2008

Differential Pathogenicity of Xanthomonas campestris pv. phaseoli and X. fuscans subsp. fuscans Strains on Bean Genotypes with Common Blight Resistance

N. Mutlu
University of Nebraska-Lincoln

A. K. Vidaver
University of Nebraska-Lincoln, avidaver1@unl.edu

D. P. Coyne
University of Nebraska-Lincoln

J. R. Steadman
University of Nebraska-Lincoln, jsteadman1@unl.edu

P. A, Lambrecht
University of Nebraska-Lincoln, plambrecht1@unl.edu

See next page for additional authors

Differential Pathogenicity of *Xanthomonas campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* Strains on Bean Genotypes with Common Blight Resistance

N. Mutlu, Department of Agronomy and Horticulture, University of Nebraska, Lincoln 68583 and Alata Horticultural Research Institute, Erdemli-Mersin, Turkey; **A. K. Vidaver,** Department of Plant Pathology, University of Nebraska, Lincoln; **D. P. Coyne,** Department of Agronomy and Horticulture, University of Nebraska (deceased); **J. R. Steadman** and **P. A. Lambrecht,** Department of Plant Pathology, University of Nebraska; and **J. Reiser,** Department of Agronomy and Horticulture, University of Nebraska

ABSTRACT

Mutlu, N., Vidaver, A. K., Coyne, D. P., Steadman, J. R., Lambrecht, P. A., and Reiser, J. 2008. Differential pathogenicity of *Xanthomonas campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* strains on bean genotypes with common blight resistance. Plant Dis. 92:546-554.

Both the common bacterial blight (CBB) pathogen (*Xanthomonas campestris* pv. *phaseoli*) and *X. fuscans* subsp. *fuscans*, agent of fuscous blight, cause indistinguishable symptoms in common bean, *Phaseolus vulgaris*. Yield losses can exceed 40%. Lack of information about the specificity between *X. campestris* pv. *phaseoli* strains and major quantitative trait loci (QTL) or alleles conferring resistance makes the task of identifying genetic changes in host–pathogen interactions and the grouping of bacterial strains difficult. This, in turn, affects the choice of pathogen isolates used for germplasm screening and complicates breeding for CBB resistance. Common bean host genotypes carrying various sources and levels of resistance to CBB were screened with 69 *X. campestris* pv. *phaseoli* and 15 *X. fuscans* subsp. *fuscans* strains from around the world. Differential pathogenicity of the CBB pathogen was identified on the 12 selected bean genotypes. The *X. fuscans* subsp. *fuscans* strains showed greater pathogenicity than *X. campestris* pv. *phaseoli* strains having the same origin. African strains were most pathogenic. The largest variation in pathogenicity came from *X. campestris* pv. *phaseoli* strains that originated in Caribbean and South American countries. Pathogenic variation was greater within *X. campestris* pv. *phaseoli* than within *X. fuscans* subsp. *fuscans* strains. Implications for breeding for CBB resistance are discussed.

Common bean (*Phaseolus vulgaris*) is a grain legume of worldwide significance for direct human consumption (38). This important crop is attacked frequently and severely by common bacterial blight (CBB), a systemic (5), seed-transmitted (2) disease caused by *Xanthomonas campestris* pv. *phaseoli* (Smith) Dye and *Xanthomonas fuscans* subsp. *fuscans* (33). *X. fuscans* subsp. *fuscans* usually is readily distinguished from *X. campestris* pv. *phaseoli* by the brown melanin pigment it produces in media containing tyrosine (18). *X. fuscans* subsp. *fuscans* strains also produce a dark internal pigment and, thus, can be distinguished from *X. campestris* pv. *phaseoli* on MXP medium (7). Both *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* are typical plant-pathogenic xanthomonads and produce mucoid yellow colonies on common culture media. However, *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* are sufficiently distinct genetically to be classified into different groups based on 16S-23S intergenic spacer (ITS) regions (32), restriction fragment length polymorphism (RFLP) analyses of genomic and plasmid DNA (16), pulsed-field gel electrophoresis and RFLP (6), DNA-DNA hybridization (19), and amplified DNA polymorphisms (4,40,44). The *X. fuscans* subsp. *fuscans* strains have been reported to be more pathogenic than the *X. campestris* pv. *phaseoli* strains (30). However, disease symptoms on common bean of both groups are usually indistinguishable. Pathogenic variation has been reported among strains of *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* (28,35) but unequivocal evidence for existence of races, identifiable on common bean differential genotypes, has yet to be found.

The disease causes yield losses as well as reduction of seed quality through discoloration of infected seed. Yield losses from CBB are estimated to range from 10 to >40% in susceptible genotypes (42,44). There is no satisfactory chemical control of this disease. Various disease management methods, including the use of clean seed, crop rotation, and deep plowing of infected straw, have been proposed, but none is fully effective under conditions highly favorable for disease (31). For effective control, combined use of resistant cultivars and cultural management is essential. The level of resistance needed in a region depends on the frequency of conditions favorable for disease and effectiveness of other management strategies. The pathogenic variation among local *X. campestris* pv. *phaseoli* strains, if present, also will determine the level and source of CBB resistance needed.

Moderate resistance to CBB has been found in common bean, with a comparatively higher level of resistance in scarlet runner bean (*P. coccineus* L.) and the highest level of resistance in tepary bean (*P. acutifolius* A. Gray) (9,26,36,45). A single recessive allele controlled resistance in a Bulgarian snap bean mutant (1); however, quantitative inheritance with predominant additive gene action was reported for resistance found in Great Northern (GN) Nebraska #1 sel. 27 (10). In contrast, a single dominant allele (12) and three linked dominant alleles, each for resistance to a different *X. campestris* pv. *phaseoli* isolate (13,15,41) were reported in tepary bean. The highest level of CBB resistance from different species recently was introgressed and pyramided into common bean (36,37). Currently, five to eight major and minor alleles and QTL are estimated to control resistance to CBB in common bean, including those introgressed from the scarlet runner (24,29) and tepary beans (14,21,27).

Races of *X. campestris* pv. *phaseoli* and a standard set of differential host genotypes with a standard rating scale were identified in separate studies by Zapata and Vidaver (49) and Opio et al. (28) for tepary bean. However, the investment in resources and time may not justify continued work with a *P. acutifolius* system (39) compared with the need for a set of differential genotypes to detect races of *X. campestris* pv. *phaseoli* in *P. vulgaris*. Pathogenic variation in *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* based on six dry bean genotypes and specificity in reactions between *X. campestris* pv. *phaseoli* and some common bean genotypes from the Caribbean area has been reported (47–49). However, Jara et al. (20) in Latin America and Opio et al. (28) working in Africa were not able to detect races of *X. campestris* pv. *phaseoli* on common bean.

Corresponding author: A. K. Vidaver
E-mail: AVIDAVER1@unl.edu

Accepted for publication 9 November 2007.

doi:10.1094/PDIS-92-4-0546

At the first international workshop on CBB in common bean, held at the University of Puerto Rico in 1996, the identification of races of *X. campestris* pv. *phaseoli* and a set of common bean differential genotypes based on compatible versus incompatible reactions to *X. campestris* pv. *phaseoli* strains using a standardized inoculation procedure and disease rating system was considered very important for future development of resistant cultivars (48).

Our objectives were to (i) determine the feasibility of identifying a set of common bean genotypes to differentiate strains of *X. campestris* pv. *phaseoli* based on low versus high virulence, (ii) elucidate the extent of pathogenic variation between and within strains of *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans*, and (iii) assess the influence of strain variation on resistance deployment.

MATERIALS AND METHODS

Thirty-four bean genotypes with different levels of resistance to CBB from diverse geographic origins were obtained from Centro Internacional de Agricultura Tropical (CIAT, Cali, Colombia) and elsewhere. The sources of resistance to CBB on common bean genotypes originated from *P. vulgaris* (GN Nebraska #1 sel. 27), *P. coccineus* (XR-235-1-1), and *P. acutifolius* (XAN 159, XAN 161, and VAX). Recently, CBB resistance from a tepary bean plant introduction (PI) via XAN 159 and G 40001 via VAX 1 and VAX 2; GN Nebraska #1 sel. 27 and PI 207262 were pyramided at CIAT to produce dry bean breeding lines XAN 263, XAN 309, VAX 3, VAX 4, VAX 5, and VAX 6 with high levels of CBB resistance (36,37).

