Chapter 12

HALO BLIGHT

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Introduction

Halo blight of beans is caused by the bacterium *Pseudomonas syringae* pv. *phaseolicola* (Burkholder) Young et al. (1978). The bacterium has a worldwide distribution: it is found in those regions of Latin America which have moderate temperatures such as the southern Andean zones of Peru and Colombia, in southern Chile and Brazil (Costa, 1972; Dubin and Ciampi, 1974), and in the Great Lakes Region of Africa (i.e., Rwanda, Burundi, and Zaire), eastern Africa, including Malawi, Kenya, and Zambia, and, occasionally, Uganda (Allen, 1983; CIAT, 1981). Yield losses of 23%-43% have occurred in research fields in Michigan (Saettler and Potter, 1970) and can be a serious problem in Colorado (Schwartz and Legard, 1986). The pathogen can infect various plant species, including the tepary bean (*Phaseolus acutifolius* A. Gray var. *acutifolius*), *Macroptilium bracteatum* (Nees ex Mart.) Maréchal *et* Baudet, scarlet runner bean (*P. coccineus* L.), lima bean (*P. lunatus* L.), *P. polyanthus* Greenman., *P. polystachyus* (L.) B.S.P., common bean (*P. vulgaris* L.), pigeonpea (*Cajanus cajan* (L.) Millsp.), hyacinth bean (*Lablab purpureus* (L.) Sweet), soybean (*Glycine max* (L.) Merrill), *Vigna angularis* (Willd.) Ohwi *et* Ohasi, mung bean (*V. radiata* (L.) Wilczek var. *radiata*), *Pueraria lobata* (Willd.) Ohwi, and siratro (*Macroptilium atropurpureum* (DC.) Urb.) (CIAT, 1987; Walker, 1969; Zaumeyer and Thomas, 1957).

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."

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Etiology

Pseudomonas syringae pv. *phaseolicola* cells are single straight rods and move by using multitrichous polar flagellae. The cells are gram-negative, strictly aerobic, and do 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. Arginine dihydrolase is absent (Doudoroff and Pallerozin, 1974). The bacterium does not use glutarate, meso-tartrate, DL-glycerate, isoascorbate, betaine, erythritol, sorbitol, meso-inositol, nor N-caproate. It does use D-gluconate, L(+)-arabinose, sucrose, succinate, DL-β-hydroxybutyrate, transaconitate, L-serine, L-alanine, and phydroxybenzoate (Misaghi and Grogan, 1969; Sands et al., 1970). It is oxidase-negative (Kovacs, 1956).

The optimal growth temperature range is 20-23 ^{0}C. On agar, the bacterium produces white to cream-colored colonies which exhibit a bluish hue and often a green fluorescent pigment (Weber, 1973).

Without altering their pathogenicity, bacterial cells can survive in liquid nitrogen at -172 ^{0}C for 30 months (Moore and Carlson, 1975), or survive on silica gel at -20 ^{0}C for 60 months (Leben and Sleesman, 1982).

Epidemiology

Pseudomonas syringae pv. *phaseolicola* survives in infected seeds and plant residue on the soil surface (Schuster and Coyne, 1975b). It is found on volunteer beans in the field early in the growing season (Legard and Schwartz, 1987). The organism survives in these habitats until environmental conditions become favorable for infection. Seed transmission is higher when infection occurs earlier in plant development (Saettler et al., 1981). Bacteria survived for nine months after passage through sheep which consumed infested plant debris (Starr and Kercher, 1969). The pathogen enters plants through wounds or stomata during periods of high relative humidity or free moisture (Saettler and Potter, 1970; Walker and Patel, 1964a; Zaumeyer and Thomas, 1957). Light intensity may

influence the plant and the nature of its response to the pathogen (Hubbeling, 1973).

Pseudomonas syringae pv. *phaseolicola* multiplies rapidly on or near the surface of foliage with or without lesions in the presence of dew (Legard and Schwartz, 1987; Stadt and Saettler, 1981). It is disseminated between leaves and plants by water splash and winds during periods of rainfall. The pathogen also multiplies on blossoms, pods, and stem internodes under experimental conditions (Stadt and Saettler, 1981). The bacterium has tremendous disease potential: a dozen infected seeds per hectare, distributed at random, are sufficient to start a general epidemic under favorable conditions (Walker and Patel, 1964a). Halo blight incidence is lower in bean-maize association than in bean monoculture (GLP, 1976). Maize probably acts as a physical barrier to bacterial spread throughout the associated cropping.

