A review of the occurrence, biology and management of common bacterial blight

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Common bacterial blight caused by *Xanthomonas axonopodis* pv. *phaseoli* (*Xap*) has been reported in many countries of the world. The disease is prevalent in areas that experience warm weather conditions, causing up to 40% yield reduction. *Xap* grows on a number of different media producing colonies that are yellow, mucoid and convex. The bacterium is single celled and motile by means of a polar flagellum. Besides infecting *Phaseolus vulgaris*, *Xap* also attacks other legumes like *Glycine max* and *Dolichos lablab*. It is capable of epiphytic survival on both leguminous and non-leguminous plants like *Chenopodium album*, *Solanum nigrum*, *Zea mays* and *Amaranthus retroflexus*. The disease causes symptoms to appear on leaves, stems, flowers and seeds. The pathogen can survive in seeds for up to fifteen years, and is also known to overwinter in crop debris. Seed infection is the primary means by which the pathogen spreads. Therefore, the production and use of certified seeds is one control measure that is effective in dealing with the disease. Besides, there are chemical and cultural control options available in the management of common bacterial blight.

Key words: common bacterial blight, *Xanthomonas axonopodis* pv. *phaseoli*, *Phaseolus vulgaris*, epidemiology, symptoms, seedborne, disease management.

Occurence of common bacterial blight

Common bacterial blight (CBB) caused by *Xanthomonas axonopodis* pv. *phaseoli* (Smith) Vauterin *et al*. (*Xap*) has been reported in many countries. Weller and Saettler (1980b) reported the disease in Michigan, USA. In other parts of the USA, the disease has been reported in Nebraska, Colorado, Wyoming (CIAT, 1981), Nebraska, New York and Texas (CABI and EPPO, undated). The disease has also been reported in Colombia, Chile (Schuster and Coyne, 1975), Brazil, Mexico (Crispin and Campos, 1976), and the Dominican

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Republic (Angeles-Ramos *et al.*, 1991). These countries produce most of the beans consumed in the world (Musana *et al.*, 1993). Amongst the EPPO countries, the disease has been confirmed in Italy, Portugal (Madeira), The Netherlands, Greece, Italy and France.

In Asia, CBB has been reported in Bangladesh, India, China, Japan and the Koreas, while Australian states of New South Wales, Queensland, Western Australia and Victoria have also confirmed the presence of CBB. Besides Australia, the disease has also been reported in the oceanic states of New Zealand and Samoa (CABI and EPPO, undated).

In Africa, CBB has been reported as a major disease in Kenya (Njungunah *et al.*, 1981), Malawi (Edje *et al.*, 1981), Uganda, Kenya, Burundi (Opio *et al.*, 1993) and Tanzania (Karel *et al.*, 1981). CBB has also been reported in Angola, Mauritius, Lesotho and Mozambique (CABI and EPPO, undated). The South African provinces of Natal (now KwaZulu Natal) and Transvaal (now Limpopo) have reported widespread occurrence of CBB (Melis, 1987). In Zimbabwe, the disease has been reported in both the smallholder and large-scale commercial farming sectors in Natural Farming Regions II, III and IV (Giga, 1989).

The disease is of major economic importance in most lowland tropical and subtropical countries (Angeles-Ramos *et al.*, 1991; Gilbertson *et al.*, 1988), causing between 10 and 40% yield reduction in susceptible varieties (Birch *et al.*, 1997). In 1972, field bean loss by CBB in Ontario (Canada) was 217 724 kg while in 1970, it was 1 251 913 kg. According to Kennedy and Alcorn (1980), CBB was the most economically important bacterial disease in the USA, causing an estimated US$4 million loss in 1976.

Taxonomy and biochemical characteristics of Xap

Classification

CBB was conventionally considered as caused by *Xanthomonas campestris* pv. *phaseoli* (Smith) Dye. Work by Vauterin *et al.* (1995) reclassified the pathogen as *Xanthomonas axonopodis* pv. *phaseoli*. *Xap* belongs to γ-proteobacteria. Below is the currently used classification for the pathogen:

Kingdom: Prokaryotae
Family: Pseudomonadaceae
Genus: *Xanthomonas*
Species: *Xanthomonas axonopodis*
Pathovar: *phaseoli*

Biochemical and Morphological Characteristics

The bacterium is characterized by single cells that are straight rods (0.4-0.7 x 0.7-1.8µm), and are motile by means of a polar flagellum. It is a gram negative and strictly aerobic bacterium which does not reduce nitrates. *Xap* is catalase positive, and does not use asparagine as the sole source of carbon and nitrogen (Schaad, 1988). It is a weak producer of acids when grown on media containing carbohydrates like glucose, arabinose, mannose, trehalose and cellabiose (Hall, 1994). The bacterium is relatively intolerant to triphenyl tetrazolium chloride (TTC) and is inhibited by 0.02% TTC (Lelliot and Stead, 1987).