To obtain pure lines for this study, single plant selections were made (without inoculation) from greenhouse-grown plants in Lincoln, NE in each of two generations from the original seed sources. Plants were grown in a greenhouse with a 14-h day length using natural and supplemental Sodium Halide-4000W lighting and 27 ± 2°C day and 20 ± 3°C night temperature regimes. Seed of these genotypes were planted in clay pots containing 1.8 liters of a potting mixture of equal parts (by volume) Sharpsburg silty clay loam soil, sand, peat moss, and vermiculite. The plants were fertilized daily after the first trifoliolate was fully expanded with a nutrient solution containing 20-10-20 (NPK) fertilizer at 200 ppm through an irrigation drip system. Fertilization was reduced to every other day after pod set.

Bacterial strains and plant inoculation. *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* strains used in experiments, including the initial 9 strains (Table 1), originated from 18 different countries (Table 2). In all, 84 strains were tested in this study. Origin of the strains was North America (n = 25), the Caribbean (n = 18), South America (n = 17), Africa (n = 14), Australia (n = 5), Europe (n = 4), and New Zealand (n = 1). The total number of strains of *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* was 69 and 15, respectively.

The strains were cultured on MXP semiselective medium (7) for 72 h at 27°C, transferred to 0.0125 M potassium phosphate buffer (pH 7.1), and diluted to an absorbance at 640 nm of 0.1 using a spectrophotometer (Spectronic 20; Bausch and Lomb, Rochester, New York). A final concentration of 10^7 CFU/ml of each strain in

Table 1. Mean percent leaf area affected by common bacterial blight for 36 dry bean genotypes inoculated with nine *Xanthomonas campestris* pv. *phaseoli* strains in the greenhouse at Lincoln, NE on 6 November 2000 and 3 March 2001 and evaluated at 21 days postinfection

		Strains									
Genotypes	**Seed color**	**FA-A2$_{US}$**	**215$_{BR}$**	**1811 NCPPB$_{RO}$**	**1X2$_{HUN}$**	**484A$_{PR}$**	**DR7$_{DR}$**	**82-1$_{US}$**	**SX 114$_{SA}$**	**X6$_{SA}$**	**Means/lines**
VAX 1	Carioca	0	10	0	3	2	2	8	7	10	5
VAX 2	Gray/tan	1	2	3	1	3	3	3	10	10	4
VAX 3	Maroon	1	8	3	3	2	3	7	4	4	4
VAX 4	Dark cream	0	8	4	0	1	4	0	2	2	2
VAX 5	Black	1	10	1	3	1	1	4	10	4	4
VAX 6	Maroon	0	1	0	0	0	0	0	2	4	1
WILK 2	White	1	9	4	23	5	9	5	12	14	9
WILK 4	White	2	7	2	23	4	2	5	5	5	6
WILK 6	White	7	6	25	11	10	9	50	20	8	16
G 6415	Dark pink	6	90	6	49	16	34	40	50	48	38
DRK-47	Maroon	5	70	4	25	5	16	6	80	85	33
G 8083	Red mottled	2	65	18	100	55	64	8	80	68	51
G 17813	Black	18	100	50	100	90	78	90	90	95	78
G 5034	Purple/gray	14	100	50	100	65	95	100	100	70	77
G 22247	Red mottled	2	65	18	100	55	64	8	80	68	51
S 31447	Purple	30	100	35	98	60	100	70	80	80	73
G 1688	Black	35	90	80	100	80	95	60	75	90	74
G 4399	Cream	25	90	40	88	32	90	11	60	90	56
G 21715	Yellow	5	95	35	65	45	85	65	80	90	60
G 6861	Gray/purple	30	100	80	98	60	98	80	100	100	80
G 5164	Pink mottled	62	90	65	95	75	95	80	90	90	82
G 22033	Pink/cream mottled	35	100	61	100	65	100	90	100	90	82
G 1373	Purple/green	13	98	60	95	80	100	75	90	95	78
G 13774	Blue/cream mottled	45	97	40	92	40	85	70	80	95	72
G 4756	Light tan	15	100	65	98	75	98	90	85	90	78
CAL 149	Red mottled	9	30	20	39	50	8	20	35	63	30
XAN 176	Black	8	75	5	4	10	67	20	35	20	25
TARS-VCR-43	Pinto	2	45	4	15	9	14	4	45	70	23
POMJOR 19	Red mottled	3	75	7	74	30	61	4	66	35	39
PR 9943-4	Pink	4	80	25	46	8	67	50	65	40	43
BAT 93	Cream/green	35	80	59	53	25	70	35	55	30	49
MONTCALM	Dark red	7	75	13	41	13	61	60	65	20	39
XR-235-1-1	White	3	90	2	46	4	4	4	20	66	27
OAC-88-1	White	4	70	4	20	10	58	6	25	61	29
PC50	Red mottled	9	90	45	100	55	90	100	80	44	68
UI 114	Pinto	55	100	75	100	80	100	90	95	80	86
Means/strains	…	13.8	64.3	27.7	55.7	34.2	54.1	39.4	54.8	54.5	…
Mean LSD[a]	…	1.64	1.64	1.64	1.64	1.64	1.64	1.64	1.64	1.64	…

[a] LSD = least significant difference.

buffer was prepared and kept on ice for short intervals (about 30 min) before inoculation.

The multiple needle method (3) was used to inoculate the first fully developed trifoliolate leaves at 21 to 28 days after planting, depending on plant development. A leaf was placed on a cheesecloth pad saturated with a bacterial suspension in a petri dish and perforated with a multiple-needle florist frog (4 cm in diameter with 61 needles 12 mm in length and 3 mm apart). Bacterial inoculum was drawn into the wounds upon removal of the multiple-needle device. Each strain was used to inoculate two leaflets of a trifoliolate leaf on each plant. Disease reactions were recorded separately for each strain. The potassium phosphate buffer was used as a control inoculation. The percentage of inoculated leaf area with CBB symptoms consisting of necrosis, water-soaking, and chlorosis was recorded visually 14 and 21 days after inoculation. The common bean genotype Pinto UI 114 was used as a universal susceptible control. The pots were arranged in a split-plot design with common bean genotypes as main plots and *X. campestris* pv. *phaseoli* or *X. fuscans* subsp. *fuscans* strains as subplots. Two plants of the same bean genotype were in a pot and there were two pots per replication. Two leaves were inoculated per plant per isolate, and four readings were made per isolate at each time. Experiments were replicated twice in time with isolates showing differential pathogenicity. Initial inoculations were made with the nine strains representing different continents on 6 November 2000, and repeated using the same strains on 3 March 2001 on 34 common bean genotypes and two susceptible checks to identify candidate common bean genotypes with differential expression to *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* strains (Table 1). Thus, 10 genotypes were selected based on their differential responses. The genotypes VAX 1, VAX 2, VAX 4, VAX 6, WILK 2, WILK 4, G 22247, TARS-VCR-43, POMJOR 19, and XR-235-1-1, and the susceptible check Pinto UI 114, and moderately resistant GN Nebraska #1 sel. 27 were evaluated with 23, 27, and 30 more strains on 23 May, 21 November, and 26 December 2001, respectively. The common bean genotype XR-235-1-1 (Table 2), which was used in the 23 May 2001 test, was replaced with XAN-159 in later tests (21 November and 26 December 2001). The final test, on 18 April 2002, was conducted to repeat the experiment with a subset of 31 strains that showed a differential pathogenicity response in the previous tests (Table 2).

