Chapter 5

ANTHRACNOSE

M. A. Pastor-Corrales and J. C. Tu*

Introduction

Bean anthracnose is caused by *Colletotrichum lindemuthianum* (Sacc. *et* Magn.) Scrib. The scientific authority has been a controversial issue and *C. lindemuthianum* (Sacc. *et* Magn.) Briosi *et* Cav. is also widely accepted (Stevenson, 1956). The perfect stage of this pathogen is *Glomerella cingulata* (Stonem.) Spauld. *et* Schrenk. (Kimati and Galli, 1970), but is rarely found in culture or in nature. Thus, the name of the imperfect stage is commonly used. Anthracnose is probably the most important disease of beans throughout the world. The disease can be devastating. It can cause complete yield losses on susceptible bean cultivars or when badly contaminated seed is planted and favorable conditions prevail during the growing season (Zaumeyer and Thomas, 1957).

Bean anthracnose has worldwide distribution. However, it causes greater losses in temperate and subtropical zones than in the tropics. Anthracnose has caused economic losses in North, Central, and South America, Europe, Africa, Australia, and Asia (Chaves, 1980; Cruickshank, 1966; Tu, 1981; Zaumeyer and Thomas, 1957). It was, at one time, considered as the most important disease in the bean-producing areas of eastern USA (Zaumeyer and Thomas, 1957). However, through widespread use of clean seed produced in areas where anthracnose does not occur, the disease has declined considerably in importance since 1925 (Zaumeyer and Thomas, 1957). Clean seed and resistant cultivars have also diminished the importance of anthracnose in western Europe (Fouilloux, 1979).

Anthracnose is an important disease of beans in Latin America and Africa. In Latin America, anthracnose has caused severe

---

* Plant pathologists, Centro Internacional de Agricultura Tropical (CIAT), Cali, Colombia, and Harrow Research Station, Agriculture Canada, Harrow, Ontario, Canada, respectively.

damage in Brazil (Costa, 1972; Vieira, 1983), Argentina (Ploper, 1983), Mexico (Crispín-Medina and Campos-Avila, 1976), Guatemala, Costa Rica, Nicaragua (Echandi, 1976), Peru, Ecuador, and Colombia (Guzmán-Vargas and de la Rosa, 1975; Olarte-M. et al., 1981). It also occurs in the Caribbean countries. In eastern Africa, anthracnose is important in Kenya, Uganda, and Tanzania. It is recurrent in the Great Lakes Region of Rwanda, Burundi, and Kivu Province of Zaire (CIAT, 1981).

Yield losses are more severe when bean plants are infected early. For example, yield losses of 95% and 38% occurred when a susceptible bean cultivar was inoculated one and six weeks after plant emergence, respectively (CIAT, 1976; Guzmán-Vargas and de la Rosa, 1975; Guzmán-Vargas et al., 1979).

Although *C. lindemuthianum* is primarily a pathogen of the common bean *Phaseolus vulgaris* L., it can infect related species and varieties such as *P. vulgaris* var. *aborigineus* (Burk.) Baudet (a South American ancestral wild form of the common bean); *P. acutifolius* var. *acutifolius* (cultivated tepary bean); *P. coccineus* L. (scarlet runner bean); *P. lunatus* L. (lima bean); *P. lunatus* var. *macrocarpus* (big lima bean); *Vigna mungo* (L.) Hepper (urd bean); *V. radiata* (L.) Wilczek var. *radiata* (cultivated mung bean); *Vigna unguiculata* (L.) Walpers ssp. *unguiculata* (cowpea); *Lablab purpureus* (L.) Sweet; and *Vicia faba* L. (horse bean) (Mordue, 1971a and 1971b; Onesirosan and Barker, 1971; Sherf and MacNab, 1986; Walker, 1950; Zaumeyer and Thomas, 1957). Common names frequently used for anthracnose in Latin America are "antracnosis," "antracnose," and "l'anthracnose" in Spanish, Portuguese, and French, respectively.

Etiology

Imperfect stage. Conidia are borne in an acervulus which may be present on pods, leaves, stems, and branches. Acervuli are round or elongated, attaining about 300 μm in diameter. They may be intra- and subepidermal, disrupting outer epidermal cell walls of the host. Occasional cells of an acervulus develop as setae which are brown, septate, and slightly swollen at the base to taper gently to the rounded paler apex. Setae are 4-9 μm wide and usually less than

Digitized by Google

100 μm long. They may be present in culture or on the host at the margin of an acervulus. Acervuli have pale salmon-colored spore masses. Conidia are unicellular, hyaline, cylindrical with both ends obtuse or with a narrow and truncate base. Conidia are uninucleate, and usually have a clear vacuole-like body near the center. Reported conidial measurements are 11-20 μm by 2.5-5.5 μm; 9.5-11.5 μm by 3.5-4.5 μm; and 4-5 μm by 13-22 μm. Conidia are formed from unbranched unicellular hyaline or faintly brown cylindrical phialidic conidiophores 40-60 μm in length. A conidium germinates in six to nine hours and produces one to four germ tubes. The germ tubes form appressoria at their tips during pathogenesis (Walker, 1950; Zaumeyer and Thomas, 1957). The appresoria, infrequently found, are pale to dark brown, clavate or circular in outline, and are borne on supporting hyphae that are hyaline and thin-walled (Mordue, 1971a and 1971b; Sutton, 1980).

Optimal fungal growth in culture occurs at 22.5 °C (Leakey and Simbwa-Bunnya, 1972). On potato dextrose agar (PDA), growth is slow, only about 6 cm in diameter in 10 days at 22-24 °C. Colonies are hyaline to gray at first, rapidly becoming dark to nearly black, and have compact aerial mycelium upon maturity. The most favorable temperature for conidial production on snap bean pods is between 14-18 °C. Production is severely limited or stops at temperatures greater than 30 °C (Zaumeyer and Thomas, 1957). Sporulation is favored at pH 5.2-6.5 and is unaffected by aeration or ultraviolet light (Mathur et al., 1950). Bean pod agar, PDA, Czapeck medium, and sterilized pods are most often used for growth and sporulation (Edgerton, 1910 and 1915; Zaumeyer and Thomas, 1957). Some isolates sporulate only when grown on a medium containing glucose, mineral salts, and neopeptone (Mathur et al., 1950). Isolates may lose viability and pathogenicity when repeatedly transferred in culture, unless occasionally reisolated from inoculated plants or stored under low temperatures. Hwang et al. (1968) stored isolates for 30 months at -150 °C to -196 °C with no loss in viability or pathogenicity.

Perfect stage. The perfect stage, consisting of perithecia and asci, was found in cultures obtained from beans with anthracnose symptoms (Shear and Wood, 1913). Although pathogenicity was not demonstrated in the perithecia-producing isolates, Shear and

Digitized by Google

Wood believed the isolates constituted the perfect stage of *C. lindemuthianum*. They named it *Glomerella lindemuthianum* Shear. The sexual stage was rediscovered in 1970 by Kimati and Galli who paired two isolates to produce perithecia. Because these asci-producing isolates were pathogenic only to beans and morphologically indistinguishable from *G. cingulata*, they named the perfect stage *Glomerella cingulata* (Stonem.) Spauld. *et* Schrenk. f. *phaseoli.*

Paradela-Filho and Pompeu (1974) reported that a different species of *Colletotrichum* was isolated from bean plants showing anthracnose symptoms in Brazil. Seedlings of Dark Red Kidney, Michelite, and Perry Marrow beans, inoculated with isolates of this pathogen, showed anthracnose symptoms. They identified the fungus as *C. dematium* f. *truncata* (Schw.) von Arx., the soybean anthracnose pathogen. This pathogen has hyaline, curved-shaped, unicellular conidia that measure 27 μm by 3.5 μm. It also has setae among the conidiophores. Dr. M. A. Pastor-Corrales (unpublished data) has also isolated a fungus very similar to that described by Paradela-Filho and Pompeu, from bean leaves in Colombia. The leaves showed long streaks of intense reddening on the leaf veins but had none of the typical sunken lesions characteristic of bean anthracnose. Further research is necessary to determine the frequency and importance of this species.

