Chapter 13

ADDITIONAL BACTERIAL DISEASES

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Bacterial Wilt

Introduction

Bacterial wilt of beans is caused by the bacterium *Corynebacterium flaccumfaciens* ssp. *flaccumfaciens* (Hedges) Dows. Recent chemotaxonomic studies (Collins and Jones, 1983) support the transfer of this bacterium to the genus *Curtobacterium*. Zaumeyer and Thomas (1957) report that the pathogen can cause severe losses in United States, but its occurrence and importance in Latin America are unknown.

Hosts include *Vigna angularis* (Willd.) Ohwi *et* Ohasi, scarlet runner bean (*P. coccineus* L.), big lima bean (*P. lunatus* f. *macrocarpus*), common bean (*P. vulgaris* L.), *Lablab purpureus* (L.) Sweet, soybean (*Glycine max* (L.) Merrill), *Vigna unguiculata* ssp. *unguiculata* var. *sesquipedalis* (L.) Verdc., mung bean (*V. radiata* (L.) Wilczek var. *radiata*), urd bean (*V. mungo* (L.) Hepper), and cowpea (*V. unguiculata* (L.) Walp. ssp. *unguiculata*) (Dye and Kemp, 1977; Zaumeyer and Thomas, 1957). Common names frequently used for bacterial wilt in Latin America are "marchitamiento bacterial," "marchitez bacterial," and "murcha bacteriana."

Etiology

Corynebacterium flaccumfaciens ssp. *flaccumfaciens* exhibits the following characteristics: cells are slightly curved rods with

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some straight and some wedge shaped. 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 (Cummins et al., 1974).

The optimal temperature for growth is 37 °C. The bacterium develops visible colonies in 48 hours or more. The colonies are yellow or orange, smooth, wet, and shiny (Dye and Kemp, 1977; Weber, 1973). Pathogenic strains of this bacterium include orange (Schuster and Christiansen, 1957; Schuster et al., 1964) and purple (Schuster and Sayre, 1967; Schuster et al., 1968) variants.

Epidemiology

Disease development is favored by temperatures above 32 °C and stress conditions such as dry weather (Coyne et al., 1965). Spread of the pathogen is similar to that for common and halo blight bacteria and is aided by irrigation water and rain-hail storms (Zaumeyer and Thomas, 1957) in association with plant wounds (Rickard and Walker, 1965), although field spread is usually slow.

The pathogen is seed-borne. It can survive up to 24 years in infected seed which may be discolored yellow, orange, or blue (Schuster and Christiansen, 1957; Schuster and Coyne, 1975; Schuster and Sayre, 1967; Zaumeyer and Thomas, 1957) (Figure 89). 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 (Schuster and Coyne, 1974).

Symptomatology

Corynebacterium flaccumfaciens ssp. *flaccumfaciens* is a vascular parasite which infects plants through infected seed, wounds on aerial organs (Coyne et al., 1971; Rickard and Walker, 1965; Walters and Starr, 1952; Zaumeyer and Thomas, 1957), or root wounds caused by nematode feeding or cultivation damage (Schuster, 1959). The rate and degree of plant infection depends upon the point of entry and stage of plant growth. Young plants are particularly susceptible—systemic invasion occurs rapidly once the bacteria reach the vascular system in the stem or petiole (Rickard

and Walker, 1965), frequently killing or stunting young bean seedlings.

The initial symptom of infection by the wilt bacterium—flaccid limp leaves—occurs during the warmest part of the day. The leaves may regain turgidity during periods of high moisture and low temperature, but usually will turn brown, with subsequent plant wilt (Figure 90) and death. The wilting is caused by the obstruction of the vascular bundles which are filled with bacterial cells (Figure 91). The golden-yellow necrotic leaf lesions that develop resemble those lesions caused by common blight bacteria, although the lesion margins are more irregular. Only one or two laterals may be affected. Stems of infected plants break readily in the wind (Dinesen, 1980; Hedges, 1926; Walters and Starr, 1952; Zaumeyer and Thomas, 1957).

