Letter to the Editor

Threshold Criteria for Model Plant Disease Epidemics. II. Persistence and Endemicity

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Onstad and Kornkven (9) defined endemicity (of a plant disease) as "the persistence or constant presence of a pathogen in an ecologically proper spatial unit over many generations." The constant presence of a pathogen was effectively taken to mean a nonzero density of infected (i.e., latent plus infectious disease), but not postinfectious, plant tissue (or plant units) over the time scale of model simulations performed with a supercomputer. This definition appears conceptually novel and potentially useful, and although the authors disavow an equilibrium approach, there are clearly long-term properties of persistence to consider.

Persistence was positively associated with potential reproduction per pathogen (the product, iR, in which i is the length of the infectious period and R is the rate parameter dimensioned plants per infectious plant per unit time, not as defined by the authors) and to increase with the sum, $i + p$ (in which p is the length of the latent period). Growth of host tissue increased the likelihood of persistence as did variations in absolute host density and in the value of iR within a particular simulation. Introduction of a spatial scale was considered essential for deriving theorems on persistence or endemicity, although the authors produced no such theorem themselves.

In a subsequent paper, Onstad (8) concluded that total disease (latent plus infectious plus postinfectious) increased under all scenarios whatever the values for host density and iR and, thus, by implication that no threshold exists in terms of disease. This problem is discussed in detail in the accompanying paper (6). Onstad argued compellingly that infected tissue (as defined above) rather than diseased tissue provides the best indicator variable for analyzing epidemic dynamics. Further probabilistic aspects of epidemic spread, not directly relevant to the present letter, also are considered in Onstad (8).

In this letter, the system of equations proposed by Onstad and Kornkven (9) is analyzed to determine qualitative properties of endemicity as it is defined. Most results can be obtained without recourse to a supercomputer, in particular the relationship between model parameters and endemicity.

THE MODEL

The model proposed by Onstad and Kornkven (9) consists of four linked differential equations:

$$\left.\begin{aligned} dS/dt &= b - RI(S/N) \\ dL/dt &= RI(S/N) - L/p \\ dI/dt &= L/p - I/i \\ dD/dt &= I/i \end{aligned}\right\} \quad (1)$$

in which L, I, and D are the densities ("the number of leaflets per plant site") of latent, infectious, and postinfectious diseased tissue within a constant spatial area; S is the density of susceptible tissue; R, i, and p are as defined above and have essentially the same meaning as in Vanderplank's differential-delay equation (5,6,11); b is a parameter describing host growth (assumed positive); and N is total plant tissue density with $N = S + L + I + D$. It follows that dN/dt, the rate of change in total plant tissue density, is equal to the constant parameter, b, and, hence, unrealistically, that N increases linearly, with slope b, from an initial population, N_0. Rather than "plant tissue density," we use the term "population" throughout. We note that the increase in newly infected tissue is proportional to the relative rather than absolute density of susceptibles in the population, an assumption that affects epidemic dynamics (4); in general, no absolute population density (N_T) exists such that infecteds only increase if $S > N_T$.

Analysis. The qualitative dynamics of equation 1 can be obtained by examining properties of particular trajectories. In particular, following the definition of persistence proposed by Onstad and Kornkven (6), we can state that persistence occurs provided $L + I$ ("infecteds") remains bound away from zero in the long run. It is possible that infecteds may cycle either regularly or irregularly without going to zero, but the possibility is not investigated here. We constructed differential equations for the new variables ($L + I$) and the proportion of the total population that is susceptible (S/N), set these equations to zero, and solved for the equilibrium values of each variable. We examined the consequences for the original variables, L, I, D, and S, both as absolute values and as proportions of the total population, N.

Considering infecteds,

$$d(L + I)/dt = dL/dt + dI/dt = RI(S/N) - I/i. \quad (2)$$

Setting equation 2 to zero implies by simple algebra that $(S/N) = (iR)^{-1}$. Thus, if $L + I$ approaches a constant value, so too does S/N, even though N is not a constant but increases linearly. It then follows that the susceptible population must also increase linearly if infecteds persist at an equilibrium value, because N increases at a constant rate, b. Because S must be less than N, we immediately have the condition $iR > 1$ if disease persists. We note that this threshold condition is exactly that for the Vanderplank differential-delay equation (11) as discussed in the accompanying paper (6) and is a necessary condition in a situation in which the host population is increasing, demonstrating the flexibility of linked differential equations (5).

