The Relation Between Total, Infectious, and Postinfectious Diseased Plant Tissue

M. J. Jeger

Department of Plant Pathology, East Malling Research Station, Maidstone, Kent ME19 6BJ, England.
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ABSTRACT

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A new mathematical analysis that links the rates of change of total, infectious, and removed (postinfectious) diseased plant tissue is presented. Equations are obtained that give the proportion of total diseased tissue that is infectious or postinfectious in terms of biologically meaningful parameters. These are the rate of increase per infectious diseased tissue, and the rates at which disease progresses from the latent to the infectious and from the infectious to the postinfectious condition. Comparisons with the parameters R, p, and i of Vanderplank's differential-difference equation are made. Reported instances in which disease increase is unexpectedly rapid after a fungicide program ends are explicable in terms of this analysis, but experimental corroboration is lacking.

Additional key words: theoretical epidemiology.

The recent development of models and simulation techniques in the epidemiology of plant diseases owes much to the conceptual and mathematical framework provided by Vankerplank (8–10). Modeling now is nearly always done by using a digital computer (an aid that Vanderplank [8–10] largely did without), which is considered by most epidemiologists to be indispensable.

The diversion of modeling effort into simulation might well prove important in the 'tactical' management of plant disease, but that should not obscure the many unresolved problems in the theory of plant disease epidemics. One such problem is the relation between infectious and total diseased tissue, in which the term "infectious" denotes those units of diseased tissue actually producing inocula, and the term "total" includes latent and "removed" (postinfectious) as well as infectious diseased plant tissue. How does this relation change as an epidemic progresses to completion under ideal environmental conditions, or is perturbed by human intervention and adverse weather? Virtually no advance has been made since Vanderplank (8) presented equations of disease increase. These will be restated; followed by a new analysis that supplements and extends Vanderplank's treatment. For brevity, Vanderplank's treatment of "removals" or progression to the postinfectious condition is omitted. The terms "infectious disease" and "removed disease" can be objected to and will not be used in this paper; they have been replaced by the less ambiguous, if cumbersome, "infectious diseased tissue" and "postinfectious diseased tissue."

In the simplest case, which involves logarithmic increase and ignores postinfectious tissue, so that there are only two categories of diseased tissue—latent and infectious,

$$dx_t/dt = rx_t \quad (1)$$

$$dx_t/dt = Rx_{t-p} \quad (2)$$

in which x_t are units of diseased tissue (latent plus infectious) at time t, p is the duration of the latent period, x_{t-p} are units of diseased tissue at time $t-p$ and hence infectious at time t, and r and R are Vanderplank's apparent and basic 'infection' rates, respectively.

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The status of these equations is arguably different. Equation 1 is an operational definition of an empirical parameter r, the rate of disease increase per diseased tissue unit, whereas equation 2 is more explanatory; both p and R, the rate of disease increase per infectious tissue unit, have biological meaning and new infections are ascribed only to infectious diseased tissue. The two equations, however, can be related. A particular solution to equation 1 can be rewritten as

$$x_{t-p}/x_t = \exp(-pr) \quad (3)$$

which gives, according to this model, the proportion of total diseased tissue that is infectious. Referring to equations 1 and 2 gives the relationship established by Vanderplank (8),

$$r = R \exp(-pr). \quad (4)$$

Unfortunately, although this transcendental equation can be solved graphically, x_{t-p}/x_t is *not* explicitly given by R and p, the parameters that have biological meaning. A similar effect is obtained when postinfectious tissues are taken into account.

If disease is measured as a proportion of an upper limit to disease and logistic versions of equations 1 and 2 are considered, then

$$dx_t/dt = rx_t(1-x_t) \quad (5)$$

and

$$dx_t/dt = Rx_{t-p}(1-x_t). \quad (6)$$

Equation 6 is now the one with biological relevance. However, unlike equations 1 and 2, equation 5 cannot be consistent with equation 6 (or its equivalent, including postinfectious tissue) unless r is a variable, which severely restricts further analysis.

Can the analysis of plant disease epidemics, which takes into account infectious diseased tissue, be developed further? Several possibilities exist: analytical treatment of the differential-difference equations (7,11), simulation (12), and matrix modeling (5). The following approach, which is used in medical epidemiology (2), specifies differential equations for categories of disease and is most relevant for polycyclic fungal epidemics. The intention is to develop relationships between infectious, postinfectious, and total diseased tissue under two sets of conditions, one set during the early stages of an epidemic and one set during the later stages when the availability of healthy tissue places an upper limit to the amount of disease. For each set of conditions, the analysis will be developed first without, and then with, the effect of postinfectious diseased tissue.