Halo blight symptoms develop in six to ten days at 24-28 °C and may be delayed two or three days at higher temperatures (Zaumeyer and Thomas, 1957). Populations of one million colony-forming units per 30 square centimetres of leaf tissue (10^6 c.f.u./30 cm^2) are apparently required for symptom development (Stadt and Saettler, 1981). Halo expression is more common at 16-20 °C than at 24-28 °C (Patel and Walker, 1963). Halo symptoms usually do not develop above 28 °C, although small and numerous water-soaked lesions may still be present (Zaumeyer and Thomas, 1957).

Symptomatology

Three to five days after infection, small water-soaked spots appear, usually on the lower leaf surface (Omer and Wood, 1969; Rudolph, 1984). A halo of greenish yellow tissue appears later around the perimeter of this water-soaked area (Figure 84). The stem and pods may also become infected during a severe epidemic (Figure 85) and produce the typical greasy spots (Figure 86). When infection occurs throughout the vascular system, interveinal leaf tissues appear water-soaked and have a reddish discoloration. Stem girdling or joint rot occurs at nodes above the cotyledons when infection originates from contaminated seed. Infected pods commonly exhibit green water-soaked spots which may develop brown

margins as they mature. Developing seed may rot or become shriveled and discolored (Zaumeyer and Thomas, 1957).

Water-soaked lesions can appear, three days after inoculation, on detached pods placed in water or nutrient solution (Pitts and Pierce, 1966).

Zaumeyer and Thomas (1957) report a snakehead symptom in which injury or destruction of the growing tip may occur after infected seed is planted. Regardless of the plant part infected, a light cream- or silver-colored bacterial exudate characteristically appears on or around lesions (Figure 87).

General plant chlorosis with leaf yellowing and malformation (Figure 88) also may develop from systemic infection without there being external infection (Zaumeyer, 1932). Hildebrand and Schroth (1971) isolated *P. syringae* pv. *phaseolicola* from chlorotic leaves. Systemic chlorosis is more pronounced and uniform at about 20 °C (Coyne and Schuster, 1974; Zaumeyer and Thomas, 1957). The general chlorosis and typical halo symptom around lesions result from a nonhost-specific toxin produced by the bacterium (Coyne et al., 1971; Hoitink et al., 1966; Walker, 1969). The toxin, identified as phaseolotoxin, contains N-phosphosulfamylornithine as the main functional component (Mitchell and Bieleski, 1977).

Patil et al. (1974) found an ultraviolet-induced mutant which was unable to produce toxin. This strain neither induced typical halos nor invaded the plant systemically. Subsequent tests have confirmed that toxin production is necessary for pathogenicity (Gnanamanickam and Patil, 1976). The toxin may suppress production of antibacterial phytoalexins such as phaseolin, phaseolinisoflavan, coumestrol, and kievitone (Gnanamanickam and Patil, 1977). Patel and Walker (1963) suggest that the toxin interferes with the urea cycle, accounting for the buildup of methionine in the halo region. Although the plant reacts to the bacterium's toxin production by producing ammonia (O'Brien and Wood, 1973), researchers do not agree on the role ammonia plays in the plant's response to infection. *P. syringae* pv. *phaseolicola* produces hemicellulases which degrade host cell-wall materials during pathogenesis (Maino, 1972).

Lesion size becomes larger if plants are infected with rust (*Uromyces phaseoli* (Reben) Wint.), before being infected with halo

blight (Yarwood, 1969). Lesion numbers may also be increased by an inoculation with *P. syringae* pv. *phaseolicola* mixed with *Achromobacter* sp. (Maino et al., 1974). A toxin-producing strain of the halo blight bacterium severely reduced nodulation by the soil bacterium *Rhizobium phaseoli* Dangeard in vitro. However, Hale and Shanks (1983) did not feel that phaseolotoxin had a direct effect upon the rhizobia.

Control by Cultural Practices

The pathogen survives between growing seasons in bean tissue on the soil surface (Schuster and Coyne, 1975b) and on volunteer beans (Legard and Schwartz, 1987). Deep-plowing and crop rotation are therefore advocated to reduce initial inoculum pressure (Zaumeyer and Thomas, 1957). In developing countries, it is also is advisable to practice sanitation, that is, to remove infested debris from the fields. Walker and Patel (1964a) reported that, in temperate zones, there is no evidence that halo blight is spread by cultivation equipment used in infected bean fields. However, foliage must be dry before moving equipment through infected fields.

The use of pathogen-free seed produced under conditions unfavorable to the organism is important in reducing the initial inoculum within a field (Zaumeyer and Thomas, 1957). Seed transmission is significantly lower in cultivars with partial to complete resistance (Katherman et al., 1980; Saettler et al., 1981). Because seed can be contaminated by bacteria present in powdered plant tissue (Grogan and Kimble, 1967; Guthrie, 1970), seed should be thoroughly cleaned of dust after threshing. Contaminated seed also can be treated with chemicals or antibiotics to destroy bacteria present on the surface (Hagedorn, 1967; Russell, 1975; Zaumeyer and Thomas, 1957). Chemical treatment is seldom effective against internally borne bacteria. Belletti and Tamietti (1982) reduced the proportion of infected seedlings by more than 70% by exposing dry seeds to 70 °C for 120 minutes or water-soaked seeds to 50 °C for 180 minutes.