Although the bacterium may enter the plant through stomata (Schuster and Coyne, 1977; Schuster and Sayre, 1967), little water-soaking occurs. This contrasts with the common bacterial blight organism (*Xanthomonas campestris* pv. *phaseoli* (Smith) Dye) and the halo blight bacterium (*Pseudomonas syringae* pv. *phaseolicola* (Burk.) Young et al.) which normally penetrate stomata and invade primarily parenchymatous tissue (Zaumeyer and Thomas, 1957).

Control by cultural practices

Such general control recommendations as planting pathogen-free seed and crop rotation (Walters and Starr, 1952; Zaumeyer and Thomas, 1957) are only partially effective because the pathogen is able to survive in plant debris or on weeds.

Schuster et al. (1964) demonstrated that, in certain resistant cultivars, bacteria can survive and multiply, and can be transmitted via infected seed. Bacteria borne on resistant cultivars can be disseminated to susceptible materials grown nearby. Clean seed is therefore still necessary, even in cultivars presumed resistant to bacterial infection.

Control by plant resistance

Germplasm resistant to *C. flaccumfaciens* (Coyne et al., 1963 and 1965) includes the following accessions: P.I. 136677, P.I. 136725,

P.I. 165078, P.I. 177510, P.I. 204600 of *Phaseolus vulgaris*; P.I. 165421, P.I. 181790 of *P. coccineus*; P.I. 213014, P.I. 214332 of *P. acutifolius* A. Gray; P.I. 247686 of *Vigna umbellata* (Thunb.) Ohwi *et* Ohashi; and various accessions of *Vigna radiata* (L.) Wilczek var. *radiata*, *Macroptilium bracteatum* (Nees ex Mart.) Maréchal *et* Baudet, *M. lathyroides* (L.) Urb., and *V. mungo* (L.) Hepper. P.I. 247686 (*V. umbellata*) exhibited no symptoms after inoculation. Although xylem vessels of resistant germplasm are larger than those of susceptible selections (Coyne et al., 1966a; Zaumeyer, 1932), researchers have concluded that xylem size is not correlated with resistance.

Inoculation methods comprise the removal of the cotyledon and inserting a needle tip, coated with inoculum, into the stem at the point of cotyledonary attachment (Coyne and Schuster, 1974); petiole inoculation (Rickard and Walker, 1965); and partial-vacuum inoculation of seeds (Goth, 1966).

Coyne and co-workers studied the inheritance of bacterial wilt resistance (Coyne et al., 1965 and 1966b). The resistant G.N. Star derives from the cross between P.I. 165078 (resistant accession from Turkey) and susceptible Great Northern Nebraska No. 1 selection 27 (Coyne and Schuster, 1976). Two complementary dominant genes conferred susceptibility and the absence of either one or both resulted in resistance. Susceptibility 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 among germplasm sources: for example, 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 (Coyne and Schuster, 1971) which has since been used for the commercial production of Great Northern beans.

Bacterial Brown Spot

Introduction

Bacterial brown spot of beans is caused by *Pseudomonas syringae* pv. *syringae* van Hall. The pathogen can be serious in

United States (Hagedorn and Patel, 1965; Hoitink et al., 1968; Patel et al., 1964) and occurs in Brazil (Robbs, 1962). However, no estimates are available for losses in Latin America where it apparently either does not exist or is of minor importance. This bacterium has an extremely wide host range, including common bean (*Phaseolus vulgaris*), lima bean (*P. lunatus* L.), *Lablab purpureus*, soybean (*Glycine max*), *Pueraria lobata* (Willd.) Ohwi, broad bean (*Vicia faba* L.), *Vigna unguiculata* ssp. *unguiculata var. sesquipedalis*, and cowpea (*V. unguiculata* ssp. *unguiculata*) (Zaumeyer and Thomas, 1957).

