8,14). It is often not possible to distinguish between these two processes based on many published studies (7,51,53,54,56).

With systemic diseases, time of infection is often considered the primary determinant of crop yield, especially for annual crops (39). For example, Watson et al. (57) showed experimentally, over 50 years ago, that the age of sugar beets when they became infected by yellowing viruses, specifically *Beet yellows virus* (BYV), a member of the genus *Closterovirus*, and *Beet mild yellowing virus* (BMV), a member of the genus *Luteovirus*, determined the magnitude of several yield components, with an increased yield as time of infection was delayed. Similar results have been found for other plant viruses (1,4,9,17,18,40,46,47,50), with various levels of experimental control over the time of infection. Researchers have also simulated the effects of plant diseases on yield losses with “artificial gapping” experiments, in which plant population density is manually reduced and the timing of the density reduction is varied (20,27). In general, yield loss can approach 100% when infection occurs at plant emergence and can be 0% when infection occurs near the end of the growing season. Major obstacles of putting results such as these into practice for populations of plants are that all plants do not become infected at a single time and the exact time of infection (or even time of first symptom appearance) for individual plants is seldom known. Thus, there is a difficulty in predicting (mean) yields for commercial fields or large experimental plots based on known information on plant responses to time of infection.

Disease progress curves, which consist of proportions of diseased plants (i.e., disease incidence) at selected times, have been determined for many plant virus diseases in experimental plots and commercial fields (38,43,52). The difference in disease incidence between pairs of assessment times gives the proportion of plants diseased during that interval, which could in principle be used in conjunction with the time-of-infection results to predict

Corresponding author: L. V. Madden; E-mail address: madden.l@osu.edu

Publication no. P-2000-0620-01R
© 2000 The American Phytopathological Society

yield for a field. Actual time of infection, rather than time of symptom appearance, can be estimated using this approach by first subtracting the mean length of the incubation period from the time of disease observations before calculating differences in incidence. The time interval between disease observations, however, is generally too long to obtain a precise estimate of the time of new plant infections based only on observed disease incidence at pairs of times. Detailed analysis of disease progress curves shows that a few epidemic models, such as the logistic, monomolecular, and Gompertz, do provide very precise descriptions of the observed data over time for a wide range of crops and cropping practices in temperate and tropical climates (34).

Madden (33) and Madden and Nutter (37) developed a theoretical approach without directly considering any observed data to predict yield or yield loss based on a logistic model for disease progress over time and a (negative) exponential model for individual plant yield in relation to time of infection. Here, we (i) give a more rigorous foundation for the model, (ii) show how model parameters can be used to evaluate disease management strategies, (iii) expand the full model to account for other disease progress and time-of-infection models, (iv) show the effects of spatial heterogeneity of disease on expected yield loss, and (v) demonstrate the applicability of the model with observations of soybean yield in relation to infection by *Soybean mosaic virus* (SMV), a member of the genus *Potyvirus* (26,48).

THEORY AND APPROACHES

Time of infection. Time during a growing season is represented by t , and yield loss per plant for plants infected at t is represented by $\zeta(t)$. We refer to $\zeta(t)$ as the "loss function." If $Y(t)$ is the yield of a plant infected at t (or mean yield of a population of plants all infected at t) and $Y(\infty)$ is yield of a plant never infected, then $\zeta(t)$ is given by $[Y(\infty) - Y(t)]/Y(\infty)$. For convenience, we generally present yield loss on a proportional scale, although terms can be rescaled easily for absolute units (e.g., kilogram per hectare). Moreover, we present plant response as loss rather than yield, although the model could be reexpressed in terms of yields (e.g., $1 - \zeta(t)$).

For the nominal situation, we assume that $\zeta(t)$ is a negative exponential function

$$\zeta(t) = \alpha e^{-\gamma t} \quad (1)$$

in which α and γ are parameters; α represents maximum per plant yield loss (i.e., yield loss of plants infected at $t = 0$) and can take values from 0 to 1; and γ represents the decline in loss (units of time⁻¹) as time of infection is delayed and can take values from 0 to ∞ . Examples are shown in Figure 1 for $\alpha = 0.9$ (90% loss if infected at time 0) and $\gamma = 0.02/\text{day}$ (curve a) and $\gamma = 0.08/\text{day}$ (curve b). When $\gamma = 0/\text{day}$ in equation 1, there is no decline in loss; i.e., the same yield loss ($=\alpha$) occurs for all times of infection for this extreme condition. At large γ ($\geq 0.15/\text{day}$), there is a very large decline in $\zeta(t)$ with an increase in time, such that there is virtually no yield loss in infected plants if time of infection is only slightly delayed past $t = 0$ (37 [Fig. 5]).

Equation 1 is chosen here because of its simplicity and ability to describe many published data sets on observed loss as a function of time of infection (40; L. V. Madden, unpublished data). Parameters are also easy to interpret, with α representing the maximum possible loss as well as a measure of the overall height of the loss function, and $\ln(2)/\gamma$ representing the time of infection at which yield loss is reduced in half (i.e., reduced to $\alpha/2$). Together, the two parameters can be considered a measure of the tolerance (5) of a plant cultivar to infection by a given disease. Many other functions for a monotonic decrease in ζ , such as a (negative) logistic, could be used if there was evidence for such a relationship. A straight line function could also be used, although this should be done with caution to avoid extrapolation errors at very early and late times of infection.

Equation 1 represents situations in which per-plant loss monotonically decreases with an increase in time of infection (Fig. 1). However, in some systems (46) there is evidence that the maximum loss occurs at $t > 0$; i.e., yield loss of plants initially increases with an increase in plant age at infection and declines at later times. This is modeled by replacing t in equation 1 with a function of t , $g(t)$, that reaches its minimum value at $t > 0$. We consider two forms of $g(t)$ here, $|t - \delta|$ and $(t - \delta)^2$, in which $|\cdot|$ represents the absolute-value function, and δ is a parameter (units of time) that corresponds to the time when maximum loss occurs. In both cases, $g(t)$ equals 0 when $t = \delta$ days. The two new versions of equation 1 can be written as

$$\zeta(t) = \alpha e^{-\gamma_1 |t - \delta|} \quad (2a)$$

and

$$\zeta(t) = \alpha e^{-\gamma_2 (t - \delta)^2} \quad (2b)$$

Examples are shown for equation 2a and 2b in Figure 1 for two values of δ (15 and 30 days). There are subscripts on the γ parameter because the scale is different for each formulation of the model. In equations 2a and 2b, maximum loss is still α , but this loss occurs at $t = \delta$, not $t = 0$ (Fig. 1). At $t = 0$, loss equals $\alpha \exp(-\gamma_1 \delta)$ which we call α_1 for equation 2a, and $\alpha \exp(-\gamma_2 \delta^2)$ which we call α_2 for equation 2b.

To compare results obtained with equation 1 for $\zeta(t)$ ($\delta = 0$) and results obtained with equation 2a or 2b for $\zeta(t)$, different values of the γ (i.e., γ_1 , γ_2) parameter are needed. Although one could choose γ values so equations 1, 2a, and 2b all give the same predicted $\zeta(t)$ at an arbitrary time (e.g., at $t = 30$ days), the more general approach, and the one taken here, is to select γ values so the area under the $\zeta(t)$ curve is the same for the three equations (Fig. 1). More formally, this means that the integration $\int_0^T \zeta(t) dt$, where T (days) is the end of the growing season (or the end of the epidemic), is equal for equations 1, 2a, and 2b. Values of γ_1 and γ_2 for $\delta = 15$ and $\delta = 30$ days are given in the legend of Figure 1 corresponding to the same area as for equation 1 with $\gamma = 0.02/\text{day}$ (i.e., curve a).

Disease progress over time. The proportion of all plants in a population that are diseased at t , known as disease incidence (5), is represented as $X(t)$. Here $X(t)$ represents both visible disease and disease that has not yet passed through an incubation (or latent) period at t . Thus, we follow the general convention of equat-

Fig. 1. Relationship between time of infection (t) and yield loss per plant (loss function, $\zeta(t)$) with a maximum per-plant loss (α) of 0.9. $\zeta(t)$ is based on: equation 1 for $\gamma = 0.02/\text{day}$ (curve a) and $\gamma = 0.08/\text{day}$ (curve b) or equations 2a (thick lines) and 2b (thin lines) for times of maximum loss (δ) of 15 (short dashed line) and 30 days (long and short dashed line). Parameter γ_1 of equation 2a equals 0.03/day for $\delta = 15$ days and 0.037/day for $\delta = 30$ days. Likewise, γ_2 of equation 2b equals 0.00095/day for $\delta = 15$ days and 0.0016/day for $\delta = 30$ days. The a and b curves also correspond to equation 2a, with $\delta = 0$ and $\gamma_1 = \gamma$. The area under the $\zeta(t)$ curves is the same for all cases except curve b.

ing infection and disease (34,36), recognizing that a diseased individual can be in the latent, infectious, or postinfectious state, or in the symptomless or visible-symptom state. For observations of disease based on symptoms, one can determine $X(t)$, at least as a first approximation in many pathosystems, by subtracting the mean length of the incubation period from the assessment time to obtain t . For example, if there is a 7-day incubation period, observed disease incidence at day 50 would correspond to total disease incidence at day 43 (i.e., $t = 43$ at day 50). If there are N plants in the population and time is measured in discrete days, $N \cdot [X(t) - X(t - 1)]$ represents the number of plants that become infected on day t . However, we consider time to be continuous here for modeling purposes.

