Ecology and Epidemiology

Empirical Estimation of the Asymptotes of Disease Progress Curves and the Use of the Richards Generalized Rate Parameters for Describing Disease Progress

E. W. Park and S. M. Lim

Graduate research assistant, and plant pathologist and professor, respectively; Agricultural Research Service, U.S. Department of Agriculture, and Department of Plant Pathology, University of Illinois at Urbana-Champaign, Urbana 61801.

Portion of a thesis submitted by the first author in partial fulfillment of the requirement for the Ph.D. degree, University of Illinois.

The authors wish to thank J. K. Pataky for critical review of the manuscript.

Accepted for publication 20 December 1984 (accepted for electronic processing).

ABSTRACT


The effect of fixing the asymptote parameter of growth functions at 1 on estimates of the rate parameter and the use of generalized rate parameters proposed by Richards are discussed. When disease progress curves have asymptotes which are less than 1, the asymptote parameter of growth functions needs to be estimated empirically; otherwise, underestimation of the rate parameter of growth functions (e.g., Vanderplank's apparent infection rate, r) and changes in the rank of estimates of the rate parameter may result from fixing the asymptote parameter of growth functions at 1. The use of the weighted mean absolute growth rate and the weighted mean relative growth rate of the Richards function as the "absolute rate of disease progress" (Ra) and the "relative rate of disease progress" (Rr), respectively, is proposed for describing and comparing epidemics with different asymptotes and shapes of disease progress curves. Since they are determined empirically without the unrealistic assumption on the upper limit of disease severity, they provide more accurate information on disease development than Vanderplank's apparent infection rate or the rate parameter of growth functions with the asymptote value of 1. Development of bacterial blight in a soybean field was described using Ra and Rr.

Additional key words: Glycine max, Gompertz model, logistic function, monomolecular function, Pseudomonas syringae pv. glycinea, Weibull model.

Since Vanderplank (28) proposed the monomolecular and the logistic functions for describing development of monocyclic and polycyclic diseases, respectively, these functions have been extensively used as models of disease development. However, disease development data often do not conform to either the logistic or the monomolecular model. This is probably because the ecological basis of the models are a crude approximation of vastly complex pathosystems and lack the flexibility necessary to accommodate the diverse aspects of disease development. Model flexibility can be enhanced by increasing the number of parameters in the model. Statistical models of the polynomial type are flexible; however, polynomial models are not often used for disease progress primarily because of difficulty in biological interpretation. As for mechanistic models, the number of parameters can be increased either by generalizing several specific models (19, 27) or by incorporating new parameters based on more detailed reasoning about the component processes (10, 29). Increase in flexibility by empirically using the Richards function (19) will be discussed in this paper. The Richards function is a generalized form of growth functions (Table 1).

Growth functions that are often used in studying plant disease epidemics include the monomolecular, the logistic, and the Gompertz functions. Disease progress is commonly modeled by empirically fitting these functions to transformed or untransformed disease progress data. In doing so, the asymptote value of the disease progress curve is fixed at 1 (100% disease severity) based on the assumption that all host area becomes diseased at the end of the epidemic. However, the maximum severity of any particular disease is a result of the interactions among the host, the pathogen, and the environmental conditions, and it often approaches an asymptote which is less than 1 (e.g., 4.6, 11, 22, 25). If disease development approaches an asymptotic maximum severity of less than 1, disease progress curves cannot be most accurately fitted by any function with the asymptote value of 1. Thus, the most commonly used forms of the monomolecular, the logistic, and the Gompertz functions are often inappropriate. The importance of determining correct asymptote values when developing appropriate disease progress models has been noted (1, 2, 12, 14, 15, 26, 27). Analysis (1, 2), and Hau and Kranz (12) noted that determination of the asymptote value on the empirical basis improved goodness-of-fit of disease progress models. Kiyosawa (14), Kushalappa and Ludwig (15), and Turner et al (27) suggested corrections for the asymptote value. For a computer simulation of disease progress, Teng and Zadoks (26) used the logistic function in which the actual maximum severity was incorporated. Empirical estimation of the asymptote value is also important in obtaining a correct estimate of the rate parameter in disease progress models. Analysis (1, 3) identified the influence of the asymptote value on the rate parameter of growth functions and demonstrated how the asymptote value could affect the calculation of the rate parameter.