The bacterium grows on several media producing characteristic yellow colonies. On nutrient agar, the colonies are yellow, mucoid, glistening and convex with entire margins (Schaad, 1988). On Yeast Dextrose Agar, the colonies are yellow, mucoid, convex and shining. On MXP, they are yellow mucoid, smooth, convex and surrounded by zones of starch hydrolysis (Mabagala and Saettler, 1992). Colonies produced on Tween B are intensely yellow, mucoid and usually lipolytic. The identification of the colonies may be enhanced by the addition of crystal violet and soluble potato starch (Schaad, 1988). Some *Xap* strains produce non-water soluble but diffusible pigments in culture. These pigments are brominated arypolyene esters (xanthomonadins) that are soluble in petroleum ether, methanol and benzene and have absorption maxima in methanol at 420, 441, and 468nm wavelength (Hall, 1994). Plate 1 below shows colonies of *Xap* on culture media.
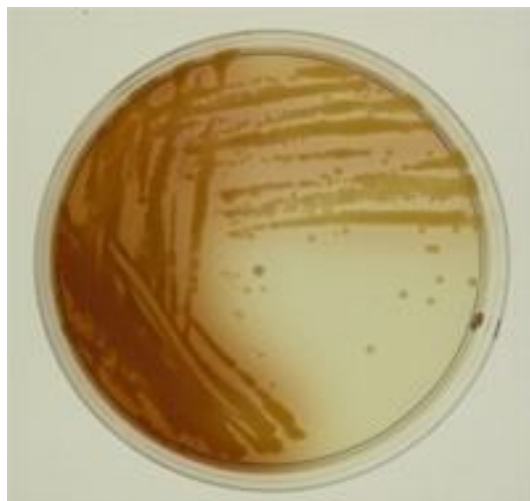

Plate 1: Colonies of *Xanthomonas axonopodis* pv. *phaseoli* on culture media.

Plant infection and symptoms of common bacterial blight

Leaf and Stem infections

Xap enter leaves through natural openings such as stomata and hydathodes or through wounds (Beattie and Lindow, 1995). The bacteria then invade intercellular spaces, causing gradual dissolution of the middle lamella. *Xap* may enter the stem through the stomata of the hypocotyls and epicotyls and reach vascular elements from infected leaves or cotyledons. The bacteria that exit through the stomata provide inocula for secondary spread. Presence of sufficient numbers of bacteria in the xylem tissue may cause plant wilting by plugging the vessels or disintegration of the cell walls (Yoshii, 1979).

Seed Infections

Xap can be haboured both within and on the seed coat (Hirano and Upper, 1983). The pathogen enters pod sutures from the vascular system of the pedicel and passes into the funiculus through the raphe leading into the seed coat. The pathogen either remains in the seedcoat or passes to the cotyledon when the seed germinates. Direct penetration through the seed coat has not been reported (Yoshii, 1979; CABI and EPPO, undated). If bacteria enter through the funiculus, only the hilum may become discolored.

Leaf and Stem symptoms

Leaf symptoms initially appear as water-soaked spots on the underside of leaves and leaflets. The spots then enlarge irregularly, and adjacent lesions frequently coalesce. The lesion can be up to 10mm in diameter (Macnab *et al.*, 1983). As the lesions enlarge and coalesce, the plants appear to be burnt. Lesions can be found at the margin and in interveinal areas of the node. Infected regions appear flaccid, and are encircled by a narrow zone of lemon-yellow tissue which later turns brown and necrotic. Serious infections may cause defoliation or stem girdling. Dead leaves may remain attached to the plant up to maturity time. Stem girdle or joint rot may develop at the cotyledonary stage, especially in plants that develop from infected seed. This causes the plants to break (Hall, 1994).

Plate 2: Pods and leaves infected by common bacterial blight disease.

Pod Symptoms

Symptoms consist of lesions that are generally circular, slightly sunken and dark red-brown. Lesions vary in shape and size depending on pod age. Under high humidity conditions, pod lesions are frequently covered with bacterial ooze (Melis, 1987). Plate 2 shows bean pods and leaves infected by common bacterial blight.