Any of the standard disease progress models (34) can be used to represent $X(t)$. We used the logistic model for the nominal situation because it describes numerous observed disease progress curves for polycyclic "compound-interest" diseases (5,34,38,43). The logistic model can be written in terms of three parameters: r , the rate parameter (units of time^{-1}); K , the maximum value of X ($0 < K \leq 1$); and X_0 , initial disease incidence ($X_0 = X(0)$; $0 < X_0 < K$) (L. V. Madden and C. L. Campbell [34] equation 20). The rate of change of $X(t)$ at t is determined by the derivative of $X(t)$ with respect to time $X'(t)$:

$$X'(t) = \frac{rKAe^{-rt}}{(1 + Ae^{-rt})^2} \quad (3)$$

in which $A = (K - X_0)/X_0$. $X'(t)$ can also be written in terms of $X(t)$ as $rX(t)[1 - X(t)/K]$.

For monocyclic or "simple-interest" diseases, in which there is no spread from plant to plant in a growing season, the monomolecular disease progress model often is more appropriate than the logistic (5,34). $X(t)$ for this model can be written in terms of r_M , the monomolecular rate parameter (units of time^{-1}), and the previously defined K and X_0 (L. V. Madden and C. L. Campbell [34] equation 17). $X'(t)$ is written as

$$X'(t) = r_M(K - X_0)e^{-r_M t} \quad (4)$$

which can also be written in terms of $X(t)$ as $r_M[K - X(t)]$.

The proportion of the plant population newly infected during a very short time interval (dt) is given by $X'(t)dt$. More formally, $X'(t)dt$ represents the new infections (proportion scale) between time t and $t + dt$.

Yield loss for a population of plants. Incremental yield loss from times t to $t + dt$ for a population of plants is determined by multiplying the amount of new disease in this interval of time, $X'(t)dt$, by the loss function for plants infected at t , $\zeta(t)$. Even though plants infected early in an epidemic (e.g., $t = 0 - 10$ days) have the greatest per-plant yield losses ($\zeta(t)$, equation 1), there are few infected plants at this early time period for logistic disease increase because $X'(t)$ (equation 3) is very low at small t . Thus, the incremental population loss in yield ($\zeta(t)X'(t)dt$) from early infections is low. Because of the logistic nature of disease progress, most of the new plant infections occur at intermediate times (L. V. Madden and F. W. Nutter, Jr. [37] Fig. 5); it is in this time interval that the greatest population yield loss occurs. There is very little population-level yield loss due to infections at long time values, because there are few new infections and the per-plant loss from these infections is very low.

The dynamics of incremental population yield loss with monomolecular disease increase (equation 4) are different from those obtained with logistic increase. $X'(t)$ for the monomolecular model has its largest value at $t = 0$ and decreases monotonically over time (5). Therefore, $\zeta(t)X'(t)dt$ decreases throughout the epidemic, indicating the greatest incremental yield loss is at the beginning of the epidemic.

Total yield loss for the population of plants at the end of a growing season (T ; days) or the end of an epidemic is given by

$$L = \int_0^T \zeta(t)X'(t)dt + \alpha X_0 \quad (5)$$

Because the disease progress and the per-plant loss function are given on proportion scales, L is a variable that ranges from 0 (no loss) to 1 (no yield), depending on the model parameters. The αX_0 term (37; L. V. Madden and F. W. Nutter, Jr., data not shown) represents the population yield loss due to initial disease (infected plants at $t = 0$). If $r = 0/\text{day}$, such that there is no increase in disease beyond X_0 , then $L = \alpha X_0$. When equation 2a or 2b are used for $\zeta(t)$ in equation 5, αX_0 is replaced by $\alpha_1 X_0$ or $\alpha_2 X_0$, respectively.

There is no obvious analytical solution to equation 5 when $X'(t)$ is described by a logistic model (equation 3) and $\zeta(t)$ is explained by an exponential model (equation 1). However, numerical solutions are easy to obtain with commercial mathematical software such as Mathcad (MathSoft Inc., Cambridge, MA). Several properties of equation 5 are presented later.

For the monomolecular disease progress (equation 4) and the exponential per-plant loss function (equation 1), there is an analytical solution to equation 5 of the form

$$L = \left[\frac{(K - X_0)r_M \alpha}{r_M + \gamma} \right] \left[1 - e^{-(r_M + \gamma)T} \right] + \alpha X_0 \quad (6)$$

Because analytical solutions are only obtained for certain combinations of models for $X'(t)$ and $\zeta(t)$, we generally restrict the presentations of results to numerical solutions of equation 5.

Heterogeneity of disease incidence. It has been shown that spatial heterogeneity of disease intensity in a field affects the relationship between mean yield (loss) and mean disease intensity (12,21,22,24). Loss can be either increased or decreased compared with the homogeneous case (37), depending on the functional relationship between yield loss and disease intensity (incidence or severity) at a given time. Therefore, equation 5 was expanded to account theoretically for disease heterogeneity. The model developed here was based on time of infection, and not directly on disease incidence at any single time. Thus, the previous approach used by Hughes (22) and Ferrandino (12) cannot be followed exactly.

To account for heterogeneity of disease, we assume that the rate parameter for disease progression (r) is not fixed for a field but is a random variable with a probability density function or distribution labeled $f(r)$. The integration of $f(r)$ from arbitrary points r_1 to r_2 gives the probability of r being in the interval $r_2 - r_1$. One can visualize this as a field comprising a large number of sites, with the increase in disease incidence at each site being of the same form (e.g., logistic) but with a different r at each site. An example of this is found in Hughes et al. (25 Figure 6b). The variation in r could be due to environmental heterogeneity or spatiotemporal population-dynamic processes among the sites (13). Following the methodology of Piantadosi (45), mean disease incidence for a population with variable r can be written as

$$X(t) = \int_0^{r_{\text{high}}} X(t, r)f(r)dr \quad (7)$$

in which $X(t, r)$ is now disease incidence at time t for a site with rate r , and the integration is over the range of possible r values; integration from 0 to $r_{\text{high}} = 2/\text{day}$ for the logistic model will encompass all possible values of r , even when mean r (μ_r) is unrealistically high. We take the approach here that r has a continuous distribution rather than a discrete distribution, although equation 7 can be modified easily for discrete distributions (45).

In this approach, we assume that X_0 and K are fixed for a field, although the heterogeneity model could be expanded to accommodate any of these terms as random variables. We focus on r as a convenient way to consider variability in disease incidence in a

field. Although there is a substantial amount of published information on the distribution of disease incidence at individual times for various diseases (5,25,35), there is virtually nothing known about the distribution of r . Thus, we consider two distributions that can represent a very wide range of possible forms for $f(r)$, namely the uniform and Weibull (16). The uniform distribution (or density function) is written as

$$f(r) = 1/(b-a) \quad (8a)$$

for r values between parameters a and b , and 0 otherwise. With the uniform distribution, all values of r between a and b are equally likely. This is a reasonable distribution to use when there is no information on a variable, other than the likely extreme values (Gilchrist [15] section 8.7). The mean of the uniform distribution is $(a+b)/2$, and the standard deviation (σ_r) is $(b-a)/\sqrt{12}$. The Weibull distribution (or density function) is written as

$$f(r) = \left(\frac{cr^{c-1}}{d^c} \right) e^{-(r/d)^c} \quad (8b)$$

in which c is a unitless shape parameter and d is a scale parameter with the same units as r (16). When $c = 1$, equation 8b reduces to the exponential distribution. The Weibull distribution is very flexible and can have many shapes; it is positively skewed at $c < 3.6$, negatively skewed at $c > 3.6$, and symmetrical at $c \approx 3.6$. The mean of the Weibull distribution is $\Gamma[(c+1)/c] \cdot d$, in which $\Gamma(\bullet)$ is the gamma function. The standard deviation of the Weibull is given by

$$d \sqrt{\left[\Gamma\left(\frac{c+2}{c}\right) - \left[\Gamma\left(\frac{c+1}{c}\right) \right]^2 \right]}$$

Because the distribution of disease incidence is positively skewed at low mean incidence in most systems (25,35) and because there is sometimes a negative relationship between estimates of X_0 and r (5), it is likely that r is either symmetrical or negatively skewed (corresponding to $c \geq 3.6$), rather than positively skewed. For instance, fitting a logistic model to each of the 75 tobacco virus disease progress curves in Hughes et al. (25 Figure 6b) resulted in a mean r of 0.13/day, standard deviation of 0.022/day, and a coefficient of skewness of -0.1 (L. V. Madden, unpublished data).

Example distributions for r based on equations 8a and 8b are shown in Figure 2A. In each case, $\mu_r = 0.18$ /day. Three values of c were used to generate the Weibull values ($c = 1.6, 3.6$, and 5.6 ; labeled W(1.6), W(3.6), and W(5.6)), and d was calculated so μ_r equaled 0.18 for each case. For the uniform distribution, two sets of a and b parameters were selected and labeled 'U(a - b)'; 'U(0.14-0.22)' and 'U(0.04-0.32)' for a narrow and wide range, respectively, with $\mu_r = 0.18$ /day. Even when the epidemics at each site are all logistic, the field-level mean disease progress is not strictly logistic (45), although a sigmoid-type disease progress curve typically is obtained. The analytical solution to equation 7 when $f(r)$ is uniform is given in L. V. Madden and C. L. Campbell (34 equation 61). For many other forms of $f(r)$, including the Weibull distribution, there is no analytical solution for equation 7 when $X(t,r)$ represents logistic disease progress (45). However, numerical solutions can be obtained, as with equation 5.

