STUDIES ON SEED TRANSMISSION OF XANTHOMONAS CAMPESTRIS PV PHASEOLI IN COMMON BEANS IN UGANDA

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ABSTRACT

Studies on the seed transmission of Xanthomonas campestris pv phaseoli (XCP) that cause common and fuscous blight of beans were conducted in Uganda. Bean seed was the main source of primary inoculum. The population of XCP in farmers', commercial and research seeds averaged 10⁵–10⁹ colony forming units (cfu) per 100 seeds; the levels of seed infection were 0.3–16.1%. There was a positive correlation between seed symptoms and population of XCP per seed. However, where seed was either slightly diseased or symptomless, XCP did not decrease bean germination irrespective of population density. The minimum population of XCP required to initiate infection in the field was 10² cfu per seed while 0.2% seed infection level resulted in serious disease incidence.

Key Words: Bacterial blight, Xanthomonas campestris beans, seed infection.

INTRODUCTION

Common bacterial blight (CBB) and fuscous blight, caused by *Xanthomonas campestris* pv phaseoli (XCP), are among the most serious seed borne diseases affecting beans (*Phaseolus vulgaris* L.) in eastern Africa (5; 6; 11). Internally infected seeds form the main source of primary

inoculum (2; 19). Worldwide distribution of these diseases is partly associated with the ability of the pathogen to infect seeds of both resistant and susceptible genotypes (1). In Canada, 0.5% seed infection level has been shown to lead to disease epidemics (21). The causes of epidemics of blight in the tropics, that occur sometimes even in newly opened areas, needs further investigation.

This study was, therefore, initiated to investigate and provide quantitative data on both common and fuscous blight transmission in Uganda.

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MATERIALS AND METHODS

The study was conducted at Kawanda Research Station, Uganda, between June 1991 and July 1992, and covered laboratory, screenhouse and field experiments.

XCP infection in farmers' and non-farm seed. Seeds used to determine levels of seed infection were obtained from the National Seed Project at Kawanda, and from 25 farmers, seven markets and eight varietal trial centers in eleven districts. A total of 62 seedlots were tested. The seeds were clean, with no obvious symptoms of disease infection, which for farmers formed part of seed for planting in the following season.

Two media; MXP (a semi selective medium for XCP) and yeast dextrose carbonate agar (YDCA) were used in all isolations and cultures of XCP. MXP was prepared following the procedure described by Claflin *et al.* (3) while YDCA was prepared as described by Vidaver (19).

The most probable number method (MPN) (4) was used to estimate the level of CBB infection in the seed. Eleven samples were drawn from each lot, one of 500 seeds, five of 100 seeds and five of 10 seeds.

The seeds were plated, hilum downwards, on both media and records taken after incubating at $28 \pm 2^{\circ}$ C for 5 days. Seeds from which XCP was recovered were recorded as positive and those from which XCP was not recovered as negative. Colonies that hydrolyzed starch on MXP were assumed to be XCP (3). The level of infection was determined using the most probable number tables (13).

From each sample, 5 or 6 colonies with typical characteristics of XCP were selected for pathogenicity testing. After a further 48 hours growth, the increased inoculum was tested for pathogenicity by inoculating the second trifoliolate leaves of 18-day old seedling of the cultivar Kanyebwa grown in the screenhouse. The plants were grown in 20 pots of 25 cm-diameter, and filled with heat sterilized soil mixed with sand in the ratio of 2:1. Five bean seeds were sown per pot, NPK fertilizer (25-5-5) was applied at the rate of 1 g/7 kg of soil. The razor blade inoculation method (7) was used,

with XCP inoculum concentration of about 10⁷ cfu/ml.

Relationship between seed symptoms, bacterial cell numbers per seed and seedling infection. Three bean cultivars: Kanyebwa, K 20, and XAN 112 were used, the first two being susceptible to XCP while the latter is resistant. A 2 kg sample of severely infected seed of each of the three cultivars was graded on the basis of visible symptoms in three categories:

Category 1: Symptomless, no lesion or discoloration on seeds.

Category 2: Slight to moderate symptoms — seeds with less than 10% butter-yellow discoloration or with discoloration in the hilum region.

Category 3: Severe symptoms — seeds with >10% discoloration with partial shrivelling.

