CHAPTER 5

SUSCEPTIBILITY OF SOUTH AFRICAN DRY BEAN CULTIVARS TO BACTERIAL DISEASES

ABSTRACT

Twenty-one locally grown commercial dry bean cultivars were evaluated at Potchefstroom during the 1998/1999 and 1999/2000 seasons for resistance to common bacterial blight, halo blight and bacterial brown spot. Results indicated that South African cultivars differed in their susceptibility to bacterial diseases. Cultivars Teebus, Cerillos, PAN 146 and PAN 159 were most susceptible to common bacterial blight with Monati and OPS-RS2 having low levels of resistance. Negative correlations between disease ratings and yields were obtained in the common bacterial blight trial. Levels of resistance to halo blight were observed with small seeded cultivars generally being more resistant than large seeded types. A negative correlation was obtained between halo blight rating and yield. Cultivars differed regarding susceptibility to bacterial brown spot with the majority having adequate resistance. Teebus, Cerillos, Bonus and PAN 159 were most susceptible, with Mkuzi exhibiting highest resistance. No correlation was obtained between disease rating and yield. Although a number of cultivars exhibited field resistance to halo blight and bacterial brown spot, all cultivars were more or less susceptible to common bacterial blight. Common bacterial blight can be considered the most important bean bacterial disease in South Africa. Improvement of common bacterial blight resistance in South African cultivars is necessary for yield stability.

INTRODUCTION

Dry beans (*Phaseolus vulgaris* L.) represent an important leguminous food crop grown in South Africa, with approximately 50 000 tons being produced annually by commercial and small scale farmers. Bacterial diseases, e.g. common bacterial blight (*Xanthomonas axonopodis* pv. *phaseoli*, Xap) (Smith) Vauterin *et al.*, halo blight (*Pseudomonas savastanoi* pv. *phaseolicola*, Psp) (Burkholder) Gardan *et al.* and bacterial brown spot (*Pseudomonas syringae* pv. *syringae*, Pss), van Hall, limit dry bean production in many international bean producing areas (CIAT 1985). Pathogens responsible are all seed-borne infecting beans at different stages of maturity. Their relative importance varies annually depending on biological and climatic factors and management practices.

Common bacterial blight (CBB) is widespread throughout the South African bean production areas (Fourie 2002). It can also be highly destructive during extended periods of warm, humid weather, resulting in yield and seed quality loss (Saettler 1991). Typical blight symptoms are visible during the crop's reproductive stage. Yield losses have been poorly documented, but vary from 22% to 45% (Wallen & Jackson 1975, Yoshii 1980).

Halo blight (HB) is restricted to cooler production areas at higher altitudes and typical symptoms are visible from seedling the stage to crop maturity. Serious yield losses have been observed, particularly where farmers grow their own seed for a number of seasons (D.Fourie: unpublished data). Yield losses of 43% have been obtained under experimental conditions (Allen *et al.* 1998). Pathogenic variation

within Psp isolates exist, with seven (races 1, 2, 4, 6, 7, 8 & 9) of the described nine races (Taylor *et al.* 1996) occurring in South Africa (Fourie 1998).

Bacterial brown spot (BBS), the most widespread bacterial disease in South Africa, occurs in all seed and commercial production areas (Fourie 2002). Sporadic losses occur in moderate to hot climatic areas, particularly where plants have been damaged by heavy rain or hail (Serfontein 1994). Yield reduction, as high as 55%, were reported (Serfontein 1994).

Bacterial bean pathogens are seed-borne and this is the primary inoculum source (Allen *et al.* 1998). Planting of pathogen-free seed is the most important primary control method (Gilbertson *et al.* 1990). Use of pathogen-free seed, however, does not guarantee disease control, as other inoculum sources exist (Allen *et al.* 1998). Additional cultural practices, such as removing, destroying or deep ploughing of debris, effective weed control, crop rotation and minimizing movement within fields when foliage is wet, may be also effective in controlling the disease (Allen *et al.* 1998, Schwartz & Otto 2000).

Copper based bactericides protect foliage against infestation and secondary pathogen spread (Oshima & Dickens 1971, Weller & Saettler 1976, Opio 1990, Schwartz *et al.* 1994). Efficacy of chemical control is limited (Allen *et al.* 1998) and resultant yield increases are minimal (Saettler 1989).

The most effective and economic bacterial control strategy in dry beans, is this use of cultivars with stable resistance (Rands & Brotherton 1925). The aim of the study was to determine susceptibility of local commercial cultivars to CBB, HB and BBS and thus to direct breeding strategies towards resistance against important bacterial diseases in South Africa.

MATERIAL AND METHODS

Twenty-one South African dry bean cultivars (Table 1) were evaluated for resistance to CBB, HB and BBS. Three field trials, one for each disease, were conducted at Potchefstroom during the 1998/1999 and 1999/2000 seasons. Cultivars were hand planted in 2 row plots, 5 m in length with 750 mm inter-row and 75 mm intra-row spacing. Trials were planted in a complete randomised block design with three replications, each surrounded by two border rows. Weed, insect and fungal control measures were applied, following standard agricultural practices.

Two Xap isolates (X6 and Xf105) were used, in a mixture to inoculate the common blight trial. A mixture of Psp isolates representing local races (races 1, 2, 6, 7, 8 & 9) was used to inoculate the halo blight trial. Race 4 isolates were not included as this race has only been identified locally from greenhouse grown seedlings. A highly aggressive Pss isolate (BV100) was used for the bacterial brown spot inoculum.

Inoculum was prepared from 48 h cultures grown on King's B medium (Psp and Pss) and yeast-extract-dextrose-calcium-carbonate agar (YDC) medium (Xap), respectively. Bacterial cells were suspended in tap water and adjusted to 10^8 CFU/ml water. Trials were irrigated prior to inoculation and repeated weekly to enhance disease development. Each trial was inoculated in the late afternoon using a motorized backpack sprayer at 21, 29 and 36 days after planting. First disease evaluations were done 10-14 days after the first inoculations on a 1-9 scale (Van Schoonhoven & Pastor-Corrales 1987) with 1 being resistant and 9 susceptible.

Evaluations were repeated at flowering and at full pod set. At maturity, two row plots of all cultivars were harvested manually and yield data recorded.

Data were analysed using a factorial analysis of variance (Statgraphics Plus 5.0) with disease ratings and yield as variables. Cofficients of linear correlation were used to determine the relationships between the measured variables.

RESULTS

Susceptibility of South African cultivars, to CBB, HB and BBS, are shown in Tables 2, 3 and 4, respectively. All cultivars screened were susceptible to CBB (Table 2). Cultivars, Teebus, Cerillos, PAN 146 and PAN 159 were susceptible differing from the other cultivars, with ratings of 7 and higher. Less disease developed on cultivars Monati and OPS-RS2 with mean ratings of 4.7 and 4.8, respectively. Small seeded cultivars were generally more susceptible to CBB than large seeded red speckled sugars. Lowest yields were recorded on Cerillos, and PAN 159, while OPS-RS3 was the highest yielding cultivar (Table 2).

Cultivars exhibited higher levels of resistance to HB than to CBB (Table 3). Teebus, PAN 150 and Mkuzi were the most resistant cultivars with PAN 182 most susceptible. Large seeded cultivars were more susceptible to HB than small seeded cultivars, with mean disease ratings averaging 4 and 5. Yields in the HB trial were generally higher than those in the CBB and BBS trials (Table 3). Lowest yielding cultivars were OPS-RS1 and PAN 159 while PAN 150 was the highest yielding cultivar. Yields of the HB trials differed significantly over the two seasons.

Cultivars differed in susceptibility to BBS (Table 4). Teebus, Cerillos, Bonus and PAN 159 were most susceptible, with Mkuzi exhibiting highest levels of resistance. The majority of cultivars had acceptable levels of resistance to BBS. Significant yield differences were obtained for cultivars in the BBS trial (Table 4). Kranskop was the lowest yielding cultivar with highest yields recorded for PAN 178. Significant differences were observed in disease rating and yield over both seasons.

DISCUSSION

Results indicated significant differences in susceptibility of South African cultivars to the economically important bacterial diseases. All cultivars were susceptible to CBB, with Teebus, Cerillos, PAN 146 and PAN 159 being most susceptible. Teebus is, currently, the only cultivar approved by the canning industry, with an acceptable canning quality. Improvement of resistance within this cultivar is extremely important.

Yields recorded for PAN 146 and PAN 159 were significantly lower than the majority of red speckled sugar cultivars. Yield reduction could be attributed to high susceptibility. Lowest yield was recorded in Cerillos, which was highly susceptible to CBB. High levels of susceptibility to CBB in Teebus, could have contributed to the reduction in yield. Negative correlations (P=-0.48) between disease ratings and yields indicate yield reduction due to CBB. No seasonal variation in disease rating and yields obtained was recorded indicating that CBB incidence and severity was not significantly influenced by the environmental conditions over the two seasons.

Acceptable levels of resistance to HB were identified in commercial cultivars. Large seeded cultivars were generally more susceptible than small seeded cultivars. Thus, attempts should be made to improve HB resistance in these cultivars.

Yields recorded in the HB trial were generally higher than those obtained in the CBB and BBS trials. A negative correlation (P=-0.56) existed between HB disease rating and yield. This disease could seriously affect yield under conducive conditions, particularly when plants are systemically infected (D. Fourie: unpublished data). Yields differed significantly over the two seasons, indicating that prevailing environmental conditions influenced yield.

Although cultivars differed significantly in their susceptibility to BBS, the majority of cultivars exhibited acceptable levels of resistance. Disease ratings and yield were, however, influenced by prevailing environmental conditions over the two seasons. Screening of cultivars for BBS resistance should, therefore, be conducted in multi-locational trials, over seasons. Although field resistance to BBS exists, this disease is the most widespread bean bacterial disease (Fourie 2002) and is a serious threat, particularly in the disease-free seed scheme. BBS is a relatively new disease in South Africa (Serfontein 1994) and studies on pathogenic variation and epidemiology of Pss need to be conducted. This could influence future screening for resistance. No significant correlation (P=-0.08) was, however, obtained between BBS rating and yield.

Although a number of cultivars exhibited field resistance to HB and BBS, all cultivars were moderately to highly susceptible to CBB. This disease is, therefore, considered the most important bean bacterial disease in South Africa. Improvement of CBB resistance in South African cultivars would largely contribute to obtain stable

yields. Improving CBB resistance in Teebus should be a priority because of its high commercial value.

REFERENCES

Allen, D.J., Buruchara, R.A. & Smithson, J.B. (1998) Diseases of common bean. In *The pathology of food and pasture legumes* (D.J. Allen & J.M. Lenné, eds) : 179-235. CAB International, Wallingford.

CIAT. (1985) Bean Programme Annual Report for 1985. Centro Internacional de Agricultura Tropical, Cali, Colombia.

Fourie, D. (1998) Characterization of halo blight races in South Africa. *Plant Disease* **82**: 307-310.

Fourie, D. (2002) Distribution and severity of bacterial diseases on dry beans (*Phaseolus vulgaris* L.) in South Africa. *Journal of Phytopathology* **150**: 220-226.

Gilbertson, R.L., Rand, R.E. & Hagedorn, D.J. (1990) Survival of *Xanthomonas campestris* pv. *phaseoli* and pectolytic strains of *X. campestris* in bean debris. *Plant Disease* **74**: 322-327.

Opio, A.F. (1990) Control of common bacterial blight of beans in Uganda. *Annual Report of the Bean Improvement Cooperative* **33**: 41-42.

Oshima, N. & Dickens, L.E. (1971) Effects of copper sprays on secondary spread of common bacterial blight of beans. *Plant Disease Reporter* **55**: 609-610.

Rands, R.D. & Brotherton, W. (1925) Bean varietal tests for disease resistance. *Journal of Agricultural Research* **31**: 110-154.

Saettler, A.W. (1989) Common bacterial blight. In *Bean Production Problems in the Tropics 2nd ed* (H.F. Schwartz & M.A. Pastor-Corrales, eds) : 261-283. CIAT, Cali, Colombia.

Saettler, A.W. (1991) Diseases caused by bacteria. In *Compendium of bean diseases* (R. Hall, ed) : 29-32. APS-Press, St. Paul, Minnesota.

Schwartz, H.F., Lienert, K. & Mcmillan, M.S. (1994) Timely and economical applications of pesticides to manage bean diseases. *Annual Report of the Bean Improvement Cooperative* **37**: 29-30.

Schwartz, H.F. & Otto, K.L. (2000) Enhanced bacterial disease management strategy. *Annual Report of the Bean Improvement Cooperative* **43**: 37-38.

Serfontein, J.J. (1994) Occurrence of bacterial brown spot of dry beans in the Transvaal province of South Africa. *Plant Pathology* **43**: 597-599.

Taylor, J.D., Teverson D.M., Allen, M.A., & Pastor-Corrales, M.A. (1996) Identification and origin of races of *Pseudomonas syringae* pv. *phaseolicola* from Africa and other bean growing areas. *Plant Pathology* **45**: 469-478.

Van Schoonhoven, A. & Pastor-Corrales, M.A. (1987) Standard system for the evaluation of bean germplasm. 53p. CIAT, Cali, Colombia.

Wallen, V.R. & 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.

Weller, D.M. & Saettler, A.W. (1976) Chemical control of common and fuscous bacterial blights in Michigan navy (pea) beans. *Plant Disease Reporter* **60**: 793-797.

Yoshii, K. (1980) Common and fuscous blights. In *Bean Production problems in the Tropics* (H.F. Schwartz & M.A. Pastor-Coralles, eds) : 157-172. CIAT, Cali, Colombia.

