A Model Relating the Probability of Foliar Disease Incidence to the Population Frequencies of Bacterial Plant Pathogens

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ABSTRACT

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A model has been developed that relates pathogen population size on individual leaves at time t_0 to disease incidence at some later time t_1. The lognormal distribution is used to describe population size of pathogenic bacteria on individual leaves, and the probit function is used to describe the probability of disease, given a bacterial population size. When integrated with respect to bacterial population frequency, the product of the probability of disease, given a bacterial population size, for individual leaves and the frequency of pathogenic bacteria on individual leaves results in a cumulative normal frequency distribution. This distribution function describes the probability of disease incidence (as frequency of diseased leaflets) in a field. To illustrate its use, the model was applied to bacterial brown spot of beans caused by *Pseudomonas syringae* pv. *syringae*. A maximum-likelihood technique was used to estimate the mean and variance of bacterial population size from censored samples.

Under laboratory conditions, the relationship between number of pathogenic bacteria introduced into leaves and disease development has been obtained via infectivity titration experiments (4). In those experiments, a series of treatments were established; each treatment consisted of a specific dose of bacteria applied uniformly to a set of leaves. Subsequently, the number of leaves with lesions in each treatment was counted and a relationship between dose (number of bacteria applied per leaf) and response (fraction of leaves that become diseased) was established.

Three conditions that commonly prevail in the field make it more difficult to establish a quantitative relationship between bacterial population size and subsequent disease. First, the "dose" (epiphytic population size of pathogenic bacteria) on individual leaves in the field frequently varies by 1,000-fold or more. Thus, to establish a quantitative relationship between bacterial population size and subsequent disease in the field it is necessary to treat the frequency of bacterial population sizes on individual leaves as an independent variable. Secondly, bacterial dose and disease response can never be determined on the same leaf because the determination of dose requires destruction of the leaf. Finally, epiphytic pathogenic bacterial (EPIPAB) populations fluctuate temporally as a result of variable environmental conditions and phenology of the crop.

While several field studies report the observation that amount of disease was associated with the size of the EPIPAB population (2,3,10,11,15,16,19,22,24), only a few studies have reported a significant correlation between some indicator of the size of the EPIPAB population on individual plant parts and subsequent disease. Thomson et al (23) found that "*Erwinia amylovora* multiplied in healthy pear flowers and was detected in individual flowers 14 days prior to disease occurrence in some orchards." Low or undetectable populations of *E. amylovora* on individual flowers correlated with low levels of disease. In this case, monitoring of epiphytic populations in flowers was useful for timing bactericide applications. In our preliminary experiments, brown spot of beans (caused by *P. syringae* pv. *syringae*), halo blight of oats (caused by *P. syringae* pv. *coronafaciens*), and bacterial blight of soybeans (caused by *P. syringae* pv. *glycinea*) were not observed until shortly after some leaves (leaflets) within the plant canopies were found to harbor relatively high EPIPAB populations (9,12,13; and Hirano et al, *unpublished*). For example, in both 1979 and 1980, symptoms of bacterial brown spot did not appear until after populations of *P. syringae* $>10^4$ had been detected on individual leaflets (13). Mean pathogen population sizes in the canopy were not necessarily associated with subsequent disease. The important criterion relating bacterial populations and subsequent disease appeared to be the frequency of high populations of the bacteria on individual leaflets. This observation is consistent with the results of infectivity titration experiments which demonstrate that the probability of disease occurrence increases with the logarithm of dose (4).

If epiphytic populations of the pathogen are the immediate source of inoculum for disease, then it is the distribution of EPIPAB population sizes on individual leaves that will determine ensuing disease incidence (proportion of diseased leaves). Thus, a theoretical explanatory model relating bacterial population size to subsequent disease development should incorporate the distribution of EPIPAB population size frequencies. Such a model may be of practical value for conditions under which both mean and variance fluctuate over time since the mean alone might be a poor predictor of the number of leaves with relatively high EPIPAB populations.

The purpose of this paper is to describe a model that relates disease incidence to plant pathogenic bacterial population size under field conditions. Both the distribution of EPIPAB population sizes among individual leaves and probability of disease given EPIPAB population size on individual leaves are taken into account. The conceptual basis for the specific model presented below has been previously reported (20).

