Use of an Apparent Infection Threshold Population of *Pseudomonas syringae* to Predict Incidence and Severity of Brown Spot of Bean

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ABSTRACT

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A single seed lot of snap bean (*Phaseolus vulgaris*) was planted at 11 sites along an east-west transect in central Wisconsin. Epiphytic population sizes of naturally occurring *Pseudomonas syringae* pv. *syringae* pathogenic to bean (Psb) on symptomless bean leaflets differed among these sites. Bacterial brown spot was not detected at any site at which $\log_{10}$ (epiphytic Psb population size) was <4.0 on every bean leaflet sampled. Thus, 10^4 colony-forming units per gram of leaflet tissue may represent an apparent infection threshold population of Psb. The frequency with which Psb populations exceeded the apparent infection threshold level was estimated graphically. A model based on this frequency estimate was highly predictive of brown spot incidence 1 wk after full flower. The presence of very high Psb populations was a more reliable predictor of disease severity than was disease incidence. This predictive model based on infection threshold is presented as preferable to models based on mean pathogen populations because infections occur on individual plant parts, rather than on some theoretical mean plant part.

Although the existence of epiphytic populations of phytopathogenic *Xanthomonas* spp., *Erwinia* spp., and *Pseudomonas* spp. has been extensively documented (10), the relationship between these populations and the epidemiology of the diseases caused by these bacteria has remained unclear. Crosse (4) concluded that the number of epiphytic cells of *P. mors-prunorum* was not related to the incidence of bacterial leaf spotting, but was related to the incidence of bacterial canker on two cherry cultivars with different "inoculum potentials." Crosse (4) hypothesized that the number of bacteria washed from leaf surfaces by rain and deposited onto leaf scars must be greater than or equal to the minimum effective dose (as determined by infectivity titration in the laboratory) before infection could occur in the field.

Weller and Saettler (16) reported that at least 5×10^6 epiphytic *Xanthomonas campestris* pv. *phaseoli* per bean leaf were necessary for development of common blight symptoms in the field since this was the lowest population of *X. campestris* pv. *phaseoli* recovered from washings of individual leaves with visible lesions. However, because bacterial multiplication may occur in lesions, the epiphytic population size present before infection occurred may have been overestimated.

No significant differences among mean epiphytic pathogen population sizes at epidemic versus nonepidemic sites were reported for *Pseudomonas syringae* pv. *garcae* on coffee leaves (13) or for *Erwinia amylovora* on pear flowers (15). However, there was a significant correlation between the percentage of pear flowers colonized by *E. amylovora* and the incidence of fire blight. In cases where an average of one infection per tree occurred, mean populations were $\geq 7.6 \times 10^5$ colony-forming units (cfu) per flower (15).

Under laboratory conditions, a clear relationship between inoculum dose and disease has been established. Theoretically, one virulent bacterium can produce one lesion. This has been concluded from infectivity titrations in which the probability that one cell would produce disease symptoms did not vary over a range of inoculum concentrations (6). Nonetheless, this probability was usually small (0.006–0.17). Frequently, large doses of inoculum may be necessary to achieve bacterial infection under laboratory conditions (1,3,7,11). These minimum effective doses have been termed "infection thresholds" (11).

To determine whether disease incidence is quantitatively related to the size of the epiphytic pathogen population in the field, the pathogen must be detected before disease symptoms appear, and different population levels of the pathogen must be present at different locations or sites so that the amount of inoculum can be quantitatively related to disease, as it is during the infectivity titration procedure under controlled conditions. This may be accomplished by choosing sites at which naturally occurring pathogen populations differ (12) or by managing a disease epidemic with a combination of inoculation and chemical control procedures (9).

Mean epiphytic populations of *Pseudomonas syringae* pv. *syringae* that were pathogenic to bean (*Phaseolus vulgaris* L.) (Psb) on the susceptible cultivar Eagle were not consistently greater at four sites where brown spot epidemics occurred than were mean Psb populations at seven nonepidemic sites (12). Since the computation of both means and variances of pathogen population sizes is heavily influenced by leaflets for which pathogen population sizes were below the limit of sensitivity of our methods, the lack of significant differences between mean populations at epidemic versus nonepidemic sites is not strong evidence for a lack of a quantitative relationship between pathogen populations and disease incidence. Rather, it may indicate that pathogen populations on individual leaves, not mean pathogen

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populations, are more reliable predictors of disease incidence. The recent finding that epiphytic bacterial population sizes are usually lognormally distributed (8) has made calculations manageable for comparing pathogen population size and subsequent disease on individual leaves. This possibility was explored by reexamining our data on a per leaflet basis.

