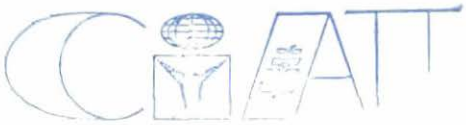

Chapter 9

Common and Fuscous Blights

Kazuhiro Yoshii

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Chapter 9

Common and Fuscous Blights

Introduction

Common blight caused by *Xanthomonas phaseoli* (E.F.Sm.) Dows. and fuscous blight caused by *X. phaseoli* var. *fuscans* (Burk.) Starr and Burk. are major bacterial diseases of dry beans. The two organisms are found frequently in association and are reported to occur in many bean production regions of the world (13, 26, 27, 47, 51, 62, 81, 92).

Yield losses due to each pathogen are difficult to estimate because their symptoms are similar. Common and fuscous blight bacteria frequently occur together in a field and probably on the same plant, increasing the difficulty of associating yield losses with a specific pathogen. In 1967, at least 75% of Michigan's 650,000 acres of Navy beans were damaged by common and fuscous blights, with 10-20% yield reductions (2).

Wallen and Jackson (82) reported a 38% yield loss in Ontario, Canada due to common and fuscous blight in two years of field trials. Aerial infrared photographic surveys suggested that losses for the bean crop grown in Ontario ranged from 1252 tons in 1970 to 218 tons in 1972 (39, 82). Yield losses estimated at 22% and 45% have been obtained by natural and artificial infections, respectively, in Colombia (88). Economic surveys, based upon field observations in the same region, estimated yield losses of 13% due to common and fuscous blight bacteria (50).

Hosts include *Phaseolus vulgaris, P. coccineus, P. mungo, P. aureus, P. acutifolius, P. aconitifolius, P. angularis, Lablab niger, Strophostyles helvula, Glycine max, Stizolobium deeringianum, Lupinus polyphyllus,* and *Vigna sinensis* (77, 92).

Common names frequently used for common bacterial blight in Latin America include bacteriosis, añublo bacterial comun, tizón comun and crestamento bacteriano.

Etiology

Laboratory isolations and purifications are necessary to distinguish the two organisms. The only significant character which distinguishes *X. phaseoli* from *X. phaseoli* var. *fuscans* is the production of a diffusible brown pigment (melanin) by the latter on a medium containing tyrosine (36). Pigment-producing isolates tend to be more virulent than those unable to produce the pigment (6); however, the pigment may not be essential for pathogenicity. Dye (30) concluded that there was little justification for separating *X. phaseoli* from *X. phaseoli* var. *fuscans*, since pigment production is common in *Xanthomonas* species not pathogenic to beans and may not even be a stable character (4).

Xanthomonas phaseoli has been described according to the following biochemical, physical and physiological characteristics: It produces single cells which are straight rods and motile by means of a polar flagellum. It is gram negative and strictly aerobic. It produces a yellow pigment due to a non-water soluble eacotenoid and a mucoid growth on nutrient glucose agar. Acid is produced as a metabolic by-product when cells grow on media containing arabinose, glucose, mannose, galactose, trehalose or cellabiose. It also causes proteolysis of milk (31).

Both organisms grow well on potato dextrose, nutrient and yeast-extract-dextrose calcium carbonate agars. The latter medium is used most commonly and consists of 10 g yeast extract, 10 g dextrose, 2.5 g calcium carbonate and 20 g agar in 1 liter distilled water (56). A relatively selective medium has been developed for isolating *Xanthomonas* sp. (40) and *X. campestris* (60), but *X. phaseoli* and *X. phaseoli* var. *fuscans* grow only in these media when mass-streaked onto the plate.

Epidemiology

X. phaseoli and *X. phaseoli* var. *fuscans* are warm temperature pathogens in contrast to *Pseudomonas phaseolicola* which is a cool temperature pathogen (34). Common and fuscous blight bacteria cause more severe damage to plants at 28°C than at lower temperatures (44, 49). *X. phaseoli* grows best *in vitro* at 28° - 32°C, and growth declines gradually as temperature is lowered. At 16°C little growth occurs. Detailed meteorological and microclimatological data are not available to determine the factors that influence development of bacterial blight epidemics. In general, however, common blight epidemics are favored by high temperature and high humidity (75).

Plant pathogenic bacteria can survive adverse environmental conditions and extended absence of host plants in the field by several means. One of the most effective means is on or within infected bean seed. Seed transmission of *X. phaseoli* has been known since 1872 (66, 69). Bacteria have been recovered from three (5), 10 (92) and 15 (71, 72)-year old bean seed. Such seed-borne isolates normally are viable and virulent when recovered from seed (56, 57, 59, 70).

