Chapter 4

ANGULAR LEAF SPOT

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Introduction

Angular leaf spot (ALS) of beans, caused by the fungus *Phaeoisariopsis griseola* (Sacc.) Ferraris (syn. *Isariopsis griseola* Sacc.), is a serious disease of beans which has occurred in such tropical and subtropical countries as Argentina, Brazil, Colombia, Costa Rica, Dominican Republic, Guatemala, Mexico, Nicaragua, Peru, Puerto Rico, Venezuela in Latin America, and Burundi, Kenya, Malawi, Rwanda, Tanzania, Uganda, Zaire, and Zambia in Africa (Barros et al., 1958a and 1958b; Bazán de Segura, 1953; CIAT, 1981; Costa, 1972; Crispín-Medina et al., 1976; Díaz-Polanco et al., 1965; Golato and Meossi, 1972; Miles, 1917; Moreno, 1977; Ploper, 1983; Schieber, 1964; Silvera-C., 1967; Stoetzer, 1983; Vieira, 1983).

Other regions where ALS has occurred are Australia, Europe, India, Iran, Israel, Japan, New Zealand, and United States (Cardona-Alvarez and Walker, 1956; Chupp, 1925; Cole, 1966; Hagedorn and Wade, 1974; Hill, 1982; Kaiser et al., 1968; Saettler and Correa-Victoria, 1983; Sharma and Sohi, 1980; Weaver and Zaumeyer, 1956; Zaumeyer and Thomas, 1957). The Commonwealth Mycological Institute lists more than 60 different countries in which ALS occurs. Yield losses can be severe and have reached 50% in the U.S. (Hagedorn and Wade, 1974), 40%-80% in Colombia (Barros et al., 1958b; Mora et al., 1985; Schwartz et al., 1981), 45% in Brazil (Rava-Seijas et al., 1985), and 80% in Mexico (Crispín-Medina et al., 1976).

The fungus has a host range which includes the common bean (*Phaseolus vulgaris* L.), lima bean (*P. lunatus* L.) (Cardona-Alvarez

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and Walker, 1956), scarlet runner bean (*P. coccineus* L.) (Brock, 1951), urd bean (*Vigna mungo* (L.) Hepper) (Golato and Meossi, 1972), tepary bean (*P. acutifolius* A. Gray var. *acutifolius*), *V. angularis* (Willd.) Ohwi *et* Ohashi, *V. umbellata* (Thunb.) Ohwi *et* Ohashi (Campos-Avila, 1979), pea (*Pisum sativum* L.) (Chupp, 1925), and cowpea (*V. unguiculata* (L.) Walp. ssp. *unguiculata*) (Díaz-Polanco et al., 1965). Abramanoff, cited by Cardona-Alvarez and Walker (1956), considered soybean (*Glycine max* (L.) Merrill) to be a host, but this has not been confirmed. The common name frequently used for angular leaf spot in Latin America is "mancha angular."

Taxonomy

Ellis (1971) followed Ferraris (1909) and recognized the ALS pathogen as *Phaeoisariopsis griseola* on the basis of characters such as conidial septation (3-6 septa), pigmentation, conidiophores, and stroma. Drs. D. Farr (U.S. Dep. Agric. Mycology Laboratory) and B. Shumaker (Biosystematics Research Institute, Canada) concur with this nomenclature which is recognized by the Commonwealth Mycological Institute in England. Thus, *P. griseola* is synonymous with *Isariopsis griseola*, *I. laxa* (Ell.) Sacc., *Graphium laxum* Ell., *Cercospora columnare* Ell. *et* Ev., *Lindaumyces griseola* Gonz. Frag., *Arthrobotryum puttemansii* Henn., and *Cercospora sthulmanni* Henn. (Cardona-Alvarez, 1956; Zaumeyer and Thomas, 1957).

The authors recognize that ALS is usually identified as *Isariopsis griseola* in plant pathology literature (Andersen, 1985), particularly since Zaumeyer and Thomas (1957) concluded that "A comparison of authentic Italian material of *I. griseola* with the other exsiccatae... and with other material of American origin... shows them to be identical. Characters compared included synnema appearance and spore morphology." However, in our opinion the more accurate designation is *Phaeoisariopsis isariopsis*, and its use, at least as a synonym, should be encouraged.