The resulting disease progress curves (equation 7) based on the logistic model for these five situations (all with $\mu_r = 0.18$ /day) are shown in Figure 2B, together with the standard (fixed) case with no variation in r (i.e., fixed $r = 0.18$ /day). At short times ($t \leq 25$ days) the curves are very similar at this μ_r , but they deviate greatly at longer times. Above $t = 25$ days, the highest incidence is for the standard case of fixed r . Increasing variation in r at $t \geq 25$ days results in decreased incidence. This variation can be represented by the uniform distribution with increasing distance

between a and b or the Weibull distribution with decreasing c . Increasing degree of right-skewness (decreasing c of the Weibull) is also associated with decreasing disease incidence at a given t .

The mean yield loss for a population of plants with heterogeneous disease progress can be determined by extending equation 5 for variable r . The yield loss model can be written as

$$L = \int_0^{r_{\text{high}}} \int_0^t \zeta(t) X'(t,r) f(r) dt dr + \alpha X_0 \quad (9)$$

which does not have an analytical solution for realistic models of $\zeta(t)$, $X(t,r)$, and $f(r)$, although numerical solutions can be obtained. As an example, calculated losses for the six epidemics in Figure 2B, with $r_{\text{high}} = 2$ /day, $T = 120$ days, $\alpha = 0.9$, and $\gamma = 0.02$ /day are 0.510 (fixed), 0.501 'U(0.14-0.22)', 0.454 'U(0.04-0.32)', 0.496 'W(5.6)', 0.484 'W(3.6)', and 0.426 'W(1.6)'.

Numerical methods. Numerical solutions were determined for the population yield loss model (equation 5) based on (negative) exponential equation 1 for the time-of-infection loss function ($\zeta(t)$) and logistic equation 3 for rate of disease increase ($X'(t)$) for a wide range of values for α , γ , r , K , and X_0 . For most presenta-

Fig. 2. A, Weibull (W) and uniform (U) probability density functions for the rate term (r) of disease progress models with a mean (μ_r) of 0.18/day. Weibull curves (equation 8b), shown as thick lines, correspond to a shape parameter (c) of 1.6 (W(1.6)), 3.6 (W(3.6)), and 5.6 (W(5.6)), and scale parameter (d) chosen so that a μ_r of 0.18/day is obtained. Values of d are 0.201, 0.2, and 0.195 for W(1.6), W(3.6), and W(5.6), respectively. Uniform curves (equation 8a), shown as thin lines, correspond to a and b parameters of either 0.04 and 0.32 (U(0.04-0.32)) or 0.14 and 0.22 (U(0.14-0.22)), respectively. **B,** Disease progress curves for a field where disease increase is logistic (L. V. Madden and C. L. Campbell (34 equation 20) at each site in a field and r is fixed (0.18/day) or has one of the five probability-density functions (distributions) in A with $\mu_r = 0.18$ /day.

tions of results, L was expressed in relation to constant r values that ranged from 0 to 0.4/day at selected values of the other parameters. Solutions were also obtained for equation 5 with alternative models for $\zeta(t)$, specifically equations 2a and 2b (when maximum loss occurs at $t = \delta$) and with the monomolecular model for $X'(t)$ (equation 4). For some situations, the partial derivatives of L with respect to the parameters ($\partial L/\partial \alpha$, $\partial L/\partial \gamma$, $\partial L/\partial r$, $\partial L/\partial K$, and $\partial L/\partial X_0$) were determined numerically with Mathcad.

To assess the effects of heterogeneity of disease on L , the uniform (equation 8a) and Weibull (equation 8b) distributions were used for $f(r)$ in equation 9, and with equation 1 for $\zeta(t)$ and logistic equation 3 for $X'(t)$. Solutions were obtained for mean r values (μ_r) between 0 and 0.4/day at selected α , γ , K , and X_0 ($r_{\text{high}} = 2/\text{day}$). For the Weibull distribution, the three previously mentioned values of the shape parameter ($c = 1.6, 3.6$, and 5.6) were used, and values of d (equation 8b) were calculated from the respective formulas for μ_r . For instance, for $\mu_r = 0.18/\text{day}$ and $c = 3.6$, d is given by $0.18/T[(3.6 + 1)/3.6] = 0.20$. For the uniform distribution, a narrow and wide range for the interval between the a and b parameters were used for each μ_r . For the narrow range, parameters were determined as $a = 0.75\mu_r$ and $b = 1.25\mu_r$; for the wide range, parameters were $a = 0.2\mu_r$ and $b = 1.8\mu_r$. For example, with $\mu_r = 0.18/\text{day}$, a and b equaled 0.14 and 0.22, respectively, for the narrow range, and 0.04 and 0.32, respectively, for the wide range. These are the same as the values used in the example shown in Figure 2. With the parameter selections made for both distributions, σ_r varied with μ_r , but the coefficients of variation and skewness were fixed for all values of μ_r (Table 1).

Example. The population yield loss model (equation 5) was demonstrated with observed epidemiological data for SMV in soybeans. Separate experiments were performed to determine (i) yield loss of plants uniformly inoculated at selected times ($\zeta(t)$); and (ii) yield loss of a population of plants infected at various times in epidemics (L), starting from a small number of infected plants.

Time of infection. A $12.6 \times 12.6\text{-m}$ field plot of ‘Williams’ soybeans was planted on 16 May in Urbana, IL (M. Irwin, unpublished data). Row spacing was 76 cm, and standard agronomic practices were followed. There were five treatments, consisting of four different times of inoculation and one uninoculated control. Each treatment comprised a randomly chosen row in the field plot. There were four uninoculated rows between all treatment rows. All plants within a treatment row were sap (mechanically) inoculated at 9, 19, 34, or 57 days after planting with the G-5 strain of SMV (6). No virus symptoms were observed in the control treatment. All inoculated plants showed symptoms a week after inoculation.

Each row was divided in 60-cm-long segments, numbered consecutively. Segments 4, 8, 12, 16, and 20 in each row were hand-harvested at 140 days after planting. Seeds from each segment were separated with a small-plot thresher, and the seeds were

cleaned, weighed, and tested for moisture content. Seed weight was standardized to 13% moisture. Mean yield and standard deviation for each inoculation time and control was determined from the five row segments, and yield loss on a proportion scale was determined as described above.

The α and γ parameters of the $\zeta(t)$ function were estimated by first transforming equation 1 to

$$\ln[\zeta(t)] = \ln(\alpha) - \gamma t \tag{10}$$

and equation 10 was fitted to the log-transformed observed data (Appendix). A 90% prediction interval (42) was calculated for $\zeta(t)$ based on the standard deviation around the fitted regression line (Appendix). A prediction interval is wider than a confidence interval and represents the interval in which 90% of future observations (i.e., new values of observed loss for plants infected at t , not the mean of several observations) are expected to occur.

Disease progress curves. In a separate study, disease incidence was determined at several times in soybean plots that had a small percentage (2.5%) of plants inoculated early in the growing season. The data sets correspond to the four epidemics shown in Figure 4 in Ruesink and Irwin (48). Field plots, consisting of 20 15-m-long rows, were established in Illinois, Virginia, Mississippi, and Georgia. Row spacing was 76 cm, except in Georgia, where spacing was 91 cm. Cv. Essex from a single seed lot was used in all states. Plots were thinned to one plant per 10 cm, and 2 weeks after planting, every 40th plant was inoculated with sap from plants infected with the G-5 strain of SMV. Separate field plots were maintained in each state that were not inoculated to serve as controls for determining yield loss.

Plants in the inner 3 m of the four center rows of each plot were observed several times during the growing season for symptoms of soybean mosaic. Each plant was scored as diseased or disease free, and the proportion of diseased plants was determined. The first assessment was 2 to 3 weeks after inoculation. The number of disease assessments varied widely among the states, from eight in Illinois to three in Georgia. At the end of the season, all plants in the central section of the plots were hand-harvested. Seeds were separated with small-plot threshers, cleaned, weighed, and tested for moisture content. Seed weight was standardized to 13% moisture content. Mean yield was determined for each plot, and yield loss was calculated based on the difference in yield between inoculated and uninoculated plots, divided by the yield of the uninoculated plots.

The linear form of the logistic disease progress model (5) was fitted to the data because there was an insufficient number of assessment times for some states to use the nonlinear form of the model directly. The logistic model can be written as

$$\ln\left(\frac{X(t)}{K - X(t)}\right) = \beta_0 + rt \tag{11}$$

which is an equation for a straight line with slope r and intercept $\beta_0 = \ln[X_0/(K - X_0)] = -\ln(A)$. Because the incubation period of SMV is ≈ 5 days (48), we subtracted 5 from the actual observation times, so $X(t)$ represents estimated total disease incidence rather than disease incidence that has passed through the incubation period. Use of equation 11 allowed estimation of β_0 and r by standard linear-equation methods, provided that a reasonable estimate of K could be supplied (5). Thus, we fitted equation 11 to the data with normal-error least-squares regression analysis (Appendix), using a few selected K 's ranging from a maximum of 1.0 down to a value slightly above the largest observed $X(t)$, in increments of 0.05. The value of K that produced the largest coefficient of determination (R^2) and acceptable residual plot was the chosen K ($\hat{K}$). Least squares estimates of r and β_0 are given as $\hat{r}$ and $\hat{\beta}_0$, respectively. We use a different ‘overstriking’ symbol for estimated K here because the estimation method was different, and not all possible K 's were considered.