In this article we examine the consequence of using a model that assumes the asymptote is equal to 1 when in fact it is less than 1, and the use of generalized rate parameters developed by Richards (19) to compare epidemics with different asymptotic values and shapes of progress curves. Computer-generated data are used in this paper for the purpose of clarity in comparing models with asymptotes of 1 and less than 1, and for introduction of Richards' generalized rate parameters. Richards' parameters are then applied to comparisons of bacterial blight epidemics in a soybean field in Illinois in 1981. An abstract describing some of the work was published (17).

CHARACTERISTICS OF RATE PARAMETERS

Asymptote (A) and rate (k) parameters. The logistic function (Table 1) is used to illustrate the consequence of fitting the model with the asymptote parameter A fixed at 1 or estimated empirically. Model 1 \[ Y = \frac{A}{1 + e^{B-ki}} \] describes the logistic function with an empirically fitted asymptote. Model 2 \[ Y = \frac{1}{1 + e^{B-ki}} \] describes the logistic function with the asymptote fixed at 1. Model 2 is commonly used by plant pathologists to determine Vanderplank's apparent infection rate, r (i.e., the rate parameter k of model 2). Suppose that model 1 is true (i.e., maximum severity is less than 1), but that B and k are estimated by assuming that model...
2 is true (i.e., maximum severity equals 1). We consider the case where there is no random error since this shows the comparison more clearly and since more complicated cases should have fewer problems. Nine sets of data were generated by model 1 with different values of $A$, $B$, and $k$ which were chosen to represent disease progress curves with three distinct aspects of development. The data sets are plotted in Figs. 1–3. Fig. 1 represents disease that progresses rapidly, reaches maximum severity early in the growing season, and remains at the maximum level during the rest of the season. Fig. 2 represents disease progress that slowly develops and asymptotically reaches maximum severity at the end of the season. Fig. 3 represents disease progress that develops slowly but that is still in the increasing phase at the end of the season. The least-

squares program for nonlinear models (NLIN Procedure with Marquardt’s compromise method) of the Statistical Analysis System (SAS) (21) was used to obtain estimates of the parameters of the models.

Estimates of the rate parameter $k$ determined from model 2 differ substantially from the true $k$ values of model 1 (Table 2). Differences are greatest when the true value of $k$ is large, and when curves approach their asymptote early in the season. In all cases, model 2 underestimated the rate parameter. When estimates of $k$ from model 2 are compared, their ranking in order of magnitude is not the same as that of the true values of $k$ from model 1. Also, estimates of $B$ from model 2 are not the same as the true values of $B$.

The coefficients of determination ($R^2$) indicate that model 2 is not appropriate, especially for curves that approach their asymptotes early in the season and have lower asymptote values. Residual plots of Model 2 for the first six cases of Table 2 (curves that approached

| TABLE 1. Growth functions that have been used for disease progress models |
|-----------------------------|-----------------------------|
| **Growth function** | **Equation** |
| Monomolecular | $Y = A[1 - e^{(B-k)t}]$ |
| Logistic | $Y = A\frac{1}{1 + e^{(B-k)t}}$ |
| Gompertz | $Y = Ae^{-e^{(B-k)t}}$ |
| Richards | $Y = A[1 - e^{(B-k)t}]_m(1 - m)$ when $0 < m < 1$ |
| | $Y = A[1 + e^{(B-k)t}]_m(1 - m)$ when $1 < m$ |

$A$ = disease severity ($0 \leq Y \leq 1$); $A$ = asymptote parameter; $B$ = base of the natural logarithm; $k$ = rate parameter; $m$ = shape parameter; $t$ = time.