Evaluation of the relationship between seed symptom, bacterial cell count/seed, and seedling infection was based on the method described by Taylor *et al.* (16).

The bacterial numbers in seed were determined using six groups of 100 seeds for each symptom category. Single seeds were ground in a sterile mortar containing 1 ml of 0.01M phosphate buffer at pH 7.2. The suspension obtained was shaken for 30 minutes and serially diluted ten-fold before plating. Aliquots of 0.1 ml (10⁻²; 10⁻⁴, 10⁻⁶ and 10⁻⁸) were spread on media in triplicates and plates incubated for 5 days. From each set of seeds, a random sample of ten bacterial colonies were selected and tested for pathogenicity.

Disease transmission to seedlings was tested in the field and in the screenhouse. One hundred seeds from each category and each genotype were sown in 6 metre long two row plots, with three replications and a 1 m spacing between the plots. The design was a split plot with bean genotypes in the main plots and symptom categories in subplots. Maize was sown between the plots and in guard rows, two weeks earlier, to reduce interplot interference (16).

The same number of seeds and replications for each infection category were used in the screenhouse and in the field. Soil preparation and sowing was as described above for the

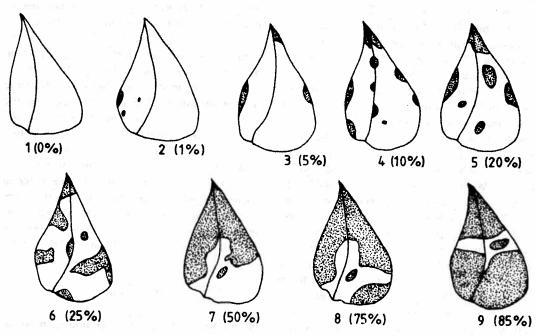


Fig. 1. Diagrammatic representation of the scale used to rate common bacterial blight of common beans on leaves.

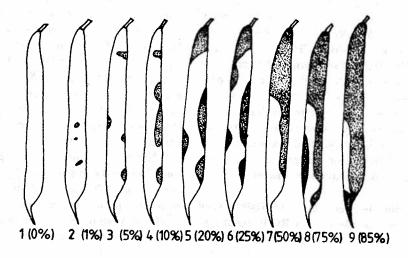


Fig. 2. Diagrammatic representation of the scale used to rate common bacterial blight of common beans on pods.

screenhouse. A total of 20 pots were used for each category for every replication.

Incidence and severity of the disease on cotyledons was recorded at seedling emergence. Thereafter, records were taken at 10, 18, 35, 42

and 56 days after sowing. Incidence was assessed by counting the number of infected plants and severity was scored using a modified CIAT 1-9 scale (Figs. 1 and 2).

Three bean genotypes: Kanyebwa, K 20 and

White haricot were used. The different levels of seed infection used were 0, 0.2, 0.4, 0.5, 1, 2, 3, 5, 10 and 20%. The clean seed used was multiplied in the off-season in the screenhouse at Kawanda. The infected seed was collected from severely diseased fields of the preceding season. Random samples of diseased and clean seed were assessed using the most probable number sampling method (14) to establish XCP inoculum levels before mixing the seeds in the proportions required. The samples were tested by plating seeds on MXP and YDCA and incubating as above. Where there was growth of XCP from presumably clean seed, such seed was discarded.

The experimental design used was a split plot, with genotypes in main plots and percentage infection in subplots, with three replications. The plot sizes were 6 x 6 m in 1991 and 12 x 12 m in 1992. Main plots and subplots were separated by 2 m space. Two rows of maize were sown between the plots to reduce interplot interference. The seedlings from infected seeds were tagged as soon as they were recognizable at two leaf stage. The presence or absence of bacterial blight symptoms on these plants was noted at tagging.

Disease incidence was determined as number of plants affected per plot, and severity was assessed using the modified CIAT 1–9 scale (Figs. 1 and 2). Incidence was determined by checking primary leaves of each plant at 10 and 18 days after sowing. Thereafter, four 1 m square quadrants were used per experimental unit and records taken weekly. Severity was assessed on 10 randomly selected plants, and a general score for the experimental unit taken. This was done at 18, 35, 42 and 56 days after sowing.

