Disease Progression as a Function of Plant Growth

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ABSTRACT

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Many plants exhibit susceptibility to infection only during their period of growth. For these pathosystems, disease progression could be expressed as a function of plant growth instead of time. The logistic, Gompertz, monomolecular, power, and other functions can be used to describe plant growth as well as the increase of disease. Functions describing these two processes were combined into single models by deriving the absolute rate of change of the proportion of disease relative to plant growth. Given this approach, four basic types of models were identified. A model was described as being either similar or dissimilar depending on whether or not the disease and plant growth patterns (i.e., their functions) were the same. Similarly, if plant growth occurred prior to onset of the epidemic, the model was considered to have a nonsynchronous temporal structure, while if both processes were initiated at the same point in time, the models were termed synchronous. Graphic comparisons of data from simulations indicated that points of inflection, concavity, and asymptotes could be readily varied through manipulation of model parameters. Although modeling disease progression as a function of plant growth does not require a cause and effect relationship, these models would, nevertheless, be particularly applicable to pathosystems in which the occurrence of disease is dependent on the production of young, susceptible plant tissue.

The classic ecological approach to the study of population dynamics is to examine changes in number or density of a species over time. The absolute rate of change of population size with respect to time was often described by the typical bell-shaped curve. The mathematical formulation of this process into the logistic function was first performed by Verhulst in 1838 (13). The logistic function was further modified for modeling plant disease progression by allowing the pathogen population to be measured as the proportion of tissue infected (12). The monomolecular, Gompertz, Richards, and Weibull functions have also been proposed as models for plant disease progression (4,9). Similarly, their rate of change of disease was determined relative to time and their dependent variable was also expressed as the proportion of tissue infected.

The expression of the proportion of disease as a function of time assumes that all plant material is susceptible to infection throughout the epidemic. However, many plants or plant parts change in their susceptibility to disease over time. Populer (10) presented a tentative system for explaining the changing pattern of susceptibility in relation to the plant part age. He classified plant part susceptibility into four groups: Type I, susceptibility during the growth period; Type II, susceptibility increases with tissue age during the adult period; Type III, susceptibility initially high during the growth period decreasing to a low level in middle life,

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then increasing as the tissue ages; and finally, no susceptibility changes during the entire life span of the plant part. We propose that for pathosystems having Type I plant susceptibility, disease progression could be expressed as a function of plant growth.

The objective of this study was to combine functions that describe disease progression and plant growth into a single model. Emphasis was not necessarily on correcting disease progression for plant growth, as recently performed (6), but rather to model it as the plant grows. Thus, the dependent variable was expressed as an amount or density of diseased tissue, and the absolute rate of change of disease was derived relative to plant growth instead of time. And because plant growth has been modeled by a variety of functions (2), many of which are used for describing disease progression, not one, but an entire set of models was derived.

DERIVATION OF MODELS

The progression of disease, a measure of the growth of the pathogen, can follow a growth pattern that is similar or dissimilar to the growth pattern followed by the suscept. Furthermore, disease progression and plant growth can either be synchronous or nonsynchronous. Thus, four types of models can be constructed, their components dependent on the form of disease progression and plant growth and the temporal relationship between these two growth patterns. Although many mathematical functions can be used for simulating their growth, we will limit our study to four possibilities: the exponential, logistic, monomolecular, and Gompertz functions (Table 1).

Similar, synchronous disease progression and plant growth. Assume that both disease progression and plant growth occur in a logistic manner, and that the pathogen initiates the epidemic when the plant begins its growth. The differential equations describing their absolute rates of growth are, respectively,

$$dy/dt = r_1 y(K_1 - y)/K_1 \quad (1)$$

$$dx/dt = r_2 x(K_2 - x)/K_2\,, \quad (2)$$

where r_1 and r_2 are intrinsic rates, y is the amount or density of diseased tissue (e.g., amount of infected leaf area per shoot), x is plant size or density (e.g., amount of leaf area per shoot), K_1 and K_2 are carrying capacities of the environment, and t is time. To determine the change in the amount of tissue diseased relative to the change in the amount of plant tissue, dy/dx, equation 1 is divided by equation 2 to yield

$$dy/dx = (dy/dt)/(dx/dt) = [r_1 y K_2(K_1 - y)]/[r_2 x K_1(K_2 - x)]. \quad (3)$$