The Richards function is the generalized form of growth functions. When $m = 0$, the Richards function becomes the monomolecular function, when $m = 2$, it becomes the logistic function. Although the Richards function is not defined when $m = 1$, as $m$ approaches 1, it closely approximates the Gompertz function. High values of $m$ represent growth curves that show prolonged exponential growth until the upper limit of growth is closely approached, then growth ceases abruptly (20).

| TABLE 2. Estimates of parameters $B$ and $k$ obtained by Model 2 when Model 1 represents the true situation |
|-----------------------------|-----------------------------|
| **Model (True)** | **Model 2** |
| $A$ | $B$ | $k$ | $A$ | $B$ | $k$ | $R^2$ |
| 0.8 | 5 | 0.4 | 1 | 0.97 | 0.051 | 0.571 |
| 0.4 | 5 | 0.4 | 1 | 1.22 | 0.011 | 0.401 |
| 0.2 | 5 | 0.4 | 1 | 2.01 | 0.008 | 0.381 |
| 0.8 | 5 | 0.1 | 1 | 3.54 | 0.060 | 0.968 |
| 0.4 | 5 | 0.1 | 1 | 3.11 | 0.031 | 0.886 |
| 0.2 | 5 | 0.1 | 1 | 3.63 | 0.025 | 0.860 |
| 0.8 | 5 | 0.05 | 1 | 5.04 | 0.047 | 0.999 |
| 0.4 | 5 | 0.05 | 1 | 5.44 | 0.041 | 0.998 |
| 0.2 | 5 | 0.05 | 1 | 6.01 | 0.039 | 0.996 |

*Model 1: $Y = A\frac{1}{1 + e^{(B-k)t}}$ |
*Model 2: $Y = [1 + e^{(B-k)t}]_m(1 - m)$ |

*Model 2 was fitted to data sets that were generated by Model 1. Therefore, parameters $B$ and $k$ of Model 2 were estimated assuming Model 2 was true when in fact Model 1 was true. It was assumed that there was no random error involved in this case.

*The coefficient of determination.

**Fig. 1. Disease progress curves generated by the logistic function $Y = \frac{A}{1 + e^{(B-k)t}}$ with $A = 5$, $B = 0.4$, and three values of $A$. These curves represent disease progress that develops rapidly, reaches asymptotic maximum severity early in the growing season, and remains at the maximum level during the rest of the growing season.**

**Fig. 2. Disease progress curves generated by the logistic function $Y = A\frac{1}{1 + e^{(B-k)t}}$ with $B = 5$, $k = 0.1$, and three values of $A$. These curves represent disease progress that develops slowly and reaches asymptotic maximum severity at the end of the growing season.**
their asymptotes) showed strong systematic trends. Residuals were negative for the tails of disease progress curves and positive for the middle part. For the last three cases of Table 2 (curves that did not approach their asymptotes) model 2 provided good fit to data and no systematic trends were observed in the residual plots of model 2.

**Proof of underestimation and faulty rank of \( k \).** Underestimation of \( k \) due to restricting the value of \( A \) to 1 can be demonstrated mathematically. Since \( k \) is the rate of change of a function of disease severity, which is \( \ln(Y/(A-Y)) \) in the case of the logistic function, \( k \) can be expressed as:

\[
k = \frac{\ln(Y_2/(A-Y_2)) - \ln(Y_1/(A-Y_1))}{(t_2 - t_1)},
\]

in which \( Y_1 \) and \( Y_2 \) are disease severities at time \( t_1 \) and \( t_2 \), respectively. If \( A \) is restricted to 1, then the rate parameter \( k \) of the logistic function becomes Vanderplank's apparent infection rate (r) (28), and

\[
r = \frac{\ln(Y_2/(1-Y_2)) - \ln(Y_1/(1-Y_1))}{(t_2 - t_1)}
\]

