European Journal of Plant Pathology **106:** 339–346, 2000.

Symptomless spread of blight-inducing strains of *Xanthomonas campestris* pv. *campestris* on cabbage seedlings in misted seedbeds

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Accepted 4 January 2000

Key words: bacteria, black rot, black veins, ELISA, epidemiology, latent spread

Abstract

Xanthomonas campestris pv. *campestris* induces two types of symptoms, namely, black rot and blight. Black rot symptoms are V-shaped lesions and black veins on the leaf, and blight symptoms are sudden collapse of interveinal tissues following the lack of veinal necrosis at early stages of infection. These two symptoms can occur simultaneously. However, the tendency to induce either symptom type is strain-dependent. Six strains were evaluated for their rate and pattern of spread in misted seedbeds by using strain-specific monoclonal antibodies and miniplate enrichment/ELISA. Data on pathogen incidence was defined as the presence of the pathogen in or on plants rather than visual symptoms. The results indicated that blight-inducing strains spread to more seedlings than black rot-inducing strains. The high incidences of blight-inducing strains in experimental plots were associated with non-randomness of spatial pattern of pathogen spread, indicating that high incidence is primarily due to the spread from adjacent plants by leaf contact and water splash. Most ELISA-positive seedlings were symptomless, indicating that the sensitivity of the system used in this study was adequate for detection of latent or epiphytic spread.

Introduction

Symptoms of black rot of crucifers, caused by *Xanthomonas campestris* pv. *campestris*, are typically described as V-shaped chlorotic lesions on leaf margins accompanied by black veins, indicative of systemic invasion (Williams, 1980). The main infection sites are hydathodes (Cook et al., 1952; Russell, 1898; Smith, 1897) although stomatal invasion was also observed (Clayton, 1925). Strains of *X. c. campestris* vary with respect to serological characteristics (Alvarez et al., 1994; Schaad, 1978), phage type (Liew and Alvarez, 1981), fatty acid analysis (Stead, 1989; Vauterin et al., 1992; Yang et al., 1993), genetic characteristics (Alvarez et al., 1994), and the symptoms produced on crucifers (Alvarez et al., 1994).

In contrast to typical foliar symptoms of black rot, blight symptoms (sudden collapse of interveinal tissues and the lack of veinal necrosis at early stages of infection) were observed in cabbage (*Brassica oleracea* var. *capitata* L.) fields in Hawaii (Yuen and Alvarez, 1985). Strains producing these symptoms were considered highly virulent variants of *X. c. campestris* (Alvarez et al., 1987). The blight strains also had different reactivity patterns with monoclonal antibodies (MAbs) and formed distinct groups by restriction fragment length polymorphism (RFLP) analysis (Alvarez et al., 1994).

Variability among *X. c. campestris* strains with respect to their capacity for spread was observed in field plots of cabbage on the island of Oahu, Hawaii (Yuen et al., 1987). Assessment of spread in the field was based on the increase of visually detectable symptoms on plants followed by strain identification with pathovar-specific MAbs. Black rot strains that appeared to be similar based on plant inoculations in greenhouse tests, spread at different rates in the field (Yuen et al., 1987). Although the reliance on symptom

expression underestimates the actual spread of the pathogen, the results demonstrated that different strains of *X. c. campestris* that cause typical black rot symptoms varied in their capacity for field spread.

The present study was conducted to examine the epidemiological potential of strains of *X. c. campestris* that cause blight symptoms and to determine whether they spread more rapidly in cabbage seedbeds than strains which cause black rot. In order to accurately assess pathogen spread, the incidence data were based on the presence of *X. c. campestris* cells on leaves, rather than relying on symptom expression. Miniplate enrichment enzyme-linked immunosorbent assay (ELISA) (Norman and Alvarez, 1994) was used with *X. c. campestris*-specific MAbs to detect the pathogen on symptomless plants.

