Models for the Spread of Disease: Model Description

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ABSTRACT

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Some relationships between the spatial and temporal characteristics of epidemics are investigated with the aid of two models of plant disease spread. Based on these models, a new measure of the disease gradient (g) is proposed, which provides a simple link between the gradient steepness, the velocity of spread (v), and the apparent infection rate (r): Specifically, the relationship is $g = r/v$. It is shown that the disease gradient is not an unambiguous measure of host resistance, and a number of relationships are proposed between the components of resistance and the spatial dynamics of the pathogen population. For example, the gradient becomes steeper as the sporulation rate or infectious period increases, but eventually it becomes independent of both variables. The gradient is little affected by the latent period. The velocity of spread is linearly dependent on the standard deviation of the probability function describing spore dispersal.

The spatial spread of a pathogen population has long been recognized as a central process in plant disease epidemics. Much effort has been directed toward understanding the factors that affect dispersal, both from experimental (eg., 8,9,14,15) and theoretical (1,6,23) points of view, and experiments specifically designed to study the spatial properties of epidemics are now common (3,7,10). However, as indicated by Vanderplank (26), the integration of spatial concepts into temporal disease progress models has been difficult. Spatial effects have been included in a few simulation models (24,27), but we lack a coherent theory of disease spread that can be applied to practical disease control problems or used to guide research. The relationships between the disease gradient and the rate of spread, on the one hand, and the sporulation rate, latent period, and infectious period, on the other, remain largely unknown.

The examination of these relationships forms the focus of this paper. Our approach is based upon the work of Mollison (19,20) and others (5,11,12) on the mathematical theory of population spread. Our principal tool is a stochastic simulation model, whose structure reflects, in a simplified way, the main processes involved in the spread of disease. We use this model as a basis for operational definitions of the disease gradient and the velocity of spread, and show how these parameters are related through the apparent infection rate. With the aid of an analogous deterministic model, we have also investigated the qualitative effects of sporulation rate, latent and infectious periods, and spore dispersal on the spatial properties of epidemics.

THE SIMULATION MODEL

The conceptual structure of the model is straightforward. The model crop is a single row of identical plants, each of which can support a fixed maximum number of lesions. Each lesion, at some time after initiation, begins to release spores, which may land on the plant of origin or on some other plant in the row. After a time the lesion becomes senescent and ceases to produce spores. A spore that has landed on a plant may or may not produce a lesion there; the probability of that event depends on how many lesions are already present.

More detailed assumptions are necessary before the probability distributions associated with each of these events can be specified. Our intent has been to provide a broad characterization of the process of disease spread that can be clearly related to conventional concepts. We have, therefore, tried to keep our assumptions as simple as possible.

Three probability distributions are necessary:

The first is the distribution $h(n)$ of the total number (n) of daughter lesions produced by a parent lesion during its lifetime. In a constant environment at low population density, n has a Poisson distribution defined by

$$h(n) = \alpha^n e^{-\alpha}/n! \qquad (1)$$

in which α is the mean number of daughter lesions per parent lesion.

The second is the distribution $z(t)$ of the times at which daughter lesions are produced. A lesion initiated at time t_0 is considered to release spores at a constant rate between $t_0 + p$ and $t_0 + p + i$; here p is the latent period and i the infectious period, sensu Vanderplank (25). The times at which daughter lesions occur will then follow a uniform distribution:

$$z(t) = \begin{cases} i^{-1}, \text{ for } t_0 + p < t < t_0 + p + i \\ 0, \text{ for } t < t_0 + p \text{ or } t > t_0 + p + i. \end{cases} \qquad (2)$$

The third is the distribution $f(x)$ of the distance a spore travels before landing; this is the spore dispersal function. It is simplest to

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assume that as a spore travels through the airspace of a particular plant, it will land on that plant with probability 'a' (which is the same for all plants), and that spore dispersal is equally likely to occur in either direction from the source plant. If so, dispersal will follow a double geometric distribution:

$$f(x) = [a/(2-a)]\,(1-a)^{|x|} \tag{3}$$

in which $|x|$ is the absolute value of the distance (number of plants) from the plant of origin, and $f(x)$ is the probability that a spore will travel that distance before landing. This function is symmetrical about the plant of origin, and decays exponentially towards zero as the magnitude of x increases.