Data analysis for clustering. The standardized CBB data of percent leaf area affected by each strain was used for analysis with the Numerical Taxonomy Multivariate Analysis System (NTSYS)-pc (ver-

Table 2. Mean percent leaf area with symptoms in common bean genotypes to *Xanthomonas campestris* pv. *phaseoli* (*Xcp*) and *X. fuscans* subsp. *fuscans* (*Xff*) strains of different geographical origin evaluated in a greenhouse at Lincoln, NE in 2000, 2001, or 2002

			Genotype[a]												
Origin	Strains	Taxon	VAX 1	VAX 2	VAX 4	VAX 6	WILK 2	WILK 4	G2224 7	Tars VCR 43	Pomjor 19	XR-235-1	XAN 159	UI 114	GN NE #1
South Africa	SX 119[b]	*Xff*	23	50	0	0	33	0	21	100	35	–	95	100	100
South Africa	X122	*Xff*	13	10	4	18	18	50	100	100	100	–	21	100	75
South Africa	X-99	*Xff*	61	38	4	21	3	1	78	88	100	–	13	98	83
South Africa	X-120[b]	*Xff*	30	21	4	11	9	0	100	98	90	–	4	100	100
South Africa	SX 111	*Xff*	56	15	8	3	4	1	78	80	93	–	4	100	95
South Africa	SX 113	*Xff*	23	26	3	3	1	1	100	69	76	–	1	88	23
South Africa	SX 127[b]	*Xff*	20	38	0	0	3	0	18	43	15	4	–	85	78
South Africa	X6	*Xcp*	15	11	0	0	8	1	57	63	14	28	–	100	90
South Africa	X 95[b]	*Xcp*	75	65	14	4	26	1	100	100	95	–	14	100	83
South Africa	SX 117	*Xcp*	4	14	0	3	19	20	95	100	100	–	39	100	58
South Africa	SX 124	*Xcp*	5	0	0	0	70	39	30	78	31	–	13	100	31
South Africa	X-97	*Xcp*	35	26	6	10	4	3	80	90	84	–	10	98	78
Zambia	1138 NCPPB	*Xcp*	11	0	4	0	1	0	50	14	60	0	–	48	16
New Zealand	2729 PDDCC	*Xcp*	85	48	9	0	19	14	40	100	73	65	–	100	93
Australia	0526[b]	*Xcp*	0	0	13	8	15	10	21	10	18	4	–	100	21
Australia	1646 NCPPB	*Xcp*	18	31	9	6	8	14	88	23	100	64	–	100	71
Australia	0386	*Xcp*	4	0	1	1	3	4	24	10	26	4	–	100	31
Australia	0708	*Xff*	85	75	13	13	8	1	45	100	28	88	–	100	100
Australia	0794	*Xff*	85	73	8	3	9	0	100	100	100	70	–	100	100
Romania	1811 NCPPB	*Xcp*	5	1	9	3	0	0	5	11	3	3	–	100	5
England	1158B[b]	*Xff*	65	44	0	0	0	0	9	70	5	9	–	40	14
England	1158A[b]	*Xff*	21	23	0	0	0	1	8	10	9	25	–	85	33
Argentina	95 Argentina[b]	*Xcp*	93	100	11	5	0	6	31	100	26	100	–	100	100
Brazil	5254[b]	*Xcp*	33	19	0	0	11	1	20	98	46	78	–	100	100
Brazil	B702	*Xcp*	0	0	3	3	0	0	80	10	93	–	1	100	5
Brazil	B704	*Xcp*	4	0	0	1	0	3	5	53	0	–	3	78	41
Brazil	B705	*Xcp*	0	0	0	0	6	20	19	13	10	–	34	100	49
Colombia	270	*Xcp*	39	26	9	0	10	3	85	93	70	45	–	100	61
Colombia	184 CBP	*Xcp*	3	1	1	5	13	5	100	38	100	–	9	100	44
Colombia	123 Colombia	*Xcp*	0	3	0	9	60	26	19	90	31	–	28	100	54
Mexico	53	*Xcp*	11	53	1	1	21	4	95	100	93	–	3	100	90
Paraguay	355	*Xcp*	14	25	0	5	20	4	35	100	61	–	8	100	100
Paraguay	354	*Xcp*	14	28	0	3	6	100	31	100	43	–	33	100	98
Honduras	95-06	*Xff*	74	83	21	16	100	54	60	100	78	–	73	100	100
Honduras	95-04[b]	*Xcp*	25	13	1	0	0	0	38	65	50	–	0	95	43
Honduras	95-03	*Xcp*	23	18	0	5	-	-	45	70	62	–	4	100	38
Nicaragua	364[b]	*Xcp*	1	3	0	0	0	10	29	11	29	–	4	100	36
Guatemala	044 CBP	*Xcp*	63	20	0	0	1	1	80	83	60	21	–	100	60
Haiti	042 CBP	*Xcp*	4	6	1	0	0	1	29	33	45	–	1	95	15

(*continued on next page*)

[a] Abbreviations: GN NE #1 = GN Nebraska #1 sel 27, – = not included, Dominican Rep. = Dominican Republic, and nc = not calculated.
[b] Strains were replicated in time (April 2002).
[c] Least significant difference (LSD) 9.1 for XR-235-1 due to missing data.
[d] LSD 6.4 for XAN 159 due to missing data.

sion 2.0; Rohlf 1998; State University of New York, Stony Brook). The distance matrix and dendrogram were constructed using the NTSYS-pc. Similarities among strains were assessed by multivariate analysis of similarity coefficients (Simint) and a matrix of similarity coefficients for each pair of strains was constructed. Principal component analysis (PCA) was performed using standardized data to obtain a graphical representation of the relationship structure of the 84 *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* strains tested. A cluster analysis (SAHN) on the similarity coefficients was performed with the unweighted pair-group method algorithm (UPGMA) contained in NTSYS-pc.

RESULTS

The initial screenings were carried out with 36 common bean genotypes, including two CBB susceptible checks from diverse origins (Table 1). The screening with nine *X. campestris* pv. *phaseoli* strains representing different geographical regions showed the extent of variation present and diversity of both host resistance and pathogen virulence (Table 1). The *X. campestris* pv. *phaseoli* strains FA-A2 (United States) and 215 (Brazil) exhibited the lowest and highest mean percent leaf damage, respectively, over all hosts tested (Table 1). WILK 2, WILK 4, and VAX genotypes consistently showed a resistant or moderate resistance response to all nine strains, whereas the CIAT G accessions ranged from susceptible to resistant in their responses. However, CIAT genotype G 22247 was selected for further testing due to its tendency for differential response to the strains tested. The checks Pinto UI 114 and PC 50 were uniformly susceptible to all strains. VAX 4 and VAX 6 were the most resistant genotypes to the nine strains tested, whereas CIAT accessions G 5164, G 22033, and G 6861 were nearly as susceptible as the checks (Table 1). Based on the results from screening with nine strains, VAX 1, VAX 2, VAX 4, VAX 6, WILK 2, WILK 4, G 22247, TARS-VCR-43, POMJOR 19, and XR-235-1-1 were selected for further evaluation. A well-studied resistance source, GN Nebraska #1 sel. 27, was added to this group and XAN 159 replaced XR-235-1-1 after the initial test because it was used as a source of resistance in our breeding program. These genotypes represented wide sources of resistance that originated from different gene pools in the common bean host background.

The disease reaction of 84 strains on 13 common bean genotypes is presented in Table 2. The mean percent disease caused by *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* differed significantly on VAX 1, VAX 2, TARS-VCR 43, POMJOR 19, and GN Nebraska # 1 sel. 27.

Table 2. (*continued from preceding page*)