The terms are rearranged and integration is then performed on both sides of the equation by expressing the denominators as partial fractions:

$$\int \left\{ 1/[r_1 y K_2(K_1 - y)] \right\} dy = \int \left\{ 1/[r_2 x K_1(K_2 - x)] \right\} dx \quad (4)$$

TABLE 1. Differential and integral forms of the growth functions used in the derivation of the composite disease/plant growth models

Growth function	Differential form[a]	Integral form[a]
Exponential	$dz/dt = rz$	$z = Ae^{rt}$
Logistic	$dz/dt = rz[(K - z)/K]$	$z = K/(1 + Ae^{-rt})$
Monomolecular	$dz/dt = r(K - z)$	$z = K(1 - Ae^{-rt})$
Gompertz	$dz/dt = rz[\ln(K) - \ln(z)]$	$z = Ke^{-Ae^{-rt}}$

[a] dz/dt is the absolute rate of growth; r is the intrinsic rate of growth; z is population size or density; K is the carrying capacity; t is time; and A is an additional parameter, its meaning dependent on the function.

$$[1/(r_1 K_1 K_2)] \left\{ \int (1/y)dy + \int [1/(K_1 - y)]dy \right\} =$$

$$[1/(r_2 K_1 K_2)] \left\{ \int (1/x)dx + \int [1/(K_2 - x)]dx \right\} \quad (5)$$

The integrals on both sides are solved by u-substitution, then equation 5 becomes

$$-[1/(r_1 K_1 K_2)] \ln[(K_1 - y)/y] + C_1/(r_1 K_1 K_2) =$$

$$-[1/(r_2 K_1 K_2)] \ln[(K_2 - x)/x] + C_2/(r_2 K_1 K_2) \quad (6)$$

where C_1 and C_2 are constants of integration. Solving for y, the amount of diseased tissue, produces

$$y = K_1/\left\{ 1 + e^{C_1} e^{-r_1 C_2/r_2} [(K_2 - x)/x]^{r_1/r_2} \right\} \quad (7)$$

Setting $B = e^{C_1} e^{-r_1 C_2/r_2}$ and $r_g = r_1/r_2$, the intrinsic rate of disease progression relative to plant growth, then equation 7 can be reparametrized to denote y more clearly as a function of x,

$$y = K_1/\left\{ 1 + B[(K_2 - x)/x]^{r_g} \right\} \quad (8)$$

Equation 8 can also be derived directly from the integral forms of equations 1 and 2, which are, respectively,

$$y = K_1/(1 + e^{C_1} e^{-r_1 t}) \quad (9)$$

$$x = K_2/(1 + e^{C_2} e^{-r_2 t}) \quad (10)$$

where C_1 and C_2 are constants of integration. We begin by solving equation 10 for time to yield

$$t = -(1/r_2) \ln[(K_2 - x)/x] + C_2/r_2. \quad (11)$$

Given that y is a function of t in equation 9, $y = f(t)$, and in equation 11, t is a function of x, $t = g(x)$, then the composite function $y = f[g(x)]$ can be readily derived by substituting equation 11 for t in equation 9:

$$y = K_1/\left\{ 1 + e^{C_1} e^{-r_1[-(1/r_2)\ln[(K_2 - x)/x] + C_2/r_2]} \right\} \quad (12)$$

Simplification of the denominator will eventually produce a function identical to equation 7. Thus, the derivation of disease progression as a function of plant growth from the differential dy/dx is equivalent to the substitution of plant growth for time via the composite function. Similar, synchronous models were also derived in the same manner for the other three growth functions (Table 2).

Dissimilar, synchronous disease progression and plant growth. In some pathosystems, disease may progress with time in a manner quite different from the growth pattern of the suscept. As an example, assume that disease progression can be modeled by the Gompertz function, whereas plant growth increases in a monomolecular fashion. The integrated form of these two functions are, respectively,

$$y = K_1 e^{-B_1 e^{-r_1 t}} \quad (13)$$

$$x = K_2(1 - B_2 e^{-r_2 t}), \quad (14)$$

where y is the amount or density of diseased tissue, x is plant size or density, K_1 and K_2 are carrying capacities, r_1 and r_2 are rate parameters, and $B_1 = \ln(K_1/y_0)$ and $B_2 = (K_2 - x_0)/K_2$ for y_0 and x_0 at time $t = 0$. Given that their growth is synchronous, equation 14 can be solved for time and substituted into equation 13 to yield the composite function

