Comparative Analysis of Flexible Two-Parameter Models of Plant Disease Epidemics

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ABSTRACT

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Eleven previously published models of plant disease epidemics, given as differential equations with a rate and a shape parameter, are compared using general model characteristics as well as their usefulness in fitting observed data. Six out of the eleven models can be solved analytically resulting in epidemic growth functions, while the others can be solved only numerically. When all 11 differential equations were fitted to two data sets, all models showed a similar goodness of fit, although the shape parameter in some models could not be estimated very precisely. With respect to useful characteristics (exponential population growth at the

beginning, ability to generate monomolecular disease progression, and flexibility of the inflection point), the models of Fleming, Kosman-Levy, Birch, Richards and Waggoner, and Rich are recommended. Formulas were established to calculate the point of inflection as well as the weighted absolute and relative rate, respectively, depending on the shape and rate parameter. These formulas allow transformation of the parameter values of one model into those of another model in many cases. If the two models are required to have the same temporal position of the disease progress curve, then the initial disease level at the start of the epidemic or the time when the inflection point is reached have to be transformed.

Additional keywords: disease progress curves, growth curves, nonlinear regression.

In 1963, Vanderplank (23) introduced simple models to describe plant disease epidemics. The graph of an epidemic is the disease progress curve, which, for instance, represents the disease severity (y) over time (t). For a mathematical description, simple differential models with only one parameter, a rate parameter ($r_s > 0$), can be used. As proposed by Vanderplank (23), the exponential (equation 1), monomolecular (equation 2), and logistic (equation 3) models are useful:

$$\frac{dy}{dt} = r_E y \quad (1)$$

$$\frac{dy}{dt} = r_M (1 - y) \quad (2)$$

$$\frac{dy}{dt} = r_L y(1 - y) \quad (3)$$

The subscript on r indicates the specific model. Later, Analytis (1) and Berger (2) added the Gompertz model (equation 4), which is also a one-parameter model:

$$\frac{dy}{dt} = r_G y (-\ln y) \quad (4)$$

For the solution of differential equations 1 to 4, the initial condition has to be specified, for instance, by setting $y(t=0) = y_0$, the initial proportion of plant tissue area diseased. The integrated forms of the differential equations are monotonically increasing growth functions, which contain two parameters; r_s and a constant of integration reflecting initial disease. Therefore, some

authors, for instance Rouse (19), have classified these models as two-parameter models.

To obtain more flexibility in analyzing disease progress curves, differential equations with an additional shape parameter have been put forward, for instance, the model of Richards (12,18):

$$\frac{dy}{dt} = \frac{r_R y (1 - y^{m-1})}{m - 1} \quad (5)$$

where $m \geq 0$ is a unit-less shape parameter. Analytis (1) had already used some special cases for m , for instance, $m = 1/2$ and $m = 2/3$ (i.e., the von Bertalanffy [24] models). The Richards growth function, resulting from the integration of equation 5, contains three parameters and was termed a three-parameter model by Rouse (19).

In this paper, the number of parameters of the differential equation is considered and, accordingly, the Richards model is termed two-parameter because only a rate and a shape parameter appear in the differential equation.

The Richards model (equation 5) was originally developed to describe plant growth, and other two-parameter models have been proposed from the kinetic theory of growth (22), which can also be applied to plant disease epidemics. On the other hand, some two-parameter models have been established specifically for plant disease epidemics, for instance, to account for the effects of spatial distribution (27), or to improve biological realism by introducing additional features of the pathogen life cycle, for instance, the latent period (5,6).

In this paper, different two-parameter models suggested to describe plant disease epidemics are compared to determine what their properties are in the general sense and to know if (or how) the models are related. Only those models were chosen that could be: (i) written as a single equation (not coupled differential equations); and (ii) written as simple scaled power functions of y and $1 - y$. The theory behind some of the models is not evaluated

or questioned. To standardize the models and to ease the comparison of the differential equations, the rate parameter is always designated by r_* and the shape parameter by s_* , both marked with an index reflecting the respective author(s) or the name of the model. In a formal way, the differential equation can then be written as

$$\frac{dy}{dt} = f(y, r_*, s_*) \tag{6}$$

For all models, it is assumed that the maximum disease level is 1, corresponding to a disease severity or incidence of 100%. The maximum value, usually the carrying capacity (K), is not taken into consideration as an additional parameter. Park and Lim (16) and Neher and Campbell (14) have investigated the effects of this parameter K on the estimation of the other parameters, especially the rate parameter.

THEORY AND APPROACHES

Models compared. Altogether, 11 models given as differential equations are included in the comparison (Table 1). The disease progress curves described by the models are sigmoidal, but not necessarily symmetrical.

The first model is Richards model (equation 7) with a shape parameter ($s_R = m$) nonnegative and not equal to 1.

Turner et al. (22) developed a generic growth model from which two special cases are also two-parametric, the hyperlogistic (equation 8) and the hyper-Gompertz (equation 9) model. According to the assumptions of Turner et al. (22), the condition for the shape parameter of the hyperlogistic model is $-1 \leq s_{HL} \leq 1$. In the hyperlogistic model (equation 8), exponents are introduced to the increasing (y) as well as to the decreasing $1 - y$ term of the logistic equation 3, but the hyper-Gompertz model (equation 9) results from equation 4 by introducing an exponent only for the decreas-

ing $-\ln(y)$ term. The shape parameter s_{HG} in equation 9 cannot be less than -1 .

Waggoner and Rich (27) tried to take into account the effect of the spatial distribution of disease on disease progression by introducing the aggregation parameter k of the negative binomial distribution into the logistic equation. Regarding $k > 0$ as shape parameter s_{WR} , their model can be written as equation 10 (Table 1).

Considering the spatial distribution of disease, Waggoner (25) as well as Waggoner and Rich (27) proposed a second equation that is an extension of the logistic equation 3 by taking the term $1 - y$ to a power greater than 1. Originally the exponent was $1 + 1/k$ in which k was the aggregation parameter of the negative binomial distribution. Taking again $k > 0$ as a shape parameter, which is now designated as $s_{Wa} > 0$, results in equation 11 (Table 1).

Yang and TeBeest (29) extended model 11 by assuming that k is proportional to the disease severity y . Regarding the proportionality factor as shape parameter $s_{YT} > 0$, this model can be written as equation 12 (Table 1).

The Weibull function (28) was also proposed to describe disease progress curves (12,17,21). In its integrated form, it is a three-parameter growth function usually given as

$$y(t) = 1 - \exp \left[- \left(\frac{t-a}{b} \right)^c \right]$$

Following Madden (12), the differential equation for this function can be written as equation 13 (Table 1) in which r_{We} corresponds to $1/b$ and s_{We} to c .

Jeger (10) used the log-logistic function, originally proposed by Hill (9), for the description of plant disease progress curves. In its integrated form, it can be written as

$$y(t) = \frac{1}{1 + \left(\frac{t-a}{b} \right)^{-c}} = \frac{1}{1 + \left(\frac{b}{t-a} \right)^c}$$

TABLE 1. Differential equations of the 11 models compared and the respective author(s)