MATHEMATICAL ANALYSIS AND INTERPRETATION OF RESULTS

Early stages of epidemic—no progression to the postinfectious condition. Suppose that disease increases according to

$$dx/dt = k_1 s \tag{7}$$

and

$$ds/dt = k_2 (x-s) \tag{8}$$

in which x represents total diseased tissue (say latent-plus-sporulating lesions), s represents infectious diseased tissue (sporulating lesions), and k_1 and k_2 are positive rate parameters.

Whereas equations 1 and 2 describe only the increase of total diseased tissue, equations 7 and 8 link both total and infectious diseased tissue.

From equations 7 and 8, linear second-order differential equations in x, or in s arise. For s,

$$d^2s/dt^2 + k_2\, ds/dt - k_1k_2s = 0. \tag{9}$$

The general solution of equation 9 is well known (4, Chapter 4-2) and because of the negative term, solutions take the form

$$s = A \exp(\lambda_1 t) + B \exp(\lambda_2 t) \tag{10}$$

with

$$\lambda_1 = -k_2/2 + \sqrt{(k_2^2 + 4\, k_1k_2)/4}$$

$$\lambda_2 = -k_2/2 - \sqrt{(k_2^2 + 4\, k_1k_2)/4}.$$

Two initial conditions are required for a particular solution. Suppose (as will be done throughout this paper) that all disease is latent on day $t=0$; ie, $s=0$, and that $ds/dt = k_2x_0$. Then solving for A and B gives $A = k_2x_0/(\lambda_1-\lambda_2)$ and $B = -k_2\, x_0/(\lambda_1-\lambda_2)$, which can be substituted in equation 10.

Note, however, that if $k_1 >> k_2$ then $\lambda_1 \simeq \sqrt{(k_1k_2)}$ and $\lambda_2 \simeq -\sqrt{(k_1k_2)}$, and a good approximation for s is given by the hyperbolic sine function

$$s = x_0\sqrt{k_2/k_1}\, \sinh(\sqrt{k_1k_2}\, t). \tag{11}$$

Substituting s into equation 7, and integrating, gives

$$x = x_0 \cosh(\sqrt{k_1k_2}\, t) \tag{12}$$

and from equations 11 and 12

$$s/x = (\sqrt{k_2/k_1}) \tanh(\sqrt{k_1k_2}\, t) \tag{13}$$

which gives the proportion of total diseased tissue that is infectious.

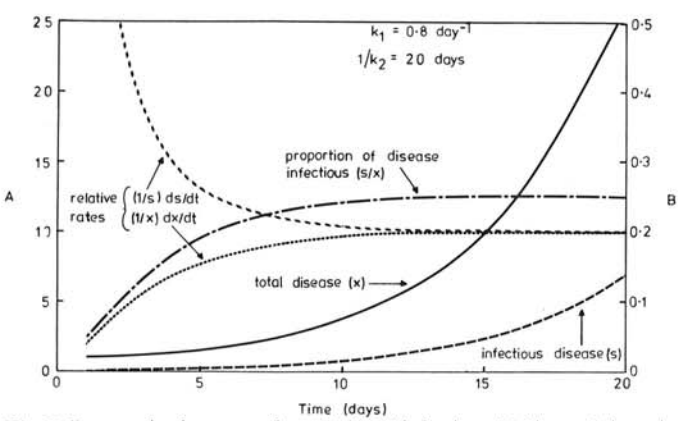

Fig. 1. Progressive increase of total (x) and infectious (s) diseased tissue in multiples of x_0 (ordinate A). Proportion of diseased tissue that is infectious (s/x) and relative rates of its increase per day (ordinate B). Parameter values are $k_1 = 0.8$ day^{-1} and $1/k_2 = 20$ days.

The relative rates are given by

$$(1/x)\, dx/dt = \sqrt{k_1k_2} \tanh(\sqrt{k_1k_2}\, t) \tag{14}$$

$$(1/s)\, ds/dt = \sqrt{k_1k_2} \tanh(\sqrt{k_1k_2}\, t). \tag{15}$$

When $\sqrt{(k_1k_2)}\, t \geqslant \sim 2.5$, then the proportion of diseased plant tissue that is infectious (s/x) will be approximately constant ($\simeq \sqrt{(k_2/k_1)}$), as will both relative rates ($\simeq \sqrt{(k_1k_2)}$).