For realistic behavior at moderate to high population levels, some form of density dependence must be introduced to the model. Here we have made the assumption that population growth rate is modified according to the amount of uncolonized susceptible tissue remaining. Specifically, we define

$$Q_j = 1 - (y_j/K) \tag{4}$$

in which Q_j is the probability that a spore, having landed on plant j, has landed on uncolonized susceptible tissue; y_j is the number of lesions on plant j, and K is the maximum number of lesions that can occur on a plant.

The epidemic is begun with a single lesion on the leftmost plant in the line. As each lesion is initiated, the potential number of daughters it may eventually produce is chosen by taking a random sample from the distribution $h(n)$. For each of these daughters a location is chosen from $f(x)$, and an initiation time from $z(t)$. Since environmental effects are not included, all of these distributions remain constant throughout the simulation. When the simulation has advanced to the time that a daughter lesion is scheduled to occur, a new lesion is initiated, with probability Q, at the appropriate location. The cycle is repeated until disease has spread throughout the line of plants.

The behavior of the model is governed by a set of five parameters: a, p, i, a, and K. It will be convenient to express some results in terms of the auxiliary parameters

$$M = \alpha/i \tag{5}$$

and

$$\alpha^2 = 2(1-a)/a^2. \tag{6}$$

M is the mean number of offspring produced per infectious lesion per unit time at low population density; it is conceptually equivalent to the corrected basic infection rate (R_c) defined by Vanderplank (25, page 100). σ^2 is the variance of the spore dispersal function $f(x)$.

SIMULATION RESULTS

Under all conditions, the simulated lesion population was found to move as a wave of constant average shape and velocity. As expected, the velocity of this wave, and the steepness of the population gradient, depended on the values of the simulation parameters; the nature of the dependence, however, was surprising. Arguments have been advanced (17,26) to suggest that increasing the multiplication rate (M) or the infectious period (i) should cause the gradient to become flatter, but the behavior of the model was just the opposite (Fig. 1B and C). An increase in the latent period, which slows the epidemic and, therefore, might be expected to make the gradient steeper, had little effect (Fig. 1D). Because the gradient is affected by some components of resistance but not by others, its usefulness as a measure of resistance (16,17) seems limited.

When the spore dispersal gradient was made flatter (by increasing the variance of the spore dispersal function), the disease gradient also flattened (Fig. 2). The velocity of spread (measured as described below) was proportional to σ; this was shown by Mollison (19) for epidemics in which there are no delays or removals, and we found it continued to hold when such phenomena were included (Fig. 3).

To investigate these effects in more detail, it is necessary to define precisely what is meant by the gradient and the velocity of spread. Until this point we have been treating these as intuitive concepts; we will now use the simulation results to develop operational definitions of these terms.