Author(s)	Model	Differential equation	Range of the shape parameter	
			Original	Extension
Richards (18)	7	$\frac{dy}{dt} = \frac{r_R y (1 - y^{(s_R - 1)})}{s_R - 1}$	$s_R \geq 0$, $s_R \neq 1$	No
Turner et al. (22)	8	$\frac{dy}{dt} = r_{HL} y^{(1 - s_{HL})} (1 - y)^{(1 + s_{HL})}$	$-1 \leq s_{HL} \leq 1$	No
Turner et al. (22)	9	$\frac{dy}{dt} = r_{HG} y [-\ln(y)]^{(1 + s_{HG})}$	$s_{HG} \geq -1$	No
Waggoner and Rich (27)	10	$\frac{dy}{dt} = r_{WR} s_{WR} \left[1 - (1 - y)^{1/s_{WR}} \right] (1 - y)$	$s_{WR} > 0$	$s_{WR} \leq -1$
Waggoner (25)	11	$\frac{dy}{dt} = r_{Wa} y (1 - y)^{(1 + 1/s_{Wa})}$	$s_{Wa} > 0$	$s_{Wa} \leq -1$
Yang and TeBeest (29)	12	$\frac{dy}{dt} = r_{YT} y (1 - y)^{(1 + 1/(s_{YT} y))}$	$s_{YT} > 0$	$s_{YT} \leq -1$
Weibull (28)	13	$\frac{dy}{dt} = r_{We} s_{We} [-\ln(1 - y)]^{(1 - 1/s_{We})} (1 - y)$	$s_{We} \geq 1$	No
Hill (9) and Jeger (10)	14	$\frac{dy}{dt} = r_{LL} s_{LL} y^{(1 - 1/s_{LL})} (1 - y)^{(1 + 1/s_{LL})}$	$s_{LL} \geq 1$	No
Fleming (5)	15	$\frac{dy}{dt} = \frac{r_F y (1 - y)}{1 + r_F s_F y}$	$s_F \geq 0$	$-\frac{1}{r_F} \leq s_F < 0$
Kosman and Levy (11)	16	$\frac{dy}{dt} = \frac{r_{KL} (s_{KL} + 1) y (1 - y)}{s_{KL} + 1 - y}$	$s_{KL} \geq 0$	$s_{KL} \leq -1$
Birch (3)	17	$\frac{dy}{dt} = \frac{r_B y (1 - y)}{1 + y (s_B - 1)}$	$s_B \geq 0$	No

The differential equation for this function can be regarded as a logistic function with a time dependent rate decreasing hyperbolically. It is, however, possible to replace the time dependence and to write the differential equation as equation 14 (Table 1) with r_{LL} equal to $1/b$ and s_{LL} equal to c (see appendix).

Fleming and Holling (6) and Fleming (5) deduced another two-parameter model with the rate r_F and a shape parameter which was explained as mean length of the latent period. Although Fleming (5) claimed his model "as the best available explanatory model," it has been neglected in epidemiology. Designating the latent period as shape parameter $s_F \geq 0$ leads to equation 15 (Table 1).

Kosman and Levy (11) proposed two coupled differential equations for the proportion of lesion area and the proportion of infectious tissue. If the transition rate from infectious to post-infectious lesions is set to 0, the Kosman-Levy model can be simplified to equation 16 (Table 1) for an epidemic without removals. Kosman and Levy (11) considered only nonnegative values of the shape parameter s_{KL} , which was interpreted as the rate of lesion increase per unit of infectious area.

The last model included in the comparison was recently proposed by Birch (3) who developed a generalized logistic growth curve with a shape parameter. With maximum y equal to 1, this model can be written as equation 17 (Table 1). Although this model has never been applied in plant disease epidemiology, it is of interest as it always tends to exponential growth at low severities, unlike the Richards model (equation 7), which has this behavior in only part of the range of its shape parameter (described below).

The unit of the rate parameters is always 1 per day, whereas the shape parameters are unit-less, except for the Fleming model (equation 15) in which the shape parameter is measured in days. The values of the rate parameters of all models are positive numbers, while the range of the shape parameter values varies (Table 1). Formally, an extension of the original range of shape parameters is possible for some models (Table 1) if the following criteria are fulfilled: (i) $dy/dt > 0$ for $0 < y < 1$; (ii) dy/dt tends to 0 when y approaches 1, and (iii) dy/dt tends to a finite nonnegative value when y approaches 0. Negative values of the shape parameters less than or equal to -1 can be considered for equations 10 to 12 and 16. The shape parameter s_F of the Fleming model (equation 15) may also vary between $-1/r_F$ and 0. However, with these extensions of the range, the biological interpretation of the shape parameters, for instance as the latent period concept in equation 15, will be lost.

Criteria used to compare the models. The comparison of the 11 models is based on the following seven criteria.

Existence of an analytical solution. The models are given as differential equations, which are difficult to handle when the models are applied to actual data. It is more convenient to use the integrated form, i.e., the three-parametric growth function, in a nonlinear regression analysis to fit the models to the data. To derive an analytical solution of a model, the corresponding initial-value problem has to be solved: the differential equation has to be integrated and then adapted to the initial condition $y(t_0) = y_0$ (usually $t_0 = 0$). However, the integration may not be possible and thus no analytical solution may exist. In this case, numerical integration methods have to be applied to fit the models to observed data.

Relation to one-parametric models. Under this criterion, it is evaluated whether the two-parametric models can be reduced to the simple one-parametric models (equations 1 to 4) by fixing the shape parameter to certain values.

Point of inflection. The inflection point of an S-shaped disease progress curve is defined as the point of maximal absolute rate of disease increase. It can be determined by calculating the first derivative of the right side of the differential equations with respect to y and by setting it equal to 0. To gain high flexibility in

describing disease progress curves, the disease level at the point of inflection should be responsive to the value of the shape parameter over a wide range of values.

Behavior at the beginning of an epidemic. The possibility of simplifying the models at low disease severities is investigated. For this purpose, the first two terms of the Taylor power series of the right side of equation 6, around $y = 0$, are calculated leading to

$$\frac{dy}{dt} \approx f(0, r_*, s_*) + f'(0, r_*, s_*) \cdot y \quad (18)$$

Thus, if $f(1, r_*, s_*) = 0$, the disease progress curve will follow an exponential curve for low y values with a rate equal to $f'(0, r_*, s_*)$, the derivative $f'(\bullet)$ with respect to y .

Asymptotic behavior. Similar to the previous criterion, the behavior at the end of an epidemic in the asymptotic phase is investigated. Again, the first two terms of the power series of equation 6, but around $y = 1$, are formed resulting in

$$\frac{dy}{dt} \approx f(1, r_*, s_*) - f'(1, r_*, s_*) \cdot (1 - y) \quad (19)$$

For $f(0, r_*, s_*)$ and $f'(1, r_*, s_*) < 0$, a monomolecular function with rate $-f'(1, r_*, s_*)$ describes the asymptotic behavior.

Weighted mean absolute rate (WMAR). WMAR is defined as the area under the curve of absolute rate of disease increase, dy/dt , in the interval from 0 to 1, so that it is given by the integral

$$WMAR = \int_0^1 \left(\frac{dy}{dt} \right) dy \quad (20)$$

This rate has been suggested for comparing rates of different growth functions, especially for Richards' functions with different shape parameters (4).

Weighted mean relative rate (WMRR). WMRR is given by the area under the relative rate of disease increase,

$$\frac{1}{y} \left(\frac{dy}{dt} \right)$$

in the interval from 0 to 1. Again, this can be determined by the following integral

$$WMRR = \int_0^1 \frac{1}{y} \left(\frac{dy}{dt} \right) dy \quad (21)$$

RESULTS

Comparison of models. The 11 models are compared based on the seven criteria.

Analytical solution. Out of the 11 models, only six can be solved analytically, while for the rest no analytical solutions are known. Richards (18) has already shown that his model (equation 7) can be solved analytically leading to the following growth function:

$$y(t) = \left[1 - A e^{-r_R t} \right]^{1/(1-s_R)}, \quad \text{where } A = 1 - y_0^{(1-s_R)} \text{ for } s_R \neq 1 \quad (22)$$

When the shape parameter s_R tends to 1, the Richards model turns into the Gompertz model (equation 4) with the following solution

$$y(t) = \exp \left(-A e^{-r_G t} \right), \quad \text{where } A = -\ln y_0$$

Turner et al. (22) provided a solution for the hyperlogistic model (equation 8), which can be written in the following way:

$$y(t) = \frac{1}{1 + (A + r_{HL} s_{HL} t)^{-1/s_{HL}}}, \quad \text{where } A = \left(\frac{y_0}{1 - y_0} \right)^{s_{HL}} \quad (23)$$

The hyper-Gompertz model (equation 9) can also be solved analytically (22):

$$y(t) = \exp\left[-(A + r_{HG}s_{HG}t)^{-1/s_{HG}}\right], \text{ where } A = \frac{1}{(-\ln y_0)^{s_{HG}}} \quad (24)$$

