Chapter 10

Miscellaneous Bacterial Diseases

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Chapter 10

Halo Blight

Introduction

Halo blight of beans is caused by the bacterium *Pseudomonas phaseolicola* (Burk.) Dows. The bacterium is distributed worldwide and is found in many regions of Latin America with moderate temperatures, such as southern Chile and Brazil (6, 20). Yield losses of 23-43% have occurred in research fields in Michigan (63). The pathogen can infect various plant species including *Phaseolus acutifolius, P. angularis, P. bracteatus, P. coccineus, P. lunatus, P. polyanthus, P. polystachyus, P. radiatus, P. vulgaris, Glycine max, Pueraria hirsuta* and *P. thunbergiana* (82, 91).

Common names frequently used for halo blight in Latin America include añublo de halo, mancha de halo, tizón de halo, hielo amarillo, crestamento bacteriano aureolado, crestamento bacteriano de halo and mancha aureolada.

Etiology

Pseudomonas phaseolicola exhibits the following characteristics: Cells are single, straight rods which are motile due to multitrichous flagella. The bacterium is gram negative, strictly aerobic and does not require growth factors. Poly-B-hydroxybutyrate is not accumulated as an intracellular carbon reserve. Cultures produce diffusable fluorescent pigments, particularly in iron-deficient media. Arginine dihydrolase is absent (19). The bacterium does not utilize glutarate, meso-tartrate, DL-glycerate, isoascorbate, betaive, erythritol, sorbitol, meso-inositol or N-caproate. It does utilize D-gluconate, L (+) arabinose, sucrose, succinate, DL- β -OH butyrate, transaconitate, L-serine, L-alanine and p-hydroxybenzoate (44, 64).

The optimum growth temperature is 20°- 23°C, and the bacterium produces white to cream colonies on agar with a bluish hue which may be accompanied by a green fluorescent pigment (86).

Bacterial cells can survive liquid nitrogen storage at -172°C for 30 months with no alteration of pathogenicity (46).

Epidemiology

P. phaseolicola survives in infected seeds and plant residue on the soil surface until environmental conditions become favorable for infection (71). *P. phaseolicola* survived for nine months after passage through sheep which consumed infested plant debris (77). The pathogen enters plants through wounds or stomata during periods of high relative humidity or free moisture (63, 83, 91). Light intensity may influence the plant and the nature of its response to *P. phaseolicola* (39).

P. phaseolicola multiplies rapidly on or near the surface of lesions in the presence of dew. It is disseminated between leaves and plants by splash dispersal and winds during periods of rainfall. The bacterium has tremendous disease potential, since a dozen infected seeds per hectare, distributed at random, are sufficient to start a general epidemic under favorable environmental conditions (83). Halo blight incidence was observed to be lower in bean/maize association than in bean monoculture (40). This implies that the maize may have served as a physical barrier to bacterium spread throughout the associated cropping.

Halo blight symptoms may develop in six to 10 days at 24° - 28°C, and may be delayed two or three days at higher temperatures (91). Halo expression is more common at 16° - 20°C than at 24° - 28°C (50). Halo symptoms usually do not develop above 28°C, although small and numerous water-soaked lesions still may be evident (91).

Symptomatology

Three to five days after infection, small water-soaked spots appear, generally on the lower leaf surface (48). A halo of greenish-yellow tissue appears later around the perimeter of this water-soaked area (Fig. 1). The

Fig. 1- Symptoms of halo blight infection on leaves.

Fig. 2- Severe plant infection during a halo blight epidemic.

stem and pods also may become infected during a severe epidemic (Fig. 2) and produce typical greasy spot symptoms (Fig. 3). When infection occurs throughout the vascular system, tissue adjacent to veins and especially branches appears water-soaked and has a reddish discoloration. Stem girdle or joint rot occurs at nodes above the cotyledons when infection originates from contaminated seed. Infected pods commonly exhibit brown or red water-soaked spots, and developing seed may rot or be shrivelled and discolored (91). Water-soaked lesions can appear three days after inoculation of detached pods placed in water or nutrient solution (55).

Zaumeyer and Thomas (91) report a snakehead symptom, in which injury or destruction of the growing tip may appear after infected seed is planted. Regardless of the plant part infected, it is common to observe a light cream or silver-colored exudate produced by the pathogen at lesion sites (Fig. 4).

Fig. 3-Greasy spot symptom produced by halo blight infection on pods.