Seed lots can be assayed for the presence of bacteria by incubation in water or a liquid medium which then is inoculated into susceptible plants by injection, watersoaking (67) or vacuum infiltration (80). Saettler and Perry (59) assayed 101 Navy bean seed lots for internal contamination with *X. phaseoli* and *X. phaseoli* var. *fuscans*. Approximately 35% of these were contaminated with *X. phaseoli*, 13% with *X. phaseoli* var. *fuscans* and 52% with both organisms. Wallen *et al.* (83) sampled 23 seed lots from Ontario, Canada and isolated virulent cultures of *X. phaseoli* var. *fuscans* from more than 50% of the samples. The minimum level of primary inoculum required to incite an epidemic is not known but should be determined for various cultural and environmental conditions.

Short term survival within healthy-appearing bean plants can occur during a growing season (76), and bacterial numbers can increase on symptomless leaves (86). Both *X. phaseoli* and *X. phaseoli* var. *fuscans* can survive between growing seasons in temperate zones within infested bean debris (64, 69). Survival occurs in bean debris placed on top of but not 20 cm below the soil surface, and survival is greater under dry than under moist environmental conditions. Bacteria are recovered from the soil up to six weeks after burial, but Schuster (64) speculated that survival occurred in infested plant debris.

Sutton and Wallen (75) could not isolate *X. phaseoli* from soil in which infected plants had been grown. Schuster and Coyne (70) believe that survival in the tropics may be greater than in temperate zones because of the opportunities to continually increase populations and to possibly survive as epiphytes on perennial hosts. Studies are needed to determine the extent of *X. phaseoli* and *X. phaseoli* var. *fuscans* survival in infested plant debris and soil under tropical conditions.

Although plant pathogenic bacteria do not form spores, many are tolerant to desiccation and can survive extended dry conditions *X. phaseoli* produces an extracellular polysaccharide in culture and in the host plant (42). It can survive in this exudate for prolonged periods under varied environmental conditions (87).

The bacteria obviously can be disseminated quite effectively on and within bean seed. Plants grown from infected seed frequently bear lesions on the cotyledons, nodes or primary leaves which serve as initial centers for pathogen spread during favorable environmental conditions (92). Infected seed or infested plant debris may be present within bean cull piles and can serve as initial sites for disease development (7). Infested bean straw residue present in fields can provide another locus from which bacteria may be disseminated to susceptible plants (69).

Secondary spread of common and fuscous blight bacteria is made easier with rain accompanied by wind (92), wind-blown soils (11), possibly by irrigation water (74), and by insects such as the white fly (55). Common and fuscous blight bacteria can survive on the bodies of insects and be transmitted to wounds caused by leaf-feeding insects such as *Diaprepes abbreviata* and *Cerotoma ruficornis* (41). Some bacterial pathogens such as *Pseudomonas glycinea* are spread within aerosols (79), but this has not been reported for *X. phaseoli* or *X. phaseoli* var. *fuscans*.

Plant Infection and Symptomatology

Xanthomonas phaseoli and *X. phaseoli* var. *fuscans* induce identical symptoms on leaves, stems, pods and seeds. Leaf symptoms initially appear as water-soaked spots on the undersides of leaves or leaflets (Fig. 1). These spots then enlarge irregularly, and adjacent lesions frequently coalesce. Infected regions appear flaccid, are encircled by a narrow zone of lemon-yellow tissue, later turn brown and necrotic (Fig. 2) and may be so extensive (Fig. 3) as to cause defoliation or stem girdle (92).

Blight bacteria enter leaves through natural openings such as stomata and hydathodes, or through wounds (92). The bacteria then invade intercellular spaces, causing a gradual dissolution of the middle lamella. The bacteria may enter the stem through stomata of the hypocotyl and

Fig. 1- Water-soaked spots caused by leaf infection of common and fuscous blight.

Fig. 2- Common blight lesions showing lemon yellow and necrotic symptoms.

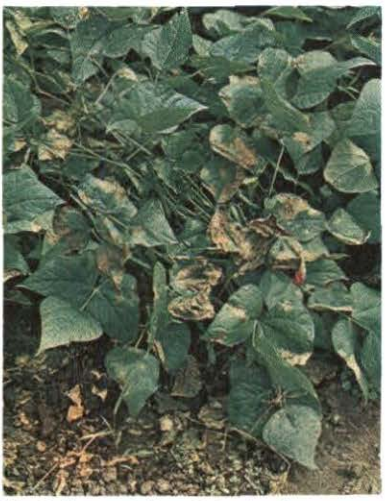

Fig. 3- Severe foliage infection by common bacterial blight.