In contradiction to the statement of Campbell and Madden (4), the model of Waggoner and Rich (equation 10) can be integrated analytically:

$$y(t) = 1 - \left[1 + A e^{r_{WR}t}\right]^{-s_{WR}}, \text{ where } A = (1 - y_0)^{(-1/s_{WR})} - 1 \quad (25)$$

As mentioned previously, the Weibull model (equation 13) has the analytical solution

$$y(t) = 1 - \exp\left[-(A + r_{We}t)^{s_{We}}\right], \text{ where } A = [-\ln(1 - y_0)]^{1/s_{We}} \quad (26)$$

Also, the initial-value problem can be solved for the log-logistic model (equation 14):

$$y(t) = \frac{1}{1 + (A + r_{LL}t)^{-s_{LL}}}, \text{ where } A = \left(\frac{y_0}{1 - y_0}\right)^{1/s_{LL}} \quad (27)$$

For the five other models (equations 11, 12, and 15 to 17), no analytical solutions for the disease severity (y) as a function of time (t) are known so that numerical methods are needed to fit the models to real data. Suitable software for the fitting of differential equations to observed data are available, for instance Model-Maker 4 (ModelKinetix, Wallingford). An analytical solution of differential equations 15 to 17 is possible, but is not expressible with $y(t)$ as a separate term. However, time (t) can be expressed as a unique function of the disease severity $y(t)$:

$$t = \frac{1}{r_F} \cdot \ln \left[A \cdot \frac{y(t)}{(1 - y(t))^{(1+r_F s_F)}} \right], \text{ where } A = \frac{(1 - y_0)^{(1+r_F s_F)}}{y_0} \quad (28)$$

for the Fleming model (equation 15);

$$t = \frac{1}{r_{KL}} \cdot \ln \left[A \cdot \frac{y(t)}{(1 - y(t))^{(s_{KL}/(1+s_{KL}))}} \right], \text{ where } A = \frac{(1 - y_0)^{(s_{KL}/(1+s_{KL}))}}{y_0} \quad (29)$$

for the Kosman-Levy model (equation 16); and

$$t = \frac{1}{r_B} \cdot \ln \left[A \cdot \frac{y(t)}{(1 - y(t))^{s_B}} \right], \text{ where } A = \frac{(1 - y_0)^{s_B}}{y_0} \quad (30)$$

for the Birch model (equation 17). These formulas allow an estimation of the time (t), needed to reach a certain level of disease y from y_0 .

Relation to one-parameter models. Richards (18) has already shown that his model (equation 7) can be reduced to the monomolecular, the Gompertz, and the logistic model when the shape parameter takes certain values (0, 1 [limit], and 2, respectively). Similarly, the other two-parameter models can be simplified to one-parameter models for special values of the shape parameter (Table 2).

The comparison shows that only the Richards model (equation 7) contains all simple asymptotic models as special cases, while the other functions can be reduced only partly to one-parameter models. Models 8, 10, 15, and 17 are reduced to the logistic model for fixed values of the shape parameter (Table 2). The logistic model is also the limit function for infinitely high values of shape parameters for the models 11, 12, and 16. When the shape parameter of equation 10 approaches infinity, a limit function also exists and is given by the equation

$$\frac{dy}{dt} = r_{WR} [-\ln(1 - y)] \cdot (1 - y)$$

Compared to the logistic model (equation 3), the driving force is then given by $[-\ln(1 - y)]$ instead of y . This term is also included as increasing force in the Weibull model (equation 13).

Point of inflection. The advantage of the two-parametric models is their flexibility with respect to the disease level at which the inflection point occurs. This level (y_{ip}) with the highest absolute rate of disease increase is a function only of the shape parameter for all models (Table 3), except for Fleming's model (equation 15) for which the product of the rate and shape parameters determines y_{ip} . No closed solution is possible for Yang and TeBeest's model (equation 12), but the point of inflection can be calculated numerically from the equation

$$s_{YT} = \frac{1}{1 - 2y_{ip}} \cdot \left[1 + \frac{(1 - y_{ip}) \ln(1 - y_{ip})}{y_{ip}} \right]$$

The relations between the disease level at the inflection point and the respective shape parameters are shown in Figure 1 (dashed lines are for the extended ranges of the shape parameters in Table 1). The rate parameter of Fleming's model (equation 15), which also affects y_{ip} , was set to 0.2 per day.

The point of inflection can occur at all disease levels between 0 and 1 for models 7, 8, 9, and 17. In all other cases, the disease level at the point of inflection is restricted to certain values: y_{ip} is less than 0.5 for the models 11, 12, 14, and 15, and less than $1 - 1/e \approx 0.632$ for models 10 and 13; for model 16, only disease values above 0.5 can occur at the point of inflection (Fig. 1). However, for models 10 to 12, 15, and 16, disease levels at the inflection point between 0 and 1 can be achieved by extending the range of the shape parameter values (Table 1, Fig. 1). On the other hand, the relationship between inflection point and shape parameter is of different form for the extended negative and the original positive s_* values, with a discontinuity in the middle (models 10 to 12 and 16), which can cause problems in curve fitting.

The time needed to reach the inflection point (t_{ip}) depends on the rate and the shape parameters as well as on the initial disease level (y_0) and is given in Table 3, but only for those models for which the corresponding differential equation can be analytically integrated (see item 1, equations 22 to 30). The time t_{ip} is inversely related to the rate parameters (r_*), while the effects of the shape parameters (s_*) and of the initial disease value (y_0) are more complicated. Knowing y_0 , the time t_{ip} can be determined

TABLE 2. Special cases of 11 two-parametric models for fixed shape parameter values

Model number	Special cases for certain shape parameter values		
7	$s_R = 0$	Monomolecular model	Equation 2
	$s_R \rightarrow 1$	Gompertz model	Equation 4
	$s_R = 2$	Logistic model	Equation 3
8	$s_{HL} = 0$	Logistic model	Equation 3
9	$s_{HG} = 0$	Gompertz model	Equation 4
10	$s_{WR} = 1$	Logistic model	Equation 3
11	$s_{Wa} \rightarrow +\infty$	Logistic model	Equation 3
12	$s_{YT} \rightarrow +\infty$	Logistic model	Equation 3
13	$s_{We} = 1$	Monomolecular model	Equation 2
14		No special cases	
15	$s_F = 0$	Logistic model	Equation 3
	$s_F = -1/r_F$	Exponential model	Equation 1
16	$s_{KL} = 0$	Exponential model	Equation 1
	$s_{KL} \rightarrow +\infty$	Logistic model	Equation 3
17	$s_B = 1$	Logistic model	Equation 3
	$s_B = 0$	Exponential model	Equation 1

also for the models 11 and 12 by numerically integrating the differential equations up to the time when the disease level is equal to y_{ip} . One should be aware that the calculated values of time t_{ip} will be negative if $y_0 > y_{ip}$, i.e., if disease observations started when the maximum value of the absolute rate of disease progress was already reached.

Behavior at the beginning of an epidemic. At the beginning of an epidemic, the right side of differential equations can be simplified by equation 18. For models 7, 10 to 12, and 15 to 17, the initial phase of the disease progress curve corresponds to the exponential growth (equation 1) for a wide range of shape parameters (Table 4). With two exceptions, the rate parameters for the exponential part of disease development are identical to those of the corresponding models describing the whole epidemic. In the two exceptions (models 7 and 12), the exponential rate is also influenced by the respective shape parameter. When the shape parameter s_R of Richards's function (equation 7) approaches 1, the corresponding rate will infinitely increase. Therefore, the Gompertz function, which is the simplified function for $s_R \rightarrow 1$, does not provide an exponential type of disease progress curve for a low disease level. For equation 12, the resulting exponential rate cannot be calculated directly but only by using the limit values when y tends to 0 in equation 18. For the hyperlogistic model (equation 8), the exponential growth can be achieved only in the special case for $s_{HL} = 0$, corresponding to the logistic model. All other models never describe disease progress as exponential outbreak when an epidemic starts.