MATERIALS AND METHODS

The plot locations and the procedures for sampling and Psb population estimation were described previously (12). All bacterial population sizes are expressed as log-transformed values (eg, $\log_{10}$ [measured value]). In 1979, brown spot incidence (percentage of leaflets in the top third of the plant canopy showing lesions) and severity (average number of lesions per diseased leaflet) on beans in the experimental plots were assessed when the beans were ready for harvest. In 1980, brown spot was assessed six times between the first trifoliolate leaf stage and harvesttime. Every plant was examined in all the plots until the disease was first detected; subsequently incidence was estimated from ~250 leaflets. The severity estimate was based on lesion counts from ~30 diseased leaflets.

It has been demonstrated that population sizes of Psb on individual bean leaflets are lognormally distributed (8). Log values of Psb populations per leaflet were ranked and plotted on a probability scale at values of y (cumulative probability) expected for samples taken from a normal distribution. The expected frequencies of leaflets with Psb populations greater than or equal to a particular value were estimated graphically from these probability plots.

RESULTS AND DISCUSSION

The incidence and severity of bacterial brown spot at harvesttime for the 11 experimental plots are shown in Table 1. The plots were divided into three groups based on brown spot incidence in 1979: no detectable disease; disease incidence of 1–5%; and disease incidence of 25–70%. Psb population sizes (as $\log_{10}$ cfu/g fresh wt) associated with each individual leaflet at both flower and harvesttime in each of the three disease incidence groups are shown in Fig. 1. Epiphytic Psb population sizes did not exceed 4.0 on any bean leaflet sampled at the plots in which no disease was detected. In contrast, Psb population sizes exceeded 4.0 on at least one of eight leaflets from each of the six plots with disease incidence of at least 1%. Since epiphytic Psb populations were quantitated only on symptomless leaflets, this suggests that the likelihood of infection increased rapidly as Psb populations reached and/or exceeded values $\approx$ 4.0.

The frequency of leaves without detectable Psb decreased as disease incidence increased in these same groups. In the disease-free plots 85% (34 of 40) leaflets had no detectable Psb, versus 62.5% (10 of 16) and 41% (13 of 32) of the leaflets where the brown spot incidence was 1–5% and >25%, respectively (Fig. 1).

Comparing frequencies of high Psb populations to disease. Since epiphytic bacterial population sizes approximate a lognormal distribution (8), the expected frequency with which a given bacterial population size is met or exceeded among a group of leaflets can be estimated from a plot of the ranked log population data on a probability scale (2). This is illustrated for the 1979 data in Fig. 2. In those plots in which brown spot disease incidence was $\geq$25%, about 30% of the leaflets were estimated to have Psb population sizes $\geq$4.0, while only ~6% of the leaflets should have a Psb population that large in plots where the brown spot incidence was $\leq$5%. Thus, the frequency of symptomless bean leaflets with Psb population sizes at or above ~4.0 appeared to be related to the frequency of leaflets with brown spot lesions.

In 1980, the plots were divided into three groups: disease incidence of $\leq$2% (not diseased); disease incidence of 24–26% (moderately diseased); and disease incidence of 95–99% (severely diseased) at harvesttime. Data from each sampling date were plotted separately (Fig. 3). On 25 June 1980, when the beans were at the first trifoliolate leaf stage, epiphytic populations of Psb were detected on at least one leaflet in all three harvesttime disease incidence categories, but were not detected on 91, 50, and 87.5% of the samples from the not diseased, moderately diseased, and severely diseased categories, respectively (Fig. 3A). Log_{10} (Psb population size) exceeded 4.0 on a few leaflets in the not diseased and moderately diseased categories, but not in the severely diseased category. It is clear from these data that the inoculum level of Psb present early in the season was not predictive of brown spot