Fig. 4- Stem girdle and breakage caused by common bacterial blight.

epicotyl and reach vascular elements from infected leaves or cotyledons. Presence of a sufficient number of bacteria in the xylem tissue may cause plant wilting by plugging the vessels or disintegration of the cell walls. *X. phaseoli* does not induce systemic infection in all *Phaseolus vulgaris* cultivars (35). Stem girdle or joint rot may develop at the cotyledonary node, especially in plants that originated from infected seed, and cause the plant to break (92) (Fig. 4).

Pod lesions appear as water-soaked spots which gradually enlarge, become dark and red and slightly sunken. If infection occurs during pod and seed development, infected seed may rot or shrivel (Fig. 5). Common and fuscous blight bacteria are harbored both within the seed and on the seed coat. They enter pod sutures from the vascular system of the pedicel 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, but it may occur. If bacteria enter through the funiculus, only the hilum may become discolored. Seed infection is difficult to see when seeds are dark in color,

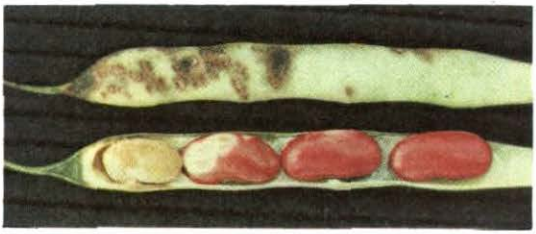

Fig. 5- Pod and seed infection by common bacterial blight.

but it is evident as butter-yellow spots on white or light-colored seeds (59, 92). Seedlings which develop from infected seed may sustain damage to the growing tip and be killed (snake head) or stunted (92).

A bean plant may be more susceptible to infection by common blight bacteria if previously infected by another pathogen. Panzer and Nickeson (48) demonstrated that common blight is more severe in the presence of bean common mosaic virus, particularly late in the season. Hedges (37) found that the virus persisted in cultures of *X. phaseoli* for six weeks. Diaz Polanco (28) also showed that a synergistic effect existed between *X. phaseoli* and *Macrophomina phaseolina* infection of bean leaves.

Symptoms of *X. phaseoli* are not significantly different from those caused by *X. phaseoli* var. *fuscans*. Zaumeyer and Thomas (92) observed that *X. phaseoli* var. *fuscans* may cause a slight hypertrophy and darkening of the stem at the point of artificial inoculation of young seedlings. Severe plant symptoms can occur from inoculations of fuscous blight bacteria (33, 92). However, inoculations with mixed inocula of fuscous and common blight bacteria can induce more severe symptoms than observed with individual inoculations (32).

Control by Cultural Practices

Cultural practices often utilized to reduce common blight are the use of pathogen-free seed, proper crop rotation and deep plowing (92). Clean or certified seed can be produced in a region free of the pathogen or where environmental conditions are unfavorable for disease development. Crop rotation with plants not susceptible to blight can reduce or eliminate blight bacteria in bean debris within a field. Such recommendations can, however, prove difficult for Latin American producers with small land holdings and limited economic resources.

Control by Chemicals

Various chemicals have been applied as a seed treatment or foliage protectant to control common blight before moderate to severe infection is apparent. They have controlled foliage infection effectively, although yield increases have been minimal. Such compounds include basic Copper Sulfate (29), Copper Hydroxide and potassium (hydroxymethyl) methyldithiocarbamate or Bunema (85). Streptomycin has given marginal control in the laboratory and field and is translocated within the plant but not into developing seeds (45, 46, 54). However, antibiotics should not be foliarly applied since resistant bacterial mutants may be induced.

Control by Plant Resistance

Isolates of *X. phaseoli* have been shown to differ in virulence within and between geographical locations from which they were collected (68). Schuster and Coyne (65) found isolates from infected bean seed from Colombia were much more virulent than standard North American isolates. Other isolates from Uganda were found to be about as virulent as the Colombian isolates (72). Isolates with even greater virulence have since been identified (33, 89). However, these differences may be complicated by variations in inoculation methods, age of isolates, and other factors. Differences in pathogenicity also can exist between sub-isolates taken from individual stock cultures of *X. phaseoli* (12, 73). Pathogenic variation occurs also in *X. phaseoli* var. *fuscans* isolates (33).

Various methods of inoculation have been used and include:

- sticking the cotyledon or cotyledonary node with a needle or scalpel dipped in inoculum (3, 8)
- rubbing the second trifoliate leaves with a cotton swab soaked with a carborundum-inoculum mixture (12)
- soaking leaves with water-inoculation at high pressure (3, 63)
- using vacuum infiltration on leaves (80)
- pricking leaves with a multiple needle cushion (1, 53)
- clipping leaves with scissors dipped in inoculum (32, 84).

Inoculum concentrations can influence the disease reaction. Optimum concentrations are reported to be in the range between 10^7 to 10^8 cells/ml (24, 32, 53).

