Relation Between Rate Parameters and Latent and Infectious Periods During a Plant Disease Epidemic

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ABSTRACT

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The observations that epidemic progress is often adequately described by a rate parameter (r) and initial amount of disease (y_o), and that calculated values of Vanderplank's R decrease during an epidemic, are used to obtain explicit time-dependent relationships between r and R during entire epidemics. Analysis of these relationships indicates that R can be calculated from epidemic data only during a finite period of time and that there are constraints on the combinations of parameter values possible in such epidemics. Further theoretical threshold results for epidemics are obtained and related to the doubling time—an important parameter in population ecology—of the epidemic. Vanderplank's R cannot easily be estimated for epidemics in which r is not approximately constant.

An interesting feature of the modeling of biological phenomena is the interplay between descriptive models and those invested with a higher level of biological realism, the former often used in conjunction with practical experimental programs and the latter serving largely theoretical purposes. Constructive interplay is often necessary for experimental and theoretical studies to advance in step rather than to diverge along separate paths. Vanderplank (13) introduced two rate parameters into plant disease epidemiology: r, the rate of disease increase per unit of disease, is essentially the intrinsic rate of disease increase dependent on the disease measure used (1,2); and R, the rate of increase per unit of infectious disease (eg, the diseased plant tissue actually producing inoculum), is more realistic biologically. The first rate, r, is defined in terms of the logistic equation

$$dy_t/dt = r\, y_t[1 - y_t], \tag{1}$$

where disease y_t is usually, but not necessarily, measured on a proportion scale and a disease asymptote of unity is assumed. The rate R is defined in terms of differential-difference equations that incorporate the time lag attributable to a latent period, p (ie, the time from onset of infection to sporulation)

$$dy_t/dt = R\, y_{t-p}[1 - y_t] \tag{2}$$

and also a finite infectious period, i (ie, the time from onset to cessation of sporulation)

$$dy_t/dt = R\,[y_{t-p} - y_{t-i-p}]\,[1 - y_t]. \tag{3}$$

Vanderplank dissociated himself from the logistic equation as a model of disease increase—see Vanderplank (14) for a recent statement of his views—largely because of the usual requirement for r to be a constant parameter. There is arguably some ambiguity in this view in that disease on a logit scale often does increase linearly with time; r is then estimated as the regression coefficient of the line and indeed this procedure was used by Vanderplank (13). The estimation of r by linear regression, although sometimes abused (4), has proven useful for comparative studies in plant breeding, fungicide evaluation, disease management, and cultural practice. Elaborations of the logistic equation with one or more additional parameters sometimes give marginally better fits to data, but this is not particularly relevant. The logistic equation with the assumption of a constant r remains the standard of comparison (10) for experimental data and other models in plant epidemiological studies. Some reasons why r often appears constant with time have been considered (16) but are not the concern here.

The differential-difference equations (equations 2 and 3), or other models that account for the various categories of disease (5,9,11), represent a higher order of biological realism and a richness of dynamic behavior not present with the logistic equation. Despite these features and despite the derivation of theoretical results of thresholds and asymptotic behavior (5,13), the use of the more realistic models has made little impact on experimental studies. Vanderplank (13) investigated the relationship of R with r during the early stages of an epidemic by ignoring the term $1 - y_t$ and equating the right-hand sides of equation 1 and either equation 2 or 3. On the assumption of constant r, and without progression from the infectious to the postinfectious condition (ie, without removals), then

$$R = r \exp(pr) \tag{4}$$

and with removals,

$$R = r/[\exp(-pr) - \exp(-(i + p)r)]. \tag{5}$$

In both cases, an assumption of constant r, p, and i implies a constant value for R, the rate parameter with biological meaning. If the entire epidemic is considered, by including the term $1 - y_t$, then the relationship between r and R has not been investigated.

Vanderplank (13) obtained constant r values for late blight epidemic data reported by Large (8). Values of R were then obtained numerically by using assumed and constant values for the latent and infectious periods of *Phytophthora infestans* (7). In the case without removals, the relationship between R and the amount of disease was linear and decreased to $R = r$. In the case with removals, the relationship between R and the amount of disease was again linear and decreased initially but reached a minimum value at $y < 1$ and then increased. Vanderplank gave convincing biological reasons why R must decrease if r remains constant during an epidemic. He then argued that because there is no known factor that could cause the late increase in the numerical value of R, then r could not be a constant at this stage of the epidemic. It is not clear whether these trends in R are real consequences of equating the dynamics of the logistic and differential-difference equations or are unique to the particular data. The purposes of this paper are to obtain explicit relationships between the two rate parameters, on the assumption that the logistic and differential-difference equations describe real epidemics; to ascertain the period of time during which reliable estimates of Vanderplank's R can be made

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from experimental data; and to explore the dependence of the relationship with the latent and infectious periods and other epidemiological parameters.