Materials and methods

Bacterial strains

The *X. c. campestris* strains used in this study were described previously (Alvarez et al., 1994). Three strains (GAC17, GAC137, and A249) were in Serogroup 1, RFLP group 1, and reported to cause typical black veins, or black rot symptoms; three other strains (G2-12, G2-17, and Xcc528^{T}) were in Serogroup 3, RFLP group 3, and were reported to induce mesophyll collapse on leaves and induce blight symptoms (Alvarez et al., 1994). Strain Xcc528^{T} (ATCC33913) is the type strain of the species isolated from brussels sprouts in Great Britain. Strain GAC137 was originally isolated from lettuce in Hawaii but produced black rot on cabbage. The other strains were isolated from cabbage on the island of Maui, Hawaii. All strains were maintained on yeast extract–dextrose–calcium carbonate (YDC) medium (Wilson et al., 1967) at 28 °C. Their reactivity patterns to MAbs X21 and A11 were confirmed (Alvarez et al., 1994) prior to the experiments.

Symptom type assessment for X. c. campestris *strains*

Cabbage plants that were approximately 6 weeks old (cultivar Tastie, Northrup King, Gilroy, California) were inoculated with 2-day-old YDC plate cultures of *X. c. campestris* strains suspended in phosphate-buffered saline (PBS) at a concentration of approximately 3×10^{8} CFU ml^{-1}. Leaf margins were clipped with sterile scissors dipped in the bacterial suspension. Four plants were used for each strain of *X. c. campestris* with four cuts on each plant. Plants were placed on greenhouse benches at a mean temperature of approximately 28 °C. Symptom types (black veins or blight) were recorded 17 days after inoculation at each inoculation site. If a symptom consisted of both black veins and blight, a predominant symptom type was recorded. The blight index (number of inoculation sites whose predominant symptom was blight ÷ total number of inoculation sites (16)) was determined for each strain.

Experimental design

We performed two experiments (Experiment I and Experiment II) with different misting conditions and sampling dates. Each experiment consisted of two runs, with two replications in each run.

One cabbage (cultivar Tastie) seed was planted in each cone of twelve trays. Trays were 60 cm long, 35 cm wide, and 11.5 cm deep (Stuewe and Sons, Corvallis, Oregon). Each tray had 96 (12 × 8) cones (diameter 4 cm) that corresponded to the configuration of microtiter plate wells. The planting medium was a mixture of Supersoil® (Rod McLellan, South San Francisco, California) and vermiculite (Thermo-o-rock Industries, Chandler, Oregon) (1 : 1 by volume) fertilized with Osmocote® (18-6-12) (Plantco, Bramalea, Ontario, Canada) at 10 g l^{-1}. Trays were placed on a greenhouse bench with a misting system in a completely randomized design. Mean temperatures ranged from 26 to 30 °C. Each tray was considered an experimental unit.

In Experiment I, plants were misted every 5 min for 10 s between 7 and 8:30 a.m., and between 4 and 5 p.m. In Experiment II, misting was every 4 min for 5 s between 7 a.m. and 7 p.m. Fourteen days after seeding, four seedlings at the center of each tray were inoculated by cutting the leaf margin with scissors dipped in a bacterial suspension of approximately 3×10^{8} CFU ml^{-1} determined spectrophotometrically (optical density $A_{600} = 0.1$). Trays were separated by transparent plastic sheets to avoid cross contamination. In Experiment I, strains A249 (typical black rot strain) and G2-12 (blight strain) were used as inoculum. In Experiment II, three typical black rot strains (A249, GAC137, and GAC17) and three blight strains (Xcc528^{T}, G2-12, and G2-17) were used.

Sampling/miniplate enrichment

Pathogen incidence data were taken at 2, 9, 16 and 23 days after inoculation in Experiment I. In Experiment II, data were taken at 2, 8, and 14 days after inoculation. Pathogen incidence was assessed by collecting from each plant leaf wash water (50 µl) that remained on leaves after misting. Leaf wash water from 32 clean seedlings were used as negative controls for ELISA. Leaf wash water samples were incubated for 5 days at 28 °C in individual wells of microtiter plates containing esculin–trehalose medium (Norman and Alvarez, 1994) to selectively amplify *X. c. campestris* cell populations (miniplate enrichment).