			Genotype[a]												
Origin	**Strains**	**Taxon**	**VAX 1**	**VAX 2**	**VAX 4**	**VAX 6**	**WILK 2**	**WILK 4**	**G2224 7**	**Tars VCR 43**	**Pomjor 19**	**XR-235-1**	**XAN 159**	**UI 114**	**GN NE #1**
Dominican Rep.	DRL-808	*Xcp*	4	0	0	1	8	8	93	4	100	–	8	100	9
Dominican Rep.	DRL-816[b]	*Xcp*	1	0	1	1	5	8	95	4	95	–	11	100	16
Dominican Rep.	AL2	*Xcp*	8	4	0	6	8	13	100	23	100	–	26	100	23
Dominican Rep.	DRS-103	*Xcp*	31	11	4	16	5	4	36	95	13	–	1	100	83
Dominican Rep.	DRA-304	*Xcp*	6	8	0	1	19	0	81	30	100	–	10	100	10
Dominican Rep.	DRL-814[b]	*Xcp*	5	0	3	20	8	13	100	35	100	–	10	100	23
Dominican Rep.	DRH-203	*Xcp*	5	3	1	0	44	36	100	14	95	–	8	100	13
Dominican Rep.	DRL-843	*Xcp*	1	4	3	0	26	81	100	21	100	–	28	100	56
Dominican Rep.	DRC-402	*Xcp*	0	1	0	0	19	11	88	25	83	–	3	100	15
Dominican Rep.	DRL-834[b]	*Xcp*	56	45	0	0	11	3	100	100	10	49	–	100	100
Dominican Rep.	DR-7	*Xcp*	5	5	3	3	14	9	100	14	100	5	–	100	36
Puerto Rico	484A	*Xcp*	0	1	1	0	5	0	85	3	88	8	–	68	3
Puerto Rico	20003	*Xcp*	6	31	1	0	5	1	21	41	29	18	–	100	46
Puerto Rico	9902[b]	*Xcp*	15	4	3	1	0	3	100	100	95	–	5	100	80
Puerto Rico	T-44[b]	*Xcp*	28	8	1	5	1	1	48	63	94	–	10	100	40
Puerto Rico	T-26	*Xcp*	1	0	0	3	0	0	48	8	20	–	0	79	5
Puerto Rico	820	*Xcp*	0	1	0	0	0	0	55	10	18	–	0	79	3
Canada	6022/112-1[b]	*Xcp*	3	5	0	0	0	11	80	85	90	–	10	75	54
Canada	60-Or	*Xcp*	1	4	5	9	20	1	49	43	43	–	60	100	73
Canada	381 NCPPB[b]	*Xff*	9	24	0	28	21	1	28	90	0	–	5	100	85
Kansas	KS-10[b]	*Xcp*	8	0	1	6	4	3	100	13	91	–	13	100	16
Nebraska	XpfS2	*Xff*	19	16	1	3	0	1	6	46	3	–	4	94	54
Michigan	2-DRK	*Xcp*	15	20	4	1	3	3	14	88	13	–	1	100	78
Nebraska	EK-11-1	*Xcp*	1	3	3	1	0	0	1	30	1	–	0	63	14
North Dakota	95-41	*Xcp*	11	0	1	0	0	0	1	20	1	–	0	93	20
Nebraska	SB7-191	*Xcp*	0	9	0	1	0	0	14	9	14	–	0	49	9
North Dakota	95-39[b]	*Xcp*	24	18	1	5	0	0	68	85	45	–	3	88	50
North Dakota	X2-99[b]	*Xcp*	0	0	0	0	0	0	10	12	0	–	0	35	2
Nebraska	SGN	*Xcp*	11	1	4	1	5	0	21	34	44	–	0	89	38
Nebraska	Xpsf seed	*Xff*	73	60	3	25	6	3	13	32	1	–	4	93	85
Colorado	S-84	*Xcp*	21	3	5	15	8	13	83	83	88	–	10	100	50
Florida	1986B5[b]	*Xcp*	4	5	0	0	43	0	0	50	5	–	3	45	11
Michigan	9712-5	*Xcp*	5	15	0	1	1	6	25	100	16	–	8	98	98
Michigan	Mich-3	*Xcp*	14	38	1	0	85	40	31	100	44	–	5	100	100
Nebraska	9807	*Xcp*	13	19	8	3	23	3	9	95	44	–	1	100	85
Nebraska	SC-4A[b]	*Xcp*	0	0	3	0	21	4	9	19	9	–	0	80	11
Nebraska	LB-2	*Xcp*	6	3	5	15	51	19	100	35	100	–	76	100	50
Florida	84-5	*Xcp*	0	0	0	40	1	3	4	40	0	–	53	41	43
Kansas	G-24[b]	*Xcp*	1	0	0	1	25	1	100	39	100	–	10	100	100
Nebraska	K3A[b]	*Xcp*	70	39	0	5	1	1	100	90	100	88	–	100	95
Florida	82-1	*Xcp*	3	3	0	0	0	0	8	9	9	13	–	100	21
Means/lines	…	…	20.3	17.7	2.7	4.7	12.7	8.9	53.4	55.6	54.3	35.6	13.8	92.2	52.7
Mean Xcp	…	…	14.9	12.6	2.4	3.6	12.2	9.1	54.0	51.2	55.5	nc	nc	92.2	47.5
Mean Xff	…	…	43.8	39.7	4.6	9.6	14.3	7.6	50.9	75.1	48.9	nc	nc	92.2	75.0
LSD	…	…	5.8	5.8	5.8	5.8	5.8	5.8	5.8	5.8	5.8	9.1[c]	6.4[d]	5.8	5.8

However, VAX 4, VAX 6, WILK 2, and WILK 4 lines showed a more consistent resistance to *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* strains. WILK 2, WILK 4, and POMJOR 19 exhibited a slightly higher resistance to *X. fuscans* subsp. *fuscans* than to *X. campestris* pv. *phaseoli* strains (Table 2). VAX 1 and VAX 2 exhibited susceptible reactions to most of the *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* strains originating from continental Africa with a mean percent CBB of 29 and 24, respectively, whereas VAX 4 and VAX 6 showed either complete or moderate resistance to the same strains with a mean of 4 and 6% CBB, respectively. WILK 2 and WILK 4 had 15 and 9% CBB, respectively, and GN Nebraska #1 sel. #27 had 70% to the same African strains. The mean percent CBB disease averaged over common bean genotypes by origin were as follows: African *X. campestris* pv. *phaseoli* strains, 38%, and *X. fuscans* subsp. *fuscans* strains, 43%; North American *X. campestris* pv. *phaseoli* strains, 27%, and *X. fuscans* subsp. *fuscans* strains, 29%; and South American *X. campestris* pv. *phaseoli* strains, 33% (Table 3). When strain origins were averaged irrespective of species, the mean % CBB were 40, 27, and 34 for African, South American, and North American strains, respectively (Table 3).

The *X. campestris* pv. *phaseoli* strains showed significant variation in virulence within and between regions (Table 4). Analysis of variance showed that strains, origin of strains, common bean genotypes, and strain–genotype interaction all were highly significant (P = 0.0001; Table 4). The greatest *X. campestris* pv. *phaseoli* strain–common bean genotype variation was seen in strains from the Caribbean and South America. The strains from the Americas showed greater within-line variation in pathogenicity than strains from Africa; however, the variation between common bean lines for resistance was high regardless of geographical origin (Table 3). Mean percent CBB was 41 and 31 for *X. fuscans* subsp. *fuscans* and *X. campestris* pv. *phaseoli* strains, respectively. African and North American *X. fuscans* subsp. *fuscans* strains caused 43 and 29% CBB, whereas *X. campestris* pv. *phaseoli* strains from the same region caused 38 and 27% CBB, respectively. The levels of virulence between *X. fuscans* subsp. *fuscans* strains originating from different continents were all significantly different. For *X. campestris* pv. *phaseoli*, however, the strains from North America and Australia were significantly less virulent than the strains from Africa and South America (Table 3). There were not enough strains from Europe to be able to generalize the mean differences of *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans*. However, differences were not significant between Australian and North American *X. campestris* pv. *phaseoli* strains (27 versus 27%) and between South American and African *X. campestris* pv. *phaseoli* strains (33 versus 38%).

A set of differential common bean genotypes based on low versus high percent disease reactions to *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* strains composed of VAX 1, VAX 2, XAN 159, WILK 2, and WILK 4 is presented in Table 5. Strains that produced a highly susceptible (high virulence) compared with a highly resistant reaction in the five differential common bean genotypes comprise nine *X. campestris* pv. *phaseoli* (123

Table 3. Summary of mean percent common bacterial blight disease on 13 common bean genotypes caused by *Xanthomonas campestris* pv. *phaseoli* and *Xanthomonas fuscans* subsp. *fuscans* strains originating from different regions of the world

Origin of strain	*X. campestris* pv. *phaseoli* mean (%)	*X. fuscans* subsp. *fuscans* mean (%)	Overall mean (%)
Africa	38 (6)[a]	42.6 (7)	40 (13)
North America	21 (21)	28.8 (3)	27 (24)
South America	33 (33)	71 (1)	34 (34)
Australia	26.6 (3)	58.5 (2)	39 (5)
Europe	12 (1)	20 (2)	17 (3)
Overall mean (%)	27.3	44.2	31.4

[a] Numbers in parentheses indicate the number of strains from which means were calculated. Least significant difference = 5.8.