Asymptotic behavior of the epidemic. In the asymptotic phase, equation 19 can approximate the right side of the differential equations, eventually leading to the monomolecular model (equation 2) (Table 4). Models 7, 10, and 15 to 17 can be simplified to a monomolecular model for a wide range of shape parameter values. Except for model 7, the corresponding rates in the asymptotic phase depend also on the respective shape parameters. For models 11, 12, and 14 the disease development for high y values can never be described by a monomolecular function. Only for some special values of shape parameters for models 8, 9, and 13, the form of disease approaching the saturation is monomolecular.

WMAR. For all models, the WMAR, defined by equation 20, depends on both parameters (Table 5). However, except for equation 15, the rate parameter, r_* , is just a multiplication factor to a more complicated term describing the effect of the shape parameter, s_* . For Fleming's model (equation 15), the effects of both parameters cannot be separated. The WMAR-values for the models 8, 9, 13, and 14 can be calculated by using the Gamma-function (Γ). For equation 12, however, no closed solution is known but numerical integration methods can be applied to calculate the WMAR if the actual values for the rate and the shape parameters are given.

The relations between WMAR and the respective shape parameters, s_* , for a rate parameter of $r_* = 0.2$ per day are shown for the 11 models in Figure 2. For two of the models (equations 8 and 9), the effect of the shape parameter on WMAR is given by non-

TABLE 3. Disease level at the inflection point (y_{ip}) and time to reach the inflection point (t_{ip}) as a function of the rate parameter (r_*) and the shape parameter (s_*), respectively, for the 11 models

Model	y_{ip}	t_{ip}
7	$\frac{1}{s_R(1-s_R)}$	$\frac{1}{r_R} \cdot \ln \left(\frac{1-y_0^{(1-s_R)}}{1-s_R} \right)$
8	$\frac{1-s_{HL}}{2}$	$\frac{1}{s_{HL}r_{HL}} \cdot \left[\left(\frac{1-s_{HL}}{1+s_{HL}} \right)^{s_{HL}} - \left(\frac{y_0}{1-y_0} \right)^{s_{HL}} \right]$
9	$\exp[-(1+s_{HG})]$	$\frac{1}{s_{HG}r_{HG}} \cdot \left[(1+s_{HG})^{-s_{HG}} - (-\ln y_0)^{-s_{HG}} \right]$
10	$1 - \left(\frac{s_{WR}}{s_{WR}+1} \right)^{s_{WR}}$	$-\frac{1}{r_{WR}} \cdot \ln \left\{ s_{WR} \left[(1-y_0)^{(-1/s_{WR})} - 1 \right] \right\}$
11	$\frac{s_{Wa}}{1+2s_{Wa}}$	No closed solution known
12	Numerical solution	No closed solution known
13	$1 - \exp \left(\frac{1-s_{We}}{s_{We}} \right)$	$\frac{1}{r_{We}} \cdot \left[\left(\frac{s_{We}-1}{s_{We}} \right)^{1/s_{We}} - (-\ln[1-y_0])^{1/s_{We}} \right]$
14	$\frac{s_{LL}-1}{2s_{LL}}$	$\frac{1}{r_{LL}} \cdot \left[\left(\frac{s_{LL}-1}{s_{LL}+1} \right)^{1/s_{LL}} - \left(\frac{y_0}{1-y_0} \right)^{1/s_{LL}} \right]$
15	$\frac{1}{1+\sqrt{1+r_F s_F}}$	$\frac{1}{r_F} \cdot \left[\ln \frac{y_{ip}}{(1-y_{ip})(1+r_F s_F)} - \ln \frac{y_0}{(1-y_0)(1+r_F s_F)} \right]$
16	$s_{KL} + 1 \mp \sqrt{s_{KL}^2 + s_{KL}}$ if $s_{KL} \geq 0$, + if $s_{KL} \leq -1$	$\frac{1}{r_{KL}} \cdot \left[\ln \frac{y_{ip}}{(1-y_{ip})^{(s_{KL}/(s_{KL}+1))}} - \ln \frac{y_0}{(1-y_0)^{(s_{KL}/(s_{KL}+1))}} \right]$
17	$\frac{1}{1+\sqrt{s_B}}$	$\frac{1}{r_B} \cdot \left[\ln \frac{y_{ip}}{(1-y_{ip})^{s_B}} - \ln \frac{y_0}{(1-y_0)^{s_B}} \right]$

monotonous functions with minima at $s_{HL} \approx 0$ and $s_{HG} \approx 0.48$, respectively. Therefore, the same *WMAR*-value can result from two different values of the corresponding shape parameter even when the rate parameter is the same. Similarly, *WMAR* for equations 13 and 14 also reaches a minimum value (minima at

$s_W \approx 1.11$ and $s_{LL} \approx 1.19$), but this is difficult to see in Figure 2. On the other hand, *WMAR* increases nearly linearly for these two models for shape parameter values above 2 ($WMAR \approx r_{We}s_{We}/4$ for equation 13 and $WMAR \approx r_{LL}s_{LL}/6$ for equation 14). For all other models (equations 7, 10 to 12, and 15 to 17), *WMAR* is an

Fig. 1. Relationships between the disease level at the point of inflection (y_{ip}) and the shape parameters s_* of models 7 to 17 (Table 1). The dashed lines are valid for the extended ranges of the shape parameters. The dotted lines represent asymptotes of the relationships.

injective function of s_* (for a fixed rate parameter r_*), i.e., any given $WMAR$ -value is related to a unique shape parameter value.

Weighted mean relative rate. The weighted mean relative rate $WMRR$, defined by equation 21, can be calculated with less complicated formulae (Table 5) than the $WMAR$. Again, $WMRR$ depends on both parameters (Table 5), but the rate parameter, r_* , is just a multiplication factor except for Fleming's model (equation 15) in which the effects of both parameters cannot be separated. For models 8, 9, and 14, $WMRR$ can be determined by means of a Γ -function (Γ). For models 10, 12, and 13, numerical integration methods are needed to calculate $WMRR$.

In Figure 3, the functions of $WMRR$ depending on the shape parameters, s_* , are displayed for all models with $r_* = 0.2$ per day. The graphs describing $WMRR$ in relation to the shape parameters are similar to those of $WMAR$ (Fig. 2) implying analogous properties. Any given $WMRR$ -value results from a unique shape parameter value for models 7, 10 to 12, and 15 to 17. $WMRR$ of model 14 increases linearly ($\approx r_{LL}(1 + s_{LL})/2$) when s_{LL} is greater than 3. However, for models 13 and 14, obviously two different values of the shape parameters can have the same $WMRR$ -value (minima at $s_{We} \approx 2.19$ and $s_{LL} \approx 2.32$). Also, for models 8 and 9, $WMRR$ reaches a flat minimum for $s_{HL} \approx -0.5$ and $s_{HG} \approx -0.54$, respectively, although this behavior is difficult to see in Figure 3.

Examples for fitting the models to observed data. The 11 models were fitted to disease progress data of potato late blight observed on two cultivars, Monona and Kennebec. These data sets, which were provided by W. E. Fry, include eight observations with four replications at each assessment for both cultivars. Although Campbell and Madden (4) fitted growth curves with linear or nonlinear regression analyses to the same data in their

temporal analyses of disease progression, we directly fitted the differential equations to the data using ModelMaker 4 (Model-Kinetix) with the ordinary least squares approach.