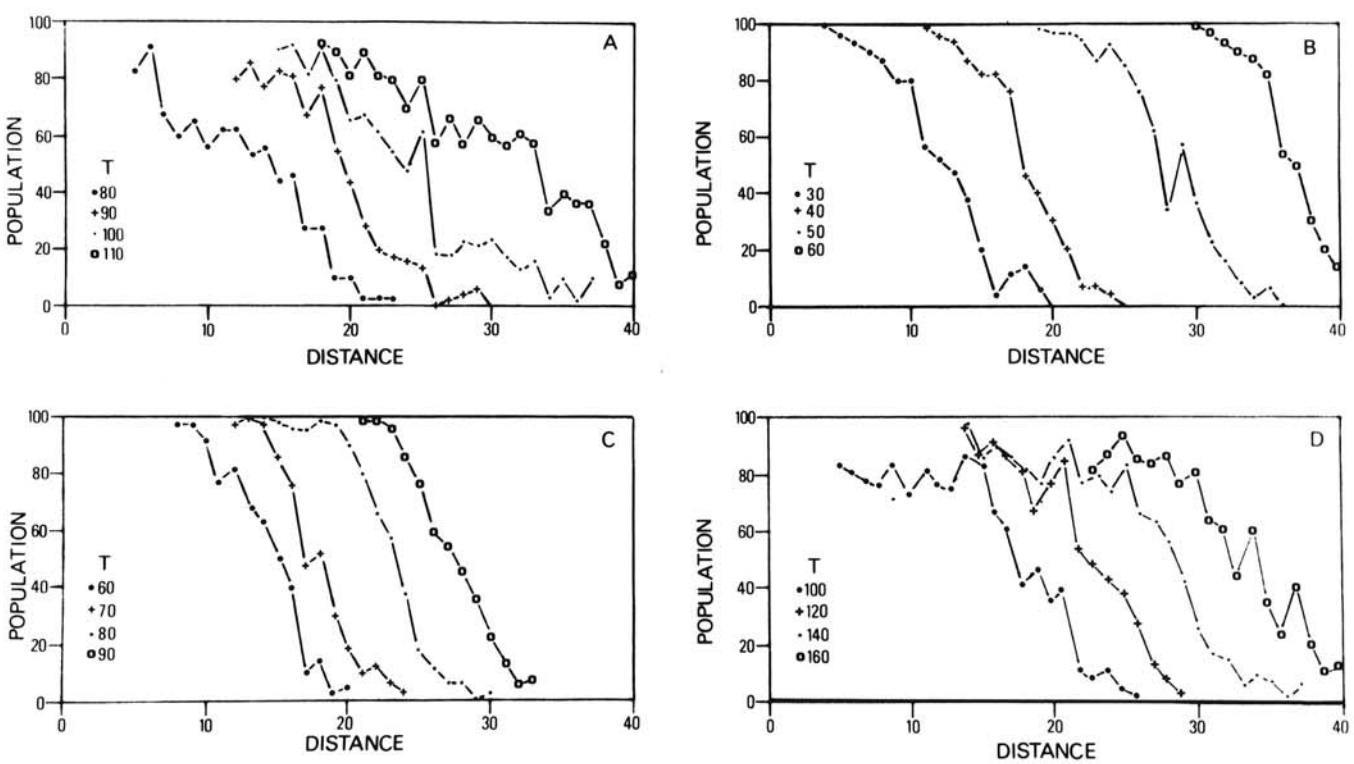

Fig. 1. Simulated population of lesions (as a percent of maximum) as a function of distance (number of plants) from the point of inoculation, for various times (T) after inoculation. Simulation parameters are: **A,** $M = 0.5, p = 3, i = 5$; **B,** $M = 1.0, p = 3, i = 5$; **C,** $M = 0.5, p = 3, i = 10$; **D,** $M = 0.5, p = 7, i = 5$. In all cases $\sigma = 2$, $K = 50$.

MEASUREMENT OF SPORE DISPERSAL GRADIENT AND VELOCITY OF SPREAD

The most widely used measures of gradient steepness are those first proposed by Gregory (7) and by Kiyosawa and Shiyomi (13). Gregory's parameter was derived for use with primary disease gradients, although it has often been applied to secondary gradients (3,17). Both measures do not allow for the nonlinear saturation phenomena that begin to appear when disease severity exceeds 10–20%, thus are applicable only at low disease levels. Attempts to apply either measure at disease severities above this level may produce misleading conclusions about the behavior of the disease gradient.