Table 4. Summary of single-factor analyses of variance of common bacterial blight (CBB) reaction on 13 common bean genotypes with 77 *Xanthomonas campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* strains in a greenhouse experiment, Lincoln, NE, USA

Source of variation	df	Mean square	Probability
Strain	74	8,588	0.0001
Bean genotype	12	237,195	0.0001
Region of strain[a]	5	19,392	0.0001
Blocks	1	85	0.60
Replication (blocks × strain)[b]	160	329	0.27
Region × bean genotypes	58	5,718	0.0001
Strain × bean genotypes	810	2,145	0.0001

[a] North America, South America, Caribbean, Africa, Asia, and Europe.
[b] Interactions between region and bean genotypes or strain and bean genotypes.

Table 5. Strains of *Xanthomonas campestris* pv. *phaseoli* (*Xcp*) and *X. fuscans* subsp. *fuscans* (*Xff*) showing differential pathogenicity and virulence on bean *(Phaseolus vulgaris)* genotypes VAX 1, VAX 2, WILK 2, WILK 4, and XAN 159[a]

Origin	Strains	Taxon	VAX 1	VAX 2	WILK 2	WILK 4	XAN 159
South Africa	SX 127[b]	*Xff*	20	38	3	0	–
South Africa	SX 119[b]	*Xff*	23	50	33	0	95
Australia	0794	*Xff*	85	73	9	0	–
England	1158B[b]	*Xff*	65	44	0	0	–
England	1158[b]	*Xff*	21	23	0	1	–
Colombia	123 Colombia	*Xcp*	0	3	60	26	28
Argentina	95 Argentina[b]	*Xcp*	93	100	0	6	–
Honduras	95-04[b]	*Xcp*	25	13	0	0	0
North Dakota	95-39[b]	*Xcp*	24	18	0	0	3
Nebraska	K3A[b]	*Xcp*	70	39	1	1	–
South Africa	SX 124	*Xcp*	5	0	70	39	13
Kansas	G-24[b]	*Xcp*	1	0	25	1	10
Florida	84-5	*Xcp*	0	0	1	3	53
Brazil	B705	*Xcp*	0	0	6	20	34
LSD	…	…	5.8	5.8	5.8	5.8	6.4

[a] Mean percentage leaf area affected by common bacterial blight was measured 21 days postinoculation in a greenhouse; – = not included and LSD = least significant difference.
[b] Strains were replicated in time.

Colombia, 95 Argentina, 95-04, 95-39, K3A, SX124, G-24, 84-5, and B-705) and five *X. fuscans* subsp. *fuscans* (1158A, 1158B, 0794, SX119, and SX127) strains (Table 5). The five *X. fuscans* subsp. *fuscans* strains in this differential group were always more virulent on VAX 1 and VAX 2 than on WILK 2 and WILK 4. Both VAX 4 and VAX 6 were highly resistant to most strains.

Clustering of *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* strains based on pathogenicity level derived from CBB severity on inoculated plants 21 days post-inoculation can be illustrated in a dendrogram (Fig. 1). Similarities among strains ranged from 0.31 to 1.00, with a mean similarity of 0.66 (Fig. 1). The UPGMA clustering algorithm grouped the strains into seven clusters of 42, 17, 17, 5, 1, 1, and 1 strain. The two clusters contained 12 of the 15 *X. fuscans* subsp. *fuscans* strains (Fig. 1). *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* strains from different geographical regions grouped together. The cophenetic coefficient was 0.68, indicating a moderate fit. A PCA was carried out using the standardized CBB disease severity (Fig. 2). The first component, which accounted for 66% of the total variation, and the second component, which accounted for 15% of the total variation, failed to group the strains according to geographic origin or *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* taxa.

DISCUSSION

This study represents the most comprehensive, largest contemporary study with respect to the number of *X. campestris* pv. *phaseoli* strains tested, diversity of their origin, and common bean host genotypes that represent known sources of resistance to CBB.

Most studies on pathogenic variation, especially with genotypes derived from GN Nebraska #1 sel. 27, indicate no hypersensitive reactions and only degrees of compatibility using the multiple-needle method of inoculation (3). Studies on compatibility have been limited mostly to regional common bean genotypes and pathogen strains and lacked a wide geographic representation. In all, 7 common bean genotypes out of 120 from a nursery showing high levels of resistance to CBB in Central America 'Vivero de Fuentes de Resistancia de Centro America' demonstrated specific susceptibility to three *X. campestris* pv. *phaseoli* strains, one each from Puerto Rico (484a), Dominican Republic (872), and Costa Rica (924) (47). From these results, Zapata (38) concluded that these three *X. campestris* pv. *phaseoli* strains represent physiological races of the bacterium and reported differential responses on *P. vulgaris* by using a pipette inoculation procedure. However, Opio et al. (28), working with 30 African *X. campestris* pv. *phaseoli* strains and 20 common bean genotypes, including some African common bean landraces, found a quantitative host-nonspecific resistance. VAX 4 showed higher resistance to *X. fuscans* subsp. *fuscans* and *X. campestris* pv. *phaseoli* than VAX 6, and a similar result was reported by Jara et al. (20) using 8 *X. fuscans* subsp. *fuscans* and 12 *X. campestris* pv. *phaseoli* strains. The amount of inoculum used in these inocula-