TABLE 1. Conditions of heterogeneity of the logistic rate parameter (r) evaluated in predicting mean yield loss for a population of plants (L) in relation to time of infection and disease progress

Label ^a	Distribution	Coefficient		90% Relative range ^b
		Variation	Skewness	
W(1.6)	Weibull	0.64	1.0	2.04
W(3.6)	Weibull	0.31	0.0	1.02
W(5.6)	Weibull	0.21	-0.3	0.68
U(wide)	Uniform	0.46	0.0	1.44
U(narrow)	Uniform	0.14	0.0	0.45

^a Number in parentheses represents the value of the shape parameter for the Weibull distribution (equation 8b) or the width of the interval between the lower and upper bounds for the uniform distribution (equation 8a). Parameter values were chosen so the coefficients of variation and skewness were fixed at all values of mean r (μ_r) from 0 to 0.4/day.

^b The difference between the 95th and 5th percentiles of the distribution divided by the mean (μ_r).

For each of the four data sets, predicted yield loss of the population (L) was calculated with equation 5 based on the estimates of r , K , and X_0 for each epidemic, with the estimates of α and γ for the independent time-of-infection study. That is, the same values

of α and γ were used for each epidemic, and these values were not based on the observed epidemics. An approximate 90% prediction interval for L was also determined (Appendix) for the four data sets.

RESULTS

Polycyclic diseases. Yield loss on a proportional scale (L ; equation 5) increased with increasing values of the logistic rate

Fig. 3. Solutions to equation 5 for population yield loss (L), based on use of the exponential function (equation 1) for time-of-infection response (loss function, $\zeta(t)$) and the logistic rate function (equation 3) for disease progress over time [$X'(t)$]. **A**, L versus the rate parameter (r) at selected γ values. **B**, L versus r at selected initial levels of disease incidence (X_0). **C**, L versus r for selected α values of the loss function and maximum possible disease incidence (K). In **C**, thick lines are for $K = 1$ and thin lines for $K = 0.5$. Fixed values of the other parameters for a given subfigure are shown above the frame. Parameter r and γ values are in units of days⁻¹. Epidemic duration (T) is 120 days. **A** and **B** adapted from Figure 6 in Madden and Nutter (37) and shown here for comparative purposes.

Fig. 4. Partial derivatives of population yield loss (L) with respect to **A**, the logistic rate parameter (r ($\partial L/\partial r$)); **B**, the time-of-infection parameter (γ ($\partial L/\partial \gamma$)); and **C**, the initial disease incidence (X_0 ($\partial L/\partial X_0$)). Partial derivatives were determined numerically from equation 5, with equation 1 for $\zeta(t)$ and equation 3 for $X'(t)$. Values of other parameters are given on the figure frames.

parameter (r), initial disease incidence (X_0), maximum disease incidence (K), and maximum per-plant loss (α) (Fig. 3A through C). Furthermore, yield loss decreased with increasing values of the time-of-infection (γ) parameter. Numerical solutions were obtained for all tested combinations of parameters when equation 3 was used for $X'(t)$ and equation 1 was used for $\zeta(t)$. As also shown previously (37), L increased rapidly with r at low to moderate r , and the increase slowed at high r (e.g., $\gamma = 0.02/\text{day}$ curve; Fig. 3A). L changed little with r when r was very close to 0/day. However, the change in L with r depended on the other parameters. This can be seen by the plot of the numerically determined $\partial L/\partial r$ versus r at two γ values (Fig. 4A). At a relatively low γ value of 0.02/day, $\partial L/\partial r$ increased with r up to $r \approx 0.05/\text{day}$ and declined rapidly at high r values. At a much higher γ of 0.08/day, $\partial L/\partial r$ did not reach a maximum until $r \approx 0.15/\text{day}$; the maximum partial derivative was low relative to the case with $\gamma = 0.02/\text{day}$.

L was very sensitive to slight changes in γ when γ was low ($0/\text{day} < \gamma \leq 0.02/\text{day}$) but insensitive to changes at high γ (Fig. 3A). For instance, a change in γ from 0.0 to 0.01/day had a larger effect on L than a change in γ from 0.02 to 0.04/day (especially at low r). The numerically determined partial derivative $\partial L/\partial \gamma$ was large and negative at small γ and rapidly approached 0 as γ increased (Fig. 4B). There also was an interaction of γ and r in terms of their effects on $\partial L/\partial \gamma$, as expected from inspection of Figure 3A. That is, at small γ values, $\partial L/\partial \gamma$ was larger (in absolute value) with small r than with large r ; at large γ values, $\partial L/\partial \gamma$ was larger with large r than with small r . A given level of L could be obtained in several ways. For instance, $L = 0.50$ was achieved with the conditions in Figure 3A with $\gamma = 0.01/\text{day}$ and $r \approx 0.09/\text{day}$ or with $\gamma = 0.02/\text{day}$ and $r \approx 0.18/\text{day}$.

L changed with X_0 in a near-logarithmic fashion at low X_0 (Fig. 3B), as also shown by Madden and Nutter (37). That is, logarithmic changes in X_0 were equivalent to linear changes in r in determining L . As an example, a 10-fold increase in X_0 from 10^{-4} to 10^{-3} had about the same effect on L as the 10-fold change in X_0 from 10^{-3} to 10^{-2} (Fig. 3B). With the parameter values in Figure 3B and $r = 0.2/\text{day}$, a change in X_0 from 0.01 to 0.001 resulted in a decline in L from 0.38 to 0.24; however, at a fixed X_0 of 0.01, r only had to be reduced from 0.2 to 0.125/day to obtain the same population yield loss. The magnitude of the numerically determined $\partial L/\partial X_0$ declined with increasing X_0 but also depended on the other parameters (Fig. 4C). For instance, $\partial L/\partial X_0$ decreased with increasing r (at $r \geq 0.03/\text{day}$) at most X_0 values (Fig. 4C; L. V. Madden, unpublished data).

K and α had similar effects on L over the range of tested r values (Fig. 3C). L was not sensitive to changes in values of K or

Fig. 5. Solutions to equation 5 for L based on the exponential function (equation 1) for time-of-infection response (loss function, $\zeta(t)$) and the monomolecular rate function (equation 4) for disease progress over time ($X'(t)$). Fixed values of the other parameters are shown above the frame. Parameter r_M and γ values are in units of days $^{-1}$. Epidemic duration (T) is 120 days.

α at small rates ($r \leq 0.07/\text{day}$) but very sensitive at high rates ($r \geq 0.20/\text{day}$). This was confirmed by the calculated partial derivatives. Specifically, $\partial L/\partial \alpha$ and $\partial L/\partial K$ were invariant to α and K , respectively, but both increased with increasing r (L. V. Madden, unpublished data).

Monocyclic diseases. For monocyclic diseases described by the monomolecular model (equation 4), the relationship between L and the parameters was similar to that found for logistic epidemics (Fig. 5). Numerical solutions of equation 5 were identical to the exact results obtained with equation 6. To make direct comparisons between results for disease progress models, one cannot use the same values of the rate parameter for two models (5), because, for example, a logistic r of 0.2/day is not equivalent to a monomolecular r_M of 0.2/day. Equivalence can be obtained by converting the rate parameter for both models to the mean absolute weighted rate of disease increase (ρ) (5). For the logistic model, $\rho = rK/6$, and for the monomolecular model, $\rho = r_M K/2$. Thus, logistic r values from 0.0 to 0.4/day (Fig. 3) are equivalent to monomolecular r_M values of 0.0 to 0.13/day.

L increased with r_M (Fig. 5) at the selected γ values in a manner very similar to that found for the logistic model, after correcting for the scale difference between r and r_M . There were only two conditions where differences were apparent. First, at low r_M , L increased with r_M faster than L increased with logistic r . This was confirmed by the numerically determined $\partial L/\partial r_M$ (Fig. 6A). Second, L was not reduced by increasing γ values to the same extent as with the logistic model. This was also evident in the plot of

Fig. 6. Partial derivatives of population-yield loss (L) with respect to **A**, the monomolecular rate parameter (r_M ($\partial L/\partial r_M$)) and **B**, the time-of-infection parameter (γ ($\partial L/\partial \gamma$)). Partial derivatives were determined numerically from equation 5, with equation 1 for $\zeta(t)$ and equation 4 for $X'(t)$. Values of other parameters are given on the figure frames.

$\partial L/\partial \gamma$ (Fig. 6B). Both results are due to the high rate of disease increase ($X'(t)$) at low t for monocyclic diseases (5). For instance, a considerable degree of incremental yield loss is realized in a population before the cumulative yield loss-reducing impact of high γ is achieved.

For the monomolecular model, L was not considered in relation to r_M at selected X_0 values because r_M is a direct function of X_0 for monocyclic diseases (5). That is, changing X_0 automatically changes r_M .

General theoretical results. Based on a direct analysis of equation 5 and assessment of numerical solutions when equation 1 was used for $\zeta(t)$, several broad results can be stated. Complete (100%) yield loss only is achievable if $\alpha = 1$ for maximum per-plant loss, $K = 1$ for the disease progress model (such that all plants are susceptible), and one of two conditions are met: (i) $\gamma = 0/\text{day}$ for the time-of-infection response; or (ii) all plants are infected at the beginning of the experiment ($X_0 = 1$). For condition one, L equals 1 if r (or r_M) and X_0 are sufficiently high so disease incidence actually reaches K by the end of the season. For condition two, r (or r_M) is not defined because all plants are instantly infected, and γ has no impact on results because there are no infections at $t > 0$. The latter situation is not considered further here.

