Persistence and Endemicity of Pathogens in Plant Populations over Time and Space

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ABSTRACT

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Endemicity is the persistence or constant presence of a pathogen in an ecologically proper spatial unit over many generations. We simulated a simple model over a grid of 8,192 sites to study persistence and the temporal and spatial dynamics of a hypothetical pathosystem of one pathogen and one host. Except for possible increase in the number of leaflets per host, the host population and environment were constant. Two characteristics of the leaf-infecting pathogen influenced persistence. As the potential reproduction per pathogen, iR, decreased, the pathogen was less likely to persist for a given number of generations. The pathogen was more likely to persist over time measured in days when it had a longer infection cycle, but persistence time was essentially constant for all values of latent and infectious periods when time was measured in generations. Several conditions of the host also influenced endemicity. Heterogeneity of iR across hosts increased persistence. Higher host densities and growth of susceptible host tissue increased persistence. Results do not support the theorem that pathogens are endemic when $iR = 1$. Theorems must include spatial scale so they can be tested. Removed diseased tissue cannot logically be used in theorems as a predictor of endemicity. Other formulas for predicting the asymptotic disease level did not predict the results of our simulations, presumably because these formulas assumed that susceptible host density would be uniformly distributed and constant.

Because plant pathologists have focused primarily on epidemics, few have defined or studied endemic diseases. Van der Plank (13) stated that endemic disease is constantly present. Van der Plank (13) also claimed that a pathogen is endemic when total production of offspring per pathogen, iR, equals 1 on average over time and space. He did not, however, provide temporal and spatial scales for evaluating persistence and endemicity. Although Van der Plank's theorem, $iR = 1$, assumes that hosts are nonlimiting, his models of disease progress do not make this assumption. Thus, iR in the theorem is actual reproduction under conditions that are rarely realistic, whereas iR in his models is potential reproduction. (Actual reproduction equals potential reproduction multiplied by the probability of propagule landing on susceptible tissue.) Zadoks and Schein (16) stated that an endemic disease is limited to a region with an average value of $iR = 1$ over the long term. Under their definition, an endemic disease has an average level for a region but may periodically increase or decrease. Zadoks and Schein (16) implied that the smallest time unit for endemicity is the growing season or host generation time, but they did not precisely describe an appropriate spatial scale.

Endemicity can be defined as the persistence or constant presence of a pathogen in an ecologically proper spatial unit over time. An ecologically proper spatial unit is determined from a quantitative understanding of the host's or pathogen's movement during its lifetime. Seem (11) also urged that a sampling or spatial unit be selected carefully and logically. He stated that the unit should be roughly the size of a typical focus for the pathogen. The spatial unit should approximate the average area over which a pathogen as a propagule disperses from the mother source. In accord with this definition, an endemic disease is neither the opposite of an epidemic disease nor necessarily usual or common. The time span for prediction must be included in the concept and expressed in terms of a series of time units related to the generation times of the host and pathogen (2). Epidemics of endemic disease can occur. An epidemic can be identified in a single, space-time unit, but persistence is likely to be of interest over many time units.

Although several plant pathologists have modeled the temporal and spatial dynamics of real or hypothetical pathogens, pathologists have not addressed the question of long-term persistence (6–9). Most often, the emphasis was on the study of dispersal gradients, focus spread, and disease (not pathogen) progress (17). Fleming et al (3) 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.

Endemicity and persistence of pathogens have not been studied for several reasons. Most plant pathologists are interested in protecting crops from economically damaging levels of disease. Natural systems and long-term population dynamics in agricultural systems are rarely investigated (1). Pathogens are likely to persist at relatively low levels that do not attract the attention of scientists.

In this paper, we investigate the persistence of pathogens in host populations that may or may not be uniformly distributed in space. We computed a numerical model to study the qualitative behavior of a hypothetical pathosystem. A numerical model was used instead of an analytical model because of the complexity of the problem (10).