From equations 13 and 14 (or directly from equation 7, $x \neq 0$),

$$(1/x)\, dx/dt = k_1 s/x. \tag{16}$$

That is, the relative rate of disease increase is directly proportional to the proportion of diseased tissue that is infectious.

Total diseased tissue (equation 12) and infectious diseased tissue (equation 11) during the early stages of an epidemic are plotted in Fig. 1 for given values of k_1 and k_2 (with k_1 16 times greater than k_2). Also shown are the proportions of diseased tissue that is infectious (equation 13) and the relative rates of increase (equations 14 and 15), each of which approaches a constant value. Fig. 2 shows this constant proportion and relative rates that arise for a representative range of values of k_1 and k_2. It can be seen that a high k_1 and $1/k_2$ will mean that a low proportion of diseased tissue is infectious. Conversely a small k_1 and $1/k_2$ will mean that a high proportion of diseased tissue is infectious. A low relative rate occurs only for small values of k_1; a high relative rate occurs only for small values of $1/k_2$.

Equations 14 and 15 give the relative rates explicitly in terms of k_1 and k_2. This is equivalent to finding an explicit expression for r in terms of R and p (which equation 4 fails to do).

The simple form of the solutions given in equations 11–15 depend on $k_1 >> k_2$ and on a particular specification of the initial state of disease. However, equation 10 can be investigated without restriction on the comparative values of k_1 and k_2, with many different initial conditions depending on the original configuration or 'age structure' of the epidemic and new formulations of equations 11–15 arise. It is not the intention here to be exhaustive.

The importance of the dimensionless parameter $\sqrt{(k_2/k_1)}$ is clear from equation 13. The question arises as to how k_1 and k_2 relate to the parameters in equations 1–6? The parameter k_1, the rate of increase in total diseased tissue per unit of infectious diseased tissue is comparable to Vanderplank's R as defined by equation 2. The parameter k_2 gives the rate at which lesions are progressing from the latent to infectious condition. On any given day at time t there are $x-s$ lesions in latency. It can be shown that the mean length of time that these lesions subsequently remain in latency is given by $1/k_2$. Hence R and $1/p$ are comparable, but not equivalent, to k_1 and k_2. Different models of infectious disease are involved; equation 2 implies a latent period of fixed and determinate duration as opposed to the mean parameter derived here. Neither model allows for the stochastic nature of the latent period and the distributions that follow. However, from the many examples given

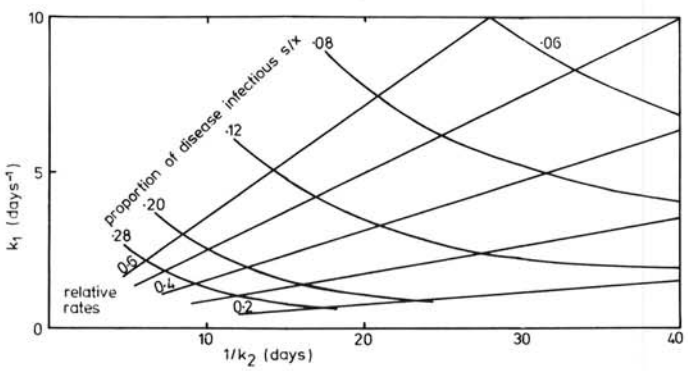

Fig. 2. Proportion of diseased tissue that is infectious (s/x) and relative rates of its increase (per day) that stabilize during the early stage of an epidemic for a representative range of values of k_1 and $1/k_2$.

by Vanderplank (8), estimates of R are usually in the range 1.0–10.0 (per day) or even higher, and for p in the range 5–30 days. This, despite the qualification above, gives some justification for an assumption that $k_1 >> k_2$.

Early stages of an epidemic—with progression to the postinfectious condition. Suppose that disease increases according to equation 7, but that x now includes postinfectious as well as latent and infectious units of diseased tissue. New equations have to be specified for the rates of increase of infectious and postinfectious diseased tissue. Suppose that

$$ds/dt = k_2 (x - (s + r)) - k_3 s \tag{17}$$

and

$$dr/dt = k_3 s \tag{18}$$

in which r are units of postinfectious diseased tissue (say sterile lesions) and k_3 is a positive rate parameter.

