Chapter 11

COMMON BACTERIAL BLIGHT

A. W. Saettler*

Introduction

Common bacterial blight is caused by the bacterium *Xanthomonas phaseoli* (Erw. Smith) Dowson and its brown pigment-producing fuscous variant, *X. phaseoli* var. *fuscans* (Burk.) Starr *et* Burk. Both bacteria are now recognized as *X. campestris* pv. *phaseoli* (Smith) Dye (Andersen, 1985) and will be referred to collectively as XCP throughout this chapter. Common blight is distributed worldwide (Costa, 1972; Crispín-Medina and Campos-Avila, 1976; Crispín-Medina et al., 1976; Mukunya et al., 1981; Orozco-Sarria, 1971; Pinto de Torres, 1968; Schieber, 1970; Vieira, 1967; Wallen and Galway, 1979). Common names frequently used for common bacterial blight in Latin America include "bacteriosis," "añublo bacterial común," "tizón común," and "crestamento bacteriano."

Yield losses caused by either of the two strains of XCP are difficult to estimate because the two bacteria frequently occur together in the same field, on the same plant, and causing identical symptoms. However, in 1967, XCP together damaged at least 75% of Michigan's 265,000 hectares of navy beans, with 10%-20% yield reductions (Focus on Michigan's bean industry, 1971). In two years of field trials, Wallen and Jackson (1975) reported a 38% yield loss in Ontario, Canada, because of XCP. Aerial infrared photographic surveys showed that these losses ranged from 1252 tons in 1970 to 218 tons in 1972 (Jackson and Wallen, 1975; Wallen and Jackson, 1975). Yield losses estimated at 22% and 45% have been obtained by natural and artificial infections, respectively, in Colombia (Yoshii et al., 1976a). Economic surveys, based upon field observations in the same region, estimated yield losses of 13% (Pinstrup-Andersen et al., 1976).

* Plant pathologist, Michigan State University, East Lansing, MI, USA.

Reported hosts of XCP are common bean (*Phaseolus vulgaris* L.), scarlet runner bean (*P. coccineus* L.), 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, *Lablab purpureus* (L.) Sweet, *Strophostyles helvola* (L.) Elliott, soybean (*Glycine max* (L.) Merrill), *Mucuna deeringiana* (Bort.) Merrill, *Lupinus polyphyllus* Lindl., and cowpea (*V. unguiculata* (L.) Walp. ssp. *unguiculata*) (Vakili et al., 1975; Zaumeyer and Thomas, 1957).

Etiology

Laboratory isolations and purifications are necessary to distinguish the two strains of XCP; the *fuscans* strain produces a diffusible brown pigment (melanin) on media containing tyrosine (Hayward and Waterston, 1965a and 1965b). Pigment-producing strains are more virulent than those not producing pigment (Basu and Wallen, 1967). However, the pigment may not be essential for pathogenicity and its production in *Xanthomonas* species not pathogenic to beans indicate that this is not a stable taxonomic character (Basu, 1974; Dye, 1962).

The XCP bacterium is a gram-negative straight rod that is strictly aerobic and motile by a polar flagellum. It produces a yellow water-insoluble carotenoid and mucoid growth on nutrient glucose agar. It produces acid on media containing arabinose, glucose, mannose, galactose, trehalose, or cellobiose. It also causes proteolysis of milk (Dye and Lelliott, 1974) and starch hydrolysis. The XCP grows well on potato dextrose, nutrient, and yeast-extract-dextrose calcium carbonate (YDC) agars. The YDC media is the most commonly used. It consists of 10 g of yeast extract, 10 g of dextrose, 2.5 g of calcium carbonate, and 20 g of agar in 1 liter of distilled water (Saettler, 1971). When glucose is deleted from YDC, the colonies of XCP are not mucoid.

Several general (Kado and Heskett, 1970; Schaad and White, 1974) or relatively selective (Claflin et al., 1985; Trujillo and Saettler, 1980) media for XCP are available which allow for rapid isolation of the pathogen and are useful for epidemiological studies. The XCP can be stored on silica gel for long periods (Leben and

Sleesman, 1982). Many bacteria are tolerant to desiccation and can survive extended dry conditions (Leben and Sleesman, 1982; Trujillo and Saettler, 1981). The XCP produces an extracellular polysaccharide in culture and in the host plant (Leach et al., 1957). The polysaccharide aids survival for prolonged periods under varied environmental conditions (Wilson et al., 1965).

Epidemiology

The XCP bacteria are warm-temperature pathogens, causing greater damage to plants at 28 °C than at lower temperatures (Goss, 1940; Mack and Wallen, 1974; Patel and Walker, 1963). They grow optimally in vitro from 28 to 32 °C and growth declines gradually as temperature is lowered until growth stops at 16 °C. Detailed meteorological and microclimatological data are not available to determine specifically which factors influence the development of bacterial blight epidemics. In general, however, common blight epidemics are favored by high temperature and humidity (Sutton and Wallen, 1970).

Infection of bean seed is the most effective means of survival for XCP. Bacteria have been recovered from bean seed that were 3, 10, and 30 years old (Basu and Wallen, 1966; Zaumeyer and Thomas, 1957; and Trujillo and Saettler, 1980, respectively). Seed-borne strains normally are virulent when recovered (Alvarez-C. et al., 1979; Saettler, 1971 and 1974; Saettler and Perry, 1972; Schuster and Coyne, 1977). Contamination by XCP is both internal and external; external contamination can be eliminated by applying bactericides such as streptomycin, to the seed.

Seed lots can be assayed for the presence of XCP by incubating seeds in water or a liquid medium and then inoculating susceptible plants with the suspension by injection, water-soaking (Schuster and Coyne, 1975a), or vacuum infiltration (Lahman and Schaad, 1985; Venette and Nayes, 1978). The most recent techniques of detection include enzyme-linked immunosorbent assay (ELISA), immunofluorescence, and a combined semiselective media and serology test (Afanador and Victoria, 1981; Malin et al., 1983; Trujillo and Saettler, 1979). Saettler and Perry (1972) assayed 101 navy bean seed lots for internal seed contamination with XCP and

about 35% of the lots were contaminated: 13% with the *fuscans* variant and 52% with both strains. Wallen et al. (1963) sampled 23 seed lots from Ontario, Canada, and isolated virulent cultures of the *fuscans* strains from more than 50% of the samples. The minimum number of infected seeds required to incite an epidemic is not known but must be determined for various cultural and environmental conditions.