MATHEMATICAL ANALYSIS

Considering the case without removals, if equations 1 and 2 are to describe the same dynamics, then

$$R = r\, y_t / y_{t-p}. \tag{6}$$

It can readily be shown that y_t/y_{t-p} cannot be constant and hence one or both of r and R must be a variable unless, trivially, $p = 0$ or $r = R = 0$.

If r is a constant, then equation 1 can be solved to give

$$y_t = 1/[1 + A \exp(-rt)], \tag{7}$$

where A is the constant of integration (if y_o is the initial amount of disease, $A = (1 - y_o)/y_o$), and at time $t - p$,

$$y_{t-p} = 1/[1 + A \exp(pr) \exp(-rt)]. \tag{8}$$

Substituting equations 7 and 8 in equation 6, and rearranging, gives

$$R = r \exp(pr)/f(t), \tag{9}$$

where $f(t) = [1 + A \exp(-rt)]/[\exp(-pr) + A \exp(-rt)]$.

In particular, for $t = 0$ and large A, $R \simeq r \exp(pr)$, and as $t \to \infty$, $R \simeq r$. Furthermore, $dR/dr < 0$ for all $t > 0$ and hence R is a strictly decreasing function of time. Values of R can be calculated from equation 9 for given values of A, r, and p.

Considering the case with removals, if equations 1 and 3 are to describe the same dynamics, then

$$R = r\, y_t / [y_{t-p} - y_{t-i-p}]. \tag{10}$$

If r is a constant, then equation 1 can be solved to give y_t (equation 7), y_{t-p} (equation 8), and

$$y_{t-i-p} = 1/[1 + A \exp((i + p)r) \exp(-rt)]. \tag{11}$$

Substituting equations 7, 8, and 11 into equation 10, and rearranging, gives

$$R = r/[\exp(-pr)\, f(t) - \exp(-(i + p)r)\, g(t)], \tag{12}$$

where $f(t)$ is as before and $g(t) = [1 + A \exp(-rt)]/[\exp(-(i + p)r) + A \exp(-rt)]$.

In particular, for $t = 0$ and large A, $R \simeq r/[\exp(-pr) - \exp(-(i + p)r)]$, but as $t \to \infty$, R increases without bound. Differentiating equation 12 with respect to time and setting to zero gives $dR/dt = 0$ when

$$g(t) = [1 + A \exp(-rt)]/[\exp(-(i + p)r) + A \exp(-rt)].$$

$$df(t)/dt = \exp(-ir)\, dg(t)/dt. \tag{13}$$

Differentiating $f(t)$ and $g(t)$ gives $df(t)/dt = -rA \exp(-rt) \left\{[\exp(-pr) - 1]/[\exp(-pr) + A \exp(-rt)]^2\right\}$ and $dg(t)/dt = -rA \exp(-rt) \left\{[\exp(-(i+p)r) - 1]/[\exp(-(i + p)r) + A\exp(-rt)]^2\right\}$. Substituting into equation 13, rearranging, and collecting terms, $A \exp(-rt) = (\beta\lambda_1 - \alpha\lambda_2)/(\lambda_2 - \lambda_1)$, where $\alpha = \exp(-pr)$, $\beta = \exp(-(i + p)r)$ and $\lambda_1 = \sqrt{\alpha(1 - \alpha)}$, $\lambda_2 = \sqrt{\beta(1 - \beta)}$. Solving for t,

$$t = \ln [A(\lambda_2 - \lambda_1)/(\beta\lambda_1 - \alpha\lambda_2)]/r. \tag{14}$$

The minimum value of R at this time can be calculated from equation 12 for given values of A, r, p, and i and the corresponding amount of disease from equation 7. It is important, however, to check that combinations of values of A, r, p, and i are permissible given the constraints of equation 14. Clearly, as natural logarithms are being taken, then the term in square brackets must be positive for t to be defined, and greater than unity for time to take only positive values.

As $\alpha = \exp(-pr)$ and $\beta = \exp(-(i + p)r)$, $\beta < \alpha$ for all $i > 0$.