Underestimation of \( k \) by restricting \( A \) to 1 when in fact \( A < 1 \) can be demonstrated by comparing \( k \) in equation 1 and \( r \) in equation 2. From equations 1 and 2,

\[
k - r = \frac{\ln((A-Y_1)(1-Y_2)/(A-Y_2)(1-Y_1))}{(t_2 - t_1)}.
\]

In equation 3, \( t_2 - t_1 \) is always greater than zero and \( \ln((A-Y_1)(1-Y_2)/(A-Y_2)(1-Y_1)) \) is always greater than zero because the numerator, \( (A-Y_1)(1-Y_2) \), is always greater than the denominator, \( (A-Y_2)(1-Y_1) \). This can be shown as follows: if \( P = (A-Y_1)(1-Y_2) \) and \( Q = (A-Y_2)(1-Y_1) \), then \( P > Q \) if \( A > 1 \), \( P < Q \) if \( A < 1 \), and \( P = Q \) if \( A = 1 \). Since \( A < 1 \), and \( Y_1 < Y_2 \), \( P \) (the numerator) is greater than \( Q \) (the denominator). Therefore, the left side of equation 3 is always greater than zero, which means \( r \) is always smaller than \( k \) if the logistic model with \( A = 1 \) is used when in fact \( A \) is less than 1.

The faulty ranking of \( k \) values due to restricting \( A \) to 1 can be explained graphically. Fig. 4 shows the change in \( \ln(Y/(A-Y)) \) over the infinitely small increment of \( Y \) (disease severity), when \( A = 0.5 \) was arbitrarily chosen for illustration. If the asymptote parameter \( A \) of the logistic function is restricted to 1 when in fact \( A = 0.5 \), the response of \( \ln(Y/(1-Y)) \) to infinitely small increases in \( Y \) is not symmetrical over all values of \( Y \). A small increase in severity results in a great increase in \( \ln(Y/(1-Y)) \) when severity is low, whereas it causes much smaller increase in \( \ln(Y/(1-Y)) \) as severity approaches the maximum severity of 0.5 used in this example. Therefore, \( k \) for slowly developing disease progress which has significantly low severity at the beginning can be faultily higher than that for rapidly developing disease progress that starts with relatively high severity and reaches a higher severity than that for slowly developing disease progress. However, if \( A = 0.5 \) is used in the model, the response of \( \ln(Y/(0.5-Y)) \) to an infinitely small increase in \( Y \) is symmetrical over \( Y \) (Fig. 4) so that \( \ln(Y/(0.5-Y)) \) is affected by high severity as much as by low severity. Therefore, \( k \) can be determined without bias caused by severity.

**The Richards rate parameters.** Table 3 shows two generalized rate parameters that were developed by Richards (19) for asymptotic growth functions. Richards proposed the use of the weighted mean absolute growth rate and the weighted mean relative growth rate of the Richards function for biological interpretation. Growth curves with different values for the shape parameter of the Richards function can be compared without danger that using those two parameters will result in any loss of biological meaning (19, 20). Mathematical derivations of these parameters were shown in detail by Richards (19).

In the context of describing plant disease development, the weighted mean absolute growth rate and the weighted mean relative growth rate will hereafter be called the “absolute rate of disease progress” (denoted by \( Ra \)) and the “relative rate of disease progress” (denoted by \( Rr \)), respectively. \( Ra \) indicates the average rate of increase in disease severity per unit time, whereas \( Rr \) is the average rate of increase in disease severity per unit disease severity per unit time. It is valuable for describing disease development to know the average rate of increase in severity not only in terms of the proportion of the total leaf area (\( Ra \)) but also in terms of the proportion of the diseased leaf area (\( Rr \)). For comparative purposes, \( Rr \) is more useful than \( Ra \) because \( Rr \) accounts for disease severity attained. In addition, \( Rr \) is a generalization of the information conveyed by \( k \) alone when curves with the same value of \( m \), i.e., single-shape curves are compared (19). The \( Ra \) and \( Rr \) values calculated from the true models (model 1) in Table 2 are
Presented in Table 4. In the first case of Table 4, for example, the $Ra$ and $Rr$ values indicate that disease severity increases at the average rate of $5.33\%$ of the total leaf area per unit time, which is an average increase of $20\%$ of already diseased leaf area per unit time.