In general terms, the maximum possible L equals αK , but this is only obtained when final incidence ($X(T)$) reaches K and $\gamma = 0/\text{day}$ (with $X_0 < 1$). With this extreme value for γ , time of infection does not affect $\zeta(t)$, such that per-plant loss is always equal to α , and L is a simple fraction of final incidence $\alpha \cdot X(T)$. When $\gamma > 0$, maximum L is less than αK , and predicted yield loss for any set of parameter values is less than $\alpha \cdot X(T)$ (Fig. 3A through C). At large γ (e.g., $\gamma \geq 0.08/\text{day}$), L is very low and is primarily determined by X_0 and α . This is because, with large γ , infections that occurred after the initial ones virtually have no effect on yield (Fig. 1). In fact, as γ increases indefinitely, L approaches $\alpha \cdot X_0$.

Alternative time-of-infection models. Population yield loss (equation 5) could be affected when time of maximum per-plant loss was delayed from $t = 0$ to $t = \delta$ days (equation 2a and 2b and Fig. 1), with maximum per-plant loss fixed at α . Direct comparisons of L are based on equal areas under the $\zeta(t)$ curves for equations 1, 2a, and 2b. At low values of the epidemic rate parameter (e.g., $r \leq 0.1/\text{day}$), shifting the time of maximum loss had only a minor effect on L . This is demonstrated in Figure 7 for fixed values of r for logistic disease progress and equation 2a for $\zeta(t)$. At higher r values ($0.1/\text{day} \leq r \leq 0.25/\text{day}$), L for the delay situation

Fig. 7. Solutions to equation 5 for population-yield loss (L) based on nominal equations 1 and 2a at two values of the delay parameter (δ ; units of days $^{-1}$) for the time-of-infection response ($\zeta(t)$) and the logistic rate function (equation 3) for disease progress over time ($X'(t)$). Maximum per-plant loss (α) was 0.9 for all curves; $\gamma = 0.02/\text{day}$ in equation 1, and $\gamma_1 = 0.03/\text{day}$ for $\delta = 15$ days and $\gamma_1 = 0.037/\text{day}$ for $\delta = 30$ days, both for equation 2a. The $\zeta(t)$ curves are described in Figure 1. Parameter r has units of days $^{-1}$. Epidemic duration (T) is 120 days.

(equations 2a and b) exceeded L for the nominal case (equation 1). L decreased at very high r values (e.g., $r \geq 0.25/\text{day}$) with $\delta > 0$ days (especially for $\delta > 15$ days). The reduction in L is because, at high values of r , a substantial proportion of the plants become infected before the time of maximum per-plant loss (δ). When the monomolecular model (equation 4) was used in conjunction with equation 2a, a reduction in L at high r_M also was evident (L. V. Madden, unpublished data). Results were very similar to those presented in Figure 7 when equation 2b was used for $\zeta(t)$ (data not shown).

Heterogeneity of disease progress curves. Variability in logistic r had an effect on population yield loss at moderate to high values of mean r (μ_r). This is demonstrated in Figure 8 for logistic epidemics (equation 3) and exponential loss function (equation 1), with three levels of variation (and skewness) according to the Weibull distribution, and two levels of variation with the uniform distribution (Table 1). At a μ_r of 0.15/day (other parameter values shown on the graph), heterogeneity had virtually no effect on L . As μ_r increased, the L curves separated, with the highest L for the nominal situation of fixed r (homogeneous disease progress throughout the fields) and lowest values of L corresponding to the highest coefficients of variation, U(wide), and positive skewness, W(1.6) (Table 1). With the lowest tested coefficient of variation or negative skewness (U(narrow) and W(5.6)), L was only slightly less than the nominal values, and the curves were too close together to label them in Figure 8. Similar effects of heterogeneity on L were found for other parameter values.

In general, increasing heterogeneity and skewness of r at fixed levels of the other parameters led to decreasing L (Fig. 8). However, an increase in heterogeneity and skewness of r also resulted in lower $X(t)$ relative to the fixed- r situation at most times (Fig. 2B shows an example). Thus, an increase in heterogeneity and skewness of r led to an increase in L at fixed $X(t)$, except at very short times (e.g., $t \leq 15$ days).

Example data analysis. Soybean yield loss was significantly ($P < 0.01$) affected by the time of infection in the controlled study

Fig. 8. Solutions to equations 5 or 9 for population yield loss (L), based on the exponential function (equation 1) for time-of-infection response (loss function, $\zeta(t)$) and the logistic rate function (equation 3) for disease progress over time ($X'(t)$). The rate parameter (r) of the logistic model was treated either as a constant (fixed) or a random variable with probability-density function ($f(r)$) defined by equation 8a (uniform) or 8b (Weibull). W(1.6) and W(3.6) = Weibull $f(r)$ with shape parameters of 1.6 and 3.6, respectively. U(wide) = uniform $f(r)$ with a wide range of r values (Table 1). The two curves immediately under 'fixed' label correspond to U(narrow) (uniform $f(r)$ with narrow range of r values) and W(5.6) (Weibull $f(r)$ with a shape parameter of 5.6). Thick lines correspond to Weibull distribution, and thin lines correspond to uniform distribution and to fixed r . At high r and mean r (μ_r), L values from high to low correspond to fixed r , U(narrow), W(5.6), W(3.6), U(wide), and W(1.6). Parameter r , μ_r , and γ values are in units of days $^{-1}$. Epidemic duration (T) is 120 days.

where all plants were uniformly inoculated (Fig. 9). The linearized form of equation 1 (equation 10) provided a satisfactory fit to the data (Table 2), resulting in parameter estimates of: $\hat{\alpha} = 0.84$ and $\hat{\gamma} = 0.015/\text{day}$.

Soybean mosaic disease progress curves were fairly similar for the four states where epidemics were initiated from a small number of inoculated plants (Fig. 10A). All epidemics were well-described by the linearized form of the logistic model (equation 11), although the selected value of K ($\bar{K}$) was different for each epidemic (Fig. 10B; Table 2). Coefficients of determination (R^2) were greater than 0.86.

Mean yield loss on a proportional basis for the population of plants (L), infected over many different days, ranged from 0.03 to 0.34 for these four data sets. L was predicted by equation 5 based on estimates of r , X_0 , and K for the corresponding epidemic and common values of $\hat{\alpha}$ and $\hat{\gamma}$ for all epidemics. Predicted L was higher than the observed L in three of the four cases, but the over-prediction was only large for the Georgia epidemic. It should be noted that these predictions were not based on curve fitting; instead, predictions were based on a quantitative description of the epidemics (independent of yield; Fig. 10B) and previously determined relationship between time of infection and yield (Fig. 9). The underprediction for the one data set (MS) was small. The 90% prediction interval for L , based on assumed uncertainty in the parameter estimates of the $\zeta(t)$ and $X(t)$ equations, included the observed L for all epidemics, except the one from Georgia (Table 2). The intervals were relatively wide because the parameter estimates were based on a small number of observations, especially for the Georgia site.

Equation 5 also could be used in an iterative curve-fitting mode. We assumed here that the logistic parameter estimates were exact (i.e., no uncertainty), and iteratively solved equation 5 with these epidemic parameter values and a range of α 's and γ 's to determine the α and γ values that resulted in a predicted L equal to the observed mean L . In general, there are many combinations of these two parameters that will produce the same predicted L for a single data set, so we choose the values of α and γ that were close to the estimates determined in the separate study. We labeled these estimates as $\hat{\alpha}$ and $\hat{\gamma}$ in Table 2. These estimates were very close to the independently determined values of α and γ for three data sets (Fig. 9). This was not attempted for the Georgia data set.

Fig. 9. Relationship between per-plant yield loss ($\zeta(t)$) of soybeans and time of infection (t ; days) by *Soybean mosaic virus* of the genus *Potyvirus*. Points represent mean, and vertical bars represent 90% confidence intervals for the means. Solid curve represents predicted values based on fitting equation 10 (or equation A1) to transformed loss data and back-transforming to obtain estimated $\zeta(t)$ (equation 1). Short dashed curves represent the 90% prediction interval for yield loss of individuals infected at the indicated times (equation A5a and A5b). It is assumed here that time of inoculation is the same as time of infection.

DISCUSSION

Expanding on the work of Madden (33) and Madden and Nutter (37), we have developed an analytical approach to characterize yield losses in crops in relation to systemic diseases, such as those caused by viruses, based on two premises supported by the literature: (i) disease incidence in a population of plants over time ($X(t)$) is described by any one of the many available nonlinear disease-progress models (5,34,43,52); and (ii) yield-loss of a plant ($\zeta(t)$) is a function of time of infection (1,4,17,18,41,48,50,57). Equation 5 is a mathematical representation of these two premises. Although the paper focused on the logistic and monomolecular models for $X(t)$ and the (negative) exponential for $\zeta(t)$, a wide range of models can be used for $\zeta(t)$ and $X(t)$, if justified on theoretical grounds or by the available data. Furthermore, the approach could be adapted for other nonsystemic diseases when severity rather than incidence is determined. Through the model parameters, one can explore the effects of attributes of disease progression (r , X_0 , and K) and the yield response of individual plants to infection (α and γ) on final yield loss for a plant population (L) in which individuals are infected at different and (unobserved) times.