Infectious viral particles have been detected in isolates of *C. lindemuthianum* and transferred to virus-free isolates by hyphal anastomosis (Delhotal et al., 1976). Radial growth and sporulation by infected isolates are reduced but there are no reports of altered pathogenicity.

Epidemiology and Plant Infection

Colletotrichum lindemuthianum can overwinter either in seed or infected crop residues. It can survive for at least two years in seed (Mordue, 1971a and 1971b). However, longevity in infected pods and seeds varies considerably, depending on environmental conditions (Tu, 1983). Moisture is an important factor that influences the survival of the fungus. The fungus survived at least 5 years on pods

Digitized by Google

and seeds that were air-dried and kept in storage at 4 °C or on dry infected plant materials left in the field in sealed polyethylene envelopes that prevented contact with water. An alternating wet-dry cycle was detrimental to fungal survival (Tu, 1983). *Colletotrichum lindemuthianum* survives as dormant mycelium within the seed coat, sometimes even in cells of cotyledons, as spores between cotyledons, or elsewhere in the seed (Zaumeyer and Meiners, 1975). It is capable of withstanding temperatures of -15 °C to -20 °C for a limited period (Mordue, 1971a and 1971b).

Temperature and humidity conditions are important for infection and expression of symptoms. Infection by *C. lindemuthianum* is favored by moderate temperatures between 13 and 26 °C (Crispín-Medina et al., 1976; Ferrante and Bisiach, 1976; Hwang et al., 1968; Lauritzen, 1919; Vieira, 1967; Zaumeyer and Thomas, 1957), with an optimum of 17 °C (Lauritzen, 1919) to 24 °C (Tu and Aylesworth, 1980). Infection by and development of the pathogen is delayed or prevented by temperatures outside the range of about 7-33 °C (Lauritzen et al., 1933; Rahe and Kuć, 1970; Salazar and Andersen 1969; Tu and Aylesworth, 1980). Humidity of more than 92% or free moisture is required during all stages of conidium germination, incubation, and subsequent sporulation (Ferrante and Biasiach, 1976; Lauritzen, 1919; Mordue, 1971a and 1971b; Tu, 1982; Zaumeyer and Thomas, 1957). Moderate rainfalls at frequent intervals, particularly when accompanied by wind or splashing rain, are essential for local dissemination of conidia and for development of severe anthracnose epidemics (Zaumeyer and Thomas, 1957). The rain dissolves the water-soluble gelatinous matrix in which the conidia rest in the acervulus.

In Ontario, the anthracnose pathogen required about 10 mm of rain to establish infection. Long-distance dissemination (3-5 m) may result from splashing raindrops blown by gusting winds (Tu, 1981). Conidia also may be dispersed within the crop by movement of insects, animals, and man, especially when plant foliage is moist (Zaumeyer and Thomas, 1957).

Araya-Fernández (1981) reported that the number of foci of the initial inoculum in the field was linearly related to the anthracnose incidence on leaves, but was not related to incidence on pods.

Digitized by Google

Similarly, under field conditions during the rainy season, anthracnose incidence was higher on leaves, whereas during the dry season, incidence was higher on pods. A conidium germinates in six to nine hours under favorable environmental conditions to form a germ tube and appressorium which attaches to the host cuticle by a gelatinous layer (Dey, 1919; Walker, 1950; Zaumeyer and Thomas, 1957). The pathogen penetrates the cuticle and epidermis mechanically with the appressorium (Dey, 1919; Leach, 1923; Zaumeyer and Thomas, 1957). Following penetration of host cells, when temperatures are favorable, infectious hyphae enlarge and grow between the cell wall and protoplast for two to four days without apparent damage to host cells.

Several days later, cell walls are degraded, probably by L-galactosidase (English and Albersheim, 1969) and protoplasts disorganize and collapse. Water-soaked lesions appear (Leach, 1923; Mercer et al., 1975; Zaumeyer and Thomas, 1957) which later turn dark brown because of a high content of tannins (Cárdenas-Soriano and Engleman, 1981). Mycelium may then mass within the lesion site and form acervuli which rupture the host cuticle. The acervulus contains a stromatic layer of three to 50 conidiophores, depending upon the lesion size (Zaumeyer and Thomas, 1957). Numerous conidia are formed and embedded in a water-soluble gelatinous matrix in each acervulus. Newly produced conidia are more infectious than older ones (Sindhan and Bose, 1981).

Symptomatology

Symptoms of anthracnose can appear on any plant part. Initial symptoms may appear on cotyledonary leaves as small, dark brown to black lesions. Conidia and hyphae are transported by rain or dew to the developing hypocotyl. The infected tissues manifest minute rust-colored specks. The specks gradually enlarge longitudinally and form sunken lesions or eye-spots. These enlarge on the hypocotyl of the young seedling, causing it to rot off. On older stems, the eye-shaped lesion is about 5-7 mm in length.

Lesions may first develop on leaf petioles and the lower surface of leaves and leaf veins as small, angular, brick-red to purple spots

Digitized by Google

which become dark brown to black (Figure 8). Later, the lesions may also appear on veinlets on the upper surface of leaves (Figure 9). Sporulation can occur in lesions on the petiole and larger leaf veins, thereby producing secondary inoculum (Zaumeyer and Thomas, 1957). Pod infections appear as flesh to rust-colored lesions. The lesions develop into sunken cankers (1-10 mm in diameter) that are delimited by a slightly raised black ring and surrounded by a reddish brown border (Figure 10).

The lesion center is light colored and, during periods of low temperature and high moisture, may contain a gelatinous mass of flesh-colored conidia. With age, the conidia dry up, becoming gray-brown or black granulations. If severely infected, young pods shrivel and dry up. The fungus can invade the pod, and the mycelia and conidia infect the cotyledons or seed coat of the developing seeds (Figure 11). Infected seeds are often discolored and may contain dark brown to black cankers (Figure 12) (Zaumeyer and Thomas, 1957).

Control by Cultural Practices

Anthracnose-free bean seed has been produced and used in various regions of the world to control the disease (Copeland et al., 1975; Costa, 1972; Crispín-Medina et al., 1976; Issa et al., 1964; Zaumeyer and Meiners, 1975; Zaumeyer and Thomas, 1957). Pathogen-free seed of susceptible cultivars is produced with surface or furrow irrigation in semiarid regions. The high temperature and low humidity conditions are unfavorable for infection by and survival of the anthracnose fungus. Although the use of pathogen-free seed considerably reduces losses, few developing countries in Latin America or Africa possess either the seed-production areas and/or the facilities necessary to produce and distribute clean seed to growers (Vieira, 1967, Zaumeyer and Thomas, 1957). Obviously, this would change if semiarid areas are found that have the right altitude and suitable isolation. Although heat treatment of contaminated seed at 50-60 °C successfully eliminates the fungus, seed viability is significantly reduced (Zaumeyer and Thomas, 1957).

Crop rotations of two to three years are recommended because the pathogen can survive in infected crop debris for two or more

Digitized by Google

years (Tu, 1983; Zaumeyer and Thomas, 1957 and 1962). However, the value of this practice has been questioned in the light of some carefully conducted experiments. When infected plant materials were placed in nylon-mesh pouches and buried in the field in November, *C. lindemuthianum* could not be isolated after mid-May (Tochinai and Sawada, 1952; Tu, 1983). An alternating 72-hr wet-dry cycle was detrimental to fungal survival. The fungus in infected pod segments lost viability after three cycles of 72 hours of dryness (Tu, 1983). Moreover, beans planted on sites where plants were heavily infected the previous year did not develop symptoms of anthracnose (Tu, 1983). Infected plant debris must be removed from the field soon after harvest (Crispín-Medina et al., 1976). It is also important to restrict the activity and movement of men and agricultural implements in a field when the foliage is wet from rain or dew (Vieira, 1967).

