Evaluation of Epidemiological Thresholds and Asymptotes with Variable Plant Densities

David W. Onstad

University of Illinois and Illinois Natural History Survey, 607 E. Peabody Dr., Champaign, IL 61820.

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ABSTRACT

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Simulations of a simple model of the temporal and spatial dynamics of a hypothetical pathogen demonstrated some limitations of Van der Plank's threshold formula, $iR > 1$. The threshold value of iR (total potential reproduction per pathogen) that determines whether the density of infected (latent and infectious) leaflets will increase over a pathogen's generation increased as the initial density of susceptible hosts decreased. In model simulations, levels of disease (sum of latent, infectious, and removed lesions) increased under all scenarios with various values of host density and iR. Thus, disease levels cannot be used in hypotheses to predict the population dynamics of the pathogen. Probabilistic formulas were developed as proposed thresholds for predicting the change in the number of infected hosts over a pathogen generation. These formulas are based on the proportion of a region occupied by hosts, the initial proportion of hosts that are infected, and the number of sites within the propagule dispersal neighborhood. The formulas of Van der Plank and Jeger that describe the asymptotic proportion of diseased tissue generally failed to match the simulated results because of the simplicity of the formulas' underlying model and assumptions.

A traditional practice in theoretical plant epidemiology has been the expression of hypotheses in simple mathematical formulas. Some of these formulas are used to predict the final or asymptotic level of diseased host tissue (6,18). Other formulas are used to predict changes or the lack of changes in the level of diseased tissue. For example, Van der Plank (16) postulated that total potential reproduction per pathogen, iR, must be greater than one for an epidemic to occur.

These hypotheses were adequate at the time of their development, but now must be investigated critically from several points of view. First, none of the hypotheses includes temporal and spatial scales that allow them to be tested or implemented (12). Second, no one has questioned the use of diseased tissue as the indicator variable in these formulas. Does this variable permit implementation of the hypotheses in disease management? Do the formulas actually predict changes in this indicator variable as the hypotheses suggest? Third, the influence of host density has never been included in these hypotheses. Hypotheses should now consider host density so that predictions can be made for many natural and managed situations in which host tissue does not uniformly cover space.

This paper addresses the issues concerning proper indicator variables and the influence of host density. Because of the complexity of the problem, a numerical model was computed to study the temporal and spatial dynamics of a hypothetical pathosystem in a heterogeneous environment (11). Although other plant pathologists have modeled the temporal and spatial dynamics of real or hypothetical pathogens, they have not addressed the issues of thresholds and asymptotes (7–10). Most often, the emphasis was on the study of dispersal gradients, focus spread, and disease (not pathogen) progress (20). Fleming et al (4) demonstrated how host field size and shape could influence the increase and persistence of pathogens, but they did not provide a temporal scale in their general conclusions, and their fields consisted of homogeneously distributed and nonlimiting susceptible tissue.

MATERIALS AND METHODS

The pathosystem described below is similar to the basic systems studied by Van der Plank (16,17) and Zadoks (19). It has one host species and one strain of pathogen that infects the host's leaves. The host's phenology does not influence its interaction with the pathogen, and the pathogen cannot kill the host. Climatic and seasonal variations in the environment do not affect the generation times of the host and pathogen.

Onstad and Kornkven (13) described the pathosystem with four differential equations. In these equations, S represents susceptible host tissue (leaflets per plant site), N symbolizes total host tissue (leaflets per plant site), and L, I, and D are the densities of leaflets with latent, infectious, and removed lesions (leaflets per plant site), respectively. The density of infected but not yet infectious lesions is L. For simplicity, I assume that a lesion covers a single small leaflet by the end of the latent period.