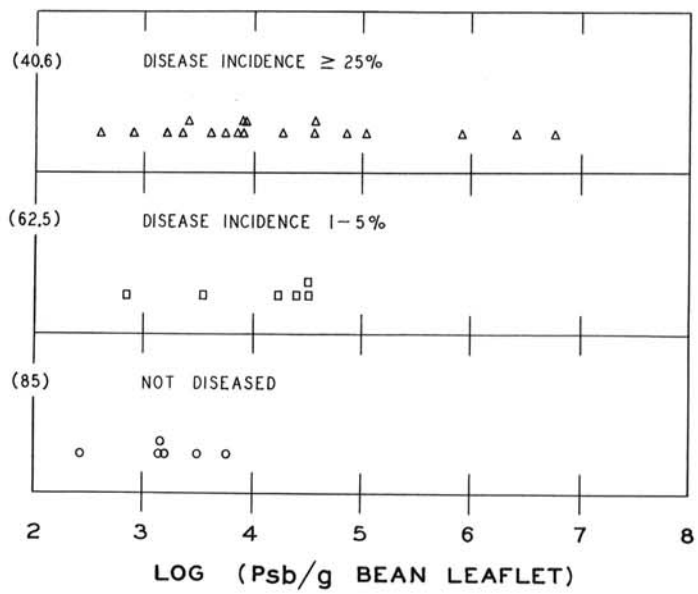

Fig. 1. Epiphytic populations of *Pseudomonas syringae* pv. *syringae* on individual symptomless cultivar Eagle bean leaflets at flowering and harvesttime in 1979 where bacterial brown spot incidence was $\geq$25% (△), 1–5% (□) and no brown spot was detected (○). The numbers in parentheses on the vertical axis at the left of the figure are the percentage of samples on which no Psb was detected.

TABLE 1. Incidence and severity of bacterial brown spot on cultivar Eagle snap beans at harvesttime in 1979 and 1980 at 11 experimental plots on an east-west transect in central Wisconsin

Year and character	Plot number[a]										
	1	2	3	4	5	6	7	8	9	10	11
1979											
Incidence[b]	1	0	0	30	70	25	30	0	5	0	0
Severity[c]	5	0	0	30	20	50	10	0	5	0	0
1980											
Incidence	0	2	0.4	99	95	24	26	0	0	0	0
Severity	0	4	5	30	31	11	10	0	0	0	0

[a] Plots 3 through 8 were located in the bean-growing area, plots 1 and 2 were west and plots 9 through 11 were east of the bean-growing area (12).
[b] Percent leaflets with lesions, estimated from ~250 leaflets in the top third of the canopy at each location.
[c] Average number of lesions per leaflet, estimated from ~30 diseased leaflets at each location.

incidence at harvesttime. Some of the Psb detected on first trifoliolate leaflets may have originated from the seed, but since populations of Psb differed at the different plot locations, either colonization of the seedling from seed varied among locations or sources of Psb other than seed were important (12).

On 7 July 1980, 1 wk before full flower, epiphytic populations of Psb exceeded 4.0 on symptomless bean leaflets from plots in all of the three disease categories (Fig. 3B). The percentage of leaflets with no detectable Psb had decreased in all disease categories in the 12 days between the samples illustrated in Fig. 3A and B. In general, the epiphytic inoculum load increased as the season progressed. However, as in 1979, the percentage of leaflets with no detectable Psb was smallest where subsequent brown spot incidence was greatest, and largest where subsequent brown spot incidence was least.

On 15 July, when the plants were in full flower, the distributions of Psb population sizes on individual leaflets (Fig. 3C) were similar to those occurring at flower and harvest in 1979 (compare Figs. 1 and 3C). Populations of Psb on samples from plots in the not diseased group were below the level of detection on 26 of 28 leaflets (93%) and none exceeded 4.0. Populations of Psb on samples from plots in the moderately diseased group were below the level of detection on 5 of 8 leaflets (63%) but exceeded 4.0 on all the remaining leaflets sampled. Of the leaflets from plots in the severely diseased group, only 1 of 8 (13%) had no detectable Psb, and 5 of 8 (63%) had Psb populations exceeding 4.0. By plotting these data on a probability scale, it was estimated that $\log_{10}$ (Psb population sizes) should have exceeded 4.0 on only 0.5% of the leaflets from the seven plots with brown spot incidence $<2\%$, but that such a population should have been exceeded on 50% of the leaflets from the moderately and severely diseased plots (Fig. 4).