THE MODEL

Relating pathogen population size to subsequent disease incidence. Assume a population of leaves borne on a uniform field of plants of a single cultivar. Let the random variable N represent the number of phytopathogenic bacteria on individual leaves for which N can take on integer values: 0, 1, 2, 3,.... Let the probability that n viable plant pathogenic bacteria reside as epiphytes on a leaf

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at time t_0 be denoted by $P(N = n)$, which is read as the probability that the random variable N takes on the particular value n. (We are using the convention that uppercase letters refer to random variables and lowercase letters to realized values.) This probability is equivalent to the proportion of leaves in the population with n phytopathogenic bacteria. The population of leaves in the field can be thought of as consisting of a series of subpopulations of leaves distinguished by the number of bacteria present on each of the leaves at time t_0. The number of leaves in each subpopulation is directly proportional to the frequency distribution for N. Assume that conditions are favorable for infection at some point in time between t_0 and t_1 and that each bacterium has an equal, but small, chance of successfully causing disease. Leaves with a small number of bacteria on them are less likely to become diseased than leaves with a large number of bacteria on them. At time t_1 each subpopulation of leaves will be divided into sets of healthy leaves and diseased leaves as a result of infection by the pathogenic bacteria present at time t_0.

For a given subpopulation of leaves defined by $N = n$, there will be associated a probability of disease equivalent to the proportion of leaves within that subpopulation that become diseased. This probability will be represented by $P_D(n)$. This quantity is the probability that leaves in the subpopulation $N = n$ will become diseased; the probability changes with n. Since the proportion of leaves in the subpopulation with n pathogenic bacteria is $P(N = n)$, the proportion of leaves in the entire field that hosted n bacteria as epiphytes at time t_0 *and* are diseased at time t_1 is given by the product of the frequency with which individual leaves bore EPIPAB population size equal to n bacteria at time t_0 and the probability that leaves bearing n bacteria became diseased, or $P(N = n) \cdot P_D(n)$. By summing this product over all subpopulations of leaves within the field, the probability that a randomly selected leaf from the overall population of leaves in the field is diseased at time t_1 can be written as

$$\begin{aligned} PD &= \text{Prob (randomly selected leaf is diseased)} \\ &= \sum_n P(N = n) \cdot P_D(n) \end{aligned} \quad (1)$$

in which $\sum_n$ represents the summation over all possible subpopulations $N = n$. Since the number of bacteria on leaves is large, the expressions for $P(N = n)$ and $P_D(n)$ are written as continuous functions and the summation in equation 1 is replaced by an integral sign.

The mathematical formulation of the model. The lognormal distribution has been shown to describe the distribution of pathogenic *P. syringae* on a leaf (8,13). The random variable $Y = \log_e N$ is normally distributed with density function

$$f(y) = (1/\sqrt{(2\pi)}\sigma)\,(e^{-(1/2)[(y-\mu)/\sigma]^2}) \quad (2)$$

in which the parameters μ and σ describe the distribution and represent the mean and standard deviation of y, the normal distribution of the logarithm of population size (1). Thus, the continuous function corresponding to $P(N = n)$ is $f(y)$ in which $y = \log_e n$.

The equation chosen to represent $P_D(n)$ can be called a dose-response function since it relates the probability of disease (response) to the number of bacteria (or dose). A plausible model for $P_D(n)$ is the probit function or cumulative distribution function for a standard normal distribution often used to describe relationships between dose and response (5). The probit model has been used in several controlled environment studies to relate plant pathogenic bacterial dose to disease response (4). Recall that $e^{-1/2z^2}/\sqrt{(2\pi)}$ is the density of the standard normal (mean = 0 and variance = 1). Let $y = \log_e n$; then

$$P_D(n) = \phi[(y - \lambda)/\tau] = \int_{-\infty}^{(y-\lambda)/\tau} [1/\sqrt{(2\pi)}][e^{-(1/2)z^2}]dz \quad (3)$$

in which λ and τ are the mean and standard deviation of the probit (normal) and ϕ denotes the cumulative distribution function for the standard normal. The relationship between the probability that a leaf from the subpopulation indexed by $N = n$ becomes diseased as n increases follows a sigmoid pattern.