$$\frac{dL_{ij}}{dt} = 0.2RI_{ij}(S_{ij}/N_{ij}) + \sum_{k=1}^{8} 0.1RI_{ik}(S_{ij}/N_{ij}) - L_{ij}/p \quad (1)$$

$$\frac{dI_{ij}}{dt} = L_{ij}/p - I_{ij}/i \quad (2)$$

$$\frac{dD_{ij}}{dt} = I_{ij}/i \quad (3)$$

$$\frac{dS_{ij}}{dt} = b - 0.2RI_{ij}(S_{ij}/N_{ij}) - \sum_{k=1}^{8} 0.1RI_{ik}(S_{ij}/N_{ij}) \quad (4)$$

with $RI_{ij}(S_{ij}/N_{ij}) \leq S_{ij}$. The total amount of host tissue N is constant when $b = 0$. Because N equals $S + L + I + D$, S/N is equivalent to $1 - (L + I + D)/N$. The latent period is p, and the infectious period is i. Both periods are expressed in days. R is the potential reproductive rate in terms of new inoculum

per infectious lesion per day. If this inoculum lands on susceptible tissue, then it will produce infected leaflets or latent lesions. On the average, a new lesion requires p days to completely cover its leaflet. The variable L is part of the infected tissue that reduces reproduction because when two or more propagules land on a latent leaflet, the leaflet will still be completely covered p days after germination of the first propagule. The final inequality expressed above prevents the amount of newly formed latent leaflets from exceeding the amount of available susceptible tissue.

To model the dynamics of the pathogen in space, I assigned the four state variables calculated by Equations 1–4 to each site in a very large two-dimensional grid. Some or all of the sites may be occupied by a host plant. Thus, the distribution of hosts is not necessarily uniform. Inoculum is dispersed among plants by assigning one-fifth of RI_t calculated at site j to site j and one-tenth to each of the eight adjacent sites. Propagules landing on nonhost sites are lost. Those landing on host plants survive and germinate with probability S_k/N_k for plant k. Hosts on the edge of the grid communicate with and are assumed to be adjacent to the sites on the opposite edge. No inoculum is dispersed out of the region.

In Equations 1 and 4, the relative density of susceptible leaflets, S/N, is used to represent the probability of survival and germination during dispersal and infection. As Van der Plank (16) mentioned, the use of S/N in a single-equation model is correct only if the susceptible hosts are homogeneous throughout a field. The use of absolute density is important when the plant canopy does not provide a uniform and complete cover of the land, such as in young crops and natural habitats, or when nonhost species are abundant. I modeled the absolute density of hosts in the model by changing the total density for the entire region.

The model was programmed in FORTRAN and computed on the Connection Machine 2 at the National Center for Supercomputing Applications at the University of Illinois. This massively parallel computer has 32,768 processors, but I only used one-quarter of the machine. These 8,192 processors were modeled as a 64 × 128 grid. Each simulation of 1,000 daily time steps with Euler integration required 2 min on the computer.

To discover which factors lead to increases in the pathogen population in the host population, I varied host density in the region and the value of iR. The number of host sites with $N_{0j} > 0$ among the 8,192 possible sites was 8,192, 3,000, 1,000, or 300. When the number of host sites was less than 8,192 (the uniform distribution), the hosts were randomly distributed across the 8,192 sites. All host sites had the same initial value of $N_{0j} = 50$. After assigning the N_{0j}, the initial values of L_{0j} and I_{0j} were randomly allocated to 1% of the hosts. For these hosts, $L_{0j} = 3$ and $I_{0j} = 2$. The initial value of D was always zero. After allocating L_{0j} and I_{0j}, the S_{0j} were calculated as the remainder of the N_{0j}. Host growth rate was zero. At least four replications were made of each simulation so that the host and pathogen were adequately mixed. If necessary, a fifth replication was made to reduce the coefficient of variation to approximately 10% of the mean for the final values of several output variables.