Using the quotient rule, the proportion of the population that is susceptible is

$$d(S/N)/dt = [(N dS/dt) - (S dN/dt)]/N^2. \quad (3)$$

Setting equation 3 to zero implies that $dS/dt = (S/N)\, dN/dt$. Substituting in the expressions for S/N (i.e., iR^{-1}) and dN/dt

$(=b)$ gives $dS/dt = b/iR$, which is always positive if $b > 0$. Thus, if disease persists, it follows that the rate of increase in susceptible tissue, S, approaches the constant and positive value b/iR, which is less than b.

Substituting in the equation for dS/dt from equation 1 and substituting in $S/N = (iR)^{-1}$ gives

$$b - I/i = b/iR.$$

Rearranging in terms of I gives the constant value

$$I^* = (b/R)(iR - 1) \quad (4)$$

in which again $iR > 1$, and the superscript indicates an equilibrium value. Also, b must be positive and greater than zero.

If I approaches the constant value I^*, and if $L + I$ approaches a constant value (the condition for persistence), it follows that L must also approach the constant value

$$L^* = (pb/iR)\,(iR - 1)$$

and thus

$$(I + L)^* = (b/iR)(iR - 1)(i + p), \quad (5)$$

again stressing the two criteria, $iR > 1$ and $b > 0$, for persistence. For large values of iR, $(I + L)^*$ approaches the limit $b(i + p)$.

We now derive an expression for each of the remaining categories of disease. The solution for N, the total population, is simply $N = N_0 + bt$, in which N_0 $(=S_0 + L_0 + I_0 + D_0)$ is the initial population at $t = 0$ (the authors specify $D_0 = 0$). In the long term, as S/N approaches the constant value $(iR)^{-1}$ we have

$$S = N(S/N) = (N_0 + bt)/iR \quad (6)$$

and by substitution

$$D = N - (S + L + I) = (iR - 1)[N_0 + b(t - i - p)]/iR. \quad (7)$$

S and D are explicitly time-dependent and do not approach equilibrium values.

Thus, the total amount of disease is

$$L + I + D = (iR - 1)(N_0 + bt)/iR$$

giving the asymptotic proportion of disease as

$$(L + I + D)/N = (iR - 1)/iR \quad (8)$$

with $iR > 1$. As noted above, persistence is not possible when $iR < 1$.

The simplicity of this asymptotic result (with host growth) is marked by comparison with the results (without host growth) obtained in the accompanying paper (6) but is a possible consequence of the simple model of linear host growth.

The infecteds population can be expressed as a proportion of the total population:

$$(I + L)/N = [(b/iR)(iR - 1)(i + p)/(N_0 + bt)]. \quad (9)$$

This proportion approaches zero for large values of t (which appears in the numerator), even though the denominator, the absolute number of infecteds (equation 5), approaches a constant nonzero value. As a check, it can be seen readily that the sum of S/N $(=iR^{-1})$ and equation 8 equals 1 (equation 8 in fact can be derived simply by substracting iR^{-1} from 1).

A numerical solution of equation 1 is shown in Figure 1 and illustrates the basic features of the analysis. After about 100 days, the increases in D are effectively linear, as given in equation 7. The amount of susceptible tissue initially falls sharply to a minimum and then increases, eventually linearly, as given by equation 6. Both I and L increase to maxima early in the epidemic and then decrease to approach the constant values given by equations 4 and 5. As t increases, D dominates the total population, N, and the proportion of infecteds, $(I + L)/N$ (equation 9), rapidly approaches zero. A further numerical example (Fig. 2) shows that the approach to linearity for D and S can be cyclical with dampening waves.