Short-term survival within or on healthy-appearing bean plants occurs during the growing season (Thomas and Graham, 1952) and bacteria multiply on symptomless leaves (Weller and Saettler, 1978 and 1980a). XCP grows epiphytically on leaves of nonhost crop species such as soybean (*Glycine max*), maize (*Zea mays* L.), beet (*Beta vulgaris* L.), and cowpea (*Vigna unguiculata* ssp. *unguiculata*), and weeds (*Chenopodium album* L., *Amaranthus retroflexus* L., *Solanum nigrum* L., *Ambrosia artemisiifolia* L., and *Echinochloa crus-galli* (L.) Beauvois). Viable populations were recovered up to 21 days after bacteria were placed on leaf surfaces. Spread of XCP from *C. album* and *A. retroflexus* to bean plants occurred within 12 days after the weeds were inoculated (Cafati and Saettler, 1980b).

Overwinter survival of XCP in infested plant debris has been reported from some temperate regions (Burkholder, 1930). In Nebraska XCP survived in bean debris placed on top of the soil surface, but not when buried 20 cm below. Survival was greater under dry than under moist environmental conditions. Bacteria were recovered from the soil up to six weeks after burial. However, Schuster (1967) speculated that survival occurred in infested plant debris. In contrast, Sutton and Wallen (1970) could not isolate XCP from soil in which infected plants had grown. Saettler et al. (1986) concluded from a 10-year study in Michigan that XCP did not survive in association with residue. Several reports mention that blight symptoms failed to develop when pathogen-free seed was planted in soil infested with XCP from the previous season (Burkholder, 1930; Hedges, 1946; Wimalajeewa and Nancarrow, 1980). However, it is believed that, under some conditions, blight organisms can survive in soil for 18 months or more.

In general, then, in temperate bean-growing regions, infested bean residue is not always an important primary inoculum source of

XCP. However, in tropical bean-growing regions, infested residue is probably important in bean blight epidemiology because of the opportunities for bacteria to multiply and survive as epiphytes on perennial hosts and because of the practice of intercropping. However, van Rheenen et al. (1981) observed a decreased incidence of XCP spread throughout beans grown in association with maize compared with monoculture. Apparently, the maize provided a biological barrier to the physical movement (e.g., by wind or rain) of bacteria between bean plants. Further research is therefore needed to study the factors that affect the survival and longevity of XCP under tropical and temperate conditions.

The XCP bacteria are disseminated effectively on and within bean seed. Seed transmission of XCP has been known since 1872 (Schuster and Coyne, 1974 and 1975c). Plants grown from infected seed frequently bear lesions on cotyledons, nodes, or primary leaves. These lesions serve as secondary sources of inoculum during favorable environmental conditions (Burkholder, 1930). Infected seed or infested plant debris may be present within bean cull piles which then act as initial sources of inoculum (Burke, 1957). Volunteer plants present in fields provide another locus from which bacteria may be disseminated to susceptible plants.

Secondary spread of common and fuscous blight bacteria is effected by rain accompanied by wind (Zaumeyer and Thomas, 1957), windblown soils (Claflin et al., 1973), irrigation water (Steadman et al., 1975), people and animals, and insects such as the whitefly (Sabet and Ishag, 1969). XCP survives on insects. Leaf-feeding insects such as the borer *Diaprepes abbreviatus* (Boh.) and the beetle *Cerotoma ruficornis* (Ol.), can transmit the bacteria to wounds caused during feeding (Kaiser and Vakili, 1978). Spread of XCP by aerosols (Venette and Kennedy, 1975) has not been reported but other bacterial pathogens are spread this way.

Symptomatology

Both strains of XCP induce identical symptoms on leaves, stems, pods, and seeds. Leaf symptoms initially appear as water-soaked spots (Figure 78) which enlarge and frequently coalesce with adjacent lesions. Infected tissues appear flaccid and lesions are often

encircled by a narrow zone of lemon-yellow tissue. Necrosis then develops (Figure 79) and may become extensive enough (Figure 80) to cause defoliation or stem girdle (Zaumeyer and Thomas, 1957).

Blight bacteria enter leaves through natural openings such as stomata and hydathodes, and wounds (Zaumeyer and Thomas, 1957). They then invade intercellular spaces, causing a gradual dissolution of the middle lamella. Bacteria enter the stem through stomata of the hypocotyl and epicotyl and reach vascular elements from infected leaves or cotyledons. Colonization of xylem tissue may cause plant wilting by plugging vessels or disintegrating cell walls. The XCP does not systemically infect all *Phaseolus vulgaris* cultivars (Haas, 1972). Stem girdle or joint rot may develop at the cotyledonary node, especially in plants that grew from infected seed, and cause the plant to break at the node (Zaumeyer and Thomas, 1957) (Figure 81).

Pod lesions appear as water-soaked spots which may enlarge and become dark, red, and slightly sunken. If infection occurs during pod and seed development, infected seed may rot or shrivel (Figure 82). Seed infection occurs when the bacteria enter pod sutures via the pedicel or pod vascular system and pass into the funiculus through the raphe leading into the seed coat. The micropyle also may serve as a point of entry into the developing seed. Direct penetration through the seed coat has not been reported. If bacteria enter through the funiculus, only the hilum may become discolored. Studies have shown that infected seed can be found even in symptomless pods (Cafati and Saettler, 1980c; Weller and Saettler, 1980b). Symptoms on seed manifest as butter-yellow spots on white or light-colored seeds (Saettler and Perry, 1972; Zaumeyer and Thomas, 1957), but are difficult to see on medium to dark-colored seeds. Seedlings which develop from severely infected seed may have damaged growing tips, be stunted, or killed (snakehead) (Zaumeyer and Thomas, 1957).