Table 1. Characteristics of 21 commercial South African dry bean cultivars screened for resistance to bacterial diseases

CV Name	Bean type	Growth habit*	Mean growing season (days)	Seed size (seeds 30g)
Teebus	Small white canning	I	92	127
Helderberg	Small white canning	II	99	180
OPS-KW1	Small white canning	II	96	156
PAN 182	Small white canning	II	90	183
PAN 185	Small white canning	II	96	183
Cerillos	Alubia	I	91	57
Kranskop	Red speckled sugar	II	97	63
OPS-RS1	Red speckled sugar	II	96	63
OPS-RS2	Red speckled sugar	I	100	61
OPS-RS3	Red speckled sugar	II	97	65
Jenny	Red speckled sugar	II	96	57
Bonus	Red speckled sugar	III	97	69
Monati	Red speckled sugar	II III	97	55
PAN 146	Red speckled sugar	I	86	70
PAN 148	Red speckled sugar	II	96	72
PAN 159	Red speckled sugar	I	85	74
PAN 178	Red speckled sugar	II	97	76
Stormberg	Red speckled sugar	III	97	70
Leeukop	Red speckled sugar	III	99	69
PAN 150	Carioca	II	95	123
Mkuzi	Carioca	II	96	143

* Type I: Determinate growth habit: flowers at end of branches stop stem growth
Type II: Intermediate growth habit: few short and upright branches, grow after flowering
Type III: Intermediate growth habit: long and low trailing branches

Table 2. Common bacterial blight reaction and yield of 21 South African dry bean cultivars in artificially inoculated field trials at Potchefstroom

Cultivar	Mean disease rating (1-9)		Yield kg.ha^{-1}	
Teebus	7.8	g	702	abcd
Helderberg	6.0	ef	645	abc
OPS-KW1	5.8	de	752	abcde
PAN 182	6.5	f	696	abcd
PAN 185	6.0	ef	983	defg
Cerillos	7.8	g	477	a
Kranskop	5.8	de	905	cdef
OPS-RS1	5.8	de	930	cdef
OPS-RS2	4.8	ab	1096	fg
OPS-RS3	5.3	bcd	1283	g
Jenny	5.2	abc	1009	defg
Bonus	5.7	cde	1077	efg
Monati	4.7	a	1000	defg
PAN 146	7.5	g	567	ab
PAN 148	5.2	abc	1001	defg
PAN 159	7.3	g	504	a
PAN 178	5.3	bcd	1053	efg
Stormberg	5.3	bcd	1080	fg
Leeukop	5.8	de	843	bcdef
PAN 150	5.8	de	1008	defg
Mkuzi	5.7	cde	1081	fg

Means followed by different letters differ significantly according to LSD (P=0.05)

Table 3. Halo blight reaction and yield of 21 South African dry bean cultivars in artificially inoculated field trials at Potchefstroom during the 1998/1999 and 1999/2000 seasons

Cultivar			Yield (kg.ha-1)			
	Mean disease rating (1-9)		1998/1999		1999/2000	
Teebus	3.0	a	2137	ef	3356	s
Helderberg	3.5	b	2703	mno	2729	no
OPS-KW1	3.2	ab	2137	ef	3103	r
PAN 182	5.3	f	1831	c	2031	de
PAN 185	4.0	c	2307	gh	3129	r
Cerillos	5.0	def	1933	cd	1956	cd
Kranskop	4.8	def	3031	qr	2636	lmn
OPS-RS1	5.0	def	1204	a	2836	op
OPS-RS2	4.7	def	2457	ik	2831	op
OPS-RS3	5.0	def	2275	fg	2347	ghi
Jenny	5.0	def	2556	klm	3103	r
Bonus	5.0	def	2723	no	2729	no
Monati	5.0	def	2627	lmn	2364	ghi
PAN 146	5.2	ef	1916	cd	1956	cd
PAN 148	5.0	def	1667	b	3636	t
PAN 159	4.8	def	1307	a	2249	fg
PAN 178	5.0	def	2516	kl	2943	pq
Stormberg	4.8	def	1884	cd	2617	lmn
Leeukop	5.0	def	1813	bc	2431	hik
PAN 150	3.0	a	4031	u	3049	qr
Mkuzi	3.0	a	3631	t	2756	no

Means followed by different letters differ significantly according to LSD (P=0.05)

Table 4. Bacterial brown spot reaction and yield of 21 South African dry bean cultivars in artificially inoculated field trials at Potchefstroom during the 1998/1999 and 1999/2000 seasons

Cultivar	Mean disease rating (1-9)				Yield (kg.ha^{-1})			
	1998/1999		1999/2000		1998/1999		1999/2000	
Teebus	6.7	l	6.0	j	840	fg	791	ef
Helderberg	3.0	c	3.0	c	929	h	1096	kl
OPS-KW1	3.0	c	3.0	c	577	ab	985	hi
PAN 182	2.7	b	3.0	c	947	hi	779	ef
PAN 185	2.7	b	3.0	c	1103	kl	767	ef
Cerillos	6.3	k	5.0	h	947	hi	1113	kl
Kranskop	4.3	g	3.0	c	543	a	1529	q
OPS-RS1	3.3	d	3.0	c	1231	mn	1291	no
OPS-RS2	2.3	a	3.0	c	631	bc	1369	op
OPS-RS3	4.0	f	3.0	c	680	cd	1359	op
Jenny	3.7	e	3.0	c	792	ef	1332	o
Bonus	6.0	j	5.0	h	920	gh	1104	kl
Monati	2.7	b	3.0	c	1076	jk	1333	o
PAN 146	4.0	f	3.0	c	1160	lm	611	ab
PAN 148	3.3	d	3.0	c	1217	mn	1724	r
PAN 159	6.0	j	5.3	i	991	hi	1168	lm
PAN 178	3.7	e	3.0	c	1587	q	2020	s
Stormberg	3.0	c	3.0	c	813	ef	1425	p
Leeukop	2.7	b	3.0	c	783	ef	1021	ij
PAN 150	2.7	b	3.0	c	1423	p	825	ef
Mkuzi	2.3	a	3.0	c	1209	m	745	de

Means followed by different letters differ significantly according to LSD (P=0.05)

CHAPTER 6

COMMON BACTERIAL BLIGHT: A DEVASTATING DISEASE OF DRY BEANS IN AFRICA

INTRODUCTION

Dry beans (*Phaseolus vulgaris* L.) are an important source of protein, B-complex vitamins and minerals (Paradez-López *et al.* 1986) and a staple food in the diet of many Latin American countries (De León *et al.* 1992). In central America, they provide between 20% and 30% of the dietary protein and are second only to maize as a staple food (Bressani *et al.* 1963). In Africa, beans are the second most important protein source after groundnuts (Technology Impact Report 1998) and production amounts to 2 049 000 t, of which 373 000 t is produced in Uganda, 332 000 t in Ethiopia, 309 000 t in Angola and 217 500 t in Tanzania. Mean annual production in South Africa over the last ten years is 58 000 t (Coetzee 2000).

Diseases are one of the most important factors reducing bean yields in most bean producing countries (Beebe & Pastor-Corrales 1991). Common bacterial blight (CBB), caused by *Xanthomonas axonopodis* pv. *phaseoli* (Smith) Vauterin, Hoste, Kosters & Swings and its fuscans variant, *Xanthomonas axonopodis* pv. *phaseoli* var. *fuscans* is a major disease limiting dry bean production in South Africa (Technology Impact Report 1998) and is considered one of the most important bean diseases worldwide (CIAT 1985). The disease is widespread throughout South African production areas (Fourie 2002) and is favoured by high temperatures and high relative

humidity (Sutton & Wallen 1970).

CBB was first reported in the USA by Beach in 1892. The same year Halsted described a bacterial disease, based on lesions on dry bean pods and seeds, and obtained similar lesions after inoculations (Zaumeyer & Thomas 1957). Smith (1897) first described the organism associated with this disease and named the bacterium *Bacillus phaseoli* E.F.Smith. After describing the cultural characteristics of the organism in 1901 he transferred it to the genus *Pseudomonas* (Zaumeyer & Thomas 1957). The name was again changed in 1905 to *Bacterium phaseoli* and later classified as *Phytomonas phaseoli* (E.F. Smith) by Bergey *et al.* (Zaumeyer & Thomas 1957). Dowson (1943) created the genus *Xanthomonas* and renamed the CBB bacterium, *Xanthomonas phaseoli*. The genus *Xanthomonas* was subdivided into five species and the causal organism renamed, *Xanthomonas campestris* pv. *phaseoli* (E.F Smith) Dye (Dye *et al.* 1980).

A similar bacterium to *Bacterium phaseoli* was isolated from bean plants, but differed in that it produced a brown diffusible pigment in culture media. The bacterium produced identical symptoms when inoculated onto bean plants and was named *Xanthomonas campestris* pv. *phaseoli* var. *fuscans* (Burkh.) Starr & Burkh. The disease was referred to as fuscous blight (Zaumeyer & Thomas 1957). Although this varietal form is often not recognized (Sutton & Wallen 1967, Leakey 1973), studies have revealed considerable genetic variation between these organisms (Birch *et al.* 1997, Toth *et al.* 1998) supporting proposals that they retain distinct taxonomic status (Chan & Goodwin 1999).

Based on DNA-DNA hybridization studies, Vauterin *et al.* (1995) suggested that the CBB organism and the fuscans variant should be reclassified as *Xanthomonas*

axonopodis pv. *phaseoli* and *X. axonopodis* pv. *phaseoli* var. *fuscans* respectively. Throughout this document, these will be referred to as Xap and Xapf. Schaad *et al.* (2000), however, rejected the transfer to *X. axonopodis* pv. *phaseoli* and recommended that it should be retained as a pathovar of *X. campestris*.

SYMPTOMOLOGY

CBB affects foliage, stems, pods and seeds of beans (Yoshii 1980). Leaf symptoms initially appear as water-soaked spots on the abaxial sides of leaves, which gradually enlarge, become flaccid and later turn brown and necrotic (Yoshii 1980, Saettler 1991). Lesions are often surrounded by a narrow zone of lemon-yellow tissue (Yoshii 1980, Saettler 1991). Lack of chlorotic zones on leaves of pompadour germplasm have, however, been reported (Beaver *et al.* 1992).

Bacteria enter leaves through natural openings such as stomata and hydathodes or through wounds (Yoshii 1980) from where they multiply and spread (Saettler 1991). Bacteria may also enter the stem and reach the vascular system of bean plants. The bacteria rapidly increase and fill xylem vessels that result in wilting of plants (Burkholder 1921). Burkholder (1921) also found bacteria in the root system of vascularly infected plants, however, no lesions have been observed below the soil surface. Systemically infected plants are in the minority (Burkholder 1921) and the pathogen does not systemically infect all *P. vulgaris* cultivars (Haas 1972).

Pod lesions are water-soaked spots which gradually enlarge, turn red-brown and are slightly sunken (Yoshii 1980, Saettler 1991). Lesions usually vary in size and shape, and are frequently covered with bacterial ooze (Saettler 1991). Infected seeds are

shrivelled and exhibit poor germination and vigour (Saettler 1991). Planting of infected seed may result in lesion development on seedling stems resulting in "snake head" symptoms, which occur (Burkholder 1921) when the plant growing tip is destroyed and only the cotyledons remain. Lesions on older stems are water-soaked spots that enlarge, discolour and may extend or girdle up the stem if infection occurs at a node. These lesions weaken stems which may break in windy conditions (Allen *et al.* 1998).

DISTRIBUTION AND ECONOMIC IMPORTANCE

CBB occurs in temperate, subtropical and tropical regions (Singh 1991) and causes severe damage under favourable environmental conditions. In Latin America it is particularly widespread in northwestern Argentina, south-central Brazil, Venezuela, central Cuba and coastal Mexico (Singh & Muñoz 1999). Although CBB was first considered a disease of minor importance in the United States of America, it was reported during 1919 to occur in all the important bean-producing states (Burkholder 1921).

In eastern and southern Africa CBB has been reported in 19 of the 20 bean producing countries (Allen 1995). It is thus considered one of five most important and widespread biotic constraints in dry bean production in sub-Saharan Africa (Gridley 1994). CBB was reported in South Africa prior to 1931 (Doidge & Bottomley 1931) while fuscous blight was first noted in 1962 (Boelema 1967). Both common and fuscous blight are widespread throughout the South African bean production area (Fourie 2002).

Other countries in which CBB occurs are Canada (Wallen *et al.* 1963, Wallen & Galway 1976, Huang *et al.* 1996), Australia (Wimalajeewa & Nancarrow 1978),

Germany (Tarigan & Rudolph 1996), France (J.J. Serfontein: personal communication), Hungary (Velich *et al.* 1991), Italy (Calzolari 1997), Bulgaria (Kiriakov *et al.* 1993), Dominican Republic (Mmbaga *et al.* 1992), India (Khandale & Kore 1979), Russia (Russkikh 1999) and New Zealand (Watson 1970). Distribution of the *X. axonopodis* pv. *phaseoli* var. *fuscans* (Xapf) seems to be more limited and does not occur in Costa Rica or Caribbean countries (CIAT 1992).

Although CBB is widely distributed, yield losses have not been well documented. Estimated losses of up to 38% have been reported in field trials in Ontario, Canada by Wallen & Jackson (1975). In Colombia, estimated yield losses of 22% and 45% have been documented after natural and artificial infections, respectively (Yoshii 1980). Moffet & Middleton (1979) obtained significant yield differences between inoculated and uninoculated plots of navy beans, despite the fact that CBB was observed in both plots. CBB in Uganda was associated with yield depression in beans and losses varied depending on susceptibility of varieties, developmental stage of crop at time of infection and climatic conditions during the season (Opio *et al.* 1992).