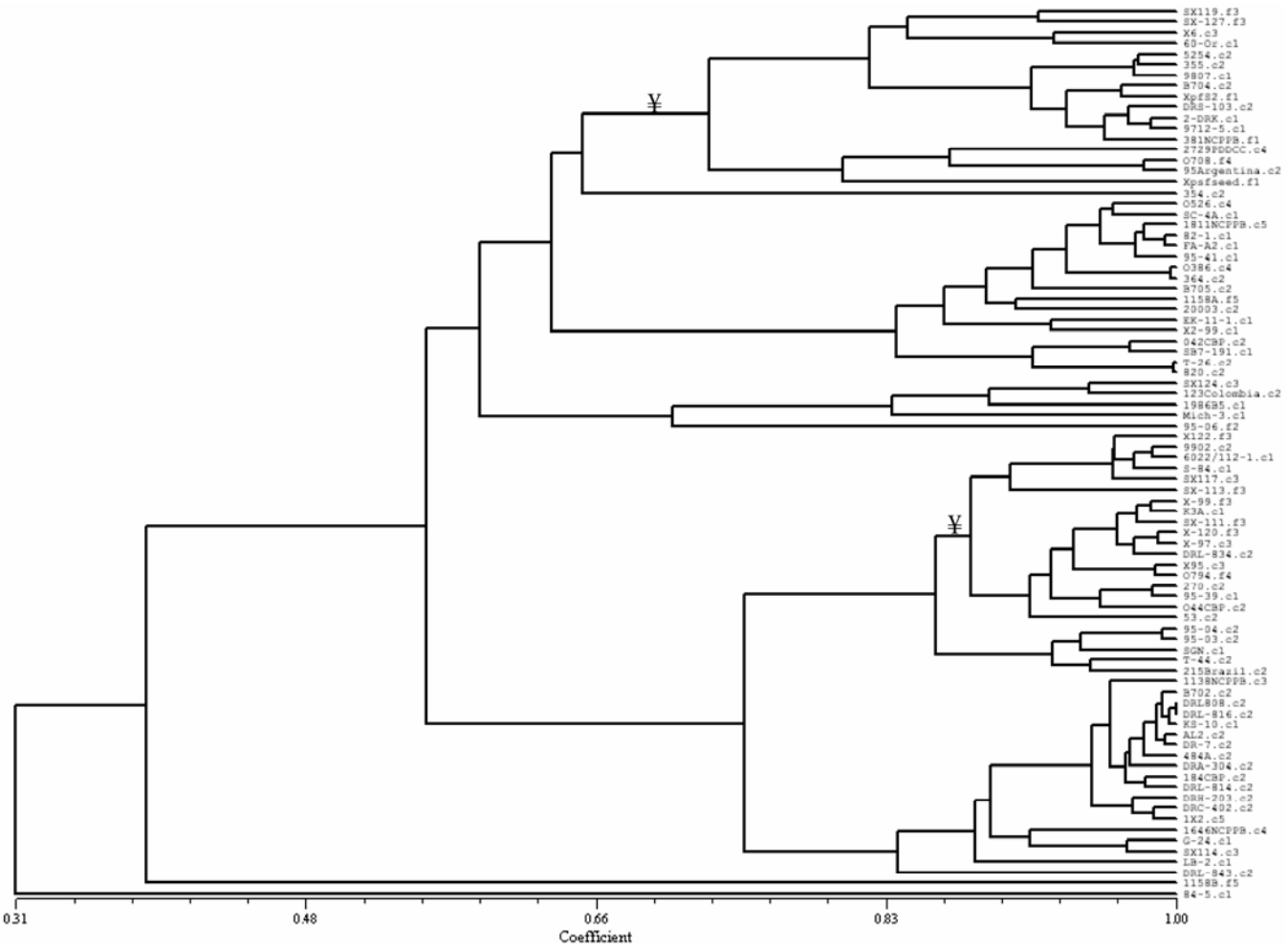

Fig. 1. Unweighted pair-group method with arithmetic averages dendrogram of genetic relationships among 84 *Xanthomonas campestris phaseoli* and *X fuscans* subsp. *fuscans* strains calculated on the basis of genetic similarity analysis by means of actual percent leaf area common bacterial blight disease on the inoculated bean leaf at 21 days after inoculation on 13 common bean genotypes. Strain numbers are followed by their geographic origin (¥ = the two clusters containing 12 of the 15 *X. fuscans* subsp. *fuscans* strains; suffix following the names of strains: c = *X. campestris* pv. *phaseoli*, f = *X. fuscans* subsp. *fuscans*, 1 = North America, 2 = South America, 3 = Africa, 4 = Australia and New Zealand, 5 = Europe, and 6 = Asia).

tion methods may have differed and this could influence the results. Differential virulence reactions of strains of *X. campestris* pv. *phaseoli* also were observed on *P. vulgaris* in Nebraska (34). However, clear differential reactions to *X. campestris* pv. *phaseoli* have been reported by several investigators on *P. acutifolius* (28,46,47).

In this study, differential pathogenicity was evident among the *X. campestris* pv. *phaseoli* strains from Africa and the Americas and between *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* strains. These results are similar to the results reported by Mkandawire et al. (25), showing that differences in pathogenicity exist between African *X. campestris* pv. *phaseoli* strains and strains from the rest of the world (Table 3). However, unlike the results of Mkandawire et al. (25), pathogenic differences were detected among *X. fuscans* subsp. *fuscans* strains originating from Africa and North America (Table 3) and, although North American *X. fuscans* subsp. *fuscans* and *X. campestris* pv. *phaseoli* strains possessed similar pathogenicity, African *X. fuscans* subsp. *fuscans* strains were more pathogenic than African *X. campestris* pv. *phaseoli* strains (Table 3). Thus, genetically distinct *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* strains exist, with differential pathogenicity determined on common bean genotypes with resistance derived from different *Phaseolus* spp. in different gene pools. It now seems clear that two distinct genetic entities (i.e., *X. c*ampestris pv. *phaseoli* and *X. fuscans* subsp. *fuscans*; 32) cause symptoms that are largely indistinguishable. *X. fuscans* subsp. *fuscans* strains were pathogenic on both Andean and Middle American germplasm (22). The practical significance of this knowledge is that examination of Middle American germplasm by breeders and plant pathologists for CBB resistance genes would benefit the breeding program in East Africa (11).

The *X. fuscans* subsp. *fuscans* strains were more pathogenic than *X. campestris* pv. *phaseoli* strains irrespective of their origin. Mean virulence differences of *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* on resistant bean lines indicate that VAX 4, VAX 6, WILK 2, and WILK 4 should be preferred as resistance sources in areas infested largely with *X. fuscans* subsp. *fuscans* strains. VAX 1 and VAX 2 do not provide adequate resistance for *X. fuscans* subsp. *fuscans* strains. They are not recommended as sole resistance sources to CBB in African breeding programs. However, VAX 4 in combination with WILK 2, WILK 4, or XAN 159 may provide complete resistance for Africa. VAX 4 or WILK 4 also would provide resistance for North America and VAX 4 or 6 would give broad resistance for the Caribbean, South America, and Australia. The differences in pathogenicity between *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* strains were illustrated on VAX 1 and VAX 2, where almost all of the 14 *X. fuscans* subsp. *fuscans* strains caused a >10% disease severity, whereas some *X. campestris* pv. *phaseoli* strains caused no symptoms regardless of their geographic origin. This result agrees with previous reports such as Opio et al. (28), where *X. fuscans* subsp. *fuscans* was shown to be more virulent than *X. campestris* pv. *phaseoli*; however, in all the cited studies, fewer strains of *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* were tested. We concur with the Mkandawire et al. (25) suggestion that, in breeding for CBB resistance in Africa, African *X. fuscans* subsp. *fuscans* or South American *X. campestris* pv. *phaseoli* strains should be utilized in order to identify germplasm with the highest levels of resistance.

The clustering of all *X. campestris* pv. *phaseoli* and most *X. fuscans* subsp. *fuscans* strains was independent of their geographical origins, which might suggest that either the dissemination of the common blight pathogen occurred frequently or pathogenic evolution was a relatively recent event. An alternative explanation could be that neither *X. campestris* pv. *phaseoli* nor *X. fuscans* subsp. *fuscans* coevolved with their common bean host. However, East African *X. campestris* pv. *phaseoli* strains were reported to be more pathogenic on Andean bean genotypes (11). *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* strains from different geographical regions grouping together suggests similarities in pathogenicity among strains from different geographical regions. However, the lack of clustering based on geographical locations may not be surprising because the primary mode of transmission of this pathogen has been contaminated seed which probably has been distributed worldwide (31).

The 20 *X. campestris* pv. *phaseoli* strains that originated from the United States did not group together. A lack of clustering of *X. campestris* pv. *phaseoli* strains representing various regions also was reported when they were genotyped using RFLP (23). Moreover, all *X. fuscans* subsp. *fuscans* strains are scattered throughout the dendrogram within *X. campestris* pv. *phaseoli* strains, which indicates a similar pattern of pathogenicity between the two species on the common bean host genotypes. Thus, even though *X.*

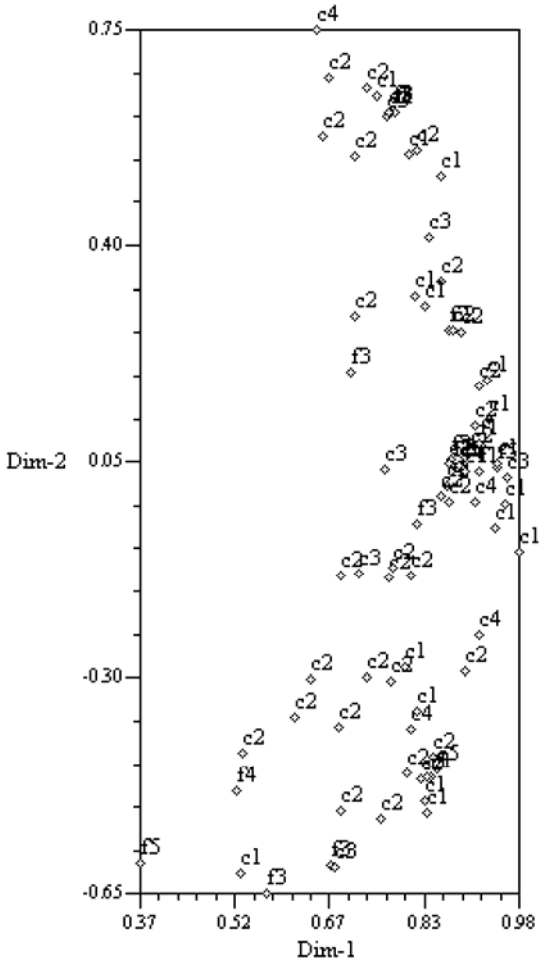

Fig. 2. Diagram showing the relationships among the 84 *Xanthomonas campestris phaseoli* and *X. fuscans* subsp. *fuscans* strains based on principal component analysis using disease reaction on 13 common bean genotypes. (c = *X. campestris* pv. *phaseoli*, f = *X. fuscans* subsp. *fuscans*, 1 = North America, 2 = South America, 3 = Africa, 4 = Australia and New Zealand, 5 = Europe, and 6 = Asia).

fuscans subsp. *fuscans* strains usually infect more bean hosts, there are *X. campestris* pv. *phaseoli* strains that are equally virulent on a given genotype. African strains of *X. campestris* pv. *phaseoli* with the highest virulence could indicate distinct pathogen genotypes in that region, as reported by Mkandawire et al. (25). RFLP analysis of genomic and plasmid DNA (17), DNA-DNA hybridization (19), and amplified DNA polymorphisms (4,43) provide evidence that *X. fuscans* subsp. *fuscans* strains are genetically distinct from *X. campestris* pv. *phaseoli*. However, genetic differences did not translate into distinct pathogenic differences, and the two species could not be definitively separated based on differential levels of pathogenicity with the common bean genotypes used in this study. The *X. fuscans* subsp. *fuscans* strains showed lower pathogenic variation and higher virulence than *X. campestris* pv. *phaseoli* strains, similar to other results (22,25). The greatest pathogenic variation was found within Caribbean and South and North American strains.