For cv. Monona, all models could be fitted very well to the data, resulting in coefficients of determinations between 99.59 to 99.66%, which corresponded to sums of squares of the residuals (SSE) of 0.0166 to 0.0200 (Table 6). The fitted curves of the best model (Weibull model, equation 13) and the worst model (Yang and TeBeest, equation 12) are displayed in Figure 4A showing only slight differences. The values of the rate parameter varied from 0.014 for equation 14 to 0.436 for equation 15. The rate parameter values of equations 15 to 17 were practically identical. The calculated values of the rate parameter r_E at the beginning of the epidemic ranged from 0.406 to 0.484 per day for models 7, 10, 11, and 15 to 17 (Table 6). On the other hand, the variability of the values of the shape parameter was quite high. The values for models 9 and 16 were negative ($s_{HG} = -0.251$ and $s_{KL} = -6.315$), whereas for models 11 and 12, the estimated values were quite large ($s_{YT} = 50.80$ and $s_{Wa} = 52.16$). The value of Richards model (equation 7) ($s_R = 1.757$) indicates that the observed disease progression was close to logistic type for which the shape parameter is 2. This closeness to the logistic growth is also reflected in the shape parameter values of some other models, for instance, close to 0 for model 8 ($s_{HL} = 0.033$), and close to 1 for models 10 and 17 ($s_{WR} = 0.877$ and $s_B = 1.182$). The extremely high shape parameter values of models 11 and 12 were due to the similarity of these models to the logistic model when the shape parameters approach infinity. Obviously, the estimation of the shape parameter for the latter models is difficult in this situation, which is reflected in the fact that the standard errors of the

TABLE 4. Disease progress at the beginning (y near 0) and at the end (y near 1) of an epidemic according to equations 18 and 19, respectively, for the 11 models

Model	Rate parameter r_E of the exponential model 1 at the beginning		Rate parameter r_M of the monomolecular model 2 at the end	
7	$r_E = \frac{r_R}{s_R - 1}$	for $s_R > 1$	$r_M = r_R$	for $s_R \geq 0, s_R \neq 1$
	Not existing	for $0 \leq s_R < 1$		
8	$r_E = 0$	for $-1 < s_{HL} < 0$	Not existing	for $-1 < s_{HL} < 0$
	$r_E = r_{HL}$	for $s_{HL} = 0$		
	Not existing	for $0 < s_{HL} < 1$		
9	Not existing	for $s_{HG} \geq -1$	Not existing	for $-1 < s_{HG} < 0$
10	$r_E = r_{WR}$	for $s_{WR} > 0, s_{WR} \leq -1$	$r_M = r_{WR} s_{WR}$	for $s_{WR} > 0$
			Not existing	for $s_{WR} \leq -1$
11	$r_E = r_{Wa}$	for $s_{Wa} > 0, s_{Wa} \leq -1$	$r_M = 0$	for $s_{Wa} > 0$
			Not existing	for $s_{Wa} \leq -1$
12	$r_E = r_{YT} e^{-\frac{1}{s_{YT}}}$	for $s_{YT} > 0, s_{YT} \leq -1$	$r_M = 0$	for $s_{YT} > 0, s_{YT} \leq -1$
13	$r_E = 0$	for $s_{We} > 1$	Not existing	for $s_{We} > 1$
	Not existing	for $s_{We} = 1$		for $s_{We} = 1$
14	Not existing	for $s_{LL} \geq 1$	$r_M = 0$	for $s_{LL} \geq 1$
15	$r_E = r_F$	for $s_F \geq -\frac{1}{r_F}$	$r_M = \frac{r_F}{1 + r_F s_F}$	for $s_F > -\frac{1}{r_F}$
			Not existing	for $s_F = -\frac{1}{r_F}$
16	$r_E = r_{KL}$	for $s_{KL} > 0, s_{KL} < -1$	$r_M = \frac{r_{KL}(s_{KL} + 1)}{s_{KL}}$	for $s_{KL} > 0, s_{KL} < -1$
	$r_E = r_{KL}$	for $s_{KL} = 0$	Not existing	for $s_{KL} = 0$
	$r_E = 0$	for $s_{KL} = -1$		for $s_{KL} = -1$
17	$r_E = r_B$	for $s_B \geq 0$	$r_M = \frac{r_B}{s_B}$	for $s_B > 0$
			Not existing	for $s_B = 0$

estimates were higher than the corresponding mean values. Only for the models 7, 9, 10, 13 and 17 were the standard errors clearly lower than the estimates of the shape parameters. The estimated value of the initial disease level on day 11 (t_0) was rather constant for the models (roughly between 0.02 and 0.025), except for models 9 and 13, for which clearly lower values were estimated (0.0108 and 0.0074, respectively).

Although the estimated values of the rate and the shape parameter varied considerably, the characteristics calculated from these values were rather stable for the different models (Table 6). The disease levels at the inflection point (y_{ip}), calculated by using the functions of Table 3, varied only from 0.473 to 0.497. Similarly, the values of the integral variables as given in Table 5 had narrow ranges; $WMAR$ varied from 0.0664 to 0.0666, and $WMRR$ from 0.201 to 0.234. The time when the point of inflection was reached (t_{ip}) corresponded to a narrow time window, ranging from 8.71 to 9.07 days after the start of disease observation on day 11.

All models could describe the observed data for cv. Kennebec well (Table 7), resulting in coefficients of determination between 98.93 and 99.18%, corresponding to SSE in the range from 0.0384 to 0.0294. The fitted curves of the best model (equation 7) (Richards) and the worst model (equation 12) (Yang and TeBeest) were again very similar (Fig. 4B), differing slightly only at the beginning of the epidemic. The rate of exponential disease progress (r_E) calculated for models 10, 11, and 15 to 17 according to Table 4 was highly variable (from 0.349 to 0.542 per day). The range of the estimated values of the shape parameter was lower

than for cv. Monona. The shape parameter value determined for Richards model ($s_R = 0.954$) indicated that the observations nearly followed a Gompertz growth function corresponding to $s_R \rightarrow 1$. Unlike the previous example with cv. Monona, in which disease progression was similar to a logistic function, here the standard error of the estimated shape parameter values was clearly lower than the estimated values, except for the hyper-Gompertz model (equation 9). The values calculated for the initial disease level vary over several magnitudes, from 6.73×10^{-3} to 1.22×10^{-15} . The curve characteristics calculated from the estimated values were again rather stable over all models (Table 7), but showed a higher variability compared with those on cv. Monona (Table 6). The disease level at the point of inflection, for instance, ranged from 0.343 to 0.459. On the other hand, the values for $WMAR$ were very similar (between 0.0402 and 0.0417), while for $WMRR$, the variation was higher (from 0.138 to 0.171). The time to reach the point of inflection (t_{ip}) varied over more than 2 days, from day 12.86 to 15.28 after the beginning of the observation on day 11.

Relations among models. Identical models. For some models, the estimated parameter values were nearly the same, for instance, the rate parameter values of the models 15 to 17 (models of Fleming, Kosman-Levy, and Birch). In fact, models 15 to 17 are formally the same model, because they are identical under the conditions

$$r_F = r_{KL} = r_B \quad \text{and} \quad r_F s_F = -\frac{1}{s_{KL} + 1} = s_B - 1 \quad (31)$$

The numerical values of the shape parameters are in accordance

TABLE 5. The weighted mean absolute rate ($WMAR$) and the weighted mean relative rate ($WMRR$) for the 11 two-parametric models