THE PATHOGEN

Cultural and morphological characteristics

Xap and Xapf can be easily isolated from CBB symptoms on leaves and pods using general isolation media (Schaad & Stall 1988). On media such as sucrose peptone agar (SPA), colonies are circular, smooth and mucoid with a yellow pigment referred to as xanthomonadin. Intensity of this yellow colour varies with medium used (Moffet &

Croft 1983). Corey & Starr (1957) described four colony types of Xap which had identical nutritional patterns and growth rates, but differed in amount of polysaccharide produced and ability to produce lesions. Differences in lesion development and morphology were correlated with polysaccharide production (Corey & Starr 1957).

Xanthomonas are non-sporing, gram-negative, aerobic rods, which are motile by means of a single polar flagellum (Moffet & Croft 1983). Characteristics are that they do not reduce nitrates, are catalase positive, asparagine is not used as a sole carbon and nitrogen source and they are weak producers of acids from carbohydrates (Schaad & Stall 1988). The organism also causes proteolysis of milk and starch hydrolysis (Saettler 1989) and does not induce a hypersensitive reaction on tobacco (Gilbertson et al. 1990).

Isolation media containing tyrosine differentiates between Xap and Xapf in that the latter produces a brown diffusible pigment (Basu & Wallen 1967). Goodwin & Sopher (1994) found this pigment to be produced due to secretion and subsequent oxidation of homogentisic acid rather than tyrosine activity.

Selective media are more effective for isolating specific bacteria from diseased material when selective at species level (Claflin, *et al.* 1987). A number of semi-selective media have been developed and improved to isolate Xap and Xapf (Kado & Heskett 1970, Schaad & White 1974, Trujillo & Saettler 1979, Claflin *et al.* 1987, Mabagala & Saettler 1992, Dhanvantari & Brown 1993, Jackson & Moser 1994, Gozczynska & Serfontein 1998).

Detection and identification

Apart from using selective media, techniques such as bacteriophage typing (Katznelson *et al.* 1954, Sutton & Wallen 1967), serology testing (Trujillo & Saettler 1979), host inoculation (Saettler 1971), ELISA (Wong 1991) and immunofluorescent staining (Malin *et al.* 1983), can be used to detect and identify Xap and Xapf. These techniques are time consuming and labourious. More sensitive, rapid and specific detection of the pathogen is often needed. This is particularly important when identification is complicated by epiphytic Xap strains (Gilbertson *et al.* 1989, Audey *et al.* 1994), that may confuse seed certification (Wong 1991, Audey *et al.* 1994).

Gilbertson *et al.* (1989) developed a plasmid DNA probe for rapid detection of pathogenic Xap strains which may be used in a breeding programme to select CBB resistant genotypes (Constabel *et al.* 1996). Based on this probe, another highly specific PCR probe, for Xap detection, was developed to detect as few as 10 colony forming units (CFU), using ethidium bromide-stained agarose gel (Audey *et al.* 1994). Audey *et al.* (1996) developed a rapid, sensitive PCR assay for detection of seedborne Xap in large bean seed samples containing as few as one infected in 10 000 healthy seeds. Birch *et al.* (1997) used RAPD-PCR to differentiate between Xap and Xapf. Toth *et al.* (1998) used primers which amplified a DNA fragment from all Xapf-isolates used, while no amplification products were obtained from Xap-isolates. These primers, therefore, provide a rapid, improved method to differentiate between these two variants.

Taxonomy and host range

The genus *Xanthomonas* consists of five species, each currently subdivided into a number of pathovars. These subdivisions remain controversial as pathovar demarcation

is often criticised as they are differentiated by inoculating host plants of that specific pathovar (Dye 1959, Lazo & Gabriel 1987), without determining the extent of host specificity (Starr 1983). Burkholder (1944) isolated *Xanthomonas* from diseased cowpeas, which were pathogenic to both beans and cowpeas. Infection was not obtained on cowpeas when inoculated with bean Xap isolates. It was suggested that the bacterium be named *X. vignicola* sp. nov. Vakili *et al.* (1975) confirmed these findings.

Schuster and Coyne (1977b) reported *X. vignicola* to be pathogenic on beans and cowpeas and that Xap, in some cases, showed a moderate degree of virulence when inoculated onto cowpeas, while *X. phaseoli* var. *sojense* was pathogenic on beans and cowpeas. Sabet (1959) found that Xap, *X. phaseoli* var. *sojense*, *X. alfalfa* and *X. vignicola* were all pathogenic on beans and suggested that all these be considered forms of Xap. Restriction fragment length polymorphisms (RFLP's) have been used to study the taxonomy of *X. campestris* (Gilbertson 1987, Lazo & Gabriel 1987, Lazo *et al.* 1987, Gabriel *et al.* 1989, Gilbertson *et al.* 1991) and results support pathovar classification.

The host range of Xap includes common bean (*Phaseolus vulgaris* L.), scarlet runner bean (*P. coccineus* L.), *P. lunatus*, urd bean (*Vigna mungo* (L.) Hepper), mung bean (*V. radiata* (L.) Wilczek var. *radiata*), tepary bean (*P. acutifolius* A. Gray var. *acutifolius*), *V. aconitifolia* (Jacq.) Maréchal, *V. angularis* (Willd.) Ohwi *et* Ohasi, *V. umbellata*, *Lablab purpureus* (L.) Sweet, *Strophostyles helvola* (L.) Elliot, soybean (*Glycine max* (L.) Merril), *Mucuna deeringiana* (Bort.) Merrill, *Lupinus polyphyllus* Lindl., cowpea (*V. unguiculata* (L.) Walp. ssp. *unguiculata*), *Macroptilum lathyroides*, *Pisum sativum*, *Strophostyles helvola* and *Mucuna deeringiana* (Saettler 1989, Allen et al.

1998).

Pathogenic and genetic diversity

Differences in virulence among pathogenic *Xanthomonas* bean strains have been confirmed in several reports (Yoshii *et al.* 1978, Schuster 1983). Small & Worley (1956) indicated that virulence differences of bacteria may be detected on culture media. Virulent Xap and *P. syringae* pv. *phaseoli* colonies were red in colour, while weakly virulent isolates were light in colour or remain white. Schuster & Coyne (1975), however, were unable to detect these visual differences. Colony types have also been used to differentiate degrees of virulence (Corey & Starr 1957, Jindal & Patel 1984).

Schuster & Coyne (1971) isolated Xap strains from Colombian seed more virulent than a Nebraskan isolate when inoculated onto three *Phaseolus* species. An equally virulent Xap strain was obtained from Uganda (Schuster *et al.* 1973). Ekpo & Saettler (1976) confirmed the observed variation in Xap and found that Xapf was more aggressive than Xap.

Several reports support the observed virulence differences between Xap and Xapf (Leakey 1973, Bozzano-Saguier & Rudolph 1994, Opio *et al.* 1996), and reports indicate that the Xapf pigment is not associated with pathogenicity (Gilbertson *et al.* 1991, Tarigan & Rudolph 1996) and considered of negligible pathological importance (Schuster & Coyne 1975). Pectolytic *Xanthomonas* associated with, but not pathogenic to beans can be distinguished from Xap and Xapf by RFLP's (Gilbertson *et al.* 1990).

Gilbertson *et al.* (1991) studied genetic diversity between Xap and Xapf, using DNA probes isolated from the genome of a single Xap strain. This was tested on a

diverse strain collection from various geographical locations. Genetic differences, based on RFLP patterns, indicated that two distinct bacterial groups exist. Similarities were revealed that were not observed when probes were hybridized to DNA from other *X. campestris* pathovars. This indicates sufficient similarities between Xap and Xapf, to consider Xapf a variety of Xap. Strains of Xap and Xapf from similar geographical locations had similar, but not identical RFLP patterns (Gilbertson *et al.* 1991). Similar results were obtained by CIAT (1992).

Although differences in isolate virulence are evident, physiological specialization on *P. vulgaris* is unknown. Zapata (1996) indicated that *P. vulgaris* genotypes exist which are useful in differentiation of Xap. Evidence suggests that interaction between Xap and *P. vulgaris* is quantitative (Opio *et al.* 1996). Host specialization of Xap based on reactions on *P. acutifolius* lines has been reported (Zapata & Vidaver 1987, Zaiter *et al.* 1989, Opio *et al.* 1996), with eight distinct physiological races identified, suggesting a gene-for-gene relationship. Different races could not be distinguished in studies conducted in South Africa (*vide* Chapter 4).

DISEASE DEVELOPMENT

CBB develops under warm, humid temperatures, causing greater damage to plants at 28°C than at lower temperatures (Saettler 1989). Bacteria enter leaves through stomata or wounds where they invade intercellular spaces causing gradual dissolution of the middle lamella (Zaumeyer & Thomas 1957). Bacteria enter stems through stomata of the hypocotyl and epicotyl, or vascular elements leading from leaves or infected cotyledons.

Plant wilting is caused by plugging of vessels or cell wall disintegration (Zaumeyer & Thomas 1957). Bacteria enter via pod sutures from the vascular system of the pedicle and pass into the funiculus through the raphe, into the seed coat where it remains until seed germination. Once the pathogen is in the seed area, the micropyle may also serve as a point of entry. Direct penetration through the seed coat has not been observed (Zaumeyer & Thomas 1957). Upon seed germination rifts are formed in the cotyledon epidermis and bacteria pass through these openings into intercellular spaces and may invade the entire cotyledon. Vascular bundles may also be invaded and hence plant wilting (Zaumeyer & Thomas 1957).

EPIDEMIOLOGY

Dissemination and survival

The most effective survival mechanism for Xap, is infected bean seed (Cafati & Saettler 1980b, Gilbertson *et al.* 1990, Arnaud-Santana *et al.* 1991, Opio *et al.*1993), within which bacteria may survive for up to thirty six years (Allen *et al.* 1998). Seed contamination may be internal or external (Saettler 1989, Allen *et al.* 1998) and even symptomless (Thomas & Graham 1952, Weller and Saettler 1980a), having serious implications for seed certification schemes.

Conflicting reports exist on the ability of Xap to survive in infested soil and plant debris (Schuster & Coyne 1976, Saettler *et al.* 1986, Gilbertson *et al.* 1990). Gilbertson *et al.* (1990) found Xap populations to overwinter in bean debris on no-tillage plots. Non-pathogenic pectolytic strains of *X. campestris* were also consistently isolated.

Experiments conducted in the Dominican Republic indicated that Xap survived up to 7 months on infected debris on the soil surface, but not in buried debris after 30 days (Arnaud-Santana *et al.* 1991). Xap survival studies conducted over ten years in Michigan indicated that infected crop debris is not the primary inoculum source for CBB (Saettler *et al.* 1986). Infected bean debris may be more important as an inoculum source in tropical and sub-tropical than in temperate areas (Gilbertson *et al.* 1990).

Survival of Xap is greater under dry conditions (Schuster & Coyne 1977a) as bacteria decline rapidly under moist conditions (Allen *et al.* 1998). Sabet & Ishag (1969) reported that Xap survived in press-dried bean leaves for more than 18 months in the laboratory, while Gilbertson *et al.* (1988) found Xap to remain viable in dry-leaf inoculum after 6 years. The longer survival under laboratory conditions as opposed to that in the field could be attributed the presence of antagonists, such as protozoa, in the soil (Habte & Alexander 1975).

Xap also survives on weeds and other host plants (Cafati & Saettler 1980c, Angeles-Ramos *et al.* 1991, Opio *et al.* 1995). Certain weed species may harbor the pathogen for up to 6 months (Opio *et al.* 1995). Angeles-Ramos *et al.* (1991) isolated epiphytic, pectolytic *Xanthomonads* from symptomless weeds where pathogenic strains were isolated from within infected fields. Epiphytic colonies survive on a wide range of plant species in families *Amaranthaceae*, *Commelinaceae*, *Compositae*, *Cruciferae*, *Gramineae*, *Oxalidaceae* and *Portulaceae* in addition to various legumes (Allen *et al.* 1998). Epiphytic Xap populations are important in the epidemiology of CBB on dry beans (Ishimaru *et al.* 1991) and are differentially affected in hosts of different genotypes (Cafati & Saettler 1980a).

The mechanisms of CBB dissemination over long distance (from one part of the

country to another), or plant to plant or field to field (Zaumeyer & Thomas 1957) vary. Seed transmission primarily disseminates CBB over international boundaries (Saettler & Perry 1972). Infections as low as 0,2% and 0,5% result in field epidemics under favourable conditions (Ednie & Needham 1973, Opio *et al.* 1993). Seedborne inoculum introduces the pathogen randomly to a field providing a number of primary infection foci. Spread from such foci is more effective than field margins (Mabagala 1997). Inoculum levels of 10^3-10^4 bacteria per seed were the minimum required to result in bacterial transfer from seed to seedling (Weller & Saettler 1980a). In Uganda even lower bacterial populations per seed (10^2 CFU/seed) were found to incite field infections (Opio *et al.* 1993).

Genotypes differ in their ability to transmit Xap from seed to seedlings (Schuster *et al.* 1979, CIAT 1994, Opio *et al.* 1994b, Mabagala 1997). Bacterial populations in resistant varieties are less than in susceptible ones, however, CBB may be transmitted through seed of resistant bean cultivars. Systemic invasion, however, does not occur in resistant varieties (Schuster *et al.* 1979).

Secondary spread of CBB depends on the number of infection foci, presence of vectors, crop growth stage, environmental conditions and cultural practices (Allen *et al.* 1998). Insects that disseminate Xap include grasshoppers (*Melanoplus* spp.), Mexican bean beetle (*Epilachna varivestis* Muts.), borers (*Diapreps abbrevialus* Boh.), *Ceratoma ruficornis* and white flies (*Bemisia tabaci*) (Zaumeyer & Thomas 1957, Sabet & Ishag 1969, Kaiser & Vakili 1978).