ELISA

After incubation, 100 µl of coating buffer (carbonate–bicarbonate buffer, pH 9.6) was added to each well of the microtiter plates, which were enriched for *X. c. campestris*. Approximately 10 min after the addition of the buffer, 50 µl of the supernatants were transferred to new microtiter plates. The plates were dried overnight at 37 °C in an incubator with circulating air. The pathogen was detected by ELISA using MAb A11 (for Xcc528^T, G2-12, and G2-17) or X21 (for A249, GAC17, and GAC137) with horseradish peroxidase-conjugate as the secondary antibody and 5-aminosalicylic acid as the substrate (Alvarez and Lou, 1985). Absorbance was read at 450 nm. Samples with absorbance of 0.25 or greater above the mean of the negative control readings were considered positive. Inoculated plants were not included in data analyses. In Experiment II, visual symptoms were also recorded at final sampling days.

The existence of possible cross contamination from neighboring experimental units was assessed at the second sampling of the first run of Experiment II. Plants inoculated with A11-positive strains were tested by MAb X21, and plants inoculated with X21-positive strains were tested by MAb A11 (reciprocal MAb assay).

Data analysis

The four replications in the two runs of each experiment was combined for statistical analyses. Pathogen incidence was defined as the percentage of ELISA-positive plants. Temporal changes in pathogen incidence were analyzed by calculating areas under the disease progress curve (AUDPC). Incidence at the final sampling date was analyzed by performing analysis of variance (ANOVA). Means were compared among strains using the Fisher's least significant difference.

For Experiment II, deviation from randomness of the pathogen occurrence patterns was analyzed by performing 'runs analysis' for each tray (Madden et al., 1982; Campbell and Madden, 1990). This analysis is appropriate when the plants are arranged in rows and the measurements are discrete and binary (positive or negative) (Campbell and Madden, 1990). The runs were counted by reading a top row (as appeared in the pathogen incidence maps) from left to right, then continuously from right to left on the next row, and so on. Inoculated seedlings were not included. Deviation from randomness was determined by calculating $Z = [U + 0.5 - E(U)]/s(U)$, where U is the number of runs. $E(U)$ is the expected number of runs under the null hypothesis of randomness and given by $E(U) = 1 + 2m(N - m)/N$, where m is the number of ELISA-positive plants and N is the total number of plants; and $s(U)$ is the standard deviation of U under the null hypothesis of randomness given by $s(U) = \{2m(N-m)[2m(N-m)-N]/N^2(N-1)\}^{1/2}$. The value of Z less than -1.64 was considered the indication of clustering ($P = 0.05$). The correlation of the values of Z and the ratios of ELISA-positive plants were also analyzed.

Results

Symptom type assessment for X. c. campestris *strains*

Chlorosis at inoculation sites appeared about 5 days after inoculation. Symptoms of most strains consisted of both blight and black veins with chlorosis rather than a single distinct type. However, strain G2-17 caused blight symptoms only, and the symptoms made by A249 consisted of black veins and chlorosis only. Reactivity with MAbs X21 and A11, symptoms on cabbage, and blight indices are summarized in Table 1.

Temporal increase in pathogen incidence

The pathogen incidence increased linearly over the experiments (Figure 1). Higher incidences for blight strains (G2-17, G2-12, and Xcc528^T) were apparent at early stages of the spread and were sustained until the end of both experiments (Figure 1). The spreading was quantitatively measured by calculating areas under

Table 1. Strains of *Xanthomonas campestris* pv. *campestris* used in this study, reactivity with MAbs A11 and X21, the symptoms induced on 6-week-old cabbage plants, and blight indices

Strain	MAb A11	MAb X21	Symptom	Blight index[a]
G2-17	+	−	Blight[b]	1.00
G2-12	+	−	More extensive blight symptoms than black veins[c]	0.63
Xcc528^T	+	−	More extensive blight symptoms than black veins	0.50
GAC17	−	+	Blight and black veins[d]	0.38
GAC137	−	+	Predominantly black veins with little blight	0.25
A249	−	+	Black veins only, no blight	0.00