Relationship between level of visible seed infection and incidence of CBB in seedlings. Two experiments were conducted in the field and two in the screenhouse, to determine the minimum population of XCP necessary for successful seedling infection.

Seeds of three bean genotypes: Kanyebwa, K 20 and XAN 112 were inoculated with XCP at nine concentrations ranging from 0 to 5×10^8 cfu/ml. The experimental design was a split plot with varieties in main plots and concentrations in subplots. One hundred seeds were sown in the screenhouse as described above. Two 5 m row

plots separated by 1 m wide space were used in the field. Maize was sown between plots and in guard rows to reduce interplot interference.

A 48-hour old culture of XCP isolate 1010, obtained from a field at Kawanda and grown on YDCA, was used as the inoculum. The bacterial culture was washed into a sterile flask with sterile phosphate buffer (0.01m; pH 7.2), and the suspension adjusted to a concentration of 5 x 108 cfu/ml at a wavelength of 620nm, using a Milton Roy 20TM spectrophotometer. Seven serial dilutions were made in the same buffer. The seeds were inoculated by soaking in the different bacterial concentrations for four hours before sowing. The control seeds were soaked in the phosphate buffer. Disease incidence and severity were scored 10, 18, 35, 42 and 56 days after sowing.

RESULTS

XCP infection in farmers and non-farm seeds.

The incidence of XCP infection in seed samples from farmers, varietal testing centres and markets varied with the genotype and location (Table 1). Kabale had the lowest level of seed infection (0.3%) while Masindi had the highest (16.1%). The seed of White haricot was more infected compared to other genotypes from the same location. Kanyebwa and K 20, the most common genotypes encountered, had infection levels ranging from 0.8 to 3.5%. MCM 5001 had the lowest seed infection among seed obtained from varietal testing centers.

The XCP population was high for all seed lots. The number recovered ranged between 10⁵ and 10⁹ cfu/100 seeds. All the XCP isolates recovered from seed induced typical bacterial blight symptoms when inoculated onto the susceptible genotype Kanyebwa.

Relationship between common bacterial blight symptoms on seed, bacterial numbers per seed and production of infected seedlings. Seeds with severe symptoms (Category 3) had the highest population of XCP cells. Large number of bacterial cells $(1.7 \times 10^6 - 5.2 \times 10^6)$ were also recovered from apparently clean seeds (Table 2). There were no significant differences in number of XCP cells recovered per seed between

TABLE 1. Incidence^a and population^b of *Xanthomonas campestris* pv *phaseoli* in bean seed collected from different parts of Uganda