Equations 7, 17, and 18 now link together the rates of increase of total, infectious, and postinfectious units of diseased tissue. From these equations, linear second-order differential equations arise. For s,

$$d^2s/dt^2 + (k_3 + k_2)\, ds/dt - k_2 (k_1 - k_3)\, s = 0. \tag{19}$$

Note immediately that if $k_3 = 0$; ie, no postinfectious diseased tissue, then equation 19 reduces to equation 9.

Solutions of equation 19 again take the form of equation 10

$$s = A \exp(\alpha_1 t) + B \exp(\alpha_2 t) \tag{20}$$

with
$$\alpha_1 = -(k_3 + k_2)/2 + \sqrt{\{(k_3 + k_2)^2 + 4 k_2 (k_1 - k_3)\}/4}$$
$$\alpha_2 = -(k_3 + k_2)/2 - \sqrt{\{(k_3 + k_2)^2 + 4 k_2 (k_1 - k_3)\}/4}\,.$$

For positive values of k_1, k_2, and k_3 then α_1 and α_2 are unequal real numbers (the term beneath the square root sign is always positive). Hence solutions for s do not oscillate but increase if $k_1 > k_3$ and decrease if $k_1 \leqslant k_3$. Note also that if $k_1 >> (k_2 + k_3)$ then $\alpha_1 \simeq \sqrt{(k_1 k_2)}$ and $\alpha_2 \simeq -\sqrt{(k_1 k_2)}$ as before. Initial conditions are required. Suppose, as before, that all disease is latent on day $t = 0$; ie, $s = 0$, $r = 0$, and that $ds/dt = k_2 x_0$. Then

$$s = k_2 x_0 (\exp \alpha_1 t - \exp \alpha_2 t)/(\alpha_1 - \alpha_2). \tag{21}$$

Substituting s into equations 7 and 18, and integrating, gives

$$x = x_0 (1 + k_1\, g(t)) \tag{22}$$

and

$$r = k_3 x_0\, g(t) \tag{23}$$

in which g is the function

$$g(t) = k_2 \left\{1 + (\alpha_2 \exp \alpha_1 t - \alpha_1 \exp \alpha_2 t)/(\alpha_1 - \alpha_2)\right\} / \alpha_1 \alpha_2.$$

From equations 22 and 23

$$r/(x - x_0) = k_3/k_1. \tag{24}$$

Hence, as x increases, the proportion of total diseased tissue that is postinfectious is approximately constant ($\simeq k_3/k_1$).

The proportions of diseased tissue that are infectious and postinfectious, calculated from equations 21–23 are plotted in Fig. 3 for the values of k_1 and k_2 used in Fig. 1, but with two values of k_3, representing a very low (0.02 day^{-1}) and a reasonably high (0.10 day^{-1}) rate of progression to the postinfectious condition. The effect of progression to the postinfectious condition on reducing the proportion of diseased tissue that is infectious is clearly shown. The importance of the dimensionless parameter k_3/k_1 is clear from equation 24. The parameter k_3 gives the rate at which lesions are progressing from the infectious to the postinfectious condition. By the same reasoning used before, the mean length of time spent infectious is given by $1/k_3$—a parameter comparable to i, the symbol used by Vanderplank (8) to denote an infectious period of fixed and determinate duration. Interestingly the result here, that $k_1 > k_3$ for s to increase, corresponds to Vanderplank's epidemic threshold theorem, $iR > 1$.

Later stages of epidemic—no progression to the postinfectious condition. Suppose now that there is an upper limit to the amount of diseased tissue and that total disease measured as a proportion of this upper limit increases according to

$$dx/dt = k_1 s (1 - x). \tag{25}$$

Infectious diseased tissue again measured as a proportion of the upper limit, increases according to equation 8. Then second-order differential equations result for x or s. For s,

$$d^2s/dt^2 + (k_2 + k_1 s)\, ds/dt - k_1 k_2 s (1 - s) = 0. \tag{26}$$

This is a nonlinear equation, less tractable analytically than those preceding and can be considered elsewhere. However, if $k_1 >> k_2$, so that s is small compared to x during the early stages of the epidemic, and given the initial conditions as before, then an approximation for s in terms of x can be derived from equations 25 and 8.