Substitution of equations 2 and 3 into the integral form of equation 1 results in

$$PD(\lambda, \tau, \mu, \sigma) = \int_{-\infty}^{\infty} \phi[(y - \lambda/\tau]\ 1/[\sigma\sqrt{(2\pi)}]\ e^{-(1/2)[y-\mu)/\sigma]^2} dy \quad (4)$$

in which P_D is now viewed as a function of the parameters μ, σ, λ, and τ. Performing the integrations (17) results in

$$PD(\lambda, \tau, \mu, \sigma) = p^* = \phi[(\mu - \lambda)/(\sigma^2 + \tau^2)] \quad (5)$$

with ϕ defined as above and p^* is shorthand for $PD(\lambda, \tau, \mu, \sigma)$. Thus, the probability that a randomly selected leaf is diseased (or equivalently the proportion of diseased leaves) is a function of the mean and variance of the distribution of log bacterial numbers on a leaf (μ and σ^2) and the mean and variance of the dose-response function (λ and τ^2).

EXAMPLE

The following example illustrates the applicability of this model to field data. Nonlinear regression using equation 5 was used to determine the relationship between epiphytic population sizes of *P. syringae* pv. *syringae* and subsequent bacterial brown spot disease of snap bean. Epiphytic populations of *P. syringae* on individual bean leaflets were measured for each of nine plots (6.1 × 7 m) three times during the growing season. Each plot represented a different treatment designed to establish different frequencies of population sizes of *P. syringae* pv. *syringae* on bean leaflets. Treatments are described in Table 1. At each sampling time, population sizes of *P. syringae* were quantitated by dilution plating of individual leaf washings of 28–30 leaflets per plot. Bacterial colonies were counted on the dilution plates after 3–4 days of incubation at room temperature. Bacteria that were fluorescent, oxidase negative, and ice nucleation active (14) were assumed to be strains of *P. syringae*. Approximately 99% of a subset consisting of 589 isolates of these presumptive *P. syringae* were pathogenic to bean pods.

For the model described by equation 5 to be applicable to a given data set, the distribution of bacterial population sizes among leaflets should be lognormal. A graphical assessment was used to determine if bacterial population sizes on individual leaflets from each treatment, expressed as $\log_{10}$ colony-forming units (cfu) per leaflet, could be described by the normal distribution. A plot of normal scores versus $\log_{10}$ bacterial population size approximated a straight line for eight of the nine treatments (Fig. 1). The $\log_{10}$ cfu from leaflets in the streptomycin seed treatment illustrated in Fig. 1A had a nonlinear relationship with normal scores indicating some departure from lognormality.

The next step in using the model was to obtain estimates of μ and σ from the bacterial population data. A problem arose in obtaining these estimates because some leaflets harbored bacterial populations too small to be detected by the leaf washing-dilution plating procedures (i.e., less than 150 cfu). In Fig. 1, numbers to the left of the data represent the numbers of leaflets on which *P. syringae* were not detected. The detection limit for the dilution plating procedure used was 2.2 as $\log_{10}$ cfu or 5.066 as $\log_e$ cfu. Population values below the detection limit are viewed as censored in that the exact values are not known. However, the value below which they fall is known. Based on the assumption that the lognormal distribution adequately fits these data, a maximum-likelihood procedure was used to obtain estimates of μ and σ for those data sets that included censored points (6). With extreme censoring (i.e., high fraction of leaflets with EPIPAB populations below the detection limit) this procedure produces biased estimates.

Procedure for estimating μ and σ for data sets with censored observations. Let k = number of observations (leaflets) with $\log_e$ cfu ≥ 5.066. The likelihood of occurrence of the ith of these k observations is

$$L = [1/\sigma\sqrt{(2\pi)}]e^{-(1/2)[(y_i-\mu)/\sigma]^2}. \quad (6)$$

Let m = total number of leaflets sampled. Then $m-k$ represents the number of leaflets with populations of bacteria below the detection limit of the assay. The likelihood of occurrence of each of these $m-k$ observations is

$$L = P(Y<d) = [1/\sigma\sqrt{(2\pi)}]\int_{-\infty}^{d} e^{-(1/2)[(z-\mu)/\sigma]^2}dz \qquad (7)$$

in which $d = 5.066$. Given the data, the overall likelihood for μ and σ is

$$L(\mu, \sigma) = \left\{\prod_{i=1}^{k} [1/\sigma\sqrt{(2\pi)}]\, e^{-(1/2)[(y_i-\mu)/\sigma]^2}\right\} \cdot \left\{[1/\sigma\sqrt{(2\pi)}]\int_{-\infty}^{d} e^{-(1/2)[(z-\mu)/\sigma]^2}\right\}^{m-k}. \qquad (8)$$

This equation is solved for μ and σ given the k values for y_i and the number of values below the detection limit, $m-k$, using a steepest gradient approximation procedure (17). We implemented this procedure with a FORTRAN program (available from D. I. Rouse).