In general, if there were some plants initially infected, L would be as low as αX_0 if there were no further disease increase (because $r = 0/\text{day}$) and as high as αK if $\gamma = 0/\text{day}$ and r was high enough so final disease incidence reached K . For the special case of maximum $\zeta(t)$ at a time later than the beginning of the season (equation 2a and 2b), minimum L was $\hat{\alpha}_1 X_0$ or $\hat{\alpha}_2 X_0$. L increased with increasing X_0 , r (or r_M), K , and α and with decreasing γ . The specific effects of the parameters on L can be determined through graphic evaluation of the numerical solutions of equation 5 (Figs. 3 and 5) or, in some cases, through an evaluation of the analytical solution (equation 6). In general, the greatest change in L with change in r (or r_M), γ , and X_0 occurred at relatively low r when $\gamma \leq 0.04/\text{day}$ (Figs. 3 through 6). The exception was when r (but not r_M) was very close to 0; in this case, L did not change much with the parameters. Conversely, the sensitivity of L to change in α and

TABLE 2. Estimates of r , β_0 , and K of equation 11 fitted to the N values of disease incidence for soybean mosaic epidemics in Illinois (IL), Virginia (VA), Mississippi (MS), and Georgia (GA), together with the standard deviation around the regression line (s), coefficient of determination (R^2) for the disease progress analysis, observed mean yield loss (L), and predicted yield loss ($\hat{L}$) based on equation 5, two forms of a 90% prediction interval for yield loss, and estimates of α and γ of equation 1 that result in equation 5 exactly producing the observed L values ($\hat{\alpha}, \hat{\gamma}$)^a

Term	IL	VA	MS	GA
$\hat{r}$ /day	0.098	0.091	0.081	0.108
β_0	-6.39	-4.69	-5.33	-4.57
$\bar{K}$ ^b	1.0	0.8	0.9	0.6
N	8	5	4	3
s	0.46	0.68	0.44	0.13
R^2	0.94	0.87	0.97	0.99
L (observed)	0.24	0.22	0.34	0.04
$\hat{L}$ (predicted)	0.31	0.32	0.29	0.27
Prediction interval				
Uncertain ζ ^c	0.24–0.42	0.25–0.41	0.22–0.38	0.22–0.35
Uncertain ζ $\hat{x}$ ^d	0.21–0.47	0.20–0.55	0.18–0.45	0.21–0.36
$\hat{\alpha}, \hat{\gamma}$	0.84, 0.020	0.84, 0.023	0.86, 0.013	... ^e

^a $\hat{L}$ is based on use of $\hat{\alpha} = 0.84$ and $\hat{\gamma} = 0.015/\text{day}$ in equation 5 for all four states.

^b $\bar{K}$ is determined iteratively with the linearized form of the logistic model (equation 11) fitted to the logits of observed disease incidence. With the selected $\bar{K}$, regression analysis was used to obtain $\hat{r}$ and β_0 . $\hat{x}_0$ can be determined from $\bar{K} (1 + e^{-\beta_0})^{-1}$.

^c The 90% prediction interval based on substituting equations A5a and A5b for $\zeta(t)$ (equation 1) in the population-yield loss model (equation 5).

^d The 90% prediction interval based on substituting equations A5a and A5b for $\zeta(t)$ (equation 1) and equation A7a and A7b for $X'(t)$ (equation 3) in the population yield-loss model (equation 5).

^e Not determined.

K was higher at large r than at small r . Moreover, at small r there was little effect of either heterogeneity of disease (equations 8a, 8b, and 9) or of a nonmonotonic time-of-infection response ($\zeta(t)$; equation 2a and 2b) on L (Figs. 7 and 8). At very high r , both of these factors could have a large effect on L .

Increasing disease intensity (incidence and severity [5]) typically leads to a reduction in yield, and the relationship can be described by one or more linear or nonlinear models (28,32,37). The traditional approach for characterizing this relationship is to express L (or yield) as a function of disease intensity at one or more times ($X(t)$) or as a function of AUDPC ($= \int X(t)dt$). Shape of the $L:X$ curve can be used, in some circumstances, to interpret the (physiological or ecological) effects that a disease has on a crop (24,31,37). This $L:X$ characterization has been done for plant virus systems (10,30,44,51). For comparative purposes, solutions to equation 5 can also be expressed as $L:X$ curves. For instance, Figure 11A shows L versus $X(90)$ (disease incidence at $t = 90$) for three of the γ curves in Figure 3A. Clearly, the value of γ has an effect on the shape of the $L:X$ curve, which varies from concave to convex depending on the magnitude of γ . The selected time during the epidemic used for determining the $L:X$ curve also can affect the results. This is shown in Figure 11B, where L is plotted versus disease incidence at three different times ($t = 60, 90$, and 120 days) for a single γ , α , X_0 , r , and K . The horizontal distance between curves reflects the increase in $X(t)$ between the indicated times. Also, in Figure 11A and B, there is a considerable range in L values at $X(t) = 1.0$. Although the $L:X$ curves provide one type of summary of the effects of disease incidence on yield loss of a population of plants, a more direct interpretation of yield loss in relation to systemic diseases can be made when L is related to α , γ , X_0 , r (or r_M), and K rather than to $X(t)$.

The model formulation for heterogeneous disease progress curves (equation 9) is based on parameter heterogeneity rather

than explicit spatial heterogeneity of disease. The model is only spatially implicit because the spatial location of the within-field sites is not utilized. Thus, the yield loss of a plant is not affected by the status of neighboring plants; only the time of infection, which is itself a function of the rate of disease increase at a given site, determines yield loss. Increasing degree of heterogeneity of r (and hence $X(t,r)$) and increasing positive skewness of the distribution of r resulted in decreasing L relative to the fixed- r situation (at constant values of the other parameters) (Fig. 8). The effect of heterogeneity was most evident, however, only at high r . This is analogous to the effect that heterogeneity of disease and pests has on the functional relationship between mean L and X for some observed and theoretical yield-loss relationships (12,21,22,37). For instance, increasing heterogeneity can result in higher or lower mean L at a given value of mean X , depending on the shape of the $L:X$ curve, and presumably, the mechanisms by which disease affects yield (14).

There is evidence in some systems that the yield (or loss) of a plant depends on the status of its neighbors (3,20,23,49,51). For instance, a healthy plant adjacent to diseased (or missing) plants may have a higher yield than a healthy plant adjacent to other healthy plants. Likewise, a diseased plant adjacent to healthy plants may have a lower yield than a diseased plant adjacent to other diseased plants. The aspatial equation 9 could be modified further to consider theoretically such spatial dependencies. One

Fig. 10. Disease progress curves for *Soybean mosaic virus* in field plots in four U.S. locations: Illinois (IL), Virginia (VA), Mississippi (MS), and Georgia (GA). **A**, Proportion of plants infected at assessment times since planting (t). **B**, Observed (points) and predicted (lines) logits of disease incidence (left side of equation 11) versus t . Logits were calculated with the values of the parameter for maximum disease incidence (K , different for each epidemic) and statistics for each epidemic given in Table 2.

Fig. 11. Solutions to equation 5 for population yield loss (L) versus disease incidence at selected time (t) values ($X(t)$). **A**, L versus $X(90)$ for three values of γ (0, 0.02, and 0.08/day) of the $\zeta(t)$ loss function (equation 1). **B**, L versus $X(t)$ for three t values (60, 90, and 120 days) at a single value of γ (0.02/day). Values of L are a subset of those in Figure 3A. Fixed values of the other parameters for a given subfigure are shown above the frames.

approach to modeling this phenomenon would be to have one or more of the $\zeta(t)$ parameters be a function of the disease-progress parameters. For example, one could make α a function of r , $\alpha(r)$. If $\alpha(r)$ decreases with increasing r , then plants at a site with high disease incidence (because of the high r) would be less affected by disease than plants at a site with low incidence. Conversely, if $\alpha(r)$ increases with increasing r , plants at a site with high incidence would be more affected by disease than plants at a site with low incidence. More complicated models could be developed, based on (i) the statistical distribution of diseased plants per field site (23,25), or (ii) spatially explicit representations of epidemics, where disease and yield status of spatially referenced individual plants are tracked and disease increases according to a spatiotemporal disease progress model (13,55). These expanded approaches for modeling yield loss are not explored further here, partly because the available data on time of infection do not justify such an analysis.

Applicability of our modeling approach was demonstrated with observed soybean yield-loss data. Yield loss of soybeans in relation to infection by SMV was well described by equation 5, using the exponential model for $\zeta(t)$, with parameters (α and γ) estimated from a separate study, and logistic model for $X(t)$ (Table 2). Discrepancies between observed and predicted L values for the four data sets could be due to uncertainty of the three parameters of the logistic model (equation 11), or more likely, misspecification of the α and γ parameter values of equation 1. The α and γ parameter values used (without curve fitting) were very close to the values that would exactly predict the observed losses found in three of the four fields ($\tilde{\alpha}$ and $\tilde{\gamma}$; Table 2), suggesting that the previously determined α and γ values were at least reasonable choices for these terms. Although (unmeasured) heterogeneity of the increase in disease incidence throughout the field could have influenced the population yield loss (equation 9), this was not of major importance for the SMV data because of the small effect heterogeneity has on predicted L at low r (Fig. 8). Likewise, possible misspecification of the functional form for $\zeta(t)$ would have had only a small impact on the soybean yield-loss predictions because of the low estimated r values (Table 2). That is, a non-monotonic loss function only has an effect on L at r values much larger than those calculated for SMV (Fig. 7). A higher precision of the predicted L (quantified by a narrower prediction interval; Appendix) could have been obtained with a larger number of inoculation times for the determination of $\zeta(t)$ or assessment times for the determination of epidemic parameters.