Control by Chemicals

Various chemical treatments have been used for seed treatment. Seed-coat infestations are controlled effectively with Ferbam, ziram (Crispín-Medina et al., 1976), thiram (Costa, 1972), and Ceresan (0.5 g/100 g of seed). However, internal seed contamination is not reduced (Zaumeyer and Thomas, 1957). Recently, formulations with benomyl or thiophanate methyl were used to treat seeds. When they were applied at 5.2 g/kg of seed, better than 95% control was achieved (Edgington and French, 1981; Edgington and MacNeill, 1978; Tu, 1986).

Preventive spraying with protective or systemic fungicides has been attempted with limited success (Issa and de Arruda, 1964; Simbwa-Bunnya, 1972; Stevenson, 1956; Zaumeyer and Thomas, 1957). Maneb (Costa, 1972; Crispín-Medina et al., 1976; Issa and de Arruda, 1964; Zaumeyer and Thomas, 1962) and zineb at 3.5 g/L (Crispín-Medina et al., 1976; Peregrine, 1971; Zaumeyer and Thomas, 1957), benomyl at 0.55 g/L (CIAT, 1977; Giroto, 1974), captafol at 3.5 kg/ha (Guzmán-Vargas and de la Rosa, 1975), carbendazim at 0.5 kg/ha (CIAT, 1977), and fentin hydroxide at 1.2 g/L (Peregrine, 1971) have been used to control anthracnose.

Digitized by Google

Combination and rotation of these fungicides is more effective than continually using a single fungicide (Guzmán-Vargas et al., 1979; Navarro-A. et al., 1981).

Crispín-Medina et al. (1976) recommended spraying foliage at flower initiation, late flowering, and pod-filling to achieve satisfactory disease control. However, continuous use of fungicides may encourage the development of resistant biotypes (Tu and Mc Naughton, 1980). Fungicides are also expensive and therefore have limited availability in Latin American or African bean production.

Control by Plant Resistance

Barrus (1911) reported that some bean cultivars were susceptible to anthracnose while others were resistant. He also reported (1918) that bean cultivars differed in their reaction to *C. lindemuthianum* and that the anthracnose fungus was pathogenically variable. He later categorized his isolates into two distinct physiologic races, calling them alpha and beta.

Since then, many surveys have been made throughout the world to identify the prevalence and distribution of specific races. The results have confirmed that extensive pathogenic variation of *C. lindemuthianum* exists on all continents. Unfortunately, workers have used different sets of differential cultivars, making it difficult to compare their data. Race designations have been based on the reactions of different host cultivars, differing in their genes for resistance, when inoculated with one or more races of the anthracnose pathogen (Zaumeyer and Meiners, 1975). In 1923, Burkholder reported from United States the gamma race. Also from the United States, Leach (1923) reported eight distinct races, apparently different from those previously reported by Barrus and Burkholder. Andrus and Wade (1942) reported the delta race.

In France, Blondet (1963), according to Charrier and Bannerot (1970), reported a new race called "epsilon" (Schnock, 1975). Fouilloux (1975) reported that an isolate of *C. lindemuthianum* obtained from Brazil was a new race: he called it alpha-brazil. A mutant of the alpha race (designated alpha-5N) was later named "lambda" by Hubbeling (1976). Schnock (1975) reported another

Digitized by Google

new physiological strain of *C. lindemuthianum* designated as "ebnet" and subsequently renamed as the "kappa" race (Krüger et al., 1977). Similarly, Hubbeling (1977) reported isolating the iota race, which apparently does not occur under field conditions, from kappa-resistant seedlings inoculated under greenhouse conditions with a mixture of gamma, delta, kappa, and lambda races. Fouilloux (1979) reported a new race he obtained from Hubbeling that was named "lambda-mutant." Races alpha, beta, gamma, delta, epsilon, and lambda have been reported in Canada, France, Holland, and Uganda (Charrier and Bannerot, 1970; Hubbeling, 1957; Leakey and Simbwa-Bunnya, 1972; Müller, 1926; Tu et al., 1984).

In France, Bannerot (1965) has designated races as PV6, D10, F8b, I4, 1, and 5. The first five correspond to alpha, beta, gamma, delta, and epsilon, respectively. The race 5 has the pathogenicity of gamma and delta. In Germany, reported races have been designated as A-E, G-N, and X by Peuser (1931) and as alpha, beta, and gamma by Schreiber (1932). In Italy, the alpha, beta, gamma, delta, and epsilon are known to occur (Ferrante and Bisiach, 1976). In Australia, races have been designated Aust-1 through to Aust-8 (Waterhouse, 1955) or simply as races 1, 2, and 3 (Cruikshank, 1966).

In Latin America, a few reports suggest that *C. lindemuthianum* is very variable pathogenically. In Mexico, most workers use three American (Michelite, Dark Red Kidney, and Perry Marrow) and five Mexican (Negro 150 and 152, Amarillo 155, Bayo 164, and Canario 101) differential cultivars to classify their isolates. Yerkes and Teliz-Ortiz (1956) reported races alpha, beta, gamma, and ten new isolates. Races MA-1 to MA-6 were classified as belonging to Mexico group I; MA-7 to Mexico group II, and MA-8 to MA-10 to Mexico group III. Yerkes (1958) reported that races MA-11 to MA-13 correspond to a group to be denominated as alpha. Gallegos cited by Villada-Ramos (1982) reported races MA-14 and MA-15 as belonging to the alpha group which correspond roughly to the alpha race; MA-16 to Mexico group I; MA-17 to group II; MA-18 to the beta race; MA-19 and MA-20 to a new group denominated as Mexico group IV. Martínez (1982) also reports MA-14 and MA-15 as new races. However, MA-15 elicited the same reaction as the

Digitized by Google

races belonging to the group alpha. Noyola et al. (1984), cited by Garrido (1986), reported races MA-21 and MA-22 as belonging to the alpha group. Garrido (1986) reported eight new races where MA-23 to MA-25 belong to the group alpha and MA-26 to MA-30 to Mexico group I.

In Brazil, reported races were alpha, beta, gamma, epsilon, lambda, kappa, zeta, teta, eta, mu, Mexico groups I and II, and Brazil groups I, II, and III. In addition, some isolates have been further characterized into 10 different races denominated as BA-1 to BA-10 and belonging the following race groups: BA-1 and BA-2 in alpha; BA-3 in Brazil II; BA-4 and BA-5 in Brazil I; BA-6, BA-7, and BA-8 in Mexico II; BA-9 in Mexico I; and BA-10 in delta (Augustin and da Costa, 1971; de Araújo, 1973a and 1973b; de Menezes, 1985; de Menezes et al., 1982; Kimati, 1966; Oliari et al., 1973; Oliveira et al., 1973; Pio-Ribero and Chaves, 1975; Ribeiro et al., 1981). None of these isolates caused symptoms on Cornell 49-242 and the reaction of BA-3 is the same as that of isolates belonging to group alpha. The separate categorizing of BA-3 is, therefore, not warranted. Races alpha, beta, and gamma occur in Chile (Mujica, 1952) and the beta and gamma races are prevalent in Colombia (CIAT, 1976 and 1977).

Other races of *C. lindemuthianum* have been detected in Latin America. In Brazil, Dr. Carlos Rava, Centro Nacional de Pesquisa de Arroz e Feijão, Goiânia (personal communication), and Dr. M. A. Pastor-Corrales (unpublished data) have collected and characterized isolates similar to alpha-Brazil (Fouilloux, 1975) which had not been previously detected in Brazil. A similar characterization was conducted for 15 isolates from Mexico. Reported races were Brazil group I, alpha, Brazil, and Mexico group 1 (Bolaños, 1984; CIAT, 1984). From Colombia, 17 isolates were characterized as beta, delta, kappa, alpha-Brazil, Mexico group II, and two isolates that did not belong to any known race (Cobo-Soto, 1986). Recently, in a cooperative effort between CIAT and the University of Costa Rica, three isolates from the northern region of Costa Rica were characterized as alpha-Brazil and three from the central region as kappa and Brazil group I.

It is therefore apparent that considerable pathogenic variation exists throughout the world. However, an international set of

Digitized by Google

differential cultivars and race designations must be developed to coordinate the research efforts of all workers and to facilitate the exchange of data and resistant germplasm.