Seed Symptoms

Symptoms on white or light-coloured seeds are evident as butter-yellow or brown spots distributed throughout the seed coat or restricted to the hilum area (Mabagala, 1997). If infection occurs during pod and seed development, infected seed may rot or shrivel or may be wrinkled (Plate 3). If sown, such seed exhibits poor germination and vigor. Seed infections are difficult to see when the seeds are dark-coloured. Seedlings that develop from infected seed may sustain damage to the growing tip and be killed or stunted.

Plate 3: Bean pods and seeds infected by common bacterial blight disease. Photograph by H.F. Schwartz

Epidemiology of Xap

Optimum conditions for disease development

Xap is a warm temperature bacterium. It causes greater damage to plants at 28-32^{0}C than at temperature lower than 16^{0}C (Macnab *et al.*, 1983). High temperature, rainfall, and humidity favour rapid disease progress in the field. The time between initial infection and production of inocula for secondary spread is 10-14 days. The pathogen is spread by windblown rain, soil and plant debris, contact between wet plant leaves, irrigation water, animals, and insects like leafminers and whiteflies (Kaiser and Vakili, 1978).

Survival in crop debris

Debris from diseased plants has always been considered a possible source for seasonal carryover of plant pathogenic bacteria (Leben, 1981b; Purseglove, 1988). Gilbertson *et al.* (1988) showed that *Xap* can survive in dry leaves under laboratory conditions for at least six years. Karavina *et al.* (2008) isolated *Xap* from bean debris kept in the greenhouse for 12 months in Zimbabwe, while Opio *et al.* (1994) reported that the pathogen survived for more than 18 months in dried leaves kept in the laboratory in Sudan. Santana *et al.* (1991) reported that pathogen survival occurs in bean debris placed on top of, but not 20 cm below the soil surface. According to Osdaghi *et al.* (2010), bean pod debris in seed lots is capable of maintaining and transmitting *Xap*. The pathogen has also been reported to overwinter in weed debris under Nebraska field conditions

(Cafati and Saettler, 1980b). Survival of *Xap* in debris is greater under dry than moist conditions.

Survival in the soil

It is known that foliar pathogens are not well adapted to survival in the soil. Bacteria found in association with leaves are known to be quite distinct from those whose normal habitat is the soil, although similar genera may be found in both communities (Hirano and Upper, 1983). *Xap* can be recovered from the soil up to six weeks after burial of infected residues (Yoshii, 1979).

Survival in seed

Over 50 different plant pathogenic bacteria, including *Xap*, were listed in Neergard's survey of seedborne bacteria (Neergard, 1989). Survival of *Xap* on or within infected bean seed is one of the most effective means of the bacteria's survival (Cafati and Saettler, 1980c; Weller and Saettler, 1980b; Leben, 1981b; Saettler *et al*., 1995). Seed transmission of *Xap* has been known since 1972 (Schuster and Coyne, 1975). Contaminated seed is the primary source of inoculum (Gilbertson *et al*., 1990; Grum *et al*., 1998), and can provide the most effective means for both local and widespread dissemination of the pathogen. *Xap* has been recovered from three, ten and fifteen year old been seed (Schuster and Coyne, 1974; Ridout and Roberts, 1997). The recovered seedborne isolates normally were viable and virulent. Seed of tolerant bean cultivars can habour *Xap* and serve as sources of inocula (Cafati and Saettler, 1980b). Seedlings arising from the contaminated seed habour high numbers of the pathogen, which can colonize developing leaves (Weller and Saettler, 1980b). Low levels of bean seed infection with *Xap* are capable of initiating heavy field infections and causing severe crop losses under favorable environmental conditions (Schaad, 1988; Weller and Saettler, 1980b). As few as five pathogen-infected seeds among 10 000 bean seeds can result in a common blight epidemic (Leben, 1981a). Weller and Saettler (1980a) reported that surface epiphytic populations of 10^3 to 10^4 cfu/seed are required for plant infection. According to Webster *et al*. (1983b), at least 10^3 viable bacterial cells per seed were necessary for seedling infection of susceptible bean cultivars in Michigan, USA. If environmental conditions are not suitable for disease development, even heavily-infected seed may produce little or no disease (Cafati and Saettler, 1980a, c).