[a](Inoculation sites that showed predominantly blight) ÷ (total number of inoculation sites).
[b]Blight symptoms refer to sudden collapse of interveinal tissues.
[c]Black veins were accompanied by typical V-shaped zones of chlorosis.
[d]Described by Hunter et al. (1987) as intermediate.

the disease progress curve (AUDPC). In Experiment I, AUDPC for the blight strain G2-12 was 48.6% · days and was significantly ($P = 0.05$) greater than that for the typical black rot strain A249 (2.5% · days). In Experiment II, AUDPCs for the three blight strains (G2-17, G2-12, and Xcc528^T) were significantly ($P = 0.05$) greater than those for two of the typical black rot strains tested (A249 and GAC137) (Table 2), indicating their faster spreading ability under the experimental conditions used in this study. GAC17 showed intermediate spreading ability.

Incidence assessment at final sampling days

In Experiment I, strain G2-12 spread to significantly greater numbers of plants than A249 by the final sampling days. The final incidence for G2-12 and A249 was 14.3% and 1.1%, respectively, and this difference was statistically significant at $P = 0.05$. In Experiment II, three blight strains, G2-12, G2-17, and Xcc528^T, spread to significantly greater numbers of plants than A249 and GAC137. Strain GAC17 showed intermediate spread. In the reciprocal MAb assay at the second sampling of the first run of Experiment II, no positive reactions were detected by MAb A11 in samples from plants inoculated with X21-positive strains, and vice versa, indicating that no cross contamination had occurred. Final pathogen incidence data for Experiment II are summarized in Table 2. Maps of pathogen incidence at the final sampling day are shown in Figure 2.

Symptom evaluation at final sampling days

Symptoms that developed on seedlings in the misted seedbeds were often atypical, including pinpoint watersoaked lesions and leaf rot, although most symptoms were consistent with those described in Table 1. At the final sampling days in Experiment II, less than 15% of the ELISA-positive plants expressed visual symptoms. The ratios of seedlings with visual symptoms to total ELISA-positive seedlings are shown in Table 2.

Runs analysis

Z values smaller than -1.64 indicate clustering at $P = 0.05$ (Campbell and Madden, 1990); 7 out of 11 (63.6%) experimental units for the blight strains showed clustering defined by the low Z values, as compared to only 2 out of 12 (16.7%) for the typical black rot strains, suggesting the non-random spatial distribution of the blight strains. Low Z values were correlated with high incidence ($R^2 = 0.597799$) (Figure 3). Observed runs and Z values for each experimental unit are summarized in Table 3.

Discussion

The extensive necrosis referred to as blight was associated with serologically and genetically distinct strains of *X. c. campestris* (Alvarez et al., 1994). In current studies, there was a continuum of symptom expressions ranging from typical black rot to blight. Therefore, a blight index system was developed to quantitatively describe a potential for a particular *X. c. campestris* strain to induce blight in cabbage. The blight index appears to be positively correlated with rapid spread in the seedbed. The pathogen incidence of both typical black rot and blight strains increased linearly. However, the blight strains spread more rapidly than the typical black rot strains in the seedbeds under misted