MATERIALS AND METHODS

The pathosystem described below is similar to the basic systems studied by Van der Plank (12,13) and Zadoks (15). 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 time of the host. Essentially, for this scenario, the host is stable, except for possible increase in the number of leaflets, for the complete period of analysis. The system is similar to a community of perennial evergreen tropical plants.

The pathosystem can be expressed 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, respectively. The density of infected but not yet infectious lesions is L. For simplicity, we assume that a lesion covers a single small leaflet by the end of the latent period.

$$dL_t/dt = RI_t(S_t/N_t) - L_t/p \quad (1)$$

$$dI_t/dt = L_t/p - I_t/i \quad (2)$$

$$dD_t/dt = I_t/i \quad (3)$$

$$dS_t/dt = b - RI_t(S_t/N_t) \quad (4)$$

with $RI_t(S_t/N_t) \leq S$. The average latent period is p, the average infectious period is i, and the host growth rate is b. 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.

Equation 1 describes the increase in latent lesions due to reproduction and the decrease due to maturation. On the average, a new lesion requires p days to completely cover its leaflet. In Equation 2, infectious leaflets are increased by maturation from the latent stage and decreased by the rate of removal or development out of the stage. Equation 4 describes host growth and infection. For host growth rate, $b > 0$, new susceptible leaflets are produced each day; for $b = 0$, N is constant. The second term in Equation 4 is the effective rate of infection or successful reproduction by the pathogen. Because N equals $S + L + I + D$, S/N is equivalent to $1 - (L + I + D)/N$. 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.

As an extension of traditional approaches (12,13), we have added an equation for calculating the density of susceptible host tissue. This density is usually not explicitly modeled for two reasons. First, it is assumed that susceptible host tissue is not limiting during early stages of disease increase (13,16). Second, total leaflet density N is assumed constant, a reasonable assumption in mature and very dense crop monocultures. Zadoks (6,15) considered including host growth in his simulation models, but he did not perform an analysis of the models with this feature. In Equation 4, we assumed that N could either be constant or could increase at a constant rate b. More complicated rates of increase could be used, including the negative effects of disease, but our emphasis is not on host growth. At $N = 50$ in the model, no bare soil is exposed for propagules to fall onto; after this state, the plant grows up, not out. Thus, propagules are lost only when they land on nonhost sites or on tissue that is not susceptible.

To model the dynamics of the pathogen in space, we assigned the four 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_j, calculated at site j, to site j, and one-tenth to each of the eight adjacent sites. Propagules landing on nonhost sites are lost. Inoculum landing on host plants survives and germinates with probability S_k/N_k for plant k. Equations 1 and 4 are transformed into

$$dL_{tj}/dt = 0.2RI_{tj}(S_{tj}/N_{tj}) + \sum_{k=1}^{8} 0.1RI_{tk}(S_{tj}/N_{tj}) - L_{tj}/p \quad (5)$$

$$dS_{tj}/dt = b - 0.2RI_{tj}(S_{tj}/N_{tj}) - \sum_{k=1}^{8} 0.1RI_{tk}(S_{tj}/N_{tj}) \quad (6)$$

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, 4, 5, and 6, 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 (12) 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 non-host species are abundant. We modeled the absolute density of hosts by changing the total density for the entire region.

Figure 1 provides two perspectives on the spatial structure of the model. Figure 1A shows the relationship between an initially infected host and its eight neighbors. The area of influence from this centralized host can be thought of as a unit of nine, although less than nine hosts may occupy the unit. This neighborhood is the ecologically proper spatial unit for this study. Figure 1B represents a small portion of the region of sites simulated with the model. The small rectangles are units of nine. The assignment of these units to the sites does not influence the model processes, is used only for analysis of results, and is arbitrary (like any other sample unit or quadrat).

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 we only used one-fourth of the machine. The 8,192 processors used were modeled as a 64×128 grid of plant sites. This grid permitted a maximum of 882 units of nine sites to be studied. Each simulation of 1,000 daily time steps with Euler integration required 2 min on the computer.