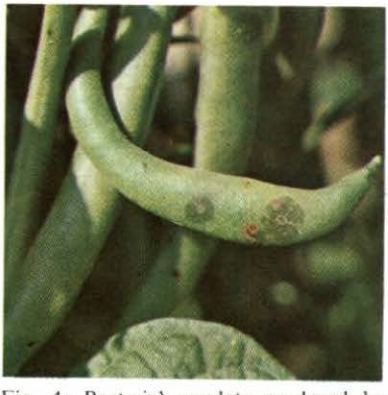

Fig. 4- Bacterial exudate produced by *Pseudomonas phaseolicola.*

Fig. 5- Systemic plant chlorosis caused by halo blight bacterial infection.

Systemic plant chlorosis with leaf yellowing and malformation (Fig. 5) also may develop without much external infection (90). Hildebrand and Schroth (35) have isolated *P. phaseolicola* from such leaves. This systemic chlorosis is more pronounced and uniform at about 20°C (9, 91). This and the typical halo symptom are due to a non host-specific toxin produced by the bacterium during infection (14, 38, 82). This toxin has been identified as phaseolotoxin, which has the main functional phytotoxin called Nδ-Phosphosulfamylornithine (45).

Patil *et al.* (54) found an ultraviolet-induced mutant which was unable to produce toxin, and neither induced typical halos nor invaded the plant systemically. Subsequent tests have confirmed that toxin production is necessary for pathogenicity (22). The toxin may suppress production of antibacterial phytoalexins such as phaseollin, phaseollinisoflavan, coumestrol and kievitone (23). Also there is a buildup of methionine in the halo region, and Patel and Walker (50) suggest that the toxin interferes with the urea cycle. Ammonia production has been associated with the plant reaction to toxin production by the bacterium (47), but researchers do not agree on whether it plays a major role in the plant's response to infection. *P. phaseolicola* is known to produce hemicellulases which degrade host cell wall materials during pathogenesis (42).

Lesion size may be increased by prior infection from the rust fungus, *Uromyces phaseoli* (89). Lesion numbers also have been increased by inoculation with a mixture of *P. phaseolicola* and *Achromobacter* sp. (43).

Control by Cultural Practices

Since the pathogen survives between growing seasons in bean tissue on the soil surface (71), deep plowing and crop rotation are advocated to reduce initial inoculum pressure (91). It also is advisable to remove infested debris (sanitation) from fields in Latin America. Walker and Patel (83) report there is no evidence that halo blight is spread by cultivation equipment in infected bean fields in temperate zones. However, movement through infected beans in fields should be delayed until free moisture has dried from the foliage.

The use of pathogen-free seed grown under conditions unfavorable to the organism is important in reducing the amount of initial inoculum within a field (91). Because seed can be contaminated by any bacteria present in powdered plant tissue (25, 27), such dust should be removed from the seed by thorough cleaning after threshing. Contaminated seed also can be treated with chemicals or antibiotics to destroy bacteria present on the seed coat surface (28, 59, 91), but it is seldom effective against internally-borne bacteria.

While current technology does not eradicate bacteria inside the seed coat or embryo, contaminated seed may be identified by exposure to ultraviolet light when a bluish-white fluorescence is evident. Wharton (88) reported that 20% of seeds exhibiting fluorescence contained *P. phaseolicola,* while 1% of non-fluorescing seeds contained the bacterium. Since other organisms can elicit this response, Parker and Dean (49) stated that this test was not definitive but could identify potentially contaminated seed lots which then could be evaluated using more critical and specific laboratory procedures.

In the United States, clean seed production is a major method to control halo blight. Clean seed production in Idaho depends upon:

- field inspection for visible evidence of infection
- laboratory inoculation of susceptible pods with preparations from seed lots
- serological evaluation of seed-borne microorganisms
- establishment of quarantines to prevent importation of bean seed from areas where the pathogen exists (4, 5).

If the bacterium is detected in a seed lot, the seed is not certified and hence is not planted by progressive growers. Despite such precautions, irrigation practices and/or environmental conditions in the region can favor pathogen development and epidemics occurred during 1963-1967 (3, 5).

Control by Chemicals

Halo blight has been controlled chemically using Bordeaux Mixture, Copper Oxychloride, Copper Sulfate, Cupric Oxide, Streptomycin Sulfate, and Dihydrostreptomycin Sulfate (33, 56, 78, 91). However, control may not always be effective or practical. Such chemicals are applied by ground or aerial spray equipment on a weekly or biweekly basis at the rate of 200-400 g/1000 m^2, or at first flower and pod set at the rate of 0.1% a.i./675 l/ha, to prevent spread and development of halo blight on leaves and pods (33, 63, 78).