Fig. 12. Contour graph of equal levels of yield loss for a population (L) based on equation 5 (with equation 1 for loss function, $\zeta(t)$, and equation 3 for rate of disease increase, $X'(t)$) in relation to common (base 10) log of initial disease incidence ($\log(X_0)$) and rate parameter (r ; units of days⁻¹) of the logistic model (equation 11). Other parameter values are $\alpha = 0.9$, $\gamma = 0.04/\text{day}$, and $K = 1.0$. Labels within the frame represent levels of L , for example, the first region represents L from 0 to 0.01, the second region represents L from 0.01 to 0.05, and the last region represents L from 0.75 to 1.0. Largest possible L for these parameters is 0.9.

Using numerical or analytical solutions to equation 5, we can develop strategies to reduce or minimize losses due to a given systemic disease. For instance, if we had information on $\zeta(t)$ based on previous experimental work or expert opinion (11), we could determine the combination of initial disease incidence (X_0) and the rate parameter (r) that would result in a given yield loss (L). This is demonstrated in Figure 12, which is based on use of equation 1 for $\zeta(t)$ and logistic equation 3 (with $K = 1$) for $X'(t)$. A yield loss of 5% (or less) could be achieved, for example, with an r of 0.125/day if X_0 was less than or equal to 6×10^{-5} (i.e., $\log(X_0) \leq -4.22$) or with an X_0 of 0.055 (i.e., $\log(X_0) = -1.26$) if $r = 0/\text{day}$ (so there was a single initial disease incidence at $t = 0$ and no further increase). Other combinations of $\log(X_0)$ and r for a yield loss of 5% can be found from the contours. Thus, we could use a graph such as Figure 12 to decide between available disease management options, according to whether it is more efficient to reduce r or X_0 to maintain yield loss below some maximum value.

Contour graphs such as Figure 12 can be used to evaluate other combinations of parameters of equation 5. For instance, if there was little opportunity to alter disease progression in a given pathosystem during a growing season, but different cultivars could be planted that differed in terms of tolerance to disease, as quantified through parameters of $\zeta(t)$, one could prepare a contour graph of L for values of α and γ at the likely (or maximum) values of r and X_0 . One then could choose the cultivar with the most desirable properties in terms of yield losses due to the disease.

In conclusion, the analytical technique developed here for modeling yield loss provides a framework for analyzing observed crop losses in relation to time of plant infection and disease progress curves and for assessing disease management strategies based on likely yield losses. Because the developed model is based on rates of disease increase, the general approach can easily accommodate more complicated and general epidemic models, such as those based on linked differential equations (29,34,36).

APPENDIX

Time of infection. Equation 10 can be written as a statistical model:

$$Z = \beta_0 + \beta_1 t + \epsilon \quad (\text{A1})$$

in which t is the independent variable (predictor), Z ($\ln[\zeta(t)]$) is the dependent variable, ϵ is the error term (unexplained variability), and β_0 ($\ln(\alpha)$) and β_1 ($-\gamma$) are parameters representing the intercept and slope, respectively. Because the variance of observed $\zeta(t)$ for the soybean mosaic data was approximately constant (L. V. Madden, unpublished data), we assumed that ϵ in equation A1 had a variance inversely proportional to $\zeta(t)^2$ (42), and mean of zero. Thus, weighted least-squares regression (42) was used to estimate the parameters, with weights equal to $(\zeta(t))^2$. Parameter estimates are given as $\hat{\beta}_0$ and $\hat{\beta}_1$.

Using the parameter estimates from regression analysis, predicted Z is given by

$$\hat{Z} = \hat{\beta}_0 + \hat{\beta}_1 t \quad (\text{A2})$$

Equation A2 can be used to predict Z ($\ln[\zeta(t)]$) at any (reasonable) t , not just for the inoculation times when observations were made. A new observation is written as t_{new} , and predicted Z is written as $(\hat{Z}_{\text{new}})$. The standard deviation of predicted Z at $t = t_{\text{new}}$ [$s(\hat{Z}_{\text{new}})$] is given by

$$s(\hat{Z}_{\text{new}}) = s \sqrt{1 + \frac{1}{n} + \frac{(t_{\text{new}} - \bar{t})^2}{\sum (t - \bar{t})^2}} \quad (\text{A3})$$

in which $\bar{t}$ is the mean time of the n observations used in estimating the parameters, the summation term is for the original observations, and s is the standard deviation around the regression line (the square-root of the mean square for error or residual mean

square). A $(1 - P) \cdot 100\%$ prediction interval for Z at time t_{new} is given by

$$\hat{Z}_{\text{new}} \pm s(\hat{Z}_{\text{new}}) \cdot t(1 - P/2; n - 2) \quad (\text{A4})$$

in which $t(1 - P/2; n - 2)$ is the student t statistic for significance level P and $n - 2$ degrees of freedom (42). The lower and upper bounds of the prediction interval determined with equation A4 can be written as $\hat{Z}_{\text{new},l}$ and $\hat{Z}_{\text{new},h}$, respectively.

A prediction interval for $\zeta(t)$ can be determined by back-transforming the predicted Z values to

$$\hat{\zeta}(t)_{\text{new},l} = e^{\hat{Z}_{\text{new},l}} \quad (\text{A5a})$$

and

$$\hat{\zeta}(t)_{\text{new},h} = e^{\hat{Z}_{\text{new},h}} \quad (\text{A5b})$$

As an alternative, the prediction interval for $\zeta(t)$ could have been based on fitting equation 1 directly to the observed loss data, and calculating predictions without the need for back-transformations. However, the limited number of inoculation times in the soybean mosaic data sets precluded this type of nonlinear regression analysis.

Disease progress curves. Equation 11 can be written as equation A1, with $Z = \ln\{X(t)/[K - X(t)]\}$, $\beta_0 = \ln[X_0/(K - X_0)]$, and $\beta_1 = r$ (5). In this formulation, equation 11 was fitted to the logits of observed disease incidence ("empirical logits") for each disease progress curve using ordinary least-squares regression and predicted logits of disease determined with equation A2 and standard errors with equation A3. A prediction interval was determined for Z at selected time values by equation A4. A prediction interval for $X(t)$ then was estimated through back-transformation as

$$\hat{X}(t)_{\text{new},l} = \bar{K} / (1 + e^{-\hat{Z}_{\text{new},l}}) \quad (\text{A6a})$$

and

$$\hat{X}(t)_{\text{new},h} = \bar{K} / (1 + e^{-\hat{Z}_{\text{new},h}}) \quad (\text{A6b})$$

in which $\bar{K}$ is the preselected K . Prediction interval for the absolute rate of change of disease incidence at time $t[X'(t)]$ can be estimated for the logistic model from

$$\hat{X}'(t)_{\text{new},l} = \hat{r} \hat{X}(t)_{\text{new},l} \left(1 - \frac{\hat{X}(t)_{\text{new},l}}{\bar{K}} \right) \quad (\text{A7a})$$

and

$$\hat{X}'(t)_{\text{new},h} = \hat{r} \hat{X}(t)_{\text{new},h} \left(1 - \frac{\hat{X}(t)_{\text{new},h}}{\bar{K}} \right) \quad (\text{A7b})$$

Prediction interval for yield loss of the population. A 90% prediction interval was calculated in two ways. For the first approach, it was assumed that the disease progress curve was known with certainty, and the only uncertain component was the exact form of $\zeta(t)$. The premise here is that the chosen disease progress model completely describes the observed incidence data, but the impact of time of infection on yield is not fully known, because the latter is not directly observed with the given data set. A prediction interval for L can be obtained by substituting equations A5a and A5b for $\zeta(t)$ in equation 5, followed by numerical integration.

For the second approach, it was assumed that there was uncertainty in both the characterization of disease progress over time and the $\zeta(t)$ function. This is probably a more realistic approach, because the coefficients of determination were generally less than 1 for the fit of equation 11 to the soybean mosaic disease progress curves discussed earlier. A prediction interval for L can be obtained here by substituting equation A5a and A5b for $\zeta(t)$ and equation A7a and A7b for $X'(t)$ in the population yield-loss equation (equation 5). This approach is only an approximation and probably gives an overly wide prediction interval because the lower (or upper) bounds of $X'(t)$ and $\zeta(t)$ are always used together. In reality, there may be some correlation in these two predicted values (34), but there is no information on this for equation 5.

ACKNOWLEDGMENTS

Salaries and research support for L. V. Madden were provided by state and federal funds appropriated to the Ohio Agricultural Research and Development Center, Ohio State University. We thank R. M. Goodman, W. G. Ruesink, G. E. Kampmeier, and G. A. Schultz for contributions toward the design and execution of the field studies.