There are several reports that other bean diseases can affect the severity of common blight. Panzer and Nickeson (1959) demonstrated that common blight is more severe in the presence of bean common mosaic virus, particularly late in the season. Hedges (1944) found that the common mosaic virus persisted in cultures of *X. phaseoli* for six weeks. Díaz-Polanco (1972) also showed that in the

infection of bean leaves a synergistic effect existed between *X. phaseoli* and the ashy stem blight fungus (*Macrophomina phaseolina* (Tassi) Goid.)

Zaumeyer and Thomas (1957) suggested that the *fuscans* variant caused a slight hypertrophy and darkening of the stem at the point of artificial inoculation of young seedlings. Moreover, several authors report severe plant symptoms following inoculation with the *fuscans* strain (Ekpo and Saettler, 1976; Zaumeyer and Thomas, 1957). Inoculation with mixtures of the two strains can induce severer symptoms than inoculation with a single strain (Ekpo, 1975).

Control by Cultural Practices

Cultural practices used to control common blight are planting pathogen-free seed (Webster et al., 1983a; Weller and Saettler, 1980b), crop rotation, and deep-plowing (Zaumeyer and Thomas, 1957). Clean or certified seed must be produced in regions free of pathogen or where environmental conditions discourage disease development. All seed must be tested for internal XCP contamination because studies have shown that symptomless bean plants can still produce contaminated seed (Cafati and Saettler, 1980c). Crop rotation with resistant crops gives time for the XCP population in bean debris within a field to decline.

Chemical Control

Various chemicals are used to protect foliage against XCP. Although some chemicals are effective in controlling foliage infection, yield increases have usually been minimal. Effective compounds include basic copper sulfate (Dickens and Oshima, 1969), copper hydroxide, and potassium *N*-hydroxymethyl-*N*-methyldithiocarbamate (Bunema) (Weller and Saettler, 1976). Streptomycin provided marginal control in laboratory and field tests; it is translocated within the plant but not into the developing seeds (Mitchell et al., 1952; 1953; and 1954). However, antibiotics should not be applied to leaves because resistant mutants of the pathogen may develop. A new approach to seed treatment, still in experimen-

tal stage, is to use organic solvents to infuse antibiotics into bean seed.

Control by Plant Resistance

Strains of XCP differ in pathogenicity and virulence within and between geographical locations (Jindal and Patel, 1984; Schuster and Coyne, 1975b; Yoshii et al., 1976b). Schuster and Coyne (1971) obtained isolates from Colombia that were more virulent than several North American strains. Strains from Uganda were as virulent as those from Colombia (Schuster et al., 1973). Isolates with even greater virulence have since been identified (Ekpo and Saettler, 1976; Jindal and Patel, 1984). Differences in pathogenicity can also exist between colonies taken from individual stock cultures of XCP (Corey and Starr, 1957; Smale and Worley, 1956). However, documenting these differences has been complicated by variation in inoculation methods, age of isolates, and other factors.

Several different methods of plant inoculation have been tested:

pricking the cotyledon or cotyledonary node with a needle or scalpel dipped in inoculum (Arp et al., 1971; Burkholder and Bullard, 1946);

rubbing the second trifoliolate leaves with a cotton swab soaked with a carborundum-inoculum mixture (Corey and Starr, 1957);

soaking leaves with inoculum at high pressure (Arp et al., 1971; Schuster, 1955);

vacuum infiltrating into leaves (Venette and Nayes, 1978);

pricking leaves with a multiple needle cushion (Andrus, 1948; Pastor-Corrales et al., 1981; Pompeu and Crowder, 1972); and

clipping leaves with scissors or razor blades dipped in inoculum (Ekpo, 1975; Webster, 1978; Webster et al., 1980).

Inoculum concentrations can influence the disease reaction. Optimal concentrations for uniform infection are between 10 million to 100 million cells/ml (Coyne et al., 1973; Ekpo, 1975; Pompeu and Crowder, 1973).

Phaseolus vulgaris cultivars and breeding materials vary in their reaction to infection by XCP (Mohan, 1981; Webster et al., 1980 and 1983b) (Figure 83). Immunity to infection has not been found, but many genotypes are resistant to infection, with little, if any, yield loss (Allen, 1983). However, bacteria can survive in tissue of resistant lines without causing symptoms (Cafati and Saettler, 1980a; Scharen, 1959). Phytoalexins, apparently, are not involved in resistance (Wyman and VanEtten, 1982). In general, beans are more susceptible to infection after the start of blossoming, that is, during the reproductive stage (Coyne and Schuster, 1973, 1974a, and 1974d; Coyne et al., 1973). Many workers, therefore, inoculate plants during flowering and evaluate reactions three to four weeks later. However, in the tropics, inoculations at three to four weeks after planting may be more useful, particularly if germplasm is variable in maturity, growth habit, and adaptation (CIAT, 1978; Webster, 1978). Coyne and Schuster (1974b) observed differential leaf and pod reactions to infection by XCP. The reactions were conditioned by different genes (Schuster et al., 1983; Valladares-Sánchez et al., 1983). Thus, the time of evaluation and design of disease rating scales must carefully account for these factors (Saettler, 1977).

Schuster (1955) first reported that *Phaseolus acutifolius* A. Gray (tepary bean) was resistant to XCP. Honma (1956) transferred genes from this resistant source into *Phaseolus vulgaris*, using embryo rescue to produce F_1 hybrid plants. Coyne and co-workers (1963 and 1973) surveyed more than 1000 plant introduction (P.I.) lines for resistance to XCP in the field. They found seven highly resistant *P. vulgaris* genotypes: P.I. 163117 (accession from India), P.I. 167399 and P.I. 169727 (accessions from Turkey), P.I. 197687 (accession from Mexico), P.I. 207262 and ICA Gualí (accessions from Colombia), and Great Northern (G.N.) Nebraska No. 1 selection 27. Yoshii et al. (1978) reported that P.I. 282086 and P.I. 313343 exhibited resistant foliage reactions, but that the former also exhibited a susceptible pod reaction.