$$y = K_1 e^{-B_1 (1/B_2)^{r_1/r_2} [(K_2 - x)/K_2]^{r_1/r_2}} \quad (15)$$

Letting $B = B_1(1/B_2)^{r_1/r_2}$ and $r_g = r_1/r_2$, the function can be rewritten as

$$y = K_1 e^{-B[(K_2 - x)/K_2]^{r_g}} \quad (16)$$

Another dissimilar, synchronous model also having Gompertz disease progression but logistic plant growth was derived in the same manner (Table 2). Note that regardless of the nature of the suscept's growth, the general form of the composite model will resemble the function describing disease progression.

Similar, nonsynchronous disease progression and plant growth. Nonsynchronous dynamics result when either the pathogen or the plant begins to grow before the other. A pathogen may initiate its growth before the suscept while on an alternate host or in a saprophytic stage. However, by definition disease cannot occur until suscept tissue is available for infection; plant growth, as our measure of time, is still set equal to zero. Thus, except for any buildup of initial inoculum that may occur, such early growth by the pathogen is inconsequential relative to the progression of disease on the plant. When plant growth does begin, its dynamics can be treated as if they were synchronous.

The initiation of plant growth before onset of the epidemic is an entirely different situation. By starting its growth before the pathogen, the plant is limiting the total seasonal amount of susceptible tissue that will be available for infection. In mathematical terms, the amount of plant tissue present at the beginning of the epidemic, x', is subtracted from the total amount of tissue, x. For example, the term $(x - x')$ is substituted for x in the synchronous logistic/logistic model (Eq. 8) to yield

$$y = K_1 / \left\{ 1 + B[(K_2 - (x - x'))/(x - x')]^{r_g} \right\} \quad (17)$$

This alteration essentially sets the plant growth clock equal to zero when disease progression begins, thus synchronizing the two growth patterns. A requirement of this transformation is that $x > x'$, which is reasonable because the pathogen does not incite disease during the period when $x < x'$. However, when $x = x'$, any nonsynchronous model having either a Gompertz or logistic plant growth component is undefined; this outcome is also true when $x = 0$ for the synchronous versions of these models.

Dissimilar, nonsynchronous disease progression and plant growth. The fourth and final model type involves a combination of the former two models. First, the dissimilar functions describing disease progression and plant growth are combined as previously described to form the single composite function. Then the $x - x'$ term is substituted for x to allow for any nonsynchronous dynamics. The nonsynchronous Gompertz/logistic model is presented as an example of this type of model (Table 2).

MODEL CALCULATIONS

Five of the models were compared graphically for a set of fictitious amounts of plant tissue ranging from $x = 1$ to $x = 100$. In addition, some of the parameters were altered to examine their effect on the shape of each function. The calculations were performed by computer programs written in the language C (5).

Similar, synchronous disease progression and plant growth. Each of the four parameters in the synchronous logistic/logistic model were separately altered to examine their effects on the epidemic (Fig. 1A and B). An increase in the rate parameter r_g from 0.25 to 4 caused the graph to rotate about its point of inflection at $x = 50$, $y = 5$ (Fig. 1A). Values of $r_g < 1$, indicating that the intrinsic rate of plant growth is faster than the intrinsic rate of disease progression, causes the curve to be concave down for $0 < x < 50$ and concave up for $50 < x < 100$. As the difference between these intrinsic rates lessens, the degree of curvature decreases until the plot becomes a straight line at $r_g = 1$. When $r_g > 1$, the rate of disease progress exceeds plant growth and the model produces the typical sigmoid shape. In Figure 1A, the curve for $r_g = 4$ is a reflection of that for $r_g = 0.25$ with respect to $r_g = 1$.

An increase in the parameter B from 0.5 to 2 for the synchronous logistic/logistic model causes a downward shift in the point of inflection (Fig. 1B). Although both epidemics eventually attain the same amount of disease at $x = 100$, larger values of B 'delayed' increase of disease during the epidemic. Decreasing K_1 and K_2 reduces the maximum amount of disease and plant tissue attained during the epidemic, respectively. In the latter case, disease increases much more rapidly to its maximum (relative to plant growth) than it would have if K_2 had been much larger; in essence, increasing K_2 increases the length of time of the epidemic.