A more generally useful gradient parameter can be derived from the following considerations. Under constant conditions, the pathogen population moves as a stable wave at constant velocity. Moving at a velocity v for a time period Δt, the wave travels a distance Δs:

$$\Delta s = v\Delta t \tag{7}$$

Let $y(s_0,t)$ be the lesion population at location s_0 at time t. Because the velocity is constant and the shape of the wave is stable,

$$y(s_0 + v\Delta t,\ t + \Delta t) = y(s_0,t),$$

or, if $s = s_0 + v\Delta t$,

$$y(s,\ t + \Delta t) = y(s - v\Delta t,\ t). \tag{8}$$

By substituting equations 7 and 8 in the standard definition of the partial derivative, we get

$$\partial y(s,t)/\partial t = -v\partial y(s,t)/\partial s. \tag{9}$$

That is, the shape of y plotted against s is the same as the shape of y against t, except for the scale factor v. In the simulated epidemics, y increases approximately logistically with time (Fig. 4); if r is the local apparent infection rate (measured at location s, not over the whole plot), then

$$\partial y/\partial s = -(r/v)y(1-y). \tag{10}$$

We can expect, therefore, that the logit of y plotted against s should be a straight line of slope $-r/v$. This is the case for the simulated epidemics (Fig. 5), except near the ends of the plot where loss of spores results in a reduction of the local apparent infection rate.

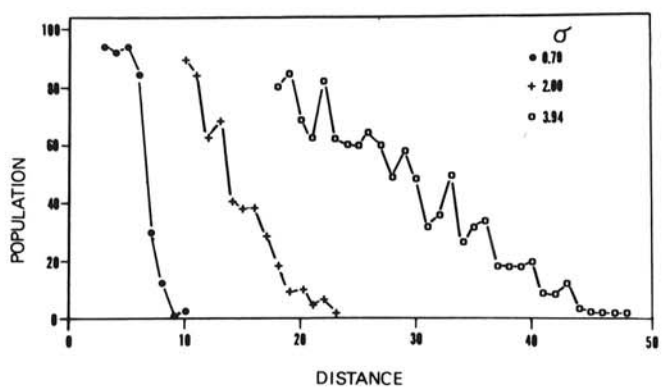

Fig. 2. Simulated population of lesions (as percent of maximum) as a function of distance from the point of inoculation, for various values of σ. Other parameters are the same as Fig. 1A. Time of observation is $T = 60$.

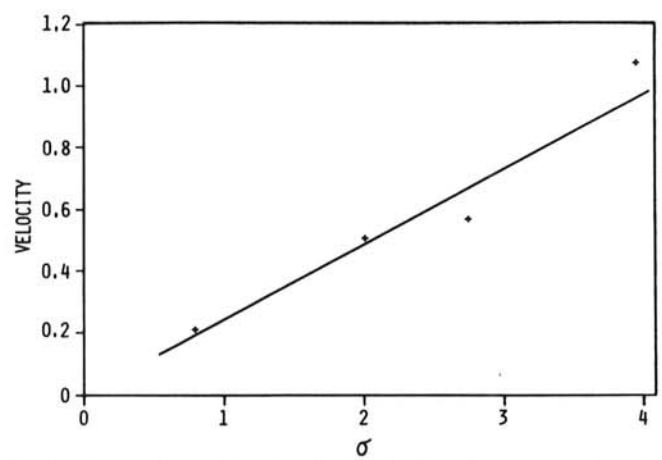

Fig. 3. Velocity of spread of the simulated epidemics as a function of σ. Other parameters are the same as Fig. 1A.

