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The effect of populations of *Xanthomonas campestris* pv. *phaseoli* in bean reproductive tissues on seed infection of resistant and susceptible bean genotypes

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Abstract

Surface and internal populations of *Xanthomonas campestris* pv. *phaseoli*, causal agent of common bacterial blight of bean, on and in flower buds, blossoms and pods of seven bean (*Phaseolus vulgaris*) genotypes were studied. Bean plants were grown in the field and artificially inoculated at the seedling stage (18 days old). The pathogen was recovered in high numbers from flower buds, blossoms, pods and seed of both resistant and susceptible bean genotypes. Significant differences (P = 0.05) in population levels of *X. c.* pv. *phaseoli* between stages of reproductive tissue development were observed. Infected seed from resistant bean genotypes had no visible symptoms. Such seed may play an important role in the epidemiology of common bacterial blight because they are difficult to detect and may occur at low frequency in seed lots, as was the case in the current study.

Introduction

Earlier studies indicate that *Xanthomonas campestris* pv. *phaseoli* (E.F. Smith) Dowson, causal agent of common bacterial blight of bean, has the ability to enter pods through the vascular system and infect the seed without causing lesions on the surface of the pods (Cafati and Saettler, 1980; Zaumeyer, 1930). In entering seed through the vascular system *X. c.* pv. *phaseoli* frequently causes a small yellow discolouration at the hilum. Such symptoms are not readily detected in coloured bean seeds. However, in white seeded beans, normally there is a yellow marking around the hilum. Seeds that are slightly infected without outward symptoms may result in severe outbreak of disease when planted the following season (Sutton and Wallen, 1970; Zaymeyer, 1930). Therefore, it is apparent that selection of pods without symptoms is not an adequate means of getting clean seed because vascular seed infection might be very important.

Foliage and stems of resistant bean cultivars are known to harbour relatively high populations of plant pathogenic bacteria without exhibiting descernible symptoms (Cafati and Saettler, 1980; Weller and Saettler, 1980a; Weller and Saettler, 1980b). Thus, there is a fear of using seed from resistant bean genotypes grown in infested areas. This fear is based on the findings that even resistant bean cultivars grown under such conditions may produce both infested and infected seeds. Cafati and Saettler (1980) and Schuster et al. (1979) demonstrated that seeds of resistant bean genotypes can become infected with *X. c.* pv. *phaseoli* to the same degree as seeds from susceptible genotypes. However, such findings were based on studies in which pod sutures were artificially inoculated.

The purpose of the current investigation was to address seed infection in resistant and susceptible bean genotypes when grown under field conditions and inoculated during the seedling stage of development.

Materials and methods

Seven bean genotypes were chosen on the basis of their reported reaction to common bacterial blight of beans. Five susceptible cultivars (Pinto UI-114, Cran-

berry Taylor Hort, Charlevoix, Black Magic and C-20) and two resistant genotypes (Valley and I-84100, a breeding line) were used. A completely randomized design (CRD) was used with four replications for each bean genotype. Pathogen-free seed were hand-planted in six meter rows at a spacing of 50 cm × 6 cm between rows and plants, respectively. Weed control was accomplished by both hand and mechanical weeding. During dry spells, soil moisture was supplemented by overhead sprinkler irrigation. The field used was under maize cultivation for three years and had not been under bean cultivation for five years.

Inoculum preparation and inoculation. Bacterial cells from 24-h old cultures of strain MI-17 grown on YCA (g/L: yeast extract 10.0; calcium carbonate 2.5; bacto-agar 15.0; glass distilled water 1000 ml) and incubated at 27 ± 1C, were washed into sterile flasks using sterile phosphate buffer (0.01M, pH 7.2). Cultures were then diluted to an optical density of 0.2 at 620 nm measured using a spectrophotometer (Model Spectronic 20, Bausch and Lomb, Rochester, NY). Bacterial suspensions were diluted further using the same buffer to a concentration of ca. 1.4×10^7 colony forming units (CFU) per ml. The inoculum was used within 40 min after preparation. Bean plants were spray-inoculated to run-off without water-soaking using a Knapsack sprayer when the second trifoliate leaf was fully open (18 days old).