We studied pathogen persistence over 1,000 days by varying absolute host density in the region, relative and absolute

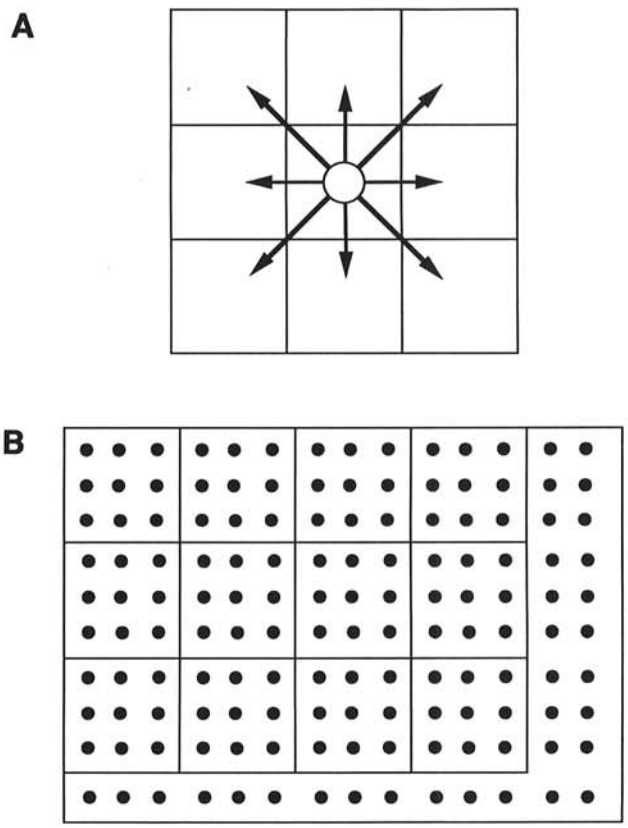

Fig. 1. Two perspectives of simulated spatial dynamics. **A,** Unit of nine sites occupied by hosts with pathogen dispersal from center source of inoculum. **B,** Portion of region of 8,192 hosts that shows grid and assignment of units.

susceptible leaflet densities per host, latent and infectious periods, and the distribution of iR values across hosts (Table 1). Host density was either 8,192 in a uniform distribution or 3,000 in a random distribution (with four replications). Each host site started with $N_{0j} = 50$, $L_{0j} = 3$, and $I_{0j} = 2$. For simulations of 8,192 hosts, half of the computations were made with no host growth ($b = 0$), and half were made with growth $b = 0.5$, causing the relative (S/N) and absolute leaflet densities to increase. All simulations of 3,000 hosts used $b = 0.5$.

Latent period p and infectious period i were 2, 10, or 20 days. All nine combinations of i and p were used in the simulations of 8,192 hosts, whereas only three combinations (2,2), (10,10), and (20,20) were used to study 3,000 hosts. For simulations of 8,192 hosts in a uniform region, one scenario used half of the hosts with $iR = 0.8$ and half with $iR = 1.2$; thus, the average value was $iR = 1$ at the start. This randomization was replicated four times. In another scenario with the 8,192 hosts, all had $iR = 1$. The distribution of iR was uniform in all simulations of 3,000 hosts either with $iR = 1$ or with $iR = 2.3$. Based on a simple calculation, we assumed that 2.3 was the average reproduction needed to break even when 3,000 out of 8,192 sites are occupied, and 20% of the propagules remain on the mother pathogen's host in a unit of nine neighbors.

We attempted to answer the following questions. Does the pathogen persist in the region for 1,000 time steps? Does the pathogen persist in all units of nine host sites? Is the period of persistence a constant number of days or a constant number of pathogen generations? Is persistence influenced by p or i? Does heterogeneity of iR affect persistence? Do host densities and host growth influence persistence? Which variables can or cannot be used as indicators of persistence?