The procedure just described was used for estimating the mean and variance of the distribution of log bacterial numbers, μ and σ, for the data in Fig. 1. These estimates are presented in Table 1 along with estimates that do not correct for censoring. Also presented in Table 1 are the disease incidence data from the field plots 4 and 8 days after leaflets were sampled for bacterial population estimation. Disease assessments were made by selecting eight plants at random from each plot and counting the number of leaflets on each plant showing bacterial brown spot disease symptoms. Data were expressed as proportion of diseased leaflets per plot.

Estimates of λ and τ from bacterial frequency and disease data. Finally, estimates for λ and τ, the mean and standard deviation of the dose-response function describing the probability of disease given the number of bacteria, are obtained from equation 5 by iteratively reweighted nonlinear regression. Using the data from Table 1 as an example, there are nine distinct data points. The dependent variable in each case is p_i^*, the proportion of diseased leaves in the ith plot. In our examples we used as separate dependent variables the proportion 4 and 8 days after leaflets were sampled for bacterial population estimation. Corresponding to p_i^* are values for μ_i and σ_i (corrected for censoring using equation 8); μ_i and σ_i can be thought of as independent variables. Thus, nine distinct values of the dependent variable, p_i^*, and of the corresponding independent variables μ_i and σ_i, are used in equation 5 for estimating λ and τ. The weights used are $s_i/p_i^*(1-p_i^*)$ in which s_i is the number of plants sampled in the ith plot. This weighting accounts for the nonconstancy of variance associated with proportions. The nonlinear regression was performed using the Statistical Analysis System (21).

RESULTS AND DISCUSSION

Estimates of λ and τ obtained by the nonlinear regression procedure for the example above are presented in Table 2 along with confidence intervals for each estimate. Results are also presented for nonlinear regressions of disease incidence versus bacterial population frequencies estimated at two other dates in 1981 and one date in 1982. In each case the disease incidence estimates were made 4–11 days after bacterial population estimates were made. All regressions were highly significant based on the F test ($P<0.01$).

Several steps were taken to confirm further the appropriateness of using this model with these data sets. One set of bacterial population sizes did not conform to our assumption of lognormality (Fig. 1A ■). Nonlinear regression was performed excluding the nonlognormal data set. The resulting estimates of λ and τ were very close to the original values. Thus, the model was at least somewhat robust with respect to violation of the lognormality assumption. The robustness of the model was examined further by observing the effect of deleting single treatments at random for each of the dates and years. The values of the parameters estimated by the nonlinear regression procedure changed only slightly when any single treatment was excluded from the analysis.

Another tool used for evaluating the appropriateness of the regression model was to plot residual values against predicted values for the fitted regression equations. No consistent patterns were observed in the plots of residuals versus predicted values that would suggest lack-of-fit. For three of the nonlinear regressions, data from a single treatment led to a large residual value. In each of these cases deletion of that single treatment from the regression had minimal effect on the parameter estimates or on the significance of the regression equation. No consistent patterns were observed by plotting residuals against seed treatment, chemical treatment, μ or σ.

The results presented above indicate the model represented by equation 5 adequately related bacterial population frequency to disease incidence 4–11 days later. Although empirically it may be possible to relate mean epiphytic bacterial population size to disease, provided that changes in the variance associated with

TABLE 1. Population sizes of *Pseudomonas syringae* on individual snap bean leaflets and bacterial brown spot incidence on each of nine treatments

Spray treatment[a]	Seed treatment[b]	m[c]	k[c]	*P. syringae* population[d] size: Uncorrected[e] μ	Uncorrected[e] σ	Corrected for censoring[f] μ_a	Corrected for censoring[f] σ_a	Disease incidence[g] DAP 27	Disease incidence[g] DAP 31
Nonsprayed	1	29	29	6.762	0.492	6.762	0.492	0.582	0.567
	2	30	24	4.448	1.432	4.382	1.630	0.393	0.394
	3	28	14	3.283	1.492	2.472	2.449	0.181	0.190
Sprayed weekly	1	29	29	6.660	0.495	6.660	0.495	0.698	0.541
	2	29	25	4.333	1.291	4.282	1.401	0.345	0.435
	3	30	9	2.601	1.035	1.080	2.423	0.160	0.196
Sprayed-on ice nucleation trigger	1	28	28	6.636	0.362	6.636	0.362	0.704	0.526
	2	30	25	3.587	1.219	3.638	1.445	0.120	0.509
	3	30	12	2.764	1.095	1.854	2.070	0.148	0.075