$$s = \sqrt{k_2/k_1}\left\{-2(\ln(1 - x) + x)\right\}^{1/2}. \tag{27}$$

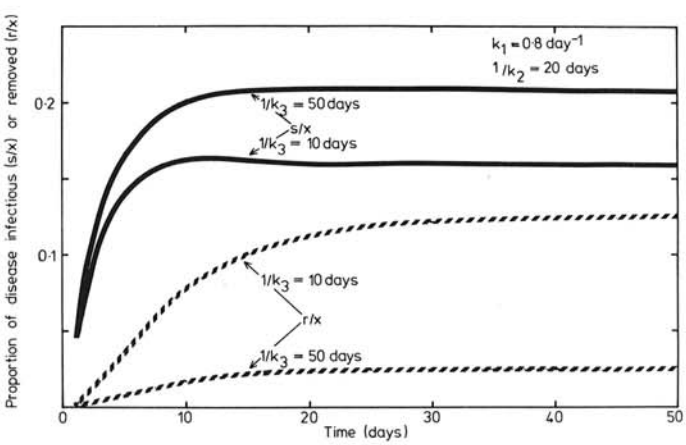

Fig. 3. Proportion of diseased tissue that is infectious (s/x) (solid lines) and removed (postinfectious) (r/x) (broken lines) for parameter values of $k_1 = 0.8$ day^{-1}, $1/k_2 = 20$ days together with a high ($1/k_3 = 10$ days) or low ($1/k_3 = 50$ days) rate of progression to the postinfectious condition.

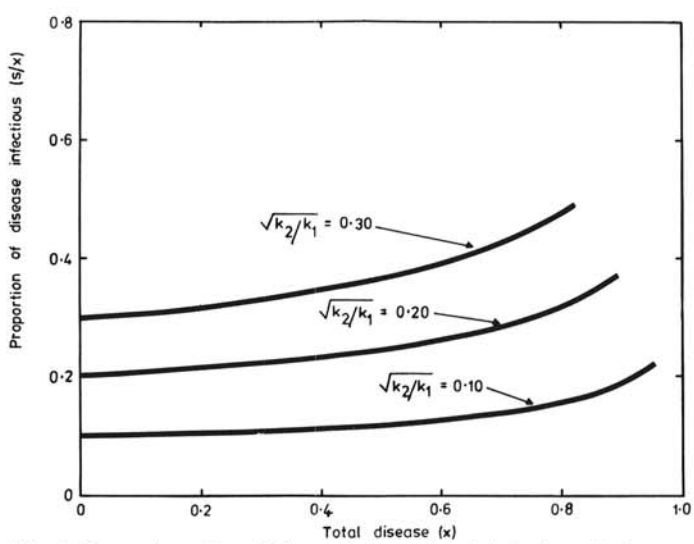

Fig. 4. Proportion of total diseased tissue that is infectious (s/x) as a function of total disease (x) for three values of $\sqrt{(k_2/k_1)}$.

From equation 27 can be obtained the proportion of diseased tissue that is infectious during the epidemic. For small x this is approximately equal to $\sqrt{(k_2/k_1)}$, but even at higher values of x the proportion of diseased tissue that is infectious is determined largely by the parameter $\sqrt{(k_2/k_1)}$; this is shown in Fig. 4 for given values of $\sqrt{(k_2/k_1)}$. The plots have been terminated at the point where the amount of latent diseased tissue $(x - s)$ is at a maximum—equivalently when ds/dx is unity. Thereafter the proportion increases rapidly and $s/x = 1$ (ie, all disease is sporulating) for some value of $x<1$. Ignoring postinfectious diseased tissue is patently unrealistic at these high levels of disease and equation 27 is of limited applicability.

Later stages of epidemic—with progression to the postinfectious condition. Suppose that disease increase is now specified by equations 25, 17, and 18. It is possible to derive a higher order, nonlinear differential equation, but this is not particularly useful. It is not, in general, possible to solve for s in terms of x. However, the proportion of diseased tissue that is postinfectious during the epidemic can be derived.

From equations 18 and 25

$$dr/dx = k_3/k_1\,(1 - x). \tag{28}$$

Integrating and specifying, as before, that at $t = 0$, $r = 0$ gives

$$r = (k_3/k_1)\,(\ln(1 - x_0) - \ln(1 - x)). \tag{29}$$

From equation 29 can be obtained the proportion of diseased tissue that became postinfectious during the epidemic. For small values of x this is approximately equal to k_3/k_1, thereafter the proportion increases until $r = x$. This is shown in Fig. 5 for given values of k_3/k_1. Note that $r/x = 1$ for some value of $x<1.0$. But at this value of x there can be no further increase in diseased tissue; all of it is postinfectious. Hence this value of x, say x_{max}, is the maximum possible amount of diseased tissue for given values of k_3 and k_1. Equation 29 may now be written as the relationship