Invasion criteria. We also note that the $iR > 1$ criterion that is required for infecteds to persist can be derived from an invasion argument. The rate of change of infecteds is given by equation 2. If disease can invade into the population from an initially very small amount and if $b > 0$, then disease will be endemic. For very small initial amounts of infecteds, we have $S \cong N$, so

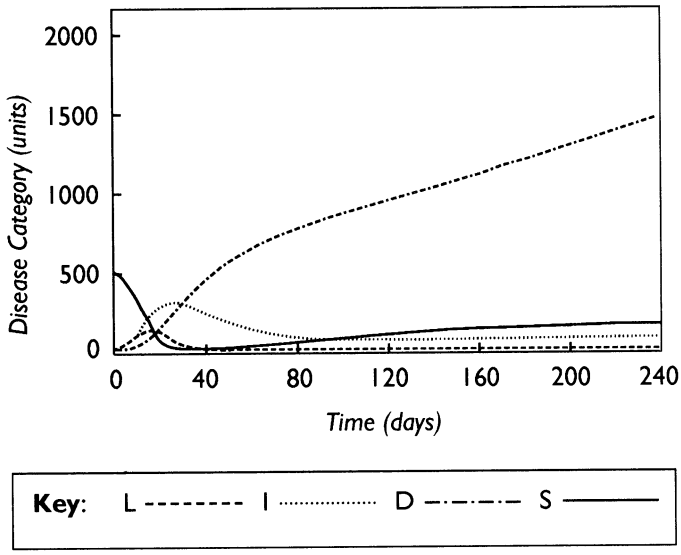

Fig. 1. Disease progress (in absolute units) with time (days) according to equation 1: *L, I, D,* and *S* represent densities of latent, infectious, postinfectious, and susceptible tissue, respectively. Numerical solutions were obtained with Runge-Kutta algorithms and a time step of 1 day; initial values of *L, I, D,* and *S* were 20, 20, 20, and 500 units, respectively; parameter values were $R = 0.5$ day^{-1}, $p = 5$ days, $i = 20$ days, and $b = 5$ day^{-1}.

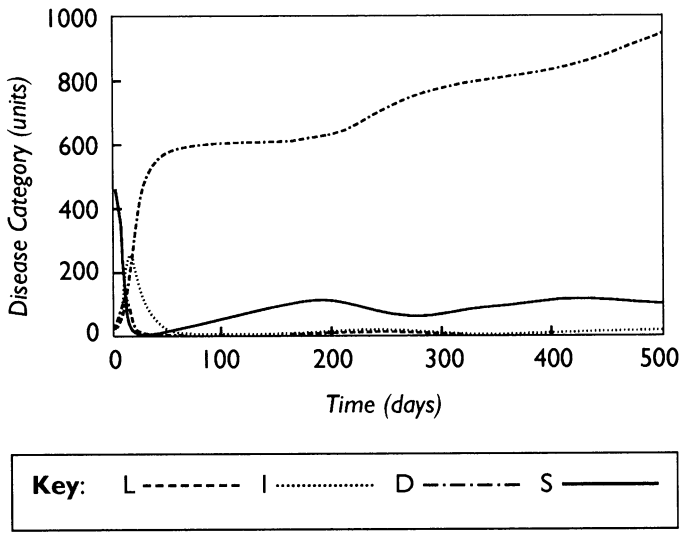

Fig. 2. Disease progress (in absolute units) with time (days) according to equation 1: *L, I, D,* and *S* represent densities of latent, infectious, postinfectious, and susceptible tissue, respectively. Numerical solutions were obtained with Runge-Kutta algorithms and a time step of 1 day; initial values of *L, I, D,* and *S* were 20, 20, 20, and 500 units, respectively; parameter values were $R = 1.0$ day^{-1}, $p = 3$ days, $i = 10$ days, and $b = 1$ day^{-1}.

that $d(L + I)/dt \cong (R - 1/i)I$. The population of infecteds ($I + L$) will increase if $R - 1/i > 0$, i.e. if $iR > 1$.

For the spatially structured model of Onstad and Kornkven (9) (with j compartments and in which disease in each compartment is related to disease in the surrounding eight compartments), the same analysis holds for $Sj = Nj$. The rate of change of infecteds is summed over all compartments, j, to give

$$d \Sigma_j(I_j + L_j)/dt = (R - 1/i) \Sigma_j I_j, \quad (10)$$

and this leads to the same threshold criterion.