Etiology

In nature, the fungus produces groups of 8-40 conidiophores (Miles, 1917) which join loosely to form the dark columnar synnemata that bear conidiospores (Barnett and Hunter, 1972). A synnemata may have a diameter of 20-40 μm and be 80-500 μm in length (Ellis, 1971; Golato and Meossi, 1972; Hocking, 1967; Miles, 1917). The conidiophores tend to separate near maturity and fructification (Chupp, 1925). Conidia are gray, cylindrical to fusiform, slightly curved, and measure 3-8 by 43-68 μm with one to six septations (Golato and Meossi, 1972; Hocking, 1967; Miles, 1917; Zaumeyer and Thomas, 1957). The conidial length of 10 isolates from Colombia, studied by Buruchara (1983), varied between 18 and 76 μm with a mean of 38.5 μm. The width varied between 3.8 and 8.8 μm with an average of 6.4 μm, whereas the number of septa varied between 0 and 7 with a mean of 3. These parameters varied significantly both within and between isolates.

Phaeoisariopsis griseola grows slowly on artificial culture media over a range of temperatures between 8 and 28 °C with an optimum of 24 °C; optimal pH is between 5 and 6. Adequate growth media include potato dextrose agar plus bean leaf extract (Cardona-Alvarez, 1956; Cardona-Alvarez and Walker, 1956), honey peptone agar, baby food (assorted vegetables)-calcium carbonate agar (Santos-Filho, 1976), and potato yeast dextrose agar. Abundant sporulation occurred in 10-15 days when the fungus was grown at 19 °C in darkness on V-8 vegetable juice agar (200 ml V-8 vegetable juice, 3 g calcium carbonate, and 18 g Bacto-agar added to sufficient distilled water to make 1 liter) (CIAT, 1979). Campos-Avila and Fucikovsky-Zak (1980) reported optimal growth of a single isolate of *P. griseola* at 24 °C on V-8 agar while maximum sporulation occurred at 16 °C. Recent studies (F. J. Correa-Victoria, unpublished data) with four different pathotypes of ALS report maximum sporulation on V-8 agar at 25 °C, no growth at 30 °C, and growth but no sporulation for one pathotype at 18 °C. The remaining 3 pathotypes sporulated at 16 °C. Similar results were reported by Buruchara (1983). Discreet colonies form on the media and single-spore isolates may exhibit variation within a petri dish for colony structure, coloration, and quantity of sporulation (Cardona-Alvarez, 1956).

Epidemiology

The pathogen infects leaf tissue by entering stomata and advancing intercellularly in the mesophyll and palisade parenchyma. Within nine days after infection, the fungus develops intracellularly throughout necrotic lesions. By 9-12 days stromata develop in the substomatal cavity and sporulation may then occur during periods (24-48 hours) of continuous moisture (Cardona-Alvarez, 1956; Cardona-Alvarez and Walker, 1956). Moisture is probably the single most important factor governing the development of ALS epidemics and is a prerequisite for infection, synnemata formation, and sporulation (Cardona-Alvarez and Walker, 1956; Sindhan and Bose, 1980a and 1980b). On the other hand, stroma formation, accompanied by spore release and dissemination, and disease development can proceed under relatively dry conditions (Cardona-Alvarez, 1956).

Infection and disease development can occur over a wide temperature range, 16-28 °C, with an optimum of 24 °C (Cardona-Alvarez, 1956; Sindhan and Bose, 1980b). Inglis and Hagedorn (1984) reported that disease was more severe when infection occurred at 16, 20, and 24 °C and plants were incubated at 20, 24, and 28 °C than when the infection and incubation temperatures were the same. Although bean plants are susceptible to *P. griseola* infection throughout the growing season (Barros et al., 1958b; Cardona-Alvarez and Walker, 1956; Costa, 1972; Santos-Filho et al., 1978; Weaver and Zaumeyer, 1956), severe disease symptoms in the field are not usually observed until soon after flowering or as plants approach maturity. Fluctuating weather conditions (temperature, relative humidity, sunlight) usually favor disease development under field conditions.

Contaminated seed constitutes one source of primary inoculum. The fungus is usually associated with the hilum area of the seed coat (Correa-Victoria, 1984; Dhingra and Kushalappa, 1980; Ellis et al., 1976; Orozco-Sarria and Cardona-Alvarez, 1959; Sharma and Sohi, 1980; Sohi and Sharma, 1974). Contamination may be external or internal (Correa-Victoria, 1984; Sohi and Sharma, 1974). Correa-Victoria (1984) found that seed infection in bean

types other than Red Kidney was associated with fungal development both in the hilum and in other areas of the seed coat. However, there was no evidence of seed infection in black-seeded bean genotypes, even after inoculation of pods. Such varietal differences in seed infection have been noted previously (Orozco-Sarria and Cardona-Alvarez, 1959; Sharma and Sohi, 1980).