Physiology of the Host-Parasite Interaction

A lot of research has focused on the host-pathogen interaction when a specific cultivar is infected by a specific race (pathogenic or nonpathogenic). Griffey and Leach (1965) inoculated cultivars of different ages which were differentially susceptible or resistant to various races. They found that the small necrotic lesions formed on old tissue of susceptible cultivars were similar to lesions on young tissue of resistant cultivars. They concluded that the former reaction was a result of plant maturation, while the latter reaction resulted from a specific protoplasmic response. The fungus develops more slowly in a resistant cultivar than in a susceptible one. The resistant plant therefore has more time to develop its defense reaction (Arnold and Rahe, 1976; Bailey, 1974; Bailey and Deverall, 1971). Also, the pathogen did not produce cell-wall degrading enzymes such as L-galactosidase, as early or as much as in susceptible cultivars (Elliston et al., 1976; English and Albersheim, 1969).

Inoculation with a nonpathogenic race may protect the host from subsequent infection by a pathogenic race (Elliston et al., 1976; Skipp and Deverall, 1973; Sutton, 1979). However, this protection is confined only to tissue actually infected previously by the nonpathogenic race (Skipp and Deverall, 1973). Also, inoculation with a pathogenic race at a low inoculum concentration or under conditions unsuitable for disease development induces a systemic cross protection against the same pathogen (Sutton, 1979). Injury by mechanical means (Arnold and Rahe, 1977; Ferrante and Bisiach, 1976) and freezing of local tissue can also induce localized protection. Such protection is probably regulated by a different mechanism than that operating in the inoculation with a nonpathogenic race (Rahe and Arnold, 1975).

Heat treatment (32-37 °C) of tissue before inoculation can also confer local and systemic protection which is not race-specific (Elliston et al., 1977; Rahe, 1973a; Rahe and Kuć, 1970). Heat treatment diminished the effectiveness of resistance of mature

Digitized by Google

tissue, but not of race-specific resistance or local protection. This suggests there may be two groups of resistance mechanisms operating (Elliston et al., 1976 and 1977). Ultraviolet irradiation applied to bean hypocotyls has altered the expression of disease response of treated cultivars. Induced resistance is accompanied by an accumulation of phytoalexins (Andebrhan and Wood, 1980).

Plant metabolites such as phaseolin (inhibitory to *C. lindemuthianum* in vivo), accumulate earlier in resistant than in susceptible plants (Bailey and Deverall, 1971; Rahe, 1973b; Rahe et al., 1969; Theodorou et al., 1982). Phaseolin and the related isoflavanoid compounds, phaseolidin, phaseolinisoflacan, and kievitone, accumulate in tissue infected by both pathogenic or nonpathogenic races (Bailey, 1974).

Phenylalanine ammonia lyase levels increase in tissue before lesion formation and is probably related to the subsequent production of compounds such as phaseolin, other isoflavonoids, and coumestrol (Rathmell, 1973). Phaseolin at low concentrations in vitro is highly inhibitory to spore germination and germ-tube growth. However, mycelial growth is less sensitive to it (Bailey, 1974) because phaseolin is metabolized into less toxic compounds such as 6a-hydroxyphaseolin, 6a-7-dihydroxyphaseolin, and others (van den Heuvel and Vollaard, 1976). Electron microscopy shows that intracellular hyphae in hypersensitive cells are dead (Landes and Hoffman, 1979). However, light microscopy suggests that some hyphae remain alive and continue to grow slowly for some time after phytoalexin accumulation has occurred (Bailey and Rowell, 1980; Erb et al., 1973; Skipp and Deverall, 1973). This apparent discrepancy may have resulted from samples being taken from different areas of a diseased lesion, or it may show that not all hyphae are killed by the hypersensitive reaction.

Inheritance and Sources of Resistance

The most appropriate and practical control of bean anthracnose, particularly in developing countries, is the use of field-resistant cultivars (Figure 13). Several resistance sources have been used extensively in United States, Canada, Europe, and in some countries of Africa and Latin America (Andersen et al., 1963;

Digitized by Google

Augustin and da Costa, 1971; Bannerot et al., 1971; Fouilloux, 1976; Hubbeling, 1957; Leakey and Simbwa-Bunnya, 1972). However, only recently has there been much effort directed toward incorporating resistance into commercial cultivars in Latin America (Augustin and da Costa, 1971; CIAT, 1984; de la Garza, 1951).

Resistance to the alpha and beta races is controlled by a single, independent dominant gene (McRostie, 1919 and 1921) which has been combined in cultivars such as Charlevoix (Andersen et al., 1963). Although Burkholder (1918) reported that resistance to the gamma race is conferred by a single dominant gene, resistance to the beta, gamma, and delta races appears more complex. It is governed by a system of 10 genes in three allelomorphic series which are composed of duplicate genes for resistance, a dominant gene for susceptibility, and interaction at three loci (Andrus and Wade, 1942). Similarly, Cárdenas et al. (1964) concluded that the resistance to races alpha, beta, and gamma was conferred by duplicate and complementary factors, as well as by multiple alleles. Muhalet et al. (1981) reported that the inheritance of resistance to beta, gamma, and delta races in crosses involving Cornell 49-242 and Kaboon was conferred by independent and complementary gene action at one or two different loci. In addition, it was also assumed that an allelomorphic series of three alleles controlled resistance to the beta race.

Among the resistance sources, Cornell 49-242 (a Venezuelan black-seeded bean) is resistant to the races alpha, beta, gamma, delta, epsilon, and lambda by virtue of a single dominant ARE gene (Ayonoadu, 1974; Bannerot, 1965; Goth and Zaumeyer, 1965; Krüger et al., 1977; Mastenbroek, 1960; McRostie, 1919; Muhalet et al., 1981). However, it is susceptible to alpha-Brazil, kappa, and jota races (Fouilloux, 1976; Hubbeling, 1977). It also has certain undesirable horticultural features (Muhalet et al., 1981; Zaumeyer and Meiners, 1975) which have been overcome by transferring the ARE gene into adapted high-yielding cultivars (Muhalet et al., 1981; Zaumeyer and Meiners, 1975). Fouilloux and Bannerot (1977) created four pairs of isogenic lines derived from Cornell 49-242 with no apparent unfavorable pleiotropic effects. However, the appearance, first, of the kappa race and, later, of alpha-Brazil in Europe and Latin America that attack Cornell 49-242 meant that

Digitized by Google

the extensive use of this gene throughout the world and, particularly, in Latin America was dangerous. This realization stimulated several scientists to identify new sources of resistance to many or all known races. In Europe, they reported that Mexico 222 and Mexico 227 contain the dominant gene Mexique 1 which may be composed of an allelic series (Bannerot et al., 1971; Fouilloux, 1979). The Mexique 1 gene, different and independent of the ARE gene, is resistant to alpha, beta, gamma, delta, epsilon, lambda, and kappa, but not to alpha-Brazil. However, only Mexico 222 has the resistance gene Mexique 1 and Mexico 227 is not resistant to either the kappa or alpha-Brazil race (Fouilloux, 1979).

In 1972, in France, six other lines obtained from Mexico and resistant to all European races were reported (Fouilloux, 1979). The line TO had the anthracnose resistance gene Mexique 2 which is different and independent of ARE and Mexique 1 resistance genes. The other five lines, TU, TV, TX, TY, and TW, have the Mexique 3 gene resistant against all European races. Mexique 3 is different and independent of resistance genes ARE, Mexique 1, and Mexique 2. Resistance to races alpha, delta, and kappa occurs in Kaboon, Coco à la Crème, Keit, Koekoek, BO-22, and Evolutie (Bannerot and Richter, 1968; Krüger et al., 1977). P.I. 150414, Titan, and Metorex are moderately resistant to kappa, while an unspecified accession of *P. coccineus* is resistant to all known races (Krüger et al., 1977). In addition, P.I. 165426 and P.I. 207262 are resistant to kappa and iota (Hubbeling, 1977).