On 22 July 1980, Psb populations were unexpectedly large in all three categories (Fig. 3D) but had declined to near the levels present at flowering time 6 days later (Fig. 3E). Since no comparably timed sample (between flower and harvest) was taken in 1979, it is not known if these changes in bacterial populations, which appeared to be transitory, were due to some change in the environment or to some change inherent to plant development.

Figure 5 represents the progress with time of the percentage of leaflets whose Psb populations exceeded 4.0 for the three different levels of brown spot incidence at harvest, 1980. The percentage of leaflets with Psb populations >4.0 increased steadily to 88% in plots where brown spot was severe, but fluctuated throughout the season without ever exceeding about 50% in plots where brown spot incidence was moderate. The increase in the percentage of symptomless leaflets with Psb $\geq$4.0 was more rapid in the plots that became severely diseased than in those that were moderately diseased. In addition, the progress of disease incidence (Fig. 6) lagged behind the growth of epiphytic Psb populations on symptomless leaflets.

A model that predicted disease. If epiphytic Psb is the immediate inoculum for brown spot disease, then, under any given set of environmental conditions the number of newly diseased leaflets at some time t_2 should be proportional to the number of leaflets with populations exceeding the apparent threshold at some earlier time, t_1. If prevailing environmental conditions were relatively similar from plot to plot at any given time across the central Wisconsin east-west transect in the week between 15 July and 22 July, then the frequency of "high" populations on 15 July (t_1) should be a predictor of new disease incidence on 22 July (t_2).

Thus, disease at time t_2 might be predicted as:

$$X_2 = X_1 + f_{(p\geq 4)}[1 - (X_1/100)]$$

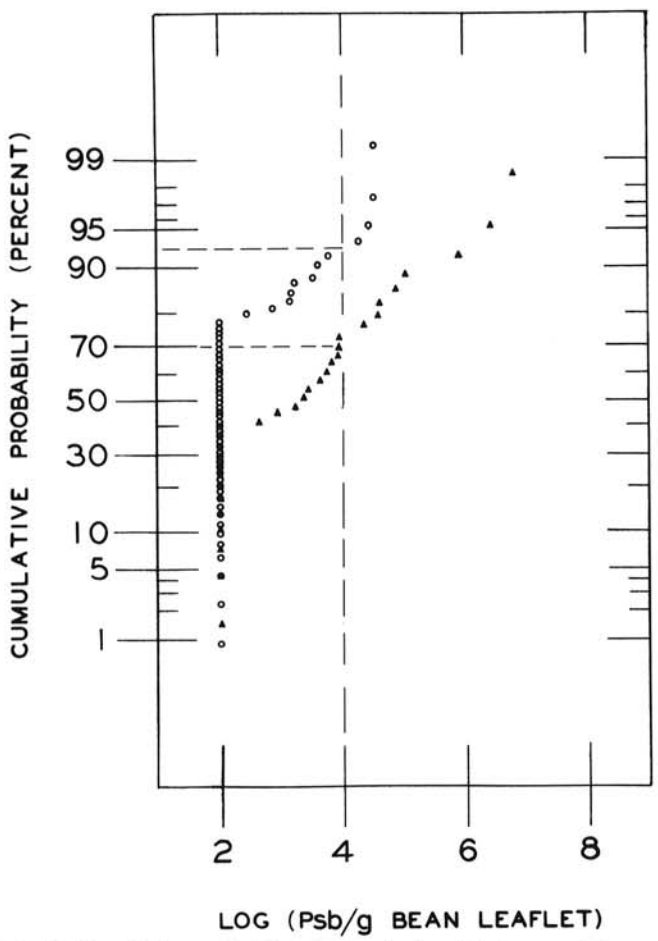

Fig. 2. Cumulative probability (percent) of epiphytic populations of *Pseudomonas syringae* pv. *syringae* on individual symptomless cultivar Eagle bean leaflets at flowering and harvesttime in 1979 where bacterial brown spot incidence was $\geq$25% (▲) and $\leq$5% (O). Values plotted at 2.0 represent leaflets on which Psb was not detected.