$$x_{max} = (k_3/k_1)\,(\ln(1 - x_0) - \ln(1 - x_{max})). \tag{30}$$

If x_0 is very small then equation 30 rearranges to

$$x_{max} = 1 - \exp(-(k_1/k_3)x_{max}) \tag{31}$$

which is directly comparable to the relationship proposed by Vanderplank (10)

$$L = 1 - \exp(-i\,R\,L) \tag{32}$$

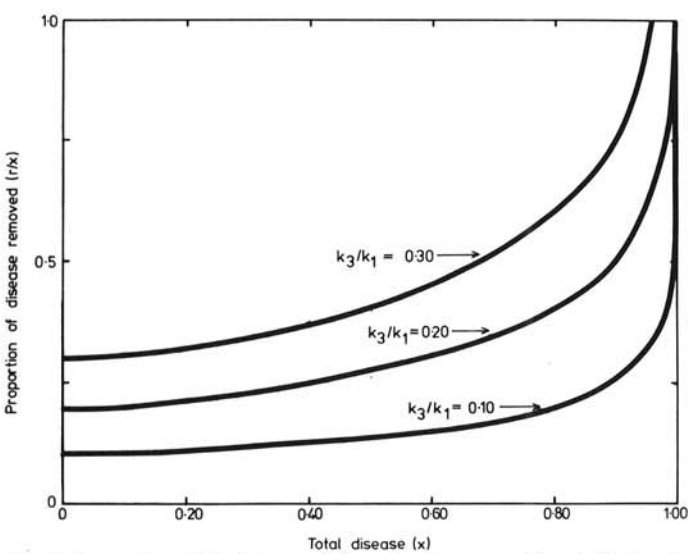

Fig. 5. Proportion of total diseased tissue that is removed (postinfectious) (r/x) as a function of total disease (x) for three values of k_3/k_1.

in which L is the maximum amount of diseased tissue. Vanderplank's derivation was by (presumably numerical) integration of the differential-difference equation (9,10). The analytical derivation here is also independent of the rate at which diseased tissue is progressing from the latent to infectious condition.

IMPLICATIONS OF THE MODEL

Any model, by itself, is incomplete; evidence is required showing how the proportion of total diseased tissue that is infectious or postinfectious varies over the time span of an epidemic, and how this is affected by weather conditions and human intervention.

For example, it has sometimes been noted that disease increase is unexpectedly fast on sprayed compared to unsprayed plants following the loss of fungicide effectiveness (3). This phenomenon is explicable in terms of the analysis given here. Suppose that disease is increasing at a given relative rate in two sets of plants. One set is sprayed with a protective fungicide and no new infections occur over the period of fungicide effectiveness; ie, $k_1 = 0$. Infections, however, are still progressing from the latent to the infectious condition. Hence, at the end of the period of fungicide effectiveness, the proportion of diseased tissue that is infectious (s/x) will be higher than at the beginning, if there has been no progression to the postinfectious condition. If the value of k_1 subsequently returns to the value prior to spraying, then by equation 16 the relative rate, $(1/x)\,dx/dt$, will of necessity be higher in the sprayed compared to the unsprayed plants at the same level of disease. If there has been progression to the postinfectious condition, then the result depends on the rates at which lesions are progressing from the latent to the infectious and from the infectious to the postinfectious condition. If the latter rate of progression of infectious host tissue to the noninfectious state is the lower of the two rates, then the proportion that is infectious will increase; if the higher, then the proportion that is infectious will decrease. If the rates are equal, then the proportion that is infectious will remain the same. This has obvious implications for the epidemiological action of fungicides. For example, it points to the inadvisability of a reduced spray program if only protective fungicides are available. Similar implications hold for any situation in which the dynamics and 'age structure' of disease have been perturbed (1).

The assessment of the proportion of diseased tissue that is infectious has rarely been attempted and, undoubtedly, will require much technical ingenuity. It is *not* sufficient to compare, say, the ratio of observed disease at times t and $t-p$—this merely presupposes the validity of equations 2 and 6. Similarly latent and infectious period distributions need to be established experimentally and compared with theoretical distributions. A start has recently been made by Shaner (6) with latent period distributions. It could well be that numerical methods are indispensable in further detailed work. The contention of this paper is that analytical approaches can achieve valuable, even if simplified, insights into this area of epidemic theory; insights that are not readily obtained by numerical methods alone. Arguably, this interplay between theory and experiment is still one of the most valuable uses of modeling for the epidemiologist.

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