Nine *X. campestris* pv. *phaseoli* strains (123 Colombia, 95 Argentina, 95-04, 95-39, K3A, SX 124, G24, 84-5, and B-705) and five *X. fuscans* subsp. *fuscans* strains (1158A, 1158B, 0794, SX 119, and SX 127) were able to differentiate host resistance based on low versus high virulence reactions (Table 5) using a uniform inoculation method as proposed at the first international workshop on common bacterial blight (8). Our findings, where clear differential expression was shown with the two CBB pathogens on common bean, *P. vulgaris*, are similar to Mkandawire et al. (25) and Lopez et al. (22). Based on virulence levels, *X. campestris* pv. *phaseoli* and *X. fuscans* subsp. *fuscans* strains did not cluster according to their geographical origin or in genetic diversity from rep-polymerase chain reaction (PCR) and PCR-RFLP (23). However, Mahuku et al. (23) implied that lack of genetic geographical differentiation means that available host resistance genes likely will be effective in diverse geographical areas. Our virulence data indicates significant differences among geographical regions and effectiveness of resistance genes, although VAX 4 has broad resistance over all geographic regions.

ACKNOWLEDGMENTS

This manuscript is a contribution of the University of Nebraska Agricultural Research Division, Lincoln, Journal Series No. 14514. We acknowledge financial support for this research from the Bean/Cowpea CRSP (USAID Contract No. DAN-1310-G-SS-6608-00). Seed of the bean lines were kindly provided by D. P. Coyne, S. Singh, S. Beebe, J. C. Rosas, P. Miklas, J. R. Smith, J. Beaver, P. Gepts, E. Vallejos, and S. Park. Limited seed of the common bean genotypes and strains are available by request from J. Steadman and A. K. Vidaver, respectively, Department of Plant Pathology, University of Nebraska, Lincoln.

LITERATURE CITED

1. Adams, M. W., Kelly, J. D., and Saettler, A. W. 1988. A gene for resistance to common blight (*Xanthomonas campestris* pv. *phaseoli*.) Annu. Rep. Bean Improv. Coop. 31:73-74.
2. Aggour, A. R., Coyne, D.P., Vidaver, A.K., and Eskridge, K. M. 1989. Transmission of the common blight pathogen in bean seed. J. Am. Soc. Hortic. Sci. 114:1002-1008.
3. Andrus, C. F. 1948. A method of testing beans for resistance to bacterial blights. Phytopathology 38:757-759.
4. Birch, P. R. J., Hyman, L. J., Taylor, R., Opio, A. F., Bragard, C., and. Toth, I. K. 1997. RAPD PCR-based differentiation of *Xanthomonas campestris* pv. *phaseoli* and *Xanthomonas campestris* pv. *phaseoli* var. *fuscans*. Eur. J. Plant Pathol. 103:809-814.
5. Burkholder, W. H. 1921. The bacterial blight of the bean: a systemic disease. Phytopathology 11:61-69.
6. Chan, J. W. Y. F., and Goodwin, P. H. 1999. Differentiation of *Xanthomonas campestris* pv. *phaseoli* from *Xanthomonas campestris* pv. *phaseoli* var. *fuscans* by PFGE and RFLP. Eur. J. Plant Pathol. 105:867-878.
7. Claflin, L. E., Vidaver, A. K., and Sasser, M. 1987. MXP, a semi-selective medium for *Xanthomonas campestris* pv. *phaseoli*. Phytopathology 77:730-734.
8. Coyne, D. P., Navarrete-Maya, R., Pastor-Corrales, M. A., Vidaver, A. K., and Zapata, M. 1996. Proposed minimum standards for race designation of *Xanthomonas campestris* pv. *phaseoli* (Xcp) in *Phaseolus* species. Annu. Rep. Bean Improv. Coop. 39:288-289.
9. Coyne, D. P., and Schuster, M. L. 1973. *Phaseolus* germplasm tolerant to common blight bacterium *Xanthomonas phaseoli*. Plant Dis. Rep. 57:111-114.
10. Coyne, D. P., and Schuster, M. L. 1983. Genetics of and breeding for resistance to bacterial pathogens in vegetable crops. Hortic. Sci. 18:30-36.
11. Coyne, D. P., Steadman, J. R., Godoy-Lutz, G., Gilbertson, R., Arnaud-Santana, E., Beaver, J. S., and Myers, J. R. 2003. Contributions of the Bean/Cowpea CRSP to management of bean diseases. Field Crop Res. 82:155-168.
12. Drijfhout, E., and Blok, W. J. 1987. Inheritance of resistance to *Xanthomonas campestris* pv. *phaseoli* in tepary bean *(Phaseolus acutifolius)*. Euphytica 36:803-808.
13. Dursun, A., Coyne, D. P., Steadman, J. R., Godoy-Lutz, G., Gilbertson, R., Arnaud-Santana, E., Beaver, J. S., Myers, J. R., Mohamed, M. F., and Jung, G. 1996. Inheritance of resistance to common bacterial blight in tepary beans. Annu. Rep. Bean Improv. Coop. 39:162-163.
14. Eskridge, K. M., and Coyne, D. P. 1996. Estimating and testing hypotheses about the number of genes using inbred-backcross data. J. Hered. 87:410-412.
15. Freytag, G. F. 1989. Inheritance of resistance to three strains of common bacterial blight *(Xanthomonas campestris)* in the cultivated tepary bean *(Phaseolus acutifolius* var. *latifolius)*. Annu. Rep. Bean Improv. Coop. 32:101-102
16. Gabriel, D. W., Kingsley, M. T., Hunter, J. E., and Gottwald, T. 1989. Reinstatement of *Xanthomonas citri* (ex Hasse) and *X. phaseoli* (ex Smith) to species and reclassification of all *X. campestris* pv. *citri* strains. Int. J. Syst. Bacteriol. 39:14-22.
17. Gilbertson, R. L., Maxwell, D. P., Hagedorn, D. J., and Leong, S. A. 1989. Development and application of a plasmid DNA probe for detection of bacteria causing common bacterial blight of bean. Phytopathology 79:518-525.
18. Goodwin, P. H., and Sopher, C. R. 1994. Brown pigmentation of *Xanthomonas campestris* pv. *phaseoli* associated with homogentisic acid. Can. J. Microbiol. 40:28-34.
19. Hilderbrand, D. C., Palleroni, N. J., and Schroth, M. N. 1990. Deoxyribonucleic acid relatedness of 24 xanthomonad strains representing 23 *Xanthomonas campestris* pathovars and *Xanthomonas fragariae*. J. Appl. Bacteriol. 68:263-269.
20. Jara, C., Mahuku, G., Teran, H., and Singh, S. P. 1999. Reaction of common bean lines VAX 4, VAX 5, and VAX 6, derived from interspecific hybridization and gene pyramiding, to 20 *Xanthomonas campestris* pv. *phaseoli* isolates of different geographical origins. Annu. Rep. Bean Improv. Coop. 42:1-2.
21. Jung, G. W., Coyne, D. P., Skroch, P. W., Nienhuis, J., ArnaudSantana, E., Bokosi, J., Ariyarathne, H. M., Steadman, J. R., Beaver, J. S., and Kaeppler, S. M. 1996. Molecular markers associated with plant architecture and resistance to common blight, web blight, and rust in common beans. J. Am. Soc. Hortic. Sci. 121:794-803
22. Lopez, R., C. Asensio, and Gilbertson, J. L. 2006. Phenotypic and genetic diversity in strains of common blight bacteria. (*Xanthomonas campestris* pv. *phaseoli* and *X. campestris* pv. *phaseoli* var. *fuscans*) in a secondary center of diversity of the common bean host suggests multiple introduction events. Phytopathology 96:1204-1213.
23. Mahuku, G. S., Jara, C., Henriquez, A., Castellanos, G., and Cuasquer, J. 2006. Genotypic characterization of the common bean bacterial blight pathogens, *Xanthomonas axonopodis* pv. *phaseoli* and *Xanthomonas axonopodis* pv. *phaseoli* var. *fuscans* by rep-PCR and PCR-RFLP of the ribosomal genes. J. Phytopathol. 154:35-44.
24. Miklas, P. N., Beaver, J. S., Grafton, K. F., and Freytag, G. F. 1994. Registration of TARS VCI-4B multiple disease resistant dry bean germplasm. Crop Sci. 34:1415.
25. Mkandawire, A. B. C., Mabagala, R. B., Guzman, P., Gepts, P., and Gilbertson, R. L. 2004. Genetic diversity and pathogenic variation of common blight bacteria (*Xanthomonas campestris* pv. *phaseoli* and *X. campestris* pv. *phaseoli* var. *fuscans*) suggests pathogen coevolution with the common bean. Phytopathology 94:593-603.
26. Mohan, S. T., and Mohan, S. K. 1982. Breeding French bean (*Phaseolus vulgaris* L.) for resistance to common bean blight. Pages 53-56 in: Anais, 1 a reuniao nacional de pesquisa de feijao. Embrapa-Cnpaf, Goiania, Brazil.
27. Nodari, R. O., Tsai, S. M., Guzman, P., Gilbertson, R. L., and Gepts, P. 1993. Toward an integrated linkage map of common bean. 3. Mapping genetic factors controlling host-bacteria interactions. Genetics 134:341-350.
28. Opio, A. F., Allen, D. J., and Teri, J. M. 1996. Pathogenic variation in *Xanthomonas campestris* pv. *phaseoli*, the causal agent of common bacterial blight in *Phaseolus* beans. Plant Pathol. 45:1126-1133.
29. Park, S. J., and Dhanvantari, B. N. 1987. Transfer of common blight (*Xanthomonas campestris* pv. *phaseoli*) resistance from *Phaseolus coccineus* Lam. to *P. vulgaris* L. through interspecific hybridization. Can. J. Plant Sci. 67:685-695.
30. Rudolph, K. 1990. Toxins as taxonomic features. Pages 251-267 in: Methods in Phytobacteriology. Z. Klement, K. Rudolph, and D. C. Sands, eds. Academiai Kiado, Budapest.
31. Saettler, A. W. 1989. Common bacterial blight. Pages 261-283 in: Bean Production Problems in the Tropics. H. F. Schwartz and M. A. Pastor-Corrales eds. Center Internacional de Agricultura Tropical, Cali, Columbia.
32. Schaad, N. W., Postnikova, E., Lacy, G. H., Sechler, A., Agarkova, I., Stromberg, P. E., Stromberg, V. K., and Vidaver, A. K. 2005. Reclassification of *Xanthomonas campestris* pv.