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

Etiology

The cells of *Pseudomonas syringae* pv. *syringae* are single straight rods and are motile by 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 diffusible fluorescent pigments, particularly in iron-deficient media. Thus, the bacterium is a typical fluorescent pseudomonad of the *P. syringae* group. Arginine dihydrolase is absent (Doudoroff and Palleroni, 1974). The bacterium uses D-gluconate, glutarate, meso-tartrate, DL-glycerate, isoascorbate, betaine, sorbitol, meso-inositol, sucrose, N-caproate, N-caprylate, N-caprate, DL-β-hydroxybutyrate, citrate, glycerol, and L-proline (Misaghi and Grogan, 1969; Sands et al., 1970).

The optimal growth temperature is 28-30 ^{0}C. The bacterium produces white, convex, and transparent colonies on agar. It also produces a green fluorescent pigment (Weber, 1973). A bacteriocin, named syringacin W-1, is produced by the pathogen in infected bean plant tissue (Smidt and Vidaver, 1982).

Epidemiology

The bacterium has a wide host range but only isolates from beans are highly virulent to beans (Saad and Hagedorn, 1972). Bean

isolates can infect other crops such as peas (*Pisum sativum* L.) or lima beans (*Phaseolus lunatus*), especially when grown in fields with a recent history of bean infection (Hagedorn and Patel, 1965; Patel et al., 1964). The bacterium can survive and multiply on weeds such as hairy vetch, which then act as primary inoculum sources to infect beans, especially during rainstorms (Daub and Hagedorn, 1981; Ercolani et al., 1974). *P. syringae* pv. *syringae* can undergo an important epiphytic-resident phase during which it can survive, and even multiply, on the leaves (Figure 92) and buds of healthy bean plants (Leben et al., 1970; Legard and Schwartz, 1987). It can also survive on such nonhost plants as oak, black locust, winter rye, and sow thistle, that grow within a bean-growing area (Lindemann et al., 1984a). It can also survive in plant residue and volunteer beans (Legard and Schwartz, 1987; Schuster and Coyne, 1975). Infection by, and spread of, the pathogen is favored by sprinkler irrigation practices (Hagedorn and Patel, 1965; Hoitink et al., 1968; Patel et al., 1964) and/or by rainstorms accompanied by strong winds. The pathogen can infest seed. The leaf infection threshold population was found to be 10,000 c.f.u. per gram of leaflet tissue (Lindemann et al., 1984b).

Symptomatology

Pseudomonas syringae pv. *syringae* produces flecks or necrotic brown lesions of varying size which may (Coyne and Schuster, 1969) or may not (Patel et al., 1964) be surrounded by a yellow zone (Figure 93). Macroscopically obvious water-soaked tissue or bacterial exudate may or may not be produced in these lesions (Patel et al., 1964; Webster and Sequeira, 1976). The pathogen can become systemic and cause stem lesions (Zaumeyer and Thomas, 1957). Patel et al. (1964) observed that pods from infected plants grown under field conditions may be bent or twisted (Figure 94). Zaumeyer and Thomas (1957) report that ring spots may form on infected pods. Older plants are usually more resistant (Zaumeyer and Thomas, 1957), but can, at the sixth or seventh trifoliolate leaf stage, be inoculated in the field (Coyne and Schuster, 1974). Plants can be successfully inoculated in the greenhouse when low moisture conditions are present (Saad and Hagedorn, 1971).

Control by chemicals

Hagedorn et al. (1969) report that various chemicals such as copper sulfate or copper hydroxide (86% cupric hydroxide with 56% metallic copper), can be applied at 200-400 g/1000 m^2 to control foliage and pod lesions. This control required weekly sprays after the emergence of the first trifoliolate leaf and resulted in a significant yield response only during severe epidemics. Detailed studies on epiphyte development (Legard and Schwartz, 1987) and disease incidence and severity on foliage revealed significantly less disease in sprayed irrigated beans (Morris et al., 1981).