Values of $k$ versus $Ra$ and $Rr$. The linear form of the Richards function is:

$$-\ln \left\{ 1 - \left( \frac{Y}{A} \right)^{\left( \frac{m}{1-m} \right)} \right\} = B + kt \quad \text{when} \quad 0 < m < 1,$$

$$-\ln \left\{ 1 - \left( \frac{Y}{A} \right)^{\left( \frac{m}{1-m} \right)} \right\} = B + kt \quad \text{when} \quad m > 1.$$

The rate parameter $k$ in these equations expresses the rate of change of a function of $Y$, which is $-\ln \left\{ 1 - \left( \frac{Y}{A} \right)^{\left( \frac{m}{1-m} \right)} \right\}$ or $-\ln \left\{ 1 - \left( \frac{Y}{A} \right)^{\left( \frac{m}{1-m} \right)} \right\}$. Since $A$ and $m$ are included in these functions of $Y$, $k$ is specific for each curve, depending on $A$ and $m$. Therefore, $k$ is not interfunctionally comparable and it is difficult to interpret differences between values of $k$ derived from curves with different values of $A$ and $m$. In contrast, $Ra$ and $Rr$ are interfunctionally comparable because they are determined from the instantaneous absolute growth rate ($dY/dt$) and the instantaneous relative growth rate ($dY/Y$) at time $t$, respectively (19). $Ra$ and $Rr$ are also biologically more meaningful than $k$ because the absolute and the relative growth rates are defined as the absolute and the relative rates at which $Y$ (disease severity) changes, respectively, whereas $k$ is the rate at which a function of $Y$, i.e., $-\ln \left\{ 1 - \left( \frac{Y}{A} \right)^{\left( \frac{m}{1-m} \right)} \right\}$ or $-\ln \left\{ 1 - \left( \frac{Y}{A} \right)^{\left( \frac{m}{1-m} \right)} \right\}$, changes.

**APPLICATION**

Data collection. Bacterial blight epidemics were induced by inoculating soybean cultivars Wells II and Williams 79 in the field at the six-node vegetative stage (V6 stage [9]) and the reproductive stage at which pods 0.5 cm long were formed at one of the four uppermost nodes with a completely unrolled leaf (R3 stage [9]). Inoculation was done by spraying inoculum of *Pseudomonas syringae pv. glycinea* (10^7 colony-forming units per milliliter) at a pressure of 7.2 kg/cm^2 until runoff occurred. The isolate of *Pseudomonas syringae pv. glycinea* used in this study was obtained from a naturally infected Gnome soybean plant in Urbana, IL in 1980. The inoculum was made from a 2-day-old culture on King's B agar plates at 24 °C and turbidometrically adjusted to approximately 10^7 colony-forming units per milliliter of distilled water. Approximately 2.4 and 3.3 L of inoculum was used for each plot at the V6 and the R3 stages, respectively. Naturally occurring bacterial blight was also observed on both cultivars in check plots. The experiment was replicated four times in a split-plot arrangement of a randomized complete block design in which the two cultivars were whole plots and the inoculations and natural infections were subplots. Each subplot consisted of six rows which were 6.1 m long and 76 cm apart. The seeding rate was eight seeds per 30 cm of row.

Disease severity for each of the middle two rows of each plot was rated eight times at 6- to 10-day intervals from the day when bacterial blight symptoms were first observed in the field. A modified Horsfall-Barratt scale (Table 5) was used for visual rating of disease severity. The ratings for each of the middle two rows were converted to proportion of leaf area diseased ($0 \leq Y \leq 1$) and then severity of individual plots was obtained by averaging the converted severities of the middle two rows.