Several bean varieties resistant to many or all known European races of the anthracnose pathogen such as Mexico 222, TO, and TU, which have the single resistance genes Mexique I, Mexique II, and Mexique III, respectively, and lines such as P.I. 207262, which are resistant to kappa and iota races, are nevertheless susceptible to several Latin American isolates. Because of the extensive pathogenic variation of *C. lindemuthianum*, particularly in the Americas, and because so many bean varieties and lines are susceptible to American isolates of the pathogen, scientists at CIAT, Colombia, have evaluated several thousand lines. They identified better and different sources of resistance (CIAT, 1984; Schwartz et al., 1982) under field and greenhouse conditions. Among those bean lines and

Digitized by Google

germplasm accessions that showed broad resistance are A 193, A 252, A 321, A 475, A 483, AB 136, K 2, G 811, G 984, G 2333, G 2338, G 2641, G 3367, Ecuador 1056 (G 12488), and Gloriabamba (G 2829). Similarly, it has been possible to identify lines with excellent resistance in several, although not all, locations such BAT 841, BAT 93, and G 5653.

Workers have relied completely upon race-specific resistance to manage specific races of *C. lindemuthianum*. However, the fungus has expressed considerable pathogenic variation by mutation, natural selection, or other mechanisms. Mycelium of nonpathogenic races can also survive in lesions in resistant tissue for as many as 25 days. Possibly, this facility leads to the development and selection of new pathogenic races (Erb et al., 1973). Therefore, bean pathologists and breeders must work together to effectively identify better and broader sources of resistance in many locations throughout the world. They must incorporate a very broad and diverse group of anthracnose resistance sources into breeding programs. It is also essential that uniform methodology be used to evaluate bean germplasm reactions to the anthracnose pathogen in order to select lines or cultivars that are truly resistant and not to discard useful germplasm. For example, the cultivar ICA Llanogrande (Ecuador 1056) has been evaluated as resistant by the senior author under field conditions in many locations of Latin America and Africa. However, it is very susceptible to the same isolates under greenhouse conditions.

Because anthracnose is important in many large bean-producing regions of the world, because the fungus has extensively pathogenic variation, and because European resistance sources are susceptible to Latin American races of the pathogen, bean workers must coordinate their efforts to properly evaluate the extent of the pathogenic variation in the different regions where anthracnose occurs recurrently. Bean workers must also use identical bean differential varieties to permit the development of an international race designation that can compare results and can evaluate, in many sites, the resistance sources. In this manner, bean varieties that are resistant to a broad range of anthracnose isolates can be identified. This, in turn, would allow the development of a broad and diverse

Digitized by Google

strategy, that emphasizes genetic resistance, to manage this very important bean disease.

References

Andebrhan, T. and Wood, R. K. S. 1980. The effect of UV radiation on the reaction of *Phaseolus vulgaris* to species of *Colletotrichum*. Physiol. Plant Pathol. 17(1):105-110.

Andersen, A. L.; Adams, M. W.; and Whitford, G. [1963]. Charlevoix anthracnose-resistant Dark Red Kidney bean. Mich. Agric. Exp. Stn. Res. Rep. 6:1-3.

Andrus, C. F. and Wade, B. L. 1942. The factorial interpretation of anthracnose resistance in beans. Technical bulletin 810. United States Department of Agriculture, Washington, DC, USA. 29 p.

Araya-Fernández, C. M. 1981. Influência do número de focos de inóculo inicial de *Colletotrichum lindemuthianum* (Sacc. & Magn.) Scrib. no desenvolvimento de antracnose em folhas, vagens e sementes de *Phaseolus vulgaris* L. M.Sc. thesis. Universidade Federal de Viçosa, Viçosa, MG, Brazil. 40 p.

Arnold, R. M. and Rahe, J. E. 1976. Effects of 15% CO_2 on the susceptibility of *Phaseolus vulgaris* to *Colletotrichum lindemuthianum*. Can. J. Bot. 54(10):1035-1043.

——— and ——— 1977. Effects of 15% CO_2 on the accumulation of the phytoalexin phaseollin in *Phaseolus vulgaris* in response to mechanical injury and to infection by *Colletotrichum lindemuthianum*. Can. J. Bot. 55(8):867-871.

Augustin, E. and da Costa, J. G. Cáprio. 1971. Fontes de resistência a duas raças fisiológicas de *Colletotrichum lindemuthianum* no melhoramento do feijoeiro no Sul do Brasil. Pesqui. Agropecu. Bras. 6:265-272.

Ayonoadu, U. W. U. 1974. Races of bean anthracnose in Malawi. Turrialba 24(3):311-314.

Bailey, J. A. 1974. The relationship between symptom expression and phytoalexin concentration in hypocotyls of *Phaseolus vulgaris* infected with *Colletotrichum lindemuthianum*. Physiol. Plant Pathol. 4(4):477-488.

Digitized by Google

——— and Deverall, B. J. 1971. Formation and activity of phaseollin in the interaction between bean hypocotyls (*Phaseolus vulgaris*) and physiological races of *Colletotrichum lindemuthianum.* Physiol. Plant Pathol. 1(4):435-449.

——— and Rowell, P. M. 1980. Viability of *Colleotrichum lindemuthianum* in hypersensitive cells of *Phaseolus vulgaris.* Physiol. Plant Pathol. 17(3):341-345.

Bannerot, H. 1965. Résultats de l'infection d'une collection de haricots par six races physiologiques d'anthracnose. Ann. Amélior. Plant. (Paris) 15(2):201-222.

——— and Richter, R. 1968. Étude de la descendance de deux croisements de haricot pour leur résistance à différentes races physiologiques d'anthracnose. Ann. Amélior. Plant. (Paris) 18(2):171-179.

———; Derieux, M.; and Fouilloux, G. 1971. Mise en évidence d'un second gène de résistance totale à l'anthracnose chez le haricot. Ann. Amélior. Plant. (Paris) 21(1):83-85.

Barrus, M. F. 1911. Variation of varieties of beans in their susceptibility to anthracnose. Phytopathology 1(6):190-195.

———. 1918. Varietal susceptibility of beans to strains of *Colletotrichum lindemuthianum* (Sacc. & Magn.) B. & C. Phytopathology 8(12):589-614.

Blondet, A. 1963. L'anthracnose du haricot: étude des races physiologiques du *Colletotrichum lindemuthianum.* Thesis. Faculté de Science, Paris, France. 160 p.

Bolaños, J. I. 1984. Variación patogénica de aislamientos mexicanos de *Colletotrichum lindemuthianum* (Sacc. & Magn.) Scrib., agente causal de la antracnosis del frijol común (*Phaseolus vulgaris*). Thesis. Universidad Nacional de Colombia, Palmira, Colombia. 56 p.

Burkholder, W. H. 1918. The production of an anthracnose-resistant White Marrow bean. Phytopathology 8(7):353-359.

———. 1923. The gamma strain of *Colletotrichum lindemuthianum* (Sacc. *et* Magn.) B. *et* C. Phytopathology 13(7):316-323.

Cárdenas, F.; Adams, M. W.; and Andersen, A. L. 1964. The genetic system for reaction of field beans (*Phaseolus vulgaris* L.) to infection

Digitized by Google

by three physiologic races of *Colletotrichum lindemuthianum.* Euphytica 13(2):178-186.

Cárdenas-Soriano, E. and Engleman, E. M. 1981. Cambios anatómicos en la semilla de *Phaseolus vulgaris* durante la infección por *Colletotrichum lindemuthianum.* Rev. Mex. Fitopatol. 1(1):9-15.

Charrier, A. and Bannerot, H. 1970. Contribution à l'étude des races physiologiques de l'anthracnose du haricot. Ann. Phytopathol. 2:489-506.

Chaves, G. 1980. Anthracnose. In: Schwartz, H. F. and Gálvez, G. E. (eds.). Bean production problems: disease, insect, soil and climatic constraints of *Phaseolus vulgaris.* CIAT series no. 09EB-1. Centro Internacional de Agricultura Tropical (CIAT), Cali, Colombia. p. 37-54.

CIAT (Centro Internacional de Agricultura Tropical). [1976]. Bean Production Systems. In: Annual report 1975. Cali, Colombia. p. C-41 to C-43.