Wind-blown soil and debris not only disseminate bacterial plant pathogens, but also wound host plants allowing bacterial penetration (Claflin *et al.* 1973). CBB incidence in 2-week-old bean plants was 25 and 55% after exposure to soil blown 13,9

m/sec for 3 and 5 minutes respectively (Claflin *et al.* 1973). Wind disseminated Xap bacterial infections may be restricted by the pathogen's inability to survive in soil (Burke 1957). Rain, dew, hail and irrigation water are also important factors in disease dissemination (CIAT 1992) as is mechanical dissemination by means of implements, animals and humans.

Growth stage

Appearance of CBB in bean fields is closely related to plant developmental stage (Weller & Saettler 1980b). Although blight symptoms sometimes appear on seedlings, symptoms are generally not seen during the vegetative growth stage. Under field conditions, symptoms usually occur during the reproductive stage, initially observed on the lower, older leaves. Secondary pathogen spread occurs rapidly following primary infection.

Inoculation of plants under controlled conditions, indicated that leaf age affects Xap responses (Patel & Walker 1963). Susceptibility to Xap increases with leaf age (Goss 1940), however, Patel & Walker (1963) found younger leaves to be more susceptible. These plants were in the vegetative stage and infections did not simulate natural field infection.

Environmental influences

Temperature

CBB is generally regarded a high-temperature disease with greatest damage occurring at 28°C (Goss 1940, Patel & Walker 1963). Goss (1940) found that CBB symptoms appeared on inoculated plants within 6 days at 32°C, 10 days at 28°C, 14 days at 24°C and no visible symptoms after 17 days at 20°C and 16°C respectively. Symptoms were most severe at 28°C which agrees with Patel & Walker (1963) and Arnaud Santana *et al.* (1993a). *In vitro* bacterial growth is greatest at 28° and 32°C, gradually decreasing as temperatures are reduced with little growth at 16°C (Patel & Walker 1963).

Although classified a high-temperature disease, CBB infections may occur at relatively low temperatures but the incubation period is prolonged. This explains disease outbreaks under conditions generally unfavourable for infection (Goss 1940).

Humidity

High humidity is preferable for CBB development (Goss 1940, Sutton & Wallen 1970), however, CBB was also reported to spread rapidly during dry weather (Goss 1940). After artificial inoculation of bean plants, Goss (1940) found infections were more severe on plants kept at low-relative humidity. Plant pathogenic bacteria do not form spores, but may tolerate dessication and survive under extended dry conditions. Xap can survive for relatively long periods under varied environmental conditions, in an extracellular polysaccharide it produces in culture (Leach *et al.* 1957).

Photoperiod

Photoperiod affects expression of common bean reactions to Xap, which have serious

implications in resistance breeding. Disease reactions in growth chamber studies were more severe under short photoperiod and high temperatures than under long photoperiod and low temperatures (Arnaud Santana *et al.* 1993a). No significant interactions were detected. Short photoperiod increased disease severity in the field (Arnaud-Santana 1993a). Schuster *et al.* (1985) found lines adapted to temperate zones did not increase in susceptibility under short daylight, however, two tropical lines increased in susceptibility. Similarly Webster *et al.* (1983), found lines with moderate resistance in temperate zones were susceptible in the tropics.

DISEASE MANAGEMENT

CBB remains a major dry bean production constraint as it is difficult to control. An integrated disease management approach, including cultural practices, chemical sprays and resistant varieties, is needed to adequately control disease.

Cultural practices

Xap contaminated seed is considered the primary inoculum source. Planting of pathogen-free seed is the most important primary control method (Gilbertson *et al.* 1990). Disease-free seed is generally produced in areas where climatic conditions and rigid quarantine minimize infestation risk and has been successfully implemented in the USA, Canada (Copeland *et al.* 1975), Australia (Redden & Wong 1995) and South Africa (D. Fourie: unpublished data). Apart from field inspections, success of seed certification programmes depends on accurate pathogen detection in seed (Audey *et*

al. 1996). Several methods for bacterial detection in seed have been reported (Ednie & Needham 1973, Lachman & Schaad 1985, Venette *et al.* 1987, Aggour *et al.* 1988, Roth 1988, Redden & Wong 1995, Audey *et al.* 1996).

Use of disease-free seed does not guarantee disease control as other inoculum sources exist (Allen *et al.* 1998). Additional cultural practices such as removing, destroying or deep ploughing of debris, effective weed control, crop rotation and minimized movement in fields, especially when foliage is wet, may be effective (Allen *et al.* 1998, Schwartz & Otto 2000). Intercropping with maize decrease incidence and severity of CBB (Fininsa 1996). Crop rotation may be less effective if epiphytic bacteria survive on non-host rotation plants.

Chemical control

Copper based bactericides protect foliage against Xap and secondary pathogen spread and include copper sulphate, copper ammonium carbonate (Oshima & Dickens 1971), copper hydroxide, potassium (hydroxymethyl) methyldithiocarbamate (Weller & Saettler 1976), cupric carbonate, cupric sulphate (Opio 1990), and cupric hydroxide (Schwartz *et al.* 1994). Efficacy of CBB chemical control is limited (Allen *et al.* 1998) and resultant yield increases are minimal (Saettler 1989).

Early season disease detection can improve efficacy of bactericide applications (Schwartz *et al.* 1994). Schwartz *et al.* (1994) effectively controlled bacterial diseases by applying cupric hydroxide early in the season, thereby reducing bacterial populations before they establish within diseased tissue. An average of three applications provided average yield increases of between 5% and 9%.

No methods are available to eradicate internal seed populations, however, external contamination may be controlled by streptomycin sulphate and sodium hypochlorite (Liang *et al.* 1992). Liang *et al.* (1992) investigated the potential of osmotic conditioning in reducing internal Xap populations from seeds, using polyethylene glycol (PEG) and glycerol as antibiotic carriers. They found that tetracycline and chlorotetracycline in PEG solutions effectively reduced Xap, but were phytotoxic. PEG solutions containing streptomycin reduced, but did not eradicate internal bacterial populations from naturally infected seeds with few phytotoxic effects.

Streptomycin is rapidly absorbed into bean stems and translocated to leaves but there is no indication that antibiotics are translocated downward through stems, trifoliate leaves or peduncle into the pod (Mitchell *et al.* 1954). Antibiotics should not be applied to leaves as resistant mutants may develop (Saettler 1989), which is the major reason why antibiotic use is prohibited in South Africa. Development of resistance to chemicals (Romeiro *et al.* 1998), costs involved and efficacy limit use of chemical control which may be feasible under certain circumstances, such as seed production or as a component of an integrated control strategy (Allen *et al.* 1998).

Biological control

Resistance in susceptible plants induced by inoculation with avirulent isolates does exist. Bean leaf extract with avirulent isolates, evaluated at CIAT (1989) significantly reduced CBB under field conditions. Mabagala (1999) identified two *Bacillus* spp. and a *Pseudomonas fluorescens* isolate that exhibited *in vitro* and *in vivo* antagonism to Xap.

Genetic resistance

The most effective and economic bean CBB control strategy is use of genetic resistance (Rands & Brotherton 1925). CBB resistance breeding has been extensively researched (Beebe & Pastor-Corrales 1991). Rands & Brotherton (1925) identified lines with resistance to CBB. Subsequent efforts only yielded moderate levels of resistance (Yoshii *et al.* 1978) with no immunity in *P. vulgaris*. Wild populations of *P. vulgaris* also gave intermediate Xap resistance reactions (Navarrete-Maya & Acosta-Gallegos 1997). Higher levels of resistance were found in scarlet runner bean (*P. coccineus*) while highest levels were identified in tepary beans (*P. acutifolius*) (Singh & Muñoz 1999).

Honma (1956) made interspecific crosses between *P. vulgaris* and *P. acutifolius* to derive the resistant line GN #1 Nebr. sel. 27 (Coyne & Schuster 1974a). This line has been used many breeding programmes as a resistance source (Coyne & Schuster 1974a, Mohan & Mohan 1983) and resulted in development of resistant lines such as Jules (Coyne & Schuster 1970), Harris (Coyne *et al.* 1980), Tara, Valley (Coyne & Schuster 1974b) and Starlight (Coyne *et al.* 1991).

Another resistance source commonly used is PI 207262 which was developed in Colombia (Coyne & Schuster 1973). GN #1 Nebr. sel. 27 and PI 207262 have limited use as GN #1 Nebr. sel. 27 is susceptible to isolates from Colombia and Uganda (Schuster *et al.* 1973, Yoshii *et al.* 1978). Both lines and derivates are poorly adapted to tropical conditions (Webster *et al.* 1983). XAN 112, developed from crosses between Jules and PI 207262, had greater resistance and was better adapted to tropical conditions (Schuster & Coyne 1981, Silva *et al.* 1989). XAN 112 has been extensively evaluated as a resistance source in many countries (Argentina, Brazil, Colombia, Cuba,

Guatemala, France and USA) (CIAT 1987).

Germplasm is continuously screened at CIAT to find more suitable resistance sources. From approximately 15 000 lines screened, only a few lines with moderate resistance levels were identified (CIAT 1988). Hybridization between common (*P. vulgaris*) and tepary beans (*P. acutifolius*) was initiated at CIAT in 1989 where they used congruity backcrossing to overcome hybridization barriers such as genotype incompatibility, early embryo abortion, hybrid sterility and lower frequencies of hybridization (Mejía-Jiménez *et al.* 1994).

Near-immune lines (XAN 159, XAN 160, XAN 161 and OAC 88-1) were derived from crosses between *P. acutifolius* and *P. vulgaris* (Beebe & Pastor-Corrales 1991). Although resistance instabilities were reported in XAN 159 and its progeny (Beebe & Pastor-Corrales 1991), it is still widely used in resistance breeding programmes (Beebe & Pastor-Corrales 1991, Fourie & Herselman 2002, Park *et al.* 1998a, Mutlu *et al.* 1999, Singh & Muñoz 1999). Resistant varieties were also developed from interspecific crosses between *P. vulgaris* and *P. coccineus* (Freytag *et al.* 1982, Park & Dhanvantari, 1987, Miklas *et al.* 1994).

New resistant lines (Vax 1 Vax 2, Vax 3, Vax 4, Vax 5 and Vax 6) were recently developed at CIAT from interspecific hybridization of *P. vulgaris* and *P. acutifolius* and gene pyramiding (Singh & Muñoz 1999). These lines showed high resistance when tested against isolates from various geographical origins (Zapata *et al.* 1998, Jara *et al.* 1999). Vax 1 and Vax 2 were susceptible when evaluated in Uganda (R. Buruchara, CIAT: personal communication) and South Africa (D. Fourie: unpublished data). Resistance levels in Vax 3, Vax 4 and Vax 6 are as high as those found in *P. acutifolius* (Singh & Muñoz 1999). Substantial progress has been made through gene pyramiding.

Lines developed through pyramiding are often not of suitable commercial seed type and resistance must be transferred to cultivars of different market classes (Singh & Muñoz 1999). Sources of CBB resistance are shown in Table 1.

Adams *et al.* (1988) reported that a single major recessive gene confers resistance in a snap bean line, A-8-40. Eskridge & Coyne (1996) found CBB resistance in common bean to be controlled by one to five genes. Genetic markers indicated CBB resistance to be linked from two to six quantitative trait loci (QTL) (Nodari *et al.* 1993, Jung *et al.* 1996, Miklas *et al.* 1996, Jung *et al.* 1997, Park *et al.* 1998b, Tsai *et al.* 1998).

Depending on resistance sources used and evaluation methodology, one to three genes appear to confer resistance in *P. acutifolius* to CBB (McElroy 1985, Drijfhout & Blok 1987, Silva *et al.* 1989). Based on resistance of F1, segregation in F2 and reaction of F3 plants and lines, Drijfhout & Blok (1987) concluded that resistance was governed by a single dominant gene which was confirmed by Silva *et al.* (1989). McElroy (1985) indicated that resistance in XAN 159, XAN 160, and XAN 161 is controlled by one major and a few minor genes. A single QTL explained 62% of the total phenotypic variation in a line derived from XAN 159, confirming that one major gene control blight resistance (Yu *et al.* 1999).

Welsh & Grafton (1997) concluded that resistance derived from *P. coccineus* is conferred by one recessive gene. Range of reaction varied in susceptible plants indicating presence of minor genes modifying expression of CBB resistance. Yu *et al.* (1998), however, detected two resistance genes in the line XR-235-1-1which carries *P. coccineus*-derived CBB resistance.

Kolkman & Michaels (1994) found that PI 440 795 and PI 319 443 from which

XAN 159, XAN 161 and OAC 88-1 were derived, carried identical genes for CBB resistance. Segregation for susceptibility in F2 generations obtained from crosses between these lines suggested that more than one resistance gene is transferred from the tepary parent and these genes should be pyramided to confer durable resistance (Michaels 1992). Resistance in XAN 159 and OAC 88-1 is, however, linked to the same RAPD marker (Singh & Muñoz 1999).

CBB resistance is quantitatively inherited with dominance for susceptibility (Coyne *et al.* 1966, Coyne *et al.* 1973, Finke *et al.* 1986). Although gene action is primarily additive, dominance and epistatic effects have been observed (Beebe & Pastor-Corrales 1991). Low estimates of narrow sense heritability have been reported (Coyne & Schuster 1974a, Arnaud-Santana *et al.*1994). Selection for resistance in advanced lines should therefore be conducted in replicated trials under uniform disease pressure (Arnaud-Santana *et al.* 1994).

Differential reactions of pods and leaves to Xap have been reported (Coyne & Schuster 1974c, Valladarez-Sanchez *et al.* 1979, Schuster *et al.* 1983, Park & Dhanvantari 1987, Aggour *et al.* 1989). Pod susceptibility in large seeded bean types (Andean origin) seems to be more problematic (Beebe & Pastor-Corrales 1991). From 18 *P. vulgaris* germplasm lines evaluated against four Xap strains, XAN 159, BAC 6 and XAN 112 had the best combined leaf and pod resistance (Arnaud-Sanata *et al.* 1993b). Lack of association between leaf and pod disease reactions, indicates the importance of evaluating both reactions to develop a resistant plant.