Disease rating. Disease rating was done progressively at 10, 16 and 40 days after inoculation. For each disease rating, plants were carefully examined for development of typical common bacterial blight symptoms on all foliage and on pods. Disease rating on pods was, however, done at physiological maturity. A CIAT scale of 0–9 was used; where: 0 = immune, no symptoms; 9 = very susceptible, 50 percent of the leaf area or more covered with lesions (CIAT, 1987).

Measurement of X. c. *pv.* phaseoli *populations*. Bacterial population studies in reproductive tissues of bean genotypes were performed at a flower bud, blossom, flat and bumpy (half grown) pod stages of plant development. At flower bud formation, 25 flower buds were sampled from each replication using steam-sterilized forceps (one forceps per replication and placed in sterile 2.5 cm diameter glass test tubes. The same sampling procedure was used for blossoms. All samples were kept cold in the refrigerator at 8 °C until processed with 12 h.

Surface populations of *X. c.* pv. *phaseoli* on flower buds and blossoms were estimated by shaking the samples for 30 min in 40 ml of 0.01 M phosphate buffer containing 10 mM Magnesium Sulphate and 0.01 percent Tween 20 (Claflin et al., 1987) on a horizontal shaker set at 75 × 1.5-inch strokes per minute. After ten-fold serial dilution of the washates in the same buffer, 0.1 ml portions of each dilution were plated on a semi-selective medium M-SSM (Mabagala and Saettler, 1992) in triplicate and inoculated plates were incubated at 27 ± 1C (unless otherwise stated all incubations were done at this temperature). Plates were observed daily, and colonies surrounded by zones of starch hydrolysis were counted by marking the back of the plates over the range of 5 days. From each bean genotype, 10 representative *X c.* pv. *phaseoli* – like colonies were selected at random and tested for pathogenicity.

Determination of internal populations of *X. c.* pv. *phaseoli* was done by weighing and surface-sterilizing fresh samples of 25 flower buds and blossoms in 2.6 percent NaOCl for 3 min followed by rinsing three times in sterile glass distilled water. Samples were then comminuted using sterile mortors and pestles in 30 ml of phosphate buffer containing 10 mM Magnesium sulfate and 0.01 percent Tween 20. The macerates were allowed to stand for 15 min and decimal serial dilutions prepared. Aliquots of 0.1 ml from each dilution were plated in triplicate on M-SSM and incubated as earlier described. Colonies surrounded by zones of starch hydrolysis were counted 2–5 days after plating. Bacterial populations were calculated and expressed as CFU/g of fresh weight tissue. Presumed *X. c.* pv. *phaseoli* colonies from each genotype were purified further on YCA and tested for pathogenicity.

Surface populations of *X. c.* pv. *phaseoli* on flat and bumpy (half grown) pods were determined by using six pods per replicate. Symptomless pods were randomly sampled and placed in sterile plastic bags. Samples were kept cold as previously described until processed within 12 h. To determine the surface populations, pods were shaken for 30 min in 60 ml of phosphate buffer as earlier described. Washates were decimally diluted serially and 0.1 ml aliquots of the washates were plated in triplicate on M-SSM and incubated at 27 ± 1C. Colonies of *X. c.* pv. *phaseoli* were counted as earlier described and expressed as CFU/cm^2 of pod tissue. Pods were traced on paper and the area was measured using Lambda instrument, Model LI 3000.

Pathogenicity test. Pathogenicity of presumed *X. c.* pv. *phaseoli* colonies was tested using a susceptible variety Charlevoix. Inocula were prepared in sterile phosphate buffer from 24-h-old YCA cultures and adjusted to contain ca. 1.5×10^6 CFU/ml. Fourteen-day-old green house grown bean plants were inoculated with bacterial suspension by water-soaking an area of 10 mm diameter at 4 sites per leaflet using a sterile hypodermic syringe. Each isolate was replicated four times using one plant per replicate. Strain MI-17 of *X. c.* pv. *phaseoli* and sterile phosphate buffer were used as positive and negative controls, respectively. Inoculated plants were incubated in the greenhouse with temperature ranging from 24 to 30 °C and observed on daily basis. Pathogenicity was evaluated 10 and 14 days after inoculation. Appearance of typical common bacterial blight symptoms confirmed identity of isolates as *X. c.* pv. *phaseoli*.