Manipulating this basic inequality eventually yields

$$\beta\lambda_1 < \alpha\lambda_2. \tag{15}$$

Hence the denominator of the term in square brackets (equation 14) is negative; this implies that $\lambda_2 - \lambda_1$ must also be negative because A is necessarily positive. Manipulating the inequality yields $\lambda_2 < \lambda_1$ if and only if

$$\beta + \alpha < 1. \tag{16}$$

Hence the first restriction to be placed on r, p, and i is that $\exp(-(i + p)r) + \exp(-pr) < 1$. This inequality is equivalent to

$$i > -\ln [\exp(pr) - 1]/r. \tag{17}$$

Because i is positive, then the inequality given by equation 17 necessarily holds unless $\ln [\exp(pr) - 1] < 0$, in which case it may not. Again, manipulation of the inequality shows than $\ln[\exp(pr) - 1]$ is negative if and only if $p < \ln 2/r = t_d$, the doubling time during the early logarithmic stage of the epidemic. If p is greater than the doubling time, then there is no lower restriction on the length of the infectious period, but if p is less than the doubling time, then a lower limit results as given by equation 17. Note that the inequality given by equation 17 cannot be inferred from the normal threshold result for an epidemic to increase, ie, $iR > 1$, which also must hold.

The time taken for R to be at a minimum (equation 14) is also dependent on the value of A, and hence of y_o, the initial amount of disease. If the time is to take only positive values, then we require

$$y_o < [1 + (\beta\lambda_1 - \alpha\lambda_2)/(\lambda_2 - \lambda_1)]^{-1}. \tag{18}$$

A sufficient condition to ensure the inequality given by equation 18 is that $y_o < |\lambda_2 - \lambda_1|/2$, where the modulus symbol indicates an absolute value. Details of the derivation of the inequalities given by equations 15–18 are available from the author on request.

RESULTS

Figs. 1 and 2 show, respectively, in the case with no removals (equation 9), the variation of R with time for values of A and of the product (pr) giving the same value of R at $t = 0$. Values of A are about equal to $1/y_o$, where y_o is the amount of initial disease: the smaller the value of y_o the longer R remains approximately constant (Fig. 1). However, it takes an order of magnitude change in A (and thus in y_o) to achieve a twofold to threefold change in R after 20 days of the epidemic. When the product (pr) is varied, there is a more pronounced effect on R (Fig. 2). For each value of pr, the curves eventually approach the lower limit set by r but do so at a rate inversely proportional to p. The length of time for R to decrease to r is longer for higher values of pr.

Fig. 3 shows the influence of introducing an infectious period of varying length (equation 12); the shorter the infectious period the higher the value of R initially and the earlier the minimum value of R is reached. The curves approach the lower limits set by the r values but then increase.

Equation 14 was solved for permissible combinations of parameter values. The positive time values that resulted are plotted in Fig. 4 in relation to r for different lengths of the infectious period. The intriguing feature of this plot is that at low r values, there are large increases in the time taken for R to be a minimum for only small increases in r. There is an optimal value of r that maximizes the length of time before R is a minimum and then a steady decline in time values for higher values of r. An expression for the optimal value of r can be obtained by differentiating equation 14 with respect to r, setting to zero, and solving, but the expression is cumbersome and of little direct value.

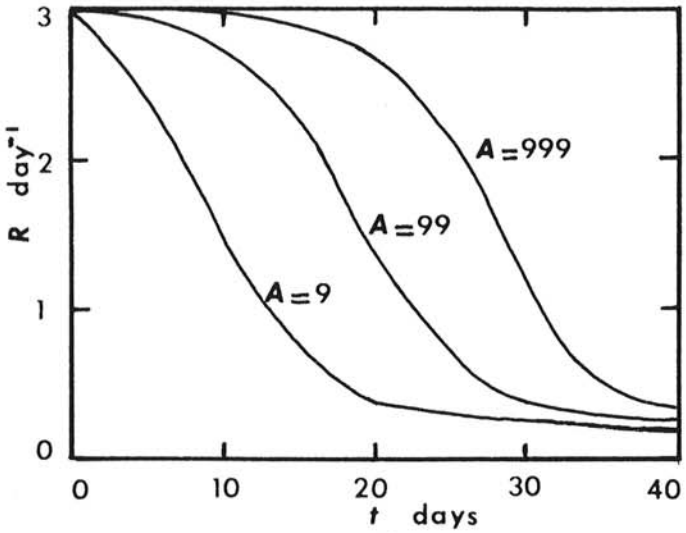

Fig. 1. Values of Vanderplank's R (day^{-1}) as a function of time t (days) (equation 9) for three values of $A = (1 - y_o)/y_o$, where y_o is initial disease at $t = 0$ ($r = 0.25$ day^{-1}, $p = 10$ days).