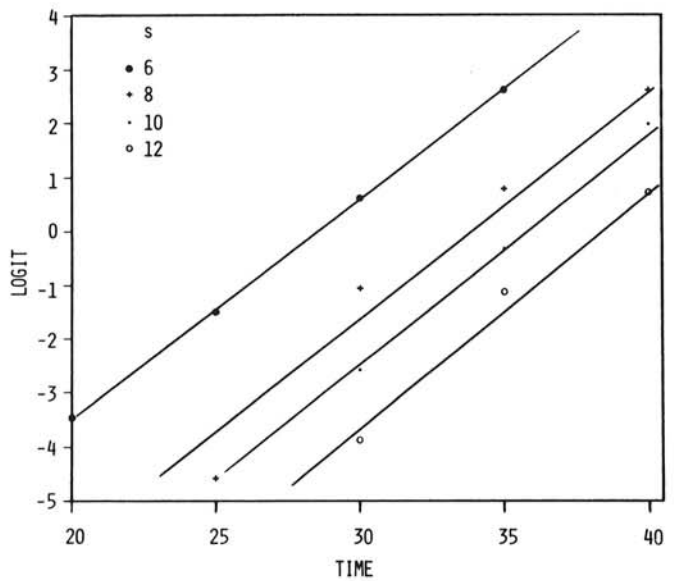

Fig. 4. Logit of simulated lesion population as a function of time since inoculation, at various distances (S) from the point of inoculation. Lines are the least squares regression of the logit on time, fitted with a common slope.

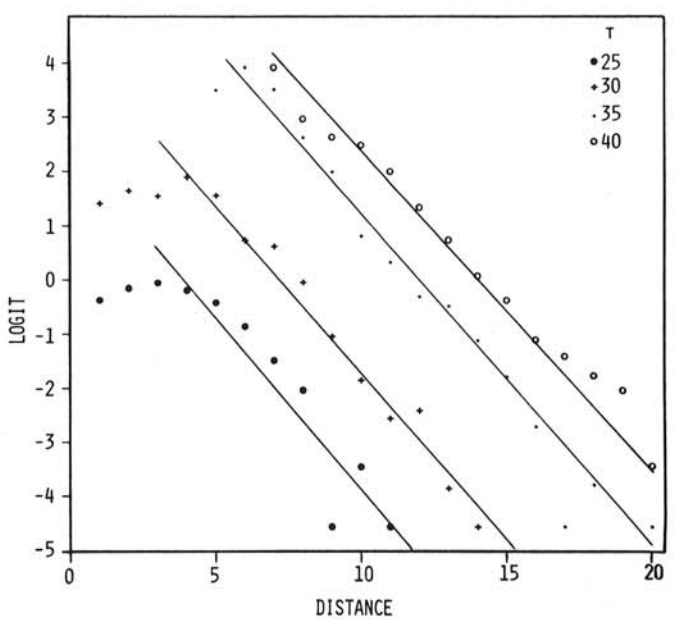

Fig. 5. Logit of simulated lesion population as a function of distance from the point of inoculation, for various times after (T) inoculation. Lines are the least squares regression of the logit on distance, fitted with a common slope.

We can use the relationship implied by equation 10 to define the gradient parameter

$$g = r/v, \tag{11}$$

which is dimensioned per unit length (eg, m^{-1}). This ratio is the amount by which the logit of disease severity falls in a unit distance. At low disease levels, g is equivalent to Kiyosawa and Shiyomi's b (13), but it remains applicable even at higher levels. The utility of the measure lies partly in this wider range of applicability, and partly in the simple and direct relationship it provides between the gradient, the velocity of spread, and the apparent infection rate. Its use depends, of course, on the adequacy of the logistic equation as a summary of the spatial and temporal characteristics of the epidemic. The results of field experiments to test its adequacy for modeling potato late blight are reported in another paper (18).

The parameters in equation 11 are defined operationally as least squares regression slopes: r is estimated by regressing logit (y) on time since inoculation, and g by regressing logit (y) on distance from the focal center. In practice, estimates are made with severity measurements pooled (4) for several locations (for r) or several times (for g). The regression on distance also provides estimates of s_{50}, the distance at which y falls to 50%; v is estimated by regressing s_{50} on time.