[a] Spray treatments consisted of application of Kocide 606 at a rate of 1.89 L/ha (2 qt/acre) on the following schedules: not sprayed, sprayed weekly beginning 16 DAP, and sprayed when more than 5% of the leaflets bore ice nuclei active at or above −2.5 C (7).
[b] Seed treatments were: 1 = infected powdered bean leaves mixed with seeds at time of planting, 2 = commercially treated seeds, and 3 = seeds treated with streptomycin liquid suspension.
[c] m = number of leaflets sampled and k = number of leaflets with $\log_{10}$ CFU ≥ 2.2.
[d] Units are $\log_{10}$ (colony-forming units per leaflet) 23 days after planting.
[e] μ = mean and σ = standard deviation of $\log_{10}$ CFUs calculated by using a value of 2.2 for each censored datum.
[f] μ_a = mean and σ_a = standard deviation of $\log_{10}$ CFUs calculated by a maximum likelihood procedure accounting for censored data.
[g] Disease incidence expressed as proportion of leaflets with visible bacterial brown spot symptoms (DAP = days after planting).

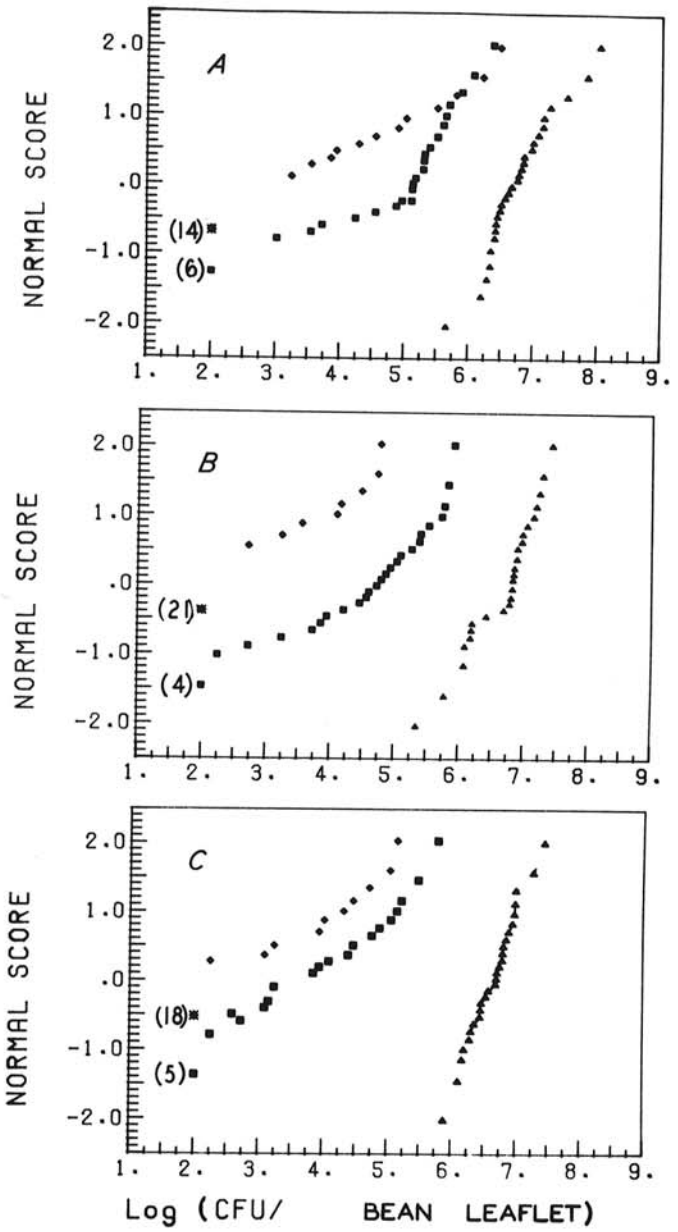

Fig. 1. Plot of logarithm (base 10) of fluorescent, oxidase-negative, ice-nucleation-active colony-forming units per leaflet obtained by dilution plating from cultivar Eagle snap bean leaflets 23 days after planting versus normal score (cumulative normal probability scale) for: **A,** nonsprayed treatments; **B,** treatments sprayed weekly with copper hydroxide; and **C,** treatments sprayed when more than 5% of leaflets had frozen. Data on each graph represent treatments planted with seed inoculated (◆); as obtained from commercial bags (■); treated with a streptomycin soak prior to planting (▲).