Phaseolus acutifolius "Tepary Buff" (Coyne and Schuster, 1974a) and P.I. 169932 (Yoshii et al., 1978) had high degrees of resistance with no symptoms observed. Several lines of *P. coccineus* were also resistant, but less so than tepary (Coyne and Schuster, 1974a). McElroy (1985) showed that three major genes determined the

reaction to a Colombian isolate of XCP of a cross of resistant with susceptible tepary beans. He successfully transferred resistance derived from the resistant source (Thomas and Waines, 1984) in a backcross program to different susceptible *P. vulgaris* cultivars.

Several of these resistant materials have been tested at various locations and exposed to bacterial isolates more virulent than those originally used. Although G.N. Nebraska No. 1 selection 27 and P.I. 207262 were also resistant to Brazilian isolates of XCP *fuscans* (Cafati and Kimati, 1972), the former was susceptible to a Colombian XCP isolate (Coyne et al., 1973). Poor plant adaptation to tropical growing conditions in Colombia apparently prevented the expression of resistance by G.N. Jules and P.I. 207262 (CIAT, 1978; Webster, 1978), until the plants became agronomically adapted through breeding and selection. Arnaud-Santana (1985) observed that *P. vulgaris* cv. Pompadour Checa is susceptible in the Dominican Republic (short days), but was moderately resistant in Nebraska (long days). However, susceptibility was expressed again when crossed to resistant adapted germplasm. Coyne et al. (1965 and 1973) found an association between delayed flowering and common blight resistance in Nebraska (long photoperiods), while Mohan (1981) found no association in Brazil (short photoperiods).

Inheritance of resistance to XCP recently has been reviewed (Coyne and Schuster, 1974a; Leakey, 1973; Schuster and Coyne, 1981; Zaumeyer and Meiners, 1975). Honma (1956) made the original interspecific cross between resistant *P. acutifolius* "Tepary 4" and susceptible *P. vulgaris* and found that resistance was quantitatively inherited. Coyne et al. (1965) further studied the inheritance of resistance in crosses to an early maturing, susceptible cultivar G.N. 1140. The resistant reaction was inherited quantitatively and linked to delayed flowering under a long photoperiod and high temperature (Coyne et al., 1973).

The late-maturing G.N. Tara and G.N. Jules (Coyne and Schuster, 1969 and 1970) and early maturing G.N. Valley (Coyne and Schuster, 1974c) cultivars, derived from the cross with G.N. 1140, are resistant to XCP in most temperate regions of United States. G.N. Starr is an early maturing cultivar in which genes for resistance in P.I. 165078 (also tolerant to the bacterial wilt (*Corynebacterium flaccumfaciens* ssp. *flaccumfaciens*)) were trans-

ferred through six backcrosses to the recurrent parent G.N. Nebraska No. 1 selection 27 (tolerant to *X. phaseoli*) (Coyne and Schuster, 1976).

Coyne et al. (1966 and 1973) report that the cross between G.N. 1140 and G.N. Nebraska No. 1 selection 27 exhibited partial dominance for susceptibility. Similar inheritance patterns also were reported by Pompeu and Crowder (1972) for crosses between G.N. Nebraska No. 1 selection 27 and local susceptible parents. Crosses between resistant P.I. 207262 and susceptible cultivars such as G.N. 1140, revealed that the resistant reaction was completely dominant in the F_1 generation (Coyne and Schuster, 1974d). Transgressive segregation has been observed in these crosses (Coyne et al., 1966 and 1973; Pompeu and Crowder, 1972; Valladares-Sánchez et al., 1979 and 1983). Breeders should therefore be able to increase the levels of resistance within promising germplasm.

Suggestions for the Integrated Control of XCP

There are a number of practices which bean growers can use to minimize losses from XCP. These practices are described in the form of instructions:

Plant high-quality disease-free seed. Use the highest quality seed that is free of internal XCP infection. Discard all seed showing spotting or discoloration characteristic of XCP.

Treat seed with a bactericide. Treat all bean seed prior to planting with a slurry containing a bactericide that will kill bacteria infesting the seed surface.

Avoid cropping beans after beans. Practice a 2- to 3-year crop rotation to protect seed from blight organisms and other soil-borne pathogens that build up when beans follow beans too closely in rotation.

Deep-plow all bean refuse after harvest. Deep-plow fields with infected bean straw as soon as possible after harvest. This will prevent infested leaf tissue and straw from being transported to those parts of the farm where beans may be planted in the following year. This practice is especially important if a 2- to 3-year crop

rotation cannot be followed. If necessary, infected debris must be removed manually and destroyed by burning.

Isolate infected fields. Do not plant beans grown for seed next to commercial bean fields. This will avoid the spread of XCP from adjacent fields by wind, water, man, or animals. Do not grow beans where the water runoff from last year's contaminated bean fields can contaminate the new (unused) fields. The more isolated the field, the greater the chances are of avoiding infection. Avoid unnecessary activity in bean fields.

Use good herbicides to control weeds. Weed-free fields permit aeration around the plants so that they dry off more quickly. The shorter the exposure to continual wetness, the shorter the blight infection periods and so the lesser the infection in plants. In addition, some weeds may actually harbor bean blight bacteria.

Stay out of the fields as much as possible. Never work in the fields while the plants are wet with dew or rain because bacteria spread and infection takes place most readily under these conditions. Remember that every time you enter a field there is a chance of spreading pathogens by animals, humans, or equipment.

References

Afanador, L. and Victoria, J. I. 1981. Specificity of Elisa to *Xanthomonas campestris* pv. *phaseoli* identification. In: Lozano, J. C. (ed.). Proceedings of the 5th international conference on plant pathogenic bacteria, August 16-23, 1981, at CIAT, Cali, Colombia. Centro Internacional de Agricultura Tropical (CIAT), Cali, Colombia, and University of Missouri, Columbia, MO, USA. p. 113-121.

Allen, D. J. 1983. The pathology of tropical food legumes: disease resistance in crop improvement. Wiley, Chichester, England. 413 p.

Alvarez-C., E.; Vanegas-G., G.; and Victoria-K., J. I. 1979. Transmisión por semilla de bacterias fitopatógenas del frijol (*Phaseolus vulgaris* L.) en Colombia. Acta Agron. (Colombia) 29(1-4):11-20.