**Comparison of bacterial blight epidemics.** The Richards function was fitted to mean severity values of the four replications. Because maximum severity due to natural infection on Williams 79 was less than 0.5%, it was excluded in the analysis. The NLIN procedure of SAS (21) with the Marquardt's compromise method was used to estimate $A$, $B$, $k$, and $m$ of the Richards function. Initial values of the four parameters from which the iterative procedure of the nonlinear fitting started were obtained by Causton's (7) method. Estimates of the parameters of the Richards function and observed final severity ($Y_i$) for each bacterial blight development are presented in Table 6.

Estimates of $A$ were close to their final severity. The estimates of $m$ indicated that bacterial blight development on plants of Williams 79 that were inoculated at the R3 stage followed a curve between the logit and the Gompertz curves, whereas the other bacterial blight progress curves had a prolonged exponential phase with an abrupt cessation of increase in severity. The highest value for the rate parameter $k$ was obtained from the epidemic which was artificially induced at the V6 stage of Williams 79 and the lowest from the epidemic which was artificially induced at the R3 stage of Williams 79.

**TABLE 4.** The absolute ($Ra$) and the relative ($Rr$) rates of disease progress of true models (Model 1) in Table 2

<table>
<thead>
<tr>
<th>$A$</th>
<th>$B$</th>
<th>$k$</th>
<th>$Ra$</th>
<th>$Rr$</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.8</td>
<td>5</td>
<td>0.4</td>
<td>0.053</td>
<td>0.2</td>
</tr>
<tr>
<td>0.4</td>
<td>5</td>
<td>0.4</td>
<td>0.027</td>
<td>0.2</td>
</tr>
<tr>
<td>0.2</td>
<td>5</td>
<td>0.4</td>
<td>0.013</td>
<td>0.2</td>
</tr>
<tr>
<td>0.8</td>
<td>5</td>
<td>0.1</td>
<td>0.013</td>
<td>0.05</td>
</tr>
<tr>
<td>0.4</td>
<td>5</td>
<td>0.1</td>
<td>0.007</td>
<td>0.05</td>
</tr>
<tr>
<td>0.2</td>
<td>5</td>
<td>0.1</td>
<td>0.003</td>
<td>0.05</td>
</tr>
<tr>
<td>0.8</td>
<td>5</td>
<td>0.05</td>
<td>0.007</td>
<td>0.025</td>
</tr>
<tr>
<td>0.4</td>
<td>5</td>
<td>0.05</td>
<td>0.003</td>
<td>0.025</td>
</tr>
<tr>
<td>0.2</td>
<td>5</td>
<td>0.05</td>
<td>0.002</td>
<td>0.025</td>
</tr>
</tbody>
</table>

*Model 1: $Y = A \left[ 1 + e^{-(B-k)} \right]$.

$Ra$ = severity/(unit time).

$Rr$ = severity/(unit severity)/(unit time).
The Ra and the R (Table 7) indicated that naturally occurring bacterial blight on Wells II developed slowly compared to those which were artificially induced. When a bacterial blight epidemic was initiated at the R3 stage of both cultivars, it developed more rapidly and reached higher severity than when induced at the V6 stage. The Ra values for the epidemics induced at the R3 stage of both cultivars were approximately 2-4 times greater than those for the epidemics induced at the V6 stage. In contrast, there was little difference in the RV values for the artificially induced epidemics. Ra and RV values for epidemics induced on Wells II were greater than those for corresponding epidemics induced on Williams 79.