It should be pointed out that the definition of g in terms of the logistic function is largely a matter of convenience. In other circumstances, it may be appropriate to define g in terms of some other growth function. For example, one could set $g_G = k/v$, in which k is the rate parameter of the Gompertz function (2), or $g_W = 1/vb$, in which b is the scale parameter of the Weibull function (22). We have not investigated the conditions under which such definitions may prove useful.

DETERMINANTS OF SPATIAL BEHAVIOR

We have characterized the spatial behavior of an epidemic by the population parameters r, v, and g, but these parameters are ultimately determined by the processes of spore production and dispersal that occur at the individual level. We have discussed briefly the effects of M, p, i, and σ on the qualitative behavior of simulated epidemics; however, simulation of many combinations

TABLE 1. Velocity measured by simulation (v_m) and predicted by equation 15 (v_p)[a]

M	σ	p	i	v_m	v_p
0.5	2.00	3	5	0.506	0.616
1.0	2.00	3	5	0.887	0.922
0.5	2.00	5	5	0.439	0.441
0.5	2.00	7	5	0.330	0.344
0.5	2.00	10	5	0.175	0.260
0.5	2.00	3	10	0.562	0.696
0.5	0.79	3	5	0.215	0.253
0.5	2.74	3	5	0.570	0.841
0.5	3.94	3	5	1.074	1.208

[a] Abbreviations: M is the multiplication rate, σ is the standard deviation of the spore dispersal function, and p and i are the latent and infectious periods, respectively.