Andersen, A. L. 1985. Common names for plant diseases: bean (*Phaseolus vulgaris* L.). Plant Dis. 69(8):653.

Andrus, C. F. 1948. A method of testing beans for resistance to bacterial blights. Phytopathology 38(9):757-759.

Arnaud-Santana, E. 1985. Genetic variation, inheritance and relationship of the reaction to common blight (*Xanthomonas campestris* pv. *phaseoli* (Smith) Dye, and number of days to flowering in dry beans (*Phaseolus vulgaris* L.). M.Sc. thesis. University of Nebraska, Lincoln, NE, USA.

Arp, G.; Coyne, D. P.; and Schuster, M. L. 1971. Disease reaction of bean varieties to *Xanthomonas phaseoli* and *Xanthomonas phaseoli* var. *fuscans,* using two inoculation methods. Plant Dis. Rep. 55(7):577-579.

Basu, P. K. 1974. Glucose inhibition of the characteristic melanoid pigment of *Xanthomonas phaseoli* var. *fuscans.* Can. J. Bot. 52(10):2203-2206.

——— and Wallen, V. R. 1966. Influence of temperature on the viability, virulence, and physiologic characteristics of *Xanthomonas phaseoli* var. *fuscans* in vivo and in vitro. Can. J. Bot. 44:1239-1245.

——— and ———. 1967. Factors affecting virulence and pigment production of *Xanthomonas phaseoli* var. *fuscans.* Can. J. Bot. 45:2367-2374.

Burke, D. W. 1957. Incidence of bacterial pathogens in dry beans in irrigated districts of Nebraska, Wyoming, and Colorado in 1954 and 1955. Plant Dis. Rep. 41:488-490.

Burkholder, W. H. 1930. The bacterial diseases of the bean: a comparative study. Memorandum no. 127. New York Experiment Station, Cornell University, Ithaca, NY, USA. 88 p.

——— and Bullard, E. T. 1946. Varietal susceptibility of beans to *Xanthomonas phaseoli* var. *fuscans.* Plant Dis. Rep. 30:446-448.

Cafati, C. R. and Kimati, H. 1972. Reacción de variedades de frejol a *Xanthomonas phaseoli* (E. F. Sm.) y *Xanthomonas phaseoli* var. *fuscans* (Burk.) Starr and Burk. Agric. Tec. (Santiago) 32(3):153-160.

——— and Saettler, A. W. 1980a. Effect of host on multiplication and distribution of bean common blight bacteria. Phytopathology 70(7):675-679.

——— and ———. 1980b. Role of nonhost species as alternate inoculum sources of *Xanthomonas phaseoli*. Plant Dis. 64(2):194-196.

——— and ———. 1980c. Transmission of *Xanthomonas phaseoli* in seed of resistant and susceptible *Phaseolus* genotypes. Phytopathology 70(7):638-640.

CIAT (Centro Internacional de Agricultura Tropical). 1978. Bean Program. In: Annual report 1977. Cali, Colombia. p. B-1 to B-85.

Claflin, L. E.; Sasser, M. D.; and Vidaver, A. K. 1985. MXP, a semi-selective medium for *Xanthomonas campestris* pv. *phaseoli*. Phytopathology 75(11):1360. (Abstr.)

———; Stuteville, D. L.; and Armbrust, D. V. 1973. Wind-blown soil in the epidemiology of bacterial leaf spot of alfalfa and common blight of bean. Phytopathology 63(11):1417-1419.

Corey, R. R. and Starr, M. P. 1957. Colony types of *Xanthomonas phaseoli*. J. Bacteriol. 74:137-140.

Costa, A. S. 1972. Investigações sobre moléstias do feijoeiro no Brasil. In: Anais do I simpósio brasileiro de feijão, Campinas, 22 à 29 de agôsto de 1971, 2 vols. Universidade Federal de Viçosa, Viçosa, MG, Brazil. Vol. 2, p. 303-384.

Coyne, D. P. and Schuster, M. L. 1969. 'Tara': a new Great Northern dry bean variety tolerant to common blight bacterial disease. SB 506. Agriculture Experiment Station, College of Agriculture and Home Economics, University of Nebraska, Lincoln, NE, USA. 10 p.

——— and ———. 1970. 'Jules', a Great Northern dry bean variety tolerant to common blight bacterium (*Xanthomonas phaseoli*). Plant Dis. Rep. 54(7):557-559.

——— and ———. 1973. Phaseolus germplasm tolerant to common blight bacterium (*Xanthomonas phaseoli*). Plant Dis. Rep. 57(2):111-114.

——— and ———. 1974a. Breeding and genetic studies of tolerance to several bean (*Phaseolus vulgaris* L.) bacterial pathogens. Euphytica 23(3):651-656.

——— and ———. 1974b. Differential reaction of pods and foliage of beans (*Phaseolus vulgaris*) to *Xanthomonas phaseoli*. Plant Dis. Rep. 58(3):278-282.

——— and ———. 1974c. 'Great Northern Valley' dry bean. HortSci. 9(5):482.

——— and ———. 1974d. Inheritance and linkage relations of reaction to *Xanthomonas phaseoli* (E. F. Smith) Dowson (common blight), stage of plant development and plant habit in *Phaseolus vulgaris* L. Euphytica 23(2):195-204.

——— and ———. 1976. 'Great Northern Star' dry bean tolerant to bacterial diseases. HortSci. 11(6):621.

———; ———; and Al-Yasiri, S. 1963. Reaction studies of bean species and varieties to common blight and bacterial wilt. Plant Dis. Rep. 47(6):534-537.

———; ———; and Harris, L. 1965. Inheritance, heritability, and response to selection for common blight (*Xanthomonas phaseoli*) tolerance in *Phaseolus vulgaris* field bean crosses. Proc. Am. Soc. Hortic. Sci. 86:373-379.

———; ———; and Hill, K. 1973. Genetic control of reaction to common blight bacterium in bean (*Phaseolus vulgaris*) as influenced by plant age and bacterial multiplication. J. Am. Soc. Hortic. Sci. 98(1): 94-99.