Ralph (56) reported that a 0.2% Streptomycin soak for two hours eliminated transmission of halo blight bacteria in contaminated seed but reduced plant emergence more than 20% of that obtained from water-soaked controls. Hagedorn (28) found that Streptomycin seed treatment was not always beneficial, although the chemical appeared to afford some residual protection against subsequent plant *infection*. Taylor and Dudley (79) reduced 98% of the primary infection from infected seed when it was slurry-treated with Streptomycin (2.5 g a.i./kg seed) or Kasugamycin (0.25 g a.i./kg seed). Streptomycin-resistant mutants have been obtained *in vitro* but often were not pathogenic or did not survive in bean tissue (59).

Control by Plant Resistance

Pathogenic variation occurs in *P. phaseolicola* populations (39, 65, 71, 72) with two major race groups identified (51). All isolates tested had a similar rate of multiplication regardless of their race designation (22). Variation in virulence of strains belonging to either race is attributed to differences in the rate at which they produce toxin (39, 53, 59). Many workers feel the race designation is not valid (65, 71). In addition, serological tests indicated that *P. phaseolicola* antiserum is not race specific (26). Schuster and Coyne (71) report that more virulent strains of *P. phaseolicola* are better adapted for survival than less virulent strains.

Various inoculation methods have been used. They include partial vacuum inoculation of seeds (24), atomizing and watersoaking leaves at 15 p.s.i. in the glasshouse and 150 p.s.i. in the field (50, 66, 67), and rubbing leaves with inoculum-carborundum (39). Inoculum concentrations of 10^6-10^7 cells/ml have been used (67).

Plant resistance to *P. phaseolicola* is well known. It encompasses specific and general resistance (referred to as tolerance by earlier workers) mechanisms to both race groups or strains which vary greatly for their virulence. In general, older plants are more resistant to infection (48, 50, 52, 91). Bacteria are known to multiply in the xylem of susceptible and

resistant plants (48). Hubbeling (39) stated that field resistance may occur when there is a reduced rate of bacterial multiplication in vascular tissue and a necrotic response of parenchymatous tissue or meristems to the bacterial toxin. No qualitative differences exist between the free amino acid content present in uninfected susceptible plants and those which are resistant (50).

Independent genes govern resistance for the leaf, pod and plant systemic chlorotic reactions (2, 9, 13, 14). Pod susceptibility may occur frequently in plants which possess leaf resistance. Linkage has been detected between different genes controlling the leaf and plant systemic chlorotic reactions (14, 36). Russell (60) reports that resistance to the halo blight bacterium encompasses two phenomena: resistance to growth of the bacterial cells *in vivo*, and resistance to toxin production.

Bean germplasm has been identified which is resistant to races 1 and 2 in field and greenhouse tests. Resistance to both races exists in Great Northern (G.N.) Nebraska #1 selection 27, G.N. #16, California Small White 59, FM 51, FM-1 Blue Lake, a Nebraska selection from P.I. 150414, P.I. 203958 and OSU 10183 (2, 9, 13, 36, 80, 84). Red Mexican U.I. 3, 34 and 35 are resistant to race 1 (39).

Schuster (66) reported that Arikara Yellow and Mexican Red conferred one or two homozygous recessive genes for resistance in progeny depending upon which susceptible parent was used. Patel and Walker (52) report that P.I. 150414 possesses recessive resistance to races 1 and 2, and that Red Mexican is dominantly resistant to race 1. Hill *et al.* (36) showed that P.I. 150414 and G.N. Nebraska #1 selection 27 contain the same dominant allele responsible for resistance to race 1 but different genes control the reaction to race 2.

Coyne *et al.* (12) proposed a breeding scheme based upon a backcross and sib-cross design to combine resistance to *P. phaseolicola* (qualitative inheritance) and *Xanthomonas phaseoli* (quantitative inheritance). Hagedorn *et al.* (34) recently developed Wis. HBR 40 and 72 which are resistant to race 1 and 2 of halo blight. In addition, Wis. BBSR 130 is resistant to both races of halo blight, to common blight, to bacterial brown spot and to various fungal pathogens (31). Coyne and Schuster (9) stress that it is important to select germplasm which has a resistant pod, leaf and non-systemic plant reaction.