While current technology cannot eradicate bacteria inside the seed coat or embryo, it can identify highly contaminated seed by exposure to ultraviolet light. Wharton (1967) reported that 20% of

seeds exhibiting a bluish-white fluorescence contained *P. syringae* pv. *phaseolicola*, while 1% of nonfluorescent seeds contained the bacterium. Because other organisms can elicit this fluorescence, this test can only identify potentially contaminated seed lots which then need to be evaluated by more specific laboratory procedures (Parker and Dean, 1968). Other diagnostic tests include the enzyme-linked immunosorbent assay (ELISA) and immunofluorescence microscopy which can detect 10,000 bacteria/ml of solution from seeds and leaves (Barzic and Trigalet, 1982; van Vuurde et al., 1983).

In United States, clean-seed production is a major method for controlling halo blight. Clean-seed production in Idaho depends upon: field inspection for visible evidence of infection; laboratory inoculation of susceptible pods with suspensions from seed lots; serological tests for seed-borne pathogens; and quarantines to prevent importation of bean seed from areas where the pathogen exists (Butcher et al., 1968 and 1971). If the bacterium is detected in a seed lot, the seed is not certified and hence not planted by progressive growers. Despite such precautions, irrigation practices and/or environmental conditions in the region can favor pathogen development as, for example, during the epidemics of 1963-1967 (Butcher et al., 1968 and 1969).

Chemical Control

Ralph (1976) reported that soaking bean seed in a 0.2% streptomycin solution for two hours prevented the transmission of halo blight bacteria by contaminated seed. However, the solution also reduced plant emergence by more than 20% compared with water-soaked controls. Hagedorn (1967) found that although streptomycin seed treatment was not always beneficial, it provided some residual protection against later plant infection. Taylor and Dudley (1977b) reduced primary infection from infected seed by 98% 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 nor survived in bean tissue (Russell, 1975).

Halo blight has been controlled chemically with Bordeaux mixture, copper oxychloride, copper sulfate, copper oxide, streptomy-

cin sulfate, and dihydrostreptomycin sulfate (Hagedorn et al., 1969; Ralph, 1976; Saettler and Potter, 1970; Taylor and Dudley, 1977a; Zaumeyer and Thomas, 1957). Such chemicals are applied 7 to 10 days with ground or aerial spray equipment at rates of 200-400 g/1000 m^2. They are also applied at first flower and pod set at the rate of 0.1% a.i. per 675 litres per hectare to prevent the spread and development of halo blight on leaves and pods (Hagedorn et al., 1969; Saettler and Potter, 1970; Taylor and Dudley, 1977a). The application of antibiotics to the foliage may induce the development of resistant mutants. Their use should therefore be reduced or avoided. Legard and Schwartz (1987) demonstrated that timely copper hydroxide sprays significantly reduce or limit the establishment of syringae-type pseudomonads on bean foliage.

Control by Plant Resistance

Pathogenic variation occurs in *P. syringae* pv. *phaseolicola* populations (Buruchara and Pastor-Corrales, 1981; Hubbeling, 1973; Schroth et al., 1971; Schuster and Coyne, 1975a and 1975b). Two major race groups (1 and 2) have been identified in the Americas and Europe (Hubbeling, 1973; Patel and Walker, 1965). However, a new race from Africa named as race 3 has been recently reported (CIAT, 1986 and 1987). All strains tested had similar rates of multiplication, regardless of race (Gnanamanickam and Patil, 1976). Variation in virulence of strains belonging to either race is attributed to differences in the rate of toxin production (Hubbeling, 1973; Patel et al., 1964; Russell, 1975). However, many workers feel that the race designation is not valid (Schroth et al., 1971; Schuster and Coyne, 1975b), for example, serological tests show that *P. syringae* pv. *phaseolicola* antiserum is not race specific (Guthrie, 1968). Schuster and Coyne (1975b) report that the more virulent strains are better adapted for survival than the less virulent strains.

Various inoculation methods have been used to test beans for halo-blight resistance. They include partial-vacuum infiltration of seeds (Goth, 1966), atomizing bacterial suspensions onto leaves and water-soaking them at 15 psi in the greenhouse and 150 psi in the field (Patel and Walker, 1963; Schuster, 1950 and 1955; Zaiter and Coyne, 1984), multiple needle-punctures, and rubbing leaves with inoculum-carborundum suspensions (Hubbeling, 1973). Zaiter and

Coyne (1984) reported that the water-soaking method provided the most severe reaction for which inoculum concentrations of 10^6-10^7 cells/ml have been used (Schuster, 1955).