For the simulations of 8,192, 1,000, and 300 hosts, only two values of iR were used: 1 and 5. With 3,000 hosts, iR equaled 0.5, 1.0, 2.3, and 5.0. The latent and infectious periods, p and i, were combined in three sets in which p and i were equal (2, 10, or 20 days). I analyzed the following variables in each simulation: regional counts of hosts with densities of L, I, and D greater than one-thousandth of a leaflet; and sums of leaflets S, $L + I$, $L + I + D$, and $I + D$ for the entire region. A variety of measures were used to avoid the problem identified by Burdon and Chilvers (3) of relying on only one variable for identification of relationships.

When hosts are randomly distributed and initial infections are independently and randomly assigned as in this study, simple formulas can be developed to describe the likelihood of a pathogen spreading from an infected host to a susceptible one. Perhaps the simplest estimate is the probability, $P1$, of finding a site that is part of a cluster of two or more hosts. This value is calculated by subtracting the sum of the probabilities of finding only zero or single clusters (i.e., sites with no host or with an isolated host) from 1.0. Thus, $P1 = 1 - ([1 - f] + f[1 - f]^r)$ is the formula based on f, the initial proportion of the region occupied by the host and r, the number of sites surrounding an infected host that receive dispersed propagules.

A more appropriate formula is based on a binomial distribution in which the occupation status of a site is a trial, and the occupation of that site by a host is a success. The probability that a site is surrounded by at least one healthy host in the neighborhood of propagule dispersal is

$$P2 = \sum_{h=1}^{r} f^h (1-f)^{r-h} (1 - y^h)\, r! \,/\, (h!\,(r-h)!) \qquad (5)$$

in which f, y, and r are the proportion of the region occupied by the host, the initial proportion of hosts infected, and the number of sites surrounding an infected host that receive dispersed propagules, respectively. The first term in Equation 5 is the probability of h sites being occupied. The next term is the probability of $r - h$ sites being vacant. The third term is the probability that not all h hosts are infected (at least one is healthy). The final term is the binomial coefficient. The probability of finding a site with an infected host surrounded by at least one healthy host within the dispersal neighborhood is $P3 = yf \times P2$. For $P2$ one assumes, if one expects possible spread of a pathogen, that the chosen site is occupied by an infected host; for $P3$ this assumption is not made because $P3$ explicitly accounts for it. If $8{,}192yf < h < r$, then Equation 5 should be modified to account for the cases in which no more than $8{,}192yf$ sites in a neighborhood

TABLE 1. Maximum numbers of infected (latent [L] and infectious [I]) and diseased (latent, infectious, and removed [D]) leaflets in region, proportion of infected leaflets, and maximum number of infected leaflets per host in region

Total host density[a]	iR	Infected	Diseased	Proportion infected	Infecteds per host
300	1	15 ± 0[b]	19 ± 1[b]	0.0010[c]	0.050[d]
	5	18 ± 6[b]	94 ± 56[b]	0.0012	0.059
1,000	1	50 ± 0	73 ± 1	0.0010[c]	0.050[d]
	5	192 ± 8	1,064 ± 114	0.0038	0.192
3,000	0.5	150 ± 0	198 ± 3	0.0010[c]	0.050[d]
	1	150 ± 0	300 ± 11	0.0010[c]	0.050[d]
	2.3	648 ± 103	25,130 ± 3,366[e]	0.0043	0.216
	5	4,956 ± 1,000	85,650 ± 5,258	0.0330	1.65
8,192	1	410 ± 0	12,061 ± 23[e]	0.0010[c]	0.050[d]
	5	121,196 ± 2,304	407,282 ± 0	0.2959	14.8

[a] One percent of hosts were initially infected with $N = 50$, $L = 3$, and $I = 2$; latent period, p, and infectious period, i, were equal to 10 days.
[b] These means and standard deviations are based on five replications; all others are based on four.
[c] A value of 0.0010 indicates that $L + I$ leaflets did not increase.
[d] A value of 0.050 indicates that $L + I$ leaflets did not increase.
[e] Because infected plants and leaflets persisted, this value is for $L + I + D$, not just D, leaflets.

can be inhabited by infecteds. The variable y^h should be replaced by v, in which $v = y^h$ for $h \leq 8{,}192yf$ and $v = 0$ for $h > 8{,}192yf$.