Successful and long-term control of *P. phaseolicola* in Latin America will require bean production regions to adopt integrated control programs. A combination of field sanitation of infested plant debris, crop rotation,

planting clean seed, progressive cultural practices, limited use of chemical applications and greater reliance upon resistant cultivars should allow growers to realize higher yields from their crop.

Bacterial Wilt

Introduction

Bacterial wilt of beans is caused by the bacterium *Corynebacterium flaccumfaciens* (Hedges) Dows. Zaumeyer and Thomas (91) report that the pathogen can cause severe losses in the United States, but its presence and importance in Latin America are unknown.

Hosts include *Phaseolus angularis, P. aureus, P. coccineus, P. lunatus* f. *macrocarpus, P. mungo, P. vulgaris, Lablab niger, Glycine max, Vigna sesquipedalis* and *V. sinensis* (91). Common names frequently used for bacterial wilt in Latin America are marchitamiento bacterial and marchitez bacterial.

Etiology

Corynebacterium flaccumfaciens exhibits the following characteristics: Cells are slightly curved rods with some straight rods and wedge-shaped forms. The bacterium is gram positive, strictly aerobic and motile by one or rarely two or three polar or subpolar flagella. The bacterium also causes hydrolysis of esculin (17).

The optimum growth temperature is 37°C. The bacterium produces yellow or orange, smooth, wet and shiny agar colonies (86). Pathogenic strains of this bacterium include an orange-colored isolate, *C. flaccumfaciens* var. *aurantiacum* Schuster and Christiansen (69, 75) and a purple-colored isolate, *C. flaccumfaciens* var. *violaceum* Schuster, Vidaver and Mandel (74, 76).

Epidemiology

Disease development is favored by temperatures above 32°C and stress conditions such as dry weather (16). Spread of the pathogen is similar to that for common and halo blight bacteria and is aided by irrigation water and rain-hail storms (91) in association with plant wounds (58).

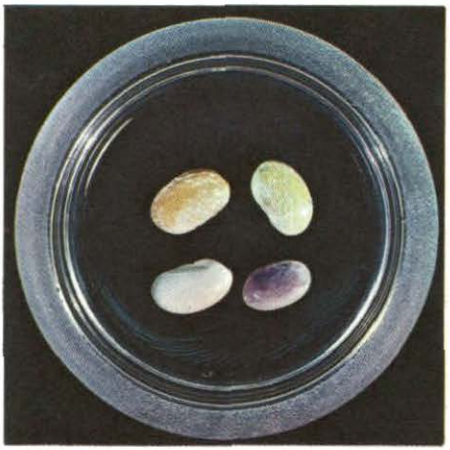

Fig. 6- Seed discoloration due to bacterial wilt infection by different pathogenic strains.

C. flaccumfaciens is seed-borne and can survive five to 24 years in infected seed, which may have yellow, orange or blue discoloration (69, 70, 74, 91) (Fig. 6). The bacterium does not overwinter well in soil but can survive between growing seasons in plant debris or on weeds. More virulent strains are better adapted for survival (71).

Symptomatology

C. flaccumfaciens is a vascular parasite which infects plants through infected seed and wounds on aerial plant organs (14, 58, 85, 91), or root wounds caused by nematode feeding or cultivation damage (68). The rate and degree of plant infection depends upon the point of entry and the stage of plant growth. Young plants are particularly susceptible and systemic development occurs rapidly once the bacteria reach the vascular system in the stem or petiole (58).

The initial symptom of infection by the wilt bacterium occurs during the warmest part of the day when leaves appear flaccid and hang limply. These leaves may regain their turgidity during periods of high moisture and low temperature but usually will turn brown, with subsequent plant wilt and death. The wilting is caused by obstruction of the vascular bundles filled with multiplying bacteria. Golden yellow necrotic leaf lesions which resemble those caused by common blight bacteria may develop but the lesion margins are more irregular (85, 91).

Although *C. flaccumfaciens* may enter the plant through stomata (73, 74), little water-soaking occurs. This contrasts with common blight *(Xanthomonas phaseoli* and *X. phaseoli* var. *fuscans)* and halo blight *(Pseudomonas phaseolicola)* bacteria, which normally penetrate through stomata and primarily invade parenchymatous tissue (91).

Control by Cultural Practices

General control recommendations have included planting pathogen-free seed and crop rotation (85, 91) which, however, are relatively ineffective because of the pathogen's ability to survive in plant debris or on weeds.