———. [1977]. Bean Production Systems Program. In: Annual report 1976. Cali, Colombia. p. A-9.

———. 1981. Potential for field beans in eastern Africa: proceedings of a regional workshop held in Lilongwe, Malawi, 9-14 March 1980. CIAT series 03EB-1. Cali, Colombia. 226 p.

———. [1984]. Annual report 1983: Bean Program. Cali, Colombia. p. 22-29.

Cobo-Soto, F. 1986. Variación patogénica y fuentes de resistencia a *Colletotrichum lindemuthianum* (Sacc. & Magn.) Scrib., patógeno de la antracnosis del frijol en Colombia. Thesis. Universidad Nacional de Colombia, Palmira, Colombia. 93 p.

Copeland, L. O.; Adams, M. W.; and Bell, D. C. 1975. An improved seed programme for maintaining disease-free seed of field beans *(Phaseolus vulgaris).* Seed Sci. Tech. 3:719-724.

Costa, A. S. 1972. Investigações sobre moléstias do feijoeiro no Brasil. In: Anais do I simpósio brasileiro de feijão, Campinas, 22 à 29 de agôsto de 1971, 2 vols. Universidade Federal de Viçosa, Viçosa, MG, Brazil. Vol. 2, p. 306-311.

Digitized by Google

Crispín-Medina, M. A. and Campos-Avila, J. 1976. Bean diseases of importance in Mexico in 1975. Plant Dis. Rep. 60(6):534-535.

———; Sifuentes-A., J. A.; and Campos-Avila, J. 1976. Enfermedades y plagas del frijol en México. Rev. ed. Folleto de divulgación no. 39. Instituto Nacional de Investigaciones Agrícolas, Secretaría de Agricultura y Ganadería, Mexico City, Mexico. p. 4-6.

Cruickshank, I. A. M. 1966. Strains of *Colletotrichum lindemuthianum* (Sacc. and Magn.) in eastern Australia. J. Aust. Inst. Agric. Sci. 32(2):134-135.

de Araújo, I. D. 1973a. Identificação das raças de antracnose *Colletotrichum lindemuthianum* (Sacc. & Magn.) Scrib. e pesquisa, de fontes de resistência no Paraná. Pesqui. Agropecu. Bras. 8(1):13.

———. 1973b. Identificação da raça alfa do *Colletotrichum lindemuthianum* e reação de cultivares de feijoeiro (*Phaseolus vulgaris*). Pesqui. Agropecu. Bras. 8(7):159-162.

de la Garza, M. de los A. M. 1951. Reacción de frijol en México a tres razas de *Colletotrichum lindemuthianum.* Folleto técnico 9. Secretaría de Agricultura y Ganadería, Mexico City, Mexico. 29 p.

de Menezes, J. R. 1985. Variabilidad patogénica de *Colletotrichum lindemuthianum* (Sacc. & Magn.) Scrib. en *Phaseolus vulgaris* L. M.Sc. thesis. Universidade de Brasília, Brasília, DF, Brazil. 65 p.

———; Mohan, S. K.; and Bianchini, A. 1982. Identificação de raças fisiológicas de *Colletotrichum lindemuthianum* (Sacc. Magn.) Scrib., no Estado do Paraná. In: Anais da primera reunião nacional de pesquisa de feijão, 10 à 15 de janeiro de 1982, Goiânia, Goiás. Brazil. p. 297-299.

Delhotal, P.; Le Grand-Pernot, F.; and Lecoq, H. 1976. Étude des virus de *Colletotrichum lindemuthianum,* II: transmission des particules virales. Ann. Phytopathol. 8(4):437-448.

Dey, P. K. 1919. Studies in the physiology of parasitism, V: infection by *Colletotrichum lindemuthianum.* Ann. Bot. (Lond.) 33:305-312.

Echandi, E. 1976. Principales enfermedades de hongo del frijol (*Phaseolus vulgaris*) en los trópicos americanos en diferentes zonas ecológicas. Fitopatol. Bras. 1(3):171-177.

Digitized by Google

Edgington, L. V. and French, B. 1981. Failure of benzamidazole seed treatment to control anthracnose of bean. In: Proceedings of the Phytopathological Society Western Ontario Region. Canada. 4 p.

——— and MacNeill, B. H. 1978. Control of bean anthracnose by seed treatment with systemic fungicides. Phytopathol. News 12(10):235. (Abstr.)

Edgerton, C. W. 1910. The bean anthracnose. Louisiana Agric. Exp. Stn. Bull. 119. 55 p.

———. 1915. Effect of temperature on *Glomerella.* Phytopathology 5(5):247-259.

Elliston, J.; Kuć, J.; and Williams, E. B. 1976. A comparative study of the development of compatible, incompatible, and induced incompatible interactions between *Colletotrichum* spp. and *Phaseolus vulgaris.* Phytopathol. Z. 87(4):289-303.

———; ———; and ———. 1977. Effect of heat treatment on the resistance of *Phaseolus vulgaris* to *Colletotrichum lindemuthianum* and *Colletotrichum lagenarium.* Phytopathol. Z. 88(1):43-52.

English, P. D. and Albersheim, P. 1969. Host-pathogen interactions, I: a correlation between α-galactosidase production and virulence. Plant Physiol. (Bethesda) 44(2):217-224.

Erb, K.; Gallegly, M. E.; and Leach, J. G. 1973. Longevity of mycelium of *Colletotrichum lindemuthianum* in hypocotyl tissue of resistant and susceptible bean cultivars. Phytopathology 63(11):1334-1335.

Ferrante, G. M. and Bisiach, M. 1976. Comparazione di metodi per l'infezione sperimentale di *Colletotrichum lindemuthianum* sul fagiolo. Riv. Patol. Veg. 12(3-4):99-118. (English summary.)

Fouilloux, G. 1975. L'anthracnose du haricot: étude des relations entre les pathotypes anciens et nouveaux; étude de nouvelles sources de résistance "totale." In: Réunion Eucarpia Haricot, Versailles, France. Centre National de Recherches Agronomiques, Paris, France. p. 81-92.

———. 1976. Bean anthracnose: new genes of resistance. Bean Improv. Coop. (USA) Annu. Rep. 19:36-37.

———. 1979. New races of bean anthracnose and consequences on our breeding programs. In: Maraite, H. and Meyer, J. A. (eds.). Diseases

Digitized by Google

of tropical food crops. Université Catholique de Louvain-La-Neuve, Belgium. p. 221-235.

——— and Bannerot, H. 1977. Four pairs of isogenic lines except for "Are gene." Bean Improv. Coop. (USA) Annu. Rep. 20:59-60.

Garrido, E. R. 1986. Identificación de razas fisiológicas de *Colletotrichum lindemuthianum* (Sacc. & Magn.) Scrib. en México y búsqueda de resistencia genética a este hongo. Thesis. Colegio de Postgraduados, Institución de Enseñanza e Investigación en Ciencias Agrícolas, Montecillos, Mexico.

Giroto, R. 1974. Evaluación de fungicidas en el control de la "antracnosis" y de la "mancha angular" en poroto. IDIA 308:29-32.

Goth, R. W. and Zaumeyer, W. J. 1965. Reactions of bean varieties to four races of anthracnose. Plant Dis. Rep. 49(10):815-818.

Griffey, R. T. and Leach, J. G. 1965. The influence of age of tissue on the development of bean anthracnose lesions. Phytopathology 55(8):915-918.

Guzmán-Vargas, P. and de la Rosa, M. D. 1975. Estudios sobre la antracnosis del frijol (*Phaseolus vulgaris* L.) causada por *Colletotrichum lindemuthianum* (Sacc. & Magn.) Scrib., en la zona de Popayán. Ing. Agr. thesis. Universidad Nacional de Colombia, Palmira, Colombia. 111 p.

———; Donado, M. R.; and Gálvez, G. E. 1979. Pérdidas económicas causadas por la antracnosis del frijol *Phaseolus vulgaris* en Colombia. Turrialba 29:65-67.