	Genotype	****	Incidence (%	6)	Population of XCP			
District		VTC	Farmers	Markets	VTC	Farmers	Markets	
Arus	K 20		_	1.8			4.8 x 10 ⁹	
	White haricot		_	2.8	_		1.7 x 10 ⁹	
	Black haricot		_	1.1		•••	8.4 x 10 ⁹	
	K 20	-	3.5	1.8	-	8.4 x 10 ⁸	4.8 x 10 ⁹	
Tororo	Kanyebwa	-	1.7	1.4	_	5.8 x 10 ¹	3.8 x 10 ⁷	
Mpigi	K 20	2.8			1.3 x 10 ⁸	_		
(Bukalasa)	CAL 96	1.4	-	-	5.2 x 10 ⁸		-	
Kabale	K 20	-	1.1	0.8		5.3 x 10 ⁸	E 4 × 409	
• •	Mixture	<u></u> -	0.3	1.1		4.0 x 10 ⁷	5.4 x 10 ⁹	
	G 5435	-	0.8	-	_	6.2 x 10 ⁸	5.3 x 10 ⁵	
Kampala	K 20	_	2.2	5.4		4.4 x 10 ⁸		
	White haricot	***	_	9.2	***	4.4 X 10°	5.3 x 10 ⁸	
	Kanyembwa		1.4	1.7		6.3 x 10 ⁷	7.7 x 10 ⁹	
٠.	Kahura	-		1.4	_	0.3 x 10°	4.5 x 10 ⁷ 3.4 x 10 ⁷	
	Kanyinja			1.3				
	Kulyembarukye	 ,	-	2.2		<u>-</u>	4.5 x 10 ⁷ 3.4 x 10 ⁷	
Masindi	White haricot	-	3.5	16.1	***	3.6 x 10 ⁸	8.4 x 10 ⁸	
	Kanyebwa	·	1.8	2.8	4	5.2 x 10 ⁶	1.8 x 10 ⁸	
	K 20	2.8	3.5	3.5	2.8×10^{8}	3.5 x 10 ⁷	4.7 x 10 ⁸	
	Mutike	****	1.7	-			2.4 x 10 ⁶	
	CAL 96	1.1			7.5 x 10 ⁶	,	- × 10	
Jinja	Kanyebwa	· _		1.7	_	_	4.8 x 10 ⁶	
	K 20	1.1	-	2.8	4.8×10^{8}	_	2.3 x 10 ⁸	
Mpigi								
Kawanda RS)		2.8	***			***	3.4 x 10 ⁹	
	Kanyebwa	0.5		_			4.4 x 10 ⁷	
	White haricot	9.2	men.	_			7.8×10^7	
	CAL 96	1.1		-			5.4 x 10 ⁷	
	RWR 36	1.3		-	_		-	
.owero	Mutike	Minor	1.4	_		3.3 x 10 ⁷	_	
	CAL 96	-	2.2			5.2×10^7		
	CAL 98	_	1.8		-	1.9×10^7		
	K 20	-	1.7			4.7×10^9		
1asaka	MCM	0.7	-	_	3.8 x 10 ⁹	_		
	CAL 96	1.1	-		7.8×10^9			
	CAL 98	1.0	Auditor.		6.2 x 10 ⁹	_	_	
Mbale	K 20	-		2.8	_			
	Kanyebwa	- ,		5.4	_	_	_	
	MCM 5001	0.5			4.4 x 10 ⁷		7.4 x 10 ⁹	
	CAL 96	2.2	_	_	5.4×10^7		3.9 x 10 ⁹	
	K 20	3.5	_	_	6.8 x 10 ⁸		J.3 X 1U"	
asese	K 20	2.4	_		2.5 x 10 ⁸			

^aIncidence is the percentage of seeds infected with XCP as determined by the most probable number method and interpreted using tables of Taylor and Phelps (14). ^bThe population of XCP was determined as colony forming units (cfu) per 100 seeds. VTC: varietal testing centres.

symptom Categories 1 and 2. Seed infection were highest for K 20, and lowest for XAN 112, their infection level being similar for all symptom categories (Table 2). For both Kanyebwa and XAN 112, infection for categories 1 and 2 were low, and statistically similar, but were significantly (P < 0.05) higher than infection for Category 3.

Seed germination failure ranged from 47.7% for symptom category 3 seeds of Kanyebwa and XAN 112, to only 0.3% for Category 1 of XAN 112 (Table 2). Higher seed infection significantly reduced germination. There were also significant ($P \le 0.05$) differences among categories for seedling infection at 10 days after sowing (Table 2). Symptom category 3 had the highest incidence while Category 1 had the lowest incidence. Differences in number of infected plants between slightly diseased and symptomless seeds were not significant ($P \le 0.05$).

Symptomless seeds contributed between 24.5-27.3% to the CBB transmission for Kanyebwa and 27-35% for K 20 (Table 2). Corresponding figures for slightly diseased seed

were 30–34% for Kanyebwa and 34% for K 20. No seed transmission occurred for these categories of seed for XAN 112. There was a positive and significant ($P \le 0.05$) correlation between the number of infected seeds and the percentage of infected seedlings at 10 days after sowing (r = 0.72). Correlations between the number of XCP cells per seed with the number of infected seedlings were not significant. The ratio of seed infection to primary infection ranged from zero to 19:10.