Model	$WMAR$ (equation 20)	$WMRR$ (equation 21)
7	$\frac{r_R}{2} \cdot \frac{1}{s_R + 1}$	$\frac{r_R}{s_R}$
8 ^a	$\frac{r_{HL}}{6} \cdot \Gamma(2 - s_{HL}) \Gamma(2 + s_{HL})$	$\frac{r_{HL}}{2} \cdot (1 + s_{HL}) \Gamma(1 - s_{HL}) \Gamma(1 + s_{HL})$
9	$r_{HG} \cdot \frac{\Gamma(2 + s_{HG})}{2(2 + s_{HG})}$	$r_{HG} \cdot \Gamma(2 + s_{HG})$
10	$\frac{r_{WR}}{2} \cdot \frac{s_{WR}}{2s_{WR} + 1}$	No closed solution known
11	$r_{Wa} \cdot \frac{s_{Wa}^2}{(2s_{Wa} + 1)(3s_{Wa} + 1)}$	$r_{Wa} \cdot \frac{s_{Wa}}{2s_{Wa} + 1}$
12	No closed solution known	No closed solution known
13	$r_{We} \cdot \frac{s_{We} \Gamma(2 - 1/s_{We})}{2(2 - 1/s_{We})}$	No closed solution known
14 ^a	$\frac{r_{LL}}{6} \cdot s_{LL} \Gamma\left(2 - \frac{1}{s_{LL}}\right) \Gamma\left(2 + \frac{1}{s_{LL}}\right)$	$\frac{r_{LL}}{2} \cdot (1 + s_{LL}) \Gamma\left(1 - \frac{1}{s_{LL}}\right) \Gamma\left(1 + \frac{1}{s_{LL}}\right)$
15	$\frac{1}{s_F} \cdot \left[\frac{1}{2} + \frac{1}{r_F s_F} - \frac{r_F s_F + 1}{r_F^2 s_F^2} \cdot \ln(r_F s_F + 1) \right]$	$\frac{1}{s_F} \cdot \left[\frac{r_F s_F + 1}{r_F s_F} \cdot \ln(r_F s_F + 1) - 1 \right]$
16	$r_{KL} \cdot (s_{KL} + 1) \left[(s_{KL} + 1) \left(1 + s_{KL} \cdot \ln \frac{s_{KL}}{s_{KL} + 1} \right) - \frac{1}{2} \right]$	$r_{KL} \cdot (s_{KL} + 1) \left(1 + s_{KL} \cdot \ln \frac{s_{KL}}{s_{KL} + 1} \right)$
17	$r_B \cdot \frac{s_B^2 - 1 - 2s_B \ln s_B}{2(s_B - 1)^3}$	$r_B \cdot \frac{s_B \ln s_B - s_B + 1}{(s_B - 1)^2}$

^a Formulas for models 8 and 14 can be expressed using the sin-function (described in mathematical handbooks), which may allow easier calculations.

with equation 31, for instance, for Monona (Table 6): $r_F s_F = 0.188$, $-1/(s_{KL} + 1) = 0.188$, and $s_B - 1 = 0.182$. The rate and the shape parameters of the Kosman-Levy model 16 and Birch model 17 are independent, while the rate and the shape parameters of the Fleming model 15 cannot be separated.

Two other models, the hyper-logistic model 8 and the log-logistic model 14, are identical under the condition

$$r_{HL} = r_{LL} s_{LL} \text{ and } s_{HL} = \frac{1}{s_{LL}} \quad (32)$$

Also, in this case, the numerical values are in accordance with equation 32, for instance, for Kennebec (Table 7): $r_{HL} = 0.238$ per day versus $r_{LL} s_{LL} = 0.237$, and $s_{HL} = 0.233$ versus $1/s_{LL} = 0.232$. It is worth mentioning that the absolute rate of disease progress

Fig. 2. Relationships between the weighted mean absolute rates (WMAR) and the shape parameters s^* of models 7 to 17 (Table 1) for $r^* = 0.2$ per day. The dashed lines are valid for the extended ranges of the shape parameters. The dotted lines represent asymptotes of the relationships.

(i.e., the right side of the differential equations) of both models as well as of model 11 can be regarded as special cases of the probability density functions of the Beta distribution (7). Thus, the characteristics of the Beta distribution can be used in the comparison and transformation of these models.

Transforming one model into another. As different two-parametric models can be fitted to the same data (examples

provided in Tables 6 and 7), it would be desirable to determine the values of the rate and shape parameters of a certain model knowing both values of another model, when the two models are not identical. As two parameters have to be calculated, this problem can be solved by assuming that both models should fulfill two conditions. The two models could be identical with respect to the disease level at the inflection point (y_{ip}) or to the two integral

Fig. 3. Relationships between the weighted mean relative rates ($WMRR$) and the shape parameters s^* of models 7 to 17 (Table 1) for $r^* = 0.2$ per day. The dashed lines are valid for the extended ranges of the shape parameters. The dotted lines represent asymptotes of the relationships.

characteristics of the disease progress curve, the *WMAR* and the *WMRR*. The identity of the rate of exponential growth (r_E), if it exists (Table 4), at the very early stage of an epidemic could also be considered as an additional condition for comparison of parameters. However, the exponential rate (r_E) cannot be recommended for the comparison because it is rather variable. In the two examples given above (Tables 6 and 7), for instance, the rate r_E , which is equal to the corresponding model rate parameter r_s (except for Richards model 7, Table 4), varied over a wide range.

The characteristic y_{ip} allows a direct transformation from shape to shape parameter value, because y_{ip} does not depend on the rate parameter except for Fleming's model 15 (Table 3). Thus, given the actual value of y_{ip} for a model and assuming the same value for a second model, its shape parameter value can be calculated by rearranging the formula of y_{ip} to yield s_s as a function of y_{ip} . Except for models 7 and 10 for which numerical solutions are necessary, simple equations to calculate the shape parameter value can be derived (Table 8). Instead of y_{ip} , the quotient $Q =$

TABLE 6. Estimated rate (r_s) and shape parameter (s_s) values (with standard errors [SE]), initial disease level (y_0), coefficient of determination (R^2), sums of squared deviations (SSE), exponential rate (r_E), disease level (y_{ip}) and time (t_{ip}) at the point of inflection, weighted mean absolute rate (*WMAR*), and weighted mean relative rate (*WMRR*) of the 11 models fitted to the disease progress data of potato late blight on cv. Monona (data from Campbell and Madden [4] page 178) (integration started at day 11 [$t_0 = 11$])

Model	r_s (SE)	s_s (SE)	y_0	R^2 (%)	SSE ($\times 10^{-4}$)	r_E	y_{ip}	t_{ip}^a	<i>WMAR</i>	<i>WMRR</i>
7	0.366 (0.036)	1.757 (0.225)	0.0196	99.60	191.58	0.484	0.475	8.747	0.0664	0.208
8	0.399 (0.014)	0.033 (0.038)	0.0213	99.60	195.29	n.e. ^b	0.483	8.851	0.0665	0.206
9	0.243 (0.009)	-0.251 (0.051)	0.0108	99.65	171.99	n.e.	0.473	8.710	0.0664	0.223
10	0.418 (0.037)	0.877 (0.193)	0.0236	99.60	198.53	0.418	0.487	8.906	0.0665	0.203
11	0.406 (0.029)	52.16 (203.95)	0.0251	99.59	200.14	0.406	0.495	9.000	0.0665	0.201
12	0.412 (0.064)	50.80 (259.17)	0.0253	99.59	200.49	0.404	0.497	9.068	0.0666	0.201
13	0.075 (0.010)	3.115 (0.455)	0.0074	99.66	166.73	0	0.493	8.964	0.0664	0.234
14	0.014 (0.015)	28.02 (29.28)	0.0209	99.60	195.32	n.e.	0.482	8.856	0.0664	0.207
15	0.436 (0.048)	0.432 (0.481)	0.0218	99.60	195.79	0.436	0.479	8.804	0.0665	0.205
16	0.436 (0.048)	-6.315 (6.524)	0.0219	99.60	195.79	0.436	0.479	8.793	0.0665	0.205
17	0.435 (0.047)	1.182 (0.225)	0.0219	99.60	195.80	0.435	0.479	8.815	0.0665	0.205

^a Number of days after the day $t_0 = 11$ (start of disease observation).

^b n.e. = not existing.

Fig. 4. Disease progress curves of the best (solid lines) and worst (dotted lines) models fitted to the data of **A**, cv. Monona and **B**, cv. Kennebec.