DISCUSSION

The model proposed by Onstad and Kornkven (9) (equation 1), although complex, has quite simple dynamics when restricted to outcomes in which disease persists as defined. We imposed the simple condition that the number of infecteds ($I + L$) is constant or approaches a constant positive value, implying that the proportion of susceptibles (S/N) approaches the constant value $(iR)^{-1}$, which in turn implies that $iR > 1$. Similarly, the persistence of disease implies that the growth rate, b, is greater than zero. Equation 5 encapsulates several of the major conclusions drawn from Onstad and Kornkven's original simulations (8,9). The degree of persistence (higher values of $I + L$) is directly related to values of growth rate (b) and to length of the infection cycle ($i + p$). For persistence to occur, iR must be greater than one and the higher the value of iR the larger the persisting densities of infecteds.

The model also includes a provision for host growth and effectively follows the procedure described by Jeger (5), in which host growth is combined with an epidemiological model describing the different disease categories, although the simple linear form in equation 1 is not generally tenable as a biological property. As might be expected, continuing host growth has a marked effect in enabling disease to persist rather than become locally extinct. Also, because the overall increase in the total host tissue is linear, the only end result for an infected leaf is to become a removed leaf, whereas some additional mortality might be expected. Moreover, no qualitative analysis of the effects of introducing additional growth and mortality factors was made. An approach that can be followed in these respects is outlined by Chan and Jeger (3). Introduction of more realistic representations of host growth in Vanderplank's differential-delay equation can lead to decreased asymptotes (2), but the linked differential equation approach offers more flexibility and tractibility. This was clearly demonstrated by the simple derivation of the epidemic threshold criterion in this paper, a criterion identical to that derived from Vanderplank's differential-delay equation in the accompanying paper (6), which holds under conditions in which the host population increases linearly. However, when other forms of host growth (including mortality and replanting) are included, other parameters enter into the threshold criterion (3).

The other two factors considered by Onstad and Kornkven (9) were spatial dynamics and heterogeneity. Because plant populations are generally fixed positionally (except for natural or introduced weed species that may be expanding their range), spatial effects in plant disease epidemics may be expected to be important. Theoretically, the asymptotic result established for Kermack and McKendrick-type equations can be generalized to include spatial (diffusion) effects, and an equivalent threshold for a pandemic is obtained (1). Onstad and Kornkven (9) introduced a spatial component into their linked differential equation model by dispersing inoculum from a spatial unit, according to the product RI (defined above), to its eight neighbors with a simple proportionality rule. Because of this model structure, only numerical analysis of spatial effects was possible. The definition of spatial scale was reported to affect the persistence of disease, but comparisons with disease dynamics defined on a purely temporal scale were not made. Techniques for modeling spatial spread of plant disease, without the confining spatial grid representation used by Onstad and Kornkven (9), are now well established (10).

May (7) looked at the effects of heterogeneity on the final size of an epidemic through the variation in distribution of spatial position across a plant population. As the coefficient of variation of this distribution increased, there were large effects on the asymptotic proportion of disease, especially at large values of the basic reproductive rate—equivalent to the iR of this paper. Onstad and Kornkven (9) took a different approach; rather than looking at the effects of heterogeneity in plant populations on epidemic processes, they looked at spatial variation in values (two) of iR in a simple fashion. They concluded that heterogeneity increased the likelihood of persistence compared with use of average values. This interesting result is of obvious relevance to studies of disease in mixtures of different host genotypes and suggests that in a closed system such mixtures may be counterproductive, unless the long-term "persistent" disease level is suitably low. Considerably more research is needed to understand the dynamics of disease in heterogeneous populations, and modeling should play a key role.

Numerical simulation approaches will rarely, however, allow claims (such as "always") to be made on the effects of heterogeneity. Further analytical modeling of stochastic effects, such as those imposed by heterogeneity, are needed and provide, as noted by Jeger (5), a major challenge to quantitative epidemiologists.

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