Coyne & Schuster (1974c), found genes controlling late maturity and resistance to be linked in crosses with GN #1 Nebr. sel. 27, and that susceptibility increased with onset of plant maturity. Adams *et al.* (1988) indicated that reaction to Xap was not

associated with flower colour or with days to flower. Purple flower colour (V gene) and RAPD markers, however, have been reported to be associated with QTL affecting leaf and pod resistance in a bean cross (Jung *et al.* 1997, Mutlu *et al.* 1999, Park *et al.*, 1999).

Assessment of resistance

Different inoculation techniques described to evaluate CBB resistance include aspersion (inoculum sprayed under pressure onto leaves) and wounding of leaves using scissors, razor blades, needles, surgical blades etc. (Andrus 1948, Schuster 1955, Pastor-Corrales *et al.* 1981, Opio *et al.* 1994a). Vacuum infusion of bean seed with a bacterial suspension gave significantly higher incidence and severity scores than spraying of bacterial suspension on plants (Bett & Michaels 1992). Gilbertson *et al.* (1988) successfully used infected dry leaves as a source of inoculum and suggested it to be an effective inoculation method where laboratory facilities are limited.

Opio *et al.* (1994a) indicated that inoculum concentrations between 10^6 and 10^8 CFU/ml water, were adequate for disease development using several inoculation techniques. Aggour *et al.* (1988) found a significant interaction between methods of inoculation, inoculum concentration and genotype. Saettler (1977) indicated that bacterial concentrations ranging from 3-6x10^7 CFU/ml gave reactions that correlated with those in the field.

Mohamed *et al.* (1993) developed a detached leaf technique for bioassay of Xap reaction over a wide range of bean genotypes and environmental conditions. Navarrete-Maya *et al.* (1995) however, found that spray inoculation of detached leaves did not

produce reliable results. Detached pods (Ariyarathane *et al.* 1996) and detached seedling stem inoculation assays (Lienert & Schwartz 1994) can also be used effectively for evaluation of resistance against CBB.

Various rating scales have been developed for evaluating and quantifying disease reaction on leaves and pods (Saettler 1977, Yoshii *et al.* 1978, Valladarez-Sanchez *et al.* 1983, Park & Dhanvantari 1987, Van Schoonhoven & Pastor Corrales 1987, Mohamed *et al.* 1993, Arnaud-Santana *et al.* 1994). Rating scales should be standardized and utilized uniformly when comparing lines with CBB resistance (Saettler 1977).

Marker assisted selection (MAS)

Evaluation of field reactions is costly in terms of time and space. Molecular markers linked to resistance were developed for indirect selection in breeding for resistance (Bai *et al.* 1996, Beattie *et al.* 1998, Park *et al.* 1999, Yu *et al.* 1999). Yu *et al.* (1999) screened 138 F5 lines derived from HR67 (resistance derived from XAN 159), using a SCAR-marker and subsequently tested it for CBB resistance in the greenhouse. Based on marker information, 28 of the 138 lines had the SCAR band present and were predicted to be resistant. On comparing SCAR results with field inoculation test data, 23 of 28 plants gave a resistant phenotypic reaction (DSI<2.0) indicating an accuracy of 82%. Only 3.6% of the lines were mis-classified as resistant plants. Cost estimates further indicated that use of marker assisted selections costed approximately one third less than greenhouse testing (Yu *et al.* 1999).

Expression of QTL may differ over environments or populations in various crops

and only one QTL affecting resistance to Xap was consistently expressed in four common bean populations (Park *et al.* 1999). Marker-QTL associations need to be confirmed in a breeding programme, particularly for traits like CBB resistance that have complex inheritance patterns, low narrow-sense heritabilities and a number of genes involved (Park *et al.* 1999).

Pyramiding of resistance genes into a single cultivar is necessary to achieve stable resistance. Use of marker assisted selection can contribute considerably when pyramiding genes (Kelly & Miklas 1999, Sing & Muñoz 1999, Dursun *et al.* 1995). Independence of resistance genes to be combined, however, need to be closely monitored as many lines and cultivars have common sources of CBB resistance (Kelly & Miklas 1999). Use of SCAR-markers linked with three independent QTL derived from XAN 159 and GN #1 Nebr. sel. 27, has resulted in advanced cranberry, pinto and snap bean germplasm with combined resistance to CBB. MAS should therefore expedite improvement of blight resistance in other market classes of bean (Miklas *et al.* 2000).

CONCLUSION

Although CBB has been studied extensively, it continues to be a major constraint in dry bean production in many parts of the world. Many contradictory results have been reported and work confirming various aspects are required. Disease management is complicated by the pathogen being seed borne and that widely adapted sources of resistance are limited. Good progress, however, has been made recently to improve resistance to CBB by combining genes from different *Phaseolus* species into a common bean type. Lines obtained from gene pyramiding (i.e. Vax 3, Vax 4 and Vax 6) possess

levels of CBB resistance that are as high as those found in *P. acutifolius* accessions (Singh & Muñoz 1999). QTL mapping contributed significantly to understanding the genetic control of a trait as complex as CBB resistance. Continued efforts in finding new sources of resistance and improvement of current levels of resistance in cultivars are needed.

It is indicated in the review that a number of different rating scales are being used in disease assessment. An internationally accepted scale needs to be standardized to allow meaningful comparison of results over time and in different parts of the world.

Existence of Xap races remains controversial. Races have been identified in some bean growing areas. Pathogenic variation may have serious implications in development of blight resistant varieties. An attempt was made during the First International Workshop on CBB (Coyne *et al.* 1996) in which minimum standards for race designation were proposed. During the Second International Workshop on CBB held in South Africa in 2002, it was, however, decided that there is a greater need to have differentials in *P. vulgaris*. The investment in time and resources does not justify working with a tepary system and *P. vulgaris* does not appear to have that degree of specificity (Steadman et al. 2002).

CBB, however, can only be effectively managed if a comprehensive integrated management strategy is developed. Studies on epidemiology and control of this devastating disease have been well documented and these technologies need to be transferred to producers and resource poor farmers.

REFERENCES

Adams, M.W., Kelly, J.D. & Saettler, A.W. (1988) A gene for resistance to common blight (*Xanthomonas campestris* pv. *phaseoli*). *Annual Report of the Bean Improvement Cooperative* **31**: 73-74.

Aggour, A., Coyne, D.P. & Vidaver, A.K. (1988) A new technique for detecting internal infection of bean (*Phaseolus vulgaris*) seed by *Xanthomonas campestris* pv. *phaseoli*. *Annual Report of the Bean Improvement Cooperative* **31**: 132-133.

Aggour, A.R., Coyne, D.P. & Vidaver, A.K. (1989) Comparison of leaf and pod disease reactions of beans (*Phaseolus vulgaris* L.) inoculated by different methods with strains of *Xanthomonas campestris* pv. *phaseoli* (Smith) Dye. *Euphytica* **43**: 143-152.

Allen, D.J. (1995) An annotated list of diseases, pathogens and associated fungi of the common bean (*Phaseolus vulgaris*) in Eastern and Southern Africa. *Phytopathological Papers* **34**. CAB International/Centro Internacional de Agricultura Tropical, Cali, Colombia.

Allen, D.J., Buruchara, R.A. & Smithson, J.B. (1998) Diseases of common bean. In *The pathology of food and pasture legumes* (D.J. Allen & J.M. Lenné, eds) : 179-235. CAB International, Wallingford.

Andrus, C.F. (1948) A method of testing beans for resistance to bacterial blights. *Phytopathology* **38**: 757-759.

Angeles-Ramos, R., Vidaver, A.K. & Flynn, P. (1991) Characterization of epiphytic *Xanthomonas campestris* pv. *phaseoli* and pectolytic xanthomonads recovered from symptomless weeds in the Dominican Republic. *Phytopathology* **81**: 677-681.

Ariyarathne, H.M., Coyne, D.P., Vidaver, A.K. & Eskridge, K.M. (1996) Effect of prior

inoculation of leaves of dry beans with the common blight pathogen on the disease reaction of subsequently inoculated leaves and pods and the reaction of detached versus attached pods. *Annual Report of the Bean Improvement Cooperative* **39**: 124-125.

Arnaud-Santana, E., Pena-Matos, E., Coyne, D.P. & Vidaver A.K. (1991) Longevity of *Xanthomonas campestris* pv. *phaseoli* in naturally infested dry bean (*Phaseolus vulgaris*) debris. *Plant Disease* **75**: 952-953.

Arnaud-Santana, E., Coyne, D.P., Beaver, J.S. & Zaiter, H.Z. (1993a) Effect of photo period and temperature on common blight disease of common beans (*Phaseolus vulgaris* L.). *Euphytica* **66**: 211-216.

Arnaud-Santana, E., Mmbaga, M.T., Coyne, D.P. & Steadman, J.R. (1993b) Sources of resistance to common bacterial blight and rust in elite *Phaseolus vulgaris* L. germplasm. *Horticultural Science* **28**: 644-646.

Arnaud-Santana, E., Coyne, D.P., Steadman, J.R., Eskridge, K.M. and Beaver, J.S. (1994) Heritabilities of seed transmission, leaf and pod reactions to common blight, leaf reaction to web blight and plant architecture and their associations in dry beans. *Annual Report of the Bean Improvement Cooperative* **37**:46-47.

Audey, P., Laroche, A., Saindon, G., Huang, H.C. & Gilbertson, R.L. (1994) Detection of the bean common blight bacteria *Xanthomonas campestris* pv. *phaseoli* and *X. c. phaseoli* var. *fuscans* using the polymerase chain reaction. *Phytopathology* **84**: 1185-1192.

Audey, P., Braat, C.E., Saindon, G., Huang, H.C. & Laroche, A. (1996) A rapid and sensitive PCR-based assay for concurrent detection of bacteria causing common and halo blights in bean seed. *Phytopathology* **86**: 361-366.

Bai, Y., Michaels, T.E. & Pauls, K.P. (1996) Identification of RAPD markers linked to bacterial blight resistance genes in *Phaseolus vulgaris* L. *Annual Report of the Bean Improvement Cooperative* **39**: 164-165.

Basu, P.K. & Wallen V.R. (1967) Factors affecting virulence and pigment production of *Xanthomonas phaseoli* var. *fuscans*. *Canadian Journal of Botany* **45**: 2367-2374.

Beattie, A., Michaels, T.E. & Pauls, K.P. (1998) An efficient, reliable method to screen for common bacterial blight (CBB) resistance in *Phaseolus vulgaris* L. *Annual Report of the Bean Improvement Cooperative* **41**: 53-54.

Beaver, J.S., Steadman, J.R. & Coyne, D.P. (1992) Atypical symptom expression of common bacterial blight in tropical Pompadour germplasm. *Annual Report of the Bean Improvement Cooperative* **35**: 110.

Beebe, S.E. & Pastor-Corrales, M.A. (1991) Breeding for disease resistance. In *Common Beans, Research for Crop Improvement* (A. Van Schoonhoven & O. Voysest, eds) : 561-610. CAB International, Wallingford.

Bett, K.E. & Michaels, T.E. (1992) Effectiveness of field inoculation procedures when evaluating common blight resistance in navy beans. *Annual Report of the Bean Improvement Cooperative* **35**: 81-82.

Birch, P.J., Hyman, L.J., Taylor, R., Opio, A.F., Bragard, C. & Toth, I.K. (1997) RAPD-PCR-based differentiation of *Xanthomonas campestris* pv. *phaseoli* and *Xanthomonas campestris* pv. *phaseoli* var. *fuscans*. *European Journal of Plant Pathology* **103**: 809-814.

Boelema, B.H. (1967) Fuscous blight of beans in South Africa. *South African Journal of Agricultural Science* **10**: 1059-1063.

Bozzano-Saguier, G. & Rudolph, K. (1994) Differential reactions of bush bean cultivars towards common and fuscous blight (*Xanthomonas campestris* pv. *phaseoli* and *X. c. phaseoli* var. *fuscans*). *Annual Report of the Bean Improvement Cooperative* **37**: 227-228.

Bressani, R., Elías, L.G. & Valente, A-T. (1963) Effect of cooking and of amino acid supplementation on the nutritive value of black beans (*Phaseolus vulgaris* L.). *Brittish Journal of Nutrition* **17**: 69-78.

Burke, D.W. (1957) Incidence of bacterial pathogens in dry beans in irrigated districts of Nebraska, Wyoming and Colorado in 1954 and 1955. *Plant Disease Reporter* **41**: 488-490.

Burkholder, W.H. (1921) The bacterial blight of the bean: a systemic disease. *Phytopathology* **11**: 61-69.

Burkholder, W.H. (1944) *Xanthomonas vignicola* sp. nov., pathogenic on cowpeas and beans. *Phytopathology* **34**: 430-432.

Cafati, C.R. & Saettler, A.W. (1980a) Effect of host on multiplication and distribution of bean common blight bacteria. *Phytopathology* **70**: 675-679.

Cafati, C.R. & Saettler, A.W. (1980b) Transmission of *Xanthomonas phaseoli* in seed of resistant and susceptible *Phaseolus* genotypes. *Phytopathology* **70**: 638-640.

Cafati, C.R. & Saettler, A.W. (1980c) Role of nonhost species as alternate inoculum sources of *Xanthomonas phaseoli*. *Plant Disease* **64**: 194-196.

Calzolari, A. (1997) Halo and common spot of bean. *Informatore Agriario* **53**: 66-67.

Chan, J.W.Y.F. & Goodwin, P.H (1999) Differentiation of *Xanthomonas campestris* pv. *phaseoli* from *Xanthomonas campestris* pv. *phaseoli* var. *fuscans* by PAGE and RFLP. *European Journal of Plant Pathology* **105**: 867-878.