bacterial population size are small relative to changes in mean population, the model presented by equation 5 represents a more complete explanation for disease development as it relates to pathogen population size. This is true because the model explicitly incorporates three facts about disease occurrence. First, disease occurs on individual leaves or leaflets as the result of inoculum present on that leaf. Second, the frequencies of EPIPAB populations are described by the two parameters of the lognormal distribution. Third, the probability a leaf will become diseased, given that it harbors some number of bacteria, is defined by a specific dose-response relationship.

The model developed in this paper has four parameters, two characterizing the dose-response function (λ and τ) and two characterizing the bacterial population frequency on individual leaves (μ and σ). The parameters λ and τ are equivalent to the ED_{50} value and slope of the probit regression equation, respectively (5). Peto (18) has demonstrated mathematically that if bacterial cells are functioning independently during the infection process, then τ (slope of the probit function) should have a value of approximately 2.0 using the $\log_{10}$ population as the independent variable. Slope values of approximately 2.0 have been reported from infectivity titration experiments under controlled environmental conditions (4). The model presented in equation 5 provides a means of testing the concept of independent action under field conditions. Our results (Table 2) are consistent with the hypothesis of independent action since values of τ were not significantly different from 2.0 (4). By assuming $\tau = 2.0$ to be an estimate of λ, the ED_{50} value can be obtained independently. When the nonlinear regression procedure was used to estimate λ with τ set equal to 2.0 in equation 5, estimated values of λ were not significantly different from estimated values of λ in the original model with τ unconstrained.

TABLE 2. Estimates of the parameters λ (mean) and τ (standard error) in $\log_{10}$ units for the dose-response function (cumulative normal distribution) used in the model $\phi(x; \mu, \sigma, \lambda, \tau)$[a]

Year	DAP[b] Bacterial population	DAP[b] Disease	λ	τ	F[c]
1981	23	27	5.589 ±0.574[d]	2.747 ±1.131[d]	133.89
		31	5.780 ±1.293	4.747 ±3.110	69.30
	44	52	6.215 ±2.816	5.288 ±4.267	39.15
		55	5.079 ±1.426	3.522 ±2.598	35.50
	51	55	5.025 ±0.628	2.045 ±1.127	68.95
		60	4.959 ±0.834	1.843 ±1.471	31.99
1982	47	52	7.116 ±1.780	3.545 ±1.841	48.071
		55	5.016 ±0.655	2.300 ±0.984	82.309

[a] Estimates were obtained by nonlinear regression of proportion diseased bean leaflets versus μ and σ (the estimated mean and standard deviation of bacterial population size per leaflet).
[b] DAP = days after planting on which data were collected.
[c] Calculated F statistic for each nonlinear regression. All regressions had 2 degrees of freedom (df) for the model and 7 df for error.
[d] Asymptotic 95% confidence interval.

Specific parameters describe the expression of disease symptoms given the presence of known levels of inoculum (λ and τ) and separately the epiphytic pathogen population size (μ and σ). Thus, the model may be useful as a means of understanding the effects of environment and host on disease development and on pathogen population dynamics. Environmental parameters may influence susceptibility of the plant (λ or τ) independently of any effect on growth rate of epiphytic bacterial colonists or vice versa there may be environmental parameters that affect epiphytic bacterial growth without having any effect on susceptibility of the plant. The data sets analyzed represent sampling dates following different environmental conditions. Although variability was observed for estimates of λ, none of these values were significantly different from 5.5 (Table 2). This may suggest that environmental factors have a relatively greater impact on epiphytic population dynamics than on host susceptibility.

The host may resist disease by its influence on any one or a combination of the parameters λ, τ, μ, and σ. Daub and Hagedorn (3) found that epiphytic populations of *P. syringae* were lower on a field-resistant bean line compared with a susceptible cultivar. In their case host resistance apparently affected μ and/or σ. Many disease screening programs utilize seedling assays or inoculation of high populations of phytopathogenic bacteria onto plants in the greenhouse as a means of detecting resistant genotypes. The form of resistance usually being identified in these cases would most likely affect λ, the ED_{50} of the host.

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