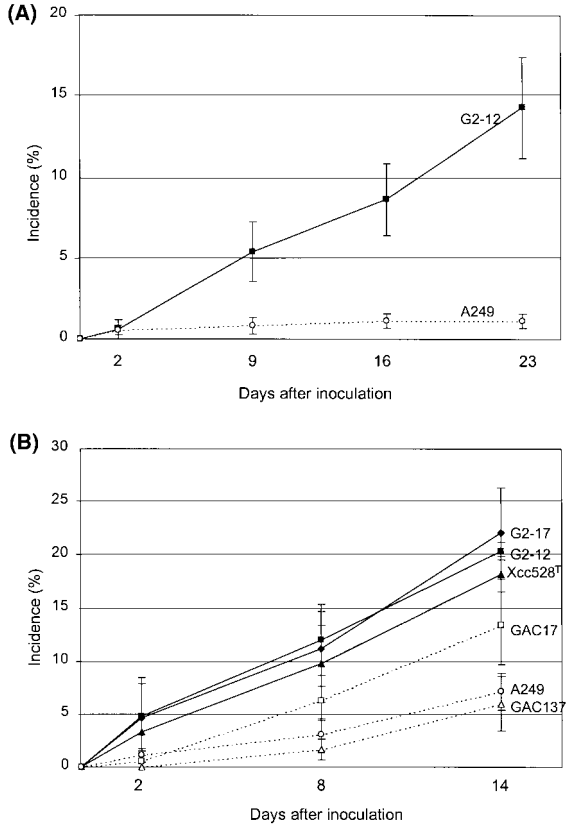

Figure 1. Temporal increase in percent pathogen incidence on cabbage seedlings inoculated with typical black rot strains (GAC17, GAC137, and A249) and atypical blight strains (G2-17, G2-12, and Xcc528^T) of *X. c. campestris*. Percent incidence is defined as percentage of ELISA-positive seedlings among all the seedling samples. Data are means of four replications (two replications in each of two runs combined) and the bars indicate standard errors of the mean. A, Experiment I; B, Experiment II. Closed square, G2-12; closed diamond, G2-17; closed triangle, Xcc528^T; open circle A249; open square, GAC17; open triangle, GAC137. A solid line and a dashed line indicate a blight strain and a black rot strain, respectively.

conditions used in this study. The greater epidemiological potential of this distinct group of *X. c. campestris* strains is thus strongly suggested.

Interestingly, high pathogen incidence was correlated with high non-randomness of the spatial pattern of pathogen spread based on runs analysis. This indicates that the high incidence observed in this study was primarily due to spread to adjacent plants, suggesting the importance of spread by direct contact of leaves or water splash. Contribution of aerosols to the dispersal of the pathogen appears to be less important

Table 2. Pathogen incidence at the final sampling day, AUDPC, and the numbers of seedlings that expressed symptoms among ELISA-positive seedlings at the final sampling days in Experiment II

Strain[a]	Pathogen incidence (%)	AUDPC (% · days)	Symptoms[b]
G2-17	22.1 a	57.0 a	19/80 (23.8%)
G2-12	20.4 ab	51.6 a	10/57 (17.5%)
Xcc528^T	18.2 ab	48.1 a	6/65 (9.2%)
GAC17	13.4 bc	39.1 ab	4/51 (7.8%)
A249	7.1 c	19.2 b	4/26 (15.4%)
GAC137	6.0 c	18.0 b	1/21 (4.8%)

Pathogen incidence and AUDPC data represent the means of four replications (two replications in each of the two runs). Figures followed by the same letter in a column are not significantly different at 5% rejection level according to the Fisher's least significant difference test. [a]G2-12, G2-17, and Xcc528^T are blight strains, and GAC17, A249, and GAC137 are typical black rot strains. [b]Overall, the ratio was 44/300 or 14.7%.

than direct contact of leaves or water splash. Aerosol spread could have caused cross contamination with the pathogen from adjacent trays, but such contamination was not observed in the reciprocal antibody assay.

Misting and dense planting of seedlings increased the potential for bacterial spread in the seedbed, especially in Experiment II, in which misting was applied for a longer period of time. The increase in incidence of pathogen was often greater than 1% per day (Figure 1B), and the blight strains in particular, showed high dispersal in every experiment.

The majority of the ELISA-positive plants were symptomless. The miniplate enrichment/ELISA method was sufficiently sensitive to detect the latent or epiphytic spread of the pathogen in the misted seedbed. Thus, the actual spread of the pathogen extended beyond plants with visual symptoms. These results are consistent with an earlier report (Mochizuki and Alvarez, 1992) that viable *X. c. campestris* cells were detected in guttation droplets on symptomless leaves of cabbage seedlings 9 and 20 days after inoculation of roots or seeds with the pathogen. To the best of our knowledge, the epidemiological application of MAbs along with pathogen enrichment for the detection of bacterial pathogens on symptomless plants has not been reported before. Our method could be easily applied to other host–pathogen combinations if appropriate antibodies and selective media were available.