Relationship between level of visual seed infection and incidence of CBB in seedlings. The 20% seed infection gave the highest number of infected seedlings at 10 days after sowing (Table 3). There were no significant (≤ 0.05) differences in number of infected seedlings produced at 10 days. The control remained free from common bacterial blight. At pod formation, and pod filling stages (42 and 56 days) 20% seed infection gave the highest incidence (97.1%) while 0.2% gave the lowest incidence (47.1%) at

TABLE 2. Relationship^a between common bacterial blight seed symptoms, *Xanthomonas* campestris pv phaseoli (XCP) population per seed and seedling infection in 1991 in the field (F) and screenhouse (S)

Var.	Seed symptoms category	(%) infected seeds	Mean XCP seed	Seedling emergence (%)		Seedling infection at 10 days (%)		Contribution to primary transmission (%)		Seed infection to primary infection ratio	
				F	s	F	S	F	s	F	S
Kany	yebwa		_				۰.	04.5	27.3	1.9:1	1.5:1
	1	4.3	2.0 x 10 ⁶	90.3	92.5	2.8	3.5	24.5		1.91	1.3:1
	2	7.3	4.7 x 10 ⁷	79.7	81.5	4.2	4.2	32.5	30.4		1:1:1
	3	16.0	9.0 x 10 ⁸	48.0	47.7	5.3	5.5	43.0	42.3	1:1	1.1.1
K 20)							040	04.4	1.5:1	1.5:1
	1	15.7	5.2 x 10 ⁶	83.7	92.7	3.6	3.5	34.9	34.1		1.4:1
	2	16.7	7.7 x 10 ⁶	70.3	80.3	4.4	4.0	35.7	37.2	1.5:1	
	3	16.0	1.3 x 10 ⁸	56.3	54.5	4.5	5.0	29.4	28.7	1.2:1	1.1:1
XAN	N 112									4.0.4	•
	1	0.7	1.7 x 10 ⁶	98.3	99.7	0.0	0.0	0.0	0.0	1.2:1	0
	2	1.7	1.3 x 10 ⁶	78.7	80.0	0.0	0.0	0.0	0.0	0	0
	3	6.0	2.3 x 10 ⁶	47.7	52.5	0.7	0.6	100.0	100.0	1:1	1:2:

^aEstimated level of infection by MPN method and interpreted with tables by Taylor and Phelps (14). Kanyebwa = 5.4; K 20 = 5.4; XAN 112 = 0.7.

TABLE 3. The effect of number of infected seeds in a seed lot on incidence of common bacterial blight in the subsequent bean crop at 10, 42 and 56 days after sowing

Percentage infected seed	Incidence of CBB (%)									
	10 days		42 (days	56 days					
	1991	1992	1991	1992	1991	1992				
20	14.6a	12.0a	72.8a	65.2a	97.1a	93.6a				
10	10.0b	8.1b	68.8b	61.0b	92.3b	89.7b				
5	6.5c	4.7c	49.4c	45.9c	88.8c	81.2c				
3	3.4d	2.0d	40.5d	31.7d	66.4d	58.4d				
2	1.6e	0.8e	26.3e	20.4e	64.0e	56.9e				
1	1.0ef	0.4ef	12.6f	10.0f	54.0h	56.1e				
0.5	0.4efg	0.3ef	13.1f	9.3f	54.6h	48.9f				
0.4	0.8efg	0.2ef	9.3f	7.1g	57.0g	48.1fg				
0.2	0.2g	0.2ef	13.2g	7.0g	57.0g 59.7f	47.1g				
0	0.0h	0.0f	0.0h	0.0h	9.8i	7.0h				

Means in a column followed by the same letter are not significantly different at 5% Duncan's Multiple Range Test.

TABLE 4. The effect of population of *Xanthomonas campestris* pv *phaseoli* on seedling infection at 10 days (d) after sowing and adult (XCP) plant infection at 56 days after sowing

Population of XCP	1991				1992				
	% infection		severity ^a		% plants infected		severity		
	10 d	56 d	10 d	56 d	10 d	56 d	10 d	56 d	
10 ⁸	8.2a	80.7a	2.1a	5.4ab	15.8ab	84.7a	2.3a	6.3a	
10 ⁷ 10 ⁶	8.6a 8.3a	79.0a 78.4a	2.2a 1.9ab	5.8a 5.2ab	17.4a 15.5ab	84.5a 82.4ab	2.7ab 2.2ab	6.4a	
10 ⁵ 10 ⁴	8.0ab	76.1ab	2.0ab	5.0b	11.3ab	82.1ab	2.2ab 1.7b	6.3a 6.1a	
10 ³	7.2ab 6.8abc	77.1ab 76.4ab	2.0ab 1.4bc	4.9bc 4.9bc	12.0ab 9.5ab	81.1ab 77.0b	2.1b 2.1b	6.3a 6.1a	
10 ² 10 ¹	4.6c 0.0d	75.5b	1.2c	4.4c	7.6b	768b	1.5b	6.1a	
Control	0.0d 0.0d	55.1d 31.2d	1.0c 1.0c	1.8c 1.2d	0.0c 0.0c	61.1c 54.4d	1.0b 1.0b	1.6b 1.2b	

^aSeverity assessed on a scale of 1-9.