TABLE 7. Estimated rate (r_s) and shape parameter (s_s) values (with standard errors [SE]), initial disease level (y_0), coefficient of determination (R^2), sums of squared deviations (SSE), exponential rate (r_E), disease level (y_{ip}) and time (t_{ip}) at the point of inflection, weighted mean absolute rate (*WMAR*), and weighted mean relative rate (*WMRR*) of the 11 models fitted to the disease progress data of potato late blight on cv. Kennebec (data from Campbell and Madden [4] page 178) (integration started at day 11 [$t_0 = 11$])

Model	r_s (SE)	s_s (SE)	y_0	R^2 (%)	SSE ($\times 10^{-4}$)	r_E	y_{ip}	t_{ip}^a	<i>WMAR</i>	<i>WMRR</i>
7	0.163 (0.016)	0.954 (0.159)	1.65e-5	99.18	294.19	n.e. ^b	0.359	13.220	0.0417	0.171
8	0.238 (0.009)	0.233 (0.056)	0.0002	99.11	317.91	n.e.	0.383	13.662	0.0411	0.161
9	0.166 (0.007)	-0.008 (0.076)	0.0001	99.18	294.68	n.e.	0.371	13.447	0.0415	0.165
10	0.457 (0.077)	0.285 (0.066)	0.0026	99.11	317.22	0.457	0.349	13.009	0.0415	0.155
11	0.349 (0.038)	2.091 (0.605)	0.0067	98.97	370.47	0.349	0.404	14.061	0.0404	0.141
12	0.673 (0.215)	1.413 (0.444)	0.0078	98.93	384.45	0.332	0.414	14.269	0.0402	0.138
13	0.054 (0.001)	2.589 (0.121)	1.22e-15	99.01	355.01	0	0.459	15.278	0.0410	0.157
14	0.055 (0.013)	4.312 (0.986)	0.0002	99.11	317.90	n.e.	0.384	13.691	0.0410	0.160
15	0.535 (0.118)	4.874 (0.934)	0.0015	99.17	298.03	0.535	0.345	13.006	0.0416	0.159
16	0.542 (0.121)	-1.375 (0.156)	0.0015	99.17	298.07	0.542	0.343	12.866	0.0416	0.160
17	0.532 (0.112)	3.577 (1.000)	0.0016	99.17	298.04	0.532	0.346	12.958	0.0416	0.159

^a Number of days after the day $t_0 = 11$ (start of disease observation).

^b n.e. = not existing.

WMAR/WMRR can be used to determine the shape parameter value, because Q is also only a function of the shape parameter for all models (except for model 15). These functions $Q(s_*)$ are monotonous and of simple form for some models (not including the Gamma-function) even if WMAR and WMRR are more complicated. The inverse function $s_*(Q)$ for some models are listed in Table 8, showing that for Richards model 7 a simple expression can be derived to determine the shape parameter value.

After the determination of the shape parameter value, the value of the rate parameter can be calculated by assuming an identical value of WMAR (or WMRR) for both models and rearranging the formulas in Table 5 to give the rate parameter as a function of the shape parameter and WMAR or WMRR, respectively.

The values of the rate and the shape parameters describe the appearance of the progress curve, but not its position in time, which is determined by either the initial value y_0 at time t_0 or the time when the point of inflection (t_{ip}) is reached. For all models, both characteristics are related by functions given in Table 3. It is not possible to use the same value for y_0 in calculating the progression according to different growth functions as y_0 estimated for the same data set can vary over several magnitudes (examples provided in Tables 6 and 7). On the other hand, the time to reach the point of inflection is rather stable across models fitted to the same data (like in Tables 6 and 7). Thus, assuming that t_{ip} of a second model will be identical to that of a given model, the formula for t_{ip} in Table 3 can be rearranged to give y_0 as a function of t_{ip} .

Example for transforming models. As an example, the fitted disease progress curve of Fleming's model 15 for Monona was transformed into a Richards progress curve obeying equation 7. Based on the values of r_F and s_F in Table 6, the curve characteristics of Fleming's model can be calculated using the formulas from Tables 3 and 5 to attain y_{ip} , WMAR, and WMRR in Table 6 and then $Q = 0.0665/0.2053 = 0.3239$. Assuming that the disease level at the point of inflection (y_{ip}) should be the same for the unknown Richards function, the equation $0.479 = s_R^{1/(1-s_R)}$ (Table 3) has to be solved numerically leading to $s_R = 1.790$. When, however, it is assumed that the unknown Richards function has the same Q value, the corresponding value of the shape parameter

s_R can be easily calculated using the formula (Table 8) $s_R = 2Q/(1-2Q) = 1.839$. Thus, the two estimates of Richards shape parameter differ slightly. Knowing the value of the shape parameter, the value of the rate parameter can be determined with the formula of Table 5 as $r_R = WMRR \cdot s_R$. This leads to the two estimates for the rate $r_R = 0.378$ or 0.367 per day depending on the two methods applied to determine the value of the shape parameter. All parameter values of the Richards model calculated from Fleming's model are close to those values, which were determined by directly fitting Richards model to the data ($r_R = 0.366$ per day and $s_R = 1.757$ in Table 6).

To determine the temporal position of the Richards function, first the time to reach the inflection point for Fleming's model has to be calculated: $t_{ip} = 8.804$ (Table 6). Then, the formula for the Richards model 7 in Table 3 has to be rearranged leading to

$$y_0 = (1 - (1 - s_R)e^{r_R t_{ip}})^{1/(1-s_R)}$$

Using the rate and shape parameter values determined above with two methods, the initial value y_0 for the Richards function can be calculated as 0.0223 or 0.0211, respectively. Again, these values are close to the value $y_0 = 0.0196$ given in Table 6 for the direct fitting of the Richards model. The progress curves of Fleming's model (fitted to the data) as well as of Richards model (with parameter values determined from Fleming's model using Q) are nearly identical (figures not shown).

DISCUSSION

In this paper, different differential-equation models that all contain a rate and a shape parameter to describe plant disease epidemics were discussed. Therefore, the problem arises of deciding which out of the "cafeteria of equations" (26) can be recommended for progress curve analysis. Waggoner (26) emphasized the biological meaning of the differential equations as the primary criterion to decide the suitability of a model, while Campbell and Madden (4) pointed out that the fit of the models to data also matters a lot, and that statistical criteria are necessary to select models.

In our comparison, general mathematical characteristics of the models were developed, but also the usefulness of models in fitting two observed epidemics was tested. In the numerical examples, the differential equations were fitted to the data, while in similar studies in literature, the growth functions resulting after integration were fitted to the observations, either directly with nonlinear regression (4) or indirectly with linear regression after the data have been transformed appropriately (8). The use of transformations, however, is not useful for two-parameter models because the transformations depend on the shape parameter, as already shown for both the Richards and Weibull function (12). On the other hand, nonlinear regression analysis using the growth functions is only possible for models having analytical solutions. Closed solutions are known for 6 out of the 11 models in our comparison, whereas for five other models, the numerical fitting of the differential equation to the data is absolutely necessary. Today, however, this disadvantage is less important because suitable methods and software are now available for the numerical integration (ModelMaker 4, ModelKinetix).

For the development of model characteristics, only the rate and shape parameters are used, but for the numerical integrations, the initial value of the disease was estimated as an additional parameter y_0 . The alternative would have been to specify the first non-zero disease severity as initial value of the disease, like Ngugi et al. (15) did when they applied the same software to fit the Lotka-Volterra competition equations to two epidemics of sorghum diseases. In fact, the estimated values of y_0 varied over several orders of magnitude for the different models, especially for the Kennebec data (Table 8). Fixing the initial disease value would

TABLE 8. The shape parameters s_* as functions of the disease level at the inflection point (y_{ip}) and of the quotient $Q = MMAR/WMRR$, respectively

Model	$s_*(y_{ip})$	$s_*(Q)$
7	Numerical solution	$\frac{2Q}{1-2Q}$
8	$1-2y_{ip}$	$1-3Q$
9	$-(1-\ln y_{ip})$	$-(2+\ln Q/\ln 2)$
10	Numerical solution	No closed solution known
11	$\frac{y_{ip}}{1-2y_{ip}}$	$\frac{Q}{1-3Q}$
12	$\frac{1}{1-2y_{ip}} \cdot \left[1 + \frac{(1-y_{ip}) \ln(1-y_{ip})}{y_{ip}} \right]$	No closed solution known
13	$\frac{1}{1+\ln(1-y_{ip})}$	No closed solution known
14	$\frac{1}{1-2y_{ip}}$	$\frac{1}{1-3Q}$
15	$[(1-y_{ip})^2/y_{ip}^2 - 1]/r_F$	Numerical solution
16	$\frac{y_{ip}^2}{2y_{ip}-1} - 1$	Numerical solution
17	$(1-y_{ip})^2/y_{ip}^2$	Numerical solution

therefore not allow comparisons of the other parameters, specifically the rate parameters which are usually highly negatively correlated with the initial disease level, as shown, for example, for the logistic model (20) or the Weibull model (21).