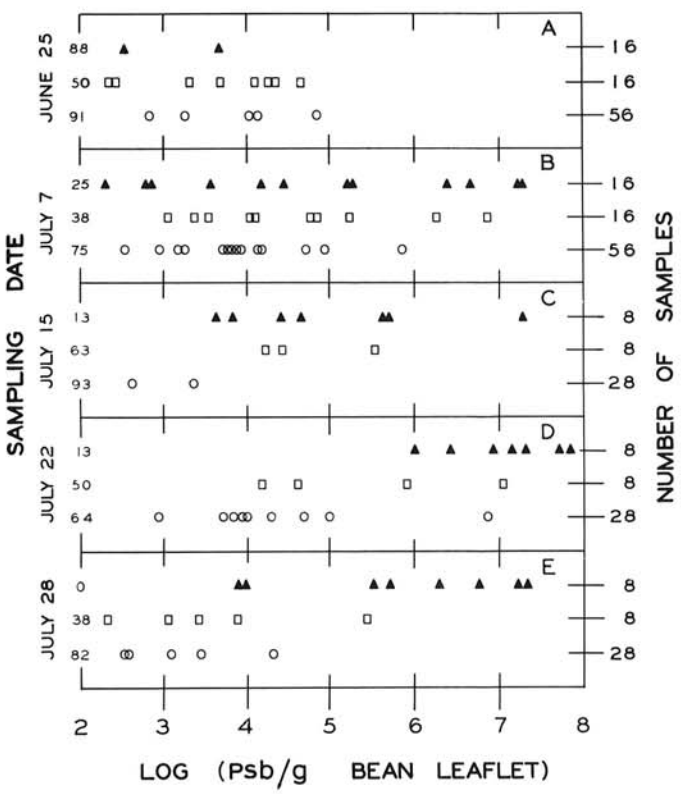

Fig. 3. Epiphytic populations of *Pseudomonas syringae* pv. *syringae* on individual symptomless cultivar Eagle bean leaflets on five sampling dates in 1980, where the bacterial brown spot incidence at harvesttime was severe, 95–99% (▲); moderate, 24–26% (□); and not diseased, 0–2% (O). The numbers on vertical axis at left of figure represent the percentage of samples on which Psb was not detected.

in which X_2 and X_1 are percent disease at times t_2 and t_1, $f_{(p \geq 4)}$ is the frequency (as percentage) with which Psb populations on individual symptomless leaflets exceed 4.0 at time t_1 in a given plot, 4.0 is an estimate of the apparent threshold, and $1-(X_1/100)$ is the proportion of leaflets that are symptomless at t_1. In simple terms, the brown spot incidence at time t_2 should be equal to the brown spot incidence at time t_1 plus the new infections that occur on symptomless leaflets where the epiphytic Psb population exceeds the infection threshold.

The frequency with which Psb exceeded 4.0 on leaflets in 1980 was determined graphically on a probability scale by using Psb populations from four leaflets for each of the six locations where Psb populations were detected. At the remaining five plot locations, $f_{(p \geq 4)}$ was assumed to be zero. The actual disease incidences at 15 and 22 July were measured quantities. The model was highly predictive of measured disease incidence on 22 July ($R^2 = 0.939$, $P < 0.001$).

Mean populations of Psb per plot on 15 July also were highly predictive of brown spot incidence on 22 July ($R^2 = 0.903$, $P < 0.001$). For this data set, mean Psb populations and the frequency of high populations on individual leaflets were closely related ($R^2 = 0.988$, $P < 0.001$). However, since infections occur on individual leaflets and not on some hypothetical average leaf, it seems appropriate to relate disease incidence to pathogen populations on individual leaves rather than to mean pathogen populations. In either case, the probability of infection increased linearly with the log Psb population size, and the probability that any one Psb cell would cause an infection was quite small.

Interpretation of the model. That our data fit the "model" as well as they do suggests that a simple threshold concept may well be applicable. Surely, however, it would be naive to assume that there is some pathogen population size that, if met or exceeded, must always lead to disease, but if not met will never produce disease. Indeed, this is not the case (6). However, if the probability of disease is near zero at some population size and increases to near one at some larger population, and if this increase in probability of disease for a given population size is moderately symmetrical, then at some population size near the midpoint of this shift in conditional probability the frequency for diseased leaves caused by populations below that value will be equal to the frequency of healthy leaves associated with larger populations. If such a population size replaced threshold in the simple threshold model, the data should fit the model very well. Thus, we propose that the threshold may resemble an ED_{50} for epiphytic Psb populations. A model for quantitative evaluation of the ED_{50} values for these epiphytic populations, and for determining the distribution of the conditional probabilities of disease given population size from field data is under development (14).