Control by plant resistance

Phaseolus germplasm resistant to infection by *P. syringae* pv. syringae includes Tempo, G.N. 1140 (Coyne and Schuster, 1971), Wisconsin BBSR 130 (Hagedorn and Rand, 1977), WBR 133 (Daub and Hagedorn, 1976), Earliwax, P.I. 186497, P.I. 326353, P.I. 326419, P.I. 339377 (Hagedorn et al., 1972), P.I. 313234, P.I. 313390, P.I. 313416, P.I. 313297, and P.I. 313404 (Antonius and Hagedorn, 1978).

Inoculation methods are dusting seeds with pulverized infected tissue (Hagedorn et al., 1972) and spraying bacterial suspensions at 15 psi in the greenhouse and 150 psi in the field (Coyne and Schuster, 1969; Saad and Hagedorn, 1971). Injection of inoculum into very small seedlings in the crook neck stage of development has also been successful (Antonius and Hagedorn, 1981). Inoculations (1000-10,000 c.f.u./ml) identified lines with high levels of resistance (for example, WBR 133 and Wisconsin BBSR 130) more effectively than lines with moderate field resistance (for example, Wisconsin BBSR 17 and 28). Seedlings became increasingly susceptible during 3-4 days after emergence. Best results were obtained when seedling development was uniform (Antonius, 1982; Antonius and Hagedorn, 1981). Inoculum concentrations as high as 10^5-10^6 c.f.u./ml have been used in the greenhouse (Coyne and Schuster, 1969; Saad and Hagedorn, 1971).

Some researchers believe the resistance of WBR 133 is recessive and polygenic (Hagedorn and Rand, 1975), but other researchers

have suggested that a more highly additive genetic system is involved. Bacterial growth in F_1 leaf and pod tissue was intermediate between resistant (P.I. 313234 and 313297) and susceptible (Tender White) parents. Estimates of narrow-sense heritability depended on the source of resistance and method of inoculation. Using Wisconsin BBSR 130 as the resistant parent, estimates were low in the field and seedling assay (0.16 and 0.29, respectively; parent-offspring regression, adjusted for inbreeding) and high in the greenhouse (0.73, generation variances) (Antonius, 1982).

Correlations between pod and foliage reactions of F_2 individuals and progeny tests within F_3 and F_4 families suggested that a common genetic system controls the reaction in both foliage and pods (Antonius, 1982; Antonius and Hagedorn, 1982). In crosses involving either Wisconsin BBSR 17 or 28 genotype, assay estimates of the number of genes involved were 1-2 for both pod and foliage reaction at the 1% significance level. At the 5% level estimates of the number of genes for pod reaction were 3-5 (Antonius, 1982; Antonius and Hagedorn, 1983).

Pod resistance of WBR 133 to low inoculum concentrations was higher than its pod resistance to high concentrations. Resistance was adversely affected by increased soil moisture (Daub and Hagedorn, 1976). Symptom expression in susceptible (Tender White) and resistant (WBR 133) beans was different at all inoculum concentrations tested. However, there were almost no differences in bacterial growth rates and final bacterial populations in the two hosts at high inoculum levels (Daub and Hagedorn, 1980). In the field, about one million cells/g of fresh weight were isolated from leaves of susceptible Eagle beans compared with the 1000 cells/g isolated from leaves of resistant WBR 133. Epiphytic populations on resistant bean-breeding lines were intermediate (Daub and Hagedorn, 1981). Wisconsin BBSR 130 was derived from a cross between a resistant selection, WBR 133 (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 the anthracnose pathogen, two rust races, and Fusarium yellows (Hagedorn and Rand, 1977). These and other germplasm sources should provide useful levels of resistance that can be incorporated effectively into commercially acceptable cultivars.

Wildfire

Introduction

Bean wildfire, caused by *Pseudomonas syringae* pv. *tabaci* (Wolf *et* Foster) Young et al. occurs in different bean-growing regions of Brazil (Mohan, 1984; Ribeiro et al., 1974 and 1979). In 1986, the disease was observed for the first time in Argentina (State of Salta) (M. A. Pastor-Corrales, personal communication). However, it has not been reported from elsewhere in Latin America. The bean strain also attacks the garden pea (Ribeiro and Hagedorn, 1976). The common name used for wildfire in Latin America is "fogo selvagem."