Phaseolus vulgaris cultivars and breeding materials have been noted to vary in their reaction to infection by common and fuscous blight bacteria (Fig. 6). Immunity to infection has not been found, but many lines are resistant (referred to as tolerant by earlier workers) to infection, with little if any yield loss. However, bacteria can survive in this resistant tissue

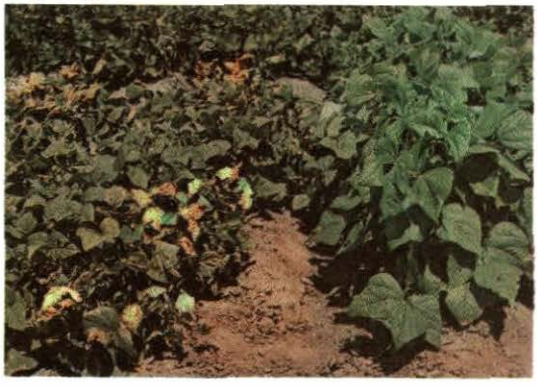

Fig. 6- Variation shown by *Phaseolus vulgaris* germplasm for its resistance to infection by common blight bacteria (susceptible left, resistant right).

without inciting disease symptoms (61). In general, beans are more susceptible to infection after the start of the blossoming or reproductive stage of plant development (17, 20, 24). Most workers inoculate plants during flowering and evaluate three to four weeks later. However, inoculations at three to four weeks after planting may be more effective in the tropics if germplasm is quite variable for its maturity, growth habit and adaptation (10, 84). In addition, Coyne and Schuster (18) reported a differential leaf and pod reaction to infection by *X. phaseoli* which was conferred by different genes. Therefore, time of evaluation and disease rating scales must be designed carefully to account for the previously mentioned factors (58).

Schuster (63) first reported that *Phaseolus acutifolius* (tepary bean) was resistant to *X. phaseoli*. Honma (38) then used the tepary bean as a source of resistance to incorporate in *Phaseolus vulgaris*. Coyne and co-workers (16, 22) surveyed more than 1,000 plant introduction (P.I.) lines for resistance to common and fuscous blight infection in the field.

The following *Phaseolus vulgaris* lines and cultivars had a high degree of resistance: 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-Guali (accessions from Colombia) and Great Northern (G.N.) Nebraska No. 1 selection 27. Yoshii *et al.* (90) reported that P.I. 282086 and P.I. 313343 had resistant foliage, but the former had susceptible pods. *P. acutifolius* "Tepary Buff" (16) and P.I. 169932 (90) had high degrees of resistance with no symptoms observed. Some *P. coccineus* lines also were quite resistant, but less so than Tepary (16).

These resistant materials have been tested at various locations and exposed to more virulent bacterial isolates than originally used. Thus, while G.N. Nebraska No. 1 selection 27 and P.I. 207262 also were resistant to Brazilian isolates of *X. phaseoli* and *X. phaseoli* var. *fuscans* (9), the former was susceptible to Colombian and Ugandan isolates of *X. phaseoli* (65, 71). P.I. 207262 also was susceptible to a Colombian isolate of *X. phaseoli* and moderately susceptible to some *X. phaseoli* var. *fuscans* isolates (33). Poor plant adaptation to tropical growing conditions in Colombia inhibited the expression of resistance by Jules and P.I. 207262 (10, 84), until their resistance was transferred to agronomically adapted and susceptible backgrounds.

Inheritance of common blight resistance recently has been reviewed (17, 43, 91). Honma (38) made the interspecific cross between resistant *Phaseolus acutifolius* "Tepary 4" and susceptible *P. vulgaris* and found that resistance was quantitatively inherited. Coyne *et al.* (23) further studied inheritance of the resistant selections crossed 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 (24).

The late-maturing G.N. Tara and Jules (14, 15) and early-maturing Valley (19) cultivars were derived from the cross with G.N. 1140. They possess resistance to common blight in temperate regions of the United States. G.N. Starr in an early maturing cultivar derived from six backcrosses of P.I. 165078 (tolerant to *Corynebacterium flaccumfaciens*) to G.N. Nebraska #1 sel. 27 (tolerant to *X. phaseoli),* resulting in resistance to both bacterial pathogens (21). Coyne *et al.* (24, 25) report that the cross between G.N. 1140 and G.N. Nebraska #1 sel. 27 exhibited partial dominance for susceptibility. This inheritance also was reported by Pompeu and Crowder (52) for similar crosses between G.N. Nebraska #1 sel. 27 and 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 (20). Transgressive segregation has been observed in these crosses (24, 25, 52, 78) and should allow breeders to increase the levels of resistance within promising germplasm.

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