———; ———; and Shaughnessy, L. 1966. Inheritance of reaction to halo blight and common blight bacteria in a *Phaseolus vulgaris* variety cross. Plant Dis. Rep. 50(1):29-32.

Crispín-Medina, A. and Campos-Avila, J. 1976. Bean diseases of importance in Mexico in 1975. Plant Dis. Rep. 60(6):534-535.

———; Sifuentes-A., J. A.; and Campos-Avila, J. 1976. Enfermedades y plagas del frijol en México. Rev. ed. Folleto de divulgación no. 39. Instituto Nacional de Investigaciones Agrícolas, Secretaría de Agricultura y Ganadería, Mexico City, Mexico. 42 p.

Díaz-Polanco, C. 1972. Synergistic effect between *Macrophomina phaseoli* and *Xanthomonas phaseoli* on bean foliage. Phytopathology 62(1):11. (Abstr.)

Dickens, L. E. and Oshima, N. 1969. Protective sprays inhibit secondary spread of common bacterial blight in snap beans. Plant Dis. Rep. 53(8):647.

Dye, D. W. 1962. The inadequacy of the usual determinative tests for the identification of *Xanthomonas* spp. N. Z. J. Sci. 5(4):393-416.

——— and Lelliott, R. A. 1974. Genus II: *Xanthomonas Dowson* 1939, 187. In: Buchanan, R. E. and Gibbons, N. E. (eds.). Bergey's manual of determinative bacteriology. 8th ed. Bailliére, London, England. p. 243.249.

Ekpo, E. J. A. 1975. Pathogenic variation in common (*Xanthomonas phaseoli*) and fuscous (*Xanthomonas phaseoli* var. *fuscans*) bacterial blights of bean (*Phaseolus vulgaris* L.). Ph.D. dissertation. Michigan State University, East Lansing, MI, USA. 127 p.

——— and Saettler, A. W. 1976. Pathogenic variation in *Xanthomonas phaseoli* and *X. phaseoli* var. *fuscans*. Plant Dis. Rep. 60(1):80-83.

Focus on Michigan's bean industry. 1971. In: Michigan Science Action No. 16. Michigan Agricultural Experimental Station, Michigan State University, East Lansing, MI, USA. 6 p.

Goss, R. W. 1940. The relation of temperature to common and halo blight of beans. Phytopathology 30(12):258-264.

Haas, J. H. 1972. *Xanthomonas phaseoli* nonsystemic in some *Phaseolus vulgaris* cultivars. Phytopathology 62(7):761. (Abstr.)

Hayward, A. C. and Waterston, J. M. 1965a. *Xanthomonas phaseoli*. CMI (Commonw. Mycol. Inst.) Descr. Pathog. Fungi Bact. No. 48. 2 p.

——— and ———. 1965b. *Xanthomonas phaseoli* var. *fuscans*. CMI (Commonw. Mycol. Inst.) Descr. Pathog. Fungi Bact. No. 49. 1 p.

Hedges, F. 1944. Association of *Xanthomonas phaseoli* and the common bean-mosaic virus, *Marmor phaseoli*, I: effect of pathogenicity of the seed-borne infective agents. Phytopathology 34(7):662-693.

———. 1946. Experiments on the overwintering in the soil of bacteria causing leaf and pod spots of snap and lima beans. Phytopathology 36(8):677-678.

Honma, S. 1956. A bean interspecific hybrid. J. Hered. 47:217-220.

Jackson, H. R. and Wallen, V. R. 1975. Microdensitometer measurements of sequential aerial photographs of field beans infected with bacterial blight. Phytopathology 65(9):961-968.

Jindal, J. K. and Patel, P. N. 1984. Variability in xanthomonads of grain legumes, IV: variations in bacteriological properties of 83 isolates and pathogenic behavior of cultural variants. Phytopathol. Z. 110(1):63-68.

Kado, C. I. and Heskett, M. G. 1970. Selective media for isolation of *Agrobacterium, Corynebacterium, Erwinia, Pseudomonas,* and *Xanthomonas.* Phytopathology 60(6):969-976.

Kaiser, W. J. and Vakili, N. G. 1978. Insect transmission of pathogenic xanthomonads to bean and cowpea in Puerto Rico. Phytopathology 68(7):1057-1063.

Lahman, L. K. and Schaad, N. W. 1985. Evaluation of the "dome test" as a reliable assay for seedborne bacterial blight pathogens of beans. Plant Dis. 69(8):680-683.

Leach, J. G.; Lilly, V. G.; Wilson, H. A.; and Purvis, M. R., Jr. 1957. Bacterial polysaccharides: the nature and function of the exudate produced by *Xanthomonas phaseoli.* Phytopathology 47(3): 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(1):132-140.

Leben, C. and Sleesman, J. P. 1982. Preservation of plant-pathogenic bacteria on silica gel. Plant Dis. 66(4):327.

McElroy, J. B. 1985. Breeding dry beans, *Phaseolus vulgaris* L., for common bacterial blight resistance derived from *Phaseolus acutifolius* A. Gray. Ph.D. dissertation. Cornell University, Ithaca, NY, USA. 45 p.

Mack, A. R. and Wallen, V. R. 1974. Effects of various field levels of soil temperature and soil moisture on the growth of beans infected with bacterial blight. Can. J. Soil Sci. 54(2):149-158.

Malin, E. M.; Roth, D. A.; and Belden, E. L. 1983. Indirect immunofluorescent staining for detection and identification of *Xanthomonas campestris* pv. *phaseoli* in naturally infected bean seed. Plant Dis. 67(6):645-647.

Mitchell, J. W.; Zaumeyer, W. J.; and Anderson, W. P. 1952. Translocation of streptomycin in bean plants and its effect on bacterial blights. Science (Wash. DC) 115(2979):114-115.

———; ———; and 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(1):25-30.

———; ———; and ———. 1953. Movement of streptomycin in bean plants. Phytopathology 43(9):480. (Abstr.)

Mohan, S. T. 1981. Breeding dry beans (*Phaseolus vulgaris* L.) for common bacterial blight resistance: relation of days "to flowering" to blight reaction. Turrialba 31(2):109-112.