Epiphytic and Endophytic survival of Xap

Epiphytic bacteria are those bacteria capable of living (i.e. multiplying) on plant surfaces. They can be removed from above ground plant parts by washing or are killed by ultraviolet radiation or chemical surface disinfection (Beattie and Lindow, 1995). A wide range of bacteria, including *Xap*, have been detected on both upper and lower leaf surfaces (Morris and Rouse, 1982; Karavina *et al.*, 2011). Larger numbers of bacteria were found on the lower than on the upper leaf surface (Ishmaru *et al.*, 1991; Leben, 1981b). This was possibly due to the high density of stoma and/or trichomes on lower leaf surfaces, to a thinner cuticular layer on the lower surface, or to reduced exposure to ultraviolet radiation (Gilbertson *et al.*, 1987; Hirano and Upper, 1983). Leaf imprint studies have shown that bacteria are localized in particular sites on leaf surfaces. In scanning electron microscopy studies, the most common sites were bases of trichomes, at stomata, and epidermal cell wall junctions, especially in the grooves along the veins (Beattie and Lindow, 1995). Bacteria have also been observed in depressions in the cuticle, beneath the cuticle, near hydathodes and in stomatal pits.

Large epiphytic populations have been associated with times of disease onset and with increased amounts of disease for CBB (Weller and Saettler, 1980a). It has generally been accepted that disease symptoms are correlated rather closely with bacterial multiplication in the intercellular spaces. Large endophytic populations are needed in disease induction. The bacteria must reach internal tissues and establish endophytic populations for infection to occur (Beattie and Lindow, 1995). The endophytic population, not the epiphytic population, is responsible for disease induction.

Large populations of *Xap* can develop on leaf surfaces in the absence of the disease. A large population of *Xap* may increase the probability of large endophytic populations, but their presence does not ensure development of the endophytic populations that are sufficiently large to induce disease outbreak. The major factor influencing disease progress in the presence of sufficiently large epiphytic population is the amount of disease ingress, which depends on the number of entry points available and environmental conditions (Hirano and Upper, 1983). The number of natural entry sites is influenced by host genotype, leaf age and position on the leaf surfaces. For example, high stomatal frequency and wider stomatal aperture are correlated with host susceptibility. The major function of epiphytic populations in disease development is probably as sources of inocula for endophytic populations and for spread to surfaces of other host and nonhost plant parts.

Disease symptoms are often induced in susceptible hosts when the endophytic populations achieve a threshold level of 10^6 to 10^7 bacterial cells

per cm^2 (Weller and Saettler, 1980a; Wyman and van Etten, 1982). Disease induction occurs when either the pathogen population reaches the threshold size or the virulence of the pathogen or the susceptibility of the host changes. *Xap* can develop endophytic populations in resistant cultivars and in nonhost species (Karavina *et al*., 2011), but the size of the population is smaller in resistant than in susceptible cultivars (Ishmaru *et al*., 1991).

Dissemination of Xap

Insect transmission of Xap

Insect injury to bean foliage is generally prevalent during the rainy season. Insects are disseminators of bean bacterial pathogens. In the USA, *Melanoplus* spp (grasshoppers) and *Epilachna varivestis* (Mexican bean beetle) are considered important vectors of *Xap*. In studies by Kaiser and Vakili (1978), some isolates of *Xap* remained and retained their pathogenicity to beans after passing through the alimentary canal of *Chalcodermus ruficornis* (Erichson) and *Diaprepes abbreviata* (LeConte). Strong winds and wind-driven rains may transport bacterial blight-infected insects within and among susceptible crops, and facilitate the spread of bacteria and the establishment of new infections. It has been shown that leaf-chewing insects are more efficient disseminators of *Xap* than sucking insects.

The role of water in inoculum dispersal

The importance of water in the dispersal of microbes was first demonstrated in the 1880s by Pierre Miquel in Paris (Fitt *et al*., 1989). The first experiments to demonstrate dispersal of plant pathogenic inocula by rain were those by Faulwetter, who showed that windborne rain was responsible for the dispersal of *Xanthomonas campestris* pv. *malvacearum*, the causal agent of angular leaf spot in cotton. Rain is the principle agent in the dispersal of pathogens by splash.

Epiphytic bacterial populations tend to increase when plant surfaces are wet. The bacteria are transported from leaves by water. *Xap* has been found in leaf runoff water during rainfall or overhead irrigation. Weller and Saettler (1980a) estimated that at least 10% of common and fuscous blight pathogens present on bean leaves are removed during rainfall. The removal of these epiphytic bacteria has no net negative effect since bacterial multiplication tends to be high after rain or irrigation.