CIAT. (1983) Bean Programme Annual Report for 1988. Centro Internacional de Agricultura Tropical, Cali, Colombia.

CIAT. (1984) Bean Programme Annual Report for 1988. Centro Internacional de Agricultura Tropical, Cali, Colombia.

CIAT. (1985) Bean Programme Annual Report for 1985. Centro Internacional de Agricultura Tropical, Cali, Colombia.

CIAT. (1987) Bean Programme Annual Report for 1987. Centro Internacional de Agricultura Tropical, Cali, Colombia.

CIAT. (1988) Bean Programme Annual Report for 1988. Centro Internacional de Agricultura Tropical, Cali, Colombia.

CIAT. (1989) Bean Programme Annual Report for 1989. Centro Internacional de Agricultura Tropical, Cali, Colombia.

CIAT. (1992) Bean Programme Annual Report for 1992. Centro Internacional de Agricultura Tropical, Cali, Colombia.

CIAT. (1994) Bean Programme Annual Report for 1994. Centro Internacional de Agricultura Tropical, Cali, Colombia.

CIAT. (1997) Bean Programme Annual Report for 1997. Centro Internacional de Agricultura Tropical, Cali, Colombia.

Claflin, L.E., Vidaver, A.K. & Sasser, M. (1987) MXP, a semi-selective medium for *Xanthomonas campestris* pv. *phaseoli*. *Phytopathology* **77**: 730-734.

Claflin, L.E., Stuteville, D.L. & Armbrust, D.V. (1973) Wind-blown soil in the epidemiology of bacterial leaf spot of alfalfa and common blight of bean. *Phytopathology* **63**: 1417-1419.

Coetzee, C. (2000) Droëboneproduksies, die verlede, die hede en 'n scenario vir 2001.

SA Droëbone/Dry beans **16**: 2-3.

Constabel, E.C., Michaels, T.E., Goodwin, P.H., Mayer, J.E. & Pastor-Corrales, M.A. (1996) Evaluation of a DNA probe for the quantitative detection of common bean and its application in a breeding program. *Euphytica* **90**: 129-135.

Copeland, L.O., Adams, M.W. & Bell, D.C. (1975) An improved seed programme for maintaining disease-free seed of field beans (*Phaseolus vulgaris*). *Seed Science and Technology* **3**: 719-724.

Corey, R.R. & Starr, M.P. (1957) Colony types of *Xanthomonas phaseoli*. *Journal of Bacteriology* **74**: 137-140.

Coyne, D.P., Schuster, M.L. & Shaughnessy, L. (1966) Inheritance of reaction to halo blight and common blight bacteria in a *Phaseolus vulgaris* variety cross. *Plant Disease Reporter* **50**: 29-32.

Coyne, D.P. & Schuster, M.L. (1970) 'Jules', a great northern dry bean variety tolerant to common blight bacterium (*Xanthomonas phaseoli*). *Plant Disease Reporter* **54**: 557-559.

Coyne, D.P. & Schuster, M.L. (1973) *Phaseolus* germplasm tolerant to common blight bacterium (*Xanthomonas phaseoli*). *Plant Disease Reporter* **57**: 111-114.

Coyne, D.P. & Schuster, M.L. (1974a) Breeding and genetic studies of tolerance to several bean (*Phaseolus vulgaris* L.) bacterial pathogens. *Euphytica* **23**: 651-656.

Coyne, D.P. & Schuster, M.L. (1974b) 'Great Northern Valley' dry bean. *Horticultural Science* **9**: 482.

Coyne, D.P. & Schuster, M.L. (1974c) Differential reaction of pods and foliage of beans (*Phaseolus vulgaris*) to *Xanthomonas phaseoli*. *Plant Disease Reporter* **58**: 278-

282.

Coyne, D.P., Nuland, D.S., Schuster, M.L. & Anderson, F.N. (1980) 'Great Northern Harris' dry bean. *Horticultural Science* **15**: 531.

Coyne, D.P., Steadman, J.R., Lindgren, D.T. & Nuland, D.S. (1991) 'Starlight' - Great Northern dry bean. *Horticultural Science* **26**: 441-442.

Coyne, D.P., Navarrete-Maya, R., Pastor-Corrales, M.A., Vidaver, A.K. & Zapata, M. (1996) Proposed minimum standards for race designation of Xanthomonas campestris pv. phaseoli (Xcp) in Phaseolus species. *Annual Report of the Bean Improvement Cooperative* **39**: 288-289.

De León, L.F., Elías, L.G. & Bressani, R. (1992) Effect of salt solutions on the cooking time, nutritional and sensory characteristics of common bean. *Food Research International* **25**: 131-136.

Dhanvantari, B.N. & Brown, R.J. (1993) YSSM-XP Medium for *Xanthomonas campestris* pv. *phaseoli*. *Canadian Journal of Plant Pathology* **15**: 168.

Doidge, E.M. & Bottomley, A.M. (1931) A revised list of plant diseases occurring in South Africa. *Botanical Survey of South Africa, Memoir* **11**: 78p.

Dowson, W.J. (1943) On the generic names *Pseudomonas*, *Xanthomonas* and *Bacterium* for certain bacterial plant pathogens. *Transactions of the British Mycological Society* **26**: 4-14.

Drijfhout, E. & Blok, W.J. (1987) Inheritance of resistance to *Xanthomonas campestris* pv. *phaseoli* in tepary bean (*Phaseolus acutifolius*). *Euphytica*, **36**: 803-808.

Dursun, A., Coyne, D.P., Mohamed, M.F. & Jung, G. (1995) Inheritance of resistance to common bacterial blight in tepary bean. *Annual Report of the Bean Improvement Cooperative* **38**: 162-163.

Dye, D.W., Bradbury, J.F., Goto, M., Hayward, A.C., Lelliot, R.A. & Schroth, M.N. (1980) International standards for naming pathovars of phytopathogenic bacteria and a list of pathovar names and pathotypes. *Review of Plant Pathology* **59**: 153-168.

Dye, D.W. (1959) The Genus *Xanthomonas*. *Commonwealth Phytopathological News* **5**: 1-3.

Ednie, A.B. & Needham, S.M. (1973) Laboratory test for internally-borne *Xanthomonas phaseoli* and *Xanthomonas phaseoli* var. *fuscans* in field bean (*Phaseolus vulgaris* L.) seed. *Proceedings of the Association of Official Seed Analysts* **63**: 76-82.

Ekpo, E.J.A. & Saettler, A.W. (1976) Pathogenic variation in *Xanthomonas phaseoli* and *X. phaseoli* var. *fuscans*. *Plant Disease Reporter* **60**: 80-83.

Eskridge, K.M. & Coyne, D.P. (1996) Estimation and testing hypothesis about the number of genes in using inbred-backcross data. *Journal of Hereditary* **87**: 410-412.

Fininsa, C. (1996) Effect of intercropping bean with maize on bean bacterial blight and rust diseases. *International Journal of Pest Management* **42**: 51-54.

Finke, M.L., Coyne, D.P. & Steadman, J.R. (1986) The inheritance and association of resistance to rust, common bacterial blight, plant habit and foliar abnormalities in *Phaseolus vulgaris* L. *Euphytica* **35**: 969-982.

Fourie, D. (2002) Distribution and severity of bacterial diseases on dry beans (*Phaseolus vulgaris* L.) in South Africa. *Journal of Phytopathology* **150**: 220-226.

Fourie, D. & Herselman, L. (2002) Breeding for common blight resistance in dry beans in South Africa. *Annual Report of the Bean Improvement Cooperative* **45**: 50-51.

Freytag, G.F., Bassett, M.J. & Zapata, M. (1982) Registration of XR-235-1-1 bean germplasm. *Crop Science* **22**: 1268-1269.

Gabriel, D.W., Kingsley, M.T., Hunter, J.E. & 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. *International Journal of Systematic Bacteriology* **39**: 14-22.

Gilbertson, R.L., Leong, S.A., Hagedorn, D.J. & Maxwell, D.P. (1987) Molecular epidemiology of *Xanthomonas campestris* pv. *phaseoli* and *Xanthomonas campestris* pv. *phaseoli* var. *fuscans*. *Phytopathology* **77**: 1718.

Gilbertson, R.L., Rand, R.E., Carlson, E. & Hagedorn, D.J. (1988) The use of dry-leaf inoculum for establishment of common bacterial blight of bean. *Plant Disease* **72**: 385-389.

Gilbertson, R.L., Maxwell, D.P., Hagedorn, D.J. & 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.

Gilbertson, R.L., Rand, R.E. & Hagedorn, D.J. (1990) Survival of *Xanthomonas campestris* pv. *phaseoli* and pectolytic strains of *X. campestris* in bean debris. *Plant Disease* **74**: 322-327.

Gilbertson, R.L., Otoya, M.M., Pastor-Corrales, M.A. & Maxwell, D.P. (1991) Genetic diversity in common blight bacteria is revealed by cloned repetitive DNA sequences. *Annual report of the Bean Improvement Cooperative* **34**: 37-38.

Goodwin, P.H. & Sopher, C.R. (1994) Brown pigmentation of *Xanthomonas campestris* pv. *phaseoli* associated with homogentisic acid. *Canadian Journal of Microbiology* **40**: 28-34.

Goss, R.W. (1940) The relation of temperature to common and halo blight of beans. *Phytopathology* **30**: 258-264.

Goszczynska, T. & Serfontein, J.J. (1998) Milk-Tween agar, a semi-selective medium for isolation and differentiation of *Pseudomonas syringae* pv. *syringae*, *Pseudomonas syringae* pv. *phaseolicola* and *Xanthomonas axonopodis* pv. *phaseoli*. *Journal of Microbiological Methods* **32**: 65.

Gridley, H.E. (1994) Bean production constraints in Africa with special reference to breeding for resistance to bean common mosaic virus in Uganda. In *Proceedings of a Pan-African working group meeting on bacterial and virus diseases of common bean. CIAT African Workshop Series* (D.J. Allen & R.A. Buruchara, eds) **34**: 33-39. Kampala, Uganda.

Haas, J.H. (1972) *Xanthomonas phaseoli* nonsystemic in some *Phaseolus vulgaris* cultivars. *Phytopathology* **62**: 761. (Abstract).

Habte, M. & Alexander, M. (1975) Protozoa as agent responsible for the decline of *Xanthomonas campestris* in soil. *Applied Microbiology* **29**: 159-164.

Honma, S. (1956) A bean interspecific hybrid. *Journal of Heredity* **47**: 217-220.

Huang, H.C., Saindon, R.S. Erickson, R.S. & Ma, P. (1996) Survey of diseases of dry bean in southern Alberta in 1995. *Canadian Plant Disease Survey* **76**: 93-94.

Ishimaru, C., Eskridge, K.M. & Vidaver, A.K. (1991) Distribution analysis of naturally occurring epiphytic populations of *Xanthomonas campestris* pv. *phaseoli* on dry beans. *Phytopathology* **81**: 262-268.

Jackson, L.E. & Moser, P.E. (1994) Development of a medium for isolation and differentiation of three bacterial plant pathogens. *Bean Improvement Cooperative* **37**: 23-24.

Jara, C., Mahuku, G., Terán, H. & 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. *Annual Report of the Bean Improvement Cooperative* **42**: 1-2.

Jindal, J.K. & Patel, P.N. (1984) Variability in Xanthomonads in grain legumes. IV. Variations in bacteriological properties of 83 isolates and pathogenic behaviour of cultural variants. *Phytopathology* **110**: 63-68.

Jung, G., Coyne, D.P., Scroch, P.W., Nienhuis, J., Arnaud-Santana, E., Bokosi, J., Ariyarathne, H.M., Steadman, J.R., Beaver, J.S. & Kaeppler, S.M. (1996) Molecular markers associated with plant architecture and resistance to common blight, web blight, and rust in common beans. *Journal of the American Society for Horticultural Science* **121**: 794-803.

Jung, G., Skroch, P.W., Coyne, D.P., Nienhuis, J., Arnaud-Santana, E., Ariyarathne, H.M., Kaeppler, S.M. & Bassett, M.J. (1997) Molecular- marker-based genetic analysis of tepary bean-derived common bacterial blight resistance in different developmental stages of common bean. *Journal of the American Society for Horticultural Science* **122**: 329-337.

Kado, C.I. & Heskett, M.G. (1970) Selective media for isolation of *Agrobacterium*, *Corynebacterium*, *Erwinia*, *Pseudomonas* and *Xanthomonas*. *Phytopathology* **60**: 969-976.

Kaiser, W.J. & Vakili, N.G. (1978) Insect transmission of pathogenic xanthomonads to bean and cowpea in Puerto Rico. *Phytopathology* **68**: 1057-1063.

Katznelson, H., Sutton, M.D. & Bayley, S.T. (1954) The use of bacteriophage of

Xanthomonas phaseoli in detecting infection in beans, with observations on its growth and morphology. *Canadian Journal of Microbiology* **1**: 22-29.

Kelly, J.D. & Miklas, P.N. (1999) Marker-assisted selection. In *Common Bean Improvement in the Twenty-First Century* (S.P. Singh, ed) : 93-123. Kluwer Academic Publishers, London.

Khandale, D.Y. & Kore, S.S. (1979) Screening of bean varieties for resistance to *Xanthomonas phaseoli*. *Indian Journal of Mycology and Plant Pathology* **9**: 121.

Kiriakov, I., Genchev, D. & Kiriakova, V. (1993) Common blight of dry bean (*Phaseolus vulgaris* L.) in north-east Bulgaria. *Annual Report of the Bean Improvement Cooperative* **36**: 152-153.

Kolkman, J.M & Michaels, T.E. (1994) Major gene control of common bacterial blight in *Phaseolus vulgaris*. *Annual Report of the Bean Improvement Cooperative* **37**: 73-74.