We measured a variety of variables to avoid the problem of relying on only one variable for the identification of relationships (2). We analyzed regional counts of hosts with densities of I, L, and D greater than one-thousandth of a leaflet and counts of units of nine containing at least one host with 10^{-3} leaflets of I, L, and D. Because the differential equations produce continuous variables that may be very small fractions of leaflets, we used this threshold of 10^{-3} to identify infected or removed leaflets, but values in the model were unaffected. Other variables were the sums of leaflets S, $L + I$, $L + I + D$, $I + D$, for the entire region.

RESULTS

The general pattern, with few exceptions, in the changes in pathogen density was a continuous decline from the initial density to a much lower value (Figs. 2,3). A similar pattern of decline was observed for the changes in the number of hosts and units of nine with infection. As results described below indicate, no more than 40% of the leaflets in the region were diseased during the simulation. Thus, susceptible tissue was always available even without host growth. We inferred that the pathogen declined to extinction in the region when its actual rate of reproduction dropped below the necessary replacement level.

In the region of 8,192 uniformly distributed nongrowing hosts ($b = 0$), the pathogen represented by L or I never persisted for all 1,000 days (Table 2). Latent and infectious leaflets disappeared from the region by day 78 when i and p equaled 2 and by day 822 when they both equaled 20. As the sum of i and p (the infection cycle) increased, the duration of persistence in terms of days increased greatly. The pathogen disappeared approximately 20 cycles ($i + p$) after the start of the simulation. The extinction dates were earlier under the homogeneous distribution of iR than they were under the heterogeneous distribution.

The asymptotic proportion of diseased (i.e., equals removed at the asymptote) leaflets in the region of 8,192 nongrowing hosts was generally 0.39 for the homogeneous distribution of iR and $0.40 + 0.00006$ for the heterogeneous distribution. With all hosts having $iR = 1$ and both i and p equal to 2, the final proportion was 0.40.

When the 8,192 hosts were allowed to grow ($b = 0.5$), the pathogen persisted for the 1,000 days in most cases. For $p + i \geq 20$, the pathogen persisted at the regional scale and in all 882 units of nine hosts for both distributions of iR. Persistence within the region or a single unit does not require persistence on all hosts within that spatial unit. With $iR = 1$ in a homogeneous distribution, the pathogen did not persist as latents or infectants when $p + i$ was 12 or less (i.e., three cases). For iR distributed heterogeneously with two values, the pathogen only disappeared from the region when both p and i were 2 days. For $p = 10$ and $i = 2$, the numbers of infected hosts and units were 5,885 $\pm$ 80 and 740 $\pm$ 11, respectively, on day 1,000 with the heterogeneous distribution. The values dropped to 0 by day 818 for the homogeneous distribution. Similar results in both cases were

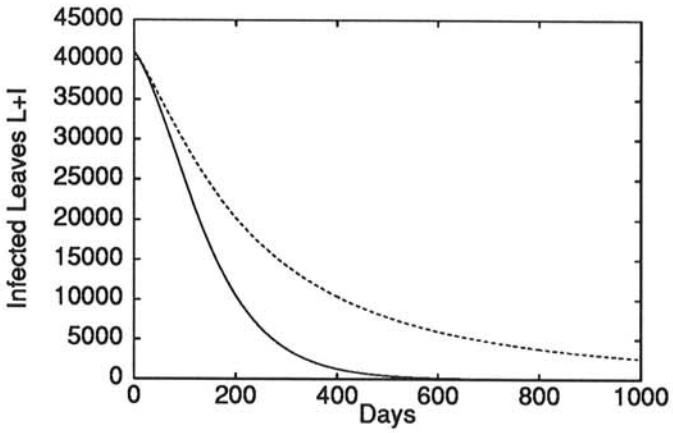

Fig. 2. Density of infected leaves ($L + I$) in the field of 8,192 uniformly distributed hosts with no growth ($b = 0.0$, ——) or with growth of new leaves ($b = 0.5$, - - -). Simulations had constant $iR = 1.0$ and latent and infectious periods of 20 days.