LITERATURE CITED

1. Avilla, C., Collar, J. L., Duque, M., and Fereres, A. 1997. Yield of bell pepper (*Capsicum annuum*) inoculated with CMV and/or PVY at different time intervals. *Z. Pflanzenkr. Pflanzenschutz* 104:1-8.
2. Bergamin-Filho, A., Carneiro, S. M. T. P. G., Godoy, C. V., Amorim, L., Berger, R. D., and Hau, B. 1997. Angular leaf spot of Phaseolus beans: Relationships between disease, healthy leaf area, and yield. *Phytopathology* 87:506-515.
3. Blodgett, F. M. 1941. A method for the determination of losses due to diseased or missing plants. *Am. Potato J.* 18:132-135.
4. Bosque-Perez, N. A., Olojede, S. O., and Buddenhagen, I. W. 1998. Effect of maize streak virus disease on the growth and yield of maize as influenced by varietal resistance levels and plant stage at time of challenge. *Euphytica* 101:307-317.
5. Campbell, C. L., and Madden, L. V. 1990. *Introduction to Plant Disease Epidemiology*. Wiley-Interscience, New York.
6. Cho, E. K., and Goodman, R. M. 1979. Strains of soybean mosaic virus: Classification based on virulence in resistant soybean cultivars. *Phytopathology* 69:467-470.
7. Chun, S.-C., and Schneider, R. W. 1998. Sites of infection by *Pythium* species in rice seedlings and effects of plant age and water depth on disease development. *Phytopathology* 88:1255-1261.
8. De Koeijer, K. J., and van der Werf, W. 1995. Effect of beet yellowing viruses on light interception and light use efficiency of the sugarbeet crop. *Crop Prot.* 14:291-297.
9. Doodson, J. K., and Saunders, P. J. W. 1970. Some effects of barley yellow dwarf virus on spring and winter cereals in field trials. *Ann. Appl. Biol.* 66:361-374.
10. Eckel, R. V. W., and Lampert, E. P. 1993. Effect of tobacco etch virus on the seasonal growth of flue-cured tobacco. *Crop Prot.* 12:505-510.
11. Edwards-Jones, G. 1993. Knowledge-based systems for crop protection: Theory and practice. *Crop Prot.* 12:565-578.
12. Ferrandino, F. J. 1989. Spatial and temporal variation of a defoliating plant disease and reduction in yield. *Agric. For. Meteorol.* 47:273-289.
13. Filipe, J. A. N., and Gibson, G. J. 1998. Studying and approximating spatiotemporal models for epidemic spread and control. *Philos. Trans. R. Soc. Lond. B. Biol. Sci.* 53:2153-2164.
14. Gaunt, R. E. 1995. The relationship between plant disease severity and yield. *Annu. Rev. Phytopathol.* 33:119-144.
15. Gilchrist, W. 1984. *Statistical Modeling*. John Wiley & Sons, Ltd., Chichester, England.
16. Hastings, N. A. J., and Peacock, J. B. 1974. *Statistical Distributions*. Butterworths, London.
17. Heijbroek, W. 1988. Factors affecting sugar beet losses caused by beet mild yellowing virus and beet yellows virus. *Meded. Fac. Landbouwwet. Rijksuniv. Gent.* 53:507-514.
18. Herbert, D. A., Stromberg, E. L., Chappell, G. F., and Malone, S. M. 1999. Reduction of yield components by barley yellow dwarf infection in susceptible winter wheat and winter barley in Virginia. *J. Prod. Agric.* 12:105-109.
19. Hills, F. J., Chiarappa, L., and Geng, S. 1980. Powdery mildew of sugar beet: Disease and crop loss assessment. *Phytopathology* 70:680-682.
20. Hirst, J. M., Hide, G. A., Stedman, O. J., and Griffith, R. L. 1973. Yield compensation in gappy potato crops and methods to measure effects of fungi pathogenic on seed tubers. *Ann. Appl. Biol.* 73:143-150.
21. Hughes, G. 1990. Characterizing crop responses to patchy pathogen attack. *Plant Pathol.* 39:2-4.
22. Hughes, G. 1997. Incorporating spatial pattern of harmful organisms into crop loss models. *Crop Prot.* 15:407-421.
23. Hughes, G., and Madden, L. V. 1997. The determination of yield losses with aggregated patterns of diseased or missing plants. Pages 156-160 in: *Exercises in Plant Disease Epidemiology*. L. J. Francl and D. Neher,

- eds. The American Phytopathological Society, St. Paul, MN.
24. Hughes, G., and McKinlay, R. G. 1988. Spatial heterogeneity in yield-pest relationships for crop loss assessment. *Ecol. Model.* 41:67-73.
25. Hughes, G., McRoberts, N., Madden, L. V., and Nelson, S. C. 1997. Validating mathematical models of plant disease progress in space and time. *IMA J. Math. Appl. Med. Biol.* 14:85-112.
26. Irwin, M. E., and Schultz, G. A. 1981. Soybean mosaic virus. *FAO Plant Prot. Bull.* 29:41-55.
27. James, W. C., Lawrence, C. H., and Shih, C. S. 1973. Yield losses due to missing plants in potato crops. *Am. Potato J.* 50:345-352.
28. James, W. C., and Teng, P. S. 1979. The quantification of production constraints associated with plant diseases. Pages 201-267 in: *Applied Biology*, Vol. 4. T. H. Coaker, ed. Academic Press, New York.
29. Jeger, M. J., van den Bosch, F., Madden, L. V., and Holt, J. 1998. A model for analyzing plant-virus transmission characteristics and epidemic development. *IMA J. Math. Appl. Med. Biol.* 15:1-18.
30. Johnson, C. S., and Main, C. E. 1983. Yield/quality trade-offs of tobacco mosaic virus-resistant cultivars in relation to disease management. *Plant Dis.* 67:886-890.
31. Johnson, K. B. 1987. Defoliation, disease, and growth: A reply. *Phytopathology* 77:1495-1497.
32. Madden, L. V. 1983. Measuring and modeling crop losses at the field scale. *Phytopathology* 73:1591-1596.
33. Madden, L. V. 1990. Understanding and predicting losses due to virus diseases: *Virus en Planta. Rev. Invest. Agropecu.* 22:130-139.
34. Madden, L. V., and Campbell, C. L. 1990. Nonlinear disease progress curves. Pages 181-229 in: *Epidemics of Plant Diseases; Mathematical Analysis and Modeling*. 2nd ed. J. Kranz, ed. Springer-Verlag, Berlin.
35. Madden, L. V., and Hughes, G. 1995. Plant disease incidence: Distributions, heterogeneity, and temporal analysis. *Annu. Rev. Phytopathol.* 33:529-564.
36. Madden, L. V., Jeger, M. J., and van den Bosch, F. 2000. A theoretical assessment of the effects of vector-virus transmission mechanism on plant virus disease epidemics. *Phytopathology* 90:576-594.
37. Madden, L. V., and Nutter, F. W., Jr. 1995. Modeling crop losses at the field scale. *Can. J. Plant Pathol.* 17:124-137.
38. Madden, L. V., Pirone, T. P., and Raccah, B. 1987. Temporal analysis of two viruses increasing in the same tobacco fields. *Phytopathology* 77:974-980.
39. Matthews, R. E. F. 1991. *Plant Virology*. 3rd ed. Academic Press, New York.
40. Moriones, E., Aramburu, J., Riudavets, J., Arno, J., and Lavina, A. 1998. Effect of plant age at time of infection by tomato spotted wilt tospovirus on the yield of field-grown tomato. *Eur. J. Plant Pathol.* 104:295-300.
41. Mzira, C. N. 1984. Assessment of effects of maize streak virus on yield of maize. *Zimb. J. Agric. Res.* 22:141-149.
42. Neter, J., Wasserman, W., and Kutner, M. H. 1983. *Applied linear regression models*. Richard D. Irwin, Homewood, IL.
43. Nutter, F. W., Jr. 1997. Quantifying the temporal dynamics of plant virus epidemics: A review. *Crop Prot.* 16:603-618.
44. Nutter, F. W., Jr., Kuhn, C. W., and All, J. N. 1989. Models to estimate yield losses in bell pepper caused by tobacco etch virus epidemics. (Abstr.) *Phytopathology* 79(suppl.):S1213.
45. Piantadosi, S. 1987. Generalizing growth functions assuming parameter heterogeneity. *Growth* 51:50-63.
46. Rosenkranz, E. E., and Scott, G. E. 1978. Effect of plant age at time of inoculation with maize dwarf mosaic virus on disease development and yield in corn. *Phytopathology* 68:1688-1692.
47. Ross, J. P. 1969. Effect of time and sequence of inoculation of soybeans with soybean mosaic and bean pod mottle viruses on yields and seed characters. *Phytopathology* 59:1404-1408.
48. Ruesink, W. G., and Irwin, M. E. 1986. Soybean mosaic virus epidemiology: A model and some implications. Pages 295-313 in: *Plant Virus Epidemics: Monitoring, Modelling, and Predicting Outbreaks*. G. D. McLean, R. G. Garrett, and W. G. Ruesink, eds. Academic Press, Sydney, Australia.
49. Scott, G. E., and Rosenkranz, E. E. 1981. Effect of field distribution of maize dwarf mosaic-diseased corn plants on yield. *Plant Dis.* 65:802-803.
50. Scott, G. E., Rosenkranz, E. E., and Nelson, L. R. 1977. Yield loss of corn due to corn stunt disease complex. *Agron. J.* 69:92-94.
51. Thongmeeakom, P., Paschal, E. H., II, and Goodman, R. M. 1978. Yield reductions in soybeans infected with cowpea mosaic virus. *Phytopathology* 68:1549-1551.
52. Thresh, M. J. 1983. Progress curves of plant virus disease. *Appl. Biol.* 8:1-85.
53. Ullstrup, A. J., and Miles, S. R. 1957. The effects of some leaf blights of corn on grain yield. *Phytopathology* 47:393-398.
54. Van Bruggen, A. H. C., and Rubatzky, V. E. 1992. Use of transplants instead of direct seeding to reduce corky root severity and losses due to corky root in iceberg lettuce. *Plant Dis.* 76:703-708.
55. Van den Bosch, F., Metz, J. A. J., and Diekmann, O. 1990. The velocity of population expansion. *J. Math. Biol.* 4:337-351.
56. Van der Werf, W., Westerman, P. R., Verweij, R., and Peters, D. 1989. The influence of primary infection date and establishment of vector populations on the spread of yellowing viruses in sugar beet. *Ann. Appl. Biol.* 121: 57-74.
57. Watson, M. A., Watson, D. J., and Hull, R. 1946. Factors affecting the loss of yield of sugar beet caused by beet yellows virus. *J. Agric. Sci.* 36:151-166.