We do not mean to imply that 4.0 is an invariant infection threshold or that Psb population size was the only factor determining brown spot incidence. The model adequately predicts brown spot incidence when Psb populations at flowering time are used. This supports the hypothesis that epiphytic Psb populations greater than a threshold level are a primary determining factor in brown spot incidence. However, the model greatly overestimates disease incidence in most cases if Psb populations on leaflets prior to flowering are used. This suggests that the apparent infection threshold is not a constant and may be related to changes in host susceptibility, for example, those changes that may occur with the onset of flowering and seed production. The model does assume

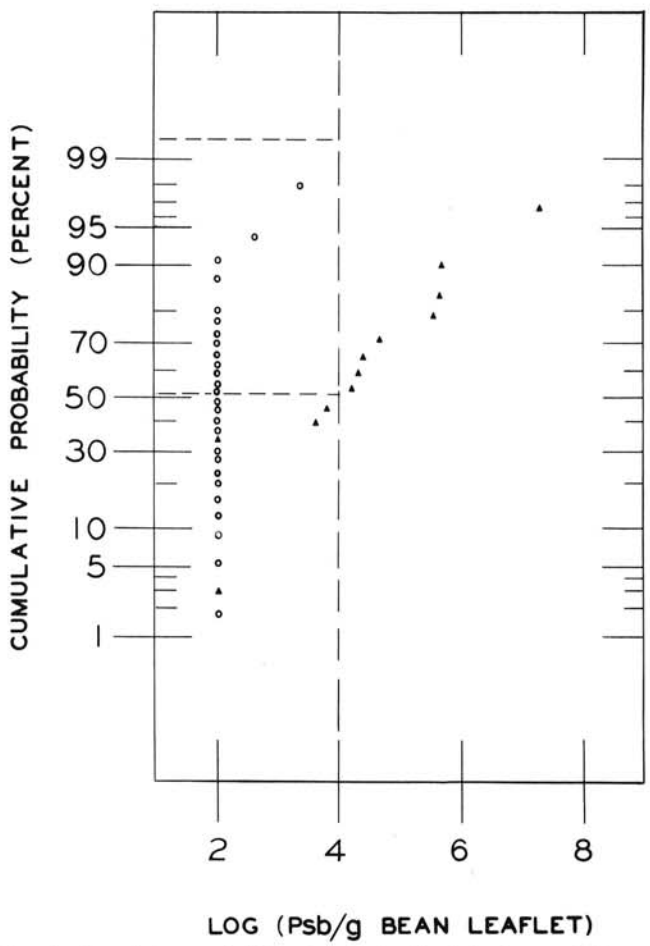

Fig. 4. Cumulative probability (percent) of epiphytic populations of *Pseudomonas syringae* pv. *syringae* on individual symptomless cultivar Eagle bean leaflets 15 July 1980, where the harvesttime bacterial brown spot incidence was moderate to severe, 24–99% (▲), and not diseased (O). Values plotted at 2.0 represent leaflets on which Psb was not detected.