The initial disease level (y_0) is also an important characteristic used in transforming one model into another. Using the actual values of the rate and shape parameter of one model, formulas have been established to calculate the values of the two parameters of another model, based on the identity of y_{ip} , $WMAR$, or $WMRR$. This procedure is an extension of the method of Campbell and Madden (4) who used $WMAR$ to compare two Richards functions with different shape parameters. It should be pointed out that this kind of transforming from one function to another just guarantees that the two functions look similar. Two Richards functions with the same $WMAR$ (for instance $WMAR = 0.025$, e.g., a logistic one with $r_L = 0.15$ per day and a Gompertz one with $r_G = 0.1$ per day) starting from an identical initial disease level (for instance $y_0 = 0.001$), have a similar appearance, but are clearly shifted in time. In this case, a disease level of $y = 0.5$ is reached by the Gompertz function after 23 days, while the logistic curve needs 46 days to reach this level. Therefore, y_0 also has to be transformed from one Richards model to another, when the real progress of the epidemics is of interest. Similar to this special case, the values of the rate and the shape parameters describe only the appearance of the progress curve, but not its position in time. Consequently, if two-parametric models are transformed into each other taking into account the temporal position, in addition to the two parameters, the initial value y_0 at time t_0 or the time t_{ip} when the point of inflection is reached also has to be transformed.

In the numerical solution, the way in which a model is mathematically written (i.e., its model parameterization) influences the precision of the estimates (13). As an example, consider the models 15 to 17, which are identical (described previously). Thus, the estimates for the rates are practically the same, but the values of the shape parameters vary among the models. In addition, the standard errors with which the shape parameters are estimated differed greatly. In the example of the late blight epidemic on cv. Monona (Table 6), the standard errors of the shape parameter of models 15 and 16 are of the same magnitude as the values of the shape parameter, while for model 17, the standard error is clearly lower than the parameter value. Thus, in this example, the shape parameter was estimated more precisely for model 17 than for models 15 and 16, although all three models are identical. However, the statistical properties (intrinsic nonlinearity and parameter-effects nonlinearity) of most models are not known and further work should address this topic.

The precision of the estimated shape parameters of the models depended on the actual values of the shape parameters and thus on the shape of the progress curve observed. For instance, the progress curve on cv. Monona (Table 6) was similar to a logistic curve, which is a special case for models 11 and 12 if the shape parameter values are infinitely high (Table 2). Therefore, the estimated shape parameter values are quite high for both models (52.2 and 50.8 in Table 6), but the standard errors are four to five times greater than the shape parameter value, showing that in this example the shape parameter cannot be estimated very precisely. In the example of the epidemic on cv. Kennebec, in which the observed progress nearly followed a Gompertz function, the shape parameter can be estimated much more precisely for models 11 and 12 (Table 7), so that the standard errors are only about one third of the parameter values.

In some cases, qualitatively different disease progress curves could be obtained for different values of the shape parameter within the confidence interval of its estimate. For the Richards model, for example, the estimate of the shape parameter $s_R = 0.954$ (SE = 0.159) with the Kennebec data (Table 7, model 7) allowed values greater and less than 1. In this case, exponential and nonexponential types of growth for $s_R > 1$ and $s_R < 1$ are

possible at the beginning of the epidemic (Table 4), a problem already mentioned by Birch (3). To avoid this situation, it is recommended to apply another model instead of, or in addition to, the Richards one, if the type of disease progress at the beginning is considered an important characteristic of the epidemic.

Many numerical approximations of disease progress curves by models 7 to 17 were realized (Tables 6 and 7; data not shown), showing that the collection of models can represent a very wide range of nonlinear disease progress curves. As they are capable of representing curves of the same shape, the question of the best model for representing recorded data does not seem practically important. Rather, a decisive issue in the choice of a suitable model should be whether estimations of various disease characteristics possess biological realism. The following three criteria can be considered for comparison of the models and selection of the most flexible ones: (i) exponential type of growth at the beginning of an epidemic; (ii) monomolecular type of growth at the end of an epidemic; and (iii) possibility for disease at the inflection point to take an arbitrary value between 0 and 1.

Although none of the considered models fully meets these conditions, the following five models fulfill them to a large extent: Richards model 7, Waggoner and Rich model 10, Fleming model 15, Kosman-Levy model 16, and Birch model 17 (Table 4). The Richards model 7 can even generate monomolecular type of growth for the whole epidemic (Table 2), but the exponential growth is impossible if values of the shape parameter s_R belong to the interval between 0 and 1 (Table 4). Therefore, the Richards model 7 just partly meets criterion (i) and also (iii) in this case, because if $s_R > 1$, the disease level at the inflection point is necessarily greater than $1/e$ (Fig. 1). A disease progress curve of the Waggoner-Rich model cannot approach its asymptote like the monomolecular function if its shape parameter is in its extended range ($s_{WR} \leq -1$) (Table 4); this eliminates the possibility of the disease level at the inflection point to be greater than $1 - 1/e$ (Fig. 1). Thus, the Waggoner-Rich model 10 only partly fulfills criterion (ii), but criterion (iii) is also only partially met in this case. However, the exponential type of growth at the beginning of an epidemic is always possible for the Waggoner-Rich model.

Summarizing the results of model comparisons with regard to the mentioned criteria, one can conclude that the Fleming model 15, Kosman-Levy model 16, Birch model 17, Richards model 7, and Waggoner-Rich model 10 are the most flexible ones. These models clearly provided a good fit to the two data sets, but future research should investigate the statistical properties of these models for different data sets to determine the best parameterization as well as the best model to use. Nevertheless, it is important to mention that other models may provide the best description of actual data. For example, the Weibull model 13, which is excellent for empirical curve fitting (3), was the best model for cv. Monona data (Table 6).

Thus, models 7, 10, and 15 to 17 (which are in fact one model) provide a good representation of actual data and possess properties for describing disease progress curves of different types. We recommend applying one of these models.

APPENDIX

Standard representations of the log-logistic disease progress curve and the corresponding differential equation are

$$y(t) = \frac{1}{1 + Bt^{-c}} \quad \text{and} \quad \frac{dy}{dt} = \frac{c}{t} \cdot y \cdot (1 - y)$$

where

$$B = \frac{1 - y_1}{y_1}$$

is determined by a value of y at $t = 1$, $y_1 = y(1)$. The replacement of t in the differential equation with its expression from the

growth function

$$t = \left(\frac{By}{1-y} \right)^{1/c}$$

results in the following form of the log-logistic model:

$$\frac{dy}{dt} = cB^{-1/c} \cdot y^{(1-1/c)} \cdot (1-y)^{(1+1/c)}$$

In a more general way, the log-logistic growth function and its differential equation can be written as

$$y(t) = \frac{1}{1 + \left(\frac{t-a}{b} \right)^{-c}} = \frac{1}{1 + b^c (t-a)^{-c}} \quad \text{and} \quad \frac{dy}{dt} = \frac{c}{(t-a)} \cdot y \cdot (1-y)$$

Solving again $y(t)$ for

$$(t-a) = b \left(\frac{y}{1-y} \right)^{1/c}$$

and replacing this term in the differential equations leads to

$$\frac{dy}{dt} = \frac{c}{b} \cdot y^{(1-1/c)} \cdot (1-y)^{(1+1/c)}$$

To be consistent with all other models, in which the exponents of y and $(1-y)$ are related to the shape parameter, the differential equation of the log-logistic model (equation 14) is rewritten as

$$\frac{dy}{dt} = r_{LL} s_{LL} y^{(1-1/s_{LL})} (1-y)^{(1+1/s_{LL})}$$

using

$$r_{LL} = \frac{1}{b} \quad \text{and} \quad s_{LL} = c$$

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