Hubbeling, N. 1957. New aspects of breeding for disease resistance in beans (*Phaseolus vulgaris* L.). Euphytica 6(2):111-141. (Errata, 6(3):272.)

———. 1976. Selection for resistance to anthracnose, particularly in respect to the "Ebnet" race of *Colletotrichum lindemuthianum*. Bean Improv. Coop. (USA) Annu. Rep. 19:49-50.

———. 1977. The new jota race of *Colletotrichum lindemuthianum*. Bean Improv. Coop. (USA) Annu. Rep. 20:58.

Hwang, S.-W.; Andersen, A. L.; Papavizas, G. C.; and Converse, R. H. 1968. Cryopreservation and pathogenicity of selected isolates of *Colletotrichum, Rhizoctonia,* and *Phytophthora.* Phytopathology 58(3):362-364.

Digitized by Google

Issa, E. and de Arruda, H. Vaz. 1964. Contribuição para contrôle da ferrugem e da antracnose do fejoeiro. Arq. Inst. Biol. São Paulo 31(4):119-126.

———; Regis, J. N. M.; Vieira, M. L.; de Araújo, J. T.; and Miyasaka, S. 1964. Primeiros estudos para produção de sementes sadias de feijão em regiões áridas do nordeste brasileiro. Arq. Inst. Biol. São Paulo 31(5):21-25.

Kimati, H. 1966. Algumas racas fisiológicas de *Colletotrichum lindemuthianum* (Sacc. *et* Magn.) Scrib. que ocorrem no Estado de São Paulo. An. Esc. Super. Agric. Luiz de Queiroz (São Paulo) 23:247-264.

——— and Galli, F. 1970. *Glomerella cingulata* (Stonem.) Spauld. *et* V. Schrenk. f. sp. *phaseoli* n. f., fase ascógena do agente causal da antracnose do feijoeiro. An. Esc. Super. Agric. Luis de Queiroz (São Paulo) 27:411-437.

Krüger, J.; Hoffman, G. M.; and Hubbeling, N. 1977. The kappa race of *Colletotrichum lindemuthianum* and sources of resistance to anthracnose in *Phaseolus* beans. Euphytica 26:23-25.

Landes, M. and Hoffmann, G. M. 1979. Ultrahistologische untersuchungen über die interaktionen bei kompatiblen und inkompatiblen systemen von *Phaseolus vulgaris* und *Colletotrichum lindemuthianum* (Ultrahistological investigations of the interactions in compatible and incompatible systems of *Phaseolus vulgaris* and *Colletotrichum lindemuthianum*). Phytopathol. Z. 96:330-351.

Lauritzen, J. I. 1919. The relation of temperature and humidity to infection by certain fungi. Phytopathology 9(1):7-35.

———; Harter, L. L.; and Whitney, W. A. 1933. Environmental factors in relation to snap-bean diseases occurring in shipment. Phytopathology 23(5):411-445.

Leach, J. G. 1923. The parasitism of *Colletotrichum lindemuthianum.* Minn. Agric. Exp. Stn. Tech. Bull. 14. 41 p.

Leakey, C. L. A. and Simbwa-Bunnya, M. 1972. Races of *Colletotrichum lindemuthianum* and implications for bean breeding in Uganda. Ann. Appl. Biol. 70(1):25-34.

McRostie, G. P. 1919. Inheritance of anthracnose resistance as indicated by a cross between a resistant and a susceptible bean. Phytopathology 9(3):141-148.

Digitized by Google

———. 1921. Inheritance of disease resistance in the common bean. Am. Soc. Agron. J. 13:15-32.

Martínez, J. L. 1982. Identificación de las razas de antracnosis *Colletotrichum lindemuthianum* (Sacc. & Magn.) Brios. & Cav. que atacan al frijol (*Phaseolus vulgaris* L.) en el área de influencia del CAEJAL. In: Informe annual de investigación del Grupo Interdisciplinario de Frijol, 1982. Centro de Investigaciones Agrícolas de El Bajío (CAEJAL), Instituto Nacional de Investigaciones Agrícolas (INIA), Mexico. p. 102-105.

Mastenbroek, C. 1960. A breeding programme for resistance to anthracnose in dry shell haricot beans, based on a new gene. Euphytica 9(2):177-184.

Mathur, R. S.; Barnett, H. L.; and Lilly, V. G. 1950. Sporulation of *Colletotrichum lindemuthianum* in culture. Phytopathology 40(1):104-114.

Mercer, P. C.; Wood, R. K. S.; and Greenwood, A. D. 1975. Ultrastructure of the parasitism of *Phaseolus vulgaris* by *Colletotrichum lindemuthianum.* Physiol. Plant Pathol. 5(3):203-214.

Mordue, J. E. M. 1971a. *Colletotrichum lindemuthianum.* CMI (Commonw. Mycol. Inst.) Descr. Pathog. Fungi Bact. Set 32, sheet no. 316, 2 p.

———. 1971b. *Glomerella cingulata.* CMI (Commonw. Mycol. Inst.) Descr. Pathog. Fungi Bact. Set 32, sheet no. 315, 2 p.

Muhalet, C. S.; Adams, M. W.; Saettler, A. W.; and Ghaderi, A. 1981. Genetic system for the reaction of field beans to beta, gamma, and delta races of *Colletotrichum lindemuthianum.* J. Am. Soc. Hortic. Sci. 106(5):601-604.

Mujica, R. F. 1952. Razas fisiológicas y susceptibilidad varietal de los frijoles chilenos a la antracnosis. Agric. Téc. (Santiago) 12:37-45.

Müller, H. R. A. 1926. Physiologic forms of *Colletotrichum lindemuthianum* (Sacc. *et* Magn.) Bri. *et* Cav. in the Netherlands. Phytopathology 16:369.

Navarro-A., R.; Puerta-E., O. D.; and Isaza, L. 1981. Uso alternado de fungicidas para el control de la antracnosis (*Colletotrichum lindemuthianum*) en frijol (*Phaseolus vulgaris*) en el oriente antioqueño. Fitopatol. Colomb. 10(1-2):3-6.

Digitized by Google

Olarte-M., D.; Osorio, G.; Puerta-E., O. D.; and Isaza, L. 1981. Mecanismos para la infección primaria de la antracnosis (*Colletotrichum lindemuthianum*) en frijol (*Phaseolus vulgaris*) en el oriente antioqueño. Fitopatol. Colomb. 10(1-2):23-28.

Oliari, L.; Vieira, C.; and Wilkinson, R. E. 1973. Physiologic races of *Colletotrichum lindemuthianum* in the State of Minas Gerais, Brazil. Plant Dis. Rep. 57(10):870-872.

Oliveira, E. Augustin; Antunes, I. Ferreira; and da Costa, J. G. Cáprio. 1973. Raças fisiológicas de *Colletotrichum lindemuthianum* identificadas no Rio Grande do Sul e em Santa Catarina de 1968 à 1972. Comunicação técnica 8. Instituto de Pesquisa Agropecuária do Sul (IPEAS), Pelotas, RS, Brazil. 5 p.

Onesirosan, P. T. and Barker, L. N. 1971. Stem anthracnose of cowpeas in Nigeria. Plant Dis. Rep. 55(9):820-822.

Paradela-Filho, O. and Pompeu, A. S. 1974. Nota no. 1: Antracnose do feijoeiro, causada por *Colletotrichum dematium* f. *truncata* (Schw.) v. Arx. Bragantia 33:I-IV.

Peregrine, W. T. H. 1971. A preliminary note on chemical control of bean anthracnose in Malawi. PANS (Pest Artic. News Summ.) 17(1): 47-50.

Peuser, H. 1931. (Continued investigations on the occurrence of biological strains in *Colletotrichum lindemuthianum* (Sacc. & Magn.) Bri. *et* Cav.). Phytopathol. Z. 4:83-112.

Pio-Ribeiro, G. and Chaves, G. M. 1975. Raças fisológicas de *Colletotrichum lindemuthianum* (Sacc. *et* Magn.) Scrib. que ocorrem em alguns municípios de Minas Gerais, Espírito Santo e Rio de Janeiro. Experientiae 19(6):95-118.