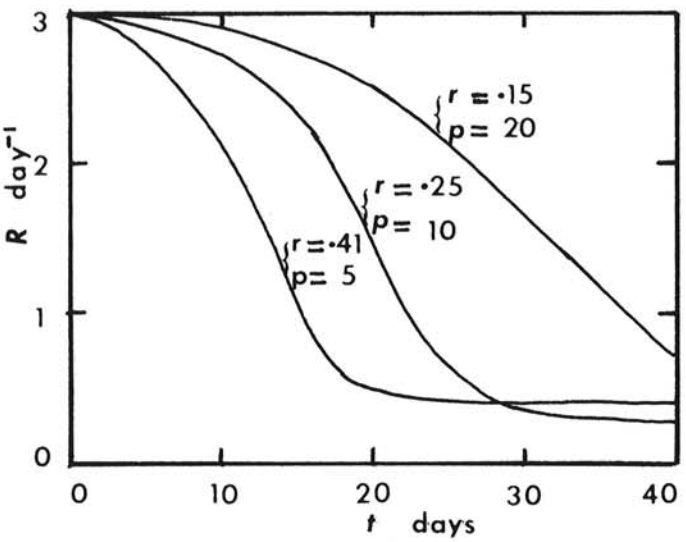

Fig. 2. Values of Vanderplank's R (day^{-1}) as a function of time t (days) (equation 9) for three values of pr giving the same value of R at $t = 0$ ($A = 99$).

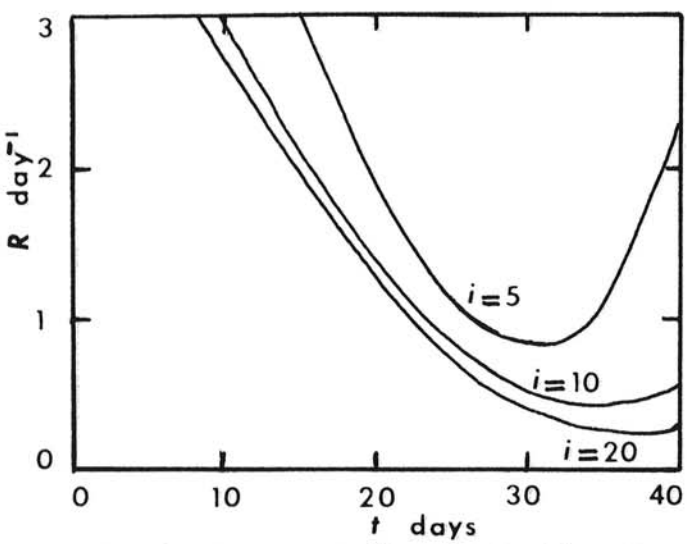

Fig. 3. Values of Vanderplank's R (day^{-1}) as a function of time t (days) (equation 12) when an infectious period i (days) of varying length is introduced ($r = 0.25$ day^{-1}, $p = 10$ days, $A = 99$).

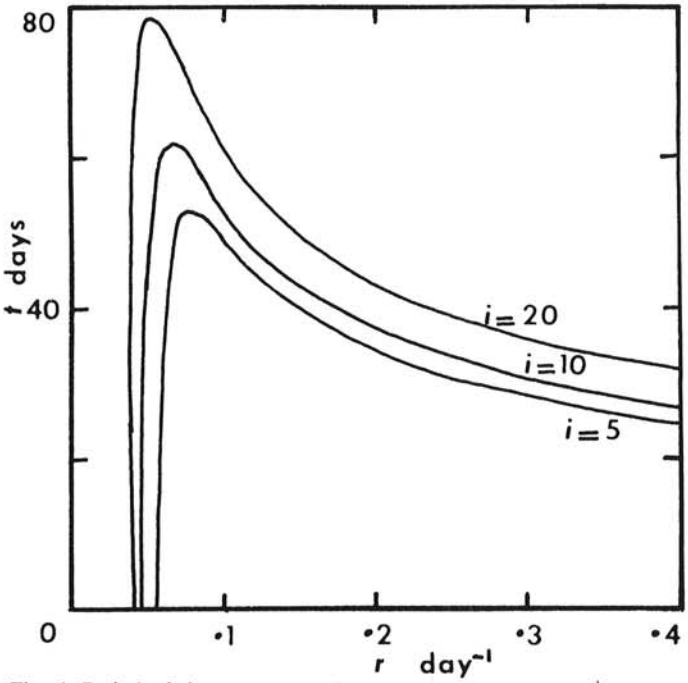

Fig. 4. Period of time t (days) before Vanderplank's R (day^{-1}) is at a minimum (equation 14) as a function of the intrinsic rate of disease increase r (day^{-1}) for infectious periods i (days) of varying length ($p = 10$ days, $A = 99$).