Etiology

Pseudomonas syringae pv. *tabaci* is a pathogen with a wide host range and exhibits a high degree of pathogenic specialization among strains isolated from different hosts (Ribeiro et al., 1979). The bacterium is a typical fluorescent pseudomonad of the *P. syringae* group (Doudoroff and Palleroni, 1974). The bean strain is characterized by its ability to hydrolyze esculin, use L-tartrate, erythritol, sorbitol, and cause pitting on polypectate gels. It is unable to use DL-lactate. It produces tabtoxin in culture, and causes the symptoms of wildfire in bean plants (Ribeiro et al., 1979).

Epidemiology

The pathogen apparently does not infect pods and seeds. Sources of primary inoculum, means of secondary spread, and other aspects of the epidemiology of this disease are not yet known.

Symptomatology

Lesions on leaves are small, necrotic, circular to angular, light to dark brown, and surrounded by the characteristically pronounced, broad, circular, bright yellow halos. The lesions may coalesce and cause a leaf blight symptom (Figure 95). Occasionally, foliar

deformation and chlorosis of the infected plants occur. However, pod infection was not found under natural conditions (Mohan, 1984; Ribeiro et al., 1979).

Control

No specific control measures are known.

Miscellaneous Bacterial Pathogens

There are other bacteria which are pathogenic to beans (*Phaseolus* spp.), but are not discussed in this book. Instead, they 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	Symptom	Literature cited
Agrobacterium tumefaciens (E.F. Smith *et* Towns.) Conn.	Crown gall	a
Azotobacter chroococcum Beijerinck	Overgrowth	b
Azotobacter indicus Starkey *et* De	Overgrowth	b
Bacillus lathyri Manns. *et* Taub.	Streak	c
Bacillus megaterium de Bary	Overgrowth	b
Bacillus pumilis Meyer *et* Gottheil	Overgrowth	b
Bacillus subtilis (Ehrenberg) Cohn	Overgrowth	b
Corynebacterium fascians (Tilford) Dows.	Gall	c
Erwinia carotovora (L.R. Jones) Holland	Market disease	a
Erwinia nulandii	Pink seed	d
Escherichia coli (Migula) Castellani *et* Chalmers	Overgrowth	b
Micrococcus luteus (Schroeter) Cohn	Overgrowth	b
Pseudomonas adzukicola		e

(Continued)

Table 1. (Continued).

Pathogen	Symptom	Literature cited
Pseudomonas aeruginosa (Schroeter) Migula	Leaf blight	f
Pseudomonas aptata (Brown *et* Jamieson) F.W. Stevens	Leaf spot	c
Pseudomonas blatchfordae	Leaf blight	g
Pseudomonas coadunata (Wright) Chester	Market disease	a
Pseudomonas flectens Johnson		h
Pseudomonas fluorescens (Trevisan) Migula	Overgrowth	b
Pseudomonas ovalis Chester	Market disease	a
Pseudomonas solanacearum (E.F. Smith) Smith	Brown rot	a
Pseudomonas viridiflava (Burk.) Clara	Gall blight	c
Staphylococcus aureus Rosenbach	Overgrowth	b
Staphylococcus epidermidis (Winslow *et* Winslow) Evans	Overgrowth	b
Staphylococcus marcescens	Overgrowth	b
Xanthomonas phaseoli var. *sojensis* (Hedges) Starr *et* Burkholder	Bacterial pustule	i
Xanthomonas phaseoli f. sp. *vignicola* (Burkholder) Sabet	Leaf blight	i

a. USDA, 1970.
b. Serrada et al., 1982.
c. Zaumeyer and Thomas, 1957.
d. Schuster et al., 1981.
e. Tanii and Baba, 1979.
f. Sirry et al., 1981.
g. Schuster et al., 1980.
h. Johnson, 1956.
i. Schuster and Coyne, 1977.

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