Viability of *P. griseola* in contaminated seed apparently decreases with time (Correa-Victoria, 1984; Orozco-Sarria and Cardona-Alvarez, 1959; Sindhan and Bose, 1979). Dhingra and Kushalappa (1980) found no consistent correlation between disease severity on pods and incidence of seed infection; diseased seeds were recovered only from areas beneath the pod suture bearing ALS lesions. The authors concluded that seed transmission of *P. griseola* is an insignificant source of primary inoculum. Díaz-Polanco et al. (1965) reported that infected seed is a minor source of primary inoculum because little possibility for seed transmission exists under low humidity and moisture conditions in the field.

However, Correa-Victoria (1984), successfully grew ALS-infected seedlings from infected seed in greenhouse studies. The transmission occurred only when seedlings were exposed to simulated wind-blown rain-splashing. Correa-Victoria observed that after germination, the seed coat harboring *P. griseola* usually stays on the soil surface. The wind-blown rain-splashing is apparently necessary to disseminate spores to infection sites on primary and/or trifoliolate leaves.

The most important source of primary inoculum for the ALS disease is pathogen-infected plant debris in the field. The fungus can survive two successive winters in temperate zones as stromatic growth on diseased plant debris (Cardona-Alvarez, 1956; Saettler and Correa-Victoria, 1985; Sohi and Sharma, 1974). Pathogen viability decreases rapidly in plant debris buried beneath the soil surface (Correa-Victoria, 1984; Saettler and Correa-Victoria, 1985). Under favorable environmental conditions, spores produced on the surface of infected tissue can disseminate to host plants (Cardona-Alvarez, 1956; Cardona-Alvarez and Walker, 1956).

Epidemic development of ALS is affected by the type of cropping system used to produce beans. There are conflicting reports in the

literature regarding the severity of ALS in beans when planted in association with other crops. Moreno (1977) reports that angular leaf spot infection is more severe in beans grown in association with maize than in association with either sweet potato or cassava, or in monoculture. However, Mora-E. (1978) and van Rheenan et al. (1981) observed less ALS in bean-maize plantings during a dry growing season.

Symptomatology

Angular leaf spot symptoms occur on all aerial parts of the plant. Lesions are most common on leaves and usually appear within six days after inoculation (Llanos-M., 1957). They may appear on primary leaves, but usually do not become prevalent on later foliage until late flowering or early pod set (Barros et al., 1958b). Lesions initially are gray or brown, may be surrounded by a chlorotic halo, and have indefinite margins. They become necrotic and well defined with the typical angular shape by nine days after infection (Figure 4). Lesions then may increase in size, coalesce, and cause partial necrosis and yellowing of leaves which then fall off prematurely. On primary leaves, lesions are usually round, larger than those found on trifoliolate leaves, and may develop concentric rings within themselves.

Lesion size is inversely related to lesion number per leaf or leaflet (CIAT, 1979). Lesions appear on pods (Figure 5) as oval to circular spots with reddish brown centers that are sometimes surrounded by darker colored borders (Barros et al., 1958b; Cardona-Alvarez; 1956, Cardona-Alvarez and Walker, 1956; Crispín-Medina et al., 1976; Vieira, 1983; Zaumeyer and Thomas, 1957). Infected pods bear poorly developed or entirely shriveled seeds (Barros et al., 1958b). Brown elongated lesions occur on plant stems, branches, and petioles (Figure 5) (Cardona-Alvarez, 1956; Cardona-Alvarez and Walker, 1956; Crispín-Medina et al., 1976). One characteristic sign of *P. griseola* is the production of dark gray to black synnemata and conidia in lesions on the lower leaf surface of trifoliolate leaves (Figure 6), on both the upper and lower surfaces of primary leaves, stems, branches, and pods during long periods of high humidity or free moisture.

Conidia can be disseminated long distances by air currents and splashing rain. Thus, the spread of conidia is the principal cause of secondary infections.

Control by Cultural Practices

The following control procedures have reduced ALS: crop rotation of at least two years between bean crops, planting in well-drained soil, removal of infected crop debris by plowing or other means, and planting pathogen-free seed (Barros et al., 1958a; Cardona-Alvarez, 1956; Correa-Victoria, 1984; Costa, 1972; Crispín-Medina et al., 1976; Saettler and Correa-Victoria, 1985). Figure 7 shows young bean plants that were infected by spores liberated from adjacent infected crop debris. The debris had not been removed from the field after the previous bean crop.