Mukunya, D. M.; Muthangya, P. M.; and Esele, J. P. 1981. Bacterial blight of beans (*Phaseolus vulgaris* L.) in Kenya. In: Lozano, J. C. (ed.). Proceedings of the 5th international conference on plant pathogenic bacteria, August 16-23, 1981, at CIAT, Cali, Colombia. Centro Internacional de Agricultura Tropical (CIAT), Cali, Colombia, and University of Missouri, Columbia, MO, USA. p. 3-8.

Orozco-Sarria, S. H. 1971. El cultivo del frijol en Colombia. Boletín de divulgación no. 2. Programa de Leguminosas de Grano y Oleaginosas Anuales, Instituto Colombiano Agropecuario (ICA), Palmira, Colombia. 22 p.

Panzer, J. D. and Nickeson, R. L. 1959. Delayed synergism of bacterial blight and bean mosaic on *Phaseolus vulgaris* L. Plant Dis. Rep. 43(2):133-136.

Pastor-Corrales, M. A.; Beebe, S. E.; and Correa, F. J. 1981. Comparing two inoculation techniques for evaluating resistance in beans to *Xanthomonas campestris* pv. *phaseoli*. In: Lozano, J. C. (ed.). Proceedings of the 5th international conference on plant pathogenic bacteria, August 16-23, 1981, at CIAT, Cali, Colombia. Centro Internacional de Agricultura Tropical (CIAT), Cali, Colombia, and University of Missouri, Columbia, MO, USA. p. 493-503.

Patel, P. N. and 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(4):407-411.

Pinstrup-Andersen, P.; Ruiz de Londoño, N.; and Infante, M. 1976. A suggested procedure for estimating yield and production losses in crops. PANS (Pest Artic. News Summ.) 22(3):359-365.

Pinto de Torres, A. 1968. Bacteriosis o tizón común de frejol en Chile. Agric. Téc. (Santiago) 29(1):14-20.

Pompeu, A. S. and Crowder, L. V. 1972. Inheritance of resistance of *Phaseolus vulgaris* L. (dry beans) to *Xanthomonas phaseoli* Dows. (common blight). Ciênc. Cult. (São Paulo) 24(11):1055-1063.

——— and ———. 1973. Methods of inoculation and bacterial concentrations of *Xanthomonas phaseoli* Dows. for the inheritance of disease reaction in *Phaseolus vulgaris* L. crosses (dry beans), under growth chamber conditions. Ciênc. Cult. (São Paulo) 25(11):1078-1081.

Sabet, K. A. and Ishag, F. 1969. Studies on the bacterial diseases of Sudan crops, VIII: survival and dissemination of *Xanthomonas phaseoli* (E. F. Sm.) Dowson. Ann. Appl. Biol. 64:65-74.

Saettler, A. W. 1971. Seedling injection as an aid in identifying bean blight bacteria. Plant Dis. Rep. 55(8):703-706.

———. 1974. Testing bean seed for internal bacterial blight contamination. Bean Improv. Coop. (USA) Annu. Rep. 17:73-74.

———. 1977. Breeding dry edible beans (*Phaseolus vulgaris* L.) for tolerance to *Xanthomonas* bacterial blights. Fitopatol. Bras. 2(3): 179-186.

———; Cafati, C. R.; and Weller, D. M. 1986. Nonoverwintering of *Xanthomonas* bean blight bacteria in Michigan. Plant Dis. 70(4):285-287.

——— and Perry, S. K. 1972. Seed-transmitted bacterial diseases in Michigan Navy (pea) beans, *Phaseolus vulgaris*. Plant Dis. Rep. 56(5):378-381.

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

Scharen, A. L. 1959. Comparative population trends of *Xanthomonas phaseoli* in susceptible, field-tolerant, and resistant hosts. Phytopathology 49:425-428.

Schieber, E. 1970. Enfermedades del frijol (*Phaseolus vulgaris*) en la República Dominicana. Turrialba 20(1):20-23.

Schuster, M. L. 1955. A method for testing resistance of beans to bacterial blights. Phytopathology 45(9):519-520.

———. 1967. Survival of bean bacterial pathogens in the field and greenhouse under different environmental conditions. Phytopathology 57(8):830. (Abstr.)

——— and Coyne, D. P. 1971. New virulent strains of *Xanthomonas phaseoli*. Plant Dis. Rep. 55(6):505-506.

——— and ———. 1974. Survival mechanisms of phytopathogenic bacteria. Annu. Rev. Phytopathol. 12:199-221.

——— and ———. 1975a. Detection of bacteria in bean seed. Bean Improv. Coop. (USA) Annu. Rep. 18:71.

——— and ———. 1975b. Genetic variation in bean bacterial pathogens. Euphytica 24(1):143-147.

——— and ———. 1975c. Survival factors of plant pathogenic bacteria. Research bulletin no. 268. Agriculture Experiment Station, University of Nebraska, Lincoln, NE, USA. 53 p.

——— and ———. 1977. Survival of plant parasitic bacteria of plants grown in tropics with emphasis on beans (*Phaseolus vulgaris*). Fitopatol. Bras. 2(2):117-130.

——— and ———. 1981. Biology, epidemiology, genetics and breeding for resistance to bacterial pathogens of *Phaseolus vulgaris* L. Hortic. Rev. 3:28-58.

———; ———; Behre, T.; and Leyna, H. 1983. Sources of *Phaseolus* species resistance and leaf and pod differential reactions to common blight. HortSci. 18(6):901-903.

———; ———; and Hoff, B. 1973. Comparative virulence of *Xanthomonas phaseoli* strains from Uganda, Colombia, and Nebraska. Plant Dis. Rep. 57(1):74-75.

Smale, B. C. and 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 Dis. Rep. 40:628.

Steadman, J. R.; Maier, C. R.; Schwartz, H. F.; and Kerr, E. D. 1975. Pollution of surface irrigation waters by plant pathogenic organisms. Water Resour. Bull. 11(4):796-804.

Sutton, M. D. and Wallen, V. R. 1970. Epidemiological and ecological relations of *Xanthomonas phaseoli* and *Xanthomonas phaseoli* var. *fuscans* on beans in southwestern Ontario, 1961-1968. Can. J. Bot. 48(7):1329-1334.