Plant resistance to *P. syringae* pv. *phaseolicola* is well known. It includes both race-specific and general resistance mechanisms that are effective against both races and virulence-variable strains. In general, older plants are more resistant to infection (Omer and Wood, 1969; Patel and Walker, 1963 and 1966; Zaumeyer and Thomas, 1957). Bacteria occasionally attach themselves to cell walls (Ebrahim-Nesbat and Slusarenko, 1983) and multiply in the xylem (Omer and Wood, 1969) of both susceptible and resistant plants. Hubbeling (1973) suggested that resistance occurs when the rate of bacterial multiplication in vascular tissue is reduced and a necrotic response to the bacterial toxin develops in parenchymatous or meristem tissue. Kinyua et al. (1981) described a resistant response as one that results in necrotic spots and partial chlorosis. A susceptible response is one that produces large water-soaked lesions with entire chlorosis. No qualitative differences exist between the free amino acid content in uninfected susceptible plants and resistant ones (Patel and Walker, 1963).

Independent genes separately govern leaf resistance, pod resistance, and plant systemic chlorotic reactions (Baggett and Frazier, 1967; Coyne and Schuster, 1974; Coyne et al., 1967 and 1971). Pod susceptibility frequently occurs in plants which possess leaf resistance. Linkage occurs between the different genes that control leaf and plant systemic chlorotic reactions (Coyne et al., 1971; Hill et al., 1972). Russell (1977) reported that resistance to the halo blight bacterium involves two phenomena: resistance to growth of bacterial cells in vivo, and suppression of toxin production.

Bean germplasm resistant to races 1 and 2 has been identified in field and greenhouse tests. Resistance to both races exists in Great Northern (G.N.) Nebraska No. 1 selection 27, G.N. No. 16, California Small White 59, FM 51, FM-1 Blue Lake, a Nebraska selection from P.I. 150414, P.I. 203958, OSU 10183, and V 4604 (Baggett and Frazier, 1967; Coyne and Schuster, 1974; Coyne et al., 1967; Hill et al., 1972; Innes et al., 1984; Mukunya and Keya, 1978; Taylor et al., 1978; Walker and Patel, 1964b). Red Mexican U.I. 3, 34, and 35 are resistant to race 1 (Hubbeling, 1973). Other resistant

materials include G 790, G 984, G 2338, G 3272, G 5272, G 6034, G 6036, G 6339 (Figueroa, 1980); Gloriabamba (G 2829), Pajuro (G 11766), Nariño 20 (G 12666), Poroto (G 12592), and Palomo (G 12669) with nonspecific resistance; BAT 590, BAT 1281, V 8010, VRA 81022, and G 5960 with specific resistance to races 1 and 3 (CIAT, 1987).

Schuster (1950) reported that Arikara Yellow and Mexican Red conferred one or two homozygous recessive genes for resistance to their progeny, depending on which susceptible parent was used. Patel and Walker (1966) report that P.I. 150414 possesses recessive resistance to races 1 and 2 and that Red Mexican, dominantly resistant to race 1. V 4604, also possesses the Red Mexican type of resistance to race 1, but has a polygenic control of its partial resistance to race 2 (Innes et al., 1984). Hill et al. (1972) showed that P.I. 150414 and G.N. Nebraska No. 1 selection 27 contain the same dominant allele responsible for resistance to race 1 but different genes control the reaction to race 2. GLP 16 and GLP-X-92 contain a recessive gene for resistance to race 2 (Kinyua et al., 1981).

Coyne et al. (1966b) proposed a breeding scheme based upon a backcross and sibcross design to combine resistance to *P. syringae* pv. *phaseolicola* (qualitative inheritance) and the common bacterial blight, *Xanthomonas campestris* pv. *phaseoli* (Smith) Dye (quantitative inheritance). Coyne and Schuster (1974) stressed that it is important to select germplasm which has a resistant pod, leaf, and nonsystemic plant reaction. Hagedorn et al. (1974) recently developed Wisconsin HBR 40 and 72 which are resistant to halo blight races 1 and 2, common bacterial blight, bacterial brown spot, and various fungal pathogens (Hagedorn and Rand, 1977).

Successful long-term control of *P. syringae* pv. *phaseolicola* requires that bean-production regions adopt integrated control programs. A combination of field sanitation (removal of infested plant debris), crop rotation, planting clean seed, progressive cultural practices (weed control, irrigation timing, planting date), limited use of chemicals, and greater reliance upon resistant cultivars should allow growers to realize higher yields from their crops.

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