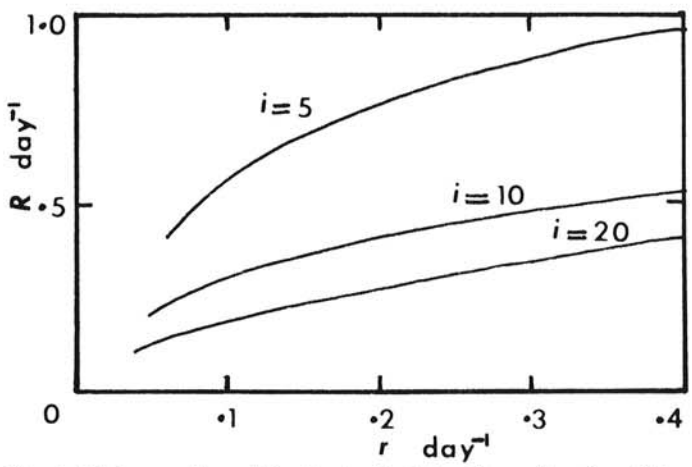

Fig. 5. Minimum values of Vanderplank's R (day^{-1}) as a function of the intrinsic rate of disease increase r (day^{-1}) for infectious periods i (days) of varying length ($p = 10$ days, $A = 99$).

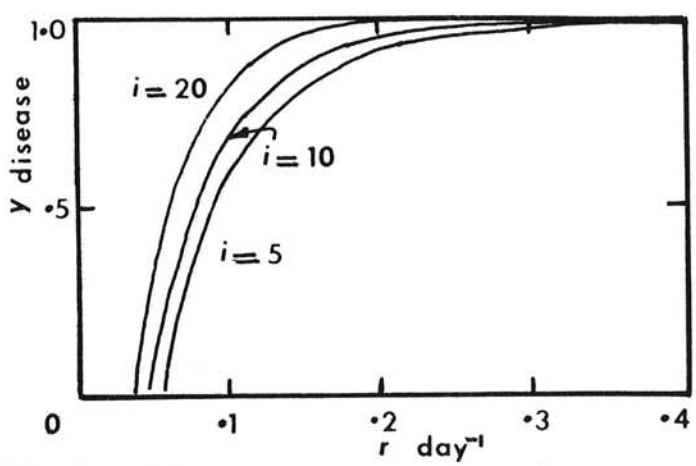

Fig. 6. Values of disease (y) at which Vanderplank's R (day^{-1}) is a minimum as a function of the intrinsic rate of disease increase r (day^{-1}) for infectious periods i (days) of varying length ($p = 10$ days, $A = 99$).

The minimum values of R (equation 12) and the corresponding amounts of disease y (equation 7) are plotted against r in Figs. 5 and 6, respectively, for different lengths of the infectious period. These figures are best viewed vertically. At low r values, the amount of disease when R is at a minimum, as with the time taken, is very sensitive to small changes in the r value; at high r values, the epidemic has virtually gone to completion before R is at a minimum; the minimum values of R, however, are less sensitive to changes in r and more sensitive to changes in the length of the infectious period.

Finally, the minimum values of i that are necessary for the inequality given by equation 17 to hold are plotted against p for values of p less than the doubling time of the epidemic (Fig. 7). Clearly, the lower the r value the longer the infectious period has to be if the differential-difference equation is to represent adequately epidemics for which r remains approximately constant.

DISCUSSION

It must be stressed that by equating the right-hand side of the logistic equation with that of equation 2 or 3 (ie, assuming the equations describe the same dynamics) immediately restricts the range of solutions of the differential-difference equations. There are infinitely many solutions for equations 2 or 3 that do not correspond to a logistic curve and the assumption of constant r. However, given the many examples of epidemic curves that are adequately described by the parameter r, then it is entirely reasonable to estimate the behavior of R, the biologically important parameter, using the techniques described here. The fact that many epidemics appear logistic, with apparently constant r, may reflect the quality of data collected. Where r can be shown to vary significantly with discontinuous random process due to environment, then the present techniques cannot be used to estimate Vanderplank's R. In those cases, it will be necessary to develop models in which r is strictly a time-dependent variable, using perhaps a sinusoidal function (15). Dependent on the tractability of the model, new versions of equations 9, 12, and 14 may be obtained but are unlikely to be any less complex.