Schuster *et al.* (75) demonstrated that bacteria survive and multiply in resistant plants and could be transmitted via infected seed of certain resistant cultivars. Microorganisms borne on resistant cultivars could be disseminated to susceptible materials grown nearby, indicating the need for clean seed, even in cultivars presumed resistant to bacterial infection.

Control by Plant Resistance

Germplasm has been identified which is resistant to *C. flaccumfaciens* (11, 16), and include the following accessions: P.I. 136677, P.I. 136725, P.I. 165078, P.I. 177510, P.I. 204600 *(Phaseolus vulgaris),* P.I. 165421, P.I. 181790 *(P. coccineus),* P.I. 213014, P.I. 214332 *(P. acutifolius),* P.I. 247686 *(P. calcaratus),* as well as accessions of *P. aureus, P. bracteatus, P. lathyroides* and *P. mungo.* P.I. 247686 had no symptoms after inoculation. Although workers have observed that the xylem vessels of resistant germplasm are larger than those of susceptible selections (12, 90), Coyne and co-workers concluded that xylem size is not correlated with resistance.

Inoculation methods have included: removal of the cotyledon and insertion of a needle tip coated with inoculum into the stem at the point of cotyledonary attachment (9), petiole inoculation (58), and partial vacuum inoculation of seeds (24).

Inheritance of bacterial wilt resistance has been studied by Coyne and co-workers (15, 16). Resistant G.N. Star was derived from the cross between P.I. 165078 (resistant accession from Turkey) and susceptible Great Northern Nebraska #1 selection 27 (10). Susceptibility was conferred by two complementary dominant genes, and the absence of either one or both resulted in resistance. Susceptibility also was dominant in a cross between P.I. 136725 (resistant accession from Canada) and susceptible G.N. 1140. In a cross between P.I. 165078 and G.N. 1140, resistance was quantitatively inherited. The degree of resistance varies between germplasm sources, since P.I. 136725 is less resistant than P.I. 165078, especially at high temperatures. P.I. 165078 was crossed with G.N. 1140 to produce the resistant cultivar Emerson (8), which has been used for commercial production of Great Northern beans.

Bacterial Brown Spot

Introduction

Bacterial brown spot of beans is caused by *Pseudomonas syringae* van Hall. The pathogen can be serious in the United States (29, 53), and Robbs reports that it occurs in Brazil (6). However, no estimates are available for losses in Latin America. This bacterial pathogen has an extremely wide host range which includes *Phaseolus vulgaris, P. lunatus, Lablab niger, Glycine max, Pueraria hirsuta, Vicia faba, Vigna sesquipedalis* and *V. sinensis* (91).

Common names frequently used for bacterial brown spot in Latin America are mancha bacteriana and punto café bacterial.

Etiology

Pseudomonas syringae exhibits the following characteristics: Cells are single, straight rods, motile by means of multitrichous flagella. The bacterium is gram negative, strictly aerobic, and does not require growth factors. Poly-β-hydroxybutyrate is not accumulated as an intracellular carbon reserve. Cultures produce diffusable fluorescent pigments, particularly in iron-deficient media. Arginine dihydrolase is absent (19). The bacterium utilizes D-gluconate, glutarate, meso-tartrate, DL-glycerate, isoascorbate, betaive, sorbitol, meso-inositol, sucrose, N-caproate, N-capryllate, N-caprate, DL-β-hydroxybutyrate, citrate, glycerol and L-proline (44, 64).

The optimum growth temperature is 28° - 30°C, and the bacterium produces white, convex and transparent colonies on agar with a green fluorescent pigment (86).

Epidemiology

The bacterium has a wide host range but only isolates from beans are highly virulent to beans (62). Bean isolates can infect other crops such as peas *(Pisum sativum),* especially when grown in fields with a history of bean infection (29, 53). The bacterium can survive and multiply on weeds such as hairy vetch and provide initial inoculum sources to infect beans, especially during rainstorms (21). *P. syringae* can undergo an epiphytic-

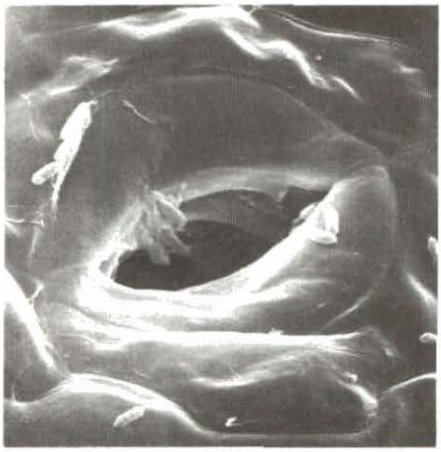

Fig. 7- Scanning electron microscope photo of *Pseudomonas syringae* cells by a plant stomata (5000x).

resident phase during which it can survive and multiply even on leaves (Fig. 7) and buds of healthy bean plants (41). It also can survive in plant residue (71). Infection by, and spread of, the pathogen is favored by sprinkler irrigation practices (29, 37, 53).