In pathosystems having synchronous monomolecular disease progression and plant growth, alternation of the relative rate parameter r_g also changes the shape of the curve (Fig. 1C). If the rate of disease progression is greater than the rate of plant growth, $r_g > 1$, the curve produced resembles that of an exponential

TABLE 2. Composite disease/plant growth models derived from various functions

Model type[a]		Growth functions[b]	
Form	Temporal relationship	Disease/plant	Model[c]
Similar	Synchronous	Logistic/logistic	$y = K_1 / \left\{ 1 + B[(K_2 - x)/x]^{r_g} \right\}$
Similar	Synchronous	Gompertz/Gompertz	$y = K_1 e^{-B(\ln K_2 - \ln x)^{r_g}}$
Similar	Synchronous	Monomolecular/monomolecular	$y = K_1 \left\{ 1 - B[(K_2 - x)/K_2]^{r_g} \right\}$
Similar	Synchronous	Exponential/exponential[d]	$y = Bx^{r_g}$
Similar	Nonsynchronous	Logistic/logistic	$y = K_1 / \left\{ 1 + B[(K_2 - (x - x'))/(x - x')]^{r_g} \right\}$
Dissimilar	Synchronous	Gompertz/monomolecular	$y = K_1 e^{-B[(K_2 - x)/K_2]^{r_g}}$
Dissimilar	Synchronous	Gompertz/logistic	$y = K_1 e^{-B[(K_2 - x)/x]^{r_g}}$
Dissimilar	Nonsynchronous	Gompertz/logisitic	$y = K_1 e^{-B[(K_2 - (x - x'))/(x - x')]^{r_g}}$

[a] Similar models have the same function for both disease progression and plant growth, whereas dissimilar models have different functions; nonsynchronous dynamics result when the plant begins growth before onset of the epidemic; synchronous dynamics result when both disease and plant growth are initiated at approximately the same point in time.

[b] The growth functions used in the derivations are presented in Table 1.

[c] y is the amount or density of diseased tissue; x is the amount or density of plant tissue; r_g is the ratio of the rate of growth of disease, r_1, to the rate of plant growth, r_2: $r_g = r_1/r_2$; K_1 and K_2 are carrying capacities of the environment for disease and plant growth, respectively; x' is the amount or density of plant tissue at the beginning of the epidemic; and B is an additional parameter that has different roles in each model.

[d] Substituting the power functions $y = B_1 t^{r_1}$ and $x = B_2 t^{r_2}$ in place of the exponential functions for disease and plant growth, respectively, produces the same composite model.

saturation function: a rapid initial increase in disease followed by a slower increase as dy/dx approaches zero at K_1 and K_2. However, if $r_1 < r_2$ then $r_g < 1$ and disease increases in an exponential fashion relative to plant growth. But unlike the exponential function, the increase of disease is nearly linear for the first half of the epidemic. If $r_1 = r_2$, the resulting plot is identical to that produced by the synchronous logistic/logistic model: a straight line.

The value of the B parameter in the synchronous monomolecular/monomolecular model is related to the initial amount of inoculum, y_0. For $x = 0$, $y_0 = K_1(1 - B)$ or $B = (K_1 - y_0)/K_1$. Thus, B is the amount that disease will increase during the epidemic. If $K_1 = 10$, then $B = 1 - y_0/10$. This latter case is demonstrated in Figure 1C, where $B = 0.7$ and $y_0 = 3$.

Dissimilar and nonsynchronous disease progression and plant growth. Synchronous and nonsynchronous forms of the logistic/logistic and Gompertz/logistic models were compared (Fig. 1D). Substitution of Gompertz disease progression for logistic disease progression in the synchronous form of the model caused an initial delay in the epidemic. This delay occurs because the term

$$e^{-B[(K_2 - x)/x]^{r_g}}$$

in the Gompertz/logistic function is initially much smaller in value than the term

$$\left\{1 + B[(K_2 - x)/x]^{r_g}\right\}^{-1}$$

in the logistic/logistic function. Because these terms are simply multiplied by K_1 to obtain y, lower values of the term yield lower values of y. However, once the Gompertz/logistic function starts to increase, it does so rapidly so that it eventually attains the same level of disease as the logistic/logistic model.