Multiplication and spread of the pathogen in the absence of symptoms may play an important role

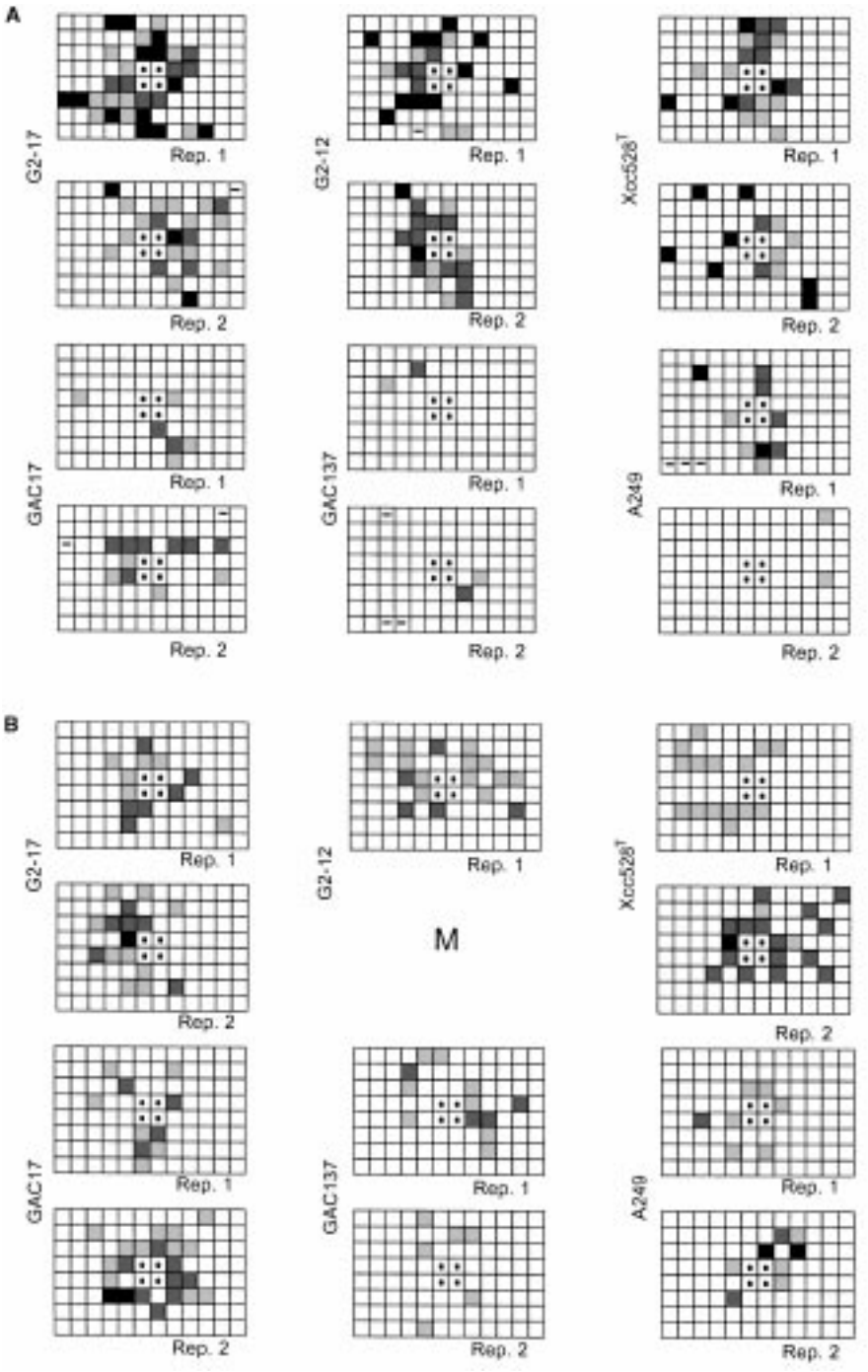

Figure 2. Spatial patterns of pathogen incidence in the plots as observed in Experiment II. G2-17, G2-12, and Xcc528^{T} are blight strains, and GAC17, GAC137, and A249 are typical black rot strains. The experimental units (trays) in the figure are grouped according to the symptom types of strains for clarity. The trays were completely randomized in the actual experiment. ■ Positive 2 days after inoculation, ■ Positive 8 days after inoculation, ■ Positive 14 days after inoculation, ⊞ Inoculated seedling, ⊟ Missing seedling, M Missing plot.

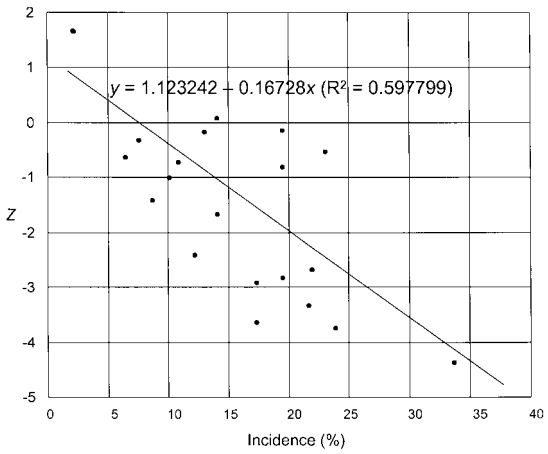

Figure 3. Scatter plot showing the correlation of non-random spatial distributions ('*Z*' values) and percent pathogen incidence. Each dot represents an experimental unit (a tray) in Experiment II. Simple linear regression line is shown in the plot.

Table 3. Summary of runs analysis for non-randomness for Experiment II

Strain[a]	Run	Rep.	'Runs'	*Z*[b]
G2-17	1	1	23	−4.37*
	1	2	31	−0.54
	2	1	19	−1.67*
	2	2	19	−2.92*
G2-12	1	1	23	−2.69*
	1	2	21	−2.84*
	2	1	29	−0.15
	2	2	M[c]	M[c]
Xcc528^T	1	1	21	−3.35*
	1	2	23	0.08
	2	1	17	−3.65*
	2	2	27	−0.82
GAC17	1	1	11	−0.64
	1	2	15	−2.41*
	2	1	17	−0.73
	2	2	21	−3.75*
A249	1	1	15	−1.01
	1	2	5	1.66
	2	1	13	−0.33
	2	2	13	−1.43
GAC137	1	1	5	−0.17
	1	2	5	−0.64