RESULTS

The influence of host density and iR on densities of infected and diseased leaflets is shown in Table 1. Diseased tissue represents the cumulative total of latent, infectious, and removed leaflets in the region. Levels of disease increased no matter how low the host density or the value of iR was. With $iR \leq 1$, the proportion and density of infected leaflets observed at any time did not increase from the initial value; the values always declined over time. The same pattern was observed for the number of infected leaflets per host on average. For $iR \geq 2.3$, the density and proportion of infected leaflets increased above the initial level. These increases were observed in all replications except with 300 hosts; three out of five replications of $iR = 5$ and 300 hosts resulted in no increase. Very few susceptible leaflets remained among the 8,192 hosts when $iR = 5$ (Table 1).

A major assumption in the iR formulas of Van der Plank is that the mother pathogen dies or is removed when reproduction stops. Thus, the lifespan or generation time of the pathogen is the time unit assumed in the threshold formulas for iR. This assumption explains why $iR = 1$ is considered the point at which a given generation exactly replaces itself in the next generation. However, this assumption of replacement must be logically based on counting only living pathogens or infected tissue, not both living and dead or diseased tissue.

Figure 1 demonstrates how the density of infected leaflets in the region changes over the first generation time as a function of iR and total host plant density. In this study, pathogen generation time is 3, 17, and 34 days for latent period p and infectious period i equal to (2,2), (10,10), and (20,20) based on median age of reproduction (12,13). The change was expressed as a percentage of the initial density, because this simplifies the identification of the threshold for increase at 0% (14). A regression line of the form $z = a + c(iR)$ was fit to each set of points on the basis of host density. This simple form was chosen because for three out of four host densities only two values of iR were tested and because the four iR values with 3,000 hosts indicated that a straight line would be appropriate. All lines had coefficients significantly different from zero ($P < 0.001$). For each set of iR and host density there are 3×4 or 3×5 (300 hosts) points based on combinations of p and i and the replications. The r^2 for the lines were 0.88, 0.97, 0.99, and 0.91, respectively, for host densities 8,192, 3,000, 1,000, and 300. Much of the variability in the values reported for 8,192 hosts is due to the approximation to the nearest day of the median age of reproduction and great potential for increase with $iR = 5$; each set of four replicates had very similar values.

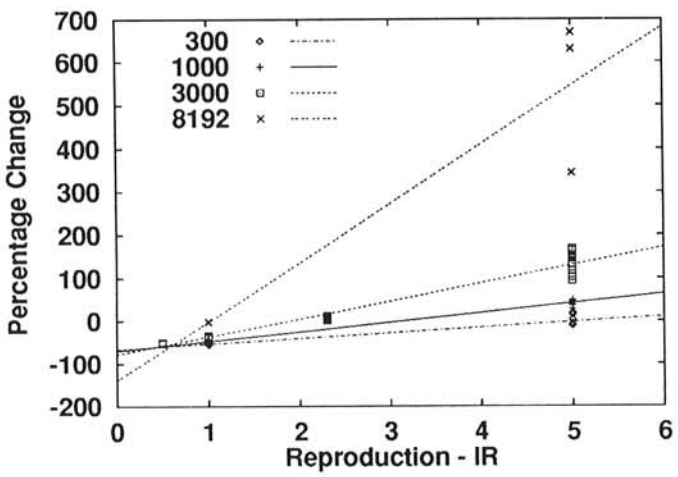

Fig. 1. Percentage of change in the density of infected leaflets ($L + I$) in the region during the first generation of the pathogen as a function of total reproduction per pathogen, iR, and host density. The lines represent the best fit to each set of points.