Lachman, L.K. & Schaad, N.W. (1985) Evaluation of the "dome test" as a reliable assay for seedborne bacterial blight pathogens of beans. *Plant Disease* **69**: 680-683.

Lazo, G.R. & Gabriel, D.W. (1987) Conservation of plasmid DNA sequences and pathovar identification of strains of *Xanthomonas campestris*. *Phytopathology* **77**: 448-453.

Lazo, G.R., Roffey, R. & Gabriel, D.W. (1987) Pathovars of *Xanthomonas campestris* are distinguishable by restriction length polymorphisms. *International Journal of Systematic Bacteriology* **37**: 214.

Leach, J.G., Lilly, V.G., Wilson, H.A. & Purvis, M.R. (1957) Bacterial polysaccharides: the nature and function of the exudate produced by *Xanthomonas phaseoli*. *Phytopathology* **47**: 113-120.

Leakey, C.L.A. (1973) A note on *Xanthomonas* blight of beans (*Phaseolus vulgaris* (L.) savi) and prospects for its control by breeding for tolerance. *Euphytica* **22**: 132-140.

Liang, L.Z., Halloin, J.M. & Saettler, A.W. (1992) Use of polyethylene glycol and glycerol as carriers of antibiotics for reduction of *Xanthomonas campestris* pv. *phaseoli* in navy bean seeds. *Plant Disease* **76**: 875-879.

Lienert, K.J. & Schwartz, H.F. (1994) Bacterial blight: screening and variability. *Annual Report of the Bean Improvement Cooperative* **37**: 59-60.

Mabagala, R.B. (1997) The effect of populations of *Xanthomonas campestris* pv. *phaseoli* in bean reproductive tissues on seed infection of resistant and susceptible bean genotypes. *European Journal of Plant Pathology* **103**: 175-181.

Mabagala, R.B. (1999) Epiphytic bacteria from various bean genotypes and their potential for biocontrol of *Xanthomonas axonopodis* pv. *phaseoli*. *Tanzania Journal of Agricultural Sciences* **2**: 19-26.

Mabagala, R.B. & Saettler, A.W. (1992) An improved semi-selective medium for recovery of *Xanthomonas campestris* pv. *phaseoli*. *Plant Disease* **76**: 443-446.

Malin, E.M., Roth, D.A. & Belden, E.L. (1983) Indirect immunofluorescent staining for detection and identification of *Xanthomonas campestris* pv. *phaseoli* in naturally infected bean seed. *Plant Disease* **67**: 645-647.

McElroy, J.B. (1985) Breeding for dry beans, *P. vulgaris* L., for common blight resistance derived from *Phaseolus acutifolius* A. Gray. PhD. Diss., Cornell University, Ithaca, New York.

Mejía-Jiménez, A., Muñoz, C., Jacobsen, H.J., Roca, W.M. & SINGH, S.P. (1994) Interspecific hybridization between common and tepary beans: increased hybrid

embryo growth, fertility, and efficiency of hybridization through recurrent and congruity backcrossing. *Theoretical Applied Genetics* **88**:324-331.

Michaels, T.E. (1992) Genetic control of common blight resistance in lines derived from *P. vulgaris* / *P. acutifolius* crosses. *Annual Report of the Bean Improvement Cooperative* **35**: 40-41.

Miklas, P.N., Beaver, J.S., Grafton, K.F. & Freytag, G.F. (1994) Registration of TARS VCI-4B multiple disease resistant dry bean germplasm. *Crop Science* **34**: 1415.

Miklas, P.N., Johnson, E., Stone, V., Beaver, J.S., Montoya, C. & Zapata, M. (1996) Selective mapping of QTL conditioning disease resistance in common bean. *Crop Science* **36**: 1344-1351.

Miklas, P.N., Smith, J.R., Riley, R., Grafton, K.F., Singh, S.P. Jung, G. & Coyne, D.P. (2000) Marker-assisted breeding for pyramiding resistance to common bacterial blight. *Annual Report of the Bean Improvement Cooperative* **43**: 39-40.

Mitchell, J.W., Zaumeyer, W.J. & Preston, W.H. Jr. (1954) Absorption and translocation of streptomycin by bean plants and its effect on the halo and common blight organisms. *Phytopathology* **44**: 25-30.

Mmbaga, M.T., Arnaud-Santana, E., Steadman, J.R. & Coyne, D.P. (1992) New sources of nonspecific resistance to rust and common bacterial blight in dry bean landrace Pompadour. *Euphytica* **61**: 135-144.

Moffet, M.L. & Middleton, K.J. (1979) Effect of common blight on yield of navy beans. *Australian Plant Pathology* **8**: 19.

Moffet, M.L. & Croft, B.J. (1983) *Xanthomonas.* In *Plant bacterial diseases: A diagnostic guide* (P.C. Fahy & G.J. Persley, eds) : 189-228. Academic Press, Australia.

Mohamed, M.F., Arnaud-Santana, E. & Coyne, D.P. (1993) Rooting of bean leaves and

use in germplasm evaluation for common bacterial blight resistance. *Euphytica* **65**: 161-166.

Mohan, S.T. & Mohan, S.K. (1983) Breeding for common bacterial blight resistance in beans. *Annual Report of the Bean Improvement Cooperative* **26**: 14-15.

Mutlu, N., Coyne, D.P., Park, S.O., Steadman, J.R., Reiser, J. & Jung, G. (1999) Backcross breeding with RAPD molecular markers to enhance resistance to common bacterial blight in pinto beans. *Annual Report of the Bean Improvement Cooperative* **42**: 7-8.

Navarrete-Maya, R., Acosta-Gallegos, J.A. & García-Espinoza, R. (1995) Screening bean genotypes for resistance to *Xanthomonas campestris* pv. *phaseoli*. *Annual Report of the Bean Improvement Cooperative* **38**: 158-159.

Navarrete-Maya, R. & Acosta-Gallegos, J.A. (1997) Common blight resistance in wild *Phaseolus vulgaris* L. *Annual Report of the Bean Improvement Cooperative* **40**: 102-103.

Nodari, R.O., Tsai, S.M., Guzman, P., Gilbertson, R.L. & Gepts, P. (1993) Towards an integrated linkage map of common bean. III. Mapping genetic factors controlling host-bacteria interactions. *Genetics* **134**: 341-350.

Opio, A.F. (1990) Control of common bacterial blight of beans in Uganda. *Annual Report of the Bean Improvement Cooperative* **33**: 41-42.

Opio, A.F., Teri, J.M. & Allen, D.J. (1992) Assessment of yield losses caused by common bacterial blight of beans in Uganda. *Annual Report of the Bean Improvement Cooperative* **35**: 113-114.

Opio, A.F., Teri, J.M. & Allen, D.J. (1993) Studies on seed transmission of *Xanthomonas campestris* pv. *phaseoli* in common beans in Uganda. *African Crop*

Science Journal **1**: 59-67.

Opio, A.F., Allen, D.J. & Teri, J.M. (1994a) Evaluation of inoculation methods and inoculum concentration for *Xanthomonas campestris* pv. *phaseoli*. *Annual Report of the Bean Improvement Cooperative* **37**:223.

Opio, A.F., Teri, J.M. & Allen, D.J. (1994b) Seed transmission efficiency for *Xanthomonas campestris* pv. *phaseoli*. *Annual Report of the Bean Improvement Cooperative* **34**: 221-222.

Opio, A.F., Allen, D.J. & Teri, J.M. (1995) The role of weeds and non-host crops in the survival of *Xanthomonas campestris* pv. *phaseoli* in Uganda. *Annual Report of the Bean Improvement Cooperative* **38**: 166-167.

Opio, A.F., Allen, D.J. & Teri, J.M. (1996) Pathogenic variation in *Xanthomonas campestris* pv. *phaseoli*, the causal agent of common bacterial blight in *Phaseolus* beans. *Plant Pathology* **45**: 1126-1133.

Oshima, N. & Dickens, L.E. (1971) Effects of copper sprays on secondary spread of common bacterial blight of beans. *Plant Disease Reporter* **55**: 609-610.

Paradez-López, O., Montes-Ribera, R., Gonzàlez Castaneda, J. & Arroyo-Figueroa, M.G. (1986) Comparison of selected food characteristics of three cultivars of bean *Phaseolus vulgaris*. *Journal of Food Technology* **21**: 487-494.

Park, S.J. & Dhanvantari, B.N. (1987) Transfer of common blight *(Xanthomonas campestris* pv. *phaseoli*) resistance from *Phaseolus coccineus* Lam. to *P. vulgaris* L. through interspecific hybridization. *Canadian Journal of Plant Science* **67**: 685-695.

Park, S.J., Michaels, T.E. & Dhanvantari, B.N. (1998a) Breeding for resistance to common bacterial blight and its effect on agronomic performance and processing

quality in dry bean. *Annual Report of the Bean Improvement Cooperative* **41**: 25-26.

Park, S.O., Coyne, D.P. & Jung, G. (1998b) Gene estimation, associations of traits, and confirmation of QTL for common bacterial blight resistance in common bean. *Annual Report of the Bean Improvement Cooperative* **41**: 145-146.

Park, S.O., Coyne, D.P., Mutlu, N., Jung, G & Steadman, J.R. (1999) Confirmation of molecular markers and flower colour associated with QTL for resistance to common blight in common beans. *Annual Report of the Bean Improvement Cooperative* **42**: 5-6.

Pastor-Corrales, M.A., Beebe, S.E. & Correa, F.J. (1981) Comparing 2 inoculation techniques for evaluating resistance in beans to *Xanthomonas campestris* pv. *phaseoli*. In *Proceedings of the fifth internacional conference on plant pathogenic bacteria* (J.C. Lozano, ed) : 493-503. CIAT, Cali, Colombia.

Patel, P.N. & Walker, J.C. (1963) Relation of air temperature and age and nutrition of the host to the development of halo and common bacterial blights of bean. *Phytopathology* **53**: 407-411.

Rands, R.D. & Brotherton, W. (1925) Bean varietal tests for disease resistance. *Journal of Agricultural Research* **31**: 110-154.

Redden, R.J. & Wong, W.C. (1995) Detection and elimination of common blight from common bean seed. *Annual Report of the Bean Improvement Cooperative* **38**:164-165.

Romeiro, R., Da, S., Moura, A.B., Oliveira, J.R., De Silva, H.S.A., Barbosa, L.S., Soares, F.M.P. & Peres, F. (1998) Evidences of constitutive multiple resistance to antibiotics in some plant pathogenic bacteria. *Summa Phytopathologica* **24**:

220-225.

Roth, D.A. (1988) Development of bean seed assays for bacterial pathogens. *Annual Report of the Bean Improvement Cooperative* **31**: 76-77.

Russkikh, I. (1999) Bacterial diseases of common beans in Belarus. *Sodininkyste ir Darzininkyste* **18**: 241-244.

Sabet, K.A. (1959) Studies on the bacterial diseases of Sudan crops. III. On the occurrence, host range and taxonomy of the bacteria causing leaf blight diseases on certain leguminous plants. *Annals of Applied Biology* **47**: 318.

Sabet, K.A. & Ishag, F. (1969) Studies on the bacterial diseases of Sudan crops. VIII. Survival and dissemination of *Xanthomonas phaseoli* (E.F. Sm.) Dowson. *Annals of Applied Bacteriology* **64**: 65-74.

Saettler, A.W. (1971) Seedling injection as an aid in identifying bean blight bacteria. *Plant Disease Reporter* **55**: 703-706.

Saettler, A.W. & Perry, S.K. (1972) Seed-transmitted bacterial diseases in Michigan navy (pea) beans, *Phaseolus vulgaris*. *Plant Disease Reporter* **56**: 378-381.

Saettler, A.W. (1977) Breeding dry edible beans (*Phaseolus vulgaris* L.) for tolerance to *Xanthomonas* bacterial blights. *Fitopatologia Brasileira* **2**: 179-186.

Saettler, A.W., Cafati, C.R. & Weller, D.M. (1986) Nonoverwintering of *Xanthomonas* bean blight bacteria in Michigan. *Plant Disease* **70**: 285-287.

Saettler, A.W. (1989) Common bacterial blight. In *Bean Production Problems in the Tropics 2nd ed* (H.F. Schwartz & M.A. Pastor-Corrales, eds) : 261-283. CIAT, Cali, Colombia.

Saettler, A.W. (1991) Diseases caused by bacteria. In *Compendium of bean diseases* (R. Hall, ed) : 29-32. APS-Press, St. Paul, Minnesota.

Schaad, N.W. & White, W.C. (1974) A selective medium for soil isolation and enumeration of *Xanthomonas campestris*. *Phytopathology* **64**: 876-880.

Schaad, N.W. & Stall R.E. (1988) Xanthomonas. In *Laboratory Guide for Identification of Plant Pathogenic Bacteria 2nd ed* (N.W. Schaad, ed) : 81-93. The American Phytopathological Society, St. Paul, MN, USA.

Schaad, N.W., Vidaver, A.K., Lacy, G.H., Rudolph, K. & Jones, J.B. (2000) Evaluation of proposed amended names of several pseudomonads and xanthomonads and recommendations. *Phytopathology* **90**: 208-213.

Schuster, M.L. (1955) A method for testing resistance of beans to bacterial blights. *Phytopathology* **45**: 519-520.

Schuster, M.L. (1983) Variability in virulence of Dominican Republic *Xanthomonas phaseoli* in CIAT *Phaseolus vulgaris* cultivars. *Fitopatologia Brasileira* **8**: 339-345.

Schuster, M.L. & Coyne, D.P. (1971) New virulent strains of *Xanthomonas phaseoli*. *Plant Disease Reporter* **55**: 505-506.

Schuster, M.L., Coyne, D.P. & Hoff, B. (1973) Comparative virulence of *Xanthomonas phaseoli* strains from Uganda, Colombia and Nebraska. *Plant Disease Reporter* **57**: 74-75.