When crop canopies become saturated by rain, mist or dew, large drops may form on the leaves. These large raindrops are the most efficient in the

dispersal of inocula by rainsplash. Hirano and Upper (1983) reported that rainsplash only accounts for short distance dispersal- from leaf to leaf of the same plant or neighbouring plant. Rain-generated aerosols may have greater potential for transporting bacteria over modest distances. Under experimental conditions in the field, epiphytic bacteria tend to die after long periods of dry weather, immediately after they are artificially introduced on plant surfaces, usually by spray application. In greenhouses, epiphytic bacteria die when plants are maintained under relatively low humidity (Fitt *et al.*, 1989).

Host range of Xap

Besides *Phaesolus vulgaris* L. (principal host), *Xap* infects other legumes like tepary bean (*P. acutifolius*) Jacq, soyabean (*Glycine max* L.), *Dolichos lablab* L., *Lupinus polyphallus* Lindl., *Stizolobium deeringianum* Bort, *Vigna angularis* (Willd) Ohwi and H. Ohashi and cowpea (*Vigna unguiculata*) (L) Wilcz (Hall, 1994). It also infects *Vigna aconitifolia* (Jacq.) Marechal., *Vigna mungo* (L) Hepper and *Vigna radiata* (L.) R. Wilcz. In Tanzania and Uganda, non-leguminous hosts like *Chenopodium album* (L.), *Solanum nigrum* (L.), *Echinochloa crusgalli* (L.) Link, *Zea mays* L., *Beta vulgaris* (L) and *Amaranthus retroflexus* (L.) also act as inoculum sources for *Xap* (Cafati and Saettler, 1980b; Saettler, 1989). In the Dominican Republic, Angeles-Ramos *et al* (1991) detected *Xap* on *Euphorbia heterophylla* (L.), *Acanthospermum hispidum* (DC) and *Portulaca oleraceae*. The pathogen could not be detected on *Eleusine indica* (L.) Gaertn, *Setaria spp*, *Panicum maximum* (Jacq.) and *Leptochloa filiformis*. Karavina *et al.* (2011) detected pathogenic *Xap* strains on *Amaranthus hybridus* and *Zea mays*, while nonpathogenic xanthomonad strains were detected on *Oxalis latifolia*, *Bidens pilosa* and *Cyperus rotundus*.

Epiphytic *Xap* populations are generally lower on resistant compared to susceptible bean cultivars. This has been found on navy and tepary bean by Cafati and Saettler (1980b). Foliage and stems of resistant bean cultivars are known to habour relatively high populations of plant pathogenic bacteria without exhibiting discernible symptoms (Weller and Saettler, 1980a, b). The fact that a certain degree of preference for certain hosts apparently exists suggests that growth of some bacteria on their host(s) is selectively slightly faster, or death or emigration slightly less frequent so that over a large number of generations, there is modest enrichment.

Management of common bacterial blight

High disease incidence and severity result from a combination of genetic vulnerability, introduction of contaminated or infected seed and sufficient rain

and wind to spread inocula over a wide area. Therefore, an integrated approach is needed to manage CBB. Below are tactics that can be implemented in the management of CBB.

Cultural Control

Practices often utilized to reduce common blight are crop rotation, use of pathogen-free seed, choice of production site, use of clean seed and field hygiene (Saettler, 1991).

Crop Rotation

Crop rotation is when crops are grown on the same piece of land at different times. Ideally, crops that follow each other in a crop rotation sequence should be from different families. This will deprive pathogens of a food source when non-susceptible crops are grown; hence pathogen is starved to death. For CBB control, Cafati and Saettler (1980a) recommended a two year rotation with non-legumes. Crop rotation is difficult to implement for farmers with small land holdings and limited economic resources.

Site selection for bean production

Clean seed can be produced in a region free of the pathogen or where environmental conditions are unfavourable for disease development. This is one of the most reliable methods of producing disease-free crops (Gilbertson *et al.*, 1990). An ideal production site should have less than 300mm annual rainfall. The daily relative humidity and mean daily temperature should be less than 60% and 25^0C respectively, and there should be a gravitational irrigation facility (Mukoko, 1997; Purseglove, 1988). The cool weather which occurs on Zimbabwe's Highveld is favourable for clean seed production. The National Centre for Bean Research in Khomein, Iran, and its related fields are located in areas where the climate is considered non-conducive to CBB. Seeds from this Centre are less contaminated, and so are more advisable for planting in non-infected areas (Osdaghi *et al.*, 2010).