The threshold value of iR that predicts or determines whether the density of infected leaflets ($L + I$) will increase over a pathogen's generation increased as the initial density of susceptible hosts in the region decreased (Fig. 1). The threshold values of iR were based on the straight lines fit to the points and were 1.01, 1.88, 3.12, and 5.14 for 8,192, 3,000, 1,000, and 300 hosts, respectively. At the beginning of all simulations, 99% of the hosts and 99.9% of the leaflets in the region were completely free of infection, so these constant percentages could not have been the reasons for the differences in the thresholds.

Two other conclusions can be drawn from the data in Figure 1 on the basis of regression analyses of all 126 points. Host density as a class variable (15) did not significantly influence the intercept but it did affect the slope ($P < 0.001$). The equation, $z = -66 + 119 \times f \times iR$ was fit to the data and produced $r^2 = 0.91$. Both coefficients were significantly different from zero ($P < 0.0001$).

The influence of host density and iR on the number and proportion of infected hosts is shown in Table 2. With 1,000 or fewer hosts, an increase in iR from 1 to 5 had very little, if any, influence on the counts and proportions. At higher host densities, the number of infected hosts apparently reached maximum levels limited by host density when iR increased. As host density increased, the peak counts increased, indicating that the increase and spread of the pathogen is constrained by host density (Table 2). At 300 hosts, the initial counts of three plants and three units of nine sites were not exceeded in three out of five and four out of five replications, respectively, at either value of iR.

Table 3 contains the values of *P1*, *P2*, and *P3* for various values of f, the proportion of the region with hosts, and y, the proportion of the hosts infected. The values of f correspond to 3,000, 1,000, 300, and 100 hosts in a region of 8,192 sites. For this study $r = 8$, but the formula can be used for any $r > 0$. *P1* is insensitive to y but decreases with decreasing f. In general, *P2* increases as y decreases and decreases with f for values less than 0.366. Probability *P3* decreases as both f and y decrease. *P1* is useful as a quick and simple indication of what proportion of sites is available for clusters of two or more. For example, in the last set of *P1* values, only one out of a thousand sites have hosts in a cluster of two or more. Without these clusters, the pathogen cannot spread from plant to plant. If we know there is an infected host at a site, then *P2* is the probability that at least one susceptible host surrounds it. Because the existence of the infected host is a given, *P2* increases as the proportion of healthy hosts increases (as y decreases). The opposite occurs for *P3*, because it must account for the probability, yf, that an infected occurs at a given site.

The asymptotic proportion of diseased leaflets ($Q = L + I + D$; $Q = D$ at asymptote) can be predicted from formulas created by Van der Plank (18) and Jeger (6). The basic formula is Q

TABLE 2. Maximum number and proportion of infected plants (with positive L and I) in region

Total host density[a]	iR	Infected plants	Proportion infected
300	1	3.8 ± 1.3[b]	0.0127
	5	3.8 ± 1.3[b]	0.0127
1,000	1	29 ± 2	0.0290
	5	30 ± 3	0.0300
3,000	0.5	219 ± 13	0.0730
	1	331 ± 32	0.1103
	2.3	1,537 ± 165	0.5123
	5	1,520 ± 177	0.5067
8,192	1	8,187 ± 10	0.9994
	5	8,192 ± 0	1.0

[a] One percent of hosts were initially infected; latent period, p, and infectious period, i, were both 10 days.

[b] These means and standard deviations are based on five replications; all others are based on four.

$= 1 - C\exp(-iRQ)$ with $C = 1 - q_0$ according to Van der Plank and $C = (1 - q_0)\exp(q_0)$ according to Jeger. The variable q_0 is the initial proportion of tissue that is diseased (latent, infected, or removed). All the simulations started with $q_0 = 0.001$. Table 4 shows the calculated values based on the two formulas and the values simulated with my model. In general, all the simulated values of Q were different from those calculated with the formulas. Because neither formula contains a variable for host density, the formula-based values were insensitive to density. For $iR = 0.5$, the value is undefined under the assumptions of Jeger's (6) formula. Levels of simulated disease increased no matter how low the host density or the value of iR was (Table 4).