**DISCUSSION**

Table 2 shows that not only the underestimation of the rate parameter k but also faulty ranking of K-values can occur when the logistic function with A = 1 is fitted to data sets with actual asymptotes differ from each other by less than 1. This is demonstrated mathematically and graphically in this paper. Systematic trends of residuals that result from using models with A = 1 also indicate that empirical estimation of A or correction for A is necessary to obtain appropriate models when disease progress approaches an asymptote less than 1. The faulty ranking of K-values that causes difficulty in biological interpretation can be also found in the literature. For example, Vanderplank’s apparent infection rates for resistant cultivars are supposed to be lower than those for susceptible cultivars (28). However, the apparent infection rates calculated for resistant cultivars were sometimes greater than those for susceptible cultivars (16, 24, 31). This serious drawback of the apparent infection rate is due to restricting A of the logistic function to 1 based on the unrealistic assumption that all host area has become diseased at the end of epidemics. As explained previously in this paper, using the ln[Y/(1 - Y)] transformation based on the unrealistic assumption results in a serious bias in calculating the apparent infection rate. This characteristic of the apparent infection rate (i.e., being affected much more greatly by low severity than by high severity) was also discussed by Shaner and Finney (23). Because of the faulty ranking of the Ra values, the area under the disease progress curve (AUDPC) has been preferred as a disease statistic, especially in slow-rusting and slow-mildewing studies (24, 31) which are conceptually based on the rate of disease development.

Although the logistic function was used in this paper, the same problem due to restricting the asymptote to 1 occurs when other asymptotic growth functions, such as the monomolecular and the Gompertz functions, are used. Also, the same problem can be faced with the Weibull function (30) which differs from the asymptotic biological growth functions. The Weibull function has flexibility of application but it does not include the asymptote parameter so that the asymptote value of the function is fixed at 1 (4-6, 18). Campbell et al. (6) used the Weibull function to characterize the development of snapbean hypocotyl rot disease severity of one snapbean hypocotyl rot progress curve in their paper (Fig. 1A of ref. 6) and they used the asymptotic value of approxiamately 36% at day 40, which indicated that the absolute rate of increase in disease severity became close to zero at day 40. However, the rate curve of the Weibull model (Fig. 1B of ref. 6) indicated the rate was approximately 0.022 at day 40 and that the maximum rate (0.024)

**Table 6. Parameter estimates of the Richards models for bacterial blight development and the final severity (Y) on two soybean cultivars**

<table>
<thead>
<tr>
<th>Cultivar</th>
<th>GS</th>
<th>Y1</th>
<th>Y2</th>
<th>A</th>
<th>B</th>
<th>k</th>
<th>m</th>
<th>R</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wells II</td>
<td>N1</td>
<td>0.034</td>
<td>0.035</td>
<td>108.24</td>
<td>3.93</td>
<td>45.29</td>
<td>0.98</td>
<td></td>
</tr>
<tr>
<td>V6</td>
<td>0.093</td>
<td>0.104</td>
<td>18.23</td>
<td>1.15</td>
<td>7.41</td>
<td>0.94</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R3</td>
<td>0.359</td>
<td>0.355</td>
<td>162.23</td>
<td>4.42</td>
<td>22.03</td>
<td>0.99</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Williams 79</td>
<td>V6</td>
<td>0.095</td>
<td>0.095</td>
<td>174.67</td>
<td>8.26</td>
<td>81.46</td>
<td>0.97</td>
<td></td>
</tr>
<tr>
<td>R3</td>
<td>0.293</td>
<td>0.280</td>
<td>6.81</td>
<td>0.23</td>
<td>1.48</td>
<td>0.95</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Growth stages of soybean plants at which plants were inoculated.

**Table 7. The absolute (Ra) and the relative (Rr) rates of bacterial blight development on two soybean cultivars**

<table>
<thead>
<tr>
<th>Cultivars</th>
<th>GS</th>
<th>Ra</th>
<th>Rr</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wells II</td>
<td>V6</td>
<td>0.005</td>
<td>0.101</td>
</tr>
<tr>
<td>R3</td>
<td>0.013</td>
<td>0.155</td>
<td></td>
</tr>
<tr>
<td>Williams 79</td>
<td>V6</td>
<td>0.005</td>
<td>0.101</td>
</tr>
<tr>
<td>R3</td>
<td>0.013</td>
<td>0.155</td>
<td></td>
</tr>
</tbody>
</table>

* Growth stages of soybean plants at which plants were inoculated.