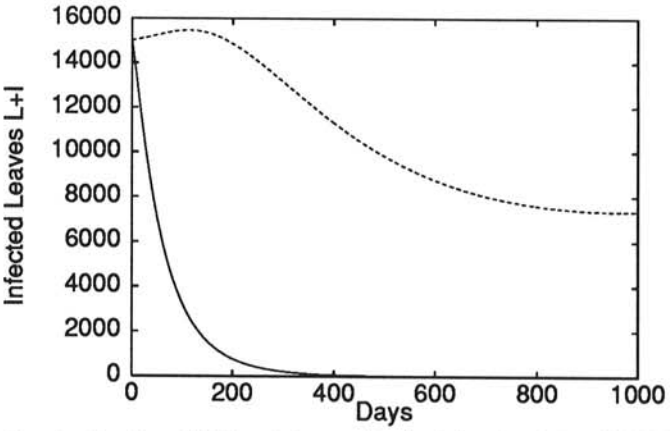

Fig. 3. Density of infected leaves ($L + I$) in the field of 3,000 heterogeneously distributed hosts with $iR = 1.0$ (——) or with $iR = 2.3$ (- - -). Simulations had $b = 0.5$ and latent and infectious periods of 20 days.

TABLE 1. Initial conditions, parameter values, and replications for all of the simulations

Initial host density	Initial density of infecteds	Growth rate b	Latent (p) and infectious (i) periods	iR	Replications
8,192	8,192	0, 0.5	Nine[a]	1	1
8,192	8,192	0, 0.5	Nine	1[b]	4
3,000	3,000	0.5	Three	1, 2.3	4

[a] Nine combinations consist of all combinations of $i = 2$, 10, or 20 days and $p = 2$, 10, or 20 days. Three combinations of i,p are (2,2), (10,10), and (20,20).

[b] Mean of half of hosts assigned $iR = 0.8$ and half $iR = 1.2$.

computed with $p = 2$ and $i = 10$. Thus, with $b = 0.5$, heterogeneity in the distribution of iR enhanced persistence just as it delayed extinction with $b = 0$.

Table 3 shows how the number of infected leaflets and proportion of diseased leaflets on day 1,000 increased as $p + i$ increased. When infected leaflets ($L + I$) persisted, the proportion or density of diseased leaflets did not reach an asymptote. Thus, diseased tissue reached an asymptote only when $p = i = 2$ (Table 3). The density of susceptible leaflets on day 1,000 declined as $p + i$ increased for both scenarios of iR: 4,328,894 ($p = 2 = i$) to 4,192,809 ($p = 20 = i$) for $iR = 1$, and 4,326,944 ± 52 ($p = 2 = i$) to 4,173,292 ± 916 ($p = 20 = i$) for $iR = 0.8$ or 1.2. As the cycle length ($p + i$) increased, the density of infected leaflets and the proportion of diseased leaflets in the region on day 1,000 increased. In general, with host growth, cumulative disease ($L + I + D$) is smaller as a proportion but larger as a density compared to the case without host growth. For example, with $b = 0$, the maximum density of diseased leaflets was 162,000 for the region; with $b = 0.5$, the maximum was 333,000.

Persistence was less likely to occur when host density decreased from 8,192 to 3,000. With 3,000 randomly distributed and constantly growing hosts with $iR = 1$, the pathogen persisted at the regional scale only when the latent and infectious periods were both 20 days (Table 4). The pathogen persisted in less than two out of 867 units of nine. When all 3,000 hosts had $iR = 2.3$, regional persistence in terms of L and I occurred in all cases (Table 4). The pathogen, however, did not remain endemic in all units of nine. For example, when both p and i equaled 2 days, counts of plants and units with positive densities of I and L declined during the first 190 days to a mean value of 370 and then increased to the counts shown in Table 4. In general, increasing the value of iR increased the likelihood of persistence. Dead or removed tissue always persisted at either spatial scale. No asymptotic disease level occurred when the pathogen persisted.