DISCUSSION

Van der Plank (16) postulated that no epidemic could start or that no increase in disease would occur unless $iR > 1$. Certainly positive reproduction is necessary for an increasing population of pathogens, but this positive value is not sufficient. Van der Plank (17) realized that his formula and theorem were appropriate only for situations in which the spread of disease is not constrained by limited amounts of susceptible host tissue. By restricting his theorem to the start of an epidemic and to situations in which S/N is close to 1 and host density is very large, he was able to omit the density of susceptible hosts from his formula.

The threshold theorem of Van der Plank (16) did not explicitly consider plant host density because agricultural monocultures have plenty of plants, they do not move, and wind or plant-to-plant contact can easily spread diseases among neighboring hosts in most situations. Nevertheless, in natural communities (1), in urban plant communities (2), in crops at seedling stage (open canopy), or in crops under significant attack from herbivores, plant density may be important.

For this study, the value of iR that is a threshold for predicting the increase in infected host tissue over a single pathogen generation is dependent on the host density (Fig. 1). The general hypothesis that a mother pathogen must produce more than one offspring to increase the population density is fine. However, for this to be true in reality or in a model, the actual successful reproduction must be above one. The term iR has always represented potential reproduction that does not account for losses in offspring during dispersal and germination. The propagules must land on hosts and on susceptible tissue. Therefore, host density is important.

Apparently, no threshold value of iR exists for an increase in the number of infected plants in the region (Table 2). Figure 1 suggests that no host density threshold exists for predicting increases in infected leaflet density. Logically, increases in infected leaflets will occur when the initial conditions provide an infectant lesion on a plant with susceptible tissue even if that host is not surrounded by other susceptible hosts. But, is there a threshold host density for predicting increases in the number of infected plants? In a probabilistic sense, there is a threshold based on host density.

A probabilistic threshold based on host density can be used to predict whether a pathogen can spread to susceptible hosts during a single pathogen generation. Three pieces of information are required in the formulas: f, the proportion of a region occupied by a host; y, the initial proportion of hosts infected; and r, the number of sites surrounding a central site within a neighborhood of propagule dispersal. The value of r is related to the size of the ecologically proper spatial unit defined by the dispersal behavior of the pathogen (12). I propose that either $P1$ or $P3$ be used to calculate probabilities that can be compared to thresholds. Consensus within the epidemiological community will be needed for deciding on the actual threshold value. When $f < 0.10$, $P1 = f - f(1 - f)^r$ may provide adequate information more quickly. A threshold value of 0.001 might be appropriate for this formula. This value would mean that only one out of 1,000 sites would be likely to have a host in a cluster of two or more. The better but more complicated formula is $P3$. This probability is a direct indicator of the probability that an infected host will be capable of dispersing the pathogen to a susceptible neighbor. A threshold value of 0.00001 may be appropriate when this formula is used. In Table 3, this value occurred when approximately 1% of the sites were occupied by hosts, and 1% of these were initially infected. Both $P1$ and $P3$ were highly correlated ($r > 0.9$) with the maximum number of infected hosts and proportion of infected hosts reported in Table 2. Both formulas were based on the assumption of random distributions of hosts and initial infection, and $P3$ is approximately equal to $yP1$ when y is small (Table 3).