Control by Chemicals

Chemical control by foliar spray applications can be achieved with a Ferbam-sulfur-adherent combination (Bazán de Segura, 1953), zineb (Barros et al., 1958a), benomyl (0.13-0.25 g/L), and thiophanate (2.0 g/L). Singh and Sharma (1976) found that disease control was best obtained and yields highest when 0.13 g/L of benomyl was used and the plants sprayed at intervals of as often as every four weeks. Multiple sprays of the systemic fungicide bitertanol increased yields by 33%-41% (Pastor-Corrales et al., 1983). Costa (1972) recommends the use of maneb, ziram, copper oxychloride, and Bordeaux mixture. González et al. (1977) obtained economic disease control from the foliage sprays mancozeb, captafol, and metiram 20, 30, and 40 days after planting.

Chemical treatment of seed is a useful approach for contaminated seed lots. For example, benomyl (6 g/kg seed) and a captan-zineb combination (3.7 g/kg seed) applied in a water-based slurry (0.11 g/ml) effectively eradicated *P. griseola* from contaminated seed (Correa-Victoria, 1984; Saettler and Correa-Victoria, 1985). Singh and Sharma (1976) obtained 100% control of ALS when contaminated seed was dry-treated with Ceresan (now discontinued), or steeped in a 1% solution of mercuric chloride for 30 min-

utes. Araya-Fernández (1977) also obtained significantly less leaf infection when seed was treated with benomyl before planting.

Control by Plant Resistance

A number of studies have reported diverse sources of resistance to ALS in bean genotypes (Brock, 1951; Campos-Avila, 1979; Costa, 1972; Díaz-Polanco et al., 1965; Hagedorn and Rand, 1985; Olave-L., 1958; Santos-Filho et al., 1976; Silvera-C., 1967; Singh and Sharma, 1975; Vieira, 1974). However, these studies were concerned primarily with resistance to local isolates of the pathogen. During the period 1978-82, Schwartz et al. (1982) evaluated about 13,000 *P. vulgaris* accessions from the CIAT germplasm bank; only 56 of the accessions exhibited a resistant or intermediate disease reaction when tested with a mixture of 15 *P. griseola* isolates obtained from eight separate regions within Colombia.

To aid the identification of new, broadly based sources of resistance to ALS, CIAT's Bean Program has distributed the Bean Angular Leaf Spot International Test (BALSIT) to interested Latin American and African researchers. Entries such as Jalo EEP 558 and BAT 332, exhibit resistance in a specific country or geographical area but are frequently susceptible in other locations. Such variation in resistance according to geographical location suggests that *P. griseola* exhibits pathogenic variation (CIAT, 1984; Saettler and Correa-Victoria, 1983). Under field conditions with sufficient disease pressure, no single *Phaseolus vulgaris* line so far evaluated exhibits immunity to the ALS pathogen.

The following bean cultivars and lines from the BALSIT have shown excellent levels of ALS resistance in more than one country at BALSIT locations: A 75, A 140, A 152, A 154, A 175, A 197, A 212, A 216, A 222, A 240, A 247, A 251, A 294, A 295, A 299, A 338, A 339, A 340, A 382, BAT 67, BAT 76, BAT 431, BAT 963, BAT 1432, BAT 1458, BAT 1510, BAT 1647, G 2959, G 3884, G 4421, and G 5653 (CIAT, 1984). When 115 commercial dry-bean cultivars were screened against a Michigan isolate of *P. griseola*, susceptibility was found associated with large- and medium-sized seeds such as those of Red Kidney and Cranberry cultivars (Correa-Victoria, 1984). Sources of resistance reported from Africa include

GLP 24, GLP-X-92, GLP-X-806, and GLP 77 (Smit et al., 1983; Stoetzer et al., 1983). Hagedorn and Rand (1985) reported that P.I. 209488 exhibited a resistance which reduces the rate of lesion development.

Inheritance of resistance is conferred by recessive and dominant genes, depending upon the parental cultivar. Santos-Filho et al. (1976) reported that the resistance of Caraota 260 is controlled by a single recessive gene. Singh and Saini (1980) also reported that the resistance of PLB 257 (*P. coccineus*) also came from a single recessive gene. Zaumeyer and Meiners (1975) showed that resistance in some genotypes is controlled by three recessive genes. Barros et al. (1957) found that, in most crosses, resistance is recessive and controlled by two or three independent factors. However, resistance was dominant in a few crosses. Cardona-Alvarez (1958) found that Line 258 possesses dominant resistance that is governed by a single gene.