If the dynamics of the above two models are nonsynchronous, as when disease progression starts at $x = x' = 30$, the curves are shifted to the right along the abscissa (Fig. 1D). Although these curves are identical in shape to their synchronous counterparts, disease does not progress to the point of reaching its carrying capacity of $K_1 = 10$. The plant has reached its carrying capacity first; its growth has stopped and it is therefore no longer producing susceptible tissue. A similar outcome also occurs for the logistic/logistic function with $x' = 50$ and $r_g = 0.5$. In this situation, nonsynchronous dynamics and an intrinsic rate of plant growth greater than that of disease progression combine to produce a curve resembling monomolecular growth.

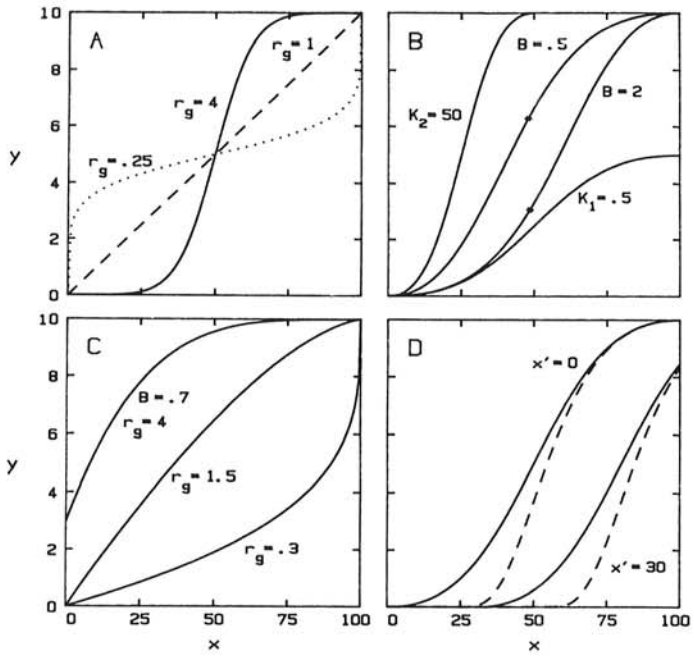

Fig. 1. Comparison of disease progression/plant growth models and examination of changes induced by altering model parameters; y = amount of diseased tissue; x = plant size or density, x' = amount or density of plant tissue at the beginning of the epidemic; $r_g = r_1/r_2$ = (intrinsic rate of disease progression)/(intrinsic rate of plant growth); K_1 and K_2 are carrying capacities of the environment for disease and plant growth, respectively; B is an additional parameter, its function dependent upon the model; values for the parameters, unless otherwise indicated, are: $r_g = 2$, $K_1 = 10$, $K_2 = 100$, and $B = 1$. **A and B,** synchronous logistic/logistic model. **C,** synchronous monomolecular/monomolecular model where $B = 0.999$ for the two lower curves. **D,** logistic/logistic (——) and Gompertz/logistic (----) models; synchronous ($x' = 0$) vs. the nonsynchronous ($x' = 30$) model types.

IMPLEMENTATION

Units of measurement. All models presented in Table 2, except the exponential/exponential function, will be dimensionally balanced regardless of the units of measurement employed for disease and plant tissue observations. Because K_2 and x are of the same units, the terms $(K_2 - x)/x$, $(K_2 - x)/K_2$, and $\ln(K_2) - \ln(x) = \ln(K_2/x)$ are all unitless. Furthermore, because both r_1 and r_2 have dimensions of t^{-1}, the relative rate r_g is also unitless. Thus, the equations are reduced to the dimensionally balanced situation of y units = K_1 units. This lack of dependency between the two variables in terms of their dimensions, however, does not necessarily imply any measurement combination will be adequate. The measurement of plant growth in terms of plant height, for example, may be a poor match for disease measured as the amount of infected tissue per plant part. The average amount of leaf area per plant part (e.g., shoot or branch) would most likely be a better indication of the production of susceptible tissue and, hence, a more accurate measure of biological time.

These models were initially conceived and reported as consisting of two components, one for pathogen growth and one for plant growth (7). Our interest was in examining how the amount of disease produced by the pathogen population changed with respect to plant growth. Thus, this approach necessitated the use of an absolute measure of disease such as the amount of surface area infected or the number of pathogen colonies per plant part. The use of a relative measure of the size of the pathogen population, such as the proportion of tissue or plant parts diseased, could be substituted without major mathematical modifications. However, the interpretation of the model is less clear since this dependent variable, by definition, includes a measure of suscept growth as its denominator.