citri (ex Hasse 1915) Dye 1978 forms A, B/C/D, and E as *X. smithii* subsp. *citri* (ex Hasse) sp. nov. nom. rev. comb. nov., *X. fuscans* subsp. *aurantifolii* (ex Gabriel 1989) sp. nov. nom. rev. comb. nov., and *X. alfalfae* subsp. *citrumelo* (ex Riker and Jones) Gabriel et al., 1989 sp. nov. nom.. rev. comb. nov.; *X. campestris* pv. *malvacearum* (ex Smith 1901) Dye 1978 as *X. smithii* subsp. *smithii* nov. comb. nov. nom. nov.; *X. campestris* pv. *alfalfae* (ex Riker and Jones, 1935) Dye 1978 as *X. alfalfae* subsp. *alfalfae*(ex Riker et al., 1935) sp. nov. nom. rev.; and "var. fuscans" of *X. campestris* pv. *phaseoli* (ex Smith, 1987) Dye 1978 as *X. fuscans* subsp. *fuscans* sp .nov. Syst. Appl. Microbiol. 28:494-518.

33. Schuster, M. L., and Coyne, D. P. 1981. Biology, epidemiology, genetics and breeding for resistance to bacterial pathogens of *Phaseolus vulgaris* L. Hortic. Rev. 3:28-58.
34. Schuster, M. L., Coyne, D. P., Behre, T., and Leyna, G. 1983. Sources of *Phaseolus* species resistance and leaf and pod differential reactions to common blight. HortScience 18:901-903.
35. Schuster, M. L., Coyne, D. P., and Hoff, B. 1973. Comparative virulence of *Xanthomonas phaseoli* strains from Uganda, Colombia, and Nebraska. Plant Dis. Rep. 57:74-75.
36. Singh, S. P., and Munoz, C. G. 1999. Resistance to common bacterial blight among *Phaseolus* species and common bean improvement. Crop Sci. 39:80-89.
37. Singh, S. P., Munoz, C. G., and Teran, H. 2001. Registration of common bacterial blight resistant dry bean germplasm VAX 1, VAX 3, and VAX 4. Crop Sci. 41:275-276.
38. Singh, S. P., Teran, H., Molina, A., and Gutierrez, J. A. 1992. Combining ability for seed yield and its components in common bean of Andean origin. Crop Sci. 32:81-84.
39. Steadman, J. R., Pastor-Corrales, M. A., and Beaver, J. S. 2002. An overview of the 3rd bean rust and 2nd bean common bacterial blight international workshops. Annu. Rep. Bean Improv. Coop. 45:120-124.
40. Toth, I. K., Hyman, L. J., Taylor, R., and Birch, P. R. J. 1998. PCR-based detection of *Xanthomonas campestris* pv. *phaseoli* var. *fuscans* in plant material and its differentiation from *X. c.* pv. *phaseoli*. J. Appl. Microbiol. 85:327-336.
41. Urrea, C. A., Miklas, P. N., and Beaver, J. S. 1999. Inheritance of resistance to common bacterial blight in four tepary bean lines. J. Am. Soc. Hortic. Sci. 124:24-27.
42. Wallen, V. R., and Jackson, H. R. 1975. Model for yield loss determination of bacterial blight of field beans utilizing aerial infrared photography combined with field plot studies. Phytopathology 65:942-948.
43. Xue, B. G., and Goodwin, P. H. 1994. Amplified DNA polymorphisms of putative 2-component regulatory genes of several *Xanthomonas campestris* pathovars. Can. J. Plant Pathol. 16:1-7.
44. Yoshii, K., Ge Galvez, E., and Alvarez, A. G. 1976. Estimation of yield losses in beans caused by common blight. Proc. Am. Phytopathol. Soc. 3:298.
45. Yoshii, K., Ge Galvez, E., and Alvarez, A. G. 1978. Screening bean germplasm for tolerance to common blight caused by *Xanthomonas phaseoli* and the importance of pathogenic variation to varietal improvement. Plant Dis. Rep. 62:343-347.
46. Zaiter, H. Z., Coyne, D. P., Vidaver, A. K., and Steadman, J. R. 1989. Differential reaction of tepary bean lines to *Xanthomonas campestris* pv. *phaseoli*. HortScience 24:134-137.
47. Zapata, M. 1997. Identification of *Xanthomonas campestris* pv. *phaseoli* races in Phaseolus *vulgaris* leaves. Agron. Mesoam. 8:44-52.
48. Zapata, M., Freytag, G. F., and Wilkinson, R. E. 1985. Evaluation for bacterial blight resistance in beans. Phytopathology 75:1032-1039.
49. Zapata, M., and Vidaver, A. K. 1987. Differentiation of *Xanthomonas campestris* pv. *phaseoli* into pathogenic races based on the tepary bean reactions. (Abstr.) Phytopathology 77:1709.