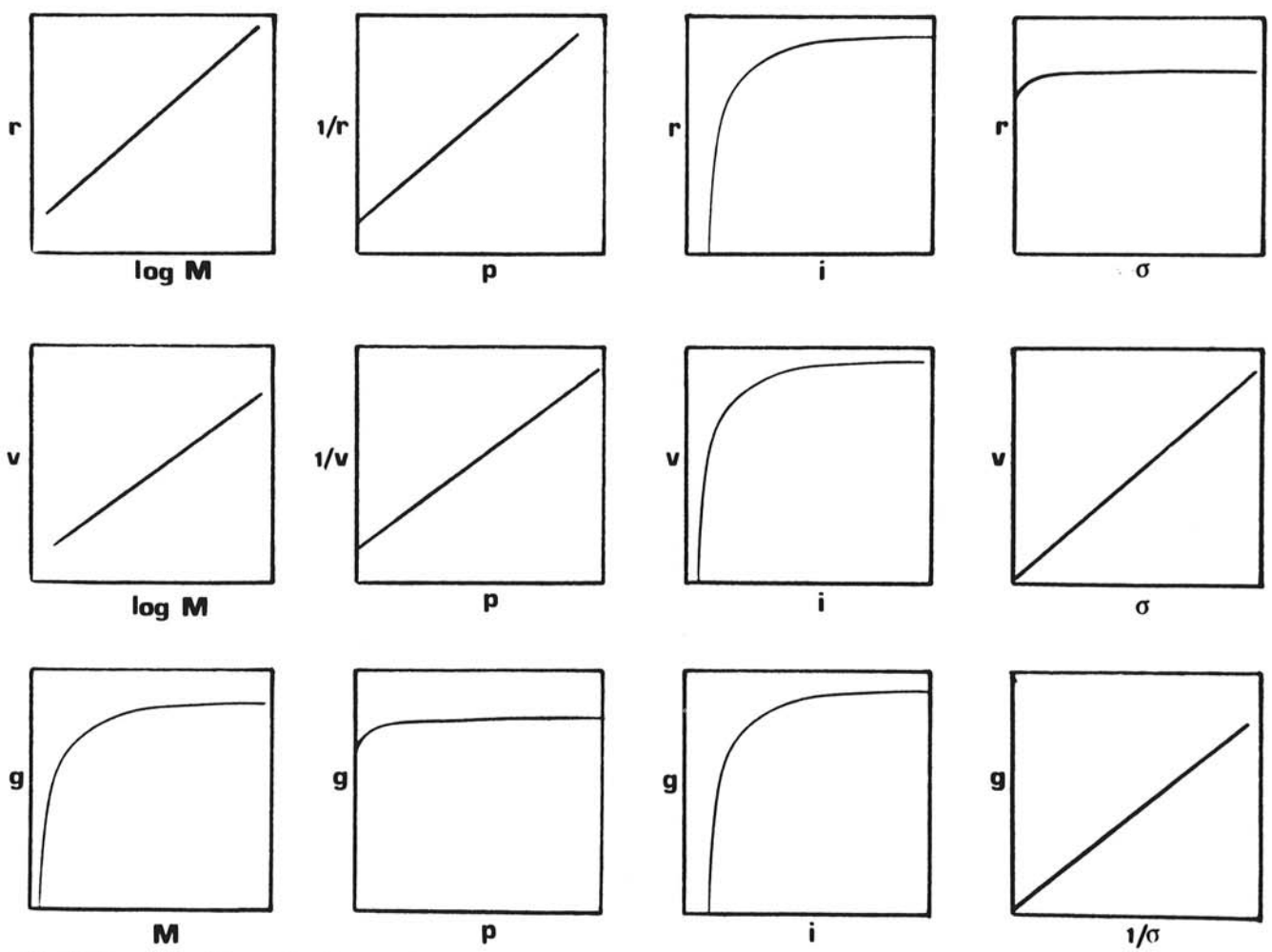

Fig. 6. Qualitative relationships predicted by equations 11 and 15 between population-level parameters (r, v, g) and individual-level parameters (M, p, i, σ) that determine spatial behavior.

of values of these parameters is expensive. Therefore, we have used an analogous deterministic model to help summarize these effects.

The model is a simple extension of Vanderplank's (25, equation 8.3) general epidemic model

$$dy(t)/dt = R_c[1-y(t)]\,[y(t-p) - y(t-p-i)] \qquad (12)$$

R_c is the corrected basic infection rate (equivalent to our M), $y(t)$ is the proportion of disease at time t, and p and i are the latent and infectious periods, respectively. To convert this to a spatial model it is only necessary to replace the term $[y(t-p) - y(t-p-i)]$, which is the proportion of infectious lesions, with a weighted average of the infectious lesions around the location s. Lesions at location $(s-x)$ exert an influence on the rate of infection at s through spore dispersal; in determining this rate of infection it is therefore appropriate to weight the amount of disease at $(s-x)$ by the probability that a spore will travel the distance x, which is given by the spore dispersal function $f(x)$. The spatial version of the model is obtained by summing over all values of x:

$$dy(s,t)/dt = M[1-y(s,t)]\,\Sigma_{x=-\infty}^{\infty}\,[y(s-x,\,t-p) - y(s-x,\,t-p-i)]. \qquad (13)$$

Although explicit solution of this equation does not appear to be possible, some useful insight can be gained by considering the results of the previous section. It was shown that the behavior of the simulation model can be described in space and time by the logistic function. At low disease levels (near the leading edge of the advancing wave), the logistic function reduces to the exponential:

$$y(s,t) = y(0,0)e^{-gs}e^{rt} \qquad (14)$$

with g and r defined as before. Substitution of equation 14 into equation 13 (again assuming low disease levels, so that the term $[1-y(s,t)]$ can be dropped) shows that it is a solution of equation 13, with the following relationship among the parameters:

$$M\psi(g) = re^{pr}/(1-e^{-ir}) \qquad (15)$$

$\psi(g)$ is the moment generating function of the distribution $f(x)$, evaluated at g. Such functions are extensively tabulated for a wide variety of distributions (eg, 21).