	2	1	21	1.66
	2	2	11	1.64

[a]G2-12, G2-17, and Xcc528^T are blight strains, and GAC17, A249, and GAC137 are typical black rot strains. [b]Asterisks indicate *Z* values smaller than −1.64, indicating clustering ($P = 0.05$). [c]Missing plot.

in development of field epidemics after transplanting seedlings to the field. However, plants infected in the seedbed are unlikely to be detected for several reasons. In early spring when seedbeds are planted, seasonal temperatures often are below the optimum for symptom expression. Even at relatively high temperatures, black rot and blight may remain latent in rapidly growing seedlings (Williams, 1980). In addition, downy mildew (caused by *Peronospora parasitica*) is common in seedbeds, and frequently causes senescence of leaves, making the detection of early symptoms of *X. c. campestris* infection difficult (Williams, 1980). The ability to detect symptomless pathogen spread will help evaluate the role of the seedbed stage of crucifer production in disease outbreaks in the field.

Acknowledgements

We would like to thank Drs. Dean W. Gabriel, Kendal D. Hirschi, Sophia Kathariou, and Suresh S. Patil for critically reading the manuscript. This research was supported in part by the U.S. Department of Agriculture under CSRS Special Grant Agreement No. 89-34135-4614, managed by the Pacific Basin Advisory Group.

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