Assessment of seed infection. At physiological maturity pods were hand-harvested, kept separate for each replication and allowed to dry under greenhouse conditions. After drying, pods were shelled by hand and seeds from each replicate were also kept separate. Seed samples were assayed for the presence of *X. c.* pv. *phaseoli* by a modification of procedures used by Cafati and Saettler (1980). A random sample of 400 seed was drown from each replicate using a slotted board containing 100 holes. Seeds were surface sterilized in 2.6 percent NaOCl for 3 min, rinsed three times in sterile distilled water and blotted dry on sterile paper towel in a laminar air flow chamber. After drying, seeds were aseptically plated, hilum down, on M-SSM five seeds per plate, incubated at 27 ± 1C and observed daily for up to 5 days. Bean seed that contained bacterial growth and were surrounded by a zone of starch hydrolysis, were counted and expressed as a percentage of the total seed plated. Bacteria from each infected seed were streaked on YCA and purified by a series of transfers. The identity of purified isolates were confirmed by physiological and biochemical tests (Schaad, 1988), and pathogenicity tests on bean variety Charlevoix earlier described. To verify the absence of *X. c.* pv. *phaseoli* in plated seed that were not surrounded by a zone of starch hydrolysis, 10 seeds from each replicate were rechecked by a six-hour incubation individually, in 4 ml of sterile phosphate buffer in test tubes. The resulting soakates were decimally diluted and plated on M-SSM and YCA. Inoculated plates were incubated at 27 ± 1C and observed for up to 5 days.

Table 1. Foliage and pod reaction of seven bean genotypes to *Xanthomonas campestris* pv. *phaseoli* under field conditions

Bean genotype	Foliage reaction[x] Days after inoculation			Pod reaction[y]
	10	16	40	
I-84100	0.0b[z]	1.4d	2.3b	1.8d
Valley	0.0b	1.5d	2.5b	1.9d
Pinto UI-114	4.5a	7.8b	6.5c	5.0c
Cranberry Taylor Hort	6.3c	8.8a	9.0a	8.0a
Charlevoix	4.8a	8.5ab	6.3c	7.3ab
Black Magic	5.0a	6.8c	4.5d	5.5c
C-20	4.3a	7.8b	6.5c	6.9b

[x] Eighteen-day-old beans plants were spray-inoculated to run-off without water-soaking with bacterial suspensions containing ca. 1.4×10^7 CFU/ml using Knapsack sprayer. Disease rating was done on all foliage by observing the whole plant at 10, 16 and 40 days after inoculation. Data are means of four replicates.
[y] Pod reaction was evaluated at physiological maturity. Disease rating based on CIAT scale of 0–9, where: 0 = immune, no symptoms, 9 = highly susceptible, 50 percent or more of pod area covered with lesions. Values are means of four replicates.
[z] Within a column, means followed by the same letter are not significantly different (P = 0.05) by Duncan's new multiple range test. Data were log-transformed before analysis.

Statistical analysis. Data analysis was done using MSTAT-C (Department of Crop and Soil Science, Michigan State University, E. Lansing). Where appropriate, data were log normal and arcsine transformed before analysis (Little and Hills, 1978). Duncan's new multiple range test was used to separate differences between means.

Results

Foliage and pod reaction. Foliage and pod reaction of the seven bean genotypes to *X. c.* pv. *phaseoli* are shown in Table 1. Under conditions of this study, the first foliage symptoms were observed 8 days after inoculation on all genotypes except I-84100 and Valley, on which symptoms appeared 15 days after inoculation. The varieties Valley and I-84100 did not differ significantly (P = 0.05) in reaction to the disease in all ratings. In addition, disease symptoms were not visible on the uninoculated leaves of the two resistant genotypes. However, disease spread very rapidly within inoculated leaves on cultivars Cranberry Taylor Hort., Pinto UI-114, Charlevoix and C-20. In addition, systemic movement of the pathogen as revealed by

Table 2. Surface and internal populations of *Xanthomonas campestris* pv. *phaseoli* on and in flower buds and blossoms of resistant and susceptible bean genotypes grown in the field and spray inoculated at seedling stage