1. **Ra** = the average increase in severity per day.

2. **Rr** = the average increase in severity per unit severity per day.

3. Natural infection.

The discrepancy between the observed data (Fig. 1A of ref. 6) and the model (Fig. 1B of ref. 6) resulted from fixing the asymptote value of the Weibull function at 1 although the disease progress curve approached 0.36 asymptotically. Thus, for the Weibull function to adequately describe disease progress with asymptotes of less than 1, a new parameter for the asymptote value needs to be included.

Disease progress curves often are still in the increasing phase at the end of the crop growing season or at the time of the last observation (Fig. 3). Data for those disease progress curves do not include information on the location of their asymptotes. Therefore, functions with the asymptote parameter are overparameterized and inadequate for the data (8). In Table 2, model 2 for the curves with a true r = 0.05 (i.e., curves from Fig. 3) has extremely high coefficients of determination (R2) and estimates of B and k are close to their true values. This indicates that the function without the asymptote parameter (i.e., A is fixed at 1) would be appropriate for disease progress curves lacking asymptotes.

Widely different values of the shape parameter m for five bacterial blight development curves suggest that there is no specific growth function that is particularly well suited for soybean bacterial blight. Because m can have different values, estimates of the parameter B for the five bacterial blight development curves cannot be compared. However, when disease progresses with the same value of m (i.e., have the same shape of disease progress curves) are compared, B indicates the time when diseases start to develop rapidly. Also, B is necessary to calculate a period of time delay to reach certain levels of severity. The time delay is important in conjunction with the concept of sanitation (28, 32).

Estimates of the asymptote parameter A can be interpreted as the magnitude of susceptibility or resistance of a host to a pathogen under certain environmental conditions. Higher A values for bacterial blight development on Wells II than for corresponding disease progress on Williams 79 indicate that Williams 79 is more susceptible than Williams 79. When A values were compared between growth stages at which plants were inoculated, higher A values were reached when plants were inoculated at the R3 stage than at the V6 stage of both cultivars. This is probably due to greater amount of inoculum applied at the R3 stage than at the V6 stage and smaller increase in the total leaf area after inoculation at the R3 stage than at the V6 stage. Since estimates of m and A for five bacterial blight progress curves are each different, the rate parameter k is specific for each progress curve and, therefore, is not comparable.

The Ra and Rr values also reflect the effects of the difference in the amount of initial inoculum applied and increase in the total leaf area after inoculation. Because of high initial severity and relatively small increases in the total leaf area after inoculation at the R3 stage, the absolute increment of severity per day (Ra) was high on both cultivars. Low initial severity and large increase in the total leaf area after inoculation at the V6 stage of both cultivars resulted in small absolute increment of severity per day. However, when the rate of increase in severity was adjusted so as to take account of severity already attained, the rates of increase in severity per unit severity per day (Rr) were not greatly different between bacterial blight development induced at the V6 and the R3 stages of both cultivars. This suggests that there may be no great difference in
susceptibility of both cultivars to bacterial blight between the two growth stages. Slow development of natural infection compared to artificially induced bacterial blight development is probably due to differences in the amount of inoculum and genetic variation between naturally occurring and inoculated populations of *Pseudomonas syringae* pv. *glycinea*.

We propose the use of the weighted mean absolute growth rate and the weighted mean relative growth rate of the Richards function (19) as the $R_a$ and $R_r$, respectively, for describing and comparing epidemics with different asymptotes and shapes of disease progress curves. Since they are not only biologically meaningful but also interfunctionally comparable, they are more useful for describing and comparing epidemics with different asymptotes and shapes of disease progress curves than Vanderplank's apparent infection rate or the rate parameter $k$ of growth functions with the asymptote value of 1. The $R_a$ and $R_r$ are descriptive parameters because they are obtained by empirically fitting the Richards function to disease progress data. Further studies are needed to relate these parameters to components of epidemiological processes such as latent period, infection efficiency, and infectious period.

**LITERATURE CITED**