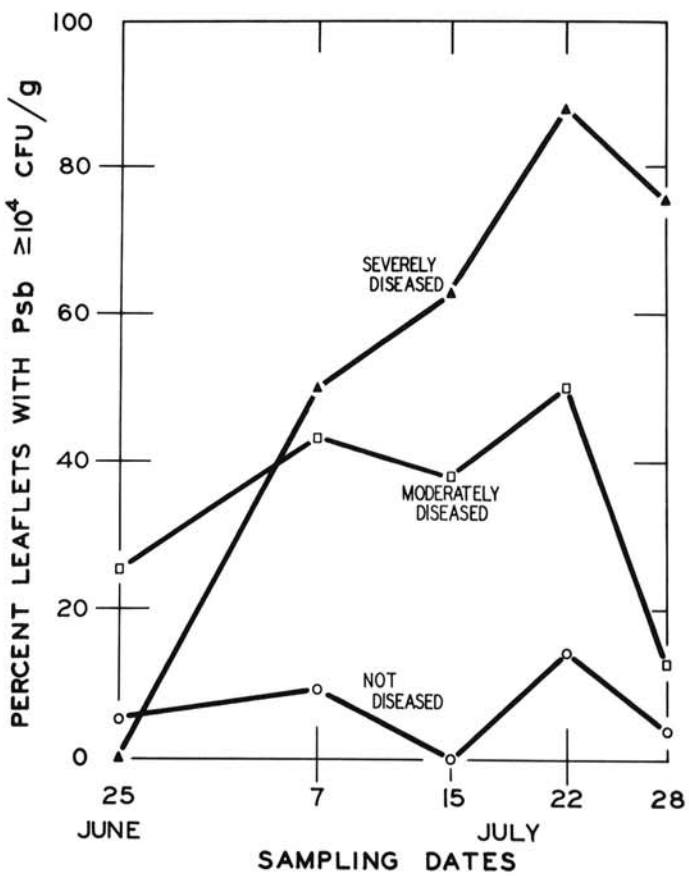

Fig. 5. The frequency (as percent) with which populations of *Pseudomonas syringae* pv. *syringae* on individual symptomless cultivar Eagle bean leaflets exceeded 10^4 cfu/g on five sampling dates in 1980 relative to the bacterial brown spot disease rating at harvest: plots 4 and 5, severely diseased (▲); plots 6 and 7, moderately diseased (□); the other seven plots, not diseased (O). The beans were at the first trifoliolate leaf stage on 25 June, in full flower on 15 July, and were ready for harvest on 28 July 1980.

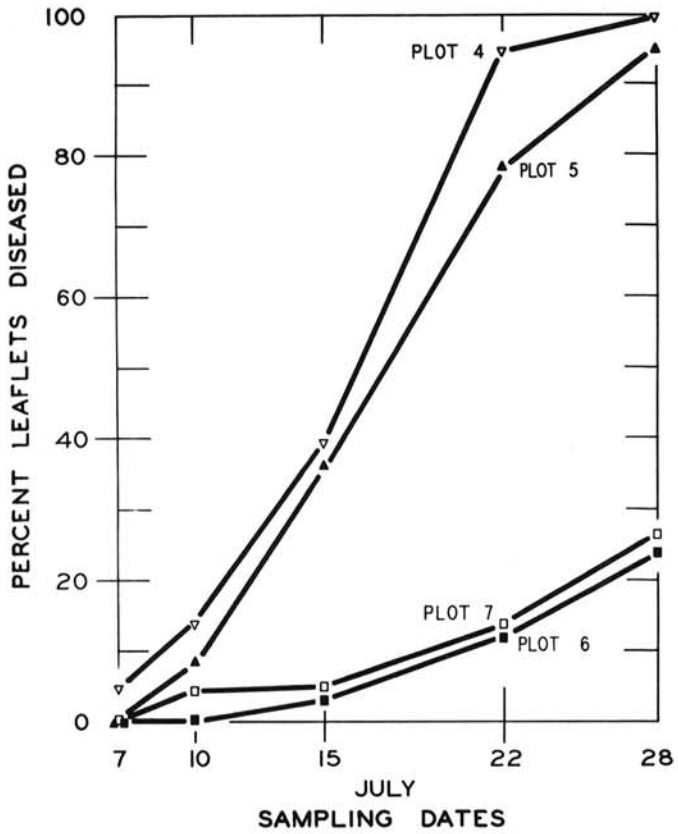

Fig. 6. Progress of bacterial brown spot incidence (percentage of leaflets with lesions) on cultivar Eagle snap bean leaflets in 1980 at four experimental plots in the bean-growing area of the central Wisconsin east-west transect. Brown spot incidence was estimated from ~250 leaflets examined in the top third of the plant canopy.

that the proportionality constant between leaflets with high populations and subsequent disease is unity. Obviously, this is not uniformly correct, since some leaflets early in the season had $\log_{10}$(Psb populations) >4.0 and never did develop disease. The physical environment probably is important for this parameter. However, since the model does not underestimate disease incidence, it is unlikely that infection occurs when high Psb populations are not present.