	CFU/g fresh weight of tissue[xyz]			
	Flower buds		Blossom	
Genotype	Surface	Internal	Surface	Internal
I-84100	2.092×10^4de	3.692×10^4cde	4.595×10^3e	4.875×10^3e
Valley	2.785×10^4bcde	3.507×10^4cde	9.465×10^4bcde	5.631×10^4de
Pinto UI-114	6.575×10^5ab	1.095×10^5abcde	3.257×10^5abc	1.975×10^5abcd
Cranberry Taylor Hort.	9.250×10^5a	2.640×10^6a	2.350×10^5abcd	1.775×10^5abcd
Charlevoix	1.387×10^6abc	7.112×10^5ab	4.712×10^5abc	8.272×10^5abc
Black Magic	1.167×10^4e	1.449×10^4bcde	4.355×10^3	7.00×10^3bcde
C-20	4.168×10^4cde	2.400×10^4	1.316×10^5cde	8.885×10^4abcd

[x] Surface populations of *X. c.* pv. *phaseoli* were estimated by shaking 25 flower buds and blossoms separately in 40 ml of 0.01M phosphate buffer, pH 7.2 containing 10 mM Magnesium sulfate and 0.01 percent Tween 20. After decimal dilutions in the same buffer, 0.1 ml aliquots of washates were plated on M-SSM in triplicate and inoculated plates incubated at 27 ± 1 C.
[y] Internal populations of *X. c.* pv. *phaseoli* were determined by comminuting 25 surface sterilized flower buds and blossoms in 30 ml of 0.01M phosphate buffer, pH 7.2, containing 10 mM Magnesium sulphate and 0.01 percent Tween 20, using sterile mortors and pestles. Resulting suspensions were allowed to stand for 15 min. and decimal serial dilutions prepared in the same buffer. Aliquots of 0.1 ml from each dilution were plated in triplicate on M-SSM and inoculated plates incubated at 27 ± 1C for up to 5 days.
[z] Within a column, means followed by the same letter are not significantly different (P = 0.05) by Duncan's new multiple range test.

symptoms on the upper portion of the stem and in petioles of uninoculated leaves was very pronounced in the most susceptible genotypes. At 16 days after inoculation (blossom stage), the entire canopy of Cranberry Taylor Hort. was affected by the disease. This continued to be the trend even at 40 days after inoculation, where except for Cranberry Taylor Hort., there was a decrease in foliage disease reaction in most bean genotypes (Table 1). On pods, Cranberry Taylor Hort. was the most susceptible, followed by Charlevoix and C-20. There was no significant difference in pod reaction (P = 0.05) between I-84100 and Valley (Table 1). In general, there was less disease at 40 than at 16 days after inoculation. At 40 days, most of the inoculated leaves had dropped down, therefore, disease rating was based on secondary infection and tended to be low.

Surface and internal populations of X. c. *pv.* phaseoli. Surface populations of *X. c.* pv. *phaseoli* on flower buds and blossoms of the seven bean genotypes are shown in Table 2. Populations of *X. c.* pv. *phaseoli* on C-20 and Black Magic were not significantly different (P = 0.05) from those of the resistant genotypes I-84100 and Valley. However, on C-20, surface populations of *X. c.* pv. *phaseoli* were higher on blossoms than on flower buds (Table 2). Resistant genotypes also supported high levels of the pathogen on flower buds and blossoms. The trend of internal populations in flower buds and blossoms was similar to surface populations (Table 2). In all bean genotypes, flower buds and blossoms were infected by the pathogen. Internal populations of *X. c.* pv. *phaseoli* on Valley, I-84100 and Black Magic, were not significantly different (P = 0.05). Oily translucent cream-coloured lesions which were more evident during sunny dry weather, were observed on blossoms and petals of highly susceptible genotypes including Charlevoix, Black Magic, Pinto UI-114 and Cranberry Taylor Hort. The pathogen was also readily isolated from such lesions.