Thomas, C. V. and Waines, J. G. 1984. Fertile backcross and allotetraploid plants from crosses between tepary beans and common beans. J. Hered. 75(2):93-98.

Thomas, W. D., Jr. and Graham, R. W. 1952. Bacteria in apparently healthy pinto beans. Phytopathology 42(3):214.

Trujillo, G. E. and Saettler, A. W. 1979. A combined semi-selective medium and serology test for the detection of *Xanthomonas* blight bacteria in bean seed. J. Seed Technol. 4(2):35-41.

——— and ———. 1980. A liquid semiselective medium for *Xanthomonas phaseoli* and *X. phaseoli* var. *fuscans*. Mich. State Univ. Agric. Exp. Stn. Res. Rep. 411:8.

——— and ———. 1981. Un nuevo método para el mantenimiento de bacterias fitopatógenas que atacan el cultivo de la caraota (*Phaseolus vulgaris* L.). Rev. Fac. Agron. (Maracay) 12(1-2):23-33.

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(4):401-403.

Valladares-Sánchez, N. E.; Coyne, D. P.; and Mumm, R. F. 1983. Inheritance and associations of leaf, external, and internal pod reactions to common blight bacterium in *Phaseolus vulgaris* L. J. Am. Soc. Hortic. Sci. 108(2):272-278.

———; ———; and 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. J. Am. Soc. Hortic. Sci. 104(5):648-654.

van Rheenen, H. A.; Hasselbach, O. E.; and Muigai, S. G. S. 1981. The effect of growing beans together with maize on the incidence of bean diseases and pests. Neth. J. Plant Pathol. 87(5):193-199.

Venette, J. R. and Kennedy, B. W. 1975. Naturally produced aerosols of *Pseudomonas glycinea*. Phytopathology 65(6):737-738.

——— and Nayes, J. B. 1978. A seed test to detect internally-borne bacterial pathogens of beans. Phytopathol. News (USA) 12(8):92. (Abstr.)

Vieira, C. 1967. O feijoeiro-comum cultura, doenças e melhoramento. Universidade Rural do Estado de Minas Gerais, Viçosa, MG, Brazil. 220 p.

Wallen, V. R. and Galway, D. A. 1979. Effective management of bacterial blight of field beans in Ontario: a 10-year program. Can. J. Plant Pathol. 1:42-46.

——— and Jackson, H. R. 1975. Model for yield loss determination of bacterial blight of field beans utilizing aerial infrared photography combined with field plot studies. Phytopathology 65(9):942-948.

———; Sutton, M. D.; and Grainger, P. N. 1963. A high incidence of fuscous blight in Sanilac beans from southwestern Ontario. Plant Dis. Rep. 47(7):652-653.

Webster, D. M. 1978. Evaluation of resistance in beans (*Phaseolus vulgaris* L.) to *Xanthomonas phaseoli*. Ph.D. dissertation. University of Wisconsin, Madison, WI, USA. 117 p.

———; Atkin, J. D.; and Cross, J. E. 1983a. Bacterial blights of snap beans and their control. Plant Dis. 67(9):935-940.

———; Temple, S. R.; and Gálvez, G. E. 1983b. Expression of resistance to *Xanthomonas campestris* pv. *phaseoli* in *Phaseolus vulgaris* under tropical conditions. Plant Dis. 67(4):394-396.

———; ———; and Schwartz, H. F. 1980. Selection for resistance to *Xanthomonas phaseoli* in dry beans. Crop Sci. 20(4):519-522.

Weller, D. M. and Saettler, A. W. 1976. Chemical control of common and fuscous bacterial blights in Michigan Navy (pea) beans. Plant Dis. Rep. 60(9):793-797.

——— and ———. 1978. Rifampin-resistant *Xanthomonas phaseoli* var. *fuscans* and *Xanthomonas phaseoli*: tools for field study of bean blight bacteria. Phytopathology 68(5):778-781.

——— and ———. 1980a. Colonization and distribution of *Xanthomonas phaseoli* and *Xanthomonas phaseoli* var. *fuscans* in field-grown navy beans. Phytopathology 70(6):500-506.

——— and ———. 1980b. Evaluation of seedborne *Xanthomonas phaseoli* and *X. phaseoli* var. *fuscans* as primary inocula in bean blights. Phytopathology 70(2):148-152.

Wilson, H. A.; Lilly, V. G.; and Leach, J. G. 1965. Bacterial polysaccharides, IV: longevity of *Xanthomonas phaseoli* and *Serratia marcescens* in bacterial exudates. Phytopathology 55(10):1135-1138.

Wimalajeewa, D. L. S. and Nancarrow, R. J. 1980. Survival in soil of bacteria causing common and halo blights of French bean in Victoria, Australia. Aust. J. Exp. Agric. Anim. Husb. 20:102-104.

Wyman, J. G. and VanEtten, H. D. 1982. Isoflavonoid phytoalexins and nonhypersensitive resistance of beans to *Xanthomonas campestris* pv. *phaseoli.* Phytopathology 72(11):1419-1424.

Yoshii, K.; Gálvez, G. E.; and Alvarez-Ayala, G. 1976a. Estimation of yield losses in beans caused by common blight. Proc. Am. Phytopathol. Soc. 3:298-299. (Abstr.)

———; ———; and ———. 1976b. Highly virulent strains of *Xanthomonas phaseoli* from Colombia. Proc. Am. Phytopathol. Soc. 3:299. (Abstr.)

———; ———; and ———. 1978. Screening bean germplasm for tolerance to common blight caused by *Xanthomonas phaseoli* and the importance of pathogenic variation to varietal improvement. Plant Dis. Rep. 62(4):343-347.

Zaumeyer, W. J. and Meiners, J. P. 1975. Disease resistance in beans. Annu. Rev. Phytopathol. 13:313-334.

——— and Thomas, H. R. 1957. A monographic study of bean diseases and methods for their control. Rev. ed. Technical bulletin no. 868. United States Department of Agriculture, Washington, DC, USA. 255 p.