Symptomatology

P. syringae produces flecks or necrotic brown lesions of varying size which may (7) or may not (53) be surrounded by a yellow zone (Fig. 8). No macroscopically obvious water-soaked tissue or bacterial exudate is produced in these lesions, according to Patel *et al.* (53); however, other workers observed watersoaked lesions (87). The pathogen can become systemic and cause stem lesions (91). Patel *et al.* (53) observed that pods from field-infected plants could be bent or twisted (Fig. 9), and Zaumeyer

Fig. 8- (above) Symptoms of leaf infection by the brown spot organism.

Fig. 9- (right) Twisted pod symptom caused by *Pseudomonas syringae* infection.

and Thomas (91) report that ring spots may form on infected pods. Older plants generally are more resistant (91), but plants at the sixth or seventh trifoliate leaf stage can be inoculated in the field (7). Plants can be inoculated successfully in the greenhouse even under low moisture conditions (61).

Control by Chemicals

Hagedorn *et al.* (33) report that various chemicals, such as Copper Sulfate or Copper Hydroxide (86% Cupric Hydroxide with 56% metallic copper), can be applied at the rate of 200-400 g/1000 m^2 to control foliage and pod lesions. This control required weekly sprays after emergence of the first trifoliate and resulted in a significant yield response only during severe epidemics.

Control by Plant Resistance

Phaseolus germplasm observed to be resistant to infection by *P. syringae* includes Tempo, G.N. 1140 (7), Wis. BBSR 130 (31), WBR 133 (18), Earliwax, P.I. 186497, P.I. 326353, P.I. 326419, P.I. 339377 (32), P.I. 313234, P.I. 313390, P.I. 313416, P.I. 313297 and P.I. 313404 (1).

Inoculation methods have included dusting seeds with pulverized infected tissue (32) and spraying at 15 p.s.i. in the glasshouse and 150 p.s.i. in the field (7, 61). An inoculum concentration of 10^6 cells/ml has been used (7).

The resistance of WBR 133 appears to be recessive and possibly polygenic (30). Pod resistance of WBR 133 was greater at low than at high inoculum concentrations, and resistance was adversely affected by increased soil moisture (18). Wisconsin (BBSR) 130 was derived from a cross between a resistant selection from P.I. 313537 and susceptible Slimgreen. It is resistant to bacterial brown spot, common bacterial blight, halo blight, bean common mosaic virus, race gamma of anthracnose, two rust races, and Fusarium Yellows (31). These and other germplasm sources should provide useful levels of resistance that can be incorporated effectively within commercially acceptable cultivars.

Miscellaneous Bacterial Pathogens

Other bacteria are reported to be pathogens of beans *(Phaseolus* spp.) but are not discussed in this book. These organisms are listed in Table 1. Little, if any, information exists in bean literature concerning their economic importance, distribution, symptomatology, epidemiology and control measures.

Table 1. **Miscellaneous bacterial pathogens of beans.**

Pathogen	Disease	Literature Cited
Agrobacterium tumefaciens (E.F. Sm. & Towns.) Conn.	Crown Gall	81
Bacillus lathyri Manns. & Taub.	Streak	91
Corynebacterium fascians (Tilford) Dows.	Gall	91
Erwinia carotovora (L.R. Jones) Holland	Market Disease	81
Pseudomonas aptata (Brown & Jameson) F.W. Stevens	Leaf Spot	91
Pseudomonas coadunata (Wright) Chester	Market Disease	81
Pseudomonas ovalis (Ravenal) Chester	Market Disease	81
Pseudomonas solanacearum E.F. Sm.	Brown Rot	81
Pseudomonas tabaci (Wolf & Foster) F.L. Stevens	Wildfire	57
Pseudomonas viridiflava (Burk.) Clara	Gall Blight	91
Xanthomonas phaseoli var. *sojense*	Bacterial Pustule	73
Xanthomonas phaseoli f. sp. *vignicola*	Leaf Blight	73

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