Differences in population levels of *X. c.* pv. *phaseoli* between bean genotypes and between the two stages of pod development within genotype were observed. Surface populations of *X. c.* pv. *phaseoli* on flat and half-grown pods expressed as CFU/cm^2 are shown in Table 3. At the flat pod stage, genotypes I-84100 and Valley supported significantly lower (P = 0.05) populations of the pathogen than the rest of the genotypes. Cranberry Taylor Hort. and Charlevoix supported the highest population of all bean genotypes. However, populations of *X. c.* pv. *phaseoli* on pod of these two varieties were not significantly different (P = 0.05).

Seed infection. To determine the presence of bacteria in bean seed, a total of 400 seed were randomly sampled from each replicate. The percentage seed infection of resistant and susceptible bean genotypes grown in the field and inoculated with *X. c.* pv. *phaseoli* is given in Table 3. Cranberry Taylor Hort. had the highest level of seed infection followed by Pinto UI-114. Some of the infected seed from these two genotypes were shriv-

Table 3. Surface populations of *Xanthomonas campestris* pv. *phaseoli* on flat and bumpy (half-grown) pods and seed infection (percent) in resistant and susceptible bean genotype

Genotype	CFU/cm^2 pod area[x]		Seed[y] infection(%)
	Flat pods	Half-grown pods	
I-84100	1.730×10^2e[z]	7.383×10^4abc	3.88cd
Valley	1.515×10^3e	9.850×10^2de	0.38e
Pinto UI-114	1.920×10^4abc	6.625×10^3bcd	16.88b
Cranberry Taylor Hort.	1.447×10^5a	6.475×10^4ab	34.38a
Charlevoix	1.779×10^5ab	1.763×10^4cd	4.88c
Black Magic	7.930×10^3cd	7.292×10^3cde	2.12d
C-20	2.550×10^3abc	1.920×10^3	6.50c

[x] Surface populations of *X. c.* pv. *phaseoli* on pods were determined using six symptomless pods per replicate. Pods were shaken for 30 min. in 60 ml of phosphate buffer and the resulting washates were decimally diluted serially. Aliquots of 0.01 were plated on M-SSM in triplicate and incubated at 27 $\pm$ 1C for up to 5 days. Data were log-transformed before analysis.
[y] Four hundred seeds per replicate were randomly drawn, surface sterlized in 2.6 percent NaOCl for 3 min., rinsed thrice in sterile distilled water and blotted dry on sterile paper towel. Seeds were then plated, hilum down, on M-SSM and incubated at 27 $\pm$ 1C for up to 5 days. Data were arcsine transformed before analysis.
[z] Within a column, means followed by the same letter are not significantly different (P = 0.05) by Duncan's new multiple range test.

elled. The genotypes Charlevoix, C-20 and I-84100 were not significantly different (P = 0.05) in seed infection (Table 3). Although foliage reaction was significantly different between Black Magic and I-84100, there was no significant difference in seed infection between these two genotypes. The lowest percentage of seed infection (0.38%) occurred in Valley.

Most of the seed which were internally infected with *X. c.* pv. *phaseoli* in the two resistant bean genotypes (I-84100 and Valley) were symptomless. Such was also the case with Black Magic, in which infected seed were neither discoloured nor shrivelled. High levels of seed infection matched with the high levels of internal *X. c.* pv. *phaseoli* population in flower buds and blossoms in Cranberry Taylor Hort, Pinto UI-114, and Charlevoix; while low levels of seed infection in Black Magic, C-20, Valley and I-84100 were consistent with low levels of *X. c.* pv. *phaseoli* on and in reproductive tissues of these genotypes (Tables 2 and 3).

All Representative isolates of *X. c.* pv. *phaseoli* tested for pathogenicity were pathogenic on bean variety Charlevoix, confirming identity.

Discussion

Previous studies indicate that *X. c.* pv. *phaseoli* is capable of colonizing young tissues as they develop from the apical meristem, thus establishing a population gradient in the bean canopy (Cafati and Saettler, 1980; Weller and Saettler, 1980(b)). At least 5×10^6 CFU/cm^2 of leaf tissue are needed for symptom development, a population requirement which creates a latent period after tissues are initially colonized by *X. c.* pv. *phaseoli* (Weller and Saettler, 1980b).