Researchers must develop methodology to produce inoculum uniformly and to screen germplasm in the laboratory, greenhouse, and field. Singh and Sharma (1975) field-screened by inoculating soil with previously infected bean debris. Inglis and Hagedorn (1984) increased disease pressure in field plots when dry infected tissue was used as inoculum instead of conidial suspensions. Spores of *P. griseola* have been harvested with good results at CIAT (1979 and 1984). The medium used was V-8 juice agar or potato dextrose agar (PDA). It was suspended in sterilized distilled water (20,000 spores/ml) and mixed with dispersing agents such as gum arabic (2-5 g/L), Triton-AE (0.1% sol.), or Tween 80 (1% wt/vol) (Alvarez-Ayala, 1979; Pastor-Corrales, 1985). The mixture was then sprayed onto plants in the greenhouse or field during optimal conditions of high moisture and moderate temperatures.

Correa-Victoria (1984) showed that disease reaction from ALS is highly dependent on such factors as pathogen isolate, inoculum concentration, host cultivar and its age, temperature, and humidity. Alvarez-Ayala and Schwartz (1979) noted that disease reactions are very dependent on inoculum concentration. Field studies at CIAT (1984) and in Brazil (Santos-Filho et al., 1978) revealed that plant age was more important than inoculum concentration in influencing disease development. Symptoms in most cultivars did not develop

until plants were about 30 days old. Recent studies in the greenhouse and field have shown that some bean genotypes exhibit different leaf and pod reactions (Correa-Victoria, 1984). Additional studies need to be performed to determine whether these differences are controlled by separate genes.

Marín-Villegas (1959) inoculated 14 differential cultivars individually with 30 single-spore isolates of *Phaeoisariopsis griseola* collected from different bean-production sites in Colombia. He concluded that the isolates contained 13 different pathogenic races, but questioned the genetical purity and uniformity of the differential cultivars he used. Hocking (1967) recovered an isolate in Tanzania which produced circular lesions and was highly virulent at 100 spores/ml. He speculated that the isolate may have been a result of a single mutation within natural isolates. Alvarez-Ayala and Schwartz (1979) differentiated among five *P. griseola* isolates from Colombia and Ecuador by inoculating the bean cultivars Caraota 260, Alabama No. 1, Red Kidney, ICA Duva, and Cauca 27a. Their isolates also appeared to differ in virulence on the same cultivar. Buruchara (1983) differentiated 21 isolates of *P. griseola* from Colombia into seven pathotypes based on differential reactions of six bean cultivars. Correa-Victoria (1984) confirmed the existence of races in *P. griseola* by dividing 30 isolates from six countries into five pathogenicity groups. He used 12 bean cultivars and found that isolates from United States and Malawi (Africa) have a narrower host range than isolates from Latin American countries (Brazil, Colombia, Dominican Republic, and Puerto Rico).

Preliminary studies were conducted at CIAT (unpublished data) on a series of 21 bean cultivars to examine the pathogenicity, virulence, and aggressiveness of 17 *P. griseola* isolates from Argentina, Brazil, Colombia, Costa Rica, Guatemala, Mexico, and Nicaragua. Differences in pathogenicity were observed among all the isolates, and within isolates from the same country. Quantitative differences (in percentages) between the cultivars were observed for disease, number of lesions, lesion size, number of spores/mm^2, and the number of days required to induce the same level of disease. Differences in the date of disease initiation, lesion size, disease progress, and severity were also observed between cultivars under field conditions. Many lines with broad resistance in several

locations throughout Latin America and Africa are characterized by small disease lesions. Studies conducted in Colombia (M. A. Pastor-Corrales, unpublished data; Santos-Filho et al., 1978) on the effects of ALS on yield components of the bean plant, suggest that the disease significantly reduces the number of seeds per pod, as well as seed weight, particularly in susceptible varieties. However, the number of pods per plot was not significantly reduced.

A standardized set of differential bean cultivars is now being developed to classify physiological races (pathotypes) of *P. griseola*. These differential cultivars, together with the BALSIT Nurseries, will permit early detection of changes in the pathogen population and the discovery of new races. A uniform disease rating scale has been developed at CIAT for use in the BALSIT, and for breeders and pathologists seeking new sources of resistance.

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