The simulated and formula-derived asymptotic proportions of diseased tissue were very different (Table 4). Jeger's (6) formula cannot account for disease levels with $iR < 1$, and it did not predict increases with $iR = 1$. Van der Plank's (18) formula and the simulations predicted qualitatively different asymptotes than Jeger's; for both $iR < 1$ and $iR = 1$, increases above the initial conditions were predicted. The reason for the difference between the two formulas is that Van der Plank's $C = 1 - q_0$ assumes that no change occurs in disease levels at $iR = 0$, whereas Jeger's formula produces no change at $iR = 1$ ($q_0 = 1 - [1 - q_0]e^{q-Q}$). Perhaps, Jeger confused the increase in infected tissue at $iR > 1$ with the increase in diseased tissue at $iR > 0$. Because both formulas were derived from typical assumptions about uniform and limitless host populations, the formula-derived values were generally higher than simulated values when $iR = 5$ or 2.3. The exception to this pattern was with 8,192 hosts and $iR = 5$, in which case the simulated value was slightly higher (Table 4).

For several reasons, infected ($L + I$) rather than diseased ($L + I + D$) tissue or hosts should be used as indicator variables

TABLE 3. Probabilities of initial spread in a region of 8,192 sites ($r = 8$) with the proportion of occupied sites equal to f and the initial proportion infected equal to y

f	y	$P1$[a]	$P2$[b]	$P3$[c]
0.366	0.50	0.357	0.80	0.14680
	0.10	0.357	0.96	0.03513
	0.01	0.357	0.97	0.00356
0.122	0.50	0.079	0.40	0.02416
	0.10	0.079	0.61	0.00740
	0.01	0.079	0.64	0.00079
0.037	0.50	0.009	0.14	0.00252
	0.10	0.009	0.24	0.00086
	0.01	0.009	0.26	0.00009
0.012	0.50	0.001	0.05	0.00029
	0.10	0.001	0.08	0.00010
	0.01	0.001	0.09	0.00001

[a] Probability of a site in the region being part of a multiple-host cluster.
[b] Probability that a site is surrounded by at least one susceptible host in a dispersal neighborhood of nine.
[c] Probability that a site has an infected host surrounded by at least one susceptible host. $P3 = yfP2$.

TABLE 4. Asymptotic (final) levels of diseased leaflets, Q, simulated with numerical model or calculated with formula $Q = 1 - C\exp(-iRQ)$, in which $C = 1 - q_0$ according to Van der Plank (VdP), or $C = (1 - q_0)\exp(q_0)$ according to Jeger

iR	Hosts[a]	VdP	Jeger	Simulated
0.5	3,000	0.002	...[b]	0.0013
1	300	0.044	0.001[c]	0.0013
	1,000	0.044	0.001[c]	0.0015
	3,000	0.044	0.001[c]	0.0020
	8,192	0.044	0.001[c]	0.0294[d]
2.3	3,000	0.860	0.862	0.1675
5	300	0.993	0.993	0.0063
	1,000	0.993	0.993	0.0213
	3,000	0.993	0.993	0.5710
	8,192	0.993	0.993	0.9943

[a] One percent of hosts initially had 10% of their leaves infected ($q_0 = 0.001$). Region has sites for 8,192 hosts.
[b] Formula is not defined for values of $iR < 1$.
[c] With $iR = 1$, $Q = q_0$.
[d] Slightly lower than asymptote because 19 out of 409,600 leaflets were either latent or infections on day 1,000.

in threshold formulas and other hypotheses concerning pathogen population dynamics. In this study, diseased leaflets always increased in density to a maximum and never declined. The same can be said about diseased hosts in the study of pathogen spread through space. In 1988, Hau (5) noted that disease levels can increase when $iR < 1$. The density of diseased leaflets will always increase above the initial value as long as the initial density includes latents or infectants. Because the density of diseased tissue never declines, Onstad and Kornkven (13) found that diseased hosts and leaflets could not be used as indicator variables for representing the pathogen population in studies of persistence and endemicity. For these reasons, I conclude that studies of pathogen increase and spread should use infected tissue and infected hosts, respectively, as indicator variables.

Results of this study demonstrate that hypotheses can now be extended to include host density. Perhaps, additional or improved formulas can be developed when studies are done of other spatial distributions of hosts and of other spatial units defined by dispersal and the parameter r.

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