Schuster, M.L. & Coyne, D.P. (1975) Genetic variation in bean bacterial pathogens. *Euphytica* **24**: 143-147.

Schuster, M.L. & Coyne, D.P. (1976) Survival mechanisms of phytopathogenic bacteria. *Annual Review of Phytopathology* **12**: 199-221.

Schuster, M.L. & Coyne, D.P. (1977a) Survival of plant parasitic bacteria of plants grown in the Tropics with emphasis on bean (*Phaseolus vulgaris*). *Fitopatologia*

Brasileira **2**: 117-130.

Schuster, M.L. & Coyne, D.P. (1977b) Characterization and variation of *Xanthomonas* and *Corynebacterium* incited diseases of beans (*Phaseolus vulgaris* L.). *Fitopatologia Brasileira* **2**: 199-209.

Schuster, M.L., Coyne, D.P., Nuland, D.S. & Smith, C.C. (1979) Transmission of *Xanthomonas phaseoli* and other bacterial species or varieties in seeds of tolerant bean (*Phaseolus vulgaris*) cultivars. *Plant Disease Reporter* **63**: 955-959.

Schuster, M.L. & Coyne, D.P. (1981) Biology, epidemiology, genetics and breeding for resistance to bacterial pathogens of *Phaseolus vulgaris*, L. *Horticultural Reviews* **3**: 28.

Schuster, M.L., Coyne, D.P., Behre, T. & Leyna, H. (1983) Sources of *Phaseolus* species resistance and leaf and pod differential reactions to common blight. *Horticultural Science* **18**: 901-903.

Schuster, M.L., Smith, C.C. & Salac, S.S. (1985) Effect of photoperiodism on common blight reactions of *Phaseolus* genotypes. *Phytopathologia Brasileira* **10**: 461-466.

Schwartz, H.F. and Otto, K.L. (2000) Enhanced bacterial disease management strategy. *Annual Report of the Bean Improvement Cooperative* **43**: 37-38.

Schwartz, H.F., Lienert, K. & Mcmillan, M.S. (1994) Timely and economical applications of pesticides to manage bean diseases. *Annual Report of the Bean Improvement Cooperative* **37**: 29-30.

Silva, L.O., Singh, S.P. & Pastor-Corrales, M.A. (1989) Inheritance of resistance to bacterial blight in common bean. *Theoretical Applied Genetics* **78**: 619-624.

Singh, SP. (1991) Bean Genetics. In *Common Beans, Research for Crop Improvement* (A. Van Schoonhoven & O. Voysest, eds) : 199-286. CAB International,

Wallingford.

Singh, S.P. & Muñoz, C.G. (1999) Resistance to common bacterial blight among *Phaseolus* species and common bean improvement. *Crop Science* **39**: 80-89.

Small, B.C. & Worley, J.F. (1956) Evaluation of 2,3,5-triphenyl tetrazolium chloride for obtaining pathogenic types from stock cultures of halo blight and common blight organisms. *Plant Disease Reporter* **40**: 628.

Smith, E.F. (1897) Description of *Bacillus phaseoli* n. sp. *Botanical Gazette* **24**: 192.

Starr, M.P. (1983) The genus *Xanthomonas*. In *The Procaryotes: a Handbook on habitats, isolation and identification of bacteria* (M.P. Starr, ed) : 742. Springer-Verlag, New York.

Steadman, J.R., Pastor-Corrales, M.A. & Beaver, J.S. (2002) An overview of the 3rd bean rust and 2nd bean common bacterial blight international workshops, March 4-8, 2002 Pietermaritzburg, South Africa. *Annual Report of the Bean Improvement Cooperative* **45**: 120-124.

Sutton, M.D. & Wallen, V.R. (1967) Phage types of *Xanthomonas phaseoli* isolated from beans. Canadian *Journal of Botany* **45**: 267-280.

Sutton, M.D. & Wallen, V.R. (1970) Epidemiological and ecological relations of *Xanthomonas phaseoli* and *X. phaseoli* var. *fuscans* on beans in southwestern Ontario, 1961-1968. *Canadian Journal of Botany* **48**: 1329-1334.

Tarigan, J.R. & Rudolph, K. (1996) Investigations on the resistance of bean genotypes to *Xanthomonas campestris* pv. *phaseoli* and *X. c.* pv. *phaseoli* var. *fuscans* and on the differentiation of bacterial strains. *Annual Report of the Bean Improvement Cooperative* **39**: 284-285.

Technology Impact Report. (1998) ARC-Grain Crops Institute, Oil and Protein Seed

Centre, Potchefstroom.

Thomas, W.D. & Graham, R.W. (1952) Bacteria in apparently healthy pinto beans. *Phytopathology* **42**: 214.

Toth, I.K., Hyman, L.J., Taylor, R. & 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*. *Journal of Applied Microbiology* **85**: 327-336.

Trujillo, G.E. & Saettler, A.W. (1979) A combined semi-selective medium and serology test for detection of *Xanthomonas* blight bacteria in bean seed. *Journal of Seed Technology* **4**: 35-41.

Tsai, S.M., Nodari, R.O., Moon, D.M., Camargo, L.E.A., Vencovsky, R. & Gepts, P. (1998) QTL mapping for nodule number and common bacterial blight in *Phaseolus vulgaris*, L. *Plant and Soil* **204**: 135-145.

Vakili, N.G., Kaiser, W.J., Pérez, J.E. and Cortés-Monllor, A. (1975) Bacterial blight of beans caused by two *Xanthomonas* pathogenic types from Puerto Rico. *Phytopathology* **65**: 401-403.

Valladarez-Sanchez, N.E., Coyne, D.P. & Schuster, M.L. (1979) Differential reaction of leaves and pods of *Phaseolus* germplasm to strains of *Xanthomonas phaseoli* and transgressive segregation for tolerance from crosses of susceptible germplasm. *Journal of the American Society for Horticultural Science* **104**: 648-654.

Valladarez-Sanchez, N.E., Coyne, D.P. & Mumm, R.F. (1983) Inheritance and associations of leaf, external, and internal pod reactions to common blight bacterium in *Phaseolus vulgaris* L. *Journal of the American Society for*

Horticultural Science **108**: 272-278.

Van Schoonhoven, A. & Pastor-Corrales, M.A. (1987) Standard system for the evaluation of bean germplasm. CIAT, Cali, Colombia.

Vauterin, L., Hoste, B., Kersters, K. & Swings, J. (1995) Reclassification of *Xanthomonas*. *International Journal of Systematic Bacteriology* **45**: 472-489.

Velich, I., Szarka, J. Néda, J. & Tóth, V. (1991) Diallel analysis of reaction of bean to *Pseudomonas* and *Xanthomonas*. *Annual Report of the Bean Improvement Cooperative* **34**: 31-32.

Venette, J.R., Lamppa, R.S., Albaugh, D.A. & Nayes, J.B. (1987) Presumptive procedure (dome test) for detection of seedborne bacterial pathogens in dry beans. *Plant Disease* **71**: 984-990.

Wallen, V.R., Sutton, M.D. & Grainger, P.N. (1963) A high incidence of fuscous blight in Sanilac beans from southwestern Ontario. *Plant Disease Reporter* **47**: 652-653.

Wallen, V.R. & 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.

Wallen, V.R. & Galway, D.A. (1976) Incidence of bacterial blight of field beans in southwestern Ontario in 1975. *Canadian Plant Disease Survey* **56**: 85-87.

Watson, D.R.W. (1970) Bean common blight and fuscous blight in New Zealand. *Plant Disease Reporter* **54**: 1068-1072.

Webster, D.M., Temple, S.R. & Galvez, G. (1983) Expression of resistance to *Xanthomonas campestris* pv. *phaseoli* in *Phaseolus vulgaris* under tropical conditions. *Plant Disease* **67**: 394-396.

Weller, D.M. & Saettler, A.W. (1976) Chemical control of common and fuscous bacterial blights in Michigan navy (pea) beans. *Plant Disease Reporter* **60**: 793-797.

Weller, D.M. & Saettler, A.W. (1980a) Evaluation of seedborne *Xanthomonas phaseoli* and X. *phaseoli* var. *fuscans* as primary inocula in bean blights. *Phytopathology* **70**: 148-152.

Weller, D.M. & Saettler, A.W.(1980b) Colonization and distribution of *Xanthomonas phaseoli* and *Xanthomonas phaseoli* var. *fuscans* in field-grown navy beans. *Phytopathology* **70**: 500-506.

Welsh, M.M. & Grafton, K.F. (1997) Identifying genes for resistance to common bacterial blight of bean. *Annual Report of the Bean Improvement Cooperative* **40**: 104-105.

Wimalajeewa, D.L.S. & Nancarrow, R.J. 1978. The incidence of bacterial blights of French bean (*Phaseolus vulgaris*) in East Gippsland, Victoria. *Australian Journal of Experimental Agriculture and Animal Husbandry* **18**: 318-320.

Wong, W.C. (1991) Methods for recovery and immunodetection of *Xanthomonas campestris* pv. *phaseoli* in navy bean seed. *Journal of Applied Bacteriology* **71**: 124-129.

Yoshii, K., Gálvez-E, G.E. & Alvarez-A, G. (1978) Screening bean germplasm for tolerance to common blight caused by *Xanthomonas phaseoli* and the 136importance of pathogenic variation to varietal improvement. *Plant Disease Reporter* **62**: 343-347.

Yoshii, K. (1980) Common and fuscous blights. In *Bean Production problems in the Tropics* (H.F. Schwartz & M.A. Pastor-Coralles, eds) : 157-172. CIAT, Cali, Colombia.

Yu, Z.H., Stall, R.E. & Vallejos, C.E. (1998) Detection of genes for resistance to common bacterial blight of beans. *Crop Science* **38**: 1290-1296.

Yu, K., Park, S.J. & Poysa, V. (1999) Application of molecular markers to assist selection of common beans resistant to common bacterial blight *(Xanthomonas campestris* pv. *phaseoli*). *Annual Report of the Bean Improvement Cooperative* **42**: 3-4.

Zaiter, H.Z., Coyne, D.P., Vidaver, A.K. & Steadman, J.R. (1989) Differential reaction of tepary bean lines to *Xanthomonas campestris* pv. *phaseoli*. *Horticultural Science* **24**: 134-137.

Zapata, H.Z. & Vidaver, A.K. (1987) Differentiation of *Xanthomonas campestris* pv. *phaseoli* into pathogenic races based on the tepary bean reactions. *Phytopathology* **77**: 1709 (Abstract).

Zapata, M. (1996) Pathogenic variability of *Xanthomonas campestris* pv. *phaseoli*. *Annual Report of the Bean Improvement Cooperative* **39**: 136-137.

Zapata, M., Rodríguez, R & Singh, S. (1998) Sources of resistance to *Xanthomonas campestris* pv. *phaseoli* from different geographical origins. *Annual Report of the Bean Improvement Cooperative* **41**: 58-59.

Zaumeyer, W.J. & Thomas, H.R. (1957) A monographic study of bean diseases and methods for their control. United States Department of Agriculture Technical Bulletin **868**: 65-74.

Table 1. Sources of resistance to common bacterial blight in dry beans

Variety	Origin	Reference
GN Nebr. # 1 Sel.27	UNL	Coyne & Schuster 1983
GN Tara	UNL	Coyne & Schuster 1983
GN Jules	UNL	Singh & Muñoz 1999
OAC 88-1	UGC	Singh & Muñoz 1999
XAN 159	CIAT	Mc Elroy, CIAT 1985
XAN 112	CIAT	CIAT 1984
XAN 91	CIAT	CIAT 1983
PI 207262	Colombia	Coyne & Schuster 1983
BAC 5	IAPAR	Arnaud-Santana *et al.* 1993
BAC 6	IAPAR	Arnaud-Santana *et al.* 1993
IAPAR 14	IAPAR	Beebe & Pastor-Corrales 1991
IAPAR 16	IAPAR	Beebe & Pastor-Corrales 1991
Tamaulipa 9-B (G 04399)	CIAT	Arnaud-Santana *et al.* 1993
MSU 183 (G 06700)	CIAT	Arnaud-Santana *et al.* 1993
Calima 9 (G 06772)	CIAT	Arnaud-Santana *et al.* 1993
PI 209.481 (G 16836	CIAT	Arnaud-Santana *et al.* 1993
RKN (G 18443)	CIAT	Arnaud-Santana *et al.* 1993
ODCSJ (G 18168)	CIAT	Arnaud-Santana *et al.* 1993
G 19195A	CIAT	Arnaud-Santana *et al.* 1993
PC 50	Dominican Republic	Schuster et al. 1983
ICA L-23	ICA, Colombia	Beebe & Pastor-Corrales 1991
Guama 23	ICA, Colombia	Beebe & Pastor-Corrales 1991
WBB-20-	UPR	CIAT 1997
G17341	CU	CIAT 1997
XAN 263	CIAT	CIAT 1997
XAN 309	CIAT	CIAT 1997
XAN 328	CIAT	Singh & Muñoz 1999
XAN 330	CIAT	Singh & Muñoz 1999
XAN 332	CIAT	CIAT 1997
Wilk 2	CU	Singh & Muñoz 1999
VAX 1	CIAT	Singh & Muñoz 1999
VAX 2	CIAT	Singh & Muñoz 1999
VAX 3	CIAT	CIAT 1997
VAX 4	CIAT	Singh & Muñoz 1999
VAX 5	CIAT	CIAT 1997
VAX 6	CIAT	CIAT 1997

UNL = University of Nebraska, Lincoln; UGC = University of Guelph; CIAT = Centro Internacional de Agricultura Tropical; IAPAR = Instituto Agronômico do Paraná; ICA = Instituto Clombiano Agropecuario; UPR = University of Puerto Rico; CU = Cornell University