DISCUSSION

In this study, we attempted to explain persistence of pathogens by using a nonequilibrium model of population dynamics. Equilibrium models identify stable scenarios of host and pathogen that last forever (10). Persistence must be measured in a finite amount of time to be able to test and develop hypotheses. Population genetics may be important for understanding persistence, but results of this study indicate that persistence may be at least partially due to typical nonevolutionary population dynamics. In our simulations, the pathogen population declines and eventually disappears or becomes extinct from a spatial unit, unless new susceptible tissue is produced in the spatial unit. Host growth with the formation of new leaflets is the mechanism by which susceptible tissue is added to our hypothetical pathosystem. Other factors like the value of iR, its heterogeneity, and host density only influence the time required for the pathogen to decline to 0 in the spatial unit.

When Van der Plank (13) and Zadoks and Schein (16) analyzed the population dynamics of disease with a simpler model, they concluded that endemic disease has $iR = 1$ on average. Our results indicate that the value of iR by itself is not enough to predict whether a pathogen will persist or not. In some cases, when $iR = 1$ as a constant or an average, the pathogen did not persist (Tables 2 and 4). The assumption of Van der Plank (13) and probably of Zadoks and Schein (16) was that the susceptible host population was not limiting. We agree with Jeger (5), who concluded that neither a constant low value of iR nor a low level of disease is sufficient for defining endemic disease.

Each 1,000-day simulation lasted at least 25 infection cycles equal to $p + i$ days. Time can also be expressed in terms of pathogen generation time. According to Burdon and Chilvers (2), the appropriate temporal scale for this type of study has a basic unit related to the reproductive cycles of the host and pathogen. Van der Plank (12,13) defined the latent period as the generation time for the pathogen. Zadoks and Schein (16) considered the infection cycle to be the basic temporal unit for plant epidemiology. For species with overlapping generations, the median age for reproduction can be used as a measure of generation time. Given that $1/i$ infectious leaflets are removed each day and that the reproductive rate, R, is constant in each simulation, the ages at which half the reproduction has occurred in a cohort are $p + 1$, $p + 7$, and $p + 14$ days for i equal to 2, 10, and 20 days, respectively. The hypothetical pathogen in this study persisted for approximately 20 infection cycles or 23.5 generations before disappearing in the region of 8,192 nongrowing hosts (Table 2). Because the product of the effective reproductive rate, $R(S/N)$, multiplied by i is less than 1 throughout all the simulations reported in Table 2 (because $S/N < 1$), the population of infecteds declined during the infectious periods of every generation or cycle. Of course, a latent period must be passed through before each reproductive period is reached. Thus, generation time is an appropriate temporal unit for analyzing endemicity.

The choice of spatial scale for the analysis determines whether the pathogen was persistent. The smallest spatial unit for the analysis of pathogen population dynamics should be based on

TABLE 2. First days on which number of infected hosts declined to zero during simulations of 8,192 nongrowing hosts initially infected with either $iR = 1$ for all hosts (one replication) or $iR = 0.8$ for half and $iR = 1.2$ for the other half (four replications)

Periods[a] (p,i)	$iR = 1$		$iR = 0.8$ or 1.2	
	Hosts with latents	Hosts with infectants	Hosts with latents	Hosts with infectants
2, 2	75[b]	77	76 ± 0[c]	78 ± 0
2, 10	259	215	263 ± 0	221 ± 0
10, 2	207	264	212 ± 0	269 ± 0
10, 10	390	401	398 ± 0.6	409 ± 0
2, 20	492	371	501 ± 0	382 ± 0.6
20, 2	354	502	364 ± 0	511 ± 0.5
10, 20	617	580	628 ± 0	592 ± 0
20, 10	560	631	573 ± 0.6	643 ± 0
20, 20	784	806	800 ± 0.6	822 ± 0

[a] Latent period is p; infectious period is i.
[b] Day on which count of hosts decreased to zero based on threshold of 10^{-3} leaflets per host.
[c] Standard deviation.