Epiphytic Psb population sizes and disease severity. If epiphytic Psb population levels on symptomless leaflets are quantitatively related to the likelihood of a leaflet bearing at least one lesion, then Psb populations also may be predictive of the number of brown spot lesions per diseased leaflet or disease severity. This idea was supported by a highly significant correlation between the largest Psb population detected at either flowering or harvesttime and the mean brown spot severity at harvest at the six plots where brown spot was detected (Table 2). If the leaflet with the largest Psb population from each sample is representative of the field leaflets with the largest Psb populations, and if these leaflets develop more lesions than leaflets with lower Psb populations, then they should make a disproportionately large contribution to the mean disease severity. In general, disease severity was correlated more consistently with Psb populations than with disease incidence.

Epiphytic populations of Psb as the immediate source of inoculum for bacterial brown spot disease. Our data support a conceptual model in which the occurrence of lesions depends upon the size of epiphytic populations of the pathogen on individual bean leaflets (14). Bacterial brown spot was not detected at plots where the $\log_{10}$ (Psb populations) on the individual bean leaflets sampled were <4.0 on every sampling date. The incidence of brown spot could be predicted from the frequency of leaflets with $\log_{10}$(Psb population) ≥4.0 at flowering time. Lastly, the mean severity at harvesttime was positively correlated with the largest Psb population detected on an individual leaflet from each plot.

TABLE 2. Comparison of regression analyses of bacterial brown spot incidence versus epiphytic populations of *Pseudomonas syringae* pv. *syringae* pathogenic to bean (Psb) on symptomless bean leaflets as predictors of brown spot severity

	Correlation of severity[a] with:				
	Disease incidence[b]			Psb population[c]	
Year	df	*r*	*P*	*r*	*P*
1979[d]	4	0.54	NS	0.94	<0.01
1980[e]	4	0.97	<0.01	0.93	<0.01
1979 & 1980	10	0.71	<0.01	0.86	<0.001

[a] Mean number of lesions per diseased leaflet.
[b] Fraction of leaflets with lesions. NS = not significant.
[c] Largest Psb population detected on a symptomless bean leaflet at flowering or harvesttime.
[d] Plots 1, 4, 5, 6, 7, and 9 as described in Table 1.
[e] Plots 2, 3, 4, 5, 6, and 7 as described in Table 1.

The question arises as to whether Psb populations >4.0 on leaflets are actually latent infections or incipient lesions and no longer merely epiphytes. This was probably not the case, at least at those four locations where large Psb populations were detected on bean leaflets early in the season yet no brown spot lesions were evident for the following 5 wk. It is improbable that infections, once initiated, would develop into visible lesions on bean leaflets only in plots within the bean-growing area. Apparent fluctuations in Psb population sizes that occurred before flowering time may have been due either to changes in population levels on individual leaflets (which were not measured) or differences in the amount of colonization of new leaves, since samples were taken from the top third of the canopy rather than from the whole plant on each sampling date. However, after flowering time fewer new leaves are formed on the determinant plants. Therefore, the apparent Psb population fluctuations that occurred between 15 and 22 July, and between 22 and 28 July 1980, probably represent real changes in population levels on individual leaflets. If the large Psb populations present on leaflets in plots 3, 8, and 10 were due to latent infections, one would not expect these populations to have declined between 22 and 28 July 1980.

With respect to Psb on bean, it appears that a susceptible plant may harbor relatively large epiphytic pathogen populations and remain symptomless (5). Given the ubiquity of *P. syringae* and its typically large epiphytic populations, it is doubtful that any host plant would survive if it were truly susceptible to the epiphytic presence of one virulent bacterial cell. Therefore, even a highly susceptible cultivar, such as Eagle, may have some measure of resistance to infection by Psb that may be quantitatively expressed in terms of the largest epiphytic Psb population size that may occur under optimum environmental conditions before lesion development becomes highly probable. In addition, since large epiphytic populations of phytopathogenic bacteria commonly occur on leaves of symptomless host plants, and since the probability of infection associated with a single virulent bacterial cell is much less than unity for several host-pathogen combinations (6), perhaps the expression of the gene(s) for pathogenicity in a cell of a phytopathogenic bacterium is a rare event.

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