Statistical considerations. Except for the synchronous exponential/exponential model, the proposed models are not intrinsically linear; their parameters cannot be made linear by transformation. Consequently, the fitting of these models to experimental data necessitates the use of nonlinear regression. Most methods for estimating the parameters of a nonlinear model, such as the Gauss–Newton or Marquardt methods, require determination of the partial derivatives of the function with respect to each parameter. However, many statistical software packages now have procedures that will numerically estimate the partial derivatives, thus eliminating the need for the differentiation.

An examination of the partial derivatives of the functions with respect to the parameter r_g reveals a limitation: The amount of plant growth cannot be greater than or equal to its carrying capacity, K_2. For example, for the synchronous monomolecular/monomolecular model, the partial derivative is

$$\partial f/\partial r_g = -K_1 B[(K_2 - x)/K_2]^{r_g} \ln[(K_2 - x)/K_2].$$

Because the techniques employed use an iterative process for minimizing the residual sum of squares, some intermediate values of $K_2 \leqslant x$ may be used during the fitting procedure. This inadvertently leads to the logarithm of zero or a negative number, and thus error statements. The same outcome occurs if the partial derivatives are numerically estimated. This limitation can be circumvented by setting the K_2 parameter equal to the maximum growth obtained by any one measurement; however, this

observation should not be an outlier. Then either that one observation can be dropped from the data set or a small fraction can be added to the value of K_2 so that it would be slightly greater than the maximum x. In either case, this process effectively changes K_2 from a parameter to a constant.

DISCUSSION

Plant pathologists have adopted and modified the basic population dynamics models developed by ecologists for use in the study of plant disease progression. But unlike the free-living species studied by most ecologists, plant parasites are inherently dependent on their hosts for existence. Although various forms of the Lotka–Volterra equations can be used for modeling symbiotic relationships (14), we suggest that plant growth itself can be used as a measure of biological time. This approach assumes that tissue is susceptible to infection only during the growth phase of the plant or plant part; Populer referred to this as a Type I pattern of susceptibility (10).

Given a pathosystem with a Type I pattern of susceptibility, the models derived in this paper can be employed in one of two ways. In the first case, plant growth is essentially envisioned as a substitute for time. Disease progression is a function of plant growth just as it would be a function of time: No cause and effect relationship is implied. In the second case, disease progression is dependent on plant growth. The pathogen can only infect young, susceptible tissue and its increase is therefore dependent on tissue production. This second approach has been demonstrated in the development of an apple powdery mildew model (8). In either case, the methodology outlined allows for the derivation of many composite models. For example, assuming the existence of only six growth functions, then there are $6!/(6 - 2)! = 30$ possible synchronous similar and dissimilar models. A total of 60 models can be derived if the nonsynchronous forms are included.

The incorporation of plant growth into a model for disease progression allows for the study of interactions between these two processes. Jeger (3) proposed a method for examining the effects of host growth on disease asymptotes. His approach, which involved "combining equations for increases in leaf area and diseased area," would allow for asymptotes less than one when disease is measured as a proportion. In the models proposed above, the effects of changes in host growth on the rate of disease progress (relative to plant growth) as well as on the disease asymptote can be examined. For example, epidemics caused by the apple powdery mildew pathogen *Podosphaera leucotricha* (Ell. & Ev.) Salm. are dependent on the availability of young tissue (1). The application of a high nitrogen fertilizer or plentiful rain could raise the growth rate of the plant, thereby decreasing r_g. However, the sudden flush of susceptible growth would allow the pathogen to increase rapidly, causing a concomitant increase in r_g. Similarly, a larger carrying capacity of the environment for plant growth should, in absolute units, produce a larger carrying capacity for the pathogen, all others factors being equal. This dependence of the pathogen's carrying capacity on plant growth can be addressed directly by making the disease asymptote a function of plant growth. Turner et al (11) derived a model in which the carrying capacity of a logistic function is itself growing logistically. Although this approach, like those proposed above, provide for the effects of plant growth on disease progression, these models do not incorporate a feedback mechanism which would allow for a decrease in plant growth as disease increases.

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