Means in a column followed by the same letter are not significantly different at 5% level using Duncan's Multiple Range Test.

56 days after sowing. This indicated that a seed infection level as low as one seed in 500 could lead to a high disease incidence provided conditions are favorable for disease development.

Minimum population of XCP necessary for successful seedling infection. The lowest XCP

population on seed that produced diseased seedlings at 10 days after sowing was 10^2 cfu/ml (Table 4). An XCP population of 10^7 cfu/ml in seed gave the highest number of diseased seedlings in all experiments. The control and 10^1 cfu/ml did not result in seedling infection at 10 days after sowing in both years.

The severity and incidence of bacterial blight was highest in treatments with XCP populations of 10⁷ cfu/ml 56 days after sowing (Table 4). Bacterial blight symptoms were observed on plants grown from seeds inoculated with 10¹ cfu/ml and control plots (buffer inoculated) at 56 days but not at other earlier stages. The appearance of blight symptoms in these plots at this stage was a result of secondary infection. The severity was, however, very low as compared to other treatments.

DISCUSSION

This study demonstrated that the bean seed used by farmers in Uganda is heavily infected by XCP. The lowest level of seed infection was 0.3% and the bacterial population per seed ranged between 10⁵ and 10⁹ cfu/100 seeds. The minimum bacterial population per seed necessary to incite infection in the field was found to be 10² cfu/seed.

There was significant correlation between symptoms on seed and the population of XCP per seed. Symptomless and slightly diseased seeds gave rise to severely infected seedlings. This demonstrated that the normal cleaning, by removing colored and shrivelled seeds, would have limited effect on reducing the XCP infection of a seed stock if it was obtained from an infected field. Similar observations were made by Saettler (8) on common bacterial blight, and by Taylor (16) on halo blight of beans.

A level of seed infection of 0.2% was found to initiate and cause severe infection in the field, similar to those reported for halo blight of beans (10; 16) and for *Xanthomonas campestris* pv campestris for crucifers (14). In Canada 0.5% seed infection was capable of causing common blight epidemics (12). Trigalet and Bidaud (17) established a tolerance level of one infected seed per 20,000 for controlling halo blight of bean in France. A seed transmission ratio of *Pseudomonas syringe* pv phaseolicola of 2:1 found by Taylor (15; 16) compares well with the ratic found in this study.

The minimum population of XCP \cdot initiate common bacterial blight infection was \cdot und to be 10^2 cfu/seed which was lower than 10^3 – 10^4 cfu/seed found by Weller and Saettler (20) in

USA. This may be attributed to differences in inoculum and bean types used, and environmental conditions. The higher temperatures involved in the present study may have enhanced development of disease symptoms from the lower populations of XCP in seed. Santana (9) reported that development of common bacterial blight was much faster on plants at higher temperatures (29.4°C) than lower temperatures (23.8°C). It is recommended that research on the XCP tolerance level needs to be carried out using more bean genotypes and locations.

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REFERENCES

- Aggour, A.R., D.P. Coyne, A.K. Vidaver and K.M. Eskridge, 1989. Transmission of the common blight pathogen in bean seed. *Journal of American Horticultural Science* 114: 1002–1008.
- 2. Cafati, C.R. and A.W. Saettler, 1980. Transmission of *Xanthomonas phaseoli* in seed of resistant and susceptible *Phaseolus* genotypes. *Phytopathology* 70: 638–640.
- 3. Caflin, L.E., A.K. Vidaver and M. Sasser, 1987. MXP, A semi selective medium for Xanthomonas campestris pv phaseoli. Phytopathology 77: 730-734.
- 4. Cochran, W.G. 1950. Estimation of bacterial densities by means of the 'most probable number'. *Biometrics* 6: 105–116.
- 5. Leakey, C.L.A. 1970. The improvement of beans (*Phaseolus vulgaris*) in beans in Eastern Africa. In: *Crop Improvement in East Africa*. Leakey, C.L.A. (Ed.), pp. 99–128. Farnham Royal, England.
- 6. Mukunya, D.M., P.M. Muthagya and J. Esele, 1981. Bacterial blight of beans (Phaseolus vulgaris L.) in Kenya. In: Proceedings of the Fifth International Conference on Plant Pathogenic Bacteria,