In the current study, development of symptoms on resistant cultivars took longer time than on susceptible cultivars. Therefore, symptoms on Valley and I-84100 were delayed for 7 days as compared to the susceptible genotypes suggesting that the pathogen multiplied at a lower rate in resistant than in susceptible genotypes. In addition, a higher population of the pathogen is required to produce symptoms in resistant than in susceptible genotypes (Cafati and Saettler, 1980). It was also noted that reproductive tissues of susceptible cultivars supported higher populations of *X. c.* pv. *phaseoli* than those of resistant genotypes (Tables 2 and 3). Except for few cases, flower buds supported lower population levels of *X. c.* pv. *phaseoli* than blossoms. These findings agree with those of Weller and Saettler (1980b) that a population build-up occurs over time. Similar trends were observed on flat and half-grown pods, especially on Cranberry Taylor Hort and I-84100 (Tables 2 and 3).

Earlier studies have presented evidence that there is systemic colonisation of pods by *X. c* pv. *phaseoli* (Cafati and Saettler, 1980; Weller and Saettler, 1980b; Zaumeyer, 1930; Zaumeyer and Thomas, 1957). However, it is not well known whether the low population

of *X. c.* pv. *phaseoli* on and in reproductive tissues of resistant bean genotypes was due to reduced systemic movement or to other internal and external factors related to the host and the environment. Both external and internal factors have been reported to influence multiplication of *X. c.* pv. *phaseoli* on and in bean tissues. Data from the current study suggest that populations of the pathogen present in and on flower buds and blossoms may play a great role in early colonization of developing pods and later, seed. Weller and Saettler (1980b) indicated that bud colonisation is an important mechanism of spread for *X. c.* pv. *phaseoli*.

The results of this study also indicate that, under field conditions, *X. c.* pv. *phaseoli* can infect seeds of both susceptible and resistant bean genotypes when the pathogen is introduced into young plants. This is contrary to previous findings by Cafati and Saettler (1980), who recovered *X. c.* pv. *phaseoli* only from seed samples harvested from symptomless pods of field grown plants inoculated at the flat pod stage of development. Such differences in results may be brought about by differences in genotypes used and by other external and internal factors related to genotypes and the environment. High levels of seed infection corresponded with high numbers of *X. c.* pv. *phaseoli* in flower buds and in blossoms for the genotypes Cranberry Taylor Hort., Pinto UI-114 and Charlevoix. In addition, low levels of seed infection in Black Magic, C-20, Valley and I-84100 corresponded with low numbers of *X. c.* pv. *phaseoli* on and in reproductive tissues of these genotypes (Tables 2 and 3). These findings further suggest that the level of *X. c.* pv. *phaseoli* in flower buds, blossoms and on pods play a role in systemic infection of pods, resulting in infected seed, since internal populations are likely more responsible with disease induction (Beattie and Lindow, 1995). The relationship between foliage reaction and internal seed infection has also been reported for some bean genotypes (Schuster, et al., 1979). However, differential reaction of foliage and pods to *X. c.* pv. *phaseoli* is also known to occur (Valladares-Sanchez et al., 1979).

Seed infected with *X. c.* pv. *phaseoli* in resistant genotypes had no visible symptoms. Such was the case for Black Magic, a susceptible variety. Symptomless infected seed have been reported to be important in the epidemiology of common bacterial blight of beans because germination and seedling growth of such seed are similar to that of uninfected seed. This is in contrast to severely infected seed which germinate poorly or produce deformed seedlings, thus reducing the effectiveness of such seed as sources of primary inocula (Weller and Saettler, 1980a). Seed infection of resistant bean genotypes is a major concern in the bean industry. This is because seed transmission is more apt to establish the pathogen in a new locality. Seed transmission also introduces the pathogen randomly throughout the field and provides numerous foci for primary infection. Such inoculum is more effective than that spreading from the margins of the field (Baker and Smith, 1966). Furthermore, detection of symptomless seed such as those from resistant varieties, is more difficult due to the low frequency in seed lots and low populations of the pathogen within such seed. Therefore, studies are needed to determine the role of infected seed from resistant bean genotypes in the epidemiology of common bacterial blight of bean under field conditions. Such studies will provide important information needed for the management of common bacterial blight in resistant bean genotypes and strenghthen our understanding of the ecology and epidemiology of *X. c.* pv. *phaseoli* in resistant bean genotypes.

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