Field Hygiene

CBB-infected bean residues can be destroyed by burning or deep burial. Residue burning involves application of a flame to residues. Any *Xap* cells contained in the residues are killed by the heat generated. This method is very effective against the pathogen (Strange, 1993). However, it is not

environmentally friendly since smoke and carbon dioxide released into the atmosphere cause pollution.

Being a foliar pathogen, *Xap* cannot survive in the soil for long periods of time. Deep burial achieved by deep ploughing is effective in CBB management. However, as the world moves towards reduced tillage practices, deep ploughing may not be a favoured practice. Besides exposing the soil to erosion, deep ploughing may also contribute to environmental pollution whereby diesel-powered tractors emit fumes into the atmosphere.

Where seed production is taking place, personnel should disinfect their boots with sodium hypochlorite and also change clothes between fields. During the growing season, diseased plants should be rogued. Basal leaves of diseased plants should be removed at weeding. Being a polycyclic disease, roguing would reduce sources of inocula for the secondary spread of the pathogen. Rogued plants should be buried, burnt or composted to kill the pathogen.

Use of clean seed

Seedborne inoculum is the primary source of *Xap* dissemination. Therefore, the use of clean seed is crucial in the management of this CBB. Clean seed can be obtained by growing bean seed in areas that are unfavourable for pathogen development (Osdaghi *et al*., 2010). For example, areas with less than 60% relative humidity and temperature below 25^0C are favourable for clean bean seed production (Mukoko, 1997). Seed can also be dressed with chemicals like quintozene to kill contaminant bacteria.

Other cultural practices

Cafati and Saettler (1980a) recommended growing different cultivars in alternating seasons, and sequential planting of adjacent fields to reduce large acreage of susceptible plants at any time during a growing season. Different cultivars have different susceptibility to *Xap*. When a tolerant cultivar is grown, lower pathogen population build up compared to when susceptible cultivars are grown.

Chemical Control

Various chemicals can be applied as seed treatment or foliage protectants to control the disease before moderate or severe infection is apparent. Chemicals like copper sulphate, copper hydroxide, and potassium methy-dithiocarbamate can control foliage infection effectively (Yoshii, 1979; Webster *et al*., 1983a). In Zimbabwe, Olivine Industries (1998/1999)

recommend early and routine sprays of copper oxychloride or copper oxide as effective control measures.

Streptomycin and kasugamycin have been used to control external contaminant bacteria (Webster *et al*., 1983b). Streptomycin has been used in Idaho, USA, to treat seed stocks to reduce the levels of contaminant bacteria. Streptomycin has given marginal control in the laboratory and field. It is translocated within the plant, but not in the developing seed (Yoshii, 1979). Antibiotics like streptomycin should, however, not be foliarly applied as resistant bacterial mutants may be induced. In Zimbabwe, seed is dressed with fungicides like quintozene, thiram and/or carboxin before planting to control contaminant pathogens. There has been no satisfactory method of seed treatment that will completely control internally-borne *Xap*.

Use of Resistant Cultivars

Although short-term control is possible using disease-free seed, chemicals and crop rotation, long term control depends on the development of disease-resistant cultivars (Saettler, 1989; Opio *et al*., 1993). Webster *et al.* (1983b) estimated that 50% of the snap beans grown in the USA were susceptible to bacterial blight diseases. These cultivars however, had favourable horticultural characteristics like good taste and high yield (Webster *et al*., 1980).

In both resistant and susceptible cultivars, pathogen populations increased after inoculation, but the increase is less in resistant than in susceptible cultivars (Cafati and Saettler, 1980a, c; Hirano and Upper, 1983). Breeding for resistance is the most effective control measure under the smallholder farming sector where farmers retain seed for the subsequent cropping cycles (Webster *et al*., 1983a). In Zimbabwe, the cultivar Mkuzi is tolerant to CBB.

Biological Control

Biological control is the reduction of inoculum density or disease producing capacity (virulence) of a pathogen or parasite in its active or dormant state, by one or more organisms, accomplished naturally or through manipulation of the environment, host or antagonist, or by mass introduction of one or more antagonists. Bioassays have been carried out in Brazil in Brazil by Zanatta *et al*. (2007) to select a biological control agent for *Xap*. Isolates from soil planted with beans, isolates from bean pods and from bean leaves offered variable control of between 80-100% to *Xap*. The identity of the isolates is yet to be determined. To date, no biological control strategies have been commercialized for CBB.

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