- 1981, Cali, Colombia. Lozano, J.C. (Ed.), pp. 3–9. Centro International de Agricultura Tropical.
- 7. Pastor Corrales, M.A., S.A. Beebe and F.J. Correa, 1981. Comparing two inoculation techniques for evaluating resistance in beans to Xanthomonas campestris pv phaseoli. In: Proceedings of the Fifth International Conference on Plant Pathogenic Bacteria, 1981, Cali, Colombia. Lozano, J.C. (Ed.), pp. 493–502. Centro International de Agricultura Tropical.
- 8. Saettler, A.W. 1989. Common bacterial blight. In: *Bean Production Problems in the Tropics*. Second edition. H.Z. Schwartz and M.A. Pastor-Corrales (Eds.), pp. 261–283.
- Santana, E.A.S. 1985. Genetic variation, inheritance and relationship of the reaction to common blight (Xanthomonas campestris pv phaseoli [(Smith) Dye] and number of days to flowering in dry beans (Phaseolus vulgaris L.). M.Sc. Thesis, University of Nebraska, Lincoln, 172pp.
- Schaad, N.W. 1988. Inoculum thresholds of seed-borne pathogens. Bacteria. *Phyto-pathology* 78: 872–875.
- Sengooba, T.N. 1985. The incidence and severity of the major diseases of beans in Uganda. Annual Report Bean Improvement Cooperative 28: 7–8.
- Sutton, M.D. and V.R. Wallen, 1970. Epidemiological and ecological relations of Xanthomonas phaseoli var fuscans in South Western Ontario 1961-1968. Canadian Journal of Botany 48: 1329-1334.
- 13. Taylor J.D. and K. Phelps, 1979. Most probable number (MPN) tables for estimation of seed infection. 6pp.
- 14. Taylor J.D. and K. Phelps, 1984. Basic methods for the detection of seed-borne

- bacteria 3. Estimation of percentage seed infection. In: Proceedings of the 1st International Workshop on Seed Bacteriology 4–9 October 1982, Angers, France. pp. 12–14. (Published by the ISTA Secretariat Zurich, Switzerland, 1984).
- Taylor, J.D., P. Kathleen and C.L. Dudley, 1979. Epidemiology and strategy for the control of halo-blight of beans. Annals of Applied Biology 93: 167-172.
- Taylor, J.D., C.L. Dudley and L. Presly (née Gray), 1979. Studies of halo blight seed infection and disease transmission in dwarf beans. Annals of Applied Biology 93: 267– 277.
- Trigalet, A. and P. Bidaud, 1978. Some aspects of epidemiology of bean halo blight. In: Proceedings of the Fourth International Conference of Plant Pathogenic Bacteria, 1978, Angers, France. Station de Pathologie Vegetale et Phytopathologie, pp. 895–902.
- 18. Vanderplank, J.E. 1963. *Plant Diseases:* Epidemics and Control. Academic Press, New York and London, 349 pp.
- Vidaver, A.K. 1967. Synthetic and complex media for the rapid detection of flourescence of photopathogenic pseudomonads. Effects of the carbon source. Applied Microbiology 15: 1523-1524.
- 20. Weller, D.M. and A.W. Saettler, 1980. Evaluation of seed-borne *Xanthomonas phaseoli* and *X. phaseoli* var. *fuscans* as primary inocula in bean blights. *Phytopathology* 70, 148-152.
- 21. Zaumeyer, W.J. and H.R. Thomas, 1957. A mono-graphic study of the bean diseases and methods for their control. United States Department of Agriculture. *Technical Bulletin* No. 868, 65-84.

