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. Coefficients 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


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

<table>
<thead>
<tr>
<th>CV Name</th>
<th>Bean type</th>
<th>Growth habit*</th>
<th>Mean growing season (days)</th>
<th>Seed size (seeds 30g)</th>
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<tbody>
<tr>
<td>Teebus</td>
<td>Small white canning</td>
<td>I</td>
<td>92</td>
<td>127</td>
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<td>99</td>
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<td>II</td>
<td>96</td>
<td>156</td>
</tr>
<tr>
<td>PAN 182</td>
<td>Small white canning</td>
<td>II</td>
<td>90</td>
<td>183</td>
</tr>
<tr>
<td>PAN 185</td>
<td>Small white canning</td>
<td>II</td>
<td>96</td>
<td>183</td>
</tr>
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<td>Alubia</td>
<td>I</td>
<td>91</td>
<td>57</td>
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<td>Kranskop</td>
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<td>97</td>
<td>63</td>
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<td>Jenny</td>
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<td>96</td>
<td>57</td>
</tr>
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<td>Bonus</td>
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<td>Mkuzi</td>
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<td>II</td>
<td>96</td>
<td>143</td>
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</table>

* 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

<table>
<thead>
<tr>
<th>Cultivar</th>
<th>Mean disease rating (1-9)</th>
<th>Yield kg ha⁻¹</th>
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</thead>
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<td>de</td>
</tr>
<tr>
<td>PAN 182</td>
<td>6.5</td>
<td>f</td>
</tr>
<tr>
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<td>ef</td>
</tr>
<tr>
<td>Cerillos</td>
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<td>g</td>
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<tr>
<td>Kranskop</td>
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<td>de</td>
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<td>PAN 148</td>
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<td>Mkuzi</td>
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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

<table>
<thead>
<tr>
<th>Cultivar</th>
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<td>mno</td>
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<td>ef</td>
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<td>c</td>
<td>2031 de</td>
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<td>gh</td>
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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

<table>
<thead>
<tr>
<th>Cultivar</th>
<th>Mean disease rating (1-9)</th>
<th>Yield (kg ha⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Teebus</td>
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<td>6.0</td>
</tr>
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</tr>
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<td>3.0</td>
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<tr>
<td>PAN 185</td>
<td>2.7</td>
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<td>3.0</td>
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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 (Doldge & 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.


**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.

**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).


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

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) methylthiocarbamate (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-May & 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 (Mejia-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.


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-235-1-1 which 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 $10^5$ 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 $3-6\times10^7$ 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.

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

<table>
<thead>
<tr>
<th>Variety</th>
<th>Origin</th>
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<tr>
<td>GN Nebr. # 1 Sel.27</td>
<td>UNL</td>
<td>Coyne &amp; Schuster 1983</td>
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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