Equation 15, in slightly rearranged form, is Vanderplank's (25) equation 8.5, modified to include spatial effects. It provides, in effect, an hypothesis about the relationships we can expect to find between the descriptors of individual behavior (M, p, i, and σ) and the descriptors of population behavior (r and g). The equation has been derived for low population levels, but clearly all of the parameters involved remain constant as long as the population continues to behave logistically; this is true because r and g are defined in terms of the logistic equation, and we have not changed their definitions in assuming low population levels. The equation is therefore useful over the entire range of conditions for which the logit plots can be considered straight.

The qualitative relationships predicted by equation 15 are shown diagrammatically in Fig. 6. This equation does not fix the values of r and g; to do so has required the ad hoc assumption that, among all the values of r and g satisfying equation 15, the realized values will be those that minimize the velocity of spread, as calculated by equation 11. This is suggested by the results of similar models (12,19), and is justified by the fact that simulated epidemics seem to propagate at or slightly below this minimum velocity (Table 1).

The hypotheses generated from the results of the simulation model are elaborated in Fig. 6; v is linearly related to σ, g increases with increasing M or i and is nearly unaffected by p. It is also clear that g rapidly becomes independent of M and i as these parameters increase, indicating again that g is not a measure of resistance. The parameters r and v, which can be seen as complementary measures of the rate of epidemic progress, differ only in their response to σ. The lack of response of r to σ suggests that spatial factors may be less important than expected under some conditions. (It is important to reemphasize that r refers to the apparent infection rate measured at a specific location s within the plot, not to the infection rate for the plot as a whole).

DISCUSSION

This research has generated two sets of hypotheses about behavior of a spreading pathogen population, derived from the stochastic and deterministic models described above.

i) Under constant conditions, the population is expected to spread as a wave at constant velocity. The properties of this wave can be described in terms of the logistic function, which permits useful definitions of the gradient and velocity parameters. These parameters are related to the apparent infection rate by equation 11: $g = r/v$.

ii) Qualitatively, the relationships between individual- and population-level parameters are described by equation 15 and Fig. 6.

Field experiments to test these hypotheses are reported in another paper (18). Because of the simplified nature of our models, we cannot make testable quantitative predictions; our approach is instead to verify the utility of the population parameters we have defined, and to show that they are related according to equation 11. We have also manipulated parameters by the use of fungicides and resistant cultivars, to investigate some features of Fig. 6.

We believe the concepts developed in this paper should provide a useful framework for the experimental investigation of the spread of plant disease. Equations 11 and 15 embody a unified view of the spatial and temporal development of epidemics, and provide a logical basis for the design of experiments. Although the derivation of equation 15 cannot be considered mathematically rigorous, the equation has proven to be a fertile source of hypotheses, and an investigation of its properties provide an illuminating excursion through the modes of behavior that might be expected of a spreading pathogen population. Besides the variety of predictions diagrammed in Fig. 6, the equation can be readily extended to cover asymmetric spore dispersal functions (in which case the solution yields two gradients and two velocities, corresponding to "upwind" and "downwind" spread), more realistic patterns of spore production (which do not involve constant sporulation rates and fixed latent and infectious periods), and spread through two dimensions. This last case is of particular interest since it is not clear to what extent our one-dimensional results can be applied to spread over a plane. A major difficulty is encountered in adequately generalizing the parametric description of the gradient from one to two dimensions. Much more theoretical and experimental work needs to be done to clarify questions such as these.

The wavelike behavior apparent in Fig. 1 provides the basis for most of the concepts discussed in this paper. For such a wave to be discernible, the scale of observation must be large enough to take in a large part of the wave. On a fine scale, especially at the leading edge of the advancing wave, stochastic effects predominate: spread of the pathogen is more usefully viewed as a series of discontinuous jumps, followed by focal formation and secondary spread, than as a smoothly propagating wave. In principle, such behavior can be investigated using the stochastic model we have presented; in practice, such investigations are of limited usefulness in the absence of appropriate unifying concepts. We see the development of such concepts as a priority for future research.

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