TABLE 3. Number of infected (latent and infectious) leaflets and proportion of diseased leaflets (latent, infectious, and removed) on day 1,000 for simulations of 8,192 growing hosts initially infected with either $iR = 1$ for all hosts (one replication) or $iR = 0.8$ for half and $iR = 1.2$ for the other half (four replications)

Periods[a] (p,i)	$iR = 1$		$iR = 0.8$ or 1.2	
	Infected leaflets	Proportion diseased	Infected leaflets	Proportion diseased
2,2	0.0	0.04[b]	0.0 ± 0.0[c]	0.04[d]
2,10	4.4[e]	0.05	22 ± 2.5	0.05
10, 2	4.4[e]	0.05	21 ± 2.4	0.05
10, 10	134	0.05	274 ± 12	0.06
2, 20	244	0.06	446 ± 16	0.06
20, 2	250	0.06	449 ± 16	0.06
10, 20	924	0.06	1,368 ± 29	0.07
20, 10	940	0.06	1,383 ± 29	0.07
20, 20	2,613	0.07	3,437 ± 48	0.07

[a] Latent period is p; infectious period is i.
[b] Total density of leaflets on day 1,000 with $N_0 = 50$ and $b = 0.5$ is 4,505,600.
[c] Standard deviation.
[d] Standard deviation based on four replications monotonically increased from 0.00001 for p and i equal to 2 days to 0.00020 for p and i equal to 20 days.
[e] Although 4.4 infected leaflets remained on day 1,000, the number per host was less than the 10^{-3} minimum level for counting hosts after dividing by 8,192.

TABLE 4. Results of simulations of 3,000 growing hosts, which were initially infected with $iR = 1.0$ or $iR = 2.3$

Periods[a] (p,i)	Number of plants with[b]			Number of units of nine with[c]		
	Latents	Infectants	Removals	Latents	Infectants	Removals or suscepts
$iR = 1.0$						
2, 2	day 70 ± 3	day 72 ± 3	3,000 ± 0	day 70 ± 3	day 72 ± 3	867 ± 5
10, 10	day 456 ± 32	day 467 ± 32	3,000 ± 0	day 456 ± 32	day 467 ± 32	867 ± 5
20, 20	3.5 ± 5	5.8 ± 6	3,000 ± 0	1.3 ± 1	1.8 ± 2	867 ± 5
$iR = 2.3$						
2, 2	2,617 ± 42[d]	2,617 ± 42[d]	3,000 ± 0	759 ± 17[d]	759 ± 17[d]	867 ± 5
10, 10	2,774 ± 13	2,774 ± 13	3,000 ± 0	799 ± 8	799 ± 8	867 ± 5
20, 20	2,868 ± 13	2,868 ± 13	3,000 ± 0	825 ± 9	825 ± 9	867 ± 5

[a] Latent period is p; infectious period is i.
[b] Number of hosts with values of L (latents), I (infectants), or D (removals) $> 10^{-3}$ on day 1,000, which normally has lowest value. If value is 0, the date on which it went to 0 is given. Means and standard deviations are based on four replications.
[c] Number of units with at least one host having values of L (latents), I (infectants), D (removals), or S (suscepts) $> 10^{-3}$ on day 1,000. Values for S and D were equal.
[d] Lowest values actually occurred about day 190. Lowest number of units of nine was 370 ± 31.

the dispersal function. For this study, the fundamental unit is the unit of nine. Sometimes the pathogen was not endemic during the 1,000 days in all units (Table 4). When the pathogen disappeared from a single unit, it persisted in the region by remaining endemic in other units. Spatial dynamics among units may keep the pathogen endemic at the regional scale. A future study should investigate how plant and leaflet densities influence the dynamics within each unit of nine.

The region of 8,192 sites could be considered a heterogeneous region of fields with N hosts at each site. Dispersal would be from a central field to the adjacent eight fields, and R would be the reproductive rate per infectious plant, not infectious leaflet. The conclusions described below are valid at any given absolute area of space (i.e., field or region of fields) as long as the spatial unit relative to dispersal is maintained.