Vanderplank (13) analyzed the particular data of Large (8) to obtain relationships between r and R for different values of the latent and infectious periods. Equations 9 and 12 show that this is possible in the general case: if r is constant, then R is an explicit function of time depending on the functions f and g. The similarity of these equations (equations 9 and 12) to the equivalent ones for the early stages of an epidemic (equations 4 and 5) can be noted by setting f and g equal to unity. Solving equations 9 and 12, using the values of A, r, p, and i assumed or computed by Vanderplank (13) from Large's data, gives R as an explicit function of time. In particular, the minimum value of R (3.51 day^{-1}) occurs, by application of equation 14, after 19 days of the epidemic and corresponds to a value of disease of 0.86 (13, Fig. 8.2). Vanderplank's conclusion that it is only reasonable to accept a constant r value while R is strictly decreasing is an important but neglected insight that can now be developed further.

From a practical standpoint, equation 14 allows for the a priori determination of the period of time for which the biologically important parameter can be estimated. It sets an upper limit to the time during which r can be assumed to remain constant, and hence estimated using conventional procedures. The threshold value of initial disease should not be exceeded if r and R are to be estimated reliably, thus emphasizing the importance of disease assessments early in the epidemic.

Further findings obtained by means of equation 14 are of note: first, there is an optimal intrinsic rate of disease increase that maximizes the time before R is at a minimum; second, when the length of the latent period is less than the doubling time of the epidemic, there is a lower limit to the length of the infectious period. These findings concerning life strategies (ie, constraints on the combinations of values for the various epidemiological parameters) may be of value in developing a more ecological perspective on plant disease epidemics. Is there any evidence for, or a biological interpretation of, an optimal intrinsic rate of disease increase in the sense described? Is there any evidence that where intrinsic rates of disease increase are low, the length of infectious periods must exceed a certain minimum value? According to Kranz (6), there are few examples of inequalities being used in epidemiology. Earlier threshold results (5,13) specify the minimum conditions for an epidemic to increase; the inequalities developed here specify stronger conditions if epidemic increase is to appear approximately logistic.

The relationships between the two rate parameters and the latent and infectious periods established in this paper are not simply of passing theoretical interest. There are many questions concerning practical epidemiological problems that require further theoretical advances. For example, models of selection for biocide resistance that incorporate latent period and the simple relationship between r and R during the logarithmic stage of an epidemic (12) have been proposed. As pointed out by Jeffery and Kable (3), selection for resistance is not likely to occur during the logarithmic phase only. It is important that relationships between r and R that hold over many epidemic cycles, especially as influenced by the length of the infectious period, be considered if only to examine the robustness of conclusions already drawn from such models.

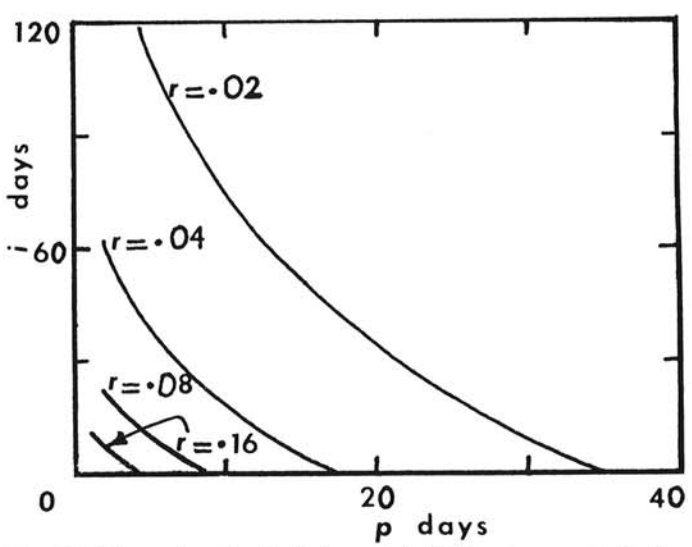

Fig. 7. Minimum length of infectious period i (days) necessary for the inequality given by equation 17 to hold, as a function of the latent period p (days), where $p <$ the doubling time during the logarithmic stage of the epidemic ($t_d = \ln 2/r$), and for different values of the intrinsic rate of disease increase r (day^{-1}).

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