Ploper, L. D. 1983. Enfermedades del poroto en el noroeste Argentino y su control. In: Hemsy, V. (ed.). 1a. reunión técnica nacional de poroto. Publicación miscelánea no. 74. Estación Experimental Agro-Industrial "Obispo Colombres," Tucumán, Argentina. p. 87-103.

Rahe, J. E. 1973a. Phytoalexin nature of heat-induced protection against bean anthracnose. Phytopathology 63(3):572-577.

———. 1973b. Occurrence and levels of the phytoalexin phaseollin in relation to delimitation at sites of infection of *Phaseolus vulgaris* by *Colletotrichum lindemuthianum.* Can. J. Bot. 51(12):2423-2430.

Digitized by Google

——— and Arnold, R. M. 1975. Injury-related phaseollin accumulation in *Phaseolus vulgaris* and its implications with regard to specificity of host-parasite interaction. Can. J. Bot. 53(9):921-928.

——— and Kuć, J. 1970. Metabolic nature of the infection-limiting effect of heat on bean anthracnose. Phytopathology 60(6):1005-1009.

———; ———; Chuang, C.-M.; and Williams, E. B. 1969. Correlation of phenolic metabolism with histological changes in *Phaseolus vulgaris* inoculated with fungi. Neth. J. Plant Pathol. 75:58-71.

Rathmell, W. G. 1973. Phenolic compounds and phenylalanine ammonia lyase activity in relation to phytoalexin biosynthesis in infected hypocotyls of *Phaseolus vulgaris*. Physiol. Plant Pathol. 3(2):259-267.

Ribeiro, S. R.; Chaves, G. M.; and Thiébaut, J. T. Lima. 1981. Reação de cultivares de feijão a nove raças fisiológicas de *Colletotrichum lindemuthianum* (Sacc. *et* Magn.) Scrib. Rev. Ceres 28(158):342-350.

Salazar, E. M. and Andersen, A. L. 1969. Effects of temperature on spore germination and host infectivity by three strains of *Colletotrichum lindemuthianum*. Phytopathology 59:1045 (Abstr.)

Schnock, M. G. 1975. A new physiological strain of *Colletotrichum lindemuthianum* infecting *Phaseolus vulgaris* L. In: EUCARPIA, section Horticole: reunion haricot, Versailles, France, 10-11-12 Septembre 1975. Centre National de Recherches Agronomiques, Paris, France. p. 71-76.

Schreiber, F. 1932. Resistenzzüchtung bei *Phaseolus vulgaris*. Phytopathol. Z. 4:415-454.

Schwartz, H. F.; Pastor-Corrales, M. A.; and Singh, S. P. 1982. New sources of resistance to anthracnose and angular leaf spot of beans (*Phaseolus vulgaris* L.). Euphytica 31(3):741-754.

Shear, C. L. and Wood, A. K. 1913. Studies of fungus parasites belonging to the genus *Glomerella*. U. S. Dep. Agric. Bur. Plant Indust. Bull. 252. 110 p.

Sherf, A. F. and MacNab, A. A. 1986. Vegetable diseases and their control. 2nd ed. Wiley, New York, NY, USA. 728 p.

Simbwa-Bunnya, M. 1972. Fungicidal control of bean diseases at Kawanda, Uganda. Plant Dis. Rep. 56(10):901-903.

Digitized by Google

Sindhan, G. S. and Bose, S. K. 1981. Epidemiology of anthracnose of French bean caused by *Colletotrichum lindemuthianum.* Indian Phytopathol. 34(4):484-487.

Skipp, R. A. and Deverall, B. J. 1973. Studies on cross-protection in the anthracnose disease of bean. Physiol. Plant Pathol. 3(3):299-313.

Stevenson, J. A. 1956. A note on the citation of the bean anthracnose fungus. Plant Dis. Rep. 40:831.

Sutton, B. C. 1979. Systemic cross protection in bean against *Colletotrichum lindemuthianum.* Aust. J. Plant Pathol. 8:4-5.

———. 1980. The Coelomycetes: fungi imperfecti with pycnidia, acervuli and stromata. Commonwealth Mycological Institute, Kew, Surrey, England. 696 p.

Theodorou, M. K.; Scanlon, J. C. M.; and Smith, I. M. 1982. Infection and phytoalexin accumulation in French bean leaves injected with spores of *Colletotrichum lindemuthianum.* Phytopathol. Z. 103(3):189-197.

Tochinai, Y. and Sawada, K. 1952. Observations on the overwintering of the bean anthracnose fungus *Colletotrichum lindemuthianum* Briosi *et* Cavara. Hokkaido Univ. Fac. Agric. Mem. 1:103-112.

Tu, J. C. 1981. Anthracnose (*Colletotrichum lindemuthianum*) on white bean (*Phaseolus vulgaris* L.) in southern Ontario: spread of the disease from an infection focus. Plant Dis. 65(6):477-480.

———. 1982. Effect of temperature on incidence and severity of anthracnose on white bean. Plant Dis. 66(9):781-783.

———. 1983. Epidemiology of anthracnose caused by *Colletotrichum lindemuthianum* on white bean (*Phaseolus vulgaris*) in southern Ontario: survival of the pathogen. Plant Dis. 67(4):402-404.

———. 1986. Control of anthracnose disease (*Colletotrichum lindemuthianum*) of navy bean (*Phaseolus vulgaris*). Meded. Fac. Landbouww. Rijksuniv. Gent 51(26):645-651.

——— and Aylesworth, J. W. 1980. An effective method for screening white (pea) bean seedlings (*Phaseolus vulgaris* L.) for resistance to *Colletotrichum lindemuthianum.* Phytopathol. Z. 99:131-137.

——— and McNaughton, M. E. 1980. Isolation and characterization of benomyl-resistant biotypes of the delta race of *Colletotrichum lindemuthianum.* Can. J. Plant Sci. 60(2):585-589.

Digitized by Google

———; Sheppard, J. W.; and Laidlaw, D. M. 1984. Occurrence and characterization of the epsilon race of bean anthracnose in Ontario. Plant Dis. 68(1):69-70.

van den Heuvel, J. and Vollaard, P. J. 1976. Metabolism of phaseollin by different races of *Colletotrichum lindemuthianum*. Neth. J. Plant Pathol. 82(3):103-108.

Vieira, C. 1967. O Feijoeiro-comum: cultura, doenças e melhoramento. Universidade Rural do Estado de Minas Gerais, Viçosa, MG, Brazil. p. 84-89.

———. 1983. Doenças e pragas do feijoeiro. Universidade Federal de Viçosa, Viçosa, MG, Brazil. 231 p.

Villada-Ramos, J. E. 1982. Asociación de caracteres morfológicos con el grado de resistencia a la antracnosis *Colletotrichum lindemuthianum* (Sacc. & Magn.) Br. & Cav. del frijol común *Phaseolus vulgaris* L. Thesis. Parasitología Agrícola, Universidad Autónoma de Chapingo, Chapingo, Mexico.

Walker, J. C. 1950. Plant pathology. McGraw-Hill, New York, NY, USA. 699 p.

Waterhouse, W. L. 1955. Studies of bean anthracnose in Australia. Proc. Linn. Soc. N. S. W. 80:71-83.

Yerkes, W. D., Jr. 1958. Additional new races of *Colletotrichum lindemuthianum* in Mexico. Plant Dis. Rep. 42:329.

——— and Teliz-Ortiz, M. 1956. New races of *Colletotrichum lindemuthianum* in Mexico. Phytopathology 46(10):564-567.

Zaumeyer, W. J. and Meiners, J. P. 1975. Disease resistance in beans. Annu. Rev. Phytopathol. 13:313-334.

——— and Thomas, H. R. 1957. A monographic study of bean diseases and methods for their control. Rev. ed. Technical bulletin no. 868. United States Department of Agriculture, Washington, DC, USA. 255 p.

——— and ———. 1962. Bean diseases: how to control them. Agriculture handbook no. 225. Agricultural Research Service, United States Department of Agriculture, Washington, DC, USA. 39 p.

Digitized by Google