Two characteristics of the pathogen influenced persistence. We conclude that as iR decreases, a pathogen is less likely to persist for a given number of generations (persistence time will be less). We also hypothesize that a pathogen is more likely to persist over a given number of days when it has a longer generation time and total reproduction per pathogen iR is held constant. In terms of pathogen generation time, the period of persistence was fairly constant. With iR constant, the longer generation times slow down any decline in the pathogen population. If R, but not iR, was held constant, total reproduction would be less for shorter infectious periods, and the ratio of p to i would be important.

Conditions of the host that influenced endemicity were increase in the number of leaflets per host, host plant density, and heterogeneity of iR. We hypothesize that heterogeneity of iR due to mixtures of cultivars will always increase persistence relative to a crop monoculture with the average iR. Another hypothesis is that smaller host densities decrease persistence over a given number of pathogen generations. No host-density threshold was observed. A final postulate is that continuous growth of susceptible host tissue increases the probability of persistence. This conclusion reminds us that the population dynamics and growth of the host are important in the long-term analysis of a pathogen.

As expected, dead or removed leaflets persisted in the region and in all the units under all scenarios. Variables that do not distinguish between infected and diseased tissue are not useful predictors of population dynamics in epidemiology. From a population dynamics perspective, predictions must be based on observations of infecteds (latents and infectants) and susceptibles. If dead or removed tissue is included in the measurement of the pathogen, then the total amount of diseased tissue will never decline (3).

Because confusion in the use of the terms diseased and infected exists, precise definitions are needed. The sum of L, I, and D equals the single variable used by Van der Plank (12,13) in most of his models and formulas. Van der Plank (12,13) generally defined epidemics in terms of all diseased tissue. Zadoks (15) calculated all three variables, but implied that an epidemic should be measured by the sum of I and D, severity of the disease. Most plant pathologists have not questioned the use of removed lesions in the measurement of pathogen population dynamics (4). In zoological epidemiology, removals are assumed to be dead or immune animals, not diseased individuals or parts of individuals. The animal systems are similar to plant systems with systemic viral diseases that infect a whole plant. Dead or removed lesions are important in crop loss assessment, but they are not useful in the measurement of pathogen population dynamics.

Van der Plank (14) postulated that the formula $Q = 1 - C\exp(-iRQ)$ describes the asymptotic proportion of diseased tissue Q, with $C = 1 - q_0$. The variable q_0 is the initial proportion of tissue that is diseased (latent, infected, or removed). Van der Plank's formula calculates 0.40 as the asymptote when $iR = 1$ and $q_0 = 0.1$. Jeger (5) used a similar formula, except that $C = (1 - q_0)\exp(q_0)$. With $q_0 = 0.1$, $C = 0.9947$, and $iR = 1$, Jeger's version of the formula calculated a value of 0.1. Thus, when 8,192 nongrowing, uniformly distributed hosts are considered, Van der Plank's formula-based prediction matched our results, but Jeger's did not. When host growth or spatial heterogeneity was included in our model, our results were different from those of both formulas. Host growth complicates the comparison between simulated and formula-based predictions, because neither formula (5,14) considers that growth and asymptotes were rarely reached in the simulations because of host growth. However, with $p = i = 2$ and $iR = 1$, the simulated asymptote is 0.04 (Table 3) or 0.02 for 8,192 or 3,000 growing hosts, respectively. These values are less than the values of 0.10 and 0.40 derived from the formulas of Jeger (5) and Van der Plank (14), respectively.

Future theoretical work concerning pathogen persistence can take several directions. The assumptions about the host population could be changed. A host with an annual growth cycle could be simulated. If the effect of disease on host reproduction and mortality was modeled, then the community dynamics of both species could be studied over many host and pathogen generations. Another subject that should be explored in more detail is the heterogeneity of host distribution. Various distributions of density or iR could be evaluated by using techniques similar to those of Mundt and Leonard (8) and Mundt et al (9). Other forms of the dispersal function must also be studied. Shallower gradients would permit the pathogen to spread to more neighbors around a given host. Finally, hypotheses should be developed from several kinds of models and tested against independent field data.

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