Bean Production Problems

Disease, Insect, Soil and Climatic Constraints of *Phaseolus vulgaris*

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Foreword

We are pleased and proud to present to the world of agricultural science this book on diseases, pests, and other problems of beans.

The book represents the combined efforts of dozens of researchers, who have contributed their expertise on this important subject. We believe the book is one of the most comprehensive works yet published on bean problems and brings together in one volume the most thorough, current knowledge available from some of the world's leading plant scientists and researchers.

Beans represent a very important component of the diets of the people of Latin America, and they are produced chiefly by small farmers. The fact that yields have remained stagnant over the past two decades has resulted in an actual decline in per capita production in Latin America. The very large gap between potential yields demonstrated on experiment stations and actual yields realized by farmers is due, to a great extent, to the many diseases and insects which besiege this crop. It is our hope that this book will contribute to the solution of these important problems.

Publication of the book is in keeping with CIAT's continued devotion to the agricultural and economic development of the lowland tropics and the improvement of living standards of its peoples.

John L. Nickel
Director General, CIAT

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The book is written as a supplement to the authoritative work published by Drs. W. J. Zaumeyer and H. R. Thomas in 1957, which was entitled "A Monographic Study of Bean Diseases and Methods for Their Control." We are deeply indebted to the personnel, past and present, of the CIAT Bean Program and other international and national institutions dedicated to improving dry bean production, whose research results provided the source from which our book was compiled.

The editors express their sincere gratitude to the other authors who contributed their expertise to the book. The editors also wish to thank the following scientists who donated illustrations, reviewed various chapters, and aided in the development of this book: Ing. Jorge Aeschlimann, Ing. Andrés Abreu, Dr. George S. Abawi, Ing. M.Sc. Germán Alvarez, Dr. Eduardo Alvarez-Luna, Dr. Barbara Ballantyne, Dr. H. Bannerot, Dr. Steven E. Beebe, Dr. Julio Bird, Dr. Howard L. Bissonnette, Mr. Charles E. Bower, Dr. Douglas W. Burke, Lic. Patricia Nieto de Calderón, Ing. Fernando Correa, Dr. Walter Correa, Dr. A. Santos Costa, Dr. Dermot P. Coyne, Dr. Onkar Dhingra, Dr. Eelco Drijfhout, Dr. George Fassuliotis, Ing. M.Sc. Carlos Flor, Dr. G. Fouilloux.

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Preface

More plant pathogens, and more aggressive or virulent isolates of these pathogens, are attacking beans (*Phaseolus vulgaris* L.) in tropical regions than in temperate regions. The pathogens vary greatly between seasons and years for their incidence and damage; hence it is difficult to obtain the economic data required for their priority ranking. Various pathogens are restricted to growing regions which possess specific environmental factors necessary for their survival and perpetuation. Other pathogens are ubiquitous throughout Latin America and other regions of the world. Additional pathogens and insects may be important in specific production regions, but they do not reduce total Latin American bean production significantly.

Since it is unlikely that resistance to all major pathogens can be combined immediately in commercially acceptable cultivars, some grouping is useful to determine priorities for specific production systems. Beans produced in cool climates frequently suffer yield losses due to some combination of bean common mosaic virus, rust, anthracnose, angular leaf spot, root rots and bacterial blights. Beans produced in warm-hot, relatively dry climates frequently suffer yield losses due to some combination of bean common mosaic virus, bean golden mosaic virus, rust, angular leaf spot, root rots, and common bacterial blight. Beans produced in warm-hot, relatively moist climates frequently suffer yield losses due to some combination of web blight, root rots, and common bacterial blight. However, it is not uncommon to encounter production regions in which conditions favor epidemics of common bacterial blight, anthracnose, web blight and other diseases simultaneously or during different stages of the bean production cycle.

Diseases such as web blight, common bacterial blight and bean golden mosaic virus have been important factors in the development of dry bean production policies throughout Latin America. Web blight and common bacterial blight are important diseases in relatively warm and humid regions and currently constrain bean cultivars from being grown profitably in many production zones. Bean golden mosaic virus has been a devastating disease in parts of Brazil, Central America, the Caribbean and Mexico.

Many of the principal insect pests, such as leafhoppers, leaf-feeding beetles and larvae, and cutworms, are encountered throughout all production regions and can damage beans seriously during various periods. Other insects, such as the Mexican bean beetle and bean pod weevil, are primarily encountered only in regions of Central America, the Caribbean and Mexico. Storage insects, or Bruchids, are very pervasive and a serious economic problem, because they often force producers to sell beans immediately after harvest when the market supply is saturated and prices are low.

Dry bean production also is affected by many constraints other than plant pathogens and insect pests. Soil fertility is extremely variable throughout Latin America and other regions of the world, and bean production often is severely limited by deficiencies and/or toxicities of elements required for plant development. Miscellaneous production problems may be induced by such factors as agricultural chemicals, air pollutants, climatic variations or extremes and genetic abnormalities.

Much literature on bean production constraints has been published by scientists in the American continents and other parts of the world since 1957, when Drs. W.J. Zaumeyer and H.R. Thomas released their authoritative monograph on bean diseases and methods for their control. Our book was written to supplement their monograph as a technical and current review of major and minor bean production constraints which occur in Latin America and other dry bean growing regions of the world. It also is intended to assist scientific, administrative and extension personnel involved in programs to improve dry bean production.

This book is divided into four general sections, each containing chapters written on specific dry bean constraints by one or more of the 20 contributing authors. Intensive reviews are presented for dry bean production and losses, fungal diseases, bacterial diseases, mycoplasma-like and viral diseases, and other production constraints including seed pathology, nematodes, miscellaneous problems, soil fertility, insects and other pests.

Three appendices are included to aid the reader in the identification of pesticides referred to throughout the book, to convert metric to U.S. units, and to clarify the current taxonomy for certain legume species.

May, 1979

H.F. Schwartz
G.E. Gálvez

Chapter 1

Bean Production and Pest Constraints in Latin America

J. H. Sanders and
H. F. Schwartz

Chapter 1

Bean Production and Pest Constraints in Latin America

Introduction

Dry beans *(Phaseolus vulgaris* L.) are exposed to a large array of yield constraints during their growth cycle in Latin America and other regions of the world. This chapter will concentrate primarily on disease and insect constraints which influence bean production in Latin America. A brief review is given on Latin American bean production, followed by a discussion on economical and pathological aspects of control strategies.

More than one-third of the dry bean production in the world occurs in Latin America. Average bean yields in Latin America are less than 600 kg/ha, compared to monoculture yields of nearly 1400 kg/ha in the United States (Table 1) and three to five tons under experimental conditions in Latin America (3). During the last decade the production growth rate of beans in Latin America was substantially less (0.27%) than the population growth rate (2.80%), and caused per capita consumption to decrease while bean imports and legume prices increased. These trends have aggravated nutritional and balance-of-payment problems in many Latin American countries (24).

Total bean production has changed relatively little in Latin America during the last decade due to a net balance realized between expanded production area and reduced crop productivity (Table 2). Not only have dry bean yields declined during the last decade, but they also have showed extreme fluctuation between years. Variable weather conditions, poor soil fertility, bean diseases and insect pests appear to be the most important factors contributing to declining and erratic yields (3, 13, 23, 25, 26, 27). The recent decline in Brazilian yields greatly influenced total productivity, since Brazil is responsible for 54% of Latin American bean production.

Recent severe disease epidemics of bean golden mosaic virus and chronic problems with anthracnose and common bacterial blight appear to have been most responsible for this decline (24).

Brazilian yield declines also have been influenced by the displacement of beans to more marginal production areas due to the influx of more profitable crops such as soybeans. This displacement also has occurred frequently throughout other regions of Latin America because of the inherent risks involved in bean production, low absolute yields and profitability, and the lack of a stable price after harvesting. These factors, plus difficulties in mechanizing the dry bean harvest, have concentrated bean production on small farms in most of Latin America (13, 16). Production on small farms usually implies low levels of purchased inputs, associated cropping, and production area shifts as soil nutrients become depleted or eroded (Table 3).

Determining Priorities Among Bean Pathogens and Pests

The importance of a plant pathogen or pest is determined by the economic loss it causes. The magnitude of this loss depends on how frequently it occurs and how severe the damage is during each crop cycle. Most estimates of yield losses in Latin America are based on experimental data and should, therefore, be regarded as estimates of yield losses under conditions of good soils, high level management, often high use of inputs and usually high disease or insect incidence. Table 4 lists estimated yield losses obtained for important bean pathogens and insect pests, primarily under these conditions. However, it is difficult to extrapolate these experiment station or glasshouse disease loss estimates to those of commercial operations.

One study of farm level pest and pathogen incidence was conducted in the major Colombian zones of bean production in 1974-1975. Based on data taken during repeated visits to 177 farms, the relative importance of various pests and pathogens was estimated by multiple regression analysis (22, 23). Table 5 summarizes the magnitude of production losses obtained during this growth cycle in various Colombian regions. For example, leafhoppers caused 1.3 million dollars damage in three regions during one semester's production. Pest and pathogen incidence is expected to vary not only by region but also between seasons and cultivars. Hence, much information is necessary for the definitive priority ranking in specific production regions in Latin America.

Bean Disease Control Strategies

Many measures are available in Latin America to control bean diseases, including cultural practices, crop rotation, sanitation and disease avoidance, production of pathogen-free or clean seed, chemical control and resistance breeding. Associated cropping with maize may reduce certain insect problems and create a physical barrier to the spread of a pathogen such as the common bacterial blight bacterium (1, 10, 11). However, it can enhance infection by other pathogens such as the angular leaf spot fungus (20).

Dry bean pathogens causing diseases such as bean common mosaic virus, common bacterial blight, angular leaf spot, and anthracnose are able to infect seed and be transmitted within seed. When compared with highly infected farmers' seed, impressive results have been obtained by planting clean seed (3, 7). In Guatemala, clean seed combined with other inputs raised yields to 1.5 tons/ha on 84 ha in two valleys compared with the national average of 515 kg/ha. Results in Colombia for certified and protected seed (produced with heavy chemical application in a high rainfall region) were not impressive. In fact, certified seed gave lower yields than farmers' seed and the protected seed was only marginally superior with a 106 kg/ha difference (3). In bean production regions with a high incidence of pathogens, pathogen-free seed may have to be combined with other control strategies to reduce disease incidence. Substantially higher yield differences will be necessary to offset the costs of implementing and maintaining clean seed production programs.

Clean seed production in semi-arid regions of the western United States undoubtedly has contributed substantially to the reduced importance of anthracnose and bacterial blights in the United States. However, clean seed programs are expensive since they require:

- specific regions unfavorable to pathogen development and survival, but favorable to plant development
- increased production costs for irrigation, inspection, chemical protection and transportation back to production regions
- distribution to farmers.

A successful clean seed production program often requires financial support by the government or a producers' cooperative to reduce seed costs and insure farmer acceptance. However, when combined with other control measures, clean seed may be a low cost and effective control measure for certain pathogens (3).

In Latin American bean production, chemical control involves multiple spraying and substantially increased production costs. However, it often results in only limited success. For example, growers in the Cauca Valley of Colombia spent large amounts for agricultural pesticides and still suffered substantial damage from rust and leafhoppers (23). Chemical control also is often associated with large farm size, since these farmers generally use more inputs and receive more technical assistance than those with smaller farms (Table 3). However, most bean production in Latin America occurs on small farms. When chemicals are used, they may be inappropriate to control specific plant pathogens or insects, since farmers often apply only those chemicals which are known to be most effective on their more profitable cash crops such as coffee or potatoes (Sanders, unpublished data). Moreover, indiscriminant application of broad spectrum chemicals can eliminate beneficial insect predators of bean pests and reduce the potential effectiveness of biological control agents. Chemical control of bean diseases and insects in Latin America, therefore, should be considered a large farmer solution, a short- term measure while resistances are being incorporated into commercially acceptable bean cultivars, and a component of integrated control.

Breeding for disease and insect resistance is an essential component if the control strategy for Latin America is to be directed toward all producers, irrespective of their economic resources. The gain from breeding for resistance to specific pathogens and insects will depend on expected yield losses from the pathogen, the probability of success in breeding resistance into a high yielding and marketable cultivar, and the period during which the resistance mechanism maintains its effectiveness. Thus, not only must sources of resistance exist and be incorporated easily into commercially acceptable cultivars, but they also must endure long enough to ensure that overall benefits are greater than the costs incurred in breeding and diffusion efforts.

When multiple races or strains of a pathogen exist, probability of the loss of effective resistance becomes an important consideration, especially in the tropics where environmental conditions in many regions favor nearly continuous disease pressure. Alternative breeding strategies for more stable resistance, for example non-race-specific resistance, also must specify the time period and cost required to develop this protection. It is not sufficient to point out only that race-specific resistance breaks down. It also is necessary to identify a higher payoff with an alternative control measure and to compare net returns during the different time periods. Race-specific resistance to rust would have been worth 1.2 million dollars, even if effective only for one season and the cultivar were distributed only throughout the Cauca Valley (Table 5). Nevertheless, a more stable and longer-term form of resistance is preferred if it has a higher economic

return than alternative controls or is the only practical control available to producers with limited economic resources.

Another problem is the increased probability of a general epidemic occurring after widescale diffusion of a new cultivar with race-specific resistance or different cultivars derived from relatively similar and uniform germplasm sources. Such an epidemic occurred during 1970 in the United States when 75-90% of the commercial maize hybrids planted were derived from a single source of cytoplasm. These hybrids were susceptible to various pathogens such as *Phyllosticta maydis* and *Helminthosporium maydis* race "T". The latter reduced U.S. yields by 15% in 1970 (4). Geographical diversity of production areas and farming systems, differences in consumer preferences for bean type, and the expected slow diffusion of new materials to the many small farmers producing beans in Latin America all reduce the danger of a widescale epidemic inherent in an agricultural system which relies on widely diffused and genetically uniform cultivars. Nevertheless, the stability of plant resistance mechanisms must be monitored continuously by research and extension personnel throughout Latin America and other dry bean production regions in the world.

Summary

Beans are attacked by a large number of plant pathogens and insect pests, many of which can reduce yields drastically. Farmers with small land holdings usually have limited resources but produce most of the beans in Latin America. Control strategies feasible for these growers may be restricted to those strategies which do not require large cash inputs, hence breeding for resistance may be the most desirable alternative available. National and international bean production programs must accurately identify yield constraints prevalent in specific production regions to provide more efficient use of the large manpower, research expenditure and time requirements necessary to implement resistance breeding.

Stability of resistant materials can be improved with an integrated control strategy consisting of resistance, cultural practices, chemicals and clean seed production for those diseases in which resistance does not confer immunity to infection. This integrated control strategy will need to be adapted to specific regional problems. As in the case of disease and insect priority identification, a more systematic collection of information is necessary to evaluate the costs and probability of success for control strategies so that the research by pathology, entomology and breeding on the experiment station is more applicable and quickly available to farmers.

Table 1. Dry bean production in the world during 1975-77 (9).

Country	Production Area ('000 ha)	Total Production ('000 ton)	Average Yields (kg/ha)
Brazil[a]	3788	1973	521
Mexico	1525	837	547
Argentina	167	187	1085
Chile	82	85	1032
Colombia	112	78	693
Guatemala	119	70	599
Paraguay	70	54	771
Nicaragua	69	51	746
Peru	64	49	772
Venezuela	95	48	493
Honduras	87	47	540
El Salvador	54	38	703
Dominican Republic	45	33	731
Ecuador	66	30	451
Cuba	35	24	686
Costa Rica	36	15	417
Panama	17	4	235
Latin America[b]	6486	3677	567
China	2605	2229	856
United States	570	779	1370
Japan	113	148	1310
Canada	68	97	1435
Far East	9472	3179	336
Africa	1961	1106	564
Western Europe	941	483	513
Near East	230	302	1313
South Africa	69	64	927
World[c]	23722	12392	522

a/ Cowpeas were deleted from the Brazilian bean data.
b/ Several Latin American countries were excluded because of inconsistent data. However, their share of production was very small.
c/ These totals include production data from the above countries plus others not listed.

Table 2. Rates of increase for production, area and yield of beans in Latin America during 1965-1976 (24).*

Country	Rate of Increase		
	Production	Area	Yield
Brazil	-0.89	1.92	-2.81
Mexico	0.99	-2.07	3.05
Argentina	16.17	14.89	1.28
Guatemala	4.21	2.24	1.97
Colombia	6.77	3.26	3.50
Chile	-0.69	2.75	-3.45
Honduras	-0.54	0.88	-1.43
Nicaragua	1.93	0.77	1.16
Haití	1.01	0.33	0.68
El Salvador	8.79	6.27	2.52
Peru	-3.80	-2.04	-1.76
Paraguay	2.04	6.65	-4.61
Venezuela	-3.76	-1.76	-2.00
Dominican Republic	3.41	1.05	2.36
Ecuador	-1.16	-0.48	-0.67
Cuba	0.35	-0.59	0.94
Costa Rica	-2.21	-4.25	2.04
Panama	-5.83	-4.01	-1.82
Uruguay	-2.66	-0.65	-2.01
Latin America	0.27	0.79	-0.52

* Estimated with the semi-log model: $LY = A + bX$, where LY is the log to the base e of production or area. A and b are the parameters of the regression, and X represents years. Differentiating LY with respect to year gives $\partial LY / \partial X = b$, thus the annual rate of change is b. When b is multiplied by 100, the geometric growth rate is obtained.

Table 3. Characteristics of bean production in the four principal production regions of Colombia (23).

Characteristic	Production Region			
	Valle	Huila	Nariño	Antioquia
Average elevation (meters above sea level)[a]	1120	1323	1309	2270
Average farm size (ha)	48.0	29.5	9.2	4.4
Area in beans (ha)	22.6	4.1	1.8	1.5
Percentage of farms using:				
Irrigation	45	2	0	0
Certified seed	52	7	5	0
Fertilizers	84	20	0	100
Herbicides	32	0	0	0
Insecticides	87	20	5	33
Fungicides	100	14	0	42
Credit	87	53	58	50
Technical assistance	70	18	5	8
Mixed cropping	0	74	95	100
Machinery	100	44	0	0
Bean yield (kg/ha)	906	680	467	533
Bean equivalent yield (kg/ha)[b]	906	825	732	723

a The range was substantital in two of the regions:

Valle	1030 - 1310m,	Nariño	865 - 1560 m.
Antioquia	2200 - 2410m.	Huila	950 - 1560 m.

b The bean equivalent yield is: $Y_B + \frac{P_c Y_c}{P_B} = Y_{B.E.}$

Where Y_B is the bean yield, Yc is the corn yield or other crop yield, $Y_{B.E.}$ is the bean equivalent yield and $\frac{P_c}{P_B}$ is the corn (or other crop price) relative to the bean price (P_B).

Table 4. Estimated bean yield losses attributed to plant pathogens and insects.

Plant Disease or Insect Pest	Estimated Yield Loss	Literature Cited
Bean Common Mosaic Virus	53-68% (U.S.A.)	15
	16-95% (Latin America)	3
Bean Golden Mosaic Virus	48-85% (Brazil)	5
Common Bacterial Blight	10-38% (U.S.A.)	28
	18-45% (Colombia)	22
Rust	38-50% (Brazil)	21
	18% (Colombia)	29
	40-80% (U.S.A.)	28
Anthracnose	38-99% (Colombia)	3
	100% (U.S.A.)	28
Angular Leaf Spot	50% (U.S.A.)	14
	40-60% (Colombia)	2
	80% (Mexico)	6
Root Rots	60% (Brazil)	12
	15-86% (U.S.A.)	17
Leafhoppers	14-23% (Wet season, Colombia)	25
	73-95% (Dry season, Colombia)	25
Bean Pod Weevil	94% (El Salvador)	18
	90% (Mexico)	8
Storage Insects (Bruchids)[a]	35% (Mexico, Central America, and Panama)	19
	7.4% (Colombia)	26

a The insect damage losses were not separated from other storage losses.

Table 5. Bean production losses caused by plant diseases and insect pests in three Colombian bean zones during 1974-1975 (23).

Production Problem	Estimated Value of Production Loss During One Crop Cycle	
	Cauca Valley [a]	Huila and Nariño [a]
Plant Diseases		
Rust	U.S.$ 1,171,000	-
Common Bacterial Blight	933,000	-
Angular Leaf Spot	552,000	-
Viruses [b]	-	400,000
Anthracnose	-	282,000
Powdery Mildew	-	250,000
Root Rot [c]	-	207,000
Insects		
Leafhoppers	749,000	537,000
Thrips	-	510,000

a The average elevation above sea level was 1120 m in the Cauca Valley and 1320 m in Huila and Nariño.

b The interviewing agronomists were unable to always differentiate between virus symptoms caused by bean common mosaic virus, bean rugose mosaic virus or other viruses.

c No attempt was made to identify the specific root rot pathogen responsible

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SECTION I.

FUNGAL DISEASES

Chapter 2

Rust

Edgar Vargas

Chapter 2

Rust

Introduction

Bean rust is caused by *Uromyces phaseoli* (Reben) Wint. (= *U. appendiculatus* (Pers.) Unger). The disease has a worldwide distribution (85). It causes one of the more important production problems in many areas of Latin America (84), including Brazil (17, 71), Colombia (86), Mexico (20), Peru (25) and the tropics in general (8). Yield losses are most severe when plants are infected during the preflowering and flowering stages of development, approximately 30-45 days after planting (1, 17, 20, 52, 74, 79). Disease loss estimates in the glasshouse and field include 40-50% plant dry weight reduction (1), and yield losses of 18-28% (25, 74, 86), 38-45% (52) and 40-100% (39, 66, 85).

Uromyces phaseoli can infect many species of *Phaseolus,* such as *P. acutifolius* var. *latifolius, P. adenanthus, P. anisotrichus, P. coccineus, P. dysophyllus, P. lunatus, P. obvallatus, P. polystachyus, P. retusus, P. sinuatus, P. vulgaris, Vigna unguiculata* (57, 85), *V. repens* and *V. vexillata* (6).

Common names frequently used for rust in Latin America include roya, ferrugem and chahuixtle.

Etiology

Uromyces phaseoli is an obligate parasite which belongs to the Basidiomycotina subdivision of fungi. It has an autoecius life cycle which is completed entirely on the bean host (5).

Aecia are rarely observed in nature (43, 85) but were studied in detail in the greenhouse by Andrus (5) and more recently by Groth and Mogen (35). After undergoing a conditioning or dormancy period, teliospores may germinate to produce basidiospores which infect bean leaves and in about

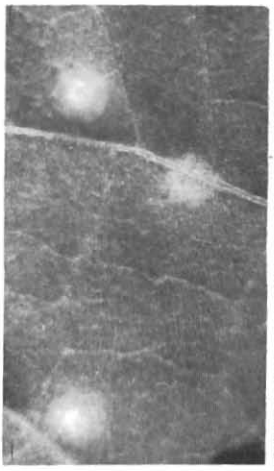

Fig. 1- (Left) Pycnica of *Uromyces phaseoli* on upper leaf surface.
Fig. 2- (Right) Aecia of *Uromyces phaseoli* on lower leaf surface.

six days at 22° - 26°C produce a small chlorotic fleck or pycnium (Fig. 1), which after approximately seven days contains droplets of cloudy white nectar, spermatia (+ or - mating type) and receptive hyphae.

Cross fertilization by pycniospores from the opposite mating type will initiate aecium formation (Fig. 2) within nine to 12 days at 22° - 26°C on the lower leaf surface. Aecia may form occasionally on the upper leaf surface also. Aeciospores form in the white aecium and, upon their release, are able to infect bean plants and eight to 10 days later produce a pustule with urediospores (5,35). Subsequent cycles of infection rely solely upon the urediospore stage. These spores are capable of germinating to provide infection hyphae which infect the plant and form new pustules wherein new urediospores and eventually teliospores may develop (5). Teliospores reportedly undergo a dormancy period and germinate six months after production and subsequent storage at 9°C (38). However, Groth and Mogen (35) were able to remove possible inhibitors by washing teliospores in running cold water for three days and observed teliospore germination on water agar within two to four weeks at 24°C.

The most commonly observed spore forms are the urediospore (summer or vegetative spore) and teliospore (winter or resting spore). Urediospores are produced in rows within a sorus or pustule on the upper or lower leaf surface. Urediospores have a short hyaline pedicel and are light brown in color, one-celled, spiny and thin-walled, and globoid to ellipsoid in shape. They may have two equatorial or superequatorial pores, and measure 22.5 μ by 28 μ. Near the end of the growing season, teliospores may form within the pustule in response to changes in light intensity, temperature, moisture,

cultivar response, race differences, leaf age or plant maturity. Teliospores have a short hyaline pedicel and are dark brown, one-celled, smooth and thick-walled, and globoid to broadly ellipsoid in shape. They may have a hyaline papilla over the pore and measure 24 μ by 30 μ (85).

Almeida (4) reports the existence of a new variety of bean rust collected from *Phaseolus longepedunculatus* Mart. in 1945 by A.P. Viegas, who named the rust *Uromyces phaseoli longepedunculati* Viegas. Almeida studied herbarium samples of the original collection, confirmed that it differs from *U. phaseoli*, and, according to current nomenclature rules, named it *Uromyces appendiculatus* (Pers.) Ung. var. *brasiliensis* R. Almeida var. nov.

Urediospores have two distinct germ pores present in a slighty superequatorial position, in contrast to *U. phaseoli* urediospores which have indistinct pores along their equator or rarely in a slightly superequatorial position. Teliospores usually are smooth-walled and rarely have small, inconspicuous warts, in contrast to *U. phaseoli* teliospores which rarely are smooth-walled and usually have numerous and prominent warts.

Although *U. phaseoli* does not grow in culture, viable spores can be preserved for varying time periods in the laboratory. Dried leaves bearing pustules and spores have been stored at -20°C for two years (37). Urediospores stored at 7°C for 26 weeks infected plants in the greenhouse (38). Viable spores (40% germination) have been recovered after storage for nearly two years at -60°C (63), and for seven years in liquid nitrogen (21). Davison and Vaughan (23) had similar results when spores were stored at -18°C, but they claim that spore viability and content of self-inhibiting chemicals were influenced by temperature and moisture conditions present during spore production. Dundas (26) reported that storage at -18°C for five to seven months reduced spore germination markedly and induced pathogenic mutations.

Epidemiology

Infection by *Uromyces phaseoli* is favored by prolonged periods (10-18 hours) of moisture conditions greater than 95% relative humidity and moderate temperatures between 17° - 27°C (7, 34, 38, 62, 85). Temperatures greater than 32°C may kill the fungus (20, 61, 62, 85), and temperatures less than 15°C may retard fungal development (20, 85). Day length and light intensity are important factors (37), and Augustin *et al.* (7) report that infection is favored by incubation in low light intensity (2×10^{-5} μe cm^{-2} sec^{-1}) for 18 hours.

Urediospore production and release also are influenced by moisture and temperature conditions. Spore production increased when infected plants

were exposed to high moisture conditions for limited periods of time (76). Sporulation increased when infected plants received at least a 12-hour photoperiod (16). *U. phaseoli* can produce 10^6 urediospores/cm^2 on leaves bearing 2-100 pustules/cm^2 (76). Nasser (52) reported that the greatest number of spores are released during temperate (greater than 21°C), dry (less than 60% relative humidity) days which are preceded by a long dew period or rain the previous night. Urediospores can survive under field conditions nearly 60 days (82).

Urediospores and teliospores can overwinter in bean debris and on wooden supports used for climbing beans (23). Urediospores can be transported long distances by wind currents and probably provide the initial and secondary inoculum during epidemics in Latin America, where multiple bean cropping and staggered planting dates provide a continuum of susceptible germplasm during favorable environmental conditions.

Bean rust incidence may be influenced by different cropping systems used to produce beans. For example, rust incidence was lower when beans were grown in monoculture than in association with maize (44). This lower rust incidence may reflect the higher relative humidity present within the maize-bean canopies.

Plant Infection

The infection process begins as an aeciospore or a urediospore produces a germ tube which develops an appressorium after physical contact with the edges of a stomata (75). An infection peg develops from the appressorium and pushes the guard cells apart until the fungal cytoplasm is transferred into the substomatal vesicle. The substomatal vesicle contains numerous glyoxysomes, lipid bodies and glycogen particles (49). The fungus develops infection hyphae and haustoria as it proceeds intercellularly throughout the host tissue, eventually forming a young pustule (85).

Plant physiology and biochemistry are affected during the infection and sporulation processes. Initially, reducing sugar, sucrose and starch contents increase in infected tissue. Later, various amino acids and sugars decrease as sporulation begins (40, 56). Various enzymes, such as peroxidase, catecoloxidase, glycolate-oxidase and glyoxalate reductase increase their activity during infection (51, 56, 65). Quinones, such as Vitamin K_1, plastoquinones A, C and O, and ubiquinone, increase during rust infection and development (50).

Infection reduces the transfer of metabolic by-products from leaves to roots and developing seeds (81). Stomatal transpiration decreases two days after infection (64), while transpiration and water vapor loss through the damaged cuticle increases as infection proceeds (27, 64). Infected plants become more sensitive to moisture stress as sporulation occurs (27).

Symptomatology

Uromyces phaseoli may infect leaves (Fig. 3), pods (Fig. 4) and rarely stems and branches (Fig. 5). Initial infection may occur on the upper or lower leaf surface. However, symptoms usually appear first on the lower surface as minute, whitish, slightly raised spots (Fig. 6) about five or six days after inoculation. These spots enlarge to form mature reddish-brown pustules which rupture the epidermis and may attain a diameter of 1-2 mm within 10-12 days after inoculation. Secondary and tertiary pustules may develop around the perimeter of this primary pustule and merge with the original pustule (85). The entire infection cycle occurs within 10-15 days, after which urediospores are released passively from pustules and scattered

Fig. 3- Mature rust pustules on infected bean leaf.

Fig. 4.- Mature rust pustules on infected bean pod.

Fig. 5- Mature rust pustule on infected bean branch

Fig. 6- Immature rust pustules five to six days after infection.

Fig. 7-(left) Mature bean rust telia which contain teliospores.

Fig. 8-(right) Interaction between bean rust and anthracnose fungi observed in the field.

by farm implements, insects, animals and wind currents (76, 85). Later, teliospores may form in these pustules, and telia appear dark-brown to black (Fig. 7). The bean rust fungus is not seed-transmitted (85).

Various interactions have been observed between infections by *Uromyces phaseoli* and other bean pathogens or non-pathogens, usually under controlled conditions. Rust infection may predispose plants to subsequent infection by bean pathogens such as *Pseudomonas phaseolicola, Colletotrichum lindemuthianum* (Fig. 8), and *Thielaviopsis basicola* and non-pathogens such as *Sphaerotheca fuligena* and Tobacco Mosaic Virus (77, 78).

A high incidence of rust infection may suppress the appearance of *P. phaseolicola* symptoms (77). Necrotic rings can occur on the perimeter of rust pustules when rust infected plants are inoculated with Tobacco Mosaic Virus (31, 73), and possibly other viruses (Fig. 9), or cucumber downy mildew caused by *Pseudoperonospora cubensis* (78). Heavily rusted sections of leaves were slowly killed during the interaction between bean rust and cucumber downy mildew. Rust spores may contain compounds which inhibit virus multiplication when the two organisms are inoculated simultaneously onto plants (31, 73).

Fig. 9- Necrotic ring development around bean rust pustules caused by interaction with unidentified virus.

Control by Cultural Practices

Cultural control recommendations include crop rotation and removal of old plant debris which may bear viable urediospores and teliospores (71, 85). Reduced plant density also may decrease rust incidence. Planting dates may be adjusted for specific production zones to avoid or reduce the incidence of rust infection during the preflowering to flowering stage of plant development.

Control by Chemicals

Bean rust reduces yields more severely when infection occurs before flowering than when it occurs after flowering. Therefore, chemical control is most effective during early plant development (79). Bean rust has been controlled by dusting plants every seven to 10 days with sulfur at a rate of 25-30 kg/ha (20, 38, 85) when the first pustules are observed. A similar time schedule is recommended for other preventative chemicals, such as Daconil or Chlorothalonil (225 g/100 l), Dithane M-22 or Maneb (4-5 kg/ha), Manzate D 80W or Maneb (4 kg/ha in 1000 l water) and Dithane M-45 or Mancozeb (3-4 kg/ha) (17, 20, 29, 32, 39, 71, 74).

Plantvax or Oxycarboxin can be somewhat therapeutic. It is effective when sprayed at the rate of 1.8-2.5 kg/ha 20 and 40 days after planting or every two weeks until the end of flowering (17, 20, 29, 32, 80). Dongo (25) reported that one preflower application of Plantvax (0.9 kg/ha) reduced rust infection by 40% and increased yields by 26%. However, seed treatment with Plantvax did not give satisfactory control (29). Oxycarboxin (4000 ppm) is therapeutic when applied up to three days after inoculation and preventive when applied less than seven days before inoculation (2, 3). However, Issa and Arruda (41) concluded that chemical control was not economically practical in Brazil.

Control by Plant Resistance

Many workers have observed that bean cultivars varied in their reaction to infection by *Uromyces phaseoli* (Fig. 10), and that the pathogen

Fig. 10- Resistant variety on left; susceptible variety on right.

possessed much pathogenic variability (37). Various sets of differential bean cultivars have been utilized (Table 1) to characterize the different races of bean rust based upon pustule size, intensity, chlorosis and necrosis. Variation in natural populations consists of 39 races identified in Brazil (13, 17), 10 races in Colombia (86), 31 races in Mexico (19), 12 races in Puerto Rico (45), four races in Nicaragua, five races in Honduras (67, 68), five races in El Salvador (69), seven races in Guatemala (70), four races in Peru (36), 11 races in Costa Rica, 11 races in Australia, eight races in East Africa and 35 races in the United States (8, 10, 28, 53). Unfortunately, it is difficult to compare these data because different rating scales (Table 2) and differential cultivars were used (18).

Most workers have relied on sources of specific resistance effective against a limited number of physiological races prevalent in specific locations (7, 8, 9, 18, 20, 46, 48, 58, 60, 83, 84, 85). Selection of resistant cultivars or germplasm usually is based on the complete absence of rust, or small pustule size. Specific resistance usually is simply inherited and dominant (7, 85). However, some sources have involved mutiple factors, incomplete dominance or transgressive segregation (83).

Many commercial cultivars possess resistance to one or more races. However, to date, no cultivar or germplasm source has been immune or resistant to all reported races or populations of rust (84). Data from the 1975-1976 International Bean Rust Nursery were gathered on 132 entries tested at 11 and 15 locations in 1975 and 1976, respectively. No entry was resistant at every location in both years. See Table 3 (14).

Coyne and Schuster (18) suggest that specific resistance may be used more effectively to provide a longer-lasting and stable protection by utilizing gene pyramiding, multilines, multiplasm and regional deployment of genes. Johnson and Allen (42) reduced the sporulation of a highly virulent race by first applying a weakly virulent race. They feel this principle may be useful in a multiline. Vieira (72) states that the diverse cultivars grown in Brazil were developed locally and, in total, provide horizontal or field resistance to rust and other bean diseases. Substitution of this mixture with a few improved, genetically uniform cultivars may place much selection pressure on pathogen populations.

The effective use of specific resistance demands that an international set of differential cultivars and rating scale be developed to coordinate research activities throughout the world. Standard techniques also must be developed for uniform procedures to inoculate differential cultivars (15, 24, 45, 47, 54). Various international efforts are now underway through the International Committee on Coordination of Rust Research, the Committee on International Bean Differentials, and the International Bean Rust Nursery.

Research also must intensify to develop forms of race non-specific resistance to supplement or replace existing sources of specific resistance. Nearly 60 years ago it was observed (30) that bean cultivars differed in their rust reaction by reduced numbers of infections, decreased pustule size and spore production, and early telia formation.

Recent workers (8, 11) have revived interest in this forgotten area of research by suggesting that factors which also may contribute to non-specific resistance include length of dew period produced on specific plant genotypes, efficiency of pathogen penetration, length of incubation period, rate of pustule development and increased resistance with plant maturity. Rodríguez *et al.* (59) report that Mexico 309 is susceptible to race CR-29 but yielded as well as resistant cultivars, many of which were early-maturing. Canessa and Vargas (12) observed cultivars were more heavily infected in the lower than the upper foliage. They feel that this type of resistance may be useful. González (33) reports that Bolita 41, Victor 8, Jicotea and Holguin 20, are late or slow-rusting. Other workers also have observed this reaction in other materials (Meiners, Ballantyne, personal communication). Methods must be designed to measure these components and incorporate useful factors into breeding programs.

Effective and stable genetic control of bean rust may be achieved by combining specific resistance genes and various factors contributing to non-specific resistance (18). Integration with other control measures, such as chemical and cultural practices, may have to be considered to achieve long-lasting and stable protection against bean rust.

Table 1. Variation in bean differential cultivars used by various researchers to distinguish physiological races of *Uromyces phaseoli*.

Harter and Zaumeyer (37)	Fisher (28)	Pereira and Chaves (55)
White Kentucky Wonder U.S. No. 3	White Kentucky Wonder U.S. No. 3	Kentucky Wonder White
Bountiful No. 181	Bountiful No. 181	Turrialba 4
California Small White No. 643	California Small White No. 643	Redlands Greenleaf C
Pinto 650	Pinto 650	Bayo Camana
Kentucky Wonder Wax 765	Kentucky Wonder Wax 765	White Kentucky Wonder U.S. No. 3
Kentucky Wonder Hybrid 780	Kentucky Wonder Hybrid 780	Canario 101
Kentucky Wonder Hybrid 814	Kentucky Wonder Hybrid 814	Cornell 49-242
	Golden Gate Wax	Kentucky Wonder Hybrid 814
Crispin and Dongo (19)	Z-4	Diacol Nutibara
Aguascalientes 13	López (45)	California Small White No. 643
Guerrero 6	California Small White No. 643	Ballantyne (10)
Guerrero 9	Cuva 168-N	California Small White No. 643
Guanajuato 10A-5	P.I. 165426 (black)	Pinto U.I. 111
Mexico 6	P.I. 152326	Sanilac
Mexico 12	Mulatinho	Golden Gate Wax
Veracruz 10	Venezuela 54	Redlands Greenleaf B
Canario 101		C.C.G.B. 44
Negro 150		Veracruz 1A6
		Epicure
		Brown Beauty
		Redlands Greenleaf C
		Bonita

Table 2. Variation in rust rating scales utilized by research workers.

Harter and Zaumeyer (37)		Crispin and Dongo (19)		Davison and Vaughan (22)	
Grade	Description	Infection types	Description	Grade	Description
0	Immune	0	Immune, no symptoms	1	Immune, no symptoms
1	Necrotic flecks, without spores	1	Small necrotic lesions, no pustules	2	Necrotic flecking without pustule or spores. Lesion size and shape may be variable.
2	Small pustules with little sporulation, may be surrounded by a necrotic fleck (highly resistant)	2	Numerous small pustules surrounded by a necrotic area	3	Pustule diameter 300 u or less.
3-10	Dependent upon the size of the spore-bearing pustule	3	Numerous small pustules barely visible on lower leaf surface, no necrosis	4	Pustule diameter 301-499 u.
3-6	Commercially resistant	4	Many good-sized pustules on upper and lower leaf surfaces, may be surrounded by a chlorotic halo	5	Pustule diameter 500 μ or more.
7-8	Tolerant	5	Numerous large pustules on upper and lower leaf surfaces; leaf margins may be dead and entire leaf may be chlorotic		
9-10	Susceptible				

Table 3. Reaction of most widely resistant entries in the 1975 and 1976 IBRN (14).

ENTRY	Number of locations where the entry was classified									
	1975					1976				
	Immune	Resistant	Intermediate	Susceptible	No Data	Immune	Resistant	Intermediate	Susceptible	No Data
Compuesto Chimaltenango 3	4	3	2	1	5	5	9	2	1	0
Turrialba 1	4	3	2	3	3	3	7	6	1	0
ICA - Pijao	3	1	4	3	4	3	6	7	1	0
Mexico 309	6	5	1	0	3	6	3	3	2	0
Mexico 235	2	1	2	0	10	6	4	4	2	1
San Pedro Pinula 72	4	3	3	2	3	4	6	5	2	0
Ecuador 299	5	7	1	0	2	3	6	6	2	0
Cornell 49-242	3	5	4	1	2	2	4	9	2	0
P.I. 226895	4	6	2	0	3	1	5	7	2	2

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Chapter 3

Anthracnose

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Introduction

Bean anthracnose is caused by *Colletotrichum lindemuthianum* (Sacc. & Magn.) Scrib. (84) and is distributed worldwide on susceptible cultivars grown in locations which have cool to moderate temperatures and high humidity or free moisture. The perfect stage of the fungus has been identified as *Glomerella cingulata* (Stonem.) Spauld. *et* V. Schrenk (52).

The anthracnose pathogen has caused economic losses in North America, Europe, Africa, Australia, Asia (91), and in such Latin American countries as Mexico (24), Costa Rica, Guatemala, Venezuela, Colombia (30) and Brazil (23, 85). Disease losses can approach 100% when badly contaminated seed is planted under conditions favorable for disease development (91). For example, yield losses of 95% or 38% occurred when a susceptible cultivar was inoculated one or six weeks after plant emergence, respectively, in the highlands of Colombia (20, 43).

Colletotrichum lindemuthianum is a pathogen of *Phaseolus vulgaris* L., *P. lunatus* L., *P. limensis* Macf., *P. acutifolius* var. *latifolius* Fre., *P. coccineus, P. aureus* Roxb., *Vigna unguiculata* and *Vicia faba* L. (67, 86, 91).

Common names frequently used for anthracnose in Latin America are antracnosis and antracnose.

Etiology

Colletotrichum lindemuthianum is a member of the Fungi Imperfecti and produces septate, branched mycelium with changes in color from hyaline to nearly black upon maturity. Unicellular hyaline conidia are produced which measure 4 to 5 by 13 to 22 μ. They usually contain a clear vacuole-like body near the center. Conidial shape may be oblong, cylindrical, kidney-like or S-like with rounded or slightly pointed ends. A

conidium may germinate in six to nine hours and produce one to four germ tubes which form appressoria at their tips during pathogenesis (86, 91).

Conidia are borne in acervuli on host tissue. En-mass, the conidia appear salmon, ochraceous or pink. Conidia are borne on hyaline, erect, unbranched conidiophores 40-60 μ in length. Setae may appear in culture among the conidiophores or on the host at the margin of an acervulus. They are pointed, stiff, septate brown hairs 30-100 μ long (91).

Optimum fungal growth occurs in culture at 22.5°C (57). Conidial production is optimum between 14°-18°C (31, 32, 86, 91), and is severely limited or prevented by temperatures greater than 30°C (31, 32, 91). Sporulation is favored at pH 5.2 - 6.5, and is unaffected by aeration, natural or ultraviolet light (59). Bean pod agar medium (31, 32), sterilized pods (91), potato-dextrose agar and Czapek medium (91) most often are used for culture growth. However, some isolates sporulate only when grown on a medium containing glucose, mineral salts and neopeptone (59). 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.* (48) stored isolates for 30 months at -150° to -196°C with no loss in viability or pathogenicity.

The perfect stage of the fungus originally was called *Glomerella lindemuthiana* Shear. (81), but recently has been renamed *G. cingulata* (52). The fungus produces perithecia with a diameter of 120-210 μ and rostrum 30-80 μ in length. Perithecia contain hyaline and filiform periphyses visible until 27 days of age and asci which measure 8 by 48-68 μ and disappear after 27-30 days. Each ascus contains eight ascospores which may be alantoid (6.5 x 20 μ) or ellipsoid (4 x 10 μ) in shape. Ascospores are ejected from the ascus (52).

Infectious viral particles have been detected in isolates of *Colletotrichum lindemuthianum* and transferred to virus-free isolates by hyphal anastomisis (28). Radial growth and sporulation by infected isolates are reduced, but there are no reports of altered pathogenicity.

Paradela Filho and Pompeu (68) report that a different species of *Colletotrichum* was isolated from anthracnose-infected plants in Brazil. The fungus was identified as *C. dematium* f. *truncata* (Schw.) V. Arx, and possessed hyaline, curved, canoe-shaped, unicellular conidia 27 x 35 μ and setae among the conidiophores. Further research is necessary to confirm this report and determine the frequency and importance of this species.

Epidemiology

Infection by *Colletotrichum lindemuthianum* is favored by moderate temperatures between 13° - 26°C (25, 37, 48, 54, 85, 91), with an optimum of 17°C (54). Temperatures greater than 30°C limit infection and development of the fungus (75, 78). High humidity (greater than 92%) or free moisture also must be present for infection to develop successfully (25, 37, 54, 85, 91). Moderate rainfalls at frequent intervals also are essential for the local dissemination of conidia present in a water-soluble gelatinous matrix and the development of severe anthracnose epidemics. Conidia also may be spread by the movement of insects, animals and man, especially when plant foliage is moist (91).

Plant Infection

C. lindemuthianum conidia may germinate in six to nine hours under favorable environmental conditions to form a germ tube and appressorium which are attached to the host cuticle by a gelatinous layer (29, 91). The pathogen penetrates the cuticle and epidermis by mechanical means applied by the appressorium and infective hyphae which develop from it (29, 56, 91). Infective hyphae enlarge and grow between the cell wall and protoplast for two to four days without apparent damage to the host cells. Several days later, the cell walls are degraded, probably by L-galactosidase (35), and the protoplast dies, leading to the appearance of water-soaked lesions (56, 62, 91). Mycelium then may aggregate within the lesion site and form an acervulus which ruptures the host cuticle. The acervulus contains a stromatic layer of three to 50 conidiophores, depending upon the lesion size (91).

Symptomatology

Symptoms of anthracnose infection may appear on any plant part depending upon time of infection and source of inoculum. Infected seed and crop debris are primary sources of inoculum for local epidemics. Initital symptoms may, in fact, appear on the cotyledonary leaves as small, dark brown to black lesions. Conidia and hyphae then may be transported by rain or dew to the developing hypocotyl where infection causes minute flesh-to-rust-colored specks. The specks gradually enlarge lengthwise along, and partially around, the hypocotyl and young stem, forming a sunken lesion.

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

become dark brown to black (Fig. 1 and Fig. 2). Sporulation can occur in lesions on the petiole and larger leaf veins, thereby producing secondary inoculum (91). Pod infections appear as flesh to rust-colored lesions which develop into sunken cankers (1-10 mm in diameter) delimited by a slightly raised black ring surrounded by a reddish brown border (Fig. 3).

The lesion center is light colored, and during periods of low temperature and high moisture may contain a gelatinous mass of flesh-colored conidia which, with age, may dry down to gray-brown or black granulations. Young pods may shrivel and dry up if severely infected. The fungus can invade the pod and infect developing seeds (Fig. 4), whereby mycelia and conidia may infect the cotyledons or seed coat. Infected seeds often are discolored and may contain dark brown to black cankers (Fig. 5) (91).

Control by Cultural Practices

Production of anthracnose-free bean seed has been accomplished in various regions of the world to control the disease (22, 23, 25, 50, 90, 91). Pathogen-free seed of susceptible cultivars is produced in semi-arid, irrigated regions where high temperatures and low humidity conditions are unfavorable for infection and survival by the anthracnose fungus. While the use of pathogen-free seed could reduce losses greatly, few countries in Latin America possess either the production areas and/or the facilities necessary to produce and distribute clean seed to growers (85, 91). Heat treatment of contaminated seed at 50° - 60°C successfully eliminated the fungus; however, seed viability was significantly reduced (91).

Because the pathogen can survive in infected crop debris for two years, crop rotations of two to three years are recommended (91, 92). Infected plant debris should be removed from fields soon after harvest (25). It also is important to restrict the activity and movement of man and agricultural implements throughout a field when the foliage is wet from rain or dew (85).

Control by Chemicals

Various chemical treatments have been examined as a control for bean anthracnose. Seed coat infestations are controlled effectively with Ferban, Ziram (25), Arasan 75 or Thiram (23) and Ceresan (0.5 g/100 g seed). However, internal seed contamination may not be reduced (92). Preventive spraying with protectant or systemic fungicides has been attempted with limited success (49, 82, 84, 92). Maneb (23, 25, 49, 92) and Zineb at 3.5 g/l (25, 69, 91), Benomyl at 0.55 g/l (21, 40), Difolatan 80 or Captafol at 3.5 kg/ha (43), Carbendazim at 0.5 kg/ha (21) and Du-Ter or Fentin Hydroxide at 1.2 g/l (69) have been used to control anthracnose.

Fig. 2 - (above) Anthracnose symptoms on upper leaf surface and petiole.

Fig. 1 - (left) Initial anthracnose lesions on veinlets of lower leaf surface.

Fig. 3- Sporulating pod lesions caused by anthracnose infection.

Fig. 4- Mycelial development by the anthracnose fungus within developing bean pod.

Fig. 5- (right) Seed infection by anthracnose.

Crispin *et al.* (25) recommended spraying foliage at flower initiation, late flowering and pod-filling to achieve satisfactory control. However, fungicides are expensive and may have limited availability in Latin American bean production.

Control by Plant Resistance

Physiologic Specialization

In 1918 it was discovered that cultivars differed in their reaction to infection by *Colletotrichum lindemuthianum* and that the pathogen possessed pathogenic variability. Barrus (14, 15) originally described two physiological races designated alpha and beta. The gamma race then was discovered (17), followed by the delta (2) and epsilon races (19). A mutant of the alpha race (designated alpha 5N) was later named lambda (46).

Recently, Schnock *et al.* (79) discovered the Ebnet strain, subsequently renamed the kappa race (53). Hubbeling (47) isolated the jota race from a greenhouse inoculation of kappa-resistant seedlings with a mixture of kappa, gamma, delta and lambda races. However, the jota race has not yet been detected in nature. Race designations have been based on the differential reactions of anthracnose isolates when inoculated onto differential host cultivars possessing different gene(s) for resistance to one or more races (90).

Numerous surveys have been made throughout the world to identify the prevalence and distribution of specific races. Unfortunately, workers have used different sets of differential cultivars and race designations, making it difficult to compare their data. For example, workers in Mexico (88, 89) used eight differential cultivars to classify isolates MA-1 through MA-10 as belonging to Mexico groups I, II, III corresponding roughly to the beta race, and races MA-11 through MA-13 corresponding roughly to the alpha race. Races in Australia have been designated Aust-1 to -8 (87), or as races 1, 2, 3 (26). Races in Germany have been designated A-E, G-N, X (70), and alpha, beta, gamma (80).

Bannerot (11) has designated races in France as PV_6 (alpha), D_{10} (beta), E_{8b} (gamma), I_4 (delta), L_1 (epsilon) and L_5 (gamma plus delta). The alpha, beta, gamma, delta and epsilon races occur in Italy (37). Races alpha, beta, gamma, delta, epsilon, lambda have been identified in France, Holland and/or Uganda (19, 45, 57, 64). Brazilian races have been identified as alpha, beta, gamma, delta, Brazilian-alpha, Brazilian I, Brazilian II, Mex I and Mex II (3, 4, 7, 51, 65, 66, 71). Races alpha, beta and gamma occur in Chile (63); and the beta and gamma races are prevalent in Colombia (20, 21, 43).

Therefore, it is apparent that much pathogenic variability exists throughout the world. However, an international set of differential cultivars and race designations must be developed to coordinate the research efforts by all workers and facilitate the exchange of data and resistant germplasm.

Physiology of the Host - Parasite Interaction

Much research has focused on the host-pathogen interaction resulting from infection of a specific cultivar by a specific race (pathogenic or non-pathogenic). Griffey and Leach (42) inoculated cultivars of different ages which were differentially susceptible or resistant to various races. They found a similarity between the small necrotic lesions formed on old tissue of susceptible cultivars and the same lesions on young tissue of resistant cultivars. They concluded that the former reaction was due to plant maturation, while the latter reaction was due to a specific protoplasmic reaction. The fungus develops slower in a resistant cultivar than in a susceptible one, thereby allowing the defense reaction of the plant to develop sufficiently (5, 9, 10). Also, the pathogen did not produce cell wall degrading enzymes, such as L-galactosidase, as early as in susceptible cultivars (33, 35).

Inoculation with a non-pathogenic race may protect the host from subsequent infection by a pathogenic race (33, 83). However, this protection is located only in tissue actually infected previously by the non-pathogenic race (83). Injury by mechanical means (6, 37) and freezing of local tissue also can induce localized protection. The latter phenomenon may be regulated by a different mechanism than that conferred by inoculation with a non-pathogenic race (74).

Heat treatment (32° - 37°C) of tissue before inoculation also can confer local and systemic protection which is non race-specific (34, 72, 75). Heat treatment decreased the effectiveness of the mature plant small lesion reaction and systemic protection, but it did not affect the effectiveness of local protection or race-specific resistance. This suggests that there may be two groups of resistance mechanisms operating (33, 34).

Resistant cultivars produce a higher quantity of plant metabolites, such as phaseollin (inhibitory to *Colletotrichum lindemuthianum in vivo*), than do susceptible plants (73, 76), and phaseollin accumulates earlier in resistant plants infected by a non-pathogenic race (10). Phaseollin, phaseollidin, phaseollinisoflavan and kievitone accumulated in tissue infected by pathogenic or non-pathogenic races (9).

Phenylalanine ammonia lyase levels increase in tissue prior to lesion formation and may be related to the subsequent production of compounds such as phaseollin, isoflavonoid and coumestrol (77). The fungus is not sensitive to phaseollin *in vitro* (9), because it can metabolize phaseollin into less toxic compounds such as 6a - hydroxyphaseollin, 6a, 7 - dihydroxy-phaseollin and others (44). However, there is little evidence that metabolic conversion of phaseollin by *C. lindemuthianum* is important during the host-pathogen interaction.

Inheritance of Resistance

Resistance to anthracnose is the most appropriate control measure (Fig. 6) (38, 46, 49) and has been used extensively in North America and Europe. While several sources of resistance have been identified in Latin America,

Fig. 6- Resistant and susceptible bean germplasm.

little effort has been directed towards incorporating resistance into commercial cultivars (3, 7, 27). Resistance to the alpha and beta races is controlled by single, independent, dominant genes (60, 61) which have been combined in cultivars such as Charlevoix (1) and Wells Red Kidney (89).

Although Burkholder (16) 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 with the presence of 10 genes in three allelomorphic series composed of duplicate genes for resistance, a dominant gene for susceptibility and interactions at three loci (2). Resistance to alpha, beta and gamma races included duplicate and complementary factors, as well as multiple alleles (18).

Recent sources of resistance include the Venezuelan black bean named Cornell 49-242 (ARE gene) which is resistant to races alpha, beta, gamma,

delta, epsilon, and lambda (8, 11, 41, 46, 53, 58, 60); but it is susceptible to Brazilian-alpha, kappa, and jota races (38, 47). The ARE source of single gene dominant resistance has been reported to have undesirable linkages (90). Leakey (57) recommended that the ARE gene from French accessions such as Confinel, Peonel and Verdon be used in place of Cornell 49-242. However, Fouilloux and Bannerot (39) created four pairs of isogenic lines derived from Cornell 49-242 with no apparent unfavorable pleiotropic effects.

Other genetic sources are resistant to many races and consist of Mexico 222 and Mexico 227 containing the dominant gene Mexique 1, which may be composed of an allelic series (13, 38). Additional genes such as Mexique 2 and Mexique 3 also are resistant to the kappa and Brazilian-alpha races (38). Resistance to alpha, delta and kappa occurs in Kaboon, Coco a la Creme, Kievit Koekoek, BO-22 and Evolutie (12, 53). However, P.I. 150414, Titan and Metorex are moderately resistant to kappa, while an unspecified accession of *Phaseolus coccineus* is resistant to all known races (53). In addition, P.I. 165422 and P.I. 207262 are resistant to the kappa and jota races (47).

Workers have relied completely upon race specific resistance to control specific races of *Colletotrichum lindemuthianum*, and the fungus has expressed much pathogenic variability by mutation, natural selection or other mechanisms. Mycelium of non-pathogenic races also can survive in lesions in resistant tissue for up to 25 days. Possibly this could result in the development and selection of new pathogenic races (36). Therefore, pathologists and breeders must work closely together to develop new and stable sources of resistance (race-specific and possibly race non-specific) which will control yield losses incited by the anthracnose fungus. In addition, a uniform race differential series and system for evaluation and inoculation of germplasm must be developed.

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Chapter 4

Angular Leaf Spot

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Introduction

Angular leaf spot of beans is caused by *Isariopsis griseola* Sacc. which is prevalent in tropical and subtropical regions such as Brazil, Colombia, Costa Rica, Guatemala, Mexico, Peru, Venezuela, and in Africa. It also exists in other regions, such as Australia, Europe, India, Iran, Israel, Japan and the United States (2, 7, 9, 12, 13, 14, 18, 20, 28, 31, 32, 33, 36). Yield losses can be quite severe and have reached 50% in the United States (18), 40 to 60% in Colombia (2), and 80% in Mexico (14).

The fungus has a host range which includes *Phaseolus vulgaris, P. lunatus* (9), *P. multiflorus* (6), *Pisum sativum* (10) and *Vigna sinensis* (15). Abramanoff, cited by Cardona-Alvarez and Walker (9), considered soybeans *(Glycine max)* 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.

Etiology

Isariopsis griseola is an imperfect fungus and is synonymous with *I. laxa* (Ell.) Sacc., *Graphium laxum* Ell., *Phaeoisariopsis griseola* (Sacc.) Ferraris, *Cercospora columnare* Ell. and Ev., *Lindaumyces griseola* Gonz. Frag., *Arthrobotryum puttemansii* Henn. and *Cercospora sthulmanni* Henn. (7, 36).

In nature the fungus produces groups of eight to 40 conidiophores, which are joined together loosely to form the dark columnar coremia or synnemata which bear conidiospores. A synnemata may have a diameter of 20 to 40 μ and be 500 μ in length. The conidiophores tend to separate near maturity and fructification (10). Conidia are gray, cylindrical to fusiform, slightly curved, and measure 7 to 8 μ x 50 to 60 μ with one to five septations (36).

Isariopsis griseola grows slowly on culture media and requires 24°C and a pH of 5 to 6 for optimum development. Adequate growth media include potato-dextrose agar plus bean leaf extract (7, 9), honey peptone agar, baby food (assorted vegetables) - calcium carbonate agar (25), 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 $CaCO_3$, and 18 g Bacto-agar added to sufficient distilled water to make 1 liter) (11). Discreet colonies form on the media, and single spore isolates may exhibit variation within a petri plate for colony structure, coloration and quantity of sporulation (7).

Epidemiology and Plant Infection

The pathogen infects leaf tissue by entering through stomata and advancing intercellularly in the mesophyll and palisade parenchyma. Within nine days after infection, the fungus develops intracellularly throughout necrotic lesions. Within nine to 12 days, stromata develop in the substomatal cavity and sporulation then may occur during periods (24 to 48 hours) of continuous moisture (7, 9). Optimum temperature conditions for development of synnemata and conidia in culture and under natural conditions range from 20° to 25°C (9, 29).

Seed transmission may occur (16, 24, 32), but the fungus survives primarily in infected plant debris on and in soil for up to 140 to 500 days (7, 9, 14, 32). The fungus may be disseminated from the debris by splashing water or wind-blown soil particles and from sporulating lesions by wind currents (7, 9).

Epidemic development may be affected by the type of cropping system used to produce beans. Moreno (22) reports that angular leaf spot infection was more severe in beans grown in association with maize than in association with sweet potato, cassava or in monoculture.

Symptomatology

Symptoms of infection are most common on leaves and usually appear within six days after inoculation (21). Lesions may appear on the primary leaves, but usually do not become prevalent on subsequent foliage until late flowering or early pod set (4). Lesions initially are gray or brown, may be surrounded by a chlorotic halo and have indefinite margins. Lesions become necrotic and well-defined with the typical angular shape by nine days after infection (Fig. 1). Lesions then may increase in size, coalesce and cause partial necrosis and yellowing of leaves, followed by premature defoliation.

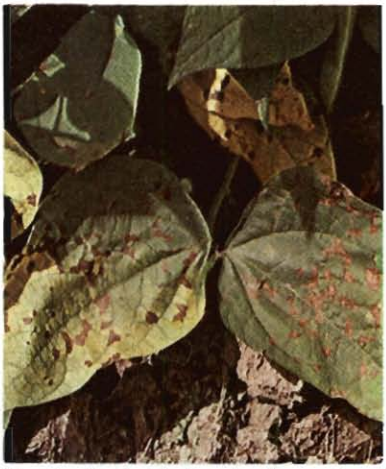

Fig. 1 - Typical lesion development and accompanying chlorosis caused by *Isariopsis griseola* infection of bean leaves.

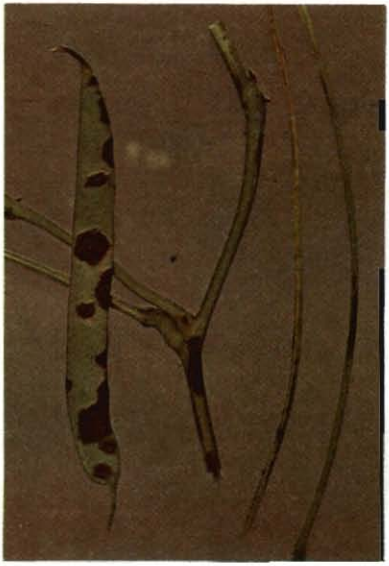

Fig. 2 - Pod, branch and petiole infection by the angular leaf spot fungus.

Lesion size may be inversely related to lesion number per leaf or leaflet (11). Lesions may appear on pods (Fig. 2) as oval to circular spots with reddish-brown centers surrounded by darker colored borders (4, 7, 9, 14, 33, 36). Infected pods may bear poorly-developed or entirely shriveled seeds (4). Brown, elongated lesions may occur on plant stems, branches and petioles as also shown in Fig. 2 (7, 9, 14). A characteristic sign of *Isariopsis griseola* is the production of dark gray to black synnemata and conidia in lesions on the lower leaf surface (Fig. 3), stems, branches and pods during long periods of high humidity or free moisture (7, 9). The pathogen can be seedborne (16, 24, 32).

Fig. 3- Synnemata production on lower surface of bean leaf.

Fig. 4- Bean plant infection from previously infested bean debris.

Control by Cultural Practices

Crop rotation for at least two years, planting pathogen-free seed, planting in well-drained soils, and removal of previously infected crop debris are advised control procedures (3, 7, 13, 14). Fig. 4 illustrates young bean plants infected by spores liberated from adjacent infected crop debris which had not been removed from the field after the previous bean production.

Control by Chemicals

Chemical control measures include Ferbam-sulfur-adherent (5), Zineb (3), Benomyl (0.5 g/l) and Thiophanate (0.2 g/l) (30). Costa (13) recommends the use of Maneb, Ziram, Copper Oxychloride and Bordeaux Mixture. González *et al.*(17) obtained control economically by applying Mancozeb, Captafol and Metiram 20, 30 and 40 days after planting. Chemical seed treatment also may be warranted if seed lots are suspected to be contaminated. Araya (1) found that seed treatment with Benomyl reduced subsequent leaf infection significantly.

Control by Plant Resistance

Various workers have identified sources of plant resistance to angular leaf spot. Brock (6) reported that Alabama No. 1, Cafe, California Small White, Case Knife *(Phaseolus coccineus),* Epicure, McCaslan, Navy Bean, Negro Costa Rica, Scotia and Rojo Chico are resistant. Other resistant cultivars include Mexico 11, Mexico 12, Cauca 27a (23), Fin de Lima (15), Caraota 260 (26, 27, 34), Cuva 168-N, Manteigao Preto 20 (13) and others (29). Schieber (28) observed field resistance in a group of Guatemalan accessions identified as 2465, 2503-12, 2504 and 2809.

Inheritance of resistance has been studied and is conferred by recessive and dominant genes, depending upon the parental cultivar. Santos-Filho *et al.* (26) reported that the resistance of Caraota 260 is controlled by a single recessive gene. Barros *et al.* (2) 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 (8) found that Line 258 possessed dominant resistance governed by a single gene.

Researchers must develop methodology to produce inoculum uniformly and to screen germplasm in the laboratory, glasshouse and field. Singh and Sharma (30) utilized field screenings by inoculating soil with previously infected bean debris. Spores of *Isariopsis griseola* have been harvested with good results at CIAT (11) from PDA or V_8 juice agar, suspended in sterilized, distilled water (2×10^4 spores/ml) plus dispersing agents such as gum arabic (2-5 g/l) or Triton - AE (0.1% soln.) and sprayed onto plants in the glasshouse or field during optimum conditions (high moisture and moderate temperature). A uniform evaluation scale also must be developed and accepted by workers. Moreno (22) classifies infection grades by the following scale: 1 = no infection; 2 = less than 5% of foliage with lesions; 3 = 25% of foliage with lesions; 4 = 50% of foliage with lesions; 5 = yellowing and death of foliage.

CIAT (11) utilizes the following leaflet evaluation scale:

- immune, no infection
- resistant, less than 2% actual leaflet area infected
- intermediate, 3-10% actual leaflet area infected
- susceptible, 11-25% actual leaflet area infected, may be accompanied by limited chlorosis
- very susceptible, more than 26% actual leaflet area infected, often accompanied by chlorosis and/or defoliation.

Villegas (35) inoculated 14 differential cultivars individually with 30 single spore isolates of the angular leaf spot pathogen which had been collected from different bean production sites in Colombia. He concluded that the isolates contained 13 different pathogenic races, but he questioned the genetical purity and uniformity of the differential cultivars he utilized. Hocking (19) recovered an isolate in Tanzania which produced circular lesions and was highly virulent at 10^2 spores/ml. He speculated that the isolate may have been due to a single mutation within natural isolates.

Most cultivars have been tested only against local isolates of the fungus and should be exposed to other populations to ascertain the specificity of the host-parasite interactions and to confirm the possible existence of different pathogenic races which could influence the breeding strategy utilized to control *Isariopsis griseola* losses.

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Chapter 5

Root Rots

H. A. Bolkan

Chapter 5

Root Rots

Introduction

Root rots of beans have been studied much less in Latin America than have foliar diseases. Specific root rot diseases are known to occur in several countries (35, 42, 59, 61, 68), but there are few reports of yield loss assessment or research concerned with developing control measures adapted for specific production regions. While root rot pathogens generally cause less conspicuous symptoms than foliage infecting pathogens, root rot diseases can greatly reduce plant development and production. This section describes various bean root rot pathogens and factors which influence their growth, pathogenicity, reproduction, survival and control.

Rhizoctonia Root Rot

Introduction

Rhizoctonia root rot, caused by *Rhizoctonia solani* Kuhn (*Thanatephorus cucumeris* (Frank) Donk), is a common root rot disease of beans in Latin America and the world (3, 41, 42, 51, 68, 87, 154, 166). The fungus is distributed throughout most agricultural soils at various levels of infestation (11, 93) and can infect a wide range of taxonomically different plants. Losses of more than 10% have occurred in the United States (166). The disease is relatively unimportant in the states of Minas Gerais and Goias in Brazil (74); but *R. solani* together with *Fusarium solani* f. sp. *phaseoli*, have caused yield losses of up to 60% in Sao Paulo (68).

Common names frequently used for Rhizoctonia root rot in Latin America include chancro, tizón, pudrición del tallo, tombamento, podredumbre del tallo and podridao radicular.

Etiology

Although highly variable for morphological characteristics, isolates of *R. solani* are commonly identified by production of:

- multinucleated cells, especially in young vegetative hyphae
- a prominent septal pore apparatus in the septum
- a constriction of hyphal branches at the junction of main hyphae and formation of a septum at the branch near the point of origin
- branching near the distal septum of cells in young hyphae
- brown coloration of mature aerial hyphae (113).

Monilioid cells and sclerotia are not produced by all isolates, and therefore are not valid criteria for identification.

When grown on potato-dextrose agar (PDA), isolates may differ in mycelium color, zonation, amount of sclerotium formation (67), amount of aerial mycelium, growth rate (109), saprophytic behavior (108), and enzyme production (110). However, they can be stable in the laboratory even after more than 100 transfers during a six-year period (Bolkan, unpublished data).

The perfect stage, *Thanatephorus cucumeris* (37), may occur and form basidia at the base of plants and/or on the underside of soil aggregates during periods of high humidity and rainfall (121). Basidia are relatively short and barrel-shaped with stout straight sterigmata, while basidiospores are smooth, thin-walled and hyaline. Some *R. solani* isolates may be induced to produce the basidial stage *in vitro* (66, 86, 135). *Rhizoctonia solani* utilizes carbon and mineral sources with a high efficiency (133). However, no specific carbon source consistently supports the growth of all isolates (8). *R. solani* isolates are generally auxotrophic (143), but some require specific growth factors (127). The optimum growth temperature is 23°- 28°C, although lower (147) and higher optima have been reported for various isolates. Specific isolates may also respond differently to varying pH levels, but most isolates attain optimum growth at pH 5-7 (134).

Epidemiology

Rhizoctonia solani contains a wide array of pathogenic isolates (145). Some isolates are specific for one crop, such as beans, while others attack a wide range of hosts (69, 110, 111, 133). Isolates vary in the degree of virulence expressed toward a single host (20, 50, 98) and disease severity is influenced by soil moisture, soil temperature (166), nutritional status of the inoculum (132, 159), and plant and root exudates which stimulate mycelial growth (55, 152).

It is reported that 18°C is the optimum soil temperature for development of hypocotyl cankers. Relatively few cankers develop at temperatures above 21°C (166). Apparently the plants emerge more rapidly at high temperatures and thus escape infection (22, 91, 166).

R. solani inoculum consists of sclerotia, mycelia and basidiospores. However, the importance of basidiospores as an inoculum source is unknown. Inoculum may survive in soil as sclerotia or thick-walled hyphae associated with plant debris (25), and/or by saprophytic growth on organic matter (122). Pathogenic variants may arise during basidiospore production or more commonly by hyphal anastomosis between different field isolates (19, 20). *R. solani* field population levels are dependent upon the presence of a susceptible crop (46). The pathogen can be disseminated into new areas by irrigation water, transplanted material, aerially disseminated sclerotia or spores, and infected seed. The fungus may be internally and externally seed-borne (21, 49, 63, 90). *R. solani* can survive in dry soil particles (128) and may possibly be transmitted via wind-blown soil particles (148).

Symptomatology

Rhizoctonia solani may induce damping-off, stem canker, root rot and pod rot. The fungus can penetrate the intact cuticle and epidermis by infection pegs produced from infection cushions (37) or by individual hyphae (55, 56) and through natural openings and wounds. Penetration is believed to occur by mechanical pressure and enzymatic degradation of host cells (17).

During initial hypocotyl and root infection, the fungus causes dark circular to oblong sunken cankers delimited by brown margins (Fig. 1). As infection progresses the sunken cankers enlarge (Fig. 2), become red,

Fig. 2- (right) Hypocotyl cankers produced by the Rhizoctonia root rot fungus.

Fig. 1- (above) Young lesions caused by *Rhizoctonia solani*.

Fig. 3- Older cankers and pith infection caused by *Rhizoctonia solani*.

rough, dry, pithy (Fig. 3) and subsequently retard plant growth. When seedlings become infected, the fungus incites necrotic lesions or girdling of the stem which may cause damping-off. Reddish-brown cankers (delimited by well-defined borders) often develop on older plant hypocotyls and occasionally extend above the soil surface. Minute brown sclerotia may develop on the surface of, or be embedded in, these cankers. *R. solani* can infect pods in contact with the soil surface, causing water-soaked and brown sunken lesions with distinct margins. These lesions may serve as an inoculum source for beans in transit and insure seed dissemination (166) as well as cause seed discoloration (49).

Control by Cultural Practices

Since *R. solani* has a worldwide distribution (93), including uncultivated soils (11), exclusion and eradication usually are not effective field control measures. Nevertheless, the local pathogenic potential can be increased upon introduction of infested soil, plants or seeds transported from other regions. *R. solani* can be eradicated from infested greenhouse soil by steaming at 60°C for 30 minutes (93).

Rhizoctonia solani infection may be reduced by various cultural practices. Seedling injury is minimized by shallow planting so that less seedling tissue is exposed to inoculum, but increased plant lodging may occur. Manning *et al.* (102) report that seed planted 7.5 cm deep developed more root rot and hypocotyl injury than seed planted only 2.5 cm deep. In the San Joaquín Valley of California, shallow planting (1.5 - 2.5 cm deep) apparently reduced disease severity without the need for fungicidal application (93).

Planting should be delayed until soil has warmed sufficiently to reduce *R. solani* infection (22, 166). Crop rotation with non-host crops can reduce the incidence of bean root rot but does not completely eliminate the pathogen. *R. solani* populations rapidly declined in soil planted to wheat,

oats, barley or corn. Population levels remained relatively high in soil planted to susceptible bean, pea or potato plants (166).

A suggested but yet unproven alternative to crop rotation is soil amendment with decomposable materials (25, 93) or the incorporation of selected plant residues (101, 111, 138). Snyder *et al.* (138) demonstrated that bean infection was significantly reduced in greenhouse studies by incorporating a barley, wheat or corn amendment. Similarly, Manning and Crossan (101) showed that a corn amendment significantly reduced hypocotyl rot under glasshouse and field conditions, the inhibitory effect lasting nearly a year. However, this control measure has not been used on a practical basis under field conditions.

Control by Chemicals

Fungicides which control *R. solani* infection include: PCNB, Benomyl, Vitavax or Carboxin, Busan, Thiram, Zineb, Demosan or Chloroneb and Captan (1-3 g a.i./Kg seed). These fungicides commonly are applied as seed treatments prior to or during planting (21, 63, 115). PCNB is the fungicide most commonly used to control *R. solani* and Crossan (44) reported that PCNB applied as a low volume spray (5.8 kg in 378 l water/ha) behind the planting shoe wetted seed and soil in the furrow during planting to provide excellent *R. solani* control. Similar results are reported by Abdel-Rahman (1) and Bristol *et al.* (27). PCNB and Demosan are highly specific towards *R. solani* and should be mixed with Captan or Pyroxychlor where *Pythium* spp. also are a problem (93). Chemical control of *R. solani* often is effective for seedling emergence and development but seldom provides protection to the expanding root zone of older plants.

Campbell and Altman (33) report that the herbicide, Cycloate, reduced the colonization of bean segments by *R. solani* and was probably due to an inhibition of the fungal growth rate. However, Grinstein *et al.* (72) report that Dinitramine reduced plant resistance to infection by *R. solani*.

Control by Plant Resistance

Older plants often are more resistant to *R. solani* infection, possibly due to increased calcium content in plant tissue (18), induction of phytoalexins (120, 137, 150) and/or decline in hypocotyl and root exudates which stimulate infection cushion formation by the fungus (48).

It has been difficult to identify a high degree of resistance to *R. solani* in dry bean germplasm. However, a lima bean line was resistant to *R. solani* infection and the resistance was inherited as a single dominant factor (166).

The dry bean cultivar Uribe Redondo was reported by Cardona (34) to be highly resistant to Rhizoctonia root rot in Colombia. Prasad and Weigle (123, 124) report that Venezuela 54 and P.I. 165426 are highly resistant to *R. solani* infection and suggest that resistance may be linked to dark seed coat color. Extracts from black seeds contained phenolic substances inhibitory to the growth of *R. solani* (125). Dickson and Boettger (54) have observed a relationship between black-seeded materials and resistance, but now have identified white-seeded materials with resistance. Recently, two dry bean breeding lines, B 3088 and B 3787, and a wax bean cultivar were reported to be highly tolerant to Rhizoctonia root rot (165). Resistance to other root rot pathogens and possibly nematodes may have to be combined with resistance to *R. solani* to provide sufficient protection against the complex of soil pathogens which commonly occurs in bean production regions of the world.

Fusarium Root Rot

Introduction

Fusarium root rot of beans is caused by *Fusarium solani* (Mart.) Appel and Wollenw. f. sp. *phaseoli* (Burk.) Snyder and Hansen. The pathogen is prevalent and causes varying degrees of damage in most bean-growing areas of the United States, such as New York, Idaho (155) and Nebraska (142). It has been reported also in Spain, Bulgaria, and England (166). In Latin America, Fusarium root rot has been identified in Brazil (41, 68, 154), Colombia (13), Peru (59), Venezuela (35), Costa Rica (61) and Mexico (43). Keenan *et al.* (85) reported that an unusually high yield reduction of 86% occurred due to a decrease in pod number/plant in Colorado. Burke and Nelson (31) found that yield losses under severe disease pressure ranged from 6-53%, depending upon the cultivar. Galli *et al.* (68) considered Fusarium root rot an important bean disease in Brazil, but they made no estimate of economic losses caused by the pathogen.

Phaseolus vulgaris L., *P. limensis* L., *P. coccineus, P. angularis* (Willd.) W.F. Wright, *P. lunatus* L., *Pisum sativum* L., *Vigna unguiculata* (38), *Onobrychus vicifolia* (10), *Phaseolus acutifolius* var. *latifolius, P. aconitifolius* Jacq., and *Pueraria thunbergiana* (Sieb. & Zucc.) Benth. (166) may be infected by *Fusarium solani* f. sp. *phaseoli.*

Common names frequently used for Fusarium root rot in Latin America are pudrición seca and podridao radicular seca.

Etiology

When the fungus is grown on artificial media such as potato-dextrose agar or Czapeks, the production of aerial mycelium is sparse and usually

grayish-white. The fungus produces chlamydospores, macroconidia and microconidia, although the latter are rarely observed. Macroconidia develop mainly from short multibranched conidiophores which emerge to form effuse minute sporodochia without a stroma. Macroconidia are hyaline and fusiform with a pointed, slightly-beaked apical cell. Size of macroconidia and number of macroconidial septa vary according to culture medium used and incubation conditions. Macroconidia generally range in length from 44 to 50 μ, in width from 5.1 to 5.3 μ (166), and are three or four septate but rarely five septate (155). Microconidia develop from sparsely-branched conidiophores. Microconidia are broad, oval and may have one septation. Chlamydospores are 6-16 μ in diameter (106) and form terminally on short lateral branches or intercalarly. They form singly, in pairs, or occasionally in short chains and may be round-subglobular or pear shaped.

Epidemiology and Plant Infection

Fusarium solani has a low mobility (28) and exists in naturally infested soil as chlamydospores associated with or embedded in tissue fragments or humus particles. Macroconidia may form on plant lesions which extend above the soil surface, and upon dissemination into soil are converted into, and survive as, chlamydospores (106). Chlamydospore germination is stimulated by exudates from non-susceptible (130) and susceptible plant roots (129). Under natural conditions, *F. solani* can exist as mycelial- or sporodochial-type vegetative clones (106). Chlamydospores provide the primary field inoculum of *F. solani*. The fungus does not grow saprophytically in the soil (106) except on organic matter (166), and chlamydospores are stimulated to germinate by seed and hypocotyl exudates from nearby plants (40).

Plant susceptibility is influenced by environmental conditions such as soil compaction, temperature and pH. Burke (29) demonstrated that Fusarium root rot is aggravated in compacted soils, the stressed roots being unable to escape infection. He concluded that the fungus has little influence on the yield of plants with vigorously growing roots. Although the fungus has an optimum growth rate on agar medium between 29°- 32°C, Chupp and Sherf (38) report field damage was more severe at 22°C that at 32°C. Infection is reported to be favored by acid soil or by soils fertilized with NH_4-N, and may be suppressed by soils fertilized with NO_3-N (136). However, Burke and Nelson (32) report that the form or rate of nitrogen applied to a field did not affect root rot severity.

Tousson *et al.* (146) demonstrated that infection is influenced by the nutritional status of the inoculum. Glucose enhanced chlamydospore germination and mycelial growth but delayed penetration and subsequent pathogenesis. Nitrogen enhanced early penetration and pathogenesis.

Root rot damage is aggravated during periods of high soil moisture when the oxygen diffusion rate is lowered (103). Hutton *et al.* (84) reported that root rot development was greater where plants inoculated with *Fusarium solani* were associated with nematodes such as *Pratylenchus penetrans* or *Meloidogyne* spp. Apparently, the nematodes influence the initiation of fungal infection. A synergistic effect also occurs between *F. solani* and *Pythium ultimum* (116).

The pathogen is disseminated primarily as chlamydospores or conidia. The fungus is not transmitted to any great extent by soil capillary water movement (28) but may be transported in drainage and irrigation water, in soil adhering to agricultural tools and animals, bean straw, manure and possibly in soil or as spores washed by rain or floods. The primary means of dissemination in New York is within bean straw and manure (38). Once introduced into a new area, the fungus may survive indefinitely as a soil saprophyte on organic matter (166) or as a mycorrhizal component of non-susceptible crops (68). Fungal incidence then may be greatly increased by repeated cultivation of a susceptible host. The fungus is not internally seed-borne, but it may be present in soil particles which adhere to the seed coat surface (166).

Symptomatology

Fusarium root rot initially appears as reddish lesions or streaks on the hypocotyl and primary root (Fig. 4) one to two weeks after seedling emergence. As infection progresses, the lesions coalesce, become brown (Fig. 5) and may extend to the soil surface but rarely beyond. The lesions have no definite margins and may be accompanied by longitudinal fissures. The primary and lateral roots frequently are killed by the fungus and When the primary root is killed, the lower stem

Fig. 4- Root and hypocotyl lesions caused by *Fusarium solani* infection.

Fig. 5- Hypocotyl and root discoloration caused by *Fusarium solani* infection.

may become pithy or hollow. There is no pronounced host wilt, though plant growth may be retarded and exhibit leaf yellowing and premature abscission. Lateral roots often develop above the initial lesions and support plant growth so that a yield still is produced if surface soil moisture is adequate, although pod number per plant and seed size may be reduced. Plants which are heavily damaged and subsequently moisture stressed, may be stunted or killed (166).

Control by Cultural Practices

When virgin soil is put into agriculture production, measures must be taken to prevent introduction of the pathogen in manure containing infested bean residue, contaminated irrigation water or soil adhering to agricultural implements. Eradication on a large scale is uneconomical and generally impossible once the pathogen becomes established within a field (151).

Beans should be grown in well-drained and fertilized soils which allow vigorous plant growth. When infection occurs, shallow cultivation will reduce pruning of lateral roots formed above the lesions sustaining the plant. High plant populations may increase disease incidence due to root competition and concentrated root exudates (Burke, personal communication). Long-term crop rotation with nonsusceptible plants reduces soil populations of, and infection by, *F. solani* (100). However, this method is seldom practical or economically feasible.

Soil amendment with various crop residues may enhance natural biological control by resident soil microorganisms. Maier (99) demonstrated that incorporation of barley straw into soil infested with *F. solani* reduced disease incidence. Adams *et al.* (5) report that Fusarium root rot was controlled under glasshouse conditions with a soil amendment of spent coffee grounds incorporated seven to 14 days before planting, but its field practicality has not been demonstrated. Actinomycete activity and suppression of *Fusarium solani* in the rhizosphere also may be influenced by the quality and quantity of amino acids released by plants (107).

Control by Chemicals

Various chemicals reported to reduce Fusarium root rot in seedling hypocotyls and young roots include: Nabam, Formaldehyde, Thiram, PCNB, Benomyl, Difolatan or Captafol and Busan. Abdel-Rahman (1) obtained good control by application of Benomyl as an overfurrow spray (0.56 kg/ha) immediately after planting. Busan 30 (2.4 l/ha) and Difolatan (4.7 l/ha) also provided adequate control (1). However, most chemical

treatments are not completely effective, since the lateral root development receives little or no benefit from the fungicide (30).

Mussa and Russell (105) report that the herbicides Treflan or Trifluralin and Basagran or Bentazon and the pesticide Metasystox or Oxdemeton-methyl stimulated growth of *F. solani* and may have aggravated root rot problems. Eptam also may increase root rot incidence (162).

Control by Plant Resistance

While root rot resistant cultivars are available, genetic linkage often is detected between resistance and undesirable plant characters (157). Statler (141) found that bean cultivars with purple hypocotyls and black seed coats were more resistant to Fusarium root rot than cultivars lacking this coloration. However, Dickson and Boettger (54) did not find an association between seed color and resistance to *Fusarium solani.*

Wallace and Wilkinson (156) report that N-203 (P.I. 203958) and N.Y. 2114-12 have a high degree of resistance when exposed to low inoculum levels. Resistance to Fusarium root rot may be controlled by three to seven dominant genes (26). Hassan *et al.* (76) confirmed these findings and noted that the gene action is mostly additive. However, a quantitative inheritance and dominant genes for susceptibility occurred in crosses between resistant P.I. 203958 and susceptible California Small White, State Half Runner or Cascade Fulton (23). They also stated that recurrent selection would be the most suitable breeding method to improve this quantitative trait. Boomstra *et al.* (24) recently tested 800 accessions and identified 18 plant introductions (primarily Mexican in origin) and various cultivars which were resistant to Fusarium root rot. There are, however, no reports of the use of tolerant or resistant cultivars in Latin American countries. Pierre (119) reports that phaseollin production inhibits germination and growth of *Fusarium solani* and may play a role in resistance.

Fusarium Yellows

Introduction

Fusarium yellows of beans is caused by *Fusarium oxysporum* Schlecht. f. sp. *phaseoli* Kendrick and Snyder. The fungus occurs in regions of the United States (166) and of countries in Latin America such as Colombia, Brazil and Panama and in Central America (45, 161).

Common names frequently used for Fusarium yellows in Latin America include marchitamiento por Fusarium, murcha de Fusarium and tizón por Fusarium.

Etiology

Fusarium oxysporum produces hyaline, non-septate chlamydospores measuring 6-15 x 2-4 μ. Elongated macroconidia are curved with two to three septations and measure 25-35 x 3-6 μ (158).

Symptomatology

Infection occurs on the roots and hypocotyls, usually at wounds (57). The vascular bundles of the root, hypocotyl, stem and petioles may become

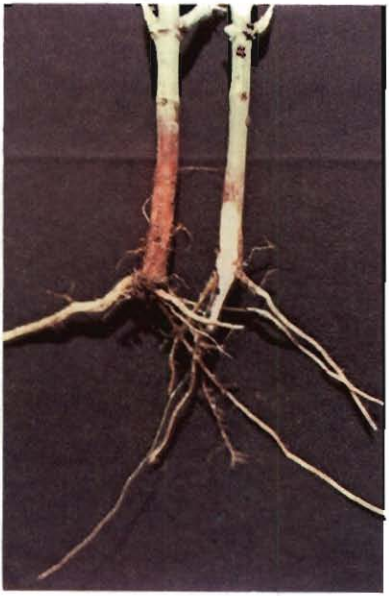

Fig. 6- Root and hypocotyl infection by *Fusarium oxysporum*.

Fig. 7- Leaf yellowing caused by *Fusarium oxysporum* infection.

discolored as tissue turns reddish-brown (Fig. 6). The fungus may cause the plant vascular system to become plugged, which results in slight yellowing and premature senescence of lower leaves (Fig. 7) resembling symptoms caused by phosphorus deficiency. This yellowing becomes more pronounced and progresses into younger leaves; however, plant wilt usually does not occur. Stunting may occur if the plant is infected during the seedling stage. The fungus also can cause water-soaked lesions on pods (71). The fungus is seedborne, probably as spores on the seed coat surface (158, 166).

Control

Control measures are similar to those advocated for *Fusarium solani* and include crop rotation, chemical seed treatment with Ceresan or Semesan, and planting resistant or tolerant cultivars (41, 45, 165).

Resistant cultivars include Manteigao Preto, Manteigao Lustroso, Manteigao 41, Pintado, Roxinho Precoce, Carioca, Pintadinho Precoce, and Rosinha Sem Cipo (45). Dongo and Muller (58) reported that resistant cultivars they have identified generally are red-seeded and produce a large number of strong lateral roots after infection.

Pythium Root Rot

Introduction

Pythium root rot is caused by several *Pythium* species such as *P. ultimum* Trow, *P. irregulare* Buism., *P. aphanidermatum* (Edson) Fits. (= *P. butleri* Gubr.) and *P. myriotylum* Drechs (35, 70, 81, 88, 96, 117, 140, 155, 166). Less common species are cited by Zaumeyer and Thomas (166) and Lumsden *et al.* (96). In Latin America, *P. aphanidermatum* appears to be a common species (35).

The disease occurs in the United States (53, 75, 78, 81, 88, 117), Brazil (47, 153), El Salvador (3), Mexico (42, 43) and Venezuela (35). The pathogen is a major problem of snap beans in the United States (53, 117), but its importance in Latin America is not reported.

Common names frequently used for Pythium root rot in Latin America are marchitamiento por Pythium and murcha de Pythium.

Etiology

Pythium species grow well on artificial media, and hyphae are coenocytic. The sexual stage (sporangium) has a filamentous, globose or oval form depending on the species. The sporangia may germinate directly by a germ tube or produce zoospores. Zoospores are kidney-shaped with two lateral flagella. Zoospore production is preceded by formation of a bubble-like vesicle at the tip of a tube which arises from the sporangium. The sexual stage is characterized by union of the oogonium and antheridium, resulting in oospore production. Oogonia are smooth-walled in some species and spiny in others. The antheridium also varies between species for shape, origin and number per oogonium. Oospores are thick-walled, smooth, plerotic (fill the oogonial cavity) or aplerotic (partially fill the oogonial cavity) and germinate by a germ tube.

Epidemiology and Plant Infection

Pythium spp. are natural soil inhabitors which survive by saprophytic growth and resistant structures such as oospores (139, 155, 160). However, they are poor competitors (79) and their saprophytic activities generally are

restricted (14, 15). *Pythium* spp. are favored by high soil moisture (79, 118). *P. ultimum* sporangia can survive for 11 months in soil, and *P. aphanidermatum* zoospores have survived up to seven days in field soil (79). Hoppe (82) reported that *P. ultimum* survived in air-dried soil for 12 years, and at -18°C for 24 months. The optimum pH and temperature for *P. aphanidermatum* oospore germination in sterile soil is 7.5 and 30°C, respectively (4), while *P. ultimum* infection is greater at 15°C (118). Species vary for temperature requirements, since *P. ultimum* and *P. debaryanum* are common at low soil temperature, while *P. aphanidermatum* and *P. myriotylum* are more common at higher soil temperatures (166). Hoch *et. al.* (81) reported that *P. ultimum* is highly pathogenic at 16°C and 28°C, but *P. aphanidermatum* is only slightly pathogenic at 16°C and highly pathogenic at 28°C. However, Pieczarka and Abawi (118) found that a low temperature species, such as *P. ultimum,* was more severe at 15°C than at higher temperatures.

Various workers have studied soil population levels of *Pythium* spp., but their data usually has been influenced by a mixture of pathogenic and non-pathogenic species. Pieczarka and Abawi (117) report that 85% of their field isolates were pathogenic and that the inoculum potential of a low temperature species, such as *P. ultimum,* ranged from 133-1560 propagules/g oven-dry soil. Subsequent greenhouse tests revealed that one propagule/g oven-dry soil was able to cause an 85% reduction in stand.

Dispersal within fields most likely occurs from zoospores which are able to swim in a film of soil water for a few millimeters, or by sporangia and mycelia which are detached and carried by wind or water splash (9). Long distance dispersal may occur by oospores and chlamydospores which are transported in plant or soil debris within irrigation water and possibly by wind-blown soil particles (78).

Penetration by *Pythium* spp. usually occurs through the unwounded host surface after formation of infection pegs (60, 64). Penetration also may occur through natural openings with or without appressorial formation and directly through wounds by individual hyphae (64). Infection is influenced by plant exudates, inoculum density, soil moisture, soil temperature and pH (89, 118). Soil temperature and moisture, however, are the most important factors since *Pythium* spp. are most active as pathogens in soils with high moisture levels (78).

In general, *Pythium* species contribute to the complex involving other root rot pathogens such as *Rhizoctonia solani, Fusarium solani* f. sp. *phaseoli* and nematodes (53, 116). Pieczarka and Abawi (116) report that *Pythium ultimum* acts synergistically with *Fusarium solani* to cause greater *Pythium* root rot, but *Rhizoctonia solani* apparently is antagonistic to *P. ultimum* and reduces Pythium root rot.

Symptomatology

Pythium spp. may infect a germinating seed, cotyledons, terminal bud, radicle and hypocotyl tissue prior to emergence, eventually leading to seedling death (pre-emergence damping off). Surviving seedlings subsequently may be killed three to five days after emergence (post-emergence damping off), or be damaged by root rot or plant wilt and death (117). Pythium root rot symptoms appear as elongated water-soaked areas on hypocotyls and roots one to three weeks following planting. The water-soaked areas may extend several cm above or below the soil level, and 25-75% of the hypocotyl region may be invaded within three weeks (81). As the

Fig. 8 - (above) Pythium root rot symptoms of plants infected (left) and non-infected (right).

Fig. 9 - (right) Sunken lesions caused by Pythium root rot.

infection progresses, lesions become dry and tan to brown (Fig. 8) with a slightly sunken surface (Fig. 9). In later stages of infection, much of the subterranean hypocotyl and fibrous root system is destroyed.

Pythium spp. also may infect seedling or mature plants (6). Plants infected before or shortly after emergence may collapse and die (Fig. 10), symptoms which may be confused with those caused by *Rhizoctonia solani* infection. When infection occurs after hypocotyl cells or main roots have developed secondary wall thickenings, damage commonly is restricted to feeder roots (Fig. 11) and/or to superficial areas on the hypocotyl near the soil surface. Hot and moist weather may induce the fungus to invade the stem cortex and lateral branches, thereby causing older plant wilt and death (166).

Control by Cultural Practices

Since *Pythium* spp. are indigenous to most soils (139), exclusion is not a practical control measure. Pythium root rot may be minimized by cultural practices such as wide plant spacing and soil amendments. Wide plant spacing provides better soil aeration, less soil shading and minimizes

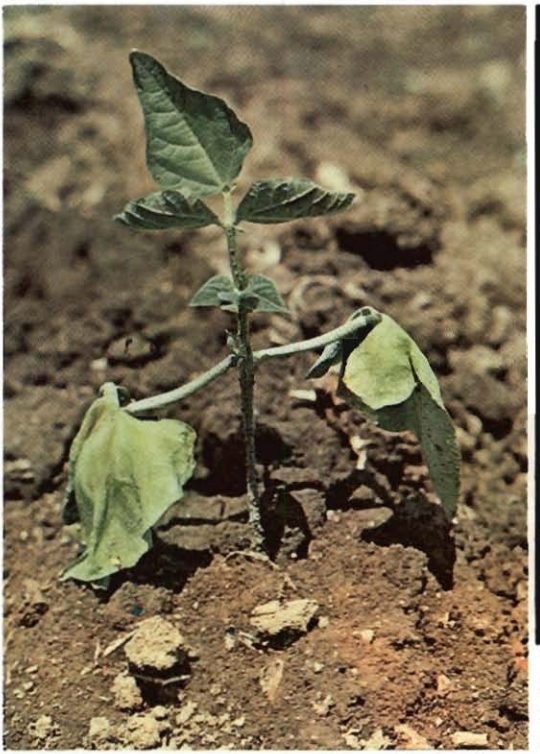

Fig. 10- Post-emergence damping-off caused by Pythium species infection.

Fig. 11 - Pythium damage to feeder roots showing healthy root, left, and damaged root, right.

pathogen spread between plants (166). Nitrogenous compounds can be toxic to and suppress *P. aphanidermatum* when incorporated into the soil (73). Rotation usually is not satisfactory because of the pathogen's wide host range. However, it can influence disease development by reducing soil populations of *Pythium* spp. Disease incidence and severity is affected by root damage (117), and practices such as soil cultivation must be carefully conducted to minimize root pruning. Pieczarka and Abawi (118) suggest that Pythium root rot incidence will be less if beans are planted in well-drained soils and in raised beds or ridges.

Control by Chemicals

Various chemicals reduce the severity of infection caused by *Pythium* spp. These include Dexon or Fenaminosulf, Demosan or Chloroneb, Pyroxychlor, Captan, Thiram, Zineb and combinations of Captan-Thiram, Thiram-Chloroneb or Captan-Chloroneb. Fumigants such as Chloropicrin and Methyl Bromide also have been used (78). Seed treatments with Prothiocarb also are effective (112). However, treatment of a large field may be economically unfeasible. In most instances, the problem is not severe enough to justify chemical control.

Control by Plant Resistance

Certain cultivars are resistant to infection by *Pythium* spp. (7, 53, 75, 164). Adegbola and Hagedorn (7) report that P.I. 203958 and Bush Green Pod are resistant to Pythium blight caused by five species of *Pythium*. The snapbean line 1273 (white seeded) is highly resistant to seed decay and pre-emergence damping-off caused by *P. aphanidermatum* in artificially inoculated soil incubated under growth chamber conditions (53, 164). Resistance was found to be polygenic and recessive in nature, and seedcoat color and resistance were broken. Specific parental combinations did yield a higher proportion of resistant F_3 progeny with colored seed coats (164). Dickson and Boettger (54) found an association between colored seed and resistance to *Pythium* spp. However, line 1273, Black Turtle Soup and P.I. 203958 all were found to be susceptible to the root rot stage of *Pythium* spp. infection, and germplasm may have to be evaluated separately for resistance to each stage of infection (117).

Southern Blight

Introduction

Southern Blight or Sclerotium root rot is caused by *Sclerotium rolfsii* Sacc. (166). The disease occurs in many countries and states between northern and southern latitudes at 38° (38). Latin American countries which have reported Sclerotium root rot as an important disease of beans include Brazil (41, 68, 87, 131, 154), Mexico (42, 43), Costa Rica (62) and Venezuela (35). Direct estimations of losses caused by this pathogen are not available.

Reported host plants include artichoke, bean, brussel sprouts, cabbage, carrot, cauliflower, sweet corn, cowpea, cucumber, egg-plant, endive, escarole, garlic, gourd, ground cherry, lettuce, muskmelon, mustard, parsley, peas, okra, onion, peppers, potato, pumpkin, radish, rhubarb, soybean, squash, sweet potato, tomato, turnip, watermelon, yam and yautia (38). There are no reports of its occurrence on grasses or small grains.

Common names frequently used for Sclerotium root rot in Latin America include añublo sureño, marchitamiento de Sclerotium, tizón sureño, maya or malla blanca, pudrición húmeda, mal de esclerocio, tizón del Sud, murcha de Sclerotium and podridao do colo.

Etiology

Sclerotium rolfsii is characterized by formation of small (approximately 0.5-1.5 mm in diameter), globose, smooth sclerotia. Recently-formed sclerotia are white but turn brown with age. Sclerotia form by occurrence of clamp connections in the hyphae and white coarse mycelium. Basidia may form on mycelial mats and produce thin-walled hyaline basidiospores on short sterigmata (155).

Epidemiology and Plant Infection

Sclerotia are the chief means of survival for *S. rolfsii*. Moisture and high temperatures are required for optimum growth. The fungus is not well adapted to low temperature conditions, but in culture it may grow at temperatures between 13° - 37°C, with an optimum at 30° - 35°C. Sclerotia germinate at 10° - 35°C, and the fungus requires relative humidity above 99%. Sclerotial germination decreases with increased soil depth due to reduced aeration (2). Germination occurs at a pH range of 2.6-7.7 with an optimum at 2.6-4.4 (39). Sclerotial germination is induced by volatiles which emanate from crop residues in the soil (94).

S. rolfsii inoculum consists of sclerotia, mycelium and basidiospores. The role of basidiospores in the life cycle is not known, but Walker considers them to be of minor importance (155). Dispersal may occur by contaminated irrigation water, soil adhering to cultural tools and animals, or seed. *S. rolfsii* sclerotia can pass through the digestive tract of animals without losing viability and, therefore, be transported relatively long distances by animals which feed on infected host materials (92).

Disease development is affected by high temperature and moisture which favor sclerotial germination and optimum mycelial growth. The fungus may penetrate host tissue through natural openings and wounds or may invade by direct penetration of intact tissue (155). Before penetration can occur, appreciable mycelial growth must take place on the plant surface (2, 39). The fungus produces protopectinase and pectinase which cause cell disintegration in bean hypocotyls (166). Bateman (16) reported the production of cellulase, and Van Etten and Bateman (149) detected enzymes which readily degraded pectic galactan, galactomannan and xylan. These substances may play a role in infection caused by *S. rolfsii*.

Symptomatology

Sclerotium rolfsii can cause damping-off, stem blight and root rot. Plant symptoms initially appear as a dark-brown, water-soaked lesion on the

Fig. 12- Hypocotyl and root lesions and sclerotia produced by *Sclerotium rolfsii*.

stem or hypocotyl just below the soil line (Fig. 12). The lesion extends downward through the stem into the tap root and may destroy the cortex (cortical rot). Foliage symptoms consist of leaf yellowing and defoliation in the upper plant branches (166), followed by a sudden wilt (155). Abundant white coarse mycelium, sclerotia and soil often are attached to the stem base. Pods which touch soil also may become infected and rot, and the fungus can be seed-borne (21).

Control by Cultural Practices

Measures should be taken to avoid introduction of *S. rolfsii* into virgin fields in contaminated seed or plant material. Eradication of susceptible weed hosts and destruction of infected host residues by burning or deep plowing reduce soil populations of *S. rolfsii*. Inoculum levels also can be reduced by selecting fields with low soil acidity and good drainage, utilizing wide plant spacing, applying lime to increase soil pH and using a crop rotation with tolerant or resistant crops such as sorghum, corn or other cereals. Soil amendment with nitrate and ammonia as a fertilizer or pre-plant treatment can reduce *S. rolfsii* infection (80, 92). Reynolds (126) reported that a soil amendment with coconut mulch reduced infection and increased yield considerably. Diaz-Polanco and Castro (52) isolated a *Penicillium* sp. which gave good biological control of *Sclerotium rolfsii* under greenhouse conditions.

Control by Chemicals

In general, sclerotia are difficult to destroy with fungicides. However, various fungicides are effective against *S. rolfsii*. They include PCNB, Difolatan 4F or Captafol, Brestanol or Fentin Chloride and Calixin 75 or Tridemorph (38, 65, 104, 144). PCNB (20% active ingredient, 17-22 kg/ha) is effective in Brazil when applied to the seed and surrounding soil in the furrow (68).

Eptam aggravated damage caused by *S. rolfsii* on ladino clover and cotton and reduced biocontrol activity by *Trichoderma viride* (114).

Control by Plant Resistance

Plant resistance has been identified and includes the cultivars Mexico 348-2 and Blanco, which are moderately tolerant to *S. rolfsii* (154). Additional research is required to identify more sources of resistance and/or tolerance to this fungus.

Black Root Rot

Introduction

Black root rot is caused by *Thielaviopsis basicola* (Berk. and Br.) Ferr., and is a relatively unknown root rot disease of beans (155, 166). No report is available on the distribution and importance of this pathogen to beans in Latin American countries, although it is known to occur in the United States, Italy and Germany (166).

Susceptible crops include alfalfa, beans, beet, carrot, celery, corn, cotton, peas, tomato, squash and sweet potato (38, 163).

Common names frequently used for black root rot in Latin America are pudrición negra and pudrición negra de la raiz.

Etiology

The fungus exhibits considerable variation when grown on culture media. Huang and Patrick (83) report that *T. basicola* isolates grown on potato-dextrose agar or V_8 juice agar were variable for colony appearance, zonation, growth rate, production of spores, and the shape and number of cells per chlamydospore. *Thielaviopsis basicola* produces endoconidia and chlamydospores. Endoconidia are borne on young mycelium and are hyaline, small and cylindrical. Chlamydospores originate in chains or clusters produced laterally or terminally on the mycelium. They are hyaline when formed but soon become thick-walled, dark brown and separate at maturity.

Epidemiology and Plant Infection

The fungus persists in soils for an indefinite period (155), and invades roots during cool and wet weather. Maier (99) reported that pathogenicity towards bean hypocotyls decreased as temperature increased, and a constant temperature of 15.5° or 18.5°C favored disease severity. The

Fig. 13- Symptoms of root infection by *Thielaviopsis basicola*.

fungus also is favored by soil alkalinity and NO_3-N (136). *T. basicola* may penetrate host tissue through wounds produced by infection from other pathogens, such as *Fusarium solani* f. sp. *phaseoli* (155), or through uninjured tissue. The fungus appears to develop easily over plant roots in natural soil (28). Apparently intact tissue is penetrated directly without prior appressorium production (36). Lumsden and Bateman (95) report that phosphatidase substances may play a role during penetration of epidermal cells. Chlamydospores are produced abundantly in infected root and hypocotyl tissue and allow fungal survival in the soil.

Symptomatology

The fungus initially infects the hypocotyl just below the soil surface and causes reddish-purple lesions which later turn brown to charcoal-black. As infection progresses, the hypocotyl discoloration extends towards the tap root and rootlets (Fig. 13), and causes plant stunting or death (155, 166).

Control

Pathogen dissemination to distant areas may occur by transportation of infected host residue and/or contaminated soil adhering to animals and agricultural tools. Well-drained soils, eradication of susceptible weeds and planting of non-susceptible crops in infested soils should reduce soil populations of *T. basicola*.

Hassan *et al.* (77) report that line 2114-12 and P.I. 203958 are resistant to black root rot. However, the pathogen seldom becomes severe enough to necessitate a control measure and usually is found in association with other soil-borne pathogens (166).

Texas Root Rot

Introduction

Texas root rot or Phymatotrichum root rot is caused by *Phymatotrichum omnivorum* (Shear) Dugger. Although the fungus has been reported in California and Utah (38), it is largely confined to alkaline soils of southwestern United States and Northern Mexico (97). Crispin and Campos (42) report it is a minor bean disease in Mexico. However, it has not been reported in other Latin American countries. Likewise, no estimation of losses caused by this fungus is available.

The fungus has a wide host range, including fruit and shade trees, ornamental shrubs, weeds and vegetables (38). However, it is principally a disease of cotton and alfalfa (166).

Common names frequently used for Texas root rot in Latin America include marchitamiento de Phymatotrichum, pudrición texana and pudrición texana de la raiz.

Etiology

The imperfect stage of *P. omnivorum* consists of mycelium, conidia and sclerotia. The mycelium may be of three forms: large-celled, fine-celled-strand and acicular hyphae (166). The conidia are hyaline, smooth, globose to ovate and borne on the swollen tip of vegetative hyphae. The function of conidia in the life cycle is unknown since they never have been observed to germinate (155). Sclerotia are dark, vary in shape and size, and are produced singly or in chains. Basidia (perfect stage) are formed in clusters and basidiospores are strongly curved (12).

Epidemiology

The fungus is primarily disseminated as sclerotia or mycelium in soil or crop residue. Sclerotia allow the fungus to survive in soil in the absence of a host. Phymatotrichum root rot is found in localized spots within a field and occurs primarily in soils with a pH of 8.0 or slightly higher (97). The fungus penetrates the host tissue after mycelial strands have enveloped the root (155). Disease development is favored by relatively dry soils at high temperatures. The fungus is not favored by sub-zero temperatures (38) and cannot produce sclerotia at a pH below 5.0 (97).

Symptomatology

The fungus is soil-borne and infects underground plant parts, causing dark, sunken soft lesions which generally are covered with coarse whitish to yellowish mycelium. A pinkish-buff color may be present on lightly infected young rootlets. The above-ground symptoms consist of stunting and sudden wilting, which usually appears during blossom initiation (166).

Control

Long crop rotation with resistant crops such as corn, small cereals, and sorghum; eradication of susceptible weeds (166); choice of soils with relatively low pH; deep plowing and soil application with NH_4-N reduce soil populations of the fungus. Dry bean germplasm should be screened to identify sources of resistance if available and practical as a control measure.

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Chapter 6

Web Blight

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Chapter 6

Web Blight

Introduction

Web blight of beans is caused by *Thanatephorus cucumeris* (Frank) Donk. (3, 24, 29), and is prevalent in tropical regions with high to moderate temperatures and moisture. The fungus was first described in 1917 as *Rhizoctonia microsclerotia* Matz as the causative agent of a fig disease in Florida (44). Since then beans have been identified as a host in the United States (41, 42, 44), Puerto Rico (12), Japan, Philippines, Burma, Ceylon (Sri Lanka), Brazil (6, 32, 44), Costa Rica (13, 37), Colombia, Ecuador, Guatemala, El Salvador, Mexico and Panama (7). Disease losses can be severe as entire crops may be destroyed (3, 23), especially in tropical lowlands and humid subtropical regions.

Thanatephorus cucumeris is a pathogen of nearly all crop plants. Its host range of 200 plant species includes bean, beet, carrot, cucumber, eggplant, melon, tomato, watermelon, and foliage and fruit of uncultivated plants (8, 23).

Common names frequently used for web blight in Latin America include mustia hilachosa, telaraña, chasparria, Rhizoctonia del follaje, murcha de teia micelica and podridao das vagens.

Etiology

The web blight fungus is homothallic and has the imperfect stage known as *Rhizoctonia solani (R. microsclerotia),* which is distributed worldwide (2, 21, 34). The perfect stage was identified in 1891, and the fungus has received a succession of names, such as *Hypochnus solani* (22, 40), *Corticium vagum* var. *solani* or *C. solani* (21, 22, 40), *Rhizoctonia microsclerotia, Corticium microsclerotia, Pellicularia filamentosa* (21, 28, 40, 44) and *P. filamentosa* f. sp. *microsclerotia* (44). The currently accepted form is *Thanatephorus cucumeris* (18). Parmeter *et al.* (35) determined that *Rhizoctonia* isolates which possess multinuclear hyphae have *T. cucumeris* as the perfect stage, while those which possess binuclear hyphae have *Ceratobasidium* as the perfect stage.

Rhizoctonia microsclerotia produces hyaline, granular hyphae (6-8 μ in width) which become septate, more or less empty and brown with maturity. It produces oval, thin-walled and hyaline basidiospores 9-11 μ in length by 5-6 μ in width. Small (0.2-0.5 mm diameter), superficial, white sclerotia also are formed and become brown to dark brown, rough and sub-globose with maturity (42).

Thanatephorus cucumeris was later described as having thin-walled, septate hyphae (5-7 μ in width) which frequently have cruciform branching. Fructifications appear whitish and form on top of a discontinuous hymenium of oblong or barrel-shaped basidia in erect terminal clusters. Basidia measure 15-18 μ in length by 8-10 μ in width, and frequently are connected. Each basidium produces four erect, slightly divergent sterigmata which measure 3 μ in width by up to 15 μ in length. A basidiospore is produced on each sterigmatum and is hyaline, thin-walled, smooth, oblong, ellipsoid with a flat edge or obvalate in shape with a truncated point. Basidiospores germinate by repetition (24, 35, 40).

The fungus grows rapidly in continuous, indirect or intermittent light, and within 24-36 hours can cover the surface of a petri plate containing artificial media incubated at 26°-29°C. Sclerotia form in culture but differ from those produced on host plants, since they are brown to dark brown, irregular in form and size (up to 1 cm in diameter), and more or less flattened (42). Heterokaryosis occurs in *T. cucumeris* and may alter the ability to form sclerotia on minimal media or the isolate pathogenicity (17, 31). Variation can occur due to anastomosis, heterokaryosis, meiosis and mutation (16, 19, 30, 33).

The perfect stage of web blight can be induced *in vitro* (14, 38, 39) with 12-16 hours of light (18, 38, 42, 43), adequate aeration (43), 20°-30°C and 40-60% relative humidity (38, 42). Self-sterile mutants frequently appear in progenies of basidiospores (37, 43), and isolates or species vary for their cultural characteristics and ability to fruit on artificial media or sterilized soil (22, 38). For example, pathogenic isolates of *T. cucumeris* fruit only on sterilized soil, while nonpathogenic isolates fruit on either substrate (38).

Pathogenic variation occurs within and between species of *Thanatephorus* isolated from specific crops, since some species are pathogenic to many crops, others to a limited number of crops. Some are non-pathogenic to all crops (15, 17, 22). Pathogenic variation also is apparent when isolates are grouped according to cultural characteristics (19, 22). Pathogenic races have been identified by their ability to infect differential hosts consisting of wheat, lettuce, tomato, beef, and cabbage (15). Races also differ in their degree of virulence, since some cause leaf death while others may produce only a few leaf spots within six days after inoculation (21, 25, 25).

Epidemiology

Fungal development in the field is favored by high to moderate air and soil temperature and moisture (42, 44), and plants with high nitrogen and low calcium content (11, 23). Isolate pathogenicity (21, 23, 25), growth in soil and ability to colonize organic matter, resistance to antagonistic microorganisms, inoculum potential, and dissemination also are important factors during epidemic development on a susceptible crop (2, 36). Sclerotia generally provide the primary inoculum which is disseminated locally by wind, rain, running water, and movement throughout a field by animals, man or agricultural implements (42). Sclerotia can remain viable in soil for one or more years (24), and the fungus also can survive as vegetative mycelium within plant residue (42).

Symptomatology

Sclerotia germinate during periods of favorable environmental conditions by producing hyphae (a few mm in length) which branch profusely until reaching young or old host tissue where an infection cushion develops and penetration occurs directly or through stomata (10, 41, 42). Subepidermal hyphae develop inter- and intracellularly and the infection appears as small, circular, reddish-brown, necrotic, water-soaked lesions which may measure 1-3 cm in diameter and are delimited by longitudinal leaf veins and veinlets.

These lesions appear to have been scalded by hot water and may appear gray-greenish to dark brown (Fig. 1). The watersoaked area may affect the entire leaf (Fig. 2) and extend to adjacent plant tissue contiguous to the infected tissue. The light brown superficial hyphae spread fan-shaped and develop on either leaf surface, but they are more prevalent on the surface which is exposed to higher moisture. The perfect stage may form on the lower leaf surface at the margin between healthy and infected tissue, at the

Fig. 1- Initial leaf infections by basidiospores and mycelia of the web blight fungus.

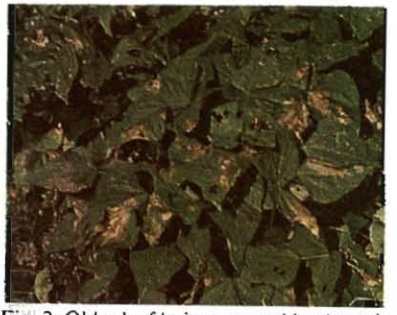

Fig. 2- Older leaf lesions caused by the web blight fungus.

Fig. 3- (above) Pod infection by the web blight fungus.
Fig. 4 - (upper right) Plant severely infected by the web blight fungus during a natural epidemic.
Fig. 5- (right) Microsclerotia produced on infected leaf tissue.

base of herbaceous plants or beneath soil aggregates (43). Basidia then form and basidiospores are dispersed during the night (12) until the leaf is disintegrated by the fungus (42). Hyphae may grow rapidly over healthy tissue of leaves, petioles, flowers and pods (Fig. 3), eventually killing plant parts or covering the entire plant with a web of mycelium (Fig. 4) and small brown sclerotia (Fig. 5) which form three to six days after infection (42, 44).

Bean pods may become infected at any stage of development, and young pod infections appear as light brown, irregular-shaped lesions which frequently coalesce and kill the pod. Lesions on older pods are dark brown, circular, lightly zonate, and sunken with a dark margin or border. Usually they do not kill the pod unless the peduncle is destroyed or the lesion is very deep (42, 44). Seeds can become infected in the endosperm and radicular end of the embryo or infested by mycelium and sclerotia on the seed coat surface (1, 3, 26, 27).

Control by Cultural Practices

Control by cultural practices includes planting seed free of internal or external contamination, sanitation of infected crop debris, and crop

rotation with non-hosts such as tobacco, maize and grasses. Planting dates should be early enough in the tropics to assure that the crop will mature before the rainy season begins. Beans should not be planted by broadcasting, but rather in spaced furrows (42, 44) to maximize air circulation and microclimatic conditions adverse to fungal development.

Control by Chemicals

Maneb (0.55 g/l) has been sprayed onto foliage twice at intervals of 15 days after symptom appearance to provide some control of web blight. Disease control also has been achieved by Benomyl (0.5 kg/ha), NF-44 (0.5 kg/ha), Derosal 60 or Carbendazim (1 kg/ha), Brestan 60 or Fentinacetate (0.8 kg/ha), and Difolatan or Captafol (3.4 kg/ha) (4, 29). The use of systemic fungicides is important where rains prevail. Beans have yielded one ton/ha when sprayed with systemic fungicides 15, 27, 39 and 51 days after germination, compared to the unsprayed check which was completely destroyed (4).

Control by Plant Resistance

Cultivars differ in their response to infection by the web blight fungus, since susceptible cultivars exude chemicals which stimulate the formation of infection cushions. Resistant or tolerant cultivars apparently do not exude these chemicals (17). Various cultivars which are tolerant to infection by *T. cucumeris* (4, 25, 29, 42) have been identified but there are no reports of cultivars which possess a high degree of resistance or immunity.

CIAT (5) has utilized the following scale to evaluate beans when leaves are inoculated with the web blight fungus under controlled conditions:

- no symptoms of infection
- little growth of pathogen, chlorosis around the inoculation point
- vein necrosis and 33% leaf chlorosis
- vein necrosis, 50% leaf chlorosis
- complete leaf necrosis.

Integrated control measures probably will be necessary to achieve satisfactory control and should consist of plant resistance or tolerance, upright plant architecture and open canopy, wide plant spacing, crop rotation and the judicious application of chemicals.

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Chapter 7

White Mold

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Chapter 7

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Introduction

Sclerotinia sclerotiorum (Lib.) de Bary, similar to *Whetzelinia sclerotiorum* (Lib) Korf and Dumont (37), is distributed worldwide. Although most important in the temperate zones of the northern hemisphere, it also can be a problem in areas with tropical or arid climates, especially during cool seasons or under favorable microclimatic conditions (59). The fungus has been reported in dry bean and vegetable fields in Argentina (32), Brazil (20, 65), Mexico (24), Peru (17), Colombia and other areas in Latin America (27).

Sclerotinia sclerotiorum is pathogenic to a wide range of host plants. Adams *et al.* (5) found 190 species from 130 genera and 45 plant families susceptible to the fungus. Schwartz (60) listed 399 hosts (unconfirmed reports in some instances) and 374 species of 237 genera in 65 plant families mentioned in the world literature. Diseases include blossom rot of fruit trees and flowers, storage rot of vegetables, and white mold of beans.

Beans can be damaged severely by the fungus. Snap bean production has been reduced greatly in New York during growing seasons conducive to fungal development (1, 51). Zaumeyer and Thomas (81) report bean losses of 30% in Virginia during 1916. Yield losses averaged 30% in Nebraska during 1970-1973, while losses in individual fields were as high as 92% (36).

Common names frequently used for white mold in Latin America include moho blanco del tallo, Sclerotinia, esclerotiniosis, salivazo, podredumbre algodonosa, mofo branco and murcha de Sclerotinia.

Etiology

Sclerotinia sclerotiorum is a member of the order Pezizales in the Ascomycete class of fungi. The fungus produces large (one to several mm

Fig. 1 - Sclerotial forms produced by the white mold fungus; left, culture produced; center, unconditioned and naturally produced; right, conditioned and naturally produced.

diameter), black and irregularly-shaped resting structures called sclerotia (Fig. 1) which germinate to form hyphae or mycelium. A sclerotium, after undergoing a conditioning period, also can germinate carpogenically to produce the sexual stage of one or more apothecia (Fig. 2). These may average 3 mm in diameter and protrude 3-6 mm above the soil surface (58).

Each apothecium contains thousands of cylindrically-shaped asci, each of which contains eight ascospores (78). The ascus measures 7-10 μ in diameter by 112-156 μ in length (18, 38, 58). Over a period of days, an apothecium may discharge more than 2 x 10^6 ascospores (62). The ascospores are ovoid and vary in width from 4-10 μ and in length from 9-16 μ (18, 38, 58, 78). *S. sclerotiorum* can produce microconidia (3-4 μ diameter) during any stage of its life cycle, but these have not been observed to function during sexual fertilization or host infection (38, 58).

Epidemiology

Fields used repeatedly for bean production, even in short crop rotations, often will contain many sclerotia. Sclerotia formed on or within diseased tissue may be dislodged onto the soil surface by wind or harvesting

Fig. 2 - Apothecia produced in field from germinated sclerotium.

operations. Subsequent land preparation redistributes them within the soil profile and over the field (19). Sclerotia also can be distributed by furrow irrigation within fields (62) and by reuse of irrigation runoff water between fields (14, 73). They can survive in sandy loam soils for at least three years (19) and are capable of producing secondary sclerotia (4, 19, 79).

The minimum quantity of soil-borne sclerotia needed to induce significant plant infection has not been intensively studied. Sclerotial populations of 0.2/30 cm^2 (1), 1-3/kg of soil (62) and 3/kg of soil (42) are known to exist in fields planted to snap, Great Northern and Pinto beans, respectively. Schwartz and Steadman (62) determined that 1 sclerotium/5 kg soil was sufficient to cause 46% plant infection in Nebraska. Suzui and Kobayashi (75) reported that 3.2 sclerotia/m^2 caused 60-95% plant infection in a kidney bean field in Japan.

Apothecia formation (carpogenic germination) is greatest at 15°-18°C with soil moisture at 50% of field capacity (Duniway, Abawi and Steadman, unpublished data). Carpogenic germination occurs in fields of dry beans, corn and sugar beet (61, 62), snap bean (1), cauliflower and tomato (40), lettuce (33,52) and table beet as well as in grassland (75). It also occurs in lemon, orange (66) and other fruit orchards (1). In a sandy loam soil studied by Schwartz and Steadman (62), numerous sclerotia germinated and formed apothecia in dry bean (11-14 apothecia/m^2) and sugar beet (7-11 apothecia/m^2) fields. An average of two apothecia were produced by each germinated sclerotium regardless of the crop beneath which it germinated. The majority of apothecia were produced on the side of, or adjacent to, plant stems in the irrigated row.

While most ascospores discharged by a germinating sclerotium are deposited close to the release point (74), crop infection has been reported in fields as far as 0.8 kilometer away (9, 15). The fungus clearly survives periods of unfavorable microclimatic conditions. Ascospores on bean leaves remained viable for 12 days in the field and mycelium in dried, colonized bean blossoms remained viable for 25 days in the laboratory (1).

Sclerotinia sclerotiorum is a cosmopolitan fungus and can be expected to occur in regions where temperature and moisture conditions are favorable (59). Brooks (13) and Moore (50) report that white mold epidemics are favored by mean temperatures less than 21°C and high humidity or moisture levels. Secondary spread of the fungus is favored by 18°C and 100% relative humidity (67, 77). Abawi and Grogan (1) feel that a surface moisture film is necessary for the fungus to develop and spread on plant tissue.

The rate of spread also can be influenced by temperature (Weiss, Kerr and Steadman, unpublished data). Gupta (30) reported that Coriander plants infected with *S. sclerotiorum* were killed in four to 10 days at 19°-

24°C but were not killed at 29°C, apparently because the plants outgrew the fungus. Microclimatic conditions may be as important as macroclimatic conditions for infection and pathogen development. Hipps (34) showed that irrigation practices significantly altered microclimatic parameters present within the dry bean canopy and conducive to development of *S. sclerotiorum*. Frequent furrow irrigation reduced daytime air and leaf temperatures 3° - 4°C and soil temperatures 10°C, and increased soil moisture content by 10%.

Plant Infection and Symptomatology

S. sclerotiorum infects bean plants by colonization of senescent plant organs such as blossoms (Fig. 3), cotyledons, seeds, leaves or injured plant tissue (1, 2, 19, 47, 51, 56). Blodgett (12) observed cotyledonary rot on bean seedlings which developed from mycelia- or sclerotia-infested seed lots planted in the greenhouse. However, Steadman (68) showed that infected seeds were completely colonized by the fungus prior to germination and/or plant emergence, and that no plant infection was observed in healthy-appearing seed from infested seed lots. Colonization of senescent tissue usually is due to germinated ascospores, but mycelial colonization also has been observed (1, 19).

After colonization of a senescent plant organ, the fungus enters the host by mechanical disruption of the cuticle with a dome-shaped infection cushion developing from an appressorium. Large vesicles form between the cuticle and epidermal layers, and infection hyphae develop intercellularly.

Fig. 3 - Bean blossoms colonized by ascospores of *Sclerotinia sclerotiorum*.

Hyphae branch from the infection hyphae and ramify inter-and intracellularly (44, 55), causing a watery soft rot. The fungus produces many enzymes and other products, including endo- and exopolygalacturonase, pectin methyl esterase (43) and oxalic acid (45) which are important to pathogenesis.

Symptoms and signs of infection appear initially as a water-soaked lesion (Fig. 4) followed by a white moldy growth on the affected organ (Fig. 5). Sclerotia form in and on infected tissue soon after infection. This infected tissue later becomes dry, light-colored and assumes a chalky or bleached appearance (Fig. 6) (12, 81). Plant wilting also may be evident

Fig. 4- Watery soft rot and sclerotia production in bean pod infected by white mold fungus.

Fig. 5 - Mycelia and sclerotia production on infected bean pod.

Fig. 6 - White or bleached symptom of bean plant severely infected by the white mold fungus.

Fig. 7- Canopy wilt caused by white mold infection of bean vegetation.

within the plant canopy after infection of the plant stem and/or vines occurs (Fig. 7).

Control by Biological Organisms

Many soil microorganisms are associated with sclerotia of *S. sclerotiorum* and may cause sclerotia to degrade or not germinate. Such organisms include *Coniothyrium minitans, Trichoderma* sp., *Aspergillus* sp., *Penicillium* sp., *Fusarium* sp., and *Mucor* sp. (35, 49, 57, 76). *S. sclerotiorum* also is inhibited by various antibiotic substances produced by *Gibberella baccata* (29), *Streptomyces* sp. (39, 41) and other actinomycetes and bacteria (25). None of these biological agents has been used effectively in reducing *S. sclerotiorum* incidence under practical field conditions.

Control by Cultural Practices

Zaumeyer and Thomas (81) recommended cultural practices such as crop rotation, flooding, reduced seeding rates, fewer irrigations and destruction of bean cull screenings which contain sclerotia as methods of controlling the pathogen. Similar recommendations have been made for control in Brazil (20). Deep plowing also has been advocated (49) and disputed (13, 28, 54) as a control measure. Crop rotation is not likely to be effective since sclerotia survive in soil, and tillage operations will assure the presence of sclerotia at or near the soil surface (19).

Irrigation frequency can influence disease incidence on cultivars with indeterminate plant growth habits and dense plant canopies (11). Growers are advised not to irrigate if white mold infection is prevalent within their bean fields (70). Re-use of irrigation water should be eliminated, or the water treated to remove sclerotial and/or ascosporic contamination which can contribute to current or future disease epidemics (73).

A survey of bean fields in Canada revealed that infected and non-infected crops were grown on soils with a pH of 7.5 and 7.0, respectively. However, the authors did not determine the nature or applicability of this association (31). Heavy fertilizer rates are not recommended, since they are associated with increased disease incidence (7), presumably because of the stimulatory effect upon plant canopy density.

Control by Chemicals

Application of Benomyl, DCNA or Dicloran, Dichlone, PCNB or Thiabendazole around early- to mid-bloom controls *S. sclerotiorum* infection on snap beans under dryland conditions (10, 16, 20, 28, 42, 48, 51). However, Partyka and Mai (53) report that repeated soil fumigation with a dichloropropene-containing compound actually increased the incidence of white mold in lettuce. Satisfactory chemical control in western Nebraska has not been obtained on indeterminate dry bean cultivars grown under irrigation (69). Sporadic results also have occurred in California, Colorado, Montana, Washington and Wyoming. Timing of the chemical application and thoroughness of coverage are critical to successful control.

Control by Plant Resistance and Architecture

An association between plant canopy development and white mold disease incidence and severity has been observed in various crops including beans. Row spacing, growth habit and plant density can influence bean canopy development and disease incidence (12, 21, 22, 23, 31, 51, 64, 71, 81). An open plant canopy which will facilitate air circulation and light penetration within the canopy helps prevent infection. It results in more rapid drying of moisture-covered leaf and soil surfaces (21).

As an example of row spacing-cultivar interaction, the cultivar Aurora escapes infection when planted at a within-row spacing of 4-5 cm because of its upright, open growth habit (22). However, when planted 30.5 cm apart within the row, it sprawls and is more severely infected. Orientation of bean rows parallel to the prevailing wind direction also may reduce disease incidence by providing improved air circulation and better light penetration (31).

Resistance to *S. sclerotiorum* has been observed in *Phaseolus vulgaris* germplasm (12, 26, 46, 58, 80), but comparative differences between cultivars were not reported until recently (8). Resistant cultivars include Black Turtle Soup, Charlevoix and Valentine (8, 63).

Resistance also has been identified in *P. coccineus* (6, 72) and *P. coccineus* x *P. vulgaris* hybrids (3). Attempts are being made to develop stable resistance by using a plant structure which maximizes disease avoidance and also possesses physiological resistance to infection by *S. sclerotiorum* (22). Such cultivars would be conducive to an integrated control program which could include fungicides and cultural practices if a greater degree of plant protection still is required.

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Chapter 8

Miscellaneous Fungal Pathogens

H. F. Schwartz

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Introduction

Dry beans are exposed to many pathogenic fungi at various stages of their plant development, and infection may occur on seedlings and mature plants throughout the growing season or post-harvest. Some of the more prevalent and economically important plant pathogenic fungi have been described previously in this book. Unfortunately, very little information exists concerning the epidemiology and control of many other fungi generally considered to be of minor importance to bean production. However, in the tropics many of these minor pathogens can become very important in specific regions of bean production. Likewise, minor pathogens may become major pathogens in the future as agricultural practices are modified. This chapter will describe briefly some of these fungi and list others reported to be pathogens of beans.

Alternaria Leaf and Pod Spot

Alternaria leaf and pod spot is caused by various *Alternaria* species including *A. alternata* (Fr.) Keissler, *A. brassicae* f. *phaseoli* Brun., *A. fasciculata* (Cke. and Ell.) L. R. Jones and Grout, and *A. tenuis* Nees (1, 15, 26, 28, 41, 46). These fungi are reported to occur in Brazil (31), Costa Rica (17), Colombia (13), Chile, Mexico, Venezuela (43), England (26), and the United States (1, 27, 28, 46). Severe epidemics may cause premature defoliation but yield losses usually are not significant. However, snap bean losses of 12% occurred in New York since infected pods were unacceptable for processing (1).

Common names frequently used for Alternaria leaf and pod spot in Latin America are mancha parda and mancha foliar por Alternaria.

Alternaria brassicae produces greenish-brown, septate and branched hyphae with erect conidiophores in culture. Conidia are smooth, long-

Fig. 1- Leaf lesions caused by Alternaria infection.

beaked, obclavate shaped with many transverse and longitudinal septations. Conidia are borne singly or in chains of two to three spores and measure 50-350 x 9-33 μ (41).

Alternaria spp. are considered to be wound parasites and usually form lesions only on older plant tissue during periods of high humidity for three or four days (1, 28), and at relatively cool temperatures such as 16°- 20°C (28). Saad and Hagedorn (27) reported that *A. tenuis* also could penetrate the leaf directly or through stomata. *A. tenuis* produces a toxin (tentoxin) in culture which induces plant chlorosis when applied to roots (11, 29). However, the fungus does not produce detectable quantities of tentoxin during natural infection of leaves or pods.

Leaf symptoms appear as small reddish-brown, irregular shaped spots or flecks which may be watersoaked and surrounded by a darker brown border. These lesions gradually enlarge and develop as concentric rings, which may become brittle and fall out, leaving a shot-hole appearance (Fig. 1). Lesions may coalesce and cover large areas of the leaf, resulting in partial or premature defoliation. *Alternaria* spp. can cause death of the central growing point of the plant or reduce plant vigor. The fungus also can blemish leaves (Fig. 2) and pods (Fig. 3) by producing a brown

Fig. 2- (left) Blemish on bean leaf caused by *Alternaria* species.

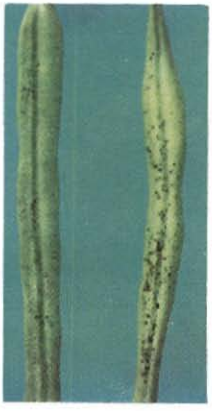

Fig. 3- (right) Blemish on bean pods caused by *Alternaria tenuis*.

discoloration on the surface and damage developing seeds (1, 17, 26, 28, 41, 46). The reddish to dark brown or black flecks may coalesce and produce streaks on infected pods (1). *Alternaria* spp. can be seed-borne (13).

Control measures seldom are warranted but wider plant and row spacing, chemicals, development of resistant cultivars (1) and crop rotation are suggested. Chemical control includes Chlorothalonil (1200 μg a.i./l) (1), Thiophanate (2 g/l) and Zineb (2.4 g/l). Workers report that *A. alternata* is insensitive to spray applications of Benomyl (1, 26).

Ascochyta Leaf and Pod Spot

Ascochyta leaf spot of beans is caused by *Ascochyta boltshauseri* Sacc. and *A. phaseolorum* Saccardo (41, 46). The fungus occurs in many regions of Latin America, such as Brazil, Colombia, Costa Rica and Guatemala (7, 12, 22, 31); the United States and other regions of the world (46). *Ascochyta pisi* Lib. occurs in Venezuela (43). The common name frequently used for Ascochyta leaf spot in Latin America is mancha de Ascochyta.

Ascochyta spp. produce hyaline, septate submerged mycelium in culture, and spores usually are two-celled and 20 x 5 μ in size (46). Sporulation and germination is optimum at 21°C, while mycelial growth is optimum at 24°C. The fungus is inactivated by temperatures above 30°C (22). The fungus produces pycnidia which measure 60-150 μ in diameter (46).

Infection by *Ascochyta* spp. is favored by high humidity and cool to moderate temperatures (12). Symptoms initially appear on leaves as brown to black zonate lesions (Fig. 4) which may later contain small black pycnidia. Lesions also may appear on the peduncle, petiole (Fig. 5) and pod

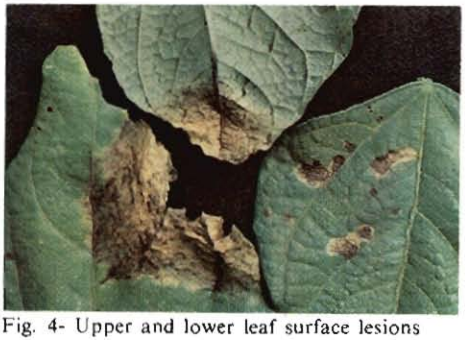

Fig. 4- Upper and lower leaf surface lesions caused by *Ascochyta* species.

Fig. 5- Petiole and pod lesions caused by *Ascochyta* species.

(Fig. 6) and cause stem girdle and plant death. Premature leaf drop may occur during severe epidemics (41), and the fungus may be seed-borne.

Control measures include crop rotation, wide plant spacing, planting clean seed, chemical treatment of seed and foliar application of sulfur fungicides (33). Other chemical control measures include Benomyl (0.55 g/l), Zineb (2.4 g/l) and Chlorothalonil (2.24 kg/ha). Dry bean germplasm should be screened to identify sources of resistance, if available and practical as a control measure.

Ashy Stem Blight

Ashy stem blight of bean is caused by *Macrophomina phaseoli* (Maubl.) Ashby or *M. phaseolina* (Tassi) Goidanich (9, 41, 46). The fungus is a warm-temperature pathogen of beans *(Phaseolus vulgaris* and *P. lunatus),* soybeans, corn, sorghum and many other crops (40). It occurs in such regions of Latin America as Brazil (7, 10, 31, 36), Mexico, Peru, Colombia, Venezuela and in Central America (43), and in other parts of the world (46). Losses of 65% have occurred in beans grown in the United States (46). However, no loss estimates are available for Latin America.

Common names frequently used for ashy stem blight in Latin America include pudrición gris de la raiz, pudrición carbonoza de la raíz, tizón cenizo del tallo, podredumbre carbonosa and podridao cinzenta do caule.

The fungus produces one-celled fusiform conidia which are pointed at one end and rounded at the other end. The straight or slightly curved conidia are 15-30 μ long and 5-8 μ wide and are produced on nearly straight conidiophores which may have a truncate tip and measure 12-20 μ in width by 6-25 μ in length (46). Sclerotia and pycnidia also are produced on infected plants.

Symptoms may appear after soil-borne mycelia or sclerotia germinate and infect seedling stems near the soil line at the base of developing cotyledons (Fig. 7). The fungus produces black, sunken cankers which have a sharp margin and often contain concentric rings. The plant-growing tip may be killed or stem breakage can occur where the stem is weakened by the canker. Infection may continue to develop into the hypocotyl and root region or the primary leaf petioles. Older seedling and plant infections may cause stunting, leaf chlorosis, premature defoliation and plant death. The infection often is more pronounced on one side of the plant (Fig. 8) (7, 9, 36, 41, 46).

A few days after infection, the fungus produces small, smooth, black sclerotia (50-150 μ in diameter) in infected tissue (Fig. 9) and inside plant

stems. Small, submerged, black pycnidia also may form in this tissue and usually are present on a gray background which has a characteristic ashen appearance (Fig. 10). The fungus may produce air-borne conidia which cause leaf spots on mature plants (10). *Macrophomina phaseolina* can be seed-borne (13, 41, 46).

Fig. 6- Older pod lesion caused by *Ascochyta* species.

Fig. 7 - Seedling infection due to *Macrophomina phaseolina.*

Fig. 8- Initial infection by ashy stem blight fungus on one side of plant.

Fig. 9 - (above, left) Sclerotia of *Macrophomina phaseolina* on infected bean stem.

Fig. 10 - (above, right) Pycnidia of *Macrophomina phaseolina* on infected bean stem.

Control measures include planting clean seed, treating seed with chemicals such as Ceresan, and using sanitation or deep plowing to bury plant debris containing pycnidia and sclerotia. Organic soil amendments (Carbon/Nitrogen ratio of 10-20) and high soil temperature (30°C) and moisture (60% moisture holding capacity) may reduce sclerotia levels (9). Sclerotia survival in soil can be reduced further by application of Benomyl (1 kg/ha) and Thiophanate-methyl (19), or by soil fumigation with Methyl Bromide and Chloropicrin (40). Resistant cultivars such as Negrito have been identified (9, 36, 46).

Cercospora Leaf Spot

Cercospora leaf spot and blotch of beans are caused by *Cercospora canescens* Ellis and Martin, and *C. cruenta* Saccardo, respectively. *C. phaseoli* Dearness and Bartholomew and *C. caracallae* (Speg.) Chupp also cause leaf spots of bean (15, 32, 41, 46). These fungi, primarily *C. canescens* and *C. cruenta,* occur in Brazil (31), Colombia (32), Puerto Rico, Trinidad, Jamaica, Venezuela, Argentina (43) and the United States (46). Yield losses are slight in the United States but can be serious in the Phillipines on *Phaseolus aureus* (46). There are no reports of serious losses in Latin America; however, defoliation has occurred in Colombia (23).

Common names frequently used for Cercospora leaf spot in Latin America include mancha de Cercospora, mancha vermelha and mancha blanca.

Cercospora spp. produce hyaline conidia with varying numbers of septations. Spores may be club, curved or straight-shaped. *C. cruenta* spores measure 50-150 μ in length by 6-9 μ in width, while *C. canescens* spores measure 50-100 μ in length by 3-4.5 μ in width (46).

Symptoms include brown or rust-colored lesions (Fig. 11) which may coalesce and vary in shape (circular to angular) and size (2-10 mm). *C.*

Fig. 11- *Cercospora* species lesions on infected bean leaves.

canescens produces irregularly-shaped light brown lesions with a gray center in leaves, pods, stems and branches (23). These lesions may contain a grayish center and be surrounded by a slightly reddish border. Lesions may dry and portions fall out, leaving a ragged appearance. Premature defoliation may occur, but vigorously growing leaves are seldom affected. *C. cruenta* may cause numerous lesions on primary leaves but seldom infect the trifoliates. Blemishes may occur on stems and pods, and the fungi can become seed-borne (23, 41, 46). A pink to purple discoloration occurred on bean seed inoculated with *Cercospora kikuchii* isolated from infected soybeans (21).

Control measures seldom are warranted but foliar applied copper fungicides are effective (46). Orozco (23) reported that Cundinamarca 116, Mexico 32, Mexico 275, Mexico 487, Mexico 507, Venezuela 42 and other cultivars were resistant to infection by *Cercospora canescens*.

Chaetoseptoria Leaf Spot

Chaetoseptoria leaf spot of beans is caused by *Chaetoseptoria wellmanii* Stevenson. It occurs in Mexico, Panama, Central America, Venezuela and the West Indies (43). The fungus has a wide host range within the Leguminoseae and may cause complete defoliation of beans with up to 50% yield reduction in regions with high humidity and moderate temperatures (42). The common name frequently used for Chaetoseptoria leaf spot in Latin America is mancha redonda.

Fig. 12- Leaf lesions caused by Chaetoseptoria leaf spot.

Chaetoseptoria wellmanii produces medium to large, circular lesions (Fig. 12) which may have a gray surface with black pycnidia in the center and be surrounded by a dark border (42). Infection is more common in

primary leaves in Mexico, and defoliation also may occur. The pathogen may be seed-borne (8).

Control measures include the development of resistant or tolerant cultivars (8). Benomyl (0.55 g/l) may provide sufficient chemical control.

Diaporthe Pod Blight

Diaporthe pod blight of beans is caused by *Diaporthe phaseolorum* (Cooke and Ellis) Saccardo (41). *D. arctii* (Lasch) Nits. is known to be pathogenic to bean stems (46). *D. phaseolorum* has a conidial stage known as *Phomopsis subcircinata* Ell. and Ev. (34). No estimates of its prevalence or importance are currently available, although Wellman (43) reports that it is a weak parasite in Honduras. Common names frequently used for Diaporthe pod blight in Latin America are añublo de vaina and tizón de la vaina.

Diaporthe phaseolorum produces hyaline, oblong ascospores with one septation and measure 10-12 μ by 2-4 μ. The ascospores are produced within black perithecia, 300 μ in diameter. Pycnidiospores are produced in the black pycnidia, and the oval spores measure 6-9 μ by 2-5 μ (41).

Symptoms appear first on leaves as irregularly-shaped, brown lesions surrounded by a distinct border. Black pycnidia and occasionally perithecia form in a zone or are scattered throughout lesions. Pod infections then may occur, and pods become discolored with pycnidia present in the lesions (41). The fungus can be seed-borne in soybeans and in beans (13).

Control measures include crop rotation, planting clean seed, and use of foliar fungicides such as Benomyl (0.55 g/l). Resistant cultivars have been developed for soybeans. Dry bean germplasm should be screened to identify sources of resistance, if available and practical as a control measure.

Downy Mildew

Downy mildew is caused by *Phytophthora parasitica* Dast. (46) and *P. phaseoli* Thaxter (8). The pathogen has caused yield losses in Mexico, Puerto Rico (8, 46), El Salvador, Venezuela, Peru and Panama (43). Infection is favored by low temperatures and high humidity. Common names frequently used for downy mildew in Latin America are mildeo velloso and mildiu velloso.

Fig. 13- Pod infection caused by *Phytophthora* species.

Fig. 14- Leaf lesions caused by *Entyloma* species

Symptoms first appear on the petioles as white spots which enlarge and eventually may cause the leaf to wilt and die. Blossoms, buds and other plant parts may be killed by the fungus. White patches of mycelium are visible on green pods, especially those in contact with the soil surface (Fig. 13). This patch usually is surrounded by a reddish-brown border. If low temperatures and high humidity persist, the entire pod may be infected, shrivel and dry up (8).

Control measures include crop rotation for three years; chemicals such as Zineb, Maneb, Nabam or Captan (8); production of pods free from soil contact (46); and development of cultivars with an upright plant architecture and open plant canopy to improve air circulation. Dry bean germplasm also should be screened to identify sources of resistance, if available and practical as a control measure.

Entyloma Leaf Smut

Entyloma leaf smut of beans is caused by a species of *Entyloma* (30, 35, 42). Entyloma leaf smut occurs in bean production regions of Costa Rica, Dominican Republic, El Salvador, Guatemala, Honduras and Nicaragua (30, 35). *Entyloma petuniae* Speg. occurs on beans in Argentina (43). The common name frequently used for smut in Latin America is carbón.

Entyloma spp. cause a blister smut which is evident as dark-colored swellings on the upper leaf surface. The swellings are filled with mycelia and teliospores of the fungus (42). Lesions are round or oval, first appear watersoaked but become gray-brown in color on the upper leaf surface and gray-blue on the lower leaf surface (Fig. 14). Lesions may coalesce and be delimited by leaf veinlets (30). Infection usually occurs only on the primary

leaves, or first and second sets of trifoliate leaves, and severe foliage infection of 40-60% may occur (35).

Chemical control may be achieved by applying a seed treatment of Carboxin (5 g/kg seed) or a foliar spray of Benomyl (0.55 g/l). Dry bean germplasm should be screened to identify sources of resistance, if available and practical as a control measure.

Floury Leaf Spot

Floury leaf spot of beans is caused by *Ramularia phaseoli* (Drummond) Deighton (41). The fungus occurs in Brazil (Minas Gerais and Espirito Santo), Nicaragua, Colombia and Venezuela (4, 5, 36, 38, 39), Ecuador, Honduras, Panama, Guatemala and the Dominican Republic (43). No estimates of yield losses caused by it are available.

Common names frequently used for floury leaf spot in Latin America include mancha harinosa, mancha farinhosa and mofo branco da folha.

Ramularia phaseoli produces hyaline, generally non-septate conidia which are oval to lemon-shaped and measure 7-18 x 4-6 μ (41).

Ramularia phaseoli produces a white growth (1-1.5 cm in diameter) of conidiophores and conidia on the lower surface of leaves (Fig. 15). It

Fig. 15- Lower leaf lesions caused by *Ramularia phaseoli*.

should not be confused with powdery mildew *(Erysiphe polygoni)* infections, which usually are present only on the upper leaf surface. Chlorosis may occur on the upper leaf surface corresponding to the lower leaf lesions. Infection generally appears first on older leaves and then progresses onto new foliage. Severe infections may cause premature defoliation (5, 41); however, this is not commonly observed, especially in Brazil.

Chemical control results by applying Benomyl (0.55 g/l) or Thiophanate (2 g/l). Dry bean germplasm should be screened to identify sources of resistance, if available and practical as a control measure.

Gray Mold

Gray mold of beans is caused by *Botrytis cinerea* Pers. *ex* Fries which has the perfect stage *Botryotinia fuckeliana* (de Bary) Whetz. (25). The fungus can be a serious problem during periods of high moisture and low temperatures in various regions of the United States and Europe (25, 46). It is a minor pathogen in Brazil and seldom causes any significant damage (7). It also is reported in Peru, Trinidad, El Salvador (43) and Colombia (13).

Common names frequently used for gray mold in Latin America are moho gris, podredumbre gris and bolor cinzento.

The fungus produces light brown mycelium and hyaline, oval conidia 12-20 x 8-12 μ in size (41). Apothecia (Fig. 16) and ascospores are formed by the perfect stage of the fungus, which provides for variability in virulence of different strains and mating types (25).

Infection usually occurs from senescent blossoms colonized by the fungus or at wounds on plant parts such as leaves, stems or pods (Fig. 17)

Fig. 16 - Apothecium and conidia produced by *Botryotinia fuckeliana*.

Fig. 17- (right) Blossom colonization and pod infection by gray mold.

and penetration occurs from an infection cushion (16). Symptoms appear as a water-soaked gray-greenish area on the affected tissue which subsequently wilts and dies. Seedlings also may become wilted and die, but damage usually is limited to a watery soft rot of pods (41, 46). Black stroma and sclerotia (up to 4 mm in diameter) may be produced in infected tissue (25), and resemble those formed by *Sclerotinia sclerotiorum*. The fungus can be seed-borne (13).

Control measures include reduced plant density, row width and irrigation frequency (20), and application of foliar fungicides. However, some strains of the fungus are resistant to fungicides (3, 25). Dry bean germplasm should be screened to identify sources of resistance, if available and practical as a control measure.

Gray Leaf Spot

Gray leaf spot of beans is caused by *Cercospora vanderysti* P. Henn. which occurs in Venezuela, Central America (43), Brazil (Minas Gerais and Espirito Santo) (31, 36, 37, 39) and Colombia, usually at elevations greater than 1000 m where high moisture and low to moderate temperature conditions persist (32). No estimates of yield losses are available. The common name frequently used for gray leaf spot in Latin America is mancha gris.

Symptoms appear on the upper leaf surface as light green to slightly chlorotic angular lesions (2-5 mm in diameter), usually delimited by the veins and veinlets (Fig. 18). Lesions may coalesce and later become covered by a fine powdery, grayish-white growth of mycelium and spores. A dense gray mat of mycelium and spores subsequently forms on the lower leaf surface (Fig. 19) and is very diagnostic for the pathogen (32, 36). Severe

Fig. 18- Upper leaf lesions caused by *Cercospora vanderysti*.

Fig. 19- Mycelium and spores produced on lower leaf surface by gray leaf spot.

Fig. 20- Severe plant infection by the gray leaf spot fungus.

infections (Fig. 20) may cause premature defoliation. Symptoms may resemble those of white leaf spot, especially during early stages of infection.

Chemical control consists of Benomyl (0.55 g/l) and Copper Hydroxide (2.24 kg/ha). Other control measures include resistant or tolerant cultivars such as Rico 23, B.H. 4935 and Porto-Alegre-Vagem-Roxa (36).

Phyllosticta Leaf Spot

Phyllosticta leaf spot is caused by *Phyllosticta phaseolina* Saccardo which is favored by high moisture and moderate temperature conditions (18, 31, 36). The fungus occurs in Brazil (15), Costa Rica, Nicaragua, El Salvador, Guatemala, Peru, Argentina, Puerto Rico (43) and the United States (18, 46). No reports are available concerning yield losses. Common names frequently used for Phyllosticta leaf spot in Latin America include mancha de Phyllosticta and queima da folhagem.

Phyllosticta phaseolina produces hyaline, one-celled pycnidiospores which are 4-6 x 2-3 μ in diameter. Pycnidia are 90 μ in diameter (42).

Symptoms generally appear only on mature leaves as small water-soaked spots which may coalesce and enlarge to 7-10 mm in diameter. Lesions have a light-colored necrotic center and are surrounded by a rusty-brown margin. The center of old lesions may fall out and leave a shot-hole appearance. Small, black pycnidia may develop throughout the lesion and along the margin. Lesions may occur on petioles and stems and turn flower buds brown. Small lesions (1 mm in diameter) with dark centers and reddish margins may develop on pods (18, 46).

Control measures consist of foliar fungicides (46). Dry bean germplasm should be screened to identify sources of resistance, if available and practical as a control measure.

Powdery Mildew

Powdery mildew of beans is caused by *Erysiphe polygoni* DC *ex* Merat. and is distributed worldwide. Infection is favored by moderate temperatures and humidity. However, it can be prevalent within a wide range of environmental conditions (46). The pathogen seldom causes extensive damage in Latin American countries such as Brazil and Costa Rica (12, 31, 36) but can seriously reduce yields in Peru (12).

Common names frequently used for powdery mildew in Latin America include oidium, oidio, mildeo polvoso, cinza, ceniza and mildio pulverulento.

The fungus produces hyaline conidia in chains on the leaf surface. The spores are ellipsoid, one-celled and measure 26-52 x 15-23 μ in size. Spherical black perithecia (120 μ in diameter), uncommon in the tropics, contain asci and ascospores which are 24-28 x 11-13 μ (41).

Symptoms first appear as slightly darkened mottled spots on the upper leaf surface, which subsequently become covered by circular growths of white, powdery mycelium (Fig. 21). The entire leaf and plant may become covered by mycelium (Fig. 22), malformed, yellow and senesce prematurely. Stems and pods can be infected (Fig. 23), resulting in yield loss and seed transmission. Pods may be stunted, malformed or killed during severe epidemics. The fungus can be seed-borne (46), probably as spores on the seed coat surface.

Control measures include planting clean seed and using foliar chemicals such as sulfur, Dinocap (1.2 g/l) or lime-sulfur (10 ml/l). Concepcion (6) did not observe significant yield increases with chemicals such as Benomyl. Resistant cultivars exist, but resistance is complicated by the existence of different physiologic races (45, 46). Sources of non race-specific resistance should be sought and utilized if practical.

White Leaf Spot

White leaf spot of beans is caused by *Pseudocercosporella albida* (Matta & Belliard) comb. nov. and recently has been observed in Guatemala (47) and in Colombian (H.F. Schwartz, personal observation) highland sites at elevations greater than 1500 m. No estimates of yield losses are available. The common name frequently used for white leaf spot in Latin America is mancha blanca.

Symptoms appear first on the lower leaf surface of older leaves as white angular spots (2-5 mm in diameter) restricted by the leaf veins.

Fig. 21 - Powdery mildew lesions on bean leaf.

Fig. 23 - Pod infection by *Erysiphe polygoni*.

Fig. 22 - Severe plant infection by *Ervsyphe polygoni*.

Fig. 24 - (above) Leaf lesions caused by the white spot fungus.

Fig. 25 - (left) Mixed leaf infection by gray and white spot fungi.

Angular white spots (Fig. 24) also may occur on the upper leaf surface and eventually enlarge and coalesce. Leaf necrosis and defoliation may occur (47). Symptoms closely resemble those of gray leaf spot, especially during the early stages of infection. Mixed infection by white and gray leaf spot has occurred in Colombia (Fig. 25).

Yoshii and Aamodt (47) report that the following cultivars were resistant to infection in Guatemala: Mexico 114, Puebla 40-4, Puebla 41-1, Puebla 138, Puebla 151-B, Puebla 199, Aguascalientes-79, Michoacan 31, Arrox I-565 and R20 Antioquia 18. No other disease control methods have been investigated.

Yeast Spot

Yeast spot or seed pitting of beans is caused by *Nematospora coryli* Pegl. and can be a seed production problem in Brazil (7, 36), Costa Rica, Ecuador, Peru, the West Indies (43) and the United States (46). Its economic importance varies from 10-100% yield loss due to its effect on seed quality and commercial appeal, which may be greatly reduced, especially in lima bean production (46). Common names frequently used for yeast spot in Latin America are mancha de levadura and pústula bacteriana.

Insects, such as the southern green stinkbug *(Nezara viridula* (L.), and lygus bugs *(Lygus hesperus* Kngt. and *L. elisus* Van Duzee), transmit the causal organism and also may damage seeds directly from toxins secreted during the feeding process (46). Galli *et al.* (15) reported in 1963 that *Nematospora coryli* also persists in weeds such as *Cassia occidentalis, Momordica charantia, Bauhinea purpurea* and *Crotalaria* sp.

The yeast organism produces a variable morphology in culture as elliptical cells 6-10 μ wide by 8-14 μ long predominate initially, followed by mature spherical cells 20 μ in diameter and mycelium-like strands which measure 2.5-3.5 μ in width by 90-140 μ in length. *Nematospora coryli* grows in culture at temperatures between 15°- 40°C, but 25°- 30°C is more favorable for infection (46).

Symptoms appear after insects feed on pods, puncture the developing seeds and transfer fungal propagules to the wound sites. The spores germinate and infect the seeds, including the embryonic cotyledonary leaves, thereby producing irregular, slightly sunken lesions about 1 mm in diameter. The lesions may be rose, tan or brown (7, 36, 41).

Control measures consist of eliminating weed hosts and controlling insect populations (46).

Additional Pathogens

Other fungi are reported to be pathogens of beans (*Phaseolus* species) and are not discussed in this book. Some of these organisms are listed in Table 1.

Table 1. Additional fungal pathogens of beans.

Pathogen	Plant Symptoms	Lit. Cited
Acrostalagmus spp.	-	13
Aristostoma oeconomicum Sacc.	Leaf Spot	46
Asteroma phaseoli Brun.	Leaf, Pod Spot	46
Botryodiplodia theobromae	Seed Decay	13
Brachysporium pisi Oud. (perhaps a *Curvularia* sp.)	Leaf Spot	34
Cephalosporium gregatum Allington and Chamberlain	Stem Rot	46
Ceratophorum setosum Kirchn.	-	46
Chaetomium indicum Cda.	-	46
Chephalosporium gregatum All. & Chamb.	Stem Rot	42
Cladosporium album Dows.	-	46
Cladosporium herbarum Pers. *ex* Fr.	Pod, Seed, Leaf Spot	34
Colletotrichum truncatum (Schw.) Andrus and Moore	Pod, Stem Spot	41
Corticum salmonicolor Berk. & Br.	Plant Rot	42
Curvularia spp.	Leaf Spot, secondary	42
Dendrophoma spp.	-	2
Dimerium grammodes (Kze.) Garman (*Parodiella perisporioides* (Berk. & Curt.) Speg.)	Leaf Spot, secondary	42
Diplodia natalensis P. Evans	Seed contaminant	46
Diplodia phaseolina Sacc.	Pod Spot	46
Elsinoe dolichi Jenkins, Bitanc, and Cheo	Leaf Spot (Scab)	41

Elsinoe phaseoli Jenkins	Leaf Spot (Scab)	41
Epicoccum neglectum Desm.	Leaf Spot	46
Fusarium culmorum (W. G. Sm.) Sacc.	Stem Rot	42
Fusarium equiseti (Cda.) Sacc.	Damping off	42
Fusarium lateritium Nees	Stem Canker	42
Fusarium macroceras Wr. and Reinking	Pods	46
Fusarium roseum Lk.	-	46
Fusarium semitectum Berk. & Rav.	Pod Decay	42
Fusarium vasinfectum Atk.	-	46
Gleosporium corallinum (Peyl.) Sacc. and Trav.	-	46
Glomerella cingulata (Ston.) Spauld and Schrenk.	-	46
Helminthosporium victoriae Meehan and Murphy	Pod Spot	46
Heterosporium spp.	Sooty Leaf Spot	46
Hypochnus centrifugus (Lev.) Tul.	-	46
Hypochnus cucumeris Frank.	Damping off	46
Leptosphaeria phaseolorum Ell. and Ev.	Stem Disease	46
Macrosporium commune Rab.	-	46
Macrosporium consortiale Theum.	-	46
(Stemphylium consortiale Theum.)	-	46
Macrosporium leguminis phaseoli P. Henn.	-	46
Macrosporium phaseoli Faut.	-	46
Microsphaera diffusa Cke. and Pk.	Leaf Spot	34
Microsphaera euphorbiae (Pk.) Berk. and Curt.	Leaf Spot	46
Monilia spp.	-	13

(continued)

Pathogen	Plant Symptoms	Lit. Cited
Mycena citricolor (Berk. & Curt.) Sacc.	Leaf Spot	42
Mycorrhizal fungi	Root Parasitism	46
Mycosphaerella phaseolicola (Desm.) Ideta.	Leaf Spot	46
Myrmaecium roridum Tode	Pod Disease	42
Nectrea spp.	-	42
Nigrospora spp.	Pod Decay	14
Periconia pycnospora Fr.	Pod Disease	42
Pestalotiopsis spp.	-	13
Peyronellaea spp.	-	13
Phakopsora vignae (Bres.) Arth. (*Phakopsora pachyrhizi* Sydow) (*Physopella concors* Arth.)	Leaf Rust (Soybean Rust)	46
Phoma terrestris Hans.	Secondary Root Rot	46
Phyllachora phaseoli (P. Henn.) Th. and Syd.	Leaf Spot (Tar Spot)	34
Phyllosticta noackiana All.	Leaf Spot	42
Phyllosticta phaseolorum Sacc. and Speg.	Leaf Spot (Ochraceous Spot)	46
Physarum cinereum (Batsch) Pers.		
Phytophthora cactorum (Leb. and Cohn) Schroet.	-	46
Phytophthora capsici Leon.	-	46
Pleiochaeta setosa (Kirchn.) Hughes	Leaf and Pod Spot (Brown Spot)	24
Pleospora herbarum (Ders. and Fr.) Rab. (*Stemphylium botryosum* Wallr.)	Leaf Spot	34

Pullularia pullulans (de By) Berkhout.	Seed Spot	34
Pythium anandrum Drechs.	-	34
Pythium arrhenomanes Drechs.	Root Rot	34
Pythium helicoides Drechs.	Root Rot	34
Pythium oligandrum Drechs.	Root Rot, Pod Rot	34
Pythium rostratum Butl.	Root Rot	34
Pythium vexans d By	-	34
Rhizoctonia dimorpha Matz.	Plant Rot	42
Rhizoctonia ferrugena Matz.	-	46
Rhizopus nigricans Ehrenberg	Pod Rot	41
Rhizopus stolonifer (Ehr. *ex* Fr.) Lind	Soft Rot	34
Rhizopus tritici K. Saito	Soft Rot	34
Sclerophoma phaseoli Karak	Pod Spot	46
Septoria phaseoli Maubl.	Leaf Spot	42
Sphaerotheca humuli var. *fuliginea* (Schlecht.) Salmon.	-	46
Stagonospora phaseoli Dearn.	Leaf Spot	34
Stagonospora hortensis Sacc. and Malbr.	Leaf Spot	34
Stemphylium botryosum Wallr.	Leaf Spot	42
Uromyces fabae (Pers.) D by	Rust	46
Vermicularia polytricha Cke.	-	46
Verticillium albo-atrum Reinke & Berth.	Root, Shoot Disease	42

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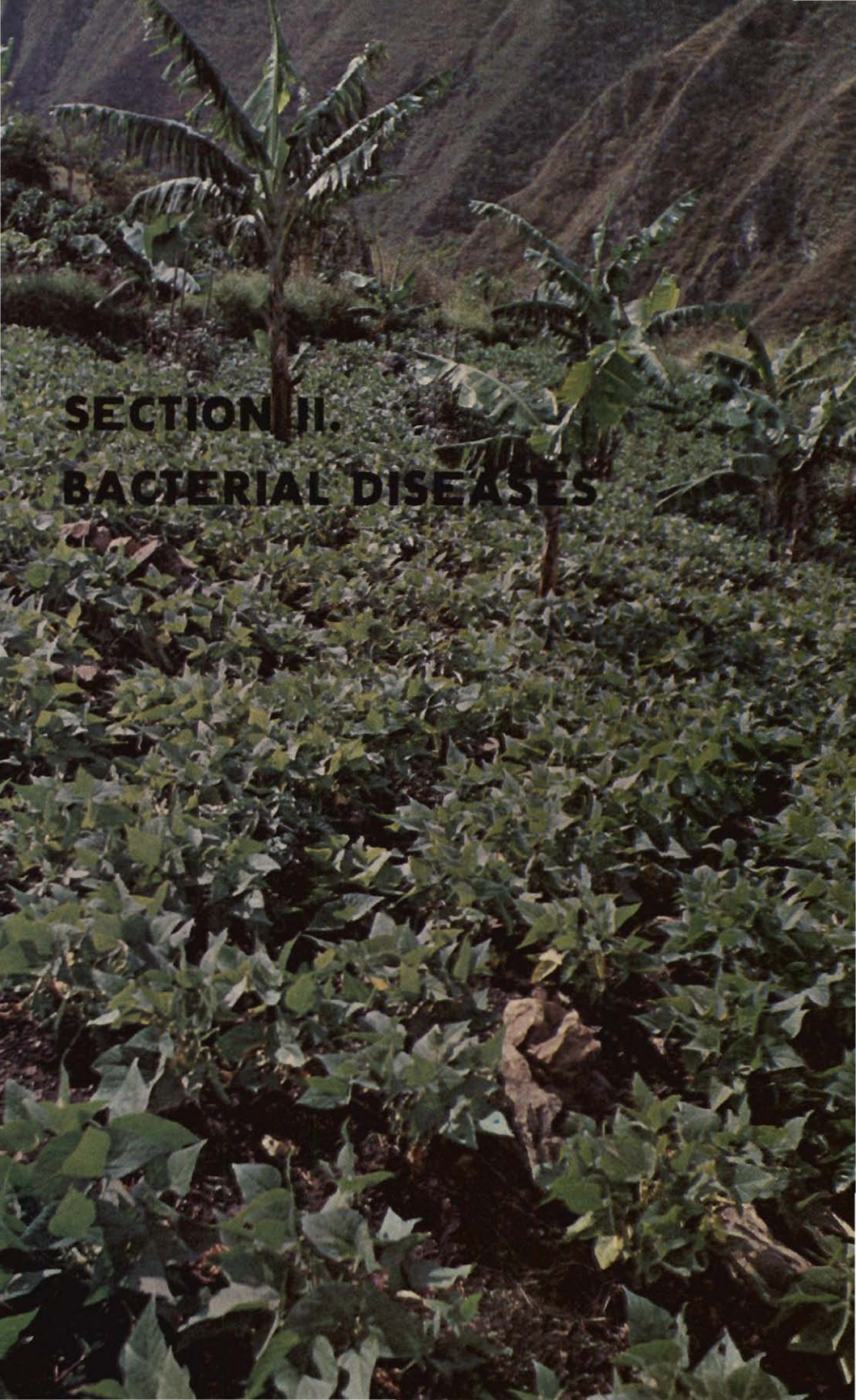

SECTION II.
BACTERIAL DISEASES

Chapter 9

Common and Fuscous Blights

Kazuhiro Yoshii

Chapter 9

Common and Fuscous Blights

Introduction

Common blight caused by *Xanthomonas phaseoli* (E.F.Sm.) Dows. and fuscous blight caused by *X. phaseoli* var. *fuscans* (Burk.) Starr and Burk. are major bacterial diseases of dry beans. The two organisms are found frequently in association and are reported to occur in many bean production regions of the world (13, 26, 27, 47, 51, 62, 81, 92).

Yield losses due to each pathogen are difficult to estimate because their symptoms are similar. Common and fuscous blight bacteria frequently occur together in a field and probably on the same plant, increasing the difficulty of associating yield losses with a specific pathogen. In 1967, at least 75% of Michigan's 650,000 acres of Navy beans were damaged by common and fuscous blights, with 10-20% yield reductions (2).

Wallen and Jackson (82) reported a 38% yield loss in Ontario, Canada due to common and fuscous blight in two years of field trials. Aerial infrared photographic surveys suggested that losses for the bean crop grown in Ontario ranged from 1252 tons in 1970 to 218 tons in 1972 (39, 82). Yield losses estimated at 22% and 45% have been obtained by natural and artificial infections, respectively, in Colombia (88). Economic surveys, based upon field observations in the same region, estimated yield losses of 13% due to common and fuscous blight bacteria (50).

Hosts include *Phaseolus vulgaris, P. coccineus, P. mungo, P. aureus, P. acutifolius, P. aconitifolius, P. angularis, Lablab niger, Strophostyles helvula, Glycine max, Stizolobium deeringianum, Lupinus polyphyllus,* and *Vigna sinensis* (77, 92).

Common names frequently used for common bacterial blight in Latin America include bacteriosis, añublo bacterial comun, tizón comun and crestamento bacteriano.

Etiology

Laboratory isolations and purifications are necessary to distinguish the two organisms. The only significant character which distinguishes *X. phaseoli* from *X. phaseoli* var. *fuscans* is the production of a diffusible brown pigment (melanin) by the latter on a medium containing tyrosine (36). Pigment-producing isolates tend to be more virulent than those unable to produce the pigment (6); however, the pigment may not be essential for pathogenicity. Dye (30) concluded that there was little justification for separating *X. phaseoli* from *X. phaseoli* var. *fuscans*, since pigment production is common in *Xanthomonas* species not pathogenic to beans and may not even be a stable character (4).

Xanthomonas phaseoli has been described according to the following biochemical, physical and physiological characteristics: It produces single cells which are straight rods and motile by means of a polar flagellum. It is gram negative and strictly aerobic. It produces a yellow pigment due to a non-water soluble eacotenoid and a mucoid growth on nutrient glucose agar. Acid is produced as a metabolic by-product when cells grow on media containing arabinose, glucose, mannose, galactose, trehalose or cellabiose. It also causes proteolysis of milk (31).

Both organisms grow well on potato dextrose, nutrient and yeast-extract-dextrose calcium carbonate agars. The latter medium is used most commonly and consists of 10 g yeast extract, 10 g dextrose, 2.5 g calcium carbonate and 20 g agar in 1 liter distilled water (56). A relatively selective medium has been developed for isolating *Xanthomonas* sp. (40) and *X. campestris* (60), but *X. phaseoli* and *X. phaseoli* var. *fuscans* grow only in these media when mass-streaked onto the plate.

Epidemiology

X. phaseoli and *X. phaseoli* var. *fuscans* are warm temperature pathogens in contrast to *Pseudomonas phaseolicola* which is a cool temperature pathogen (34). Common and fuscous blight bacteria cause more severe damage to plants at 28°C than at lower temperatures (44, 49). *X. phaseoli* grows best *in vitro* at 28°- 32°C, and growth declines gradually as temperature is lowered. At 16°C little growth occurs. Detailed meteorological and microclimatological data are not available to determine the factors that influence development of bacterial blight epidemics. In general, however, common blight epidemics are favored by high temperature and high humidity (75).

Plant pathogenic bacteria can survive adverse environmental conditions and extended absence of host plants in the field by several means. One of the most effective means is on or within infected bean seed. Seed transmission of *X. phaseoli* has been known since 1872 (66, 69). Bacteria have been recovered from three (5), 10 (92) and 15 (71, 72)-year old bean seed. Such seed-borne isolates normally are viable and virulent when recovered from seed (56, 57, 59, 70).

Seed lots can be assayed for the presence of bacteria by incubation in water or a liquid medium which then is inoculated into susceptible plants by injection, watersoaking (67) or vacuum infiltration (80). Saettler and Perry (59) assayed 101 Navy bean seed lots for internal contamination with *X. phaseoli* and *X. phaseoli* var. *fuscans*. Approximately 35% of these were contaminated with *X. phaseoli*, 13% with *X. phaseoli* var. *fuscans* and 52% with both organisms. Wallen *et al.* (83) sampled 23 seed lots from Ontario, Canada and isolated virulent cultures of *X. phaseoli* var. *fuscans* from more than 50% of the samples. The minimum level of primary inoculum required to incite an epidemic is not known but should be determined for various cultural and environmental conditions.

Short term survival within healthy-appearing bean plants can occur during a growing season (76), and bacterial numbers can increase on symptomless leaves (86). Both *X. phaseoli* and *X. phaseoli* var. *fuscans* can survive between growing seasons in temperate zones within infested bean debris (64, 69). Survival occurs in bean debris placed on top of but not 20 cm below the soil surface, and survival is greater under dry than under moist environmental conditions. Bacteria are recovered from the soil up to six weeks after burial, but Schuster (64) speculated that survival occurred in infested plant debris.

Sutton and Wallen (75) could not isolate *X. phaseoli* from soil in which infected plants had been grown. Schuster and Coyne (70) believe that survival in the tropics may be greater than in temperate zones because of the opportunities to continually increase populations and to possibly survive as epiphytes on perennial hosts. Studies are needed to determine the extent of *X. phaseoli* and *X. phaseoli* var. *fuscans* survival in infested plant debris and soil under tropical conditions.

Although plant pathogenic bacteria do not form spores, many are tolerant to desiccation and can survive extended dry conditions *X. phaseoli* produces an extracellular polysaccharide in culture and in the host plant (42). It can survive in this exudate for prolonged periods under varied environmental conditions (87).

The bacteria obviously can be disseminated quite effectively on and within bean seed. Plants grown from infected seed frequently bear lesions on the cotyledons, nodes or primary leaves which serve as initial centers for pathogen spread during favorable environmental conditions (92). Infected seed or infested plant debris may be present within bean cull piles and can serve as initial sites for disease development (7). Infested bean straw residue present in fields can provide another locus from which bacteria may be disseminated to susceptible plants (69).

Secondary spread of common and fuscous blight bacteria is made easier with rain accompanied by wind (92), wind-blown soils (11), possibly by irrigation water (74), and by insects such as the white fly (55). Common and fuscous blight bacteria can survive on the bodies of insects and be transmitted to wounds caused by leaf-feeding insects such as *Diaprepes abbreviata* and *Cerotoma ruficornis* (41). Some bacterial pathogens such as *Pseudomonas glycinea* are spread within aerosols (79), but this has not been reported for *X. phaseoli* or *X. phaseoli* var. *fuscans*.

Plant Infection and Symptomatology

Xanthomonas phaseoli and *X. phaseoli* var. *fuscans* induce identical symptoms on leaves, stems, pods and seeds. Leaf symptoms initially appear as water-soaked spots on the undersides of leaves or leaflets (Fig. 1). These spots then enlarge irregularly, and adjacent lesions frequently coalesce. Infected regions appear flaccid, are encircled by a narrow zone of lemon-yellow tissue, later turn brown and necrotic (Fig. 2) and may be so extensive (Fig. 3) as to cause defoliation or stem girdle (92).

Blight bacteria enter leaves through natural openings such as stomata and hydathodes, or through wounds (92). The bacteria then invade intercellular spaces, causing a gradual dissolution of the middle lamella. The bacteria may enter the stem through stomata of the hypocotyl and

Fig. 1- Water-soaked spots caused by leaf infection of common and fuscous blight.

Fig. 2- Common blight lesions showing lemon yellow and necrotic symptoms.

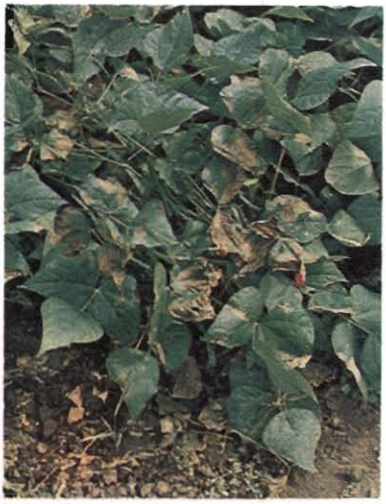

Fig. 3- Severe foliage infection by common bacterial blight.

Fig. 4- Stem girdle and breakage caused by common bacterial blight.

epicotyl and reach vascular elements from infected leaves or cotyledons. Presence of a sufficient number of bacteria in the xylem tissue may cause plant wilting by plugging the vessels or disintegration of the cell walls. *X. phaseoli* does not induce systemic infection in all *Phaseolus vulgaris* cultivars (35). Stem girdle or joint rot may develop at the cotyledonary node, especially in plants that originated from infected seed, and cause the plant to break (92) (Fig. 4).

Pod lesions appear as water-soaked spots which gradually enlarge, become dark and red and slightly sunken. If infection occurs during pod and seed development, infected seed may rot or shrivel (Fig. 5). Common and fuscous blight bacteria are harbored both within the seed and on the seed coat. They enter pod sutures from the vascular system of the pedicel and pass into the funiculus through the raphe leading into the seed coat. The micropyle also may serve as a point of entry into the developing seed. Direct penetration through the seed coat has not been reported, but it may occur. If bacteria enter through the funiculus, only the hilum may become discolored. Seed infection is difficult to see when seeds are dark in color.

Fig. 5- Pod and seed infection by common bacterial blight.

but it is evident as butter-yellow spots on white or light-colored seeds (59, 92). Seedlings which develop from infected seed may sustain damage to the growing tip and be killed (snake head) or stunted (92).

A bean plant may be more susceptible to infection by common blight bacteria if previously infected by another pathogen. Panzer and Nickeson (48) demonstrated that common blight is more severe in the presence of bean common mosaic virus, particularly late in the season. Hedges (37) found that the virus persisted in cultures of *X. phaseoli* for six weeks. Diaz Polanco (28) also showed that a synergistic effect existed between *X. phaseoli* and *Macrophomina phaseolina* infection of bean leaves.

Symptoms of *X. phaseoli* are not significantly different from those caused by *X. phaseoli* var. *fuscans*. Zaumeyer and Thomas (92) observed that *X. phaseoli* var. *fuscans* may cause a slight hypertrophy and darkening of the stem at the point of artificial inoculation of young seedlings. Severe plant symptoms can occur from inoculations of fuscous blight bacteria (33, 92). However, inoculations with mixed inocula of fuscous and common blight bacteria can induce more severe symptoms than observed with individual inoculations (32).

Control by Cultural Practices

Cultural practices often utilized to reduce common blight are the use of pathogen-free seed, proper crop rotation and deep plowing (92). Clean or certified seed can be produced in a region free of the pathogen or where environmental conditions are unfavorable for disease development. Crop rotation with plants not susceptible to blight can reduce or eliminate blight bacteria in bean debris within a field. Such recommendations can, however, prove difficult for Latin American producers with small land holdings and limited economic resources.

Control by Chemicals

Various chemicals have been applied as a seed treatment or foliage protectant to control common blight before moderate to severe infection is apparent. They have controlled foliage infection effectively, although yield increases have been minimal. Such compounds include basic Copper Sulfate (29), Copper Hydroxide and potassium (hydroxymethyl) methyldithiocarbamate or Bunema (85). Streptomycin has given marginal control in the laboratory and field and is translocated within the plant but not into developing seeds (45, 46, 54). However, antibiotics should not be foliarly applied since resistant bacterial mutants may be induced.

Control by Plant Resistance

Isolates of *X. phaseoli* have been shown to differ in virulence within and between geographical locations from which they were collected (68). Schuster and Coyne (65) found isolates from infected bean seed from Colombia were much more virulent than standard North American isolates. Other isolates from Uganda were found to be about as virulent as the Colombian isolates (72). Isolates with even greater virulence have since been identified (33, 89). However, these differences may be complicated by variations in inoculation methods, age of isolates, and other factors. Differences in pathogenicity also can exist between sub-isolates taken from individual stock cultures of *X. phaseoli* (12, 73). Pathogenic variation occurs also in *X. phaseoli* var. *fuscans* isolates (33).

Various methods of inoculation have been used and include:

- sticking the cotyledon or cotyledonary node with a needle or scalpel dipped in inoculum (3, 8)
- rubbing the second trifoliate leaves with a cotton swab soaked with a carborundum-inoculum mixture (12)
- soaking leaves with water-inoculation at high pressure (3, 63)
- using vacuum infiltration on leaves (80)
- pricking leaves with a multiple needle cushion (1, 53)
- clipping leaves with scissors dipped in inoculum (32, 84).

Inoculum concentrations can influence the disease reaction. Optimum concentrations are reported to be in the range between 10^7 to 10^8 cells/ml (24, 32, 53).

Phaseolus vulgaris cultivars and breeding materials have been noted to vary in their reaction to infection by common and fuscous blight bacteria (Fig. 6). Immunity to infection has not been found, but many lines are resistant (referred to as tolerant by earlier workers) to infection, with little if any yield loss. However, bacteria can survive in this resistant tissue

Fig. 6- Variation shown by *Phaseolus vulgaris* germplasm for its resistance to infection by common blight bacteria (susceptible left, resistant right).

without inciting disease symptoms (61). In general, beans are more susceptible to infection after the start of the blossoming or reproductive stage of plant development (17, 20, 24). Most workers inoculate plants during flowering and evaluate three to four weeks later. However, inoculations at three to four weeks after planting may be more effective in the tropics if germplasm is quite variable for its maturity, growth habit and adaptation (10, 84). In addition, Coyne and Schuster (18) reported a differential leaf and pod reaction to infection by *X. phaseoli* which was conferred by different genes. Therefore, time of evaluation and disease rating scales must be designed carefully to account for the previously mentioned factors (58).

Schuster (63) first reported that *Phaseolus acutifolius* (tepary bean) was resistant to *X. phaseoli*. Honma (38) then used the tepary bean as a source of resistance to incorporate in *Phaseolus vulgaris*. Coyne and co-workers (16, 22) surveyed more than 1,000 plant introduction (P.I.) lines for resistance to common and fuscous blight infection in the field.

The following *Phaseolus vulgaris* lines and cultivars had a high degree of resistance: P.I. 163117 (accession from India), P.I. 167399 and P.I. 169727 (accessions from Turkey), P.I. 197687 (accession from Mexico), P.I. 207262 and ICA-Guali (accessions from Colombia) and Great Northern (G.N.) Nebraska No. 1 selection 27. Yoshii *et al.* (90) reported that P.I. 282086 and P.I. 313343 had resistant foliage, but the former had susceptible pods. *P. acutifolius* "Tepary Buff" (16) and P.I. 169932 (90) had high degrees of resistance with no symptoms observed. Some *P. coccineus* lines also were quite resistant, but less so than Tepary (16).

These resistant materials have been tested at various locations and exposed to more virulent bacterial isolates than originally used. Thus, while G.N. Nebraska No. 1 selection 27 and P.I. 207262 also were resistant to Brazilian isolates of *X. phaseoli* and *X. phaseoli* var. *fuscans* (9), the former was susceptible to Colombian and Ugandan isolates of *X. phaseoli* (65, 71). P.I. 207262 also was susceptible to a Colombian isolate of *X. phaseoli* and moderately susceptible to some *X. phaseoli* var. *fuscans* isolates (33). Poor plant adaptation to tropical growing conditions in Colombia inhibited the expression of resistance by Jules and P.I. 207262 (10, 84), until their resistance was transferred to agronomically adapted and susceptible backgrounds.

Inheritance of common blight resistance recently has been reviewed (17, 43, 91). Honma (38) made the interspecific cross between resistant *Phaseolus acutifolius* "Tepary 4" and susceptible *P. vulgaris* and found that resistance was quantitatively inherited. Coyne *et al.* (23) further studied inheritance of the resistant selections crossed to an early-maturing

susceptible cultivar G.N. 1140. The resistant reaction was inherited quantitatively and linked to delayed flowering under a long photoperiod and high temperature (24).

The late-maturing G.N. Tara and Jules (14, 15) and early-maturing Valley (19) cultivars were derived from the cross with G.N. 1140. They possess resistance to common blight in temperate regions of the United States. G.N. Starr in an early maturing cultivar derived from six backcrosses of P.I. 165078 (tolerant to *Corynebacterium flaccumfaciens*) to G.N. Nebraska #1 sel. 27 (tolerant to *X. phaseoli),* resulting in resistance to both bacterial pathogens (21). Coyne *et al.* (24, 25) report that the cross between G.N. 1140 and G.N. Nebraska #1 sel. 27 exhibited partial dominance for susceptibility. This inheritance also was reported by Pompeu and Crowder (52) for similar crosses between G.N. Nebraska #1 sel. 27 and susceptible parents. Crosses between resistant P.I. 207262 and susceptible cultivars such as G.N. 1140 revealed that the resistant reaction was completely dominant in the F_1 (20). Transgressive segregation has been observed in these crosses (24, 25, 52, 78) and should allow breeders to increase the levels of resistance within promising germplasm.

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Chapter 10

Miscellaneous Bacterial Diseases

H. F. Schwartz

Chapter 10

Halo Blight

Introduction

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

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

Etiology

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

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

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

Epidemiology

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

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

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

Symptomatology

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

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

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

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

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

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

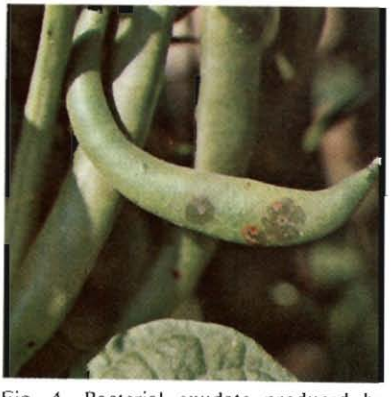

Fig. 4- Bacterial exudate produced by *Pseudomonas phaseolicola*.

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

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

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

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

Control by Cultural Practices

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

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

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

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

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

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

Control by Chemicals

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

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

Control by Plant Resistance

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

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

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

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

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

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

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

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

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

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

Bacterial Wilt

Introduction

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

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

Etiology

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

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

Epidemiology

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

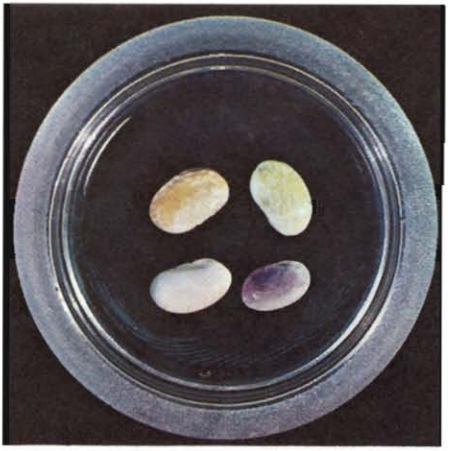

Fig. 6- Seed discoloration due to bacterial wilt infection by different pathogenic strains.

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

Symptomatology

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

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

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

Control by Cultural Practices

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

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

Control by Plant Resistance

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

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

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

Bacterial Brown Spot

Introduction

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

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

Etiology

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

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

Epidemiology

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

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

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

Symptomatology

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

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

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

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

Control by Chemicals

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

Control by Plant Resistance

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

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

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

Miscellaneous Bacterial Pathogens

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

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

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

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SECTION III. MYCOPLASMA AND VIRAL DISEASES

Chapter 11

Mycoplasma-Like Diseases

E. W. Kitajima and
G. A. Granada

Chapter 11

General Review of Mycoplasma - Like Diseases

E. W. Kitajima

Introduction

Various workers (16, 32) have used electron microscopy and antibiotics to demonstrate that some plant diseases, known as "yellows" and believed to have a viral etiology, actually were caused by mycoplasma-like microorganisms (MLM). Many disease problems have been associated with MLM since 1967, especially when symptoms have been characterized by general plant chlorosis, stunting, excessive proliferation of branches (witches' broom) and disorders of floral organs (phyllody) (4, 11, 12, 35, 37, 48). Many of these causal agents are transmitted naturally by leafhopper insects to various hosts, including cultivated crops in the family Leguminoseae (5, 6, 8, 33, 42, 45).

Mycoplasma organisms are prokaryotes, lack a cell wall, are highly pleomorphic, measure 0.2 - 1.0 μm in diameter, possess a membrane, contain ribosomes, RNA and DNA (37). MLM can be seen by electron microscopy within plant sieve tubes but may occur in the phloem parenchyma. They are difficult to grow *in vitro*. However, Sugiura *et al.* (47) have maintained and apparently multiplied MLM associated with Peach-X-disease by placing them in dead cells obtained from the salivary gland of its leafhopper vector (*Colladonus montanus* van Duzee). Since MLM lack a cell wall, they are resistant to penicillin. However, they are susceptible to other antibiotics, such as tetracycline.

Two other types of plant pathogenic prokaryotes are known to infect various hosts but have not yet been detected in beans. The first type is called a spiroplasma, which is motile, has a definite helicoid morphology and measures 0.25 x 3-25 μm. Spiroplasmas have been cultured *in vitro* (9, 17, 44, 49) and are transmitted by leafhoppers (9, 40, 49). Corn stunt (13) and stubborn disease of citrus (17) are caused by spiroplasma organisms. The other type of prokaryote is called a rickettsia-like bacterium. It has a rippled cell wall, and may be located by electron microscopy in xylem

vessels or occasionally in the phloem (28). Pierce's disease of grapes (1, 19, 29), phony disease of peaches (30) and rattoon stunting of sugar cane (38) are caused by rickettsia-like bacteria.

MLM Associated with Legume Diseases

Various MLM are known to infect beans and other legume crops and incite symptoms described generally as legume little-leaf, witches' broom and phyllody, and virescence. Various examples of these diseases are described in this section.

Legume Little-Leaf. Hutton and Grylls (31) described the little-leaf disease associated with forage legumes in Australia. This MLM is transmitted by the leafhopper *Orosius argentatus* Evans, which also is a vector of tomato big bud. Electron microscopy studies revealed the presence of MLM in sieve tubes and phloem parenchyma of naturally infected plants of siratro *(Phaseolus atropurpureus),* alfalfa *(Medicago sativa),* tomato *(Lycopersicon esculentum)* and *Vigna sinensis;* as well as in experimentally infected plants of *Nicotiana glutinosa, Datura stramonium, Catharanthus roseus* and *Phaseolus vulgaris.* They also were detected in the sieve tubes of *Cuscuta australis* used for little-leaf transmission and in the salivary gland of the leafhoppers *(O. argentatus)* that had fed on infected plants (5, 6, 8).

Trials carried out with tetracycline showed that spray applications (100 μg/ml) every two or three days for four to eight weeks eliminated little-leaf symptoms on new growth of *N. glutinosa, Callistephus chinesis* and *Lycopersicon esculentum.* However, the symptoms reappeared when the treatment was suspended. Electron microscope examinations revealed that there were no pleomorphic corpuscles present in the phloem of plants which exhibited a decrease in symptom severity. Moreover, leafhoppers were not able to transmit the pathogen from these plants (7).

Witches' Broom and Phyllody. Witches' broom disease has existed for a long time in Japan on sweet potatoes *(Ipomoea batatas),* soybeans *(Glycine max),* peanuts *(Arachis hypogea),* peas *(Pisum sativum),* beans and *Vigna sinensis* (42, 45). Shinkai (46) found that the leafhopper vector of sweet potato witches' broom was not the same as that transmitting witches' broom of legumes, although both species belonged to the genus *Nesophrosyne,* later reclassified as *Orosius.* The vector of sweet potato witches' broom infected only species in the family *Convulvulaceae* and *Vinca rosea.* The vector causing witches' broom in legumes was able to infect members of the Leguminoseae and several species of Compositae, Amaranthaceae, Cruciferae and Chenopodiaceae (42, 45). The vectors of

witches' broom in legumes and sweet potatoes now have been classified as *Orosius orientalis*, and *O. ryukyuensis*, respectively (46).

The incubation period in the vector of the causal agent of witches' broom of legumes is about one month, but this can be shortened by raising the temperature (e.g. 17 days at 30°C). Diseased bean plants exhibit the typical symptoms of witches' broom - yellowing, reduced leaflets, shoot proliferation and phylloid-like disorders of the floral organs (42, 45). Mycoplasma-like corpuscles were detected in the phloem of diseased legume plants by electron microscopy (15).

Although *Phaseolus vulgaris* was not included in the list presented by Iwaki (33), this author reported the occurrence of witches' broom and phyllody in Indonesia in several legume crops including soybeans, peanuts, mung beans *(Phaseolus mungo), Vigna sinensis* and *Crotalaria* sp. *Orosius argentatus* was identified as the vector in which the MLM has an incubation period of nearly three weeks. Transmission trials showed that the witches' broom causal agent in legumes could infect other plant species. The presence of MLM was confirmed in the tissues of affected plants by histological examinations made with the electron microscope.

Witches' broom and phyllody have caused economic damage to *Vigna sinensis* in the Philippines (3) and Thailand (14). Electron microscopy revealed the presence of MLM in the phloem of infected plants. However, there is no additional information concerning the transmission and vectors of these diseases. In the revision of virus and plant problems associated with MLM, Mishra (41) described witches' broom in *Phaseolus aureus* (Roxb.) and *P. mungo* in India but gave no information concerning the pathogen.

Kitajima and co-workers (35, 36) reported the occurrence of witches' broom in several legumes such as *Crotalaria juncea, C. paulinea, Desmodium* sp., soybeans and siratro. Electron microscopy observations demonstrated that there was a consistent association between MLM and the disease. However, no work was done on its transmission or vector identification.

A few cases (1-3%) of witches' broom and phyllody have been observed in the green-belt of the Federal District in Brazil. The infectious nature of this disease was shown by grafting trials, but its vector has not been identified. Mycoplasma-like corpuscles were found in the sieve tubes of the

vascular region of naturally or experimentally infected plants (Fig. 1 and Fig. 2).

Maramorosch *et al.* (39) reported the presence of MLM in the sieve tubes of pigeon pea *(Cajanus cajan)* plants exhibiting witches' broom symptoms. However, no details were given for its pathology or transmission.

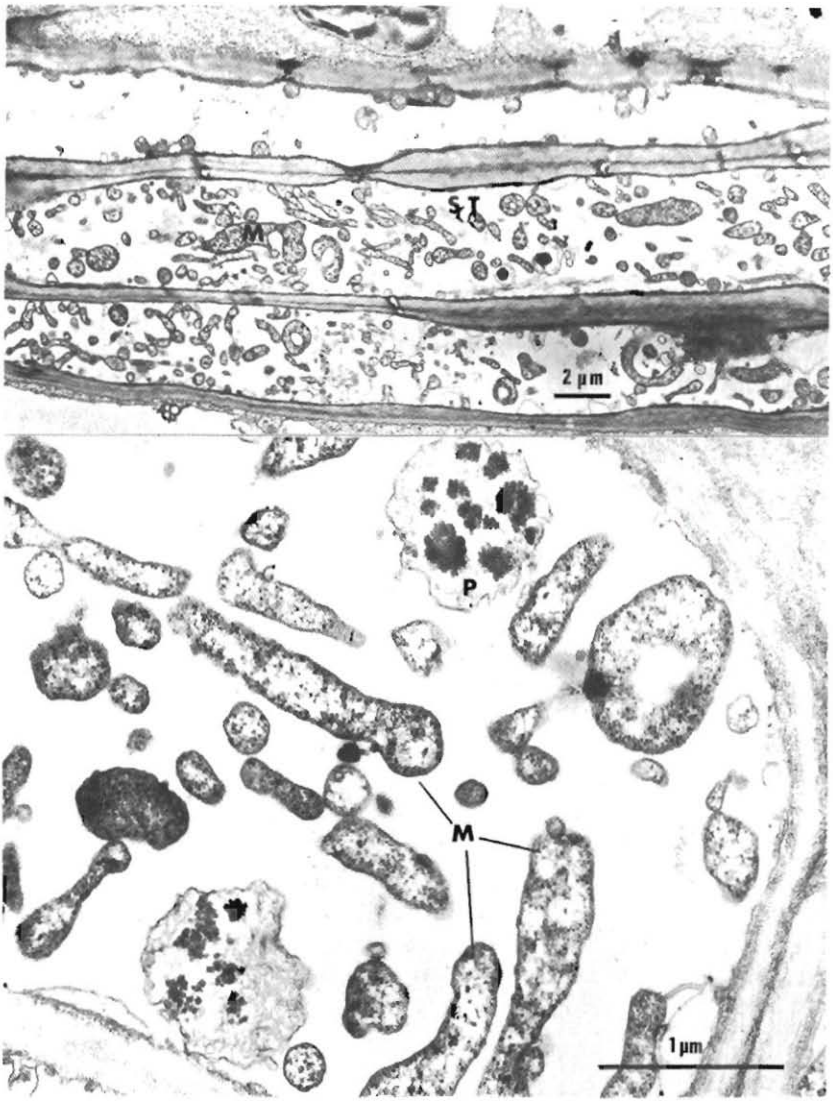

Fig. 1- (top) Electron microphotograph of longitudinal section of bean sieve tubes (ST) containing numerous pleomorphic corpuscles (M).

Fig. 2- (bottom) Electron microphotograph of mycoplasma-like corpuscles (M) showing absence of cell wall and presence of plastids (P).

Virescence. Cousin *et al.* (10) identified mycoplasma-like corpuscles which were present in the cortical parenchyma of beans exhibiting symptoms of virescence and collected in Zagora and Morocco. However, they did not furnish economical or pathological data concerning the disease and its pathogen.

Unfortunately, few data are available to indicate the identity of the MLM associated with witches' broom of legumes in different parts of the world. In the three cases studied in most detail - Australia, Japan and Indonesia - the similarity in host range and vector (31, 33, 45) suggests the possible identity of the etiological agent.

The available information on problems associated with MLM is insufficient to conclude that all of them are caused by the same or different species of a member of the mycoplasma group. Host and/or vector specialization could explain why certain MLM are associated with diseases that have a restricted host range.

Mycoplasma Disease in Colombia

G.A. Granada

Introduction

A mycoplasma-like disease was first detected in 1968 in infected soybean plants grown in the Cauca Valley of Colombia (2, 20). Since then its incidence has increased in cultivated soybean crops and can vary from 0.4-80.0% plant infection with corresponding yield losses of 8-1600 kg/ha (26). A similar disease has been observed since in beans with 8-15% plant incidence in commercial plantings grown in the Cauca Valley.

This mycoplasma-like organism can infect the following hosts: *Glycine max, Phaseolus vulgaris, P. angularis, P. calcaratus, P. lunatus, Crotalaria spectabilis, C. juncea, Desmodium* sp., *Vinca rosea, Cajanus cajan, Rhynchosia minima* and *Galactia glaucescens* (21, 25). Common names frequently used for bean mycoplasma in Latin America are machismo and amachamiento.

Etiology

Electron microscopy evaluation of infected bean or soybean tissue reveals the presence of mycoplasma-like corpuscles which lack cell walls and are located in the phloem cells. The mycoplasma-like etiology also has been confirmed by symptom expression and the remission of symptoms when infected plants are treated with tetracycline (24, 27, Granada, unpublished data).

Transmission

The mycoplasma-like organism is transmitted by the brown leafhopper *Scaphytopius fuliginosus* Osborn (Fig. 3) (20, 23). High population levels of this insect have been detected in infected soybean fields in Colombia (18). This vector has been shown to transmit the mycoplasma-like organism to bean plants grown under controlled conditions (Granada, unpublished data). Newson recently has utilized a *Scaphytopius* species to transmit a viral-like organism in soybeans (M.E. Irwin, INTSOY, personal

Fig. 3- Leafhopper vector (*Scaphytopius fuliginosus*) of bean mycoplasma-like organism in Colombia.

correspondence). Other species of *Scaphytopius* transmit mycoplasma-like diseases, such as stubborn disease of citrus (*S. nitridus*), aster yellows *(S. delongi* and *S. irroratus);* or virus diseases such as alfalfa witches' broom *(S. acutus)* and a cranberry disease *(S. magdalensis)* (34, 43).

When one to six-day-old bean seedlings were exposed to cage-reared infective adults of *S. fuliginosus* for five days, the average incubation time of the pathogen was 37 days (range of 31-43 days) (Granada, unpublished data). This is similar to the 39-day incubation period obtained in soybeans tested under the same conditions (23). The organism is not transmitted mechanically or by seed, but it is graft transmissible (Granada, unpublished data). Bowyer and Atherton (6) report that legume little leaf has an incubation period of only 19-23 days, while other insect vectors have incubation periods which range from seven to 102 days (31).

Symptomatology

Symptoms of mycoplasma infection generally become apparent during flowering and pod development when the plant reproductive stage is converted into a continuous vegetative stage. Time of infection determines the extent of this conversion within the plant.

Early infection causes flower petals to be light or dark green (virescence), and flowers are smaller but have longer sepals than normal flowers. A corrugated structure emerges from the unopened floral apex which is filiform at the upper end and resembles a rolled leaf when dissected (phyllody) (Fig. 4). Later infections may cause pods to be rigid, thin, erect, twisted, corrugated, oriented upwards, and shaped like a half-moon (Fig. 5). These pods form few if any seeds. Severe symptoms may appear as

Fig. 4- Phyllody caused by mycoplasma infection of bean.

Fig. 5- Pod deformation caused by bean mycoplasma infection.

Fig. 6- Leaf and petiole deformation caused by bean mycoplasma infection.

Fig. 7- Witches' broom symptom of infected plant.

flowers are reduced to small buds supported on a large petiole from which additional small leaves and petioles may proliferate (Fig. 6). The general plant symptom may resemble a typical witches' broom (Fig. 7). Late infection of plants bearing healthy-appearing pods may stimulate the premature germination of seeds born in the pods (Fig. 8). These germinated seeds can be transplanted and develop into normal plants free of the mycoplasma-like organism (Granada, unpublished data).

This mycoplasma-like organism induces similar symptoms during flowering in other hosts, such as *P. lunatus,* soybean (Fig. 9), *P. angularis, P. calcaratus, Galactia glaucescens* and *Desmodium* sp. However, infected *Crotalaria spectabilis* plants demonstrate abundant vegetative ramification before flowering, which does not occur in *C. juncea* (Granada, unpublished data).

Fig. 8- (above) Premature germination of bean seeds in immature pod.

Fig. 9- (right) Mycoplasma symptoms in soybean.

Control

Control measures include maintaining an adequate crop rotation and not planting continuous or simultaneous cycles of susceptible crops such as beans and soybeans. This will avoid a build-up and the continued survival of insect vector populations and sources of inoculum from infected plants. When economically feasible, infected plants should be removed from the field and destroyed. In addition, weed hosts should be eliminated from fields and surrounding borders or irrigation canals. Insecticides may reduce populations of the vector and should consist of those used to control the green leafhopper *(Empoasca kraemeri).* Plant resistance may provide an ideal control measure, but no information is available concerning varietal response to infection.

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Chapter 12

Aphid-Transmitted Viruses

G. E. Gálvez

Chapter 12

Aphid - Transmitted Viruses

General Introduction

Four aphid-borne viruses infect beans. They are bean common mosaic virus (BCMV), bean yellow mosaic virus (BYMV), cucumber mosaic virus (CMV) and alfalfa mosaic virus (AMV). This chapter will review the geographical distribution, economic importance, host range, physical properties, purification, transmission, epidemiology, symptomatology, and control measures reported for this group of bean viruses, except AMV, which has been included in the miscellaneous group of viruses.

Bean Common Mosaic Virus

Introduction

Bean common mosaic was one of the first virus diseases reported in the world, when Iwanoski (88) observed it in the Soviet Union. Since then, this seed-borne virus has been reported in nearly every country of the world. It is economically important throughout Africa, Europe, North America and Latin America (1, 2, 4, 34, 37, 38, 39, 40, 41, 42, 43, 45, 46, 47, 48, 50, 51, 52, 54, 62, 66, 67, 68, 86, 93, 96, 97, 98, 99, 100, 110, 111, 112, 113, 114, 118, 138, 139, 146, 164, 169).

Plant infection may reach 100% in fields, and yield losses are reported to range from 35-98% (28, 31, 64, 77, 169). Hampton (77) reported that pod number per plant was reduced 50-64% and seed yield per plant was reduced 53-68%, depending upon the virus strain. Gálvez and Cárdenas (64) reported that yield losses varied from 6-98%, depending upon the cultivar and time of infection.

The host range for BCMV is more limited than that reported for BYMV, but still includes: *Phaseolus vulgaris, P. limensis, P. acutifolius* var. *latifolius, P. angularis, P. aconitifolius, P. calcaratus, P. mungo, P. coccineus, P. atropurpureus, P. radiatus, P. aureus, P. lunatus, P. polyanthus, Vigna sesquipedalis, V. sinensis, Vicia faba, Crotalaria spectabilis, Canavalia ensiformis, Lupinus alba, Nicotiana clevelandii,*

Macroptilium lathyroides, Pisum sativum, Medicago sativa, Dolichos lablab, Trifolium pratense and *Rhynchosia minima* (21, 68, 91, 92, 103, 118, 130, 137, 169). *Sesbania exaltata* and *Macroptilium atropurpureum* are reported to be symptomless hosts (103). *Chenopodium quinoa, Gomphrena globosa, Tetragonia expansa* and cultivars of *Phaseolus vulgaris* serve as local lesion indicators to various strains of BCMV (21, 123, 130, 134, 135, 141, 155, 157, 166).

BCMV was called bean virus 1 and *Marmor phaseoli* Holmes by earlier workers (169). Common names frequently used for bean common mosaic virus in Latin America include mosaico común and mosaico comum.

Symptomatology

Bean common mosaic virus may incite three types of symptoms: mosaic, systemic necrosis (black root), or local lesions, depending upon the cultivar, time of infection, strain and environmental conditions. Mosaic symptoms appear in systemically infected cultivars and may cause a mottling, curling, stunting and malformation of primary leaves (Fig. 1), especially if the primary infection occurred through contaminated seed. The trifoliate leaves express leaf curling and malformation and a mosaic of yellow and various shades of green (Fig. 2). Infected leaves may appear narrower and longer than uninfected leaves, and leaf tips curl downwards and deform the leaf (Fig. 3).

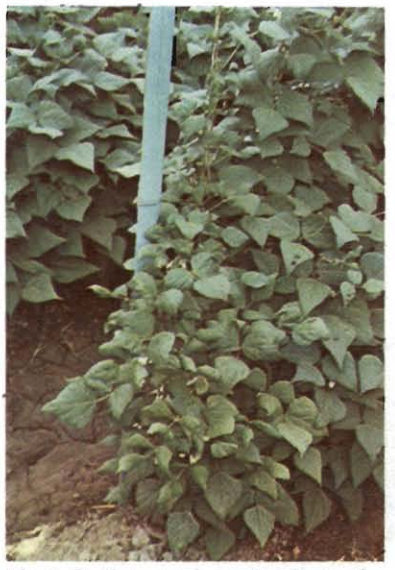

Fig. 1- Curling, stunting and malformation of leaves infected by BCMV.

Fig. 2- Leaf mosaic symptoms induced by BCMV infection.

Fig. 3- Leaf curling and malformation induced by BCMV infection.

Fig. 4- Initial leaf symptoms of black root reaction induced by BCMV.

Fig. 6- Black root induced necrosis in vascular system of bean pods.

Fig. 5- Plant wilting and systemic necrosis symptoms of black root.

Systemically infected plants may have smaller pods which contain fewer seeds than pods from uninfected plants. Infected pods occasionally may be covered with small dark green spots and mature later than uninfected pods (167, 169). Symptoms of systemic mosaic are expressed more clearly at moderate temperatures between 20° - 25°C.

Systemic necrosis or black root symptoms may appear in cultivars possessing resistance (hypersensitive I gene) to systemic mosaic and which are infected by necrosis-inducing strains at low temperatures (20°C) or other strains at high temperatures (26° - 32°C). Infection may reach 40-100%, and occurs from aphids which transmit BCMV particles from susceptible beans or other hosts to resistant plants.

Symptoms initially appear as leaf lesions (Fig. 4) or in the plant apex and young trifoliates which wilt, become dull green and then black (Fig. 5). Eventually the entire plant wilts and dies. A chararacteristic necrosis (reddish-brown to black) of the vascular system may be evident in leaves, stems, roots and pods (Fig. 6) (55, 80, 81, 82, 169). Bean southern mosaic virus, the necrosis strain of bean yellow mosaic virus and a strain of bean rugose mosaic virus also are able to induce systemic necrosis symptoms (35, 38, 169).

Local lesions may appear on leaves of cultivars resistant to systemic mosaic infection. These lesions may be induced by mechanical inoculation or aphid transmission. They are evident as reddish to dark brown necrotic lesions or spots (Fig. 7) of varying size and frequency, depending upon the cultivar, strain, and environmental conditions. Cultivars which are known local lesion hosts include Great Northern U.I. 31 and 123, Pinto U.I. 111, Potomac, Stringless Green Refugee, Plentiful and Monroe (123, 130, 134, 135, 141, 155, 157, 166).

Physical Properties and Purification

BCMV particles can be observed easily with the electron microscope in crude sap or partially purified preparations. The flexible and filamentous virus particles are 730-750 nm in length and 12-15 nm in width (26, 36, 109). These particles are similar in morphology to those produced by bean yellow mosaic virus, see Fig. 12. Cytoplasmic inclusions also are easily observed in preparations and may be present as filaments, lamellates and pinwheels (Fig. 8) (36, 79). Virus particles are transported throughout the phloem and can be detected in upper plant parts within 24-48 hours and in the root system within 60 hours after inoculation (58, 59, 60, 61).

Fig. 8- (above) Cytoplasmic inclusions or pinwheels (25,000 X) produced by BCMV.

Fig. 7- (left) Local lesions produced by BCMV in inoculated bean leaves.

BCMV particles are inactived in sap at 56° to 65°C, have a dilution end point of 10^{-3} to 10^{-4}, and are infective for one to four days (21, 67, 106, 137). Morales (109) determined that BCMV has a 260/280 absorbance ratio of 1.27 and a molecular weight of 32.5 to 34.4 x 10^3 daltons for the capsid protein subunit.

Other physical properties have not yet been determined for this virus, since it is difficult to purify. BCMV particles tend to aggregate and precipitate at low centrifugal forces and are difficult to separate from major plant contaminants (21, 68, 101, 103, 110, 158). Recently, Morales (109) developed a purification method which permits the isolation of BCMV with a high degree of purity and in adequate amounts to produce a specific

Fig.9- Winged aphid adults such as these may act as virus vectors.

antiserum. This purification procedure utilizes clarification with chloroform and carbon tetrachloride, precipitation with polyethylene glycol and equilibrium centrifugation in cesium chloride.

Transmission and Epidemiology

BCMV particles may be transmitted mechanically, in pollen and seed from infected plants, and by insect vectors. BCMV-infected leaves, used as inoculum, can be homogenized in water or buffers such as potassium phosphate and then manually applied to leaves of healthy susceptible plants (109). Many workers also have added abrasives such as carborundum powder to inoculum to facilitate the introduction of virus particles into plant cells (33, 169).

An inoculation efficiency of nearly 100% can be achieved in the glasshouse, while in the field the efficiency is lower due to adverse environmental factors which may affect both the viruses and the plants.

Virus particles can be transmitted in pollen grains, ovules and flowers of infected plants (58, 59, 163, 169). Seed transmission likewise can occur in susceptible cultivars of *Phaseolus vulgaris, P. acutifolius, P. coccineus, P. polyanthus, P. mungo, Macroptilium lathyroides* and *Rhynchosia minima* (91, 103, 117, 122, 125, 126, 131, 137, 147). The percentage of seed transmission may vary from 3 to 95%. It is affected by the cultivar and the time of infection, especially before flowering (5, 28, 39, 40, 41, 42, 43, 44, 49, 54, 64, 65, 98, 106, 107, 118, 140, 169). BCMV particles are reported to survive in bean seed for at least 30 years (169).

Insect vectors such as aphids (Fig. 9) can transmit BCMV effectively from infected plants to healthy plants. Reported aphid vectors include *Macrosiphum solanifolii, M. pisi, M. ambrosiae, Myzus persicae, Aphis rumicis, A. gossypii, A. medicaginis, Hyalopterus atriplicis* and

Rhopalosiphum pseudobrassicae (169). Studies have determined that aphid populations often are lower than those of other insect species in bean fields, but that the aphids are responsible for transmission of BCMV particles. The efficiency of transmission depends upon the leaf (source of inoculum) on which aphids feed (170) and the period of pre- and post-feeding by aphids (172).

Infected seeds and plants of susceptible bean cultivars and weed hosts serve as sources of initial inoculum for BCMV in the tropics and other regions (131, 132, 133). Aphids are responsible for the secondary transmission of the virus. In Colombia, studies determined that relatively high apterous aphid populations were able to incite 100% plant infection from a seed source that was only 15-25% contaminated (39, 40).

Control by Cultural Practices

Various cultural practices, such as planting date and clean seed production, have been used to reduce the incidence of BCMV infection in susceptible cultivars. Burke (29) found a correlation between planting date and virus incidence which was associated with aphid population levels. Therefore, bean plantings should be adjusted to minimize the period during which susceptible cultivars may be exposed to infection by aphids migrating from other crops to beans during the growing season.

Production of seed free from BCMV can effectively reduce the initial inoculum. However, it also may be necessary to control the aphids with insecticides to reduce transmission of BCMV from other infected bean plants or weed hosts (40, 136). No chemicals or other treatments are available to remove or destroy BCMV particles present within infected seed (39, 169).

Control by Plant Resistance

Plant resistance to bean common mosaic virus has been available for nearly 60 years since the cultivar Robust was discovered to be resistant. The resistance of Robust was later determined to be conferred by a single recessive gene (11, 34, 72, 78, 120, 134, 169). Cultivars subsequently derived with Robust resistance include Great Northern U.I. No. 1, No. 59, No. 81, No. 123, Red Mexican U.I. No. 3 and No. 34, Royal Red, Pinto U.I. No. 72, No. 78 and 111 (32, 148, 149, 169). These cultivars have been resistant to the type strain of BCMV for more than 50 years (165, 168).

Nearly 50 years ago another source of resistance was identified in Corbett Refugee. This resistance was determined to be conferred by a dominant gene (hypersensitive gene affected by black root). The majority

of cultivars developed in the United States have derived their resistance from Corbett Refugee and include Wisconsin Refugee, Idaho Refugee, Refugee U.S. No. 5 (169). This resistance has been effective for nearly 50 years (165), and Burke and Silbernagel (30) have suggested that the Corbett Refugee type of resistance be widely incorporated into commercial cultivars.

These sources of resistance also have been used to develop resistant cultivars in Latin America, such as ICA-Tui and ICA-Pijao in Colombia, Titan and Arroz 3 in Chile, Peru 257 in Peru, Tacarigua in Venezuela, and Jamapa and Sataya 425 in Mexico (34, 40, 55, 106, 107, 119, 156, 173).

Hagel *et al.* (75) have reported that certain BCMV resistant cultivars, such as Black Turtle Soup, also express tolerance to insect vectors such as aphids. Additional studies are necessary to determine the effectiveness of this type of aphid resistance and its applicability to commercial production.

Plant resistance to BCMV is affected by the nature of the gene(s) conferring resistance, variability between virus strains and environmental conditions. Various workers have investigated the relationships between different virus strains and sources of resistance (6, 7, 14, 55, 56, 57, 144). Drijfhout and co-workers have assigned 22 cultivars to 11 resistance groups, and divided the 15 known viral strains in seven pathogenicity groups. Gálvez *et al.* (65) have proposed a similar system of nomenclature (BCMV-1 to BCMV-7) to distinguish these seven basic viral groups (Table 1). The International Working Group on Legume Viruses has presented another viral strain classification.

Cultivars in resistance groups one to six do not express systemic necrosis to any viral strains but do express systemic mosaic symptoms to one or more of the viral groups. These cultivars, therefore, possess recessive alleles for the necrosis gene "I". Likewise, line IVT 7214 (resistance group 7) does not exhibit systemic mosaic or necrosis upon inoculation with any known viral strain and possesses recessive alleles for the necrosis gene. Cultivars in resistance groups eight to 10 exhibit systemic necrosis to one or more viral strains, and no systemic mosaic symptoms to any viral strain. These cultivars, therefore, possess dominant alleles for the necrosis gene. The IVT 7233 line likewise possesses dominant alleles for the necrosis gene but exhibits only local necrotic lesions.

Results from these investigations should allow breeders and pathologists to incorporate resistance gene(s) effective against the known pathogenicity spectrum and provide growers with resistant commercial cultivars adapted to the tropics and other regions of the world.

Bean Yellow Mosaic Virus

Introduction

Bean yellow mosaic virus is widely distributed throughout the world on beans and many other hosts. The virus is reported to occur in North America, Europe, East Africa, Japan (20, 86, 159, 169), and Latin American countries such as Chile (27, 35), Argentina (121), Brazil (46, 95), Uruguay (Juan Izquierdo, personal communication), and possibly northern Mexico. The distribution of BYMV in Latin America is not completely known, since it often has been confused with bean golden mosaic virus.

BYMV can infect up to 100% of the plants grown in a field as observed in the United States (169). Hampton (77) reported that BYMV could cause serious yield losses with a 33% and 41% reduction in pod number and seed yield, respectively. Little research has been conducted in Latin America to measure yield losses induced by BYMV. However, the existence of virus complexes has made it difficult to measure accurately the effect of individual viruses.

Bean yellow mosaic virus has been called Phaseolus virus 2, Gladiolus mosaic virus, pea mosaic virus, and bean virus 2 by earlier workers (169). Common names frequently used for BYMV in Latin America include mosaico amarillo, mosaico amarelo and moteado amarillo.

Bean yellow mosaic virus has a wide host range which includes *Phaseolus vulgaris, P. aureus, P. lunatus, Cajanus indicus, Cicer arietinum, Lathyrus odoratus, Lens esculenta, Melilotus alba, Cucurbita sativum, Pisum sativum, Vicia faba, V. americana, V. monantha, V. villosa, V. sativa, V. atropurpurea, Vigna sesquipedalis, Vigna sinensis, Trifolium pratense, T. incarnatum, T. hybridum, Medicago sativa, M. lupulina, Glycine max, Gladiolus* spp., *Trigonella foenumgraecum, Crotalaria spectabilis, Lupinus deusiflorus, Proboscidea jussievi, Cladrastis lutea, Robinia pseudoacacia, Freesia* sp., *Babiana* sp., *Ixis* sp., *Sparaxis* sp., *Tritonia* sp., *Nicotiana tabacum, N. sylvestris* and *N. rustica* (20, 90, 127, 128, 169, 171).

Symptomatology

Initial symptoms of BYMV systemic infection appear as small chlorotic spots one to three mm in diameter, which are often surrounded by a halo. These spots gradually enlarge and coalesce to produce a general chlorosis

Fig. 10- Chlorotic leaf symptoms caused by BYMV infection.

Fig. 11- Leaf malformation induced by BYMV infection.

on affected leaves (Fig. 10). Young leaves become brittle, glossy, concave on the upper leaf surface, and may be malformed (Fig. 11). Yellow and green mottling becomes more intense on leaves as they age. Infection causes shortened internodes, proliferation of branches and plant stunting. It also may delay maturity (169).

Systemic necrosis symptoms can be induced by certain strains of BYMV. Symptoms appear as a purplish coloration at the base of the lower leaves, which may be accompanied by veinal, stem and petiole necrosis, top necrosis at the terminal growing point, or plant death. These symptoms may resemble those induced by necrotic strains of BCMV (Black Root). Other BYMV strains are able to incite local necrotic lesions on leaves. The typical chlorotic leaf symptoms also may be evident (35, 169). Reddish-brown spots may form on infected pods, which can be malformed, depending upon the specific virus strain (169).

Physical Properties and Purification

Particles of BYMV resemble those of BCMV since they are long, flexible (Fig. 12), and measure 750 nm in length and 15 nm in width (25, 26, 161). Cytoplasmic inclusions may be spiral, ring or lamellate pinwheels which

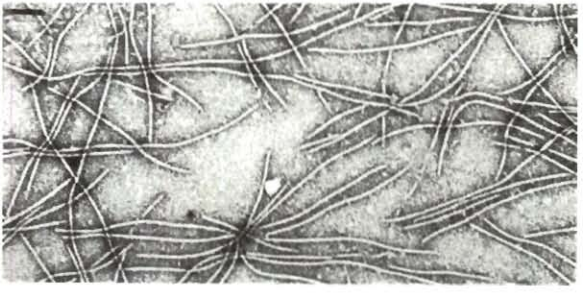

Fig. 12- Filamentous particles of BYMV.

are typical of the potyvirus group (19, 20, 27, 36, 87, 95, 153). These pinwheels are similar in morphology to those produced by bean common mosaic virus, see Fig. 8.

BYMV has a 260/280 absorbance ratio of 1.18 - 1.20 (89, 108). BYMV particles have a thermal end point between 50° to 60°C, and a dilution end point between 10^{-3} and 10^{-4}. Particles retain their infectivity for one to two days and occasionally up to seven days. These properties depend upon the virus source, host plant and experimental conditions (20, 116, 169).

Purification of BYMV was difficult in early work since particles aggregated easily and also agglutinated to plant chloroplasts. Various workers developed methods to partially purify BYMV (12, 83, 84, 162). Morales (108) developed a procedure which yields highly purified and nondenatured BYMV preparations. The purification procedure is similar to that described for BCMV. It utilizes clarification with chloroform and carbon tetrachloride, precipitation with polyethylene glycol and equilibrium centrifugation in cesium chloride. Sodium diethyldithiocarbamate (chelating agent) must be added to the extraction buffer to purify the necrotic strain of BYMV. Jones and Diachun (90) also have developed a reliable purification procedure.

BYMV has some serological similarities to BCMV but can be distinguished. BYMV also has various strains which now can be distinguished serologically (13, 14, 15, 20, 23, 24, 70, 90, 116, 169). Jones and Diachun (90) identified three BYMV subgroups within a collection of BYMV isolates obtained from infected red and white clover. These subgroups differ for serological and biological factors such as host range and symptoms. Additional work is required to establish an acceptable set of host differentials and strain classification.

Transmission and Epidemiology

BYMV particles may be easily transmitted mechanically and by insect vectors such as aphids. BYMV is not transmitted in seed of *Phaseolus vulgaris*. However, it can have a low transmission in seed of *Vicia faba* and some other legumes (20).

Aphid vectors include *Acyrthosiphon pisum, Macrosiphum euphorbiae, Myzus persicae* and *Aphis fabae* (20, 71, 150, 151, 152, 154). Aphid transmission from infected beans or other hosts is primarily responsible for natural epidemics of BYMV. Some strains of BYMV are not easily transmitted by aphids (63, 150, 154), and some BYMV strains may lose aphid transmissibility during storage or maintenance by mechanical inoculation (154).

Control

Alternate hosts of BYMV should be eliminated from bean fields and adjacent areas and as components of crop rotations. Chemical control may be utilized to reduce aphid populations present within bean fields or other host crops (74, 75, 76, 85, 132, 160, 169).

Plant resistance appears to be the most reliable control measure available (168). Resistance to specific strains is conditioned by specific plant genes such as By-2 (53, 142). Sources of resistance to the BYMV strain inducing pod malformation have been identified in various Great Northern lines such as G.N. U.I. No. 31, 59, 123 and 1140. This resistance is conferred by three recessive genes with modifiers (9, 10, 35, 73, 168). Resistance to BYMV strains and BCMV has been found in interspecific crosses between *Phaseolus vulgaris* and *P. coccineus* (8, 11, 169). Black Turtle Soup is resistant to BCMV and likewise is not a preferred host for aphids (75). Additional research is necessary to identify and incorporate sources of resistance effective against all strains of BYMV (129).

Cucumber Mosaic Virus

Introduction

Cucumber mosaic virus (CMV) is widely distributed throughout the world, including the United States, Puerto Rico, Spain, France and Brazil (16, 22, 102, 104, 105, 145, 169). The virus is not reported to be a serious or economically important disease (16, 104, 169).

Cucumber mosaic virus has been called cucumber virus 1, *Cucumis* virus 1, *Marmor cucumeris,* Spinach blight virus and tomato fein leaf virus. The common name frequently used for CMV in Latin America is virus del mosaico del pepino.

The host range of CMV includes *Phaseolus vulgaris, P. aborigeneus, P. aconitifolius, P. angularis, P. bracteatus, P. calcaratus, P. caracalla, P. coccineus, P. dumosus, P. erythroloma, P. lunatus, P. panduratus, P. phyllanthus, P. pilosus, P. polystachios, P. radiatus, Macroptilium atropurpureum, M. lathyroides, Capsicum annuum, Chenopodium album, Cucumis sativus, Nicotiana* spp., *Ocimum basilicum, Spinacia oleracea, Canavalia ensiformis, Lathyrus sativus, Pisum sativum, Vicia faba, Vigna unguiculata, Gomphrena globosa* and *Musa* spp. (22, 104, 124).

Symptomatology

Symptoms of CMV infection may consist of a mild mosaic, vein clearing, vein banding, leaf rolling, epinasty and/or apical necrosis. Symptoms may

resemble those induced by BCMV. The intensity of symptom expression may vary, depending upon the cultivar, strain and time of infection. Symptoms may become less noticeable in older tissue if infection occurred in very young plants. Pod distortion also may be evident (16, 17, 105, 124).

Physical Properties and Purification

CMV particles are isometric and may be 20-22 nm (105), 24-27 nm (104), or 30 nm (69) in diameter. The particles are present in clusters of 180 subunits which form pentameres or hexameres (69). CMV particles have a thermal end point of 70°C, a dilution end point between 10^{-4} and 10^{-5}, and are infective *in vitro* for three to six days at 23°C (105).

The virus particles have a sedimentation coefficient of 98 S, a molecular weight between 5.8 to 6.7 x 10^6 daltons, a diffusion coefficient of 1.23 at D20 x 10^{-7} cm ^{2}/sec, its isometric point at pH 4.7, and electrophoretic mobility of 8 x 10^{-5} cm^2/sec/volt in 0.1 M buffer at pH 7.0, a 260 nm absorbance of 5.0 and a 260/280 absorbance of 1.65. The virus particles contain RNA which has a molecular weight of 1 x 10^6d, protein subunits which have a molecular weight of 3.2 x 10^4d, and more than 280 amino acids (69).

Various purification procedures have been developed by workers (18, 22, 104, 115, 143). These procedures have enabled researchers to develop antisera to study CMV and its strains.

Transmission and Epidemiology

CMV particles are easily transmitted mechanically, in seed, and by insect vectors such as aphids. CMV may be transmitted mechanically from

Fig. 13- Leaf symptoms of cucumber mosaic virus in infected cucumber plants.

infected beans, tobacco, cucumbers (Fig. 13) and other hosts (16, 102, 104). Seed transmission may vary from less than 1% to 30%, depending upon the bean cultivar (16, 22, 102, 104, 124). Bos and Maat (22) reported that CMV retained its infectivity in stored bean seeds for 27 months.

More than 60 species of aphids may transmit CMV. They include *Aphis gossypii* and *Myzus persicae* (94, 104, 124). Meiners *et al.* (104) report that aphids retained infective particles of CMV for up to 40 minutes after a 10 minute accession feeding period.

Control

Control measures may include planting seed free of contamination by CMV and crop rotation to reduce the number of hosts for the virus and/or its insect vector. Chemical control may be used to reduce aphid populations in bean fields or other host crops. Cultivars may differ in their resistance. However, little research has been justified in this area since CMV is of such minor and/or currently unknown importance.

Table 1. Differentiation and grouping of BCMV strains and host resistance groups.

Host resistance group	Differential cultivar name	Pathogenicity group of the virus														
		I			II	III	IVa	IVb			Va	Vb	VIa	VI	VII	
		Westlandia NL1	Type US 1	Puerto Rico PR 1	NL 7	NL 8	Florida US 5	Western US 4	Idaho or B US 3	Colana NL 6	NY 15 US 2	Imuna NL 2	Michelite NL 3	Jolanda NL 5	Mexico US 6	Great North. NL 4
Cultivars with recessive alleles (I*I*) of the necrosis gene																
1	Dubbele Witte	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
	Str. Gr. Ref	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
2	Redl. Gr. C	-	-	-	+	-	+	+	+	+	+t	+	+t	+	+	+
	Puregold Wax	-	-	-	+	-	+	+	+	+	+t	+	+t	+	+	+
	Imuna	-	-	-	+t	-	+	+	+	+	+t	+	+t	+	+	+
3	Redl. Gr. B	-	-	-	-	-	+	+	+	+	-	-	+	+	+	+
	Gr. North. 123	-	-	-	-	-	+	+	+	+	-	-	+t	+t	+	+
4	Sanilac	-	-	-	-	+	-	-	-	-	+	+	+	+	-	-
	Michelite 62	-	-	-	-	+	-	-	-	-	+	+	+	+	-	-
	Red Mex. 34	-	-	-	-	+	-	-	-	-	+	+	+	+	-	-
5	Pinto 114	-	-	-	-	-	-	-	-	-	+	+	+	+	-	-

6	Monroe	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-
	Gr. North. 31	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	
	Red. Mex. 35	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	
7	IVT 7214	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
Cultivars with dominant alleles (II) of the necrosis gene																	
8	Widusa	-	-	-	-	+n	-	±n	±n	±n	-	-	+n	+n	-	-	
	Bl. Turtle S.	-	-	-	-	+n	-	±n	±n	±n	-	-	+n	+n	-	-	
9a	Jubila	-	-	-	-	-	-	+n	+n	+n	-	±n	+n	+n	-	-	
9b	Topcrop	-	-	-	-	-	-	±n	±n	±n	-	±n	+n	+n	-	-	
	Imp. Tendergr.	-	-	-	-	-	-	±n	±n	±n	-	±n	+n	+n	-	-	
10	Amanda	-	-	-	-	-	-	-	-	-	-	-	-	+n	-	-	
11	IVT 7233	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	

\+ Susceptible, sensitive, systemic mosaic.

+t Susceptible, tolerant, systemic symptoms questionable or very weak, virus recovered from uninoculated leaves by back-inoculation onto Dubbele Witte.

\- Resistant, no systemic symptoms, virus not recovered from uninoculated leaves by back-inoculation.

+n Susceptible, sensitive, usually all plants with systemic necrosis, not clearly dependent on temperature.

±n Susceptible or resistant, dependent on temperature, from none to all but mostly only a few plants with systemic necrosis, the number varying in repeated tests and increasing with temperature. Greenhouse mean temperature 22-26°C, day and night fluctuation at most 20-24°C in winter and 20-30°C in summer (55, 57).

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Chapter 13

Beetle-Transmitted Viruses

Rodrigo Gámez

Chapter 13

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General Introduction

One group of bean diseases with characteristic virus symptoms includes mosaics frequently associated with leaf and plant malformations and green or yellow stippling. These diseases are caused by isometric viruses, which are 25-30 nm in diameter. The viruses are easily transmitted mechanically and are very stable and highly antigenic. They belong to various groups of plant viruses distinguishable by their serological properties, host range and the number of nucleoprotein or protein components.

The most important known insect vectors of this group of bean viruses are beetles belonging to the subfamily *Galerucinae* of the family *Chrysomelidae*. This chapter will review the geographical distribution, economic importance, host range, physical properties, purification, transmission, epidemiology, symptomatology, and control measures reported for this group.

Bean Rugose Mosaic and Bean Pod Mottle Viruses

Introduction

Limited information is available on the distribution and economic importance of bean rugose mosaic virus (BRMV). The disease was first observed in Costa Rica in 1964 (18) and later in Guatemala (17) and El Salvador (24). Bean pod mottle virus (BPMV) was originally discovered on beans in 1945 in southern United States (72). Bean pod mottle and bean rugose mosaic viruses belong to the comovirus group and are serologically related.

The host range for BPMV is restricted to legumes such as the common bean (*Phaseolus vulgaris* L.), lima bean (*P. lunatus* L.) and soybean (*Glycine max* L.) (66, 72, 74). The J-10 strain of this virus, however, also has been reported to systemically infect *Chenopodium quinoa* (43). Bean rugose mosaic virus causes a systemic infection in some cultivars of *P. vulgaris, P. acutifolius, P. lathyroides, P. lunatus, Vicia faba, Trifolium incarnatum, Glycine max, Cicer arietinum* and *Pisum sativum* (18). *Vigna unguiculata* also has been reported as susceptible to BRMV (6). *Chenopodium amaranticolor* is a local lesion host.

Common names frequently used for bean rugose mosaic virus in Latin America include mosaico rugoso, ampollado, arrugamiento, and encarrugamiento. Mosaico em desenhos possibly corresponds to this disease in Brazil. The common name frequently used for bean pod mottle virus in Latin America is moteado de las vainas.

Members of the comovirus group are highly antigenic and serologically related (26, 56). Five important serogroups within the comovirus group have been identified in legumes (12) and consist of two serogroups of the cowpea mosaic virus, one serogroup of the bean rugose mosaic virus, one serogroup of the quail pea mosaic virus (42) which includes the strain that causes curly dwarf mosaic on beans (40, 67), and the serogroup of the bean pod mottle virus (43).

Cowpea mosaic virus is the type member of the comovirus group and has a large number of strains in two serogroups. The first serogroup includes strains from Arkansas, Costa Rica, El Salvador and Puerto Rico. The second serogroup contains the Sb strain and the yellow strain (12, 13). These strains also differ in virulence and host range. The BRMV group is composed of isolates from El Salvador. In the BPMV serogroup, the J-10 strain, isolated from soybeans, differs serologically and symptomatologically from that isolated from *Chenopodium quinoa* (43).

Symptomatology

Three different types of reactions have been observed in beans when infected by BPMV or BRMV. These reactions are systemic infection, local lesions and immunity (18, 74). Cultivars which are susceptible to systemic infection do not express local lesions, and cultivars which show local lesions usually do not become systemically infected.

The severity of systemic infection depends upon the virus strains and plant cultivar infected. In general, plants infected by BRMV exhibit a severe mosaic, rugosity, malformation and leaf puckering (Fig. 1). The pods of the infected plants exhibit varying degrees of malformation and mottling, although in some cultivars mottling is not evident (6, 18, 24).

Fig. 1- Leaf blisters and malformation induced by bean rugose mosaic virus infection.

Plants infected by BPMV show mottling with leaf malformation and necrosis in some cultivars but lack the rugosity characteristic of BRMV. Symptoms are most severe on pods, which exhibit an intense mottling, malformation and, frequently, a more intense green tone than healthy pods (72, 74).

Local lesions induced by both viruses are similar. On primary leaves, the local lesions appear three to four days after inoculation, are light to dark brown, necrotic, and approximately 2 mm in diameter. The size varies slightly depending upon the cultivar, plant age and number of lesions per leaf (18, 74).

Bean cultivars used as diagnostic species for BPMV and BRMV (6, 18, 43) include Pinto 111, Stringless Green Refugee, Kentucky Wonder, Sure Crop Wax, Michelite, Sanilac, Potomac, Tender Green, Top Crop, Great Northern U.I. 60, Plentiful, Bountiful, Cherokee Wax, Black Valentine, ICA-Pijao and 27R. Cowpea cultivars such as Monarch and Early Ramshorn, and soybean cultivars such as Lee, Hill, Hood, Improved Pelican, Hampton, Bienville and Biloxi, also have been used.

Numerous bean cultivars produce local lesions after inoculation with either virus. Some cultivars used to determine the properties of these viruses include Idaho Pinto, Pinto 111, Jamapa, Turrialba 2, and ICA-Pijao (1, 6, 18, 72). The bean cultivars Col. 109-R, 27R, and ICA-Guali have been used to propagate BRMV (6, 18). Bean cultivars such as Black Valentine and Cherokee Wax, and soybean cultivars such as Lee and Gibson, have been used to propagate BPMV (1, 43, 72).

Physical Properties and Purification

The particles of BRMV and BPMV are polyhedral in shape and about 25-30 nm in diameter (1, 18, 30, 32). In ultrathin sections of bean leaves infected with strain A_1 of BRMV, large crystals appear which are formed by spherical units or particles, about 20 nm in diameter (5) and regularly spaced about 30 nm from the center. In tissues infected with strain A_2 of the

same virus, the small 20 nm particles are dispersed in the cytoplasm and probably represent the virions (32). In the cytoplasm of bean plant cells infected with BPMV, 25-28 nm particles have been observed dispersed or as crystals in the tubules and vacuoles. Such particles correspond in size to those observed in pure preparations (29, 30). BPMV also produces osmiophilic globules and myelinic bodies in the cytoplasm of infected cells (31).

The thermal inactivation point of BPMV is between 70° and 75°C, and of BRMV between 65° and 70°C. Both viruses have a final dilution point between 10^{-4} to 10^{-5}. BRMV remains infective in crude extracts for 48 to 96 hours at 22°C, and BPMV is infective for 62 days at 18°C (18, 72).

Both viruses can be purified using bean or soybean as propagation plants. Frozen leaves are thawed and homogenized in 5% K_2HPO_4. A solution of 0.01 M phosphate buffer at pH 7.0 is added to the extract, and the pulp is pressed through gauze and mixed with equal parts of n-butanol and chloroform. After 30 to 60 minutes, the emulsion is separated and the virus extract is subjected to differential centrifugation. The pellet is resuspended in the same buffer solution, and the virus is precipitated by adjusting the preparation to pH 5.0 with acetic acid (10%). The virus is resuspended in the same buffer and put through a second cycle of differential centrifugation and finally resuspended in 0.2N buffer at pH 7.0 (1, 18).

Alternatively, the virus may be precipitated with polyethylene glycol (4%) and NaCl at 0.3 M (R. Gámez, unpublished information), or the method used by Gálvez *et al.* (15, 16). Further purification is obtained by sucrose density gradient centrifugation. Three centrifugal components typical of the comovirus group are separated — the top component which lacks nucleic acid, a middle component and a bottom component composed of nucleoprotein (1). The middle and bottom components are infectious only when present in a mixture, since this group of viruses has a divided genome requiring both particles to be infective (59).

The isometric particles of BPMV have sedimentation coefficients of 54, 91 and 112 S for the top, middle and bottom centrifugal components, respectively (1). These properties and the molecular weight have not been reported yet for BRMV. The molecular weight of the BPMV nucleic acid is 1.9 and 2.4 x 10^6 daltons for the middle and bottom components, respectively (51).

Bean pod mottle virus contains single stranded ribonucleic acid, 38% of which is present in the middle component and 31% in the bottom component. The base composition of the RNA is guanine 20.0%, adenine 32.1%, cytosine 16.8% and uracil 31.1% (1, 51). These properties have not been determined for BRMV.

Transmission and Epidemiology

Comoviruses can be easily transmitted mechanically in the laboratory or glasshouse. BRMV and BPMV are disseminated in the field by insect vectors in the subfamily *Galerucinae* of the family *Chrysomelidae* (13). Bean rugose mosaic virus is transmitted by *Cerotoma ruficornis, Diabrotica balteata* (Fig. 2) and *D. adelpha* (6, 12, 18). Bean pod mottle

Fig. 2- Adult beetle of *Diabrotica balteata.*

virus is transmitted by *Cerotoma trifurcata, D. balteata, D. undecimpunctata, Epilachna varivestis, Colaspis flavida, C. lata* and *Epicanta vittata* (11, 13, 27, 43, 45, 48, 61).

Both viruses can be acquired by their vectors during feeding periods of less than 24 hours. As with many virus-vector associations, a high percentage of the insects transmit the virus for up to two days. The transmission rate then drops markedly although, occasionally, some insects can transmit the virus for longer periods (13, 50, 64). In the case of BRMV, *C. ruficornis* can transmit the virus for seven to nine days, but *D. balteata* and *D. adelpha* transmit it for only one to three days (6, 18).

At the same time, *E. varivestis* rarely transmits BPMV for more than one day, while *C. trifurcata* can transmit it for several days (11). Previously it had been assumed that transmission resulted from contamination of the beetle mouthparts. However, the transmission mechanism now is considered to be a more complex biological phenomenon which is not completely understood. The virus has been detected in the hemolymph, regurgitant, and feces of viruliferous beetles (12, 13, 50).

Bean rugose mosaic and bean pod mottle viruses are not seed-borne. No other information is available on the epidemiology of BRMV or BPMV. If transmission by seed does not exist, then it must be assumed that there are other hosts from which insects acquire the virus to transmit it to beans. The identity of such plants, as well as the ecological conditions which determine their survival, need to be studied (50).

Control

Populations of insect vectors can be controlled with insecticides (see Chapter 20 for specific recommendations).

Cultivars which react with local lesions are resistant, since damage from local infection under field conditions is not important. Numerous commercial bean cultivars are immune to one or both viruses (18, 19, 72). If the viruses become a limiting factor to bean production, it would not be difficult to incorporate resistance to systemic infection into commercial susceptible cultivars.

Genetic factors which determine immunity, local lesions, and systemic infection by BPMV and BRMV are similar. Inheritance is monogenic and governed by three alleles, the first of which is dominant over the other two and confers immunity to the virus. The second is dominant over the third and confers hypersensitivity, and the third determines susceptibility to systemic infection (36, 37, 55).

Bean Southern Mosaic Virus

Introduction

Bean southern mosaic virus (BSMV) was originally observed in Louisiana and has since been found in several states in southern and western United States (71, 74). In Latin America it has been observed in Mexico (70), Colombia, Costa Rica (44) and Brazil (7). This virus can reduce bean production (74). In Costa Rica, losses of 83-94% occurred under experimental conditions, while in Mexico, Brazil and Colombia its importance is considered to be moderate (7, 70).

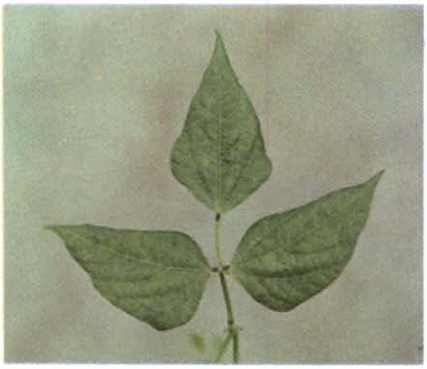
Bean southern mosaic virus infects *Phaseolus vulgaris, P. lunatus, P. acutifolius, P. coccineus, Trifolium alexandrinum, Cyamopsis* sp., *Melilotus indica,* soybeans and cowpeas. No species outside the legume family is reported to be susceptible (25, 53, 70).

Bean southern mosaic virus consists of a group of strains which are serologically related (52), the severe strain described in Mexico (70), the Ghana strain which infects beans and cowpeas (34), and the cowpea strain (25, 53) which does not infect beans.

The common name frequently used for bean southern mosaic virus in Latin America is mosaico sureño.

Symptomatology

Bean southern mosaic virus induces three major types of symptoms in bean cultivars. These are local lesions, mosaic or mottling, and systemic necrosis. The type of symptom and severity depend upon the cultivar, climatic conditions and virus strain. Local necrotic lesions which appear



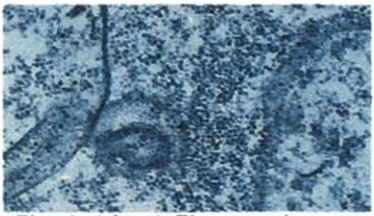


Fig. 4- (above) Electron microscope illustration of the isometric particles of bean southern mosaic virus (15,000 X).

Fig. 3- (left) Light chlorosis and leaf curling induced in leaves of the bean cultivar Diacol-Calima inoculated with BSMV.

two to three days after inoculation are dark reddish-brown and 1 to 3 mm in diameter. Their size depends upon the cultivar, leaf age and number of lesions produced per leaf.

Symptoms of systemic infection may resemble those induced by bean rugose mosaic or bean common mosaic viruses. The first symptoms of systemic infection consist of a mild mottling (Fig. 3) which may increase in severity during flowering. Vein banding, rugosity and deformation frequently occur. A reduction in plant size and severe leaf malformations commonly are observed in highly susceptible cultivars and with some virus strains (44, 70, 71, 74). Pod symptoms usually are severe, as they become distorted and acquire a dark green or mottled appearance (44, 74).

The bean cultivars Full Measure, Logan, Plentiful, US #5, Refugee, and Stringless Black Valentine are infected systemically by the typical strain and the severe strain from Mexico. The latter strain induces local lesions and systemic infection in other cultivars such as Blue Lake, Kentucky Wonder, Pinto U.I. 78 and Sutter Pink (25, 70, 74). Numerous cultivars which react with local lesions to the severe strain include Kentucky Wonder, Sutter Pink and Blanco 157 (25, 46, 70, 74). Bean cultivars susceptible to systemic infection and used to propagate the virus include Bountiful and Black Valentine. The cowpea cultivar Black Eye has been used to propagate the cowpea strain (9, 22, 25, 57).

Physical Properties and Purification

Bean southern mosaic virus has isometric particles (Fig. 4) which are 25-26 nm in diameter when observed under the electron microscope in purified preparations (25, 47). Viral particles 20.5 nm in diameter have been found in local lesions (10), while later studies described the existence of spherical particles (25-30 nm) in the cytoplasm and nucleus of plants with local lesions or systemic infection. The cowpea strain forms crystals in or near the vascular tissues, while the bean strain does not form true crystals (8, 69).

The thermal inactivation point is between 90° and 95°C, although there are slight variations within this range for different strains (53, 70, 71). The virus tolerates dilutions between 5 x 10^{-5} to 4 x 10^{-6}, depending upon the strain and test plant. The virus remains infective for 11 weeks under laboratory conditions and 32 weeks at 18°C (70, 71).

Different methods have been used to purify the virus. The initial extraction usually is done with a phosphate buffer at pH 7.5. The extract can be either heat-clarified at 60°C for 10 minutes before centrifugation at low velocity or treated with organic solvents before centrifugation. The preparations are subjected to various cycles of differential centrifugation. The virus can be further purified by precipitation by acidification to pH 5.0 with 0.1 N HCl or with ammonium sulfate (9, 25, 53, 57). Centrifugation in sucrose density gradients separates the virus as a single component with a sedimentation coefficient of 115 S (25, 41).

The molecular weight of the virus is 6.6 x 10^6d (41). The viral capsid is about 5.2 x 10^6d and the nucleic acid is approximately 1.4 x 10^6d (9, 22, 57). The ribonucleic acid of the virus is single-stranded and represents 21-23% of the virion. The composition of the bases is guanine 27.0%, adenine 23.5%, cytosine 22.5% and uracil 27.0% (22, 57). Immuno-diffusion in agar gels and other serological tests have been used to study relationships between virus strains which have been shown to be related but not serologically identical (25).

Transmission and Epidemiology

Bean southern mosaic virus has been reported to be transmitted in seed coats (38, 39). More recently, it has been detected in embryos (58). The cowpea strain also is seed-transmitted (53). The virus can be transmitted mechanically. Natural dissemination occurs by chrysomelid beetles (12, 13, 50, 64). The Coleoptera species, *Cerotoma trifurcata* and *Epilachna varivestis,* are vectors in the United States (11, 62, 63). *Diabrotica adelpha* was shown to transmit the virus in Costa Rica (44). The insects can acquire the virus after feeding on infected plants for less than 24 hours. *C. trifurcata* can retain the virus and transmit it for up to 19 days, although the percentage of insects transmitting the virus decreases after the second day (65). *E. varivestis* rarely transmits the virus for more than one day (11).

The virus also has been detected in the hemolymph of insect vectors (54). Previously, it was believed that the transmission mechanism of this virus, like those of other viruses in the comovirus group, consisted of a simple contamination of the buccal parts of the insect, but now it is believed to be a more complex biological phenomenon (12, 13).

Control

The use of insecticides or other methods to control beetles should be an effective measure, although such practices have not been proven experimentally. Planting BSMV-free seed should be an adequate control method.

Although the majority of bean cultivars are not immune to all strains of the virus, those which exhibit local lesions can be considered to be commercially resistant. Resistant cultivars include Kentucky Wonder, Blue Lake, Decatur and Great Northern No. 15, 59 and 123 (74). The Mexican cultivar Blanco 157 is also hypersensitive (70).

Bean Yellow Stipple Virus

Introduction

Bean yellow stipple virus (BYSV) was first isolated in Illinois in 1948 (73) and later in Costa Rica in 1972 (20, 21). There are no studies on the economic importance of BYSV in beans.

Only species belonging to the legumes have been reported as susceptible to systemic infection by BYSV. Susceptible plants include *Phaseolus vulgaris, P. acutifolius, P. lunatus, P. calcaratus, P. riccardianus, P. aconitifolius, P. lathyroides, Vigna sinensis, V. sesquipedalis, V. hirta, Glycine max, G. javanica,* and *Cajanus indicus* (21, 33, 60). In other studies, *Cyamopsis tetragonoloba, Phaseolus mungo,* and *Pisum sativum* also were susceptible (73).

The common name frequently used for bean yellow stipple virus in Latin America is moteado amarillo.

Symptomatology

Only systemic infection has been observed in bean cultivars inoculated with BYSV. Infected plants show initial symptoms of very light yellow stippling and, later, small yellow spots on the trifoliate leaves. These may coalesce to form spots or yellow areas with well-defined borders and an irregular shape. The spots decrease in intensity and number on the new leaves formed at flowering. Slight variations in severity occur depending upon the cultivar, time of infection and climatic conditions. Some cultivars also exhibit slight growth reduction. In general, the infected plants do not show malformation, rugosity, or mosaics commonly associated with other bean viruses (20, 21, 73).

Bean cultivars susceptible to BYSV include Stringless Green Refugee, Pinto 111, Bountiful, Michelite, Sanilac, Top Crop, Tender Crop, Tender White, Tender Green, Great Northern U.I. 60, Kentucky Wonder and Tender Long. The cowpea cultivar Black Eye also is susceptible. Several species of legumes which react to the virus with local necrotic lesions include *Dolichos lablab, Glycine max, Crotalaria juncea* and *C. paulina. Dolichos lablab* has been used in studies on virus infectivity. *Chenopodium amaranticolor* and *C. album* react with whitish local lesions. The bean cultivars Col. 109-R and Pinto U.I. 78 have been used to multiply the virus (21, 73).

Physical Properties and Purification

Bean yellow stipple virus is a member of the bromovirus group (26, 35). Typical of the bromoviruses, BYSV has isometric particles 26-30 nm in diameter (20, 21). In infected beans and cowpeas, BYSV produces amorphous inclusions, filamentous inclusions and membranous vesicles (30-100 nm in diameter) which contain virus particles (28). The virus has a thermal inactivation point of 76°C, a dilution end point between $1\text{-}5 \times 10^{-4}$, and a longevity *in vitro* of five days at 18°C, and one day at 20°C (21, 73).

The virus can be purified by homogenizing 100 g of tissue in 100 ml of 0.01 M phosphate buffer at pH 7.0, then pressing the pulp through gauze, and mixing the extract with equal parts of chloroform and n-butanol. After one hour at 4°C, the emulsion is broken by low speed centrifugation, and the virus in the aqueous phase then is subjected to two cycles of differential centrifugation. The virus pellet is resuspended in the same buffer and eventually centrifuged in a sucrose density gradient (17). The virus is separated as a single band or centrifugal component which has a sedimentation coefficient of 81 S (3, 14).

The molecular weight and chemical composition of BYSV has not been determined. Cowpea chlorotic mottle virus has a molecular weight of 4.6×10^6d and contains 24% ribonucleic acid in a single strand, with a composition of guanine 26.4%, adenine 25.3%, cytosine 20.3% and uracil 28.0% (2, 4).

The viruses of the brome mosaic virus group are serologically related. The serological reactions were determined by gel diffusion and showed differences among strains. The known strains include the type cowpea strain, a strain from Arkansas, and the yellow stipple of beans from Costa Rica (13, 14). Brome mosaic virus, the type member of the bromovirus group, is related serologically to the yellow stipple virus of cowpea (49).

Transmission and Epidemiology

Bean yellow stipple virus is not seed transmitted (21, 74) but is easily transmitted mechanically. Dissemination appears to occur principally by beetles such as *Cerotoma ruficornis* and *Diabrotica balteata* in Central America. Virus acquisition by the vector can occur in less than 24 hours. *C. ruficornis* can retain the virus from three to six days but *D. balteata* for only one to three days. As with other groups of viruses which are transmitted by Coleoptera insects, the transmission percentage decreases rapidly during the third day after virus acquisition (21). The mechanism of transmission of the virus apparently is similar to that of bean rugose mosaic virus and bean southern mosaic virus (13, 50, 64). The cowpea chlorotic mottle virus also is transmitted by *C. trifurcata, D. undecimpunctata* (65), and *E. varivestis* (J.P. Fulton, unpublished information).

No information is available on the epidemiology of this virus in bean fields. Preliminary studies carried out with cowpeas in Costa Rica have shown that ecological conditions related to the season of the year and systems of production affect vector populations and subsequent virus incidence (23).

Control

No information is available on methods of control for this virus in beans. All cultivars of beans tested experimentally have been susceptible (21, 73). The apparent natural incidence is low, and perhaps the virus does not severely affect production. Control of insect vectors could constitute an effective method to reduce virus incidence in the event that it should cause important economic problems.

Bean Curly Dwarf Mosaic and Bean Mild Mosaic Viruses

Introduction

Bean curly dwarf mosaic virus (BCDMV) and bean mild mosaic virus (BMMV) were isolated from beans in El Salvador in 1971 (40, 68). No estimates of yield losses caused by either virus are available. However, BCDMV reportedly occurred in 1-15% of plants growing in different parts of bean fields in El Salvador (40).

The host range of BCDMV includes *Phaseolus vulgaris, P. acutifolius, P. lunatus, Cajanus cajan, Cicer arietinum, Crotalaria juncea, Glycine max, Lathyrus sativus, Lens culinaris, Macroptilium lathyroides, Pisum sativum, Sesbania exaltata, Vicia faba* and *Vigna radiata* (40). The host

range of BMMV includes *Phaseolus vulgaris, P. acutifolius, Dolichos lablab, Macroptilium lathyroides, Glycine max, Canavalia ensiformis, Sesbania exaltata, Gomphrena globosa* and *Chenopodium quinoa* (68). Hosts expressed a range of symptoms after inoculation with either virus including systemic infection with or without symptom expression (its presence was detected serologically) and top necrosis (40, 68).

The common name frequently used for BCDMV in Latin America is mosaico y enanismo rizado del frijol. The literal translation of BMMV would be virus del mosaico suave del frijol.

BCDMV is serologically related to Quail Pea Mosaic Virus (QPMV) and Squash Mosaic Virus but is not related serologically to BRMV or BMMV (40). BMMV does not belong to any of the five serogroups in the comoviruses (68). However, both BMMV and BCDMV are transmitted by beetles.

Symptomatology

BCDMV induces a wide range of symptoms with varying degrees of severity, depending upon the cultivar (Fig. 5) and stage of plant development. Symptoms may resemble those induced by bean rugose mosaic virus. Plants infected by BCDMV at an early stage of development are extremely stunted and produce no yield. Older plants which become infected are less severely affected and produce limited yields. Symptoms may be observed only in the terminal growth of some cultivars with an indeterminate growth habit. Symptoms include mosaic, rugose, curling and twisting of leaves and plant dwarfing. The virus may cause chlorotic and/or necrotic local lesions, vein necrosis, top necrosis and death, depending upon the cultivar (40).

BMMV may produce a barely discernible mild mosaic (Fig. 6), slight vein-banding, roughening of the leaf surface or no visible symptoms. Chlorotic local lesions may form on inoculated primary leaves but appear to depend upon unspecified environmental conditions. BMMV does not stunt plant growth or cause severe leaf deformations. BCDMV can occur in combination with BMMV (Fig. 7) under field conditions in El Salvador and can incite greater damage to certain cultivars than BCDMV infection only (68).

Physical Properties and Purification

BCDMV may be extracted from freshly harvested leaves and concentrated by centrifugation. The virus pellet is then resuspended and clarified with activated charcoal before the next centrifugation at 8000 g for five minutes. The nearly colorless supernatant containing the virus is

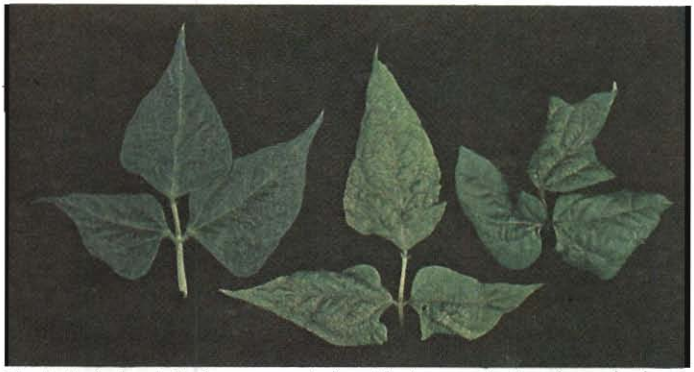


Fig. 5- Variation in leaf symptoms induced by bean curly dwarf mosaic virus infection of bean cultivars 27-R, Porrillo No. 1 and El Salvador 184 (left to right).

further purified by density gradient (5-30% sucrose) centrifugation and separated into three viral components.

The BCDMV particles are 23-25 nm in diameter and infectious in dilutions up to 1 x 10^{-5} in 0.025 M phosphate buffer. Dilutions still are infectious after incubation at room temperature for three weeks or heating at 50°C for 10 minutes (40).

BMMV may be extracted from freshly harvested leaves by blending in two to three volumes (w/v) of 0.02M sodium citrate buffer at pH 7.5 containing 0.02M 2-mercaptoethanol. Cold chloroform (20 ml/100 g tissue) is added to the homogenate before centrifugation at 1000 g for 10 minutes. The clear yellow supernatant containing the virus then is

Fig. 6- (above) Leaf symptoms induced by bean mild mottle virus infection of the bean cultivar Porrillo No. 1.

Fig. 7- (right) Plant and leaf symptoms induced in the bean cultivar Porrillo No. 1 by mixed inoculation with BCDMV and BMMV

concentrated by centrifugation at 105,000 g for 1.5 h; or by precipitation with 10% (w/v) polyethylene glycol 6000 before centrifugation at 12,000 g for 30-60 minutes. Virus pellets then are resuspended in 0.02 M citrate buffer for 4-24 h before centrifugation at 8000 g to remove plant materials. The virus is further purified on 10-40% linear sucrose gradients in 0.02 M neutral citrate before centrifuging in a swinging bucket rotor at 100,000 g for two hours. Gradients then are fractionated into a single viral component and subjected to dialysis to remove most of the sucrose. The virus then is reconcentrated by high speed centrifugation.

The BMMV particles are 28 nm in diameter and infectious in dilutions up to 1×10^{-8} in 0.25 M phosphate buffer even after incubation at room temperature for six weeks. Polyacrylamide gel electrophoresis determined that BMMV-RNA had a molecular weight of 1.27×10^{6}d. The base ratio was determined to be guanine 21.7%, adenine 25.8%, cytosine 31.5% and uracil 21.0% (68).

Transmission and Epidemiology

BCDMV and BMMV may be transmitted by the spotted cucumber beetle (*Diabrotica undecimpunctata howardi* Barber) and the Mexican bean beetle (*Epilachna varivestis* Mulsant). The banded cucumber beetle (*D. balteata* Le Conte) and a flea beetle (*Cerotoma ruficornis* Oliver) are suspected to be natural vectors of both viruses in El Salvador (40, 68). The spotted cucumber beetle and Mexican bean beetle retained BCDMV infectivity for two and three days, respectively, after a 24-hour accession feeding (40). These vectors retained particles of BMMV and were infectious for 21-40 hours after a 19-hour accession feeding (68). Both viruses are transmitted mechanically (40, 68). BCDMV was not found to be seed transmitted (40). Seed transmission studies by BMMV have not been reported.

Studies in El Salvador suggest that insect vectors transmit the viruses to beans from infected wild plant species growing on the edge of fields, since the incidence of virus-infected plants is less in the center of bean fields than in the outer edges (40). BMMV commonly occurs in mixture with BCDMV. Its economic importance may depend on the combined infection with other viruses (68).

No control measures are reported for bean curly dwarf mosaic and bean mild mosaic viruses.

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Chapter 14

Whitefly-Transmitted Viruses

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Chapter 14

Whitefly-Transmitted Viruses

General Introduction

Whiteflies belong to the order Homoptera, family Aleyrodidae, and are currently reported to transmit 28 different plant viruses of beans and other crops (71, 120). Whitefly species reported to be vectors of plant viruses include *Bemisia tabaci* Gennadius *(=B. inconspicua* Quaintance), *B. lonicerae, B. manihotis* Frappa, *B. tuberculata* Bandar, *B. vayassieri* Frappa, *Aleurotrachelus socialis* Bondar, *Aleurothrixus floccosus* Mask, *Trialeurodes abutilonea* Haldeman, *T. natalensis* Corb. and *T. vaporariorum* Westwood (13, 32, 36, 91, 106). Whitefly populations are commonly restricted to tropical zones below 1300m, where they are capable of transmitting viruses to various plant species (13, 32, 36, 61, 68, 95, 102, 119, 120).

Bemisia tabaci is the most common whitefly vector of bean viruses and is variable in its feeding habits and reproduction rates on different plant species. Flores and Silberschmidt (56) and Russell (107) characterize this variation as biotypes. However, Bird (9, 10, 11, 14) denotes the variation as races, *B. tabaci* race *jatrophae* and *B. tabaci* race *sidae*.

The virus diseases transmitted by whiteflies *(B. tabaci)* are grouped into two main types by Costa (52) according to their symptomatology. These types are mosaic and leaf curl.

A green, or more frequently yellow, mosaic of foliage is the most conspicuous symptom in the mosaic group. Yellowing may appear along the veins and develop into a yellow net or be limited by the veins. Curling or crinkling of the foliage may occur due to the abnormal or unequal growth of healthy and infected mosaic areas of the leaf. As the foliage matures, the mosaic tends to become less apparent, and for certain diseases, such as cotton common mosaic, the yellow areas may turn reddish late in the season (28). In the case of *Malva parviflora* infected with the disease agent from *Abutilon thompsonii,* the initial mosaic is followed by witches' broom symptoms (58). The characteristic yellow or golden color of infected plants is easy to distinguish from healthy plants in a field.

In the case of leaf curl, infected plants do not exhibit clear mosaic symptoms but may show a diffused yellowing of leaves and vein clearing which may be easily overlooked. The characteristic symptom caused by this group is the stunting of infected plants, curling, enation, and vein thickening of foliage.

Costa (36) recently included a third group of whitefly-transmitted viruses which produces yellowing symptoms to distinguish from similar symptoms induced by aphid-transmitted viruses or nutritional disorders. Yellowing symptoms induced by whitefly-transmitted viruses commonly appear only later during plant development.

Symptomatological differences suggest that the first group of viruses occurs in parenchymatous tissue and the second group occurs in phloem vessels (32). However, some diseases may induce symptoms of the first group in some hosts and symptoms of the second group in other hosts. For example, the disease agent from infected *Rhynchosia minima* induces a bright yellow mosaic symptom on *Rhyncosia minima* but induces leaf curl and enation on tobacco (11). Duffus (54) also mentions two major groups of whitefly-transmitted viruses identified as variegation-producing and plant malformation-producing types.

Very few whitefly-transmitted diseases have been isolated and proven to have a viral etiology. The previously mentioned groups of viral diseases have been based upon arbitrary classifications due to similarities in symptomatology and presumed insect vectors. Bird *et al.* (20) suggested that these whitefly-transmitted viruses with unknown or incomplete etiology be placed in one group, rugaceous diseases, instead of different groups primarily distinguished only by symptomatology. Much organized and collaborative research is required to characterize these whitefly-transmitted viruses and establish their true relationships.

The following viruses of beans and other plant species have been demonstrated to be whitefly-transmitted, many however, only under research conditions. These viruses are grouped in order of their decreasing economic importance: a) bean golden mosaic; b) bean chlorotic mottle, abutilon mosaic, yellow dwarf mosaic, infectious chlorosis of Malvaceae; c) euphorbia mosaic; d) rhynchosia mosaic; e) jatropha mosaic; f) jacquemontia mosaic; g) ipomoea or merremia mosaic; and h) mung bean yellow mosaic.

The following sections of this chapter will review the geographical distribution, economic importance, host range, symptomatology, physical properties, transmission, epidemiology and control measures reported for these viruses.

Bean Golden Mosaic Virus

Introduction

Bean golden mosaic virus (BGMV) was first reported in Latin America in 1961 (31), at which time it was considered to be a minor disease in Sao Paulo, Brazil. It has since occurred in practically every major bean production area in Brazil, including Minas Gerais, Parana, Bahia, Pernambuco, Ceara, Para, the Amazon, and the Valle del Río Sao Francisco (33, 44, 121). BGMV has been reported in many other bean production regions of Latin America, such as El Salvador (66, 67, 126, 127), Guatemala, Nicaragua, Costa Rica, Panama (66, 67), Puerto Rico (12, 17, 21), Jamaica, Dominican Republic (1, 2, 101, 102, 108), Colombia (63), Cuba (23), Belize, Mexico, Honduras and Venezuela (Gálvez, personal observations).

Identification and nomenclature of BGMV has been quite diverse and must be standardized between workers in different regions, since BGMV-like symptoms have been called BGMV, bean yellow mottle, bean golden yellow mosaic, bean yellow mosaic and bean double yellow mosaic (12, 17, 21, 46, 47, 48, 108, 126, 127). Gálvez *et al.* (64) utilized serology, electron microscopy and density gradient centrifugation to prove that isolates inducing similar disease symptoms in Mexico, Guatemala, El Salvador, Colombia, Cuba, Puerto Rico, Dominican Republic, Brazil and Nigeria all were bean golden mosaic virus. This relationship between isolates also should be clarified by utilization of the BGMV antisera developed by Goodman (75) from isolates collected in Puerto Rico.

Bean golden mosaic virus is an economically important disease, especially in regions of Latin America such as Brazil and parts of Central America and the Caribbean. Brazilian bean production has been reduced greatly by the virus since 1972, and its seriousness has been attributed to the increasing whitefly populations associated with the expanded production of soybeans in bean growing areas (33, 44, 121). Gámez (66, 67, 70) considers BGMV to be the principal bean disease in the Pacific coastal plains of El Salvador, where disease incidence frequently reaches 100%.

Various workers (42, 69, 101, 102) report that infection by BGMV reduces the number of pods, number of seeds per pod and seed weight. Reported yield losses consist of 57% in Jamaica (101, 102), 48-85% in Brazil (42, 90), 40-100% in Guatemala (96), and 52-100% in El Salvador (Cortez and Diaz, personal correspondence). Yield losses vary greatly depending upon plant age at the time of infection, varietal differences and possibly viral strains (33, 61).

The host range of BGMV includes *Phaseolus vulgaris, P. lunatus, P. acutifolius, P. polystachios, P. longepedunculatus, P. aborigeneus, P. coccineus, Desmodium occuleatum, Macroptilus lathyroides, Terramnus urcinatus, Vigna radiata, V. unguiculata* and *Calopogonium muconoides* (2, 4, 12, 13, 20, 21, 27, 31, 33, 34, 35, 36, 51, 57, 68, 79, 102, 122, 124).

Common names frequently used for bean golden mosaic virus in Latin America include mosaico dorado del fríjol, moteado amarillo del fríjol and mosaico dourado do feijoeiro.

Symptomatology

Symptoms of BGMV are readily visible in infected bean plants which exhibit a brilliant yellow or golden color of leaves (Fig. 1). Symptoms may appear in the primary leaves within 14 days after planting if high populations of whiteflies are present in or near the field. Bird *et al.* (20, 21) observed the presence of small yellow spots, sometimes apparent as star-shaped lesions, near the leaf veins three to four days after exposure to viruliferous whiteflies.

The primary systemic symptoms of BGMV infection are apparent as rolling of the lower leaf surface of young leaves, which later exhibit a range of mosaic symptoms (Fig. 2). These symptoms are predominant near the veins and within the leaf parenchymatous tissue, where an intense and often brilliant yellowing develops. Susceptible cultivars exhibit a marked rugosity and rolling of leaves, many of which may be completely yellowed or occasionally white to nearly bleached. Tolerant cultivars often present symptoms with less intense leaf mosaics and may exhibit some plant recuperation at a later stage of development.

Most cultivars do not show a reduction of leaf size (33). When the infection occurs during the seedling stage, susceptible plants may become stunted. Pods of infected plants may exhibit mosaic spots or be malformed (Fig. 3). Seeds may be discolored, malformed, and reduced in size and weight (24, 66, 67).

The symptomatology of BGMV appears to be similar to that reported for lima bean golden mosaic virus in Africa (122) and lima bean yellow mosaic in India; but the latter differs in its host range (95, 105). Mung bean yellow mosaic, urd bean yellow mosaic viruses and yellow mosaic of *Dolichos lablab* likewise are not able to infect the majority of *Phaseolus vulgaris* cultivars (104). However, these viruses appear to have a similar symptomatology on their respective hosts as does BGMV in beans (92, 93, 95, 104, 128).

Fig. 1- Symptoms induced by bean golden mosaic virus in beans.

Electron microscopy evaluations of infected bean tissue reveal that the principal cellular symptom is evident as a dramatic change in chloroplast morphology, particularly in the lamellar system (81). Recently Kim *et al.* (80) reported that the symptoms are limited to the phloem tissue and cells adjacent to the parenchyma tissue. Virus-like particles appear as packed hexagonal crystal arrangements or as loose aggregates in the nuclei of infected cells. Distinct changes in the nucleoli also are evident, since there is a segregation of granular complexes and fibrils which may occupy 75% of the nuclear volume (76).

Physical Properties

Bean golden mosaic virus has been classified as a viral disease because of its characteristic transmission by insects, symptomatology and mode of dissemination in the field (21, 31, 68, 85, 101). However, its viral etiology was not completed until its isolation was accomplished in 1975 by Gálvez and Castaño (62). They observed that fixed BGMV has a specific form which consists of icosahedral particles united in pairs (identical dimer particles or geminates). The bonded particles are flattened at their point of

Fig. 2- Mosaic symptoms and leaf malformation induced by BGMV infection.

Fig. 3- Pod malformation caused by BGMV infection of a susceptible bean cultivar.

union (Fig. 4) and measure 19 x 32 nm, while individual particles have a diameter of 15-20 nm. Matyis *et al.* (87) reported individual particles measured 12-13 nm in diameter. A similar particle morphology was found for the viruses causing tomato golden mosaic, euphorbia mosaic (86, 87) as well as BGMV of beans in Brazil, Colombia, El Salvador, Dominican Republic, Guatemala, Mexico, and BGMV of *P. lunatus* from Nigeria (64).

Goodman *et al.* (77) could not determine whether these geminate particles actually were the infectious entities or artifacts of fixation. However, Gálvez and co-workers (24, 62) could observe particles in unfixed preparations, and they gave the highest infectivity. When the BGMV particles were disassociated with EDTA at high molarity (0. 1M), infectivity was almost completely lost.

BGMV particles have a thermal inactivation point of 50°C (18, 19) to 55°C (62), a final dilution end-point of 10^{-1} (62) to 10^{-2} (18, 19), and an *in vitro* longevity of 48 hours at room temperature (62). Goodman and co-workers (76, 77) determined that the particles have a sedimentation coefficient value of 69 S, a molecular weight of 2.6 x 10^6 daltons, a 260 nm absorbance value of 7.7 and a 260/280 absorbance ratio of 1.4. The genome of BGMV contains DNA which has a sedimentation coefficient of 16 S, a molecular weight of 0.75 x 10^6 daltons, and composes 29% of the particle (24, 25, 72, 73, 76). Two protein components, of molecular weight 3.8 x 10^4 and 5.5 x 10^4 daltons, were isolated by Cárdenas and Gálvez (24, 25). The DNA is single stranded and resistant to exonucleases (24, 74). It has a buoyant density of 1.717 g/ml in cesium chloride and is resolved into two components during polyacrylamide gel electrophoresis in 8 M urea (74, 77).

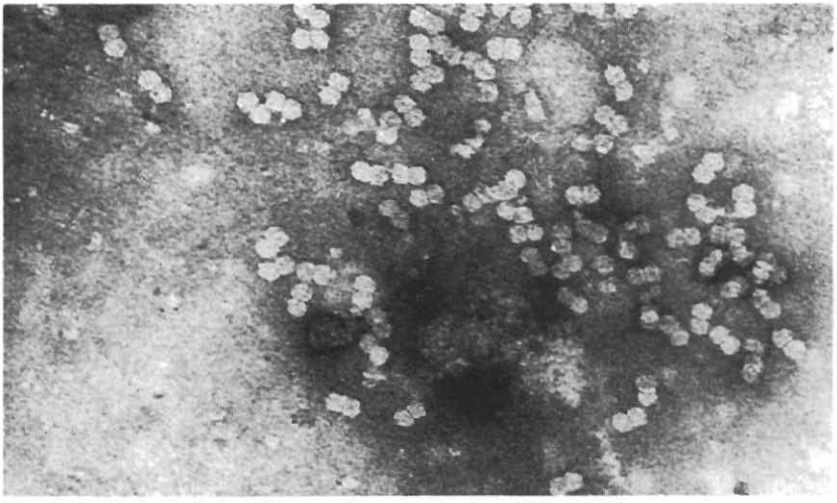

Fig. 4- Geminate particles of bean golden mosaic virus (160,000 X).

Francki and Bock (60) have included BGMV in a new virus group called the Geminivirus, based upon its particle characterization, physical-chemical properties and single-stranded DNA.

Transmission and Epidemiology

BGMV can be transmitted naturally by whiteflies and artificially by mechanical inoculation. Other whitefly-transmitted plant viruses such as euphorbia mosaic, abutilon mosaic and sweet potato virus B also have been transmitted mechanically (32, 36). However, Meiners *et al.* (88) were the first workers to mechanically transmit BGMV to beans. Successful inoculation required a high temperature of 30°C, and a 30% transmission rate was obtained at 24° - 28°C. No transmission occurred below 21°C. Bird and co-workers (16, 19) originally obtained only a 4% transmission but have since improved this efficiency.

Gálvez and Castaño (62) obtained nearly 100% transmission under glasshouse conditions at 25°C with BGMV inoculum extracted from plants infected 21 days earlier in a 0.1 M phosphate buffer at pH 7.5 and 1% 2-mercaptoethanol. Transmission was significantly reduced or zero if inoculum was extracted from plants infected after 21 days. Bird *et al.* (19) utilized a similar buffer at pH 7.0 to obtain 100% transmission by inoculation with an airbrush at 80 lb/in^2. Matyis *et al.* (87) were not able to transmit BGMV isolates mechanically in Brazil, which may reflect differences in methodology or strains. Some strains of BGMV may be transmissible only by the whitefly vector (36, 41, 76).

BGMV has not been shown to be transmissible in seed from infected bean plants. Pierre (102) tested seed from 300 infected bean plants, and Costa (31, 33, 34, 36) tested seed from 350 infected lima bean plants. None of these seeds was found to be infected by BGMV.

The principal mode of BGMV transmission, especially under field conditions, occurs from the whitefly vector, *Bemisia tabaci*. Whiteflies are able to extract plant sap, but the principal threat to crop productivity is their ability to transmit plant viruses. Costa (32) stated that the whitefly is able to transmit viruses to more than 16 plant species, including cultivated and non-cultivated plants.

Nene (94) has studied the biology of whiteflies in relation to legumes such as *Phaseolus aureus, Vigna mungo* and *Glycine max*. The insect can produce 15 generations a year, during which time populations may be restricted to a single crop species or migrate to a variety of plant species. A

whitefly may lay 38-106 eggs (Fig. 5) during its life cycle, which requires 13-20 days during March to October or 24-72 days during November to March in India. Populations of whiteflies are reduced as the mung bean crop matures. These populations then may migrate to other plants such as crucifers, lentils and peas.

The life cycle on cotton in India (107) varies from 14-107 days, is shortest during April to September (14-21 days), and is longer during November to February (69-72 days). The maximum oviposition occurred at temperatures greater than 26.5°C, and no oviposition occurred at temperatures below 24°C.

Adults of *B. tabaci* are able to transmit BGMV in a circulative manner. There is no evidence of transovarial transmission or virus multiplication within the whitefly (32, 36, 95).

Costa (32) states that whitefly-transmitted viruses are not acquired as rapidly as aphid-transmitted viruses. Inoculation efficiency increases more because of longer acquisition periods than because of differences in virus infectivity. Whitefly-transmitted viruses have a defined but shorter incubation period, and particles are retained for more than 20 days in the insect vector. Whitefly adults can acquire and transmit BGMV within 5 minutes (7, 21, 68), and the inoculation efficiency is increased as population size is increased per infected plant (7, 13, 32, 36, 68, 120). Gámez (68) found an average acquisition and incubation period of three hours each. The retention period varies according to the acquisition period but may reach 21 days or the entire life of the whitefly (7, 20, 32, 36, 68, 120). The insects occasionally have been observed to lose their capacity for transmission (68).

Immature forms (Fig. 6) are able to acquire mung bean yellow mosaic virus which persists during pupation and can be transmitted during the

Fig. 5- Eggs and immature forms of *Bemisia tabaci* on the lower leaf surface.

Fig. 6- Immature forms of *Bemisia tabaci*.

Fig. 7- The adult whitefly (*Bemisia tabaci*) vector of BGMV.

adult stage. At least 50% transmission has occurred from adults (Fig. 7) obtained from immature forms which had previously fed on infected plants (95, 105). Costa (35) reported that female whiteflies were more efficient than males as vectors of BGMV to *Phaseolus vulgaris, P. acutifolius* and *P. polystachios*. However, males were more efficient vectors on *P. lunatus* and *P. longepedunculatus*.

BGMV is not seed-transmitted and, therefore, probably exists in many regions in plant reservoirs such as lima beans and other susceptible legumes including voluntary and cultivated beans, and weeds (34, 36, 51, 52, 61, 68, 102). Pierre (102) considers that lima beans and *Macroptilium lathyroides* are natural hosts for BGMV in Jamaica, in addition to poinsettias *(Euphorbia pulcherrima)*. Increased production of soybeans has increased whitefly populations and BGMV incidence greatly in beans planted in Parana and Sao Paulo, Brazil (33, 44, 121). Tobacco, tomato and cotton plantings in El Salvador and Guatemala are responsible for the high whitefly populations in those countries (5, 6, 27, 52, 61, 78).

Bean golden mosaic virus is more prevalent in lower to intermediate elevations (13, 33), normally below 2000 m where whitefly populations, temperatures and inoculum sources are greater. BGMV incidence is less during November to March when temperatures and insect vector populations are lower in Jamaica, Cuba and the Dominican Republic. BGMV is more common and severe in Brazil at elevations between 400-800 m and near the end of the summer or dry period (January to February) when whiteflies migrate from other maturing crops, such as soybeans, to the young bean plantings. Whitefly populations decline rapidly during cooler periods of the year, when temperatures are unfavorable to the whitefly and when fewer susceptible crops exist (31, 33).

Control by Cultural Practices

The incidence of BGMV in a bean production region can be reduced by eliminating alternative plant reservoirs of inoculum such as volunteer plants of *Phaseolus vulgaris, P. lunatus, P. longepedunculatus, Calopogonium* sp. and other plant species. Crop rotation and distribution within a production region also are important. BGMV incidence is

increased greatly by planting beans near fields of soybeans which, although not susceptible to BGMV, are favorable for whitefly populations which may encounter and transmit BGMV from infected plants, such as *Sida* spp. and other hosts, to developing bean crops (33, 102). BGMV infection of beans can therefore be reduced by not planting beans near fields of other crops such as soybeans, tomatoes, tobacco and cotton, which favor the build-up of whitefly populations.

Date of planting should be varied, if possible, so that young bean plants develop during periods of lower temperature and higher moisture which are less favorable to the whitefly and its ability to transmit BGMV (5, 6, 23, 31, 32, 33, 36, 44, 70, 78, 102).

No economical and practical biological control measures are currently available (95, 109). Plant mulches have been shown to reduce whitefly populations (8), possibly due to altered air temperature near the plants.

Control by Chemicals

The whitefly vector can be controlled by applying insecticides to economically reduce the population size and incidence of BGMV tranmission to susceptible cultivars. Various insecticides are effective against whiteflies *(Bemisia tabaci* and *Trialeurodes vaporariorum).* These include Tamaron 600E (1 lt/ha), Nuvacron 60 (0.5 lt/ha), Folimat 1000 (0.5 lt/ha), Bux 360 and Thiodan 35 or Endosulfan (1.5 lt/ha) (50). Populations of whiteflies were reduced effectively in El Salvador by applying Tamaron 600 (1 lt/ha) every seven days during the first 30 days after plant emergence (53, 82, 83). Alonso (6) reported that Nutasystox R-25 (1 lt/ha), followed by Nuvacron 50 (1.5 lt/ha) and Folimat 80 (0.33 lt/ha), effectively controlled whiteflies when applied 15 and 30 days after planting.

Systemic insecticides, such as Furadan and Thimet, effectively control whitefly populations when applied at planting (6). Substantial yield increases were obtained in the Dominican Republic by applying Carbofuran (Furadan 5G) (2.5 g/m row) at planting followed by 0.15% Monocrotophos (Azodrin 60E) applied at six, 15 and 30 days after plant emergence (3, 89, 99, 100). Nene (94) obtained effective control of whiteflies in India with a mixture of (a) 0.1% Thiodan, 0.1% Metasystox and 2% mineral oil, and a mixture of (b) 0.1% Malathion, 0.1% Metasystox and 2% mineral oil. He observed that the mineral oil acted as an ovacide.

Chemical control of insect vectors can be effective and economical in areas with moderate to low disease pressure and whitefly populations. However, its effectiveness can be reduced in regions where high numbers of

viruliferous vectors migrate continuously from other infected plant species. Therefore, chemical control may have to be combined with other control measures, such as plant resistance, to achieve a higher level of protection.

Control by Plant Resistance

Plant resistance can provide an economical method of disease control. Workers have evaluated more than 10,000 accessions of *Phaseolus vulgaris*, and some accessions of *P. lunatus*, *P. acutifolius*, and *P. coccineus* under field and laboratory conditions, but they have not found any source of high resistance or immunity to BGMV (24, 26, 27, 31, 33, 43, 61, 66, 67, 68, 102, 124). However, some accessions have exhibited a low to moderate level of resistance or tolerance, including Porrillo 1 and 70, Turrialba 1, ICA-Pijao, ICA-Tuí, Venezuela 36 and 40, Puebla 441, Guatemala 388 and 417, and CIAT G-651, -716, -729, -738, -843, -951, -1018, -1069, -1080, -1157, and -1257. Various *P. coccineus* accessions from the ICTA germplasm bank are resistant in Guatemala. They include Guat. -1278, -1279, -1288, -1291, -1296, -1299, M7689A and M7719 (24, 26, 27, 79, 124, 125).

Pompeu and Kranz (103) observed field tolerance in Aete-1/37, Aete-1/38, Aete-1/40 (Bico de Ouro types), Rosinha GZ/69, Carioca 99 and Preto 143/106. Rio Tabagi and Goianio Precoce are tolerant in Capinopolis, Brazil (Rava, personal communication). Tulmann-Neto *et al.* (116, 117, 118) obtained a tolerant mutant, TDM-1, by treating seed of Carioca with 0.48% ethyl methanol sulfonate for six hours at 20°C. TDM-1 has a level of tolerance similar to that of Turrialba 1, but it is not as agronomically acceptable.

The tolerance of Turrialba 1, Porrillo 1, ICA-Tuí and ICA-Pijao has been confirmed in Guatemala, El Salvador, the Dominican Republic, Brazil and Nigeria under high disease pressure in bean nurseries interplanted between tomatoes, tobacco, cotton, and soybeans to favor high whitefly populations (Fig. 8). Glasshouse inoculations and subsequent

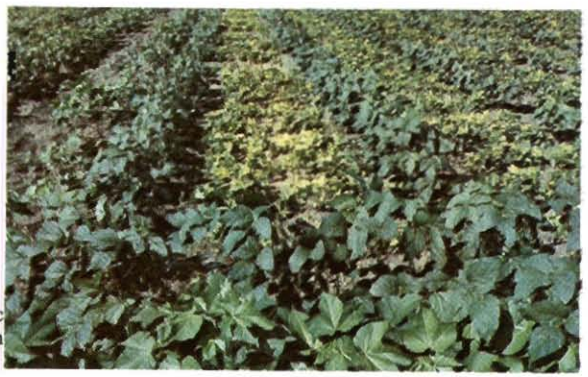

Fig. 8- Bean golden mosaic virus screening nursery in the Dominican Republic.

laboratory analyses revealed that these tolerant materials contained lower virus concentrations than highly susceptible accessions (24, 26, 27).

These tolerant materials have been utilized in breeding programs, and initial progenies appear promising (65, 129). Some progenies are highly tolerant to BGMV and produce 1,500 kg/ha under high disease pressure, as compared to yields of 1,000 (ICA-Pijao) and 650 (Turrialba 1) kg/ha for the progenitors. These progenies can produce 3,000 kg/ha in conditions where the virus is not a limiting factor to production.

Bean golden mosaic virus and its whitefly vector are able to survive on and infect various plant species, including beans. Integrated control measures can effectively reduce the incidence and severity of BGMV. These measures should consist of reducing vector populations by chemicals, eliminating alternative hosts, and using different planting dates combined with the development of agronomically acceptable cultivars with improved levels of tolerance or resistance.

Bean Chlorotic Mottle Virus

Introduction

Bean chlorotic mottle virus (BClMV), abutilon mosaic virus (AbMV), yellow dwarf mosaic virus and infectious chlorosis of Malvaceae have a similar symptomatology and are considered as a group in this section. Additional research is required to fully characterize these viruses to determine whether or not they are identical.

These viruses reportedly are widespread throughout Latin America, wherever the whitefly vector exists (4, 10, 12, 13, 14, 15, 16, 36, 38, 45, 78). They have been observed in Colombia, Mexico, Guatemala, El Salvador, Costa Rica, Cuba, Dominican Republic, Jamaica, Trinidad, Tobago, Venezuela, Ecuador, Peru, Bolivia and the United States. Often they are present in regions where bean golden mosaic virus and Rhynchosia mosaic virus exist. Their symptoms frequently are confused with those of BClMV and AbMV (27, 29, 31, 32, 36, 61, 97, 111, 113, 123).

Common names frequently used for bean chlorotic mottle virus and abutilon mosaic virus in Latin America include moteado clorótico del fríjol, enanismo amarillo, enanismo del fríjol, anao amarelo, clorosis infecciosa de las Malvaceas, and mosaico de Abutilon.

BClMV can cause 100% infection in susceptible cultivars but seldom is economically important. Its incidence normally is only 2-5% in Brazil (31). However, Costa (33) reported that BClMV caused 100% yield loss in each of five cultivars that he studied.

Fig. 9- Plant stunting and witches' broom produced by the bean chlorotic mottle virus.

This group of viruses has a wide host range which includes *Phaseolus vulgaris, P. lunatus, Abutilon hirtum* Sweet, *Althere rosea* (L.) Cav., *Bastardia viscosa* (L.) H.B.K., *Corchorus aestruans* L., *Gossypium barbadense* L., *G. hirsutum* L., *G. esculentum* Mill., *Hibiscus brasilensis* L., *H. esculentus* L., *Malva parviflora* L., *Malva silvestris* L., *Malvaviscus* sp., *Sida acuminata* D.C., *S. aggregata* Presl., *S. bradei* Ulbricht, *S. carpinifolia* L., *S. cardifolia* L., *S. glabra* Mill., *S. glomerata* Cav., *S. humilis* Cav., *S. micrantha* St. Hil., *S. procumbens* Sw., *S. rhombifolia* L., *S. urens* L., *Datura stramonium* L., *Nicandra physaloides* Gaertn., *Nicotiana glutinosa* L., *N. tabacum* L., *Solanum tuberosum* L., *Arachis hypogea* L., *Canavalia ensiformis* D.C., *Cyamopsis tetragonalobus* (L.) Taub., *Glycine max* (L.) Merr., *Lens culinaria* Medik., *L. esculenta* Moench., *Lupinus albus* L. and *Pisum sativum* L. (10, 12, 13, 14, 15, 20, 29, 30, 31, 39, 40, 45, 49, 55, 59, 61, 78, 81, 98, 110, 111, 112).

Symptomatology

BCIMV and AbMV infection can cause a severe dwarfing of susceptible plants, accompanied by a high proliferation of buds and a bunchy or rosette type of plant development. If infection occurs in young plants, a witches' broom is produced and leaves often exhibit chlorotic mottling (Fig. 9). Chlorotic spots or mottled areas may be produced on leaves of tolerant cultivars or older susceptible plants (Fig. 10). These spots may be accompanied by a rugosing of leaves (Fig. 11). Severely affected plants

Fig. 10- Chlorotic mottle symptoms produced on leaves infected by BCIMV.

Fig. 11- Leaf rugosing suspected to be induced by BCIMV.

Fig. 12- Chlorotic mottling induced by AbMV infection of *Pavonia sidaefolia*.

Fig. 13- Infectious chlorosis of Malvaceae symptoms induced in an infected *Malva* sp. plant.

produce few or no pods. Figure 12 illustrates AbMV symptoms produced in an infected *Pavonia* sp. plant, and Figure 13 illustrates symptoms of infectious chlorosis of Malvaceae in an infected *Malva* sp. plant.

Physical Properties

Sun (115) observed ultrathin cytoplasmic sections of *Abutilon striatum* var. *thompsonii* infected with AbMV and found spherical particles 80 nm in diameter. These particles consisted of an inner core 16 nm in diameter surrounded by an outer shell. Kitajima and Costa (81) observed isometric particles 20-25 nm in diameter in infected tissue of *Sida micrantha*. Additional studies are needed to compare these observations with BCIMV isolated from other infected hosts including beans.

Costa and Carvalho (39, 40) determined that AbMV had a thermal inactivation point of 55° - 60°C, a final dilution end-point of 5-6, and retained its infectivity for 48-72 hours *in vitro* in water or sodium sulfide buffer.

Transmission and Epidemiology

Mechanical transmission of AbMV has been very difficult but has been accomplished by Costa and Carvalho (39, 40) from *Malva parviflora* and *Sida micrantha* to soybeans. The virus can be propagated in these species as well as in *Sida carpinifolia*. Bird *et al.* (20) was unable to transmit AbMV mechanically and had difficulties with its natural vector, *Bemisia tabaci* race *sidae*. Strain differences may exist within the virus and whiteflies.

Whiteflies have been demonstrated to transmit BCIMV and AbMV to beans (10, 20, 29, 30, 31, 33, 36, 38, 56, 97, 113, 114). Bird *et al.* (20) showed that whiteflies could acquire the virus during a 15-20 minute feeding and retain their ability to transmit AbMV for seven days. Costa (33) was able to transmit AbMV easily from *Sida* sp. to beans but had difficulty transferring it from beans to beans via the whitefly.

Studies have not found BClMV or AbMV to be seed transmitted (20).

These viruses appear to have a wide host range, including many tropical weed species, which serve as inoculum sources from which whitefly populations acquire the virus and transmit it to beans. Epidemics of AbMV and BClMV also may occur in beans when large plantings of other susceptible crops such as soybeans and cotton, are planted nearby (27, 31, 61, 123).

Control

Very little research exists concerning control measures. However, Costa (31, 36) did not encounter any resistance within *Phaseolus vulgaris* in Brazil. Resistance was found in other species of *Phaseolus,* such as *P. angularis, P. aureus, P. calcaratus* and *P. trinervius* (31). The following *P. vulgaris* accessions were observed to be resistant to BClMV during a natural epidemic at CIAT: ICA - Tuí, Trujillo 7, Honduras 4, P.I. 307824 and P.I. 310739. Additional research is required to verify the resistance of these materials and the practicality of incorporating their resistance into agronomically desirable backgrounds.

Euphorbia Mosaic Virus

Introduction

Euphorbia mosaic virus (EMV) was isolated in 1950 from *Euphorbia prunifolia* Jacq. (37) and has since been observed in many species of *Euphorbia.* The virus has been detected in beans in Brazil but does not appear to be economically important. Common names frequently used for EMV in Latin America include mosaico de las Euforbiaceas and encarquilhamente da folha.

The host range of EMV includes *Euphorbia prunifolia, Datura stramonium, Lycopersicon esculentum, Nicandra physaloides, Nicotiana glutinosa, Canavalia ensiformis, Glycine max, Lens esculenta* and *Phaseolus vulgaris* (18, 20, 22, 31, 33, 36, 40).

Symptomatology

EMV or bean crumpling generally produces only local necrotic leaf lesions at the feeding sites of viruliferous whiteflies. Occasionally EMV may induce a systemic infection characterized by twisting or crumpling of leaves due to the unequal growth of green tissue surrounding the initial necrotic lesions. Abnormal development of auxillary buds also may occur, and plants are commonly stunted.

Physical Properties

Matyis *et al.* (86, 87) purified EMV partially and reported that it consists of identically-paired particles 25 nm in diameter and individual isometric particles which measure 12 - 13 nm in diameter. They determined that EMV belongs to the Geminivirus group.

Costa and Carvalho (39, 40) reported that EMV in sap has a thermal inactivation point of 55° - 60°C and retains its infectivity *in vitro* for more than 48 hours. Bird *et al.* (18) also report that EMV has a thermal inactivation point of 55° - 60°C but retains its infectivity *in vitro* less than 24 hours and has a dilution end point of 10^{-3}. Infectivity can be maintained in tissue dried in calcium chloride at 4°C for 12 weeks.

Transmission and Epidemiology

Euphorbia mosaic virus can be transmitted mechanically from *Euphorbia* sp. (Fig. 14) to *Datura* sp. at a rate of 31% and easily between *Datura* sp. (18, 22, 39, 40). Transmission from soybeans to soybeans is difficult. EMV is not seed-transmitted (20, 33).

Bemisia tabaci supply the natural mode of transmission, can acquire the virus during a 10-minute feeding period, but require a 20-minute period for transmission, and can retain their infectivity for 20 days (20, 31, 36, 37).

Euphorbia mosaic virus seldom is observed in bean fields unless there is a high incidence of whiteflies and infected *Euphorbia* spp. near or within the field.

Control

Very little research has been conducted on control measures for EMV, which is even less infectious to beans than BCIMV or AbMV (31, 33, 36). However, plant resistance has been identified in accessions of *Phaseolus*

Fig. 14- Leaf wrinkling and chlorosis of an *Euphorbia* sp. plant infected with Euphorbia mosaic virus.

angularis, P. aureus, P. calcaratus and *P. trinervius*. Additional research is required to determine if resistance exists within *P. vulgaris* and is practical as a control measure.

Rhynchosia Mosaic Virus

Introduction

Rhynchosia mosaic virus (RMV) was isolated in Puerto Rico and produces symptoms similar to those reported for infected *Rhynchosia minima* in other tropical countries (11, 12, 13, 14, 15, 20, 84). Symptoms of RMV are similar to those caused by BClMV and AbMV. Research is required to determine the relationship between these viruses. Rhynchosia mosaic virus is transmitted by whiteflies but is not reported to cause economic problems.

The common name frequently used for Rhynchosia mosaic virus in Latin America is mosaico de la Rhynchosia.

The virus has a host range which includes *Salvia splendeus* Sellow, *Cajanus indicus* Spreng, *Canavalia ensifomis* (L.) D.C., *C. maritima* (Aubl.) Thou., *Crotalaria juncea* L., *Glycine max* (L.) Merrill, *Macroptilium lathyroides* (L.) Urban, *Pachyrrhizus erosus* (L.) Urban, *Phaseolus aborigeneus* Burk., *P. acutifolius* A. Gray. P. I. Wright, *P. acutifolius* A. Gray *latifolius, P. coccineus* L., *P. lunatus* L., *P. trichocarpus* C. Wright, *P. vulgaris* L., *Rhynchosia minima* DC, *R. reticulata* DC, *Vigna aconitifolia* (Jacq.) Marechal, *V. angularis* (Willd.) Ohwi and Ohashi, *Abelmoschus esculentus* (L.) Moendi, *Gossypium hirsutum* L., *Malachra capitata* L., *Oxalis berrelieri* L., *Nicotiana acuminata* Hook, *N. alata* Link and Otto, *N. bonariensis* Lehmann, *N. glutinosa* L., *N. nightiana* Goodspeed, *N. maritima* Wheeler, *N. paniculata* L.and *N. tabacum* L. (11, 20).

Symptomatology

Rhynchosia mosaic virus infection of beans causes symptoms such as leaf malformation, yellowing (Fig. 15), witches' broom and plant stunting.

Fig. 15- Bean leaves infected with Rhynchosia mosaic virus.

When infection occurs in young plants, symptoms consist of a proliferation of flowers and branches and little if any seed production (14).

The virus has not yet been purified to study its physical properties.

Transmission and Epidemiology

Mechanical transmission (18%) has been demonstrated by using buffers and the tobacco cultivar, Virginia 12, as source of inoculum (12, 20). Rhynchosia mosaic virus has not been found to be seed-transmitted (20).

The virus is easily transmitted by *Bemisia tabaci* (11, 20). Transmission can be achieved in less than 24 hours and the insect retains its infectivity for seven days. Apparently, the virus survives in infected weeds such as *Rhynchosia minima* which is widespread throughout the tropics.

Control

Very little research has been conducted into control measures for RMV. Glasshouse investigations in Puerto Rico (20), revealed that the bean cultivars La Vega (R19) and Santa Ana (selection from Masaya, Nicaragua) were tolerant to the virus and had a good level of resistance in the field.

Other Whitefly-Transmitted Viruses

Bird (9,20) reports that three viruses were capable of infecting beans under controlled conditions in Puerto Rico. They were Jatropha mosaic virus, isolated from *Jatropha gossypifolia* (L.) Pohl and transmitted by *Bemisia tabaci* race (biotype) *jatropha;* Merremia mosaic virus, isolated from *Merremia quinquefolia* Hall and transmitted by *Bemisia tabaci* race (biotype) *sidae;* and Jacquemontia mosaic virus, isolated from *Jacquemontia tamnifolia* Griseb and transmitted by *Bemisia tabaci* race (biotype) *sidae*.

This chapter has reviewed briefly some of the whitefly-transmitted viruses which are reported to infect beans under natural and artificial conditions. Much confusion exists between investigators as to virus identification and relationships (20, 33, 36, 41, 61, 76, 86). Additional research is required to elucidate this complex group of viruses and to study the variability which may exist within these viruses and their whitefly vectors.

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Chapter 15

Miscellaneous Bean Viruses

G. E. Gálvez and
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Chapter 15

Miscellaneous Bean Viruses

Introduction

Previous chapters have reviewed many bean viruses transmitted by insect vectors such as aphids, beetles and whiteflies. Other bean viruses also are known to be transmitted by these vectors, or by other insects, such as thrips and leafhoppers. Some bean viruses are not known to be transmitted by any insect vector. This chapter will review briefly some miscellaneous virus diseases of *Phaseolus vulgaris.*

Alfalfa Mosaic Virus

Alfalfa mosaic virus (AMV) is an aphid-transmitted virus that was initially detected on beans in the United States (31). AMV consists of various strains including yellow dot, alfalfa yellow mosaic (31), vein necrosis (30) and spot mosaic (29). None of these strains of AMV has been reported to be economically important (31).

AMV was known previously as Lucerne mosaic virus, Alfalfa virus 1, Alfalfa virus 2, Medicago virus 2, and *Marmor medicaginis* Holmes (7, 31). Alfalfa mosaic virus has not been studied on beans in Latin America, but AMV and its strains have the Spanish names of mosaico de la alfalfa, punto amarillo, mosaico amarillo de la alfalfa, necrosis venal, mosaico de la mancha and calico.

AMV and its strains may produce a light systemic mottling, an intense chlorotic mottling of leaves, necrosis of leaves or stems, and dieback of the growing point. However, the most common symptom consists only of local necrotic lesions which may have a diameter of 0.5-3.0 mm (31).

AMV is easily transmitted mechanically and by aphids (17). It is not reported to be transmitted in bean seed, but is transmitted in seed of alfalfa (6%) and pepper (1-5%). AMV particles are bacilliform in shape, have three different lengths and contain RNA (7).

Since AMV is not an economically important virus disease of beans, little research has been conducted with control measures. However, some differences have been observed in the frequency of local lesions produced on specific bean cultivars (16). Susceptibility is also correlated with plant age, ability of the virus to induce local lesions or systemic infection, and temperatures during the pre-and post-inoculation period (3, 6, 14, 19, 28).

Curly Top

Curly top of beans is transmitted by the beet leafhopper, *Circulifer tenellus* (Baker). This virus can cause economic losses to beans and other cultivated crops, such as beets *(Beta vulgaris* L.), in the United States and Canada (4,31). Curly top has been called *Ruga verrucosous* Cars.& Bennett, and reportedly contains 10 strains which differ for their virulence (31). The common name of curly top in Latin America is ápice rizado de la remolacha.

Infected young bean plants commonly exhibit trifoliate leaf symptoms of puckering, downward curling, yellowing and death. Primary leaves of infected plants may be thicker and more brittle than those of uninfected plants. The initial symptoms of curly top may resemble those induced by bean common mosaic virus (31). Leaf curling and yellowing also may resemble damage induced by green leafhopper *(Empoasca* spp.) feeding.

Virus particles of curly top are geminate, have a sedimentation coefficient of 82 S and a 20% nucleic acid content (20, 22).

Control measures consist of resistant cultivars. This resistance is temperature-sensitive in some bean cultivars since it can be destroyed at high temperatures, regardless of plant age at the time of inoculation (25). Silbernagel (24) reports that the breeding lines, ARS-6BP-5 and ARS-5BP-7, are highly resistant to the curly top virus.

Bean Summer Death

Bean summer death is reported to occur in New South Wales, Australia (1, 2, 8). The disease agent is transmitted by the brown leafhopper, *Orosius argentatus,* which also is known to transmit various mycoplasma-like pathogens of beans and other legumes (refer to Chapter 11). Bean summer death was originally suspected to have a mycoplasma-like etiology, but Bowyer and Atherton (8) claim that the causal agent is not a mycoplasma but is similar in some respects to curly top.

The host range of bean summer death includes *Phaseolus vulgaris, Datura stramonium, Beta vulgaris* var. *vulgaris, B. vulgaris* var. *cicla* and

Callistephus chinensis (8). The Spanish name for bean summer death is muerte de verano del fríjol.

The symptomatology of this disease consists of yellowing and subsequent death of beans, commonly following a period of high temperature (1,2). The insect vector has a minimum latent period of 24-48 hours and remains infective for at least 21 days after acquisition of the causal agent during the nymphal or adult stage.

Little research has been conducted into control measures. However, Ballantyne *et al.* (2) report that various materials resistant to curly top in the United States also were resistant to bean summer death in Australia. Additional research is required to identify resistant cultivars and to fully characterize the agent responsible for bean summer death.

Tomato Spotted Wilt Virus

Tomato spotted wilt virus (TSWV) is reported to occur in Brazil and Canada on various plant species. It is not reported to cause serious economic damage to beans. However, it can affect other legumes, tomatoes, tobacco, pineapple and ornamental plants. The virus is transmitted mechanically in tomato seed and by various types of thrips, such as *Thrips tabaci, Frankliniella schultzei, F. fusca, F. paucispinosa* and *F. occidentalis* (9,10,11, 23).

Tomato spotted wilt virus also is known as Kromnek virus, *Lycopersicum* virus 3, Pineapple yellow spot virus, tomato bronze leaf virus and vira-cabeca virus. It is commonly referred to as marchitamiento manchado del tomate in Latin America.

Kitajima *et al.* (18) reported that particles of the virus were partially isometric, apparently surrounded by a membrane, contain RNA, and measure 80-120 nm in diameter. TSWV was the first plant virus reported to contain lipids (27). Its identification and characterization are reported by Best (5) and Ie (15).

Red Node

Red node has been reported to occur in the United States (31) but rarely in Latin America (11, 26). This viral disease is reported to be related to tobacco streak virus (31). The common Latin American names of red node and tobacco streak virus are nudo rojo and mosaico rayado del tabaco, respectively.

Symptoms include a reddish discoloration at the nodes of stems and pulvini of leaves, as well as reddish concentric rings on pods. Pods may be shriveled and not produce seed. Plants also may be stunted or killed (31).

The virus is transmitted mechanically and in bean seed (12, 31). There are no reports of insect vectors. The virus particles are isometric, measure 28 nm in diameter, contain three to four nucleoproteins, and have a sedimentation coefficient between 90-123 S (21).

The virus may be controlled by production of clean seed and use of resistant cultivars such as Kentucky Wonder No. 780 and Kentucky Wonder Brown No. 814 (31).

Other Bean Viruses

Many other viruses are reported to infect beans, but primarily only under controlled conditions in the laboratory or glasshouse (13, 31). A few examples of these viruses are clover blotch, clover (red) necrotic mosaic, cowpea aphid-borne mosaic, adzuki bean mosaic, pea dwarf mosaic, clover yellow bean, and Desmodium yellow mottle. Little if any information is reported concerning the natural occurrence of these minor bean viruses.

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SECTION IV.
OTHER PRODUCTION PROBLEMS

Chapter 16

Seed Pathology

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Chapter 16

Seed Pathology

Introduction

Dry beans *(Phaseolus vulgaris* L.) are not vegetatively propagated. Therefore, they depend upon seed production for perpetuation of the crop. The quality of dry bean seeds used for planting by Latin American farmers generally is low, especially among those with small land holdings.

Sánchez and Pinchinat (36) conducted a survey of seed used by farmers in Costa Rica and found an average germination of 68%. Ellis *et al.* (16) conducted a similar survey of farmers with small land holdings in Colombia and reported that germination was as low as 8% with 100% seed infected by fungi. Certified seed is difficult to obtain and rarely used by farmers in Latin America, since less than 3% of all seed sown is certified (44).

Seed Transmission of Pathogens

Seeds provide an efficient method for the transfer of plant pathogenic organisms between locations. More than 50% of the major bean diseases are seed-borne (14). As a farmer plants infested seed, he also sows the potential for future disease problems. Seed transmission of plant pathogens is of concern in Latin America because most farmers plant seed saved from previous harvests (20). The effect of seed-borne organisms upon seed germination is not well documented, but internally-borne fungi are associated with decreased seed germination and field emergence of dry beans (Figs. 1-4, p 304). Ellis *et al.* (16) found a correlation of -0.88 between percentage recovery of internally-borne fungi and seedling emergence. Seed viability, germination and contamination by micro-organisms also can be affected by mechanical damage which may occur during harvesting, threshing and/or planting (9, 39).

Seed Storage Problems

Conditions for seed storage are critical to the survival of high quality seed for long periods and to the degree of storage losses incited by various

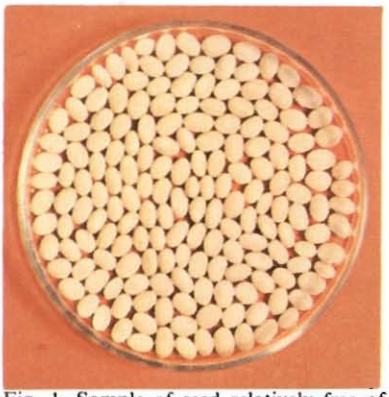

Fig. 1- Sample of seed relatively free of seed-borne organisms.

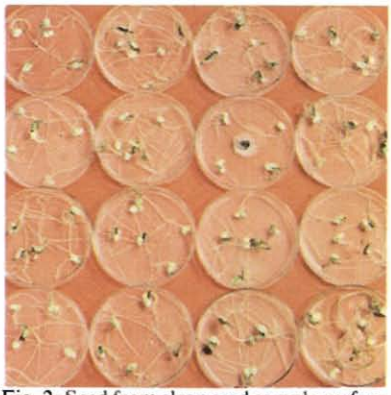

Fig. 2- Seed from clean seed sample surface disinfected and incubated on potato-dextrose agar.

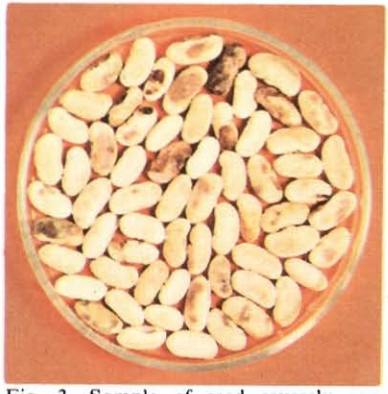

Fig. 3- Sample of seed severely contaminated by seed-borne organisms.

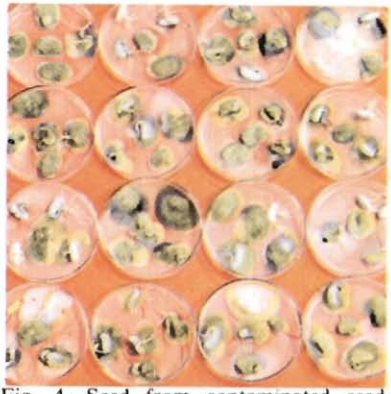

Fig. 4- Seed from contaminated seed sample surface disinfected and incubated on potato-dextrose agar.

seed contaminants and seed-borne pathogens (see Table 1). López and Christensen (26) report that the seed moisture content should be less than 15%, preferably 13%, and seed should be stored in conditions with less than 75% relative humidity. López and Crispin (27) report that cultivars vary in their resistance to storage rot organisms. Also, storage temperatures lower than 10°C should extend the viability of dry bean seed.

Control of Seed-Borne Fungi

Numerous fungi are reported to be borne internally or as surface contaminants in seed of *Phaseolus vulgaris* (Table 1). Many of these organisms also are seed-borne in other members of the Leguminoseae, such as soybeans, pigeon peas and cowpeas (16). Figure 5 illustrates the manner by which *Colletotrichum lindemuthianum* may become seed-borne in dry beans. Most internally-borne fungi are located inside the seed coat and some infection may occur in the cotyledon or embryo (1, 15).

Fig. 5- (right) Pod and seed infection by the anthracnose fungus.

Fig. 6 - (lower right) Seed sample harvested at maturity, surface disinfected and incubated on potato-dextrose agar.

Fig. 7 - (below) Seed sample harvested two weeks after maturity, surface disinfected and incubated on potato-dextrose agar.

Protectant fungicides such as Captan (Orthoside), Ceresan and Arasan or Thiram diffuse into the seed coat where many seed-borne fungi are located but do not enter dry bean cotyledons (14, 15, 40). Recommended application rates for most seed treatments is 1-2 g per kg seed. Seed treatment is relatively inexpensive and can improve germination and field emergence of seed lots with moderate levels of infected seed.

Systemic fungicides such as Benomyl can penetrate the seed coat and cotyledons of beans to provide some degree of control (1, 14). Investigations are being conducted with chemicals such as ethylene oxide (34) which has excellent biocidal and penetrative properties and may prove to be practical in removing seed-borne contaminants with little reduction in seed viability.

Systemic fungicides were foliarly applied beginning 40 days after planting, with four applications made at 9-day intervals by Ellis and co-workers. Benomyl (1 kg/ha) significantly reduced seed infection by *Colletotrichum lindemuthianum* when compared to the non-sprayed treatment (11, 13). A protectant fungicide such as Difolatan or Captafol was not as effective, because heavy rainfalls consistently washed the chemical off the plants. Fungicides may be useful for clean seed production in Latin America. However, they may not be economical for regular production operations unless growers are willing to pay for the increased production costs.

Date of harvest is important in the production of high quality and pathogen-free seed (13, 35). The percentage of seed infection by fungi increases and the percentage of seed germination decreases with prolonged time in the field after plant maturity (Fig. 6 and Fig. 7) (13). Therefore, it is

Fig. 8- Seed infection by *Sclerotium rolfsii*.

Fig. 9- Seed infection by *Macrophomina phaseolina* (black mycelia) and *Phomopsis* species (white mycelia).

important that seed production fields be harvested immediately after plant maturation. Foliar applications of Benomyl during the growing season can reduce the incidence of seed-borne fungi and low seed germination commonly associated with delayed harvest. Similar results are reported for soybean production (10).

In some dry bean cultivars, pod contact with the soil may cause significantly higher levels of seed infection by various soil-borne fungi, such as *Rhizoctonia solani, Sclerotium rolfsii* (Fig. 8), and *Macrophomina phaseolina* (Fig. 9). This may result in a significantly lower seed germination than in seeds collected from pods of the same plant free from soil contact (12, 47). When harvesting seed production fields, it would be beneficial to avoid pods which have soil contact, especially for farmers who can hand-pick desirable pods with seeds destined for future plantings.

The most efficient method of producing clean seed free from a specific pathogen is to use a cultivar that is immune or resistant to infection by that pathogen. For example, York *et al.* (46) have studied resistance to Pythium seed decay intensively. Cultivars which are tolerant to a specific pathogen may allow limited development of the pathogen and its potential to be transmitted within the seed. Therefore, seed from such cultivars must be assayed carefully to determine whether seed-borne fungi are present.

Control of Seed-Borne Bacteria

It is reported that 95 species and varieties of bacteria may be seed-borne in numerous crops (38). Various bacterial pathogens are reported to be internally seed-borne in *Phaseolus vulgaris* (Table 1). *Xanthomonas*

phaseoli and *Corynebacterium flaccumfaciens* can remain viable for two to 10 and five to 24 years, respectively, in seeds (38).

No satisfactory method of seed treatment will completely control internally-borne bacteria of dry beans. Several methods and compounds have been tested with varying results, but the general conclusion is still negative. External seed contamination can be controlled by application of Streptomycin or Kasugamycin (41).

The most reliable method of producing seed free from bacterial pathogens is to select production areas where environmental conditions and cultural practices do not favor bacterial growth and development (19). Copeland *et al.* (4) state that additional control can be achieved by long rotations of different crops, planting different cultivars in alternating seasons and sequential planting of adjacent fields to reduce large acreages of susceptible plants at one point during a growing season.

At present, no commercial cultivar is immune to infection by the common blight pathogen. However, resistance to infection has been reported and differential pod susceptibility (5, 6) may be used to further reduce seed contamination.

Control of Seed-Borne Viruses

Viruses are reported to be seed-borne in *Phaseolus vulgaris* (Table 1). Bean common mosaic virus is transmitted internally in cotyledons and embryos but not in seed coats, while southern bean mosaic virus is transmitted in embryos and seed coats (17). Once seeds are infected, no seed treatments available currently will eliminate the virus from bean seed. The most effective procedure is to produce clean seed in an area where the virus-infected plants can be eliminated and where vectors which transmit the virus can be controlled or do not exist.

Development of resistant cultivars also will allow the production and use of clean seed. However, research still is needed to determine if low levels of virus can persist in resistant or tolerant cultivars and serve as reservoirs of inoculum for infection of susceptible cultivars by insects or other vectors.

Production of Pathogen-Free Seed

Benefits derived from the use of clean seed have been demonstrated in temperate regions such as the United States (4, 19) and in Australia (28) and Latin America (2, 3, 18). Clean seed production has been difficult in Brazil (23), but programs still are being developed. Clean seed production fields should be located in areas where the environment is unfavorable for

survival, infection and spread of pathogenic organisms. An ideal production site should have an annual rainfall of less than 300 mm, a daily relative humidity less than 60%, a daily temperature regime between 25°-35°C, and gravity irrigation facilities. These production sites also should be located in regions where dry beans or other legumes are not grown commercially in order to avoid contamination by insect transmitted viruses with wide host ranges. A seed production program will require a form of inspection and certification to ensure seed cleanliness and purity.

Seed production programs often are provided with a limited seed quantity. The CIAT bean production program has used the following glasshouse and/or screen house technique (Fig. 10) to produce small quantities (10-100 g) of pathogen-free seed:

— Seed of each entry is planted (2 seeds/pot measuring 15-20 cm in diameter by 25 cm in depth) in sterilized soil in a glasshouse or fine-meshed screen house.

— Seedlings are carefully irrigated to avoid physical contact between plants and observed daily to identify the expression of bean disease symptoms. When an infected plant is identified, the data is recorded and the plant + soil + pot are immediately sterilized.

— Surviving plants are protected from outside contamination and observed daily for symptom expression.

Fig. 10- Clean seed production in screen house facilities at CIAT.

— Seedlings and/or mature plants may be assayed serologically and harvested separately to avoid contamination, especially from latent seed-borne viruses.

— Pathogen-free seed then is stored in sealed containers at less than 10°C and 13% relative humidity.

Field production and increase of pathogen-free seed should be undertaken in the proper production zone. Seed should be planted 25-30 cm apart within rows spaced 1 m apart. Plants must be inspected frequently (weekly) during their growth to detect and eliminate plants infected with diseases. Critical evaluation times after germination include 15 days to detect bean common mosaic virus; 30 days to detect common bacterial blight, angular leaf spot, and web blight; 45 and 60 days to detect common bacterial blight, angular leaf spot and anthracnose. Chemical applications may be required to prevent plant infection by pathogens or the buildup of insect vectors.

It is ideal to tolerate 0% infection by any bean pathogen which may be transmitted by seed. However, this tolerance may have to be raised to 0.5-1% infection when seed is produced in tropical environmental conditions which are marginal for successful clean seed production.

Successful production of clean seed also is dependent upon proper field management during maturation and harvest. Foliar applications of chemicals seven to 10 days before plant maturity may reduce pod infection by plant pathogens and/or saprophytes and ensure good seed viability. Mature pods which are not in contact with the soil should be harvested immediately.

A windrow inspection is advised if beans are not harvested and threshed immediately. Pods must be carefully threshed and cleaned to avoid mechanical damage and cracking, and they should be stored under proper conditions. Subsequent laboratory (serology or other detection procedures) and greenhouse tests may be conducted to verify that the seed is indeed pathogen-free (21, 29, 45). Certified seed should be planted in pathogen-free commercial production regions or protected with chemicals to assure improved production. Additional yield advances may be possible by utilization of clean seed practices for newly developed high-yielding and disease resistant cultivars.

Table 1. Examples of seed-borne and seed-contaminating organisms associated with dry beans *(Phaseolus vulgaris* L.).

Organism	Common Name	Literature Cited
FUNGI		
Acrostalagmus spp.	-	16
Alternaria spp.	Leaf and Pod Spot	37
Ascochyta spp.	Leaf and Pod Spot	1
Aspergillus candidus	Storage Rot	27
Aspergillus glaucus	Storage Rot	27
Aspergillus niger	Storage Rot	16
Aspergillus repens	Storage Rot	27
Aspergillus restrictus	Storage Rot	27
Botryodiplodia theobromae	Seed Decay	16
Botrytis cinerea	Gray Mold	16
Cercospora cruenta	Leaf Blotch	47
Chaetoseptoria wellmanii	Leaf Spot	7
Cladosporium herbarum	Cladosporium Spot	42
Colletotrichum dematium	-	16
Colletotrichum lindemuthianum	Anthracnose	47
Colletotrichum truncatum	Stem Anthracnose	25
Curvularia spp.	Leaf Spot	8
Dendrophoma spp.	-	1
Diaporthe phaseolorum	Pod and Stem Blight	16
Diplodia natalensis	Seed Contaminant	47
Erysiphe polygoni	Powdery Mildew	47
Fusarium equiseti	Damping Off	16
Fusarium moniliforme	-	32
Fusarium oxysporum f. sp. *phaseoli*	Fusarium Yellows	47
Fusarium roseum	-	8
Fusarium semitectum	Pod Decay	43
Fusarium solani	Root Rot	31
Fusarium sulphureum	-	16
Isariopsis griseola	Angular Leaf Spot	33
Macrophomina phaseolina	Ashy Stem Blight	47
Monilia spp.	-	16
Mucor spp.	-	8
Nematospora coryli	Yeast Spot	43
Nigrospora spp.	-	12
Penicillium spp.	Storage Rot	27

(continued)

Organism	Common Name	Literature Cited
Pestalotiopsis spp.	-	16
Peyronellaea spp.	-	16
Phomopsis phaseolina	Leaf and Pod Spot	16
Rhizoctonia solani	Root Rot	24
Rhizopus spp.	Soft Rot	1
Sclerotinia sclerotiorum	White Mold	47
Sclerotium rolfsii	Southern Blight	1
Sporotrichum spp.	-	37
Stemphylium spp.	Leaf Spot	37
Thanatephorus cucumeris	Web Blight	47
BACTERIA		
Achromobacter spp.	-	37
Aerobacter aerogenes	-	37
Agrobacterium radiobacter	-	37
Alcaligenes viscosus	-	37
Bacillus cereus	-	37
Bacillus megatherium	-	37
Bacillus polymyxa	-	37
Bacillus sphaericus	-	37
Bacillus subtilis	-	37
Bacterium globiforme	-	37
Corynebacterium flaccumfaciens	Bacterial Wilt	47
Corynebacterium helvolum	-	37
Micrococcus spp.	-	37
Pseudomonas fluorescens	-	37
Pseudomonas phaseolicola	Halo Blight	47
Pseudomonas syringae	Bacterial Brown Spot	47
Xanthomonas phaseoli	Common Bacterial Blight	47
Xanthomonas phaseoli var. *fuscans*	Fuscous Bacterial Blight	47
VIRUSES		
Bean Common Mosaic Virus	BCMV	47
Bean Western Mosaic Virus	Strain of BCMV	47
Bean Southern Mosaic Virus	BSMV	47
Tobacco Streak Virus	Red Node Strain	47
Cucumber Mosaic Virus	CMV-PR	30
Cherry Leaf Roll Virus	-	22

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Chapter 17
Nematodes

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Chapter 17

Nematodes

Introduction

Numerous nematodes (eelworms) have been found on the roots of beans and other plants throughout the world (Table 1). Many of these and other nematodes are reported to occur on beans worldwide, with species of *Meloidogyne* and *Pratylenchus* frequently encountered in Latin and North America (8, 10, 11, 14, 18, 20, 22, 24, 30, 31, 33, 35, 36, 37, 38, 45, 49, 51, 56). During severe infestations, yield losses may reach 10 to 80% with root lesion (35) nematodes, or 50 to 90% with root knot (14, 50, 56) nematodes. This chapter will concentrate primarily on research with species of *Meloidogyne* and *Pratylenchus*.

Common names frequently used for *Meloidogyne* species in Latin America include nematodos de los nódulos radicales and galhas das raizes. Common names frequently used for *Pratylenchus* species include nematodos de las lesiones radicales, lesiones por nematodos and definhamento de nematoide.

Epidemiology and Life Cycle

Meloidogyne species are most prevalent in light sandy soils with good drainage and an average soil temperature of 25° to 30°C (9). Numerous nematode species are transported between growing regions or fields by irrigation water, vegetative plant parts, and soil contaminated with eggs or larvae which adhere to farm implements, animals or man (7, 9, 43, 51, 52, 53). Length of survival in soil varies with the nematode species, stage of development, soil type, moisture, temperature (52, 53), soil aeration and length of the fallow period.

The life cycle of *Meloidogyne* spp. involves various developmental stages. Larvae hatch from eggs. They grow between a series of three molts into adult males and females, and the latter lay eggs in a gelatinous mass. Nematode eggs are oval, sometimes ellipsoidal and slightly concave on the

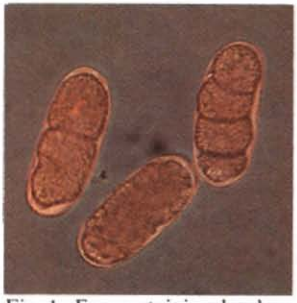

Fig. 1 - Egg containing developing larvae of *Meloidogyne* species.

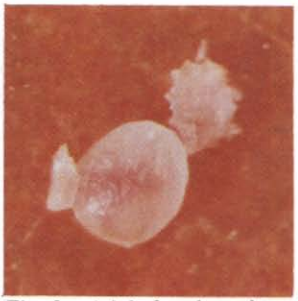

Fig. 2 - Adult female and egg mass. *Meloidogyne* species.

Fig. 3 - Young larva of *Meloidogyne* species.

Fig. 4 - Adult female of *Meloidogyne incognita.*

side (Fig. 1), and measure 30-52 by 67-128 μ (47). Eggs usually are protected from dehydration by a gelatinous mass or matrix (glycoprotein substance) secreted by the female (Fig. 2) (3).

Larvae are vermiform (Fig. 3), have a stylet which is about 10 μ long and may have an overall length of 375-500 μ and a width of 15 μ (35). Adult males are cylindroid, measure 0.03-0.36 by 1.20-1.50 mm, lack a bursa and have a well-developed stylet. Adult females are pyriform (Fig. 4), are pearly white (visible in roots without magnification), have a soft cuticle and measure 0.27-0.75 by 0.40-1.30 mm (42, 44, 53). The entire life cycle may be completed during a period of 17-57 days following inoculation (27), depending upon the soil temperature (48).

Plant Infection

Larvae of *Meloidogyne* spp. penetrate the plant root system (100-300 larvae/seedling) within 48 hours after inoculation (29) and migrate inter- and intracellularly through the cortical tissue into the stele. The larval head is inserted into the vascular system to obtain plant nutrients. Plant cells in the vicinity of the larvae increase in number (hyperplasia) and size (hypertrophy), thereby producing the root swelling or gall. Giant cells form near the larval head by the fusion and enlargement of plant cells in response to nematode feeding. Slight injury is apparent 10 days after infection, but

Fig. 5 - Plant chlorosis and stunting caused by *Meloidogyne* species infection.

within 40 days epidermal cells often collapse after females have deposited eggs near the outer root surface (28). Infection by and pathogenesis of *Meloidogyne* spp. are affected by plant age, plant susceptiblity, size of nematode populations and environmental factors (6, 16, 21, 25, 29, 40).

Larvae of *Pratylenchus* spp. penetrate the root system and migrate intracellularly through the cortical tissue causing the cell wall to break. The nematodes usually are oriented lengthwise to the vascular tissue which becomes necrotic 25-32 days after infection. Larvae also may be coiled within one or two host cells (46). The 60-day life cycle may be completed inside host tissue, where all larval and adult stages are eel-shaped migratory endoparasites (47).

Symptomatology

Symptoms of nematode feeding upon plant root systems often appear in above-ground plant parts which become chlorotic, stunted, burned at the leaf edges and may wilt during periods of moisture stress (Fig. 5). Symptoms of root infection by *Meloidogyne* spp. consist of the appearance of root galls (as large as 12 mm or greater in diameter) on primary and secondary roots (Fig. 6), reduced root systems, shortened and thickened roots, or a reduced number of lateral roots.

Fig. 6 - Root galls produced after infection by *Meloidogyne* species.

During severe infections, the roots may appear as a mass of galls. These can cause plant death, due to interference with normal root functions. They cannot be detached easily from the root system without breaking the root, in comparison to nodules formed by nitrogen-fixing bacteria which are loosely attached to the sides of roots (33). Nematodes may feed on bacterial nodules of soybeans and expose the nodules to subsequent infection and degradation by other species of bacteria, nematodes and fungi (2). Stem and hypocotyl tissue may become infected and exhibit galls when seed is planted too deeply (12).

Fig. 7 - Root damage caused by *Pratylenchus scribneri* feeding.

Lesion nematodes produce brown or black lesions on roots (Fig. 7) during their feeding activities in root epidermal and cortical tissues (28, 46).

Control by Cultural Practices

Crop rotation can reduce the population levels of parasitic nematodes when beans are planted once every two or three years in rotation with corn or other cereal crops or with canopy crops such as *Tagetes minuta* (marigolds), *Crotalaria spectabilis* (rattle box) (11, 23, 56), or *Indigofera hirsuta* (hairy indigo) (34). However, many nematode species have a wide host range and make crop rotation impractical. Other cultural practices which reduce nematode populations include long fallow periods, deep plowing and flooding for one or two weeks (9, 51).

Control by Chemicals

Chemical control can be effective but is expensive and often requires special equipment for soil application. Soil fumigants such as Dichloropropene-dichloropropane or DD, Ethylene Dibromide or EDB, Nemagon (DBCP 75% EC) (19, 32, 34, 39, 41, 56), Phenamiphos 40% (19) and Methyl Bromide plus Chloropicrin (35) have been used successfully for control.

Control by Plant Resistance

Plant resistance to root knot nematodes such as *Meloidogyne incognita* exists in many bean lines including Alabama No. 1, Alabama No. 2, Alabama No. 8, Alabama No. 19, Spartan, State, P.I. 165426, Rico 23, Manteigao Fosco 11, Porto-Alegre-Vagem-Roxa, Coffee Wonder, Manao Wonder, Spring Water Half Runner and Wingard Wonder (4, 13, 17, 34, 50, 51, 54, 56). Resistant lima bean cultivars include Hopi, L-5989, Nemagreen, Westan and White Ventura (1). P.I. 165426 is resistant to *M. incognita* (13) but is susceptible to a simultaneous infection by *M. incognita* and *M. javanica* (26). Ngundo (26) reports that the following bean lines are resistant to infection by both species: P.I. 165435, P.I. 313709, Nyakahuti, Red Haricot, Rono, Saginaw and Kibuu.

Wyatt (55) reports that resistance to galling and the build-up of nematode populations in root systems are independent characters and probably governed by separate genetic controls. Selection often is based upon root galling, egg mass formation and number of eggs produced per gram of root tissue. However, gall index is not always correlated with yield (26). Resistant reactions also may include the appearance of root necrosis four days after inoculation and an absence of giant cells (13). This reaction is influenced by soil temperature, since galling, egg mass production and female development increases as the soil temperature increases from 16° to 28°C (13, 15).

Breeding for resistance to nematodes is complicated by various factors already presented, as well as by the facts that:

- Plant resistance and galling response apparently are controlled by separate genetic mechanisms.

- Beans are very sensitive to disturbances of the root and therefore pose problems to seedling evaluations and conservation by transplanting (13).

- Resistance or tolerance to nematode species also may be complicated by the presence of different races or biotypes of nematodes. In soybeans, for example, susceptibility to one race of the root knot nematode was partially dominant, and resistance was qualitatively inherited and conditioned by one major gene in association with at least one modifying gene (5).

A modified backcross system has been used to incorporate high levels of tolerance or resistance to root knot nematodes in snap beans (13). Methodology must be developed to improve dry beans as well.

Table 1. List of nematodes frequently encountered in association with roots of dry beans and other plants.

Scientific Name*	Common Name
Aphelenchoides spp.	Bud and Leaf Nematode
Belonolaimus gracilis Steiner	Sting Nematode
Belonolaimus longicaudatus Rau	Sting Nematode
Criconemoides spp.	Ring Nematode
Ditylenchus dipsaci (Kühn) Filipjev	Stem Nematode
Ditylenchus destructor Thorne	Potato Rot Nematode
Helicotylenchus spp.	Spiral Nematode
Heterodera glycines Ichinohe	Soybean Cyst Nematode
Heterodera humuli Filipjev	Hop Cyst Nematode
Heterodera schachtii Schmidt	Sugar Beet Nematode
Heterodera trifolii Goffart	Clover Cyst Nematode
Meloidogyne arenaria (Neal) Chitwood	Root Knot Nematode
Meloidogyne hapla Chitwood	Root Knot Nematode
Meloidogyne incognita (Kofoid & White) Chitwood	Root Knot Nematode
Meloidogyne javanica (Treub) Chitwood	Root Knot Nematode
Pratylenchus brachyurus (Godfrey) Filipjev & Stekhoven	Root Lesion Nematode
Pratylenchus penetrans (Cobb) Filipjev & Stekhoven	Root Lesion Nematode
Pratylenchus scribneri Steiner	Root Lesion Nematode
Rotylenchulus reniformis Linford & Oliveira	Reniform Nematode
Trichodorus spp.	Stubby Root Nematode
Tylenchorhynchus spp.	Stunt Nematode
Xiphinema elongatum Stekhoven & Teunissen	Dagger Nematode
Xiphinema krugi Lordello	Dagger Nematode
Xiphinema setarie Luc	Dagger Nematode

* This table does not list all the important nematode species, and many are endemic to specific soils, hosts and regions.

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Chapter 18
Miscellaneous Problems

H.F. Schwartz

Chapter 18

Miscellaneous Problems

Introduction

Many other factors besides plant pathogens, insects, nematodes and nutritional disorders may damage beans severely during their growth. Parasitic plants such as dodder can attack bean plants and reduce yields. Various environmental conditions including frost, high temperatures, wind and drought can injure bean seedlings or mature plants. Variation in soil properties and drainage may produce marked differences in plant appearance and vigor within localized areas of a field. Genetic and physiological abnormalities may cause obvious or subtle changes in plant development. Improper pesticide and fertilizer applications, or toxic air pollutants may cause chemical damage.

Symptoms induced by these types of factors sometimes are confused with those caused by other problems described elsewhere in this book. Proper identification of the causal agent often requires a complete history of all past and current factors relevant to bean production in a specific region. This chapter will describe briefly some miscellaneous problems which may occur during dry bean production in Latin America and other parts of the world.

Biotic Problems

Parasitic plants such as dodder are known to cause damage to cultivated crops, including dry beans (17, 18, 20, 21). *Cassytha filiformis* is reported to parasitize bean plants under controlled conditions (20), and *Cuscuta epithymum* (clover dodder) is a general parasite of legumes (21). Dodder produces slender, nearly leafless vines (Fig. 1, page 330) which may be white, yellow, orange or reddish purple. When a vine contacts a host such as a bean plant, it wraps around the plant part and develops haustoria or suckers through which the dodder may obtain nutrients from the bean plant. The dodder vines then may extend from plant to plant and can seriously reduce yield (18). Pieces of the dodder vine and seeds can be

Fig. 1 - Parasitism of potatoes by dodder.

disseminated by animals, man, farm implements and surface irrigation water. Control measures include sanitation before the dodder produces seeds, burning residue to destroy seeds, and rotation with resistant crops such as cereals, soybeans or cowpeas (17, 21).

Algae also are known to occur on many tropically grown plants; however, there are no reports of damage caused to beans.

Climatic and Physical Problems

Beans are grown under a wide range of environmental conditions, but certain cultivars may be better adapted to growing conditions unique to specific production areas. However, cultivars that are reasonably well-adapted to a specific growing region may then be affected by extremes or variations which occur for one or more environmental factors during the course of a production season.

Moisture

Plants may be subjected to high or low moisture stresses which can influence physiological processes, plant development and susceptibility to plant pathogens. A low soil moisture content can damage plants due to the unavailability of water for plant roots, the accumulation of toxic ions such as magnesium and boron, stomatal closure, restricted uptake of CO_2, and temporary or permanent plant wilt (13).

High soil moisture and flooding may leach important nutrients required for normal plant development, reduce oxygen content, induce general

plant chlorosis, and increase levels of toxic by-products from anaerobic metabolism. If combined with high temperatures, they may increase the rate of respiration (13, 18, 25).

High soil moisture or relative humidity may induce intumescence in cultivars with abundant foliage and pods which are not directly exposed to the sun. Raised dark green spots may appear on leaves or pods due to the elongation and multiplication of cells, and the spots may burst (edema) if high moisture conditions persist (25).

Leaves may be damaged by the impact of large droplets of water during rainstorms, which may cause leaf wilt or defoliation (14). Hail and lightning damage also may occur during rainstorms and stunt plant development, provide wounds for secondary disease agents, and cause plant death (14, 18).

Temperature

Beans also are affected by soil and air temperatures, and sudden changes may influence the plant's ability to absorb soil moisture. Low temperatures may produce chilling or frost damage (Fig. 2), which appears as dark watersoaked areas on wilted leaves or plants, or they may stunt general plant development if these low temperatures persist for an extended period. High temperatures may induce flower abortion (21), increase the rate of evapotranspiration, and cause plant wilt if there is an insufficient supply of

Fig. 2 - Frost damage to climbing bean cultivar grown in association with maize.

soil moisture or limited root growth. High temperatures and winds may compound plant stresses from low soil moisture by physically inducing soil aggregation, cracks and subsequent root damage (13). Seedlings may develop basal lesions at the soil line if the soil surface layer becomes too hot (13, 18, 21, 25).

Sunscald

Sunscald of bean leaves, stems, branches and pods may occur during periods of intense sunlight (ultraviolet wave length), especially following conditions of high humidity and cloud cover (18, 25). High temperatures also may induce sunscald damage (18). Symptoms appear as small water-soaked spots on the exposed side of the plant. The spots become reddish or brown, may coalesce, and form large necrotic or discolored lesions on affected plant structures (Fig. 3). These symptoms may resemble those caused by the tropical spider mite and air pollutants.

Bean development also can be influenced by light intensity, quality and duration (photoperiod). Reduced light can cause etiolation as plants produce succulent growth with long stem internodes, and often reduced chlorophyll content and flower production (13, 18). Cultivars which are sensitive to photoperiod do not flower normally, and often produce few pods late in the growing season when planted at high latitudes. Plants often appear healthy and green unless low temperatures cause abnormalities (personal communication, Dr. D.R. Laing, CIAT Bean Physiologist). High light intensity can scorch or burn leaves and pods (russet), cause flower and pod abortion, and increase damage caused by chemical spray droplets or air pollution, especially that caused by photochemical pollutants (13, 25).

Fig. 3 - Sunscald damage on bean pods.

Wind

Wind speed and direction can affect plant development. Evapotranspiration rates may be increased by consistent winds and aggravate plant moisture stress (13). Violent plant movement may damage roots and predispose them to subsequent root rot problems, break stems and branches, and cause plant lodging, especially if soil moisture is high (13).

Beans also can be damaged by the abrasive action of wind and air-borne soil particles (2, 25). Yield losses of 8% occurred when seedlings sustained leaf damage (Fig. 4), and a 14% yield loss occurred when flowering plants sustained the loss of buds and blossoms, after a 20-minute exposure to winds (15.5 m/sec) in the field (2).

Fig. 4 - Primary leaf damage caused by wind and air-borne soil particles.

Fig. 5 - Baldhead symptoms induced by physically damaged seed.

Physical

Bean plants can be damaged physically during cultivation, application of pesticides, or preparation of irrigation furrows if not properly managed and if bean plants have produced too much vegetation. Wounds on leaves and other plant parts can provide entry sites for various bean pathogens, especially bacteria.

Bean seeds may be mechanically or physically damaged during harvesting, threshing, processing and planting operations, especially when the seed moisture content is low (4, 21, 25). External seed damage may consist of cracked seed coats and cotyledons. Internal damage may consist of detached cotyledons or injury to the hypocotyl, radicle or epicotyl and plumule. When the growing tip is injured or killed, seedlings produce the typical baldhead symptom from which plants may survive only by

Fig. 6 - Leaf variegations caused by a genetical abnormality.

producing buds in the axils of the cotyledons (Fig. 5). A similar symptom, snakehead, may occur from damage by insects or common bacterial blight. Seedlings which survive the effects of mechanical damage often are stunted and yield poorly (4, 25).

Physiological and Genetical Problems

Beans occasionally exhibit physiological and genetical abnormalities which may be confused with symptoms induced by plant pathogens or abiotic factors. Albino seedlings may occur but usually die within a few days, due to their lack of chlorophyll. Leaf variegations may appear as mosaic patterns of green, yellow and white tissue (Fig. 6), and can cause an abnormal development of the plant and pods. Individual leaves or branches may be affected, or the entire plant may express variegations (21, 25). General plant chlorosis and pseudo-mosaic symptoms can be heritable traits. Small chlorotic spots (Yellow Spot) may appear on primary and trifoliate leaves of certain cultivars which still develop normally, and the trait is heritable (25).

A heritable seedling wilt, not caused by root rot, has been reported to occur when primary leaves become pale, bronzed, curl slightly and senesce, resulting in plant death. Internal necrosis is also a heritable trait which produces brown necrotic spots on the flat surface of cotyledons (25). Cripples or abnormal plant development can occur and also may be caused by a genetic abnormality.

Seed coat splitting may take place in certain cultivars and appears to be heritable. Symptoms consist of the uneven growth of cotyledons and the seed coat, which cause the exposed cotyledons to extend beyond the seed coat and appear cone-shaped, roughened and serrated (25). Other factors, such as moisture and temperature, may be involved.

Fig. 7 - Insecticide damage to bean leaves.

Fig. 8 - Paraquat spray-drift damage to beans.

Chemical Problems

Chemical Toxicities

Chemical damage may affect beans during the growing season, especially during germination and seedling development if chemicals are not applied according to manufacturers' recommendations. Toxic concentrations of various chemicals and fertilizer may be placed too close to seeds, creating problems if chemicals do not dissolve and leach rapidly throughout the root zone (13, 25). Insecticides (Fig. 7), Paraquat spray-drift (Fig. 8) and 2,4-D spray-drift (Fig. 9) can produce distinctive necrotic or morphological symptoms on affected leaves or plant parts. Other physiological disorders may be caused by chemicals which contain impurities or products metabolized by soil microorganisms into toxic by-products, or aggravated by specific soil and environmental conditions.

Root injury by herbicides and pesticides may be increased by soil moisture stress, deep planting, soil compaction and mechanically damaged seed (22). Chemically damaged roots often are predisposed to subsequent infection and greater yield loss by root rot pathogens (12, 22, 23, 24).

Air Pollution

Air pollution has become an important problem in many parts of the world where beans are planted near small or large industries which release

Fig. 9 - Damage by 2,4-D spray-drift.

gaseous by-products produced during their processing operations. Other gaseous by-products generated by transportation vehicles or natural environmental processes also can contribute to air pollution. Air pollutants which affect beans include ozone, peroxyacetyl nitrate (PAN), sulfur dioxide, fluorides, solid particles and chlorine. Air pollutants also can influence the interactions between beans and plant pathogens.

Ozone (O_3) is a common air pollutant formed by electrical discharge during thunder storms, the action of sunlight on oxygen, gases liberated by combustion engines and as a by-product of photochemical reactions (6). Yield losses greater than 50% have been reported on dry beans (16). Ozone damage appears on the upper leaf surface first as small watersoaked or necrotic lesions which may coalesce and become bronze or reddish-brown (Fig. 10), resembling sunscald damage (6, 8, 16, 19). Premature senescence and defoliation then may occur, especially when ozone concentrations reach 100 ppb (16). The severity of plant damage is affected by the ozone concentration, cultivar sensitivity, leaf age, light (Fig. 11), temperature, humidity, soil moisture and texture, and plant nutrition (1, 6, 16).

Peroxyacetyl nitrate (PAN) is formed by the photochemical interaction between hydrocarbons emitted by the incomplete combustion of petroleum products and oxides of nitrogen. PAN damage appears on the lower leaf surface inititally as a watersoaked, shiny or silvery symptom (Fig. 12), which eventually becomes bronzed. Symptoms may resemble those induced by frost, sunscald or various insects (6), such as the tropical spider mite.

Sulfur dioxide (SO_2) is formed during the combustion of fossil fuels and can act directly as an air pollutant or combine with water to form sulfuric acid mist (6). SO_2 damage may appear on the upper or lower leaf surface as a dull dark-green watersoaked area which eventually turns necrotic or bleached (Fig. 13) (6, 8). SO_2 damage generally is more serious on younger leaves than on older ones (6), especially when temperature and relative humidity are high (18).

Other air pollutants exist which can damage beans, but generally they are not as common as ozone, PAN or SO_2. Hydrogen fluoride may damage young leaf tips and margins which become necrotic and may cause the leaf edges to curl downwards. Chlorine gas can induce dark green leaf spots or flecks on the upper leaf surface, which later become light tan or brown and may resemble ozone damage. Chlorine also may cause interveinal bleaching similar to SO_2 damage. Hydrochloric acid can cause yellow brown to brown, red or nearly black necrosis (flecks or spots) surrounded by a cream or white border of leaf margins or interveinal tissue on the upper leaf surface. HCl also may cause a glazing on the lower leaf surface which resembles PAN damage. Nitrogen oxide and nitrogen dioxide can cause

Fig. 10- (above) Ozone flecking (50 pphm for 3 hr.) of bean leaves.

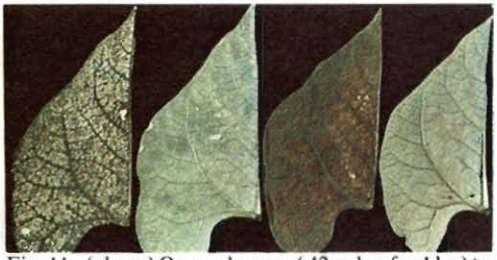

Fig. 11 - (above) Ozone damage (42 pphm for 1 hr.) to bean plants exposed in shade (left) and sun (right) at 22°C.

Fig. 12 - (right) Peroxyacetyl nitrate (PAN) damage to Pinto bean on right.

Fig. 13 - (left) Sulfur dioxide damage (1 pphm for 1 hr.) to Pinto bean.

chlorotic or bleached symptoms on the upper leaf surface. These symptoms may extend to the lower leaf surface and resemble SO_2 damage. Necrotic lesions induced by NO_2 may fall out from the leaf, leaving a shot-hole appearance (6).

Air pollutants are reported to interact with each other or with plant pathogens to alter the type or intensity of damage to beans. Additive, synergistic or antagonistic interactions have occurred between ozone-PAN and ozone-SO_2, depending upon the concentration of each pollutant and sensitivity of plants (8, 9, 10). Various pollutants influence plant pathogens and the resulting symptoms on infected or exposed plants (6).

Rust and halo blight infection can be altered by an interaction with fluorides. For example, smaller, but more numerous rust pustules developed more slowly in the presence of fluorides than in non-exposed and inoculated controls (7). Prior inoculation with bean common mosaic virus reduced the extent of ozone damage when sensitive beans were subsequently exposed to the pollutant (5).

Air pollution damage by ozone has been reduced on various crops, including tobacco and onions, by applying antioxidants such as Dichlone and the dithiocarbamates (10). Bean damage by oxidants has been reduced by application of Benomyl (11, 15) and N-[2-(2-oxo-1-imidazolidinyl) ethyl] -N^{1}- phenylurea or EDU (3). Other control measures may include the identification and development of cultivars which are less sensitive to damage by the various pollutants or their interactions.

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Chapter 19

Nutritional Disorders

R. H. Howeler

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Chapter 19

Nutritional Disorders

Introduction

In Latin America, beans are grown on many different soil types where different nutritional deficiencies or toxicities may limit plant development and yield. In Central America and western South America, beans generally are grown in mountain areas where Andosols (Inceptisols) predominate. Phosphorus and nitrogen deficiencies are most common, although deficiency of minor elements and aluminium/manganese toxicity can limit yield seriously in certain areas.

Between mountain ranges, beans are grown in valleys which generally have alluvial soils of high fertility but which may be low in certain minor elements. In many parts of Venezuela and Brazil, beans are grown on rather acid, low fertility Oxisols and Ultisols. On these soils beans may suffer from aluminium and/or manganese toxicity, as well as a deficiency of phosphorus and occasionally zinc.

A nutritional problem generally is diagnosed with the use of soil and tissue analyses and visual observation of symptoms. Soil samples are taken with a soil auger in the root zone of the plants, and several subsamples from the same general area may be combined into one sample. Leaf samples (without petioles) generally are taken at the top of the plant from the uppermost leaves present at the time of flower initiation. The leaves are oven-dried at 60° to 80°C for 24 to 48 hours, ground and analyzed. If plants show symptoms of nutritional disorders, soil and plant samples are taken from areas with and without symptoms and the analyses compared to identify the element causing the symptoms.

Sometimes, a range of different elements is applied to either soil or foliage to observe any improvement of growth or disappearance of symptoms so as to identify the element which is limiting growth. The latter method is more time-consuming but is useful if laboratories are not available to analyze soil and plant tissue.

In order to use these diagnostic techniques, researchers must recognize symptoms of nutritional disorders and know the critical levels for

deficiency and toxicity symptoms to occur in the soil and plant. These are described later for each element.

Effect of Soil pH on Nutrient Availability

Beans grow best on soils with pH's from 6.5 to 7.5. In this range, most plant nutrients have their maximum availability. However, soils in Latin America have a pH below 6.5, and there are important agricultural areas with a pH above 7.5. Beans will tolerate a low pH of about 4.5 to 5.5 but below that, generally they suffer from aluminium and/or manganese toxicity.

In alkaline soils, beans will tolerate a pH below 8.2 (19), but many soils with high pH also have problems with excess salt (salinity), excess sodium (alkalinity), deficiency of minor elements and poor drainage. According to FAO maps, there are 55 million hectares which have salt problems in South America (20). Salinity can be caused by an excess of sodium chloride, calcium chloride, sodium sulfate and magnesium sulfate. However, it is mainly chloride salts which cause stunted growth, yellowing, flower abortion, hastened maturity and low bean yields (20). Excess sodium salts reduce plant uptake and disperse clay minerals in the soil, thereby causing poor drainage. Beans will tolerate a sodium saturation percentage up to 8 or 10% and an electrical conductivity (measure of salinity) up to 1 mmho/cm. Above these levels, yields drop sharply (19).

Soil salinity problems can be controlled by planting salt-tolerant species and cultivars. In soils with good internal drainage, the application of elemental sulfur or gypsum in combination with large quantities of water may reduce the problems, but at a very high cost.

Nutrient Deficiencies and Toxicities

Aluminium Toxicity

Aluminium toxicity occurs in large areas of Latin America with acid Oxisols, Ultisols and Inceptisols.

Figure 1 shows symptoms of aluminium toxicity. If the toxicity is very severe, plants may die shortly after germination. In less severe conditions, lower leaves become uniformly yellow with necrotic margins, plant growth is stunted and yields depressed. Beans are particularly susceptible to aluminium toxicity. There are large varietal differences for susceptibility (30, 31). Black beans have been reported to be less susceptible than beans of other colors (14). However, this observation is biased by limited sampling of other colors.

Fig. 1 - Stunted plant growth and leaf margin necrosis caused by aluminium toxicity.

Aluminium toxicity is controlled by deep incorporation of agricultural lime, calcium oxide or calcium hydroxide until the pH is above 5.2 to 5.5, or the aluminium content is less than 25 to 30%(25); however, this may not always be economically feasible. Application of 1.5 to 2 ton per hectare of lime will neutralize one milligram equivalent of aluminium per 100 grams of soil. Six tons lime/ha was effective on an acidic volcanic ash soil, as indicated by improved plant growth. Application of basic slag and certain rock phosphates also may reduce aluminium toxicity, while acid-forming fertilizers such as ammonium sulfate and urea may intensify the problem.

Boron Deficiency and Toxicity

Boron deficiency commonly occurs in coarse-textured soils low in organic matter and high in aluminium and iron hydroxides (6, 64). It also can be very serious in alluvial soils with a high pH and low total boron content (15, 16, 17).

Boron deficient plants have thick stems and leaves with yellow and necrotic spots (Fig. 2 and Fig. 3). In less severe cases, leaves are crinkled and curl downwards, similar to symptoms caused by virus or Empoasca

Fig. 2 - Leaf symptoms induced by boron deficiency.

Fig. 3 - Abnormal plant growth (left) induced by boron deficiency.

Fig. 4 - Yellowing and necrosis of leaf margins caused by boron toxicity.

attack. Under conditions of severe boron deficiency, plants remain stunted or die shortly after germination. The critical level for boron deficiency is 20 to 25 ppm in leaves (38) and 0.65 ppm hot-water extractable boron in soil.

Boron deficiency can be controlled by soil application at planting of 1 to 2 kilograms of boron per hectare as Borax, Solubor or other sodium borates, or by foliar application of 1% Solubor or Borax. There are large varietal differences for susceptibility to boron deficiency. Black beans generally have been more susceptible than red beans (17).

Boron toxicity causes yellowing and necrosis of the margins of primary leaves shortly after emergence (Fig. 4), and of older leaves. The critical level for boron toxicity is 40 to 45 ppm in leaves and 1.6 ppm in soils (38). Fox (29) reported that beans are more susceptible to boron toxicity than corn, cotton and alfalfa. Toxicity symptoms appear when the soil content exceeds 5 ppm boron. The toxicity generally occurs after non-uniform application of fertilizer or when the fertilizer is band-applied too closely to the seed, especially during dry weather.

Calcium Deficiency

Calcium deficiency is seldom observed in beans, although plant growth and nitrogen fixation can be affected in many acidic soils with a low calcium status (4). Calcium deficiency generally is observed in combination with aluminium toxicity in acid Oxisols and Ultisols. Beans grown in such soils generally respond to liming. The effect is due to a decrease in exchangeable aluminium and/or manganese ions, and the increased availability of calcium, magnesium and molybdenum.

Calcium deficiency symptoms are apparent as the leaves remain dark green with only slight yellowing at the margins and tips and the leaves

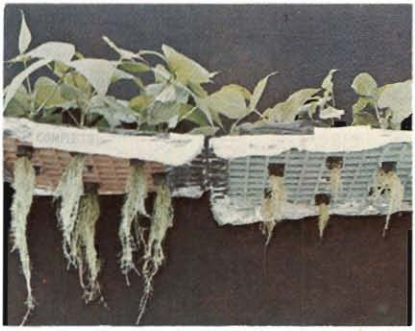

Fig. 5 - Poorly developed root system (right) caused by calcium deficiency.

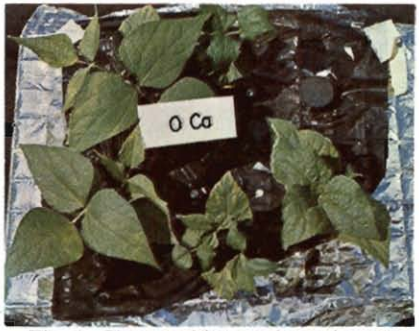

Fig. 6 - Shortened internodes and rosette-type plant growth (foreground) caused by calcium deficiency.

crinkle and curl slightly downward. Calcium deficient plants remain small and root growth may be reduced seriously (Fig. 5). Internodes often are short, producing a rosette-type of plant growth (Fig. 6). Optimum calcium levels in leaves are 2%(1), while values of 5 to 6% have been detected (7, 10).

A critical calcium level of 1.44% occurred in upper mature leaves at flower initiation (18). Since little calcium retranslocation occurs within the plant, apical leaves depend upon a continuous calcium uptake by the root system (8, 9, 41). Calcium contents of leaves decrease with increasing potassium applications (28).

Calcium deficiency is controlled by deep incorporation of calcitic or dolomitic lime or calcium oxide or hydroxide. Low rates, such as 500 kg/ha generally are sufficient to relieve calcium deficiency, but higher rates often are employed to neutralize toxic amounts of aluminium. Calcium phosphate sources, such as basic slag, rock phosphate and superphosphate, may contribute significantly to calcium nutrition.

Copper Deficiency

Copper deficiency has occurred in the Everglades of Florida (62), and in organic or very sandy soils, but it has not been studied in Latin America. Beans are relatively insensitive to copper deficiency when compared to other crops (43).

Copper deficient bean plants are stunted, have shortened internodes, and young leaves become gray or blue-green. Normal copper content of leaves is 15 to 25 ppm in upper leaves.

Copper deficiency is controlled by soil applications of 5 to 10 kg copper/ha as copper sulfate. Foliar applications of 0.1% copper as copper sulfate or copper chelates also are effective.

Iron Deficiency

Iron deficiency is not common but can occur in certain organic soils or mineral soils with a high pH, especially if free calcium carbonate is present.

Iron deficient plants have light yellow to white upper leaves with veins which are initially green (Fig. 7). Normal iron levels in bean leaves may reach 100-800 ppm (7, 10).

Iron deficiency can be controlled by applying iron ethylenediaminotetraacetic acid (EDTA) or other chelates to the soil. Inorganic iron is precipitated easily, especially in high pH soils (37). The application of EDDHA (ethylene diaminedi-o-hydroxyphenylacetic acid) increased iron transport within the plant by decreasing copper uptake, while DTPA (diethylenetriaminepentaacetic acid) increased the uptake of copper, manganese, zinc and iron (63). Foliar application of iron chelates also can control iron deficiency if initial plant growth has not been affected greatly by the deficiency.

Magnesium Deficiency

Magnesium is a basic component of chlorophyll, and optimum levels therefore are vital to photosynthesis. Magnesium deficiency commonly occurs in acid infertile soils with low base status and in volcanic ash soils relatively high in calcium and potassium.

Interveinal chlorosis and necrosis appear first on older leaves (Fig. 8), later spreading over the entire leaf and to younger foliage (Fig. 9). Magnesium is not very mobile. During stress more magnesium goes to younger leaves, thereby causing a deficiency in older leaves. Magnesium deficient plants commonly contain 0.22 to 0.3% magnesium in leaves (18, 56), while normal plants contain 0.35 to 1.3% (7, 10).

Magnesium deficiency can be controlled by soil application of 10 to 20 kilograms of magnesium per hectare as dolomitic lime, magnesium oxide or magnesium sulfate; or as a foliar application of 1% magnesium sulfate solution if the deficiency is not too serious. Lime and magnesium oxide should be broadcast and incorporated, while magnesium sulfate may be more effective when band-applied. Magnesium is absorbed rapidly by primary leaves but is not readily translocated (12). However, it is rapidly distributed throughout the plant when applied to the root system.

Manganese Deficiency and Toxicity

Manganese deficiency occurs in organic soils, mineral soils with a high pH or over-limed acidic soils (27). High calcium levels depress both iron

Fig. 7 - Interveinal chlorosis of leaves caused by iron deficiency.

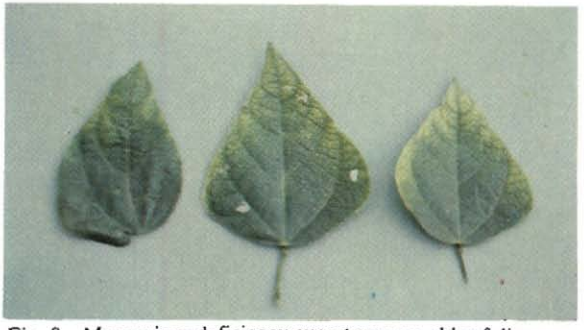

Fig. 8 - Magnesium deficiency symptoms on older foliage.

Fig. 9 - Magnesium deficiency symptoms on young foliage.

and manganese uptake. Under these conditions, the optimum iron: manganese ratio is nearly 2.

Manganese deficient plants are stunted and upper leaves become golden-yellow between small veins, giving a mottled appearance (Fig. 10). Deficient plants contain less than 30 ppm manganese, while normal plants may contain 75 to 250 ppm.

Manganese deficiency can be controlled by soil application of 5 to 10 kilograms manganese per hectare as manganese sulfate or manganous oxide (27), or by foliar applications of manganese chelates. Application of manganese-ethylenediaminotetraacetic acid (EDTA) was not effective, because it increased iron uptake and induced more severe manganese deficiency in organic soils (40). Manganese and zinc are absorbed primarily during the first 40 days of plant growth (5).

Manganese toxicity has been observed in poorly drained, acidic volcanic ash soils in Colombia (18) and in hydromorphic soils in Brazil (23).

Figure 11 shows symptoms of manganese toxicity which include interveinal chlorosis of young leaves. In more severe cases, plants become completely chlorotic, and the upper leaves are small, crinkle and curl downwards (Fig. 12). Symptoms of manganese toxicity are easily confused with those of zinc and magnesium deficiency.

Both manganese toxicity and magnesium deficiency occur in acid soils, but the former produces symptoms in the young leaves while the latter affects the older leaves. Zinc deficiency is more common in high pH soils. Beans are more susceptible to manganese toxicity than corn, and the toxicity seriously affects plant growth, nodule formation and nitrogen fixation (23). Plants suffering from manganese toxicity may contain 1000-3000 ppm (18).

Manganese toxicity can be controlled by liming (18, 23) and by improving field drainage.

Nitrogen Deficiency

Although beans are a legume and therefore capable of symbiotic nitrogen fixation with the appropriate *Rhizobium* strain (33, 34), soil, varietal or inoculation difficulties can limit fixation (16, 23, 58), thereby forcing the plant to rely on soil or fertilizer nitrogen. Nitrogen deficiency is most common in soils with low organic matter. It also is found in acidic soils in which toxic levels of aluminium or manganese, or deficient levels of calcium and magnesium, limit microbiological decomposition of organic matter and nitrogen fixation by *Rhizobium*.

Fig. 10- Manganese deficiency symptoms in bean.

Fig. 11 - Interveinal chlorosis caused by manganese toxicity.

Fig. 12 - Plant symptoms induced by severe manganese toxicity.

Fig. 13 - Plant chlorosis induced by nitrogen deficiency.

Nitrogen deficiency symptoms are evident when leaves near the bottom of the plant turn pale green and eventually yellow, and the discoloration progresses gradually upward (Fig. 13). Plant growth is stunted and yields may be affected. Upper leaves of plants which exhibit deficiency symptoms generally have a nitrogen çontent of less than 3% during flower initiation (10, 56), compared to an optimum of 5% in normal plants (44). Carvajal (13) reported that petioles are more useful in the diagnosis of nitrogen deficiency than are leaf blades. He reports critical petiole levels of 600 ppm for nitrates, 200 ppm for soluble organic nitrogen and 800 ppm for total soluble nitrogen.

Nitrogen deficiency may be controlled by applying a nitrogen fertilizer or by the incorporation of animal manure (59) and green manure (2, 48, 50, 52). No significant differences have been noted between nitrogen sources such as urea, ammonium nitrate, sodium nitrate or calcium ammonium nitrate (47, 58), or between application times (47). On acid soils, sources such as calcium ammonium nitrate, and on alkaline soils, sources such as ammonium sulfate may be beneficial. In general, however, the choice of the nitrogen source is determined by its cost per kilogram of useable nitrogen. Responses to nitrogen application rates varied from no response in many trials in Brazil (25, 35, 51), to large responses to levels as high as 200 (24) and 400 kilograms of nitrogen per hectare (17). Of 232 NPK trials conducted in Brazil, only 67 showed a positive response to nitrogen fertilization (45). Nitrogen fertilizers generally are band-applied at or shortly after seeding, or as a split application at seeding and flower initiation.

In an acid, volcanic ash soil in Colombia, a negative response (fertilizer burn) occurred to band application of urea above 80 kilograms of nitrogen per hectare during a drought. In the same soil, application of 320 and 640 kilograms of nitrogen per hectare produced negative results due to a lowering of soil pH and a subsequent induction of manganese toxicity. Manganese levels in leaves increased from 250 ppm in the check to 600 ppm with the high nitrogen application (19).

In soils where phosphorus is the principal limiting factor, beans may not respond to nitrogen until sufficient phosphorus is applied (61). Nitrogen fixation may be ineffective in the absence of adequate amounts of lime and phosphorus (16, 23, 55), since *Rhizobium* spp. are sensitive to high aluminium or manganese levels and low calcium and phosphorus levels. Liming may increase the efficiency of nitrogen fertilizer application (55) and nitrogen fixation (23). Whenever soil and temperature conditions are conducive to nitrogen fixation, it may be advisable to inoculate seed with *Rhizobium* to replace or supplement chemical nitrogen fertilizers.

Phosphorus Deficiency

Phosphorus deficiency probably is the most common nutritional problem of beans in Latin America. Phosphorus deficiency limits bean yields in many areas of Brazil, especially in the Campo Cerrado (35), in the Oxisol and Ultisol soils of Puerto Rico (1), and in Andosol soils of Colombia (16, 17) and Central America (22, 46).

Phosphorus deficient beans are stunted, have few branches (Fig. 14) and lower leaves are yellow and necrotic before senescing (Fig. 15). Upper

Fig. 14 - (left) Stunted plant growth and sparse branching induced by increasing levels of phosphorus deficiency, left to right.

Fig. 15 - (right) Chlorotic and necrotic leaf symptoms induced by phosphorus deficiency.

Fig. 16- Reduced flowering and small leaf formation caused by phosphorus deficiency.

leaves often are dark green but small. Phosphorus deficiency reduces flowering and affects maturation (Fig. 16). Plants become taller and more vigorous when more phosphorus is applied.

Deficient plants generally contain less than 0.2% phosphorus in their leaves (56). In the uppermost mature leaves, phosphorus contents of 0.2 (21) to 0.4% (44) are optimum levels during the 10% flowering stage. At CIAT (17) the critical level was calculated to be 0.35% phosphorus. The critical phosphorus content (Mehlich extractant) of soils in Minas Gerais (Brazil) was 8 ppm (11), while at CIAT (18) the critical level was found to be 10 to 15 ppm (Olson, Bray I and II extractants).

Phosphorus deficiency generally is corrected by applying phosphorus fertilizers such as triple superphosphate, single superphosphate, rock phosphate or basic slag. These materials should be broadcast and incorporated, except for the superphosphate which should be applied in bands in high phosphorus-fixing soils. Best results generally are obtained by application of triple superphosphate or single superphosphate in soils that also are sulfur deficient. Basic slag and rock phosphates are better suited to acid soils where their relatively large calcium or calcium carbonate content can have a neutralizing effect. The effectiveness of ground rock phosphates varies considerably depending on the crystalline structure of the mined rock. The phosphorus availability of each source is determined by its solubility in ammonium citrate. Bean experiments in Colombia showed a good correlation between this solubility index and the agronomic effectiveness of rock phosphates (18).

The phosphorus availability of rock phosphates can be improved by acidifying them partially with sulfuric acid or by mixing them with sulfur and sulfur-producing bacteria (17, 18). In most soils, beans respond to low

phosphorus application rates (22, 35), while in some soils beans respond to 400 kilograms P_2O_5 per hectare (18).

In a high phosphorus fixing soil of Colombia, beans responded to broadcast applications of triple superphosphate as high as 2060 kilograms P_2O_5 per hectare. However, when the phosphorus was band-applied, similar yields could be obtained with 300 kilograms of P_2O_5 per hectare (19). Thus, in phosphorus-fixing soils, highly soluble sources, such as triple superphosphate, should be band-applied to reduce the soil/fertilizer contact. Less soluble sources, such as basic slag and rock phosphates, need good soil contact to dissolve and are more effective when broadcast and incorporated (19, 57).

In Brazil, beans responded positively to phosphorus application in 103 of 232 trials (45). However, high phosphorus applications may induce zinc deficiency (3, 42).

Potassium Deficiency

Potassium deficiency seldom is observed in beans, but it can occur in infertile Oxisols and Ultisols, or in soils high in calcium and magnesium. In Brazil, a positive potassium response was obtained in only 15 of 232 NPK trials (45).

The symptoms of potassium deficiency consist typically of yellowing and necrosis of leaf tips and margins. These appear first in lower leaves and gradually extend upward (Fig. 17). Necrotic spotting may occur in cases of severe deficiency. The optimum leaf content is 2% potassium (44). Blasco and Pinchinat (10) and Berrios and Bergman (7) report that slightly higher levels occur in field-grown beans. Deficient plants have less than 2%

Fig. 17 - Leaf symptoms induced by potassium deficiency.

Fig. 18- Chlorotic leaf symptoms caused by a deficiency of sulfur.

potassium in upper leaves at flower initiation, and this level may be lower in plants grown on high calcium or magnesium soils.

Potassium deficiency can be controlled by band application at planting of 50 to 100 kilograms potash (K_2O) per hectare in the form of either potassium chloride or potassium sulfate. The sulfate form is recommended for soils which are low in available sulfur.

Sulfur Deficiency

Sulfur deficiency is not common in Latin America but may occur in infertile Oxisols and Ultisols, especially those far removed from industrial centers (49).

Symptoms of sulfur deficiency are evident as uniformly yellow upper leaves (Fig. 18), similar to symptoms caused by nitrogen deficiency. Although top growth is reduced, root growth is little affected by sulfur deficiency. Sulfur deficiency occurs in soybeans if plants contain less than 0.15% sulfur (32), while in beans the critical level is about 0.2 to 0.25% (19). A proper nitrogen: sulfur balance is important for protein formation (60). The optimum nitrogen: sulfur ratio in bean tops is near 15:1. Sulfur deficiency causes an accumulation of inorganic and amide nitrogen in leaves and inhibits protein synthesis. In sulfur deficient soils, nitrogen fertilization should be accompanied by sulfate application at a ratio of nitrogen: sulfur of 15:1.

Sulfur deficiency can be controlled by applying 10-20 kg/ha of elemental sulfur, or by using sulfur-containing fertilizers such as ammonium sulfate, simple superphosphate, potassium sulfate or the application of elemental sulfur. Certain fungicides, such as Elosal, may contribute to the sulfur nutrition of the plant.

Fig. 19 - Interveinal chlorosis of younger leaves induced by zinc deficiency.

Fig. 20 - Symptoms of zinc deficiency on older leaves.

Zinc Deficiency

Zinc deficiency occurs in soils with a high pH or in acid soils that have had high rates of lime and/or phosphorus applications.

Zinc deficiency symptoms begin as an interveinal yellowing of younger leaves (Fig. 19) and older leaves (Fig. 20) which may advance into necrotic spots at a later stage.

The critical level of zinc in bean tissue is 15 to 20 ppm (42), while normal levels are 42 to 50 ppm zinc (39). Levels greater than 120 to 140 ppm zinc can decrease yields (3). Zinc deficiency may be induced by large applications of lime, phosphorus, iron (3) or copper (53, 54).

Cultivars differ in susceptibility to zinc deficiency. A low zinc supply reduced the content of starch and soluble starch synthetase of a cultivar susceptible to zinc deficiency, suggesting that zinc may be essential for starch synthesis (39).

Zinc deficiency can be controlled by soil application of 5 to 10 kilograms of zinc/ha as zinc sulfate (3), or foliar application of 0.3 to 0.5% zinc sulfate or zinc chelates (3, 36). Soil application of zinc sources should be hand-mixed, because incorporation into fertilizer granules reduces their solubility (26), except when mixed with ammonium polyphosphates.

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Chapter 20

Insects and Other Bean Pests in Latin America

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Chapter 20

Insects and Other Bean Pests in Latin America

Introduction

Pests take their toll of bean production as in any crop, both before and after harvest. Attempts to reduce these losses through pesticides have been relied upon less in bean production than in other crops. Bean production in Latin America occurs principally on small holdings where growers often have limited economic resources, conditions not conducive to programmed pesticide use. Moreover, beans often are grown in association with other crops, which may help to stabilize insect populations. While such factors favor an integrated approach to insect control, the short growing seasons and rapid crop turnover in beans may not suit a stable ecosystem, which is desirable for effective pest management practices.

This chapter reviews pertinent literature available on bean pests in Latin America, with emphasis on bean pest ecology and non-chemical control methods. Since the Latin American literature contains no information for some pests, references are cited from other regions on crops besides beans.

Ruppel and Idrobo (100) listed a total of 208 insect species which attack beans, while Mancia and Cortez (65) list more than 400 insect species which are found on bean plants. Bonnefil (6) considers 15 insect species to be economically important in Central America. Most bean pests are omnivorous, attacking several cultivated legumes or other crops. The most important bean pests reported in the literature and according to the authors' observations are listed in Table 1. The given division cannot be maintained strictly, since the Mexican bean beetle and chrysomelids also may attack young pods while *Epinotia* and *Heliothis* spp. may also attack leaves and buds. Not all pests listed are insects, such as slugs and mites.

Fig. 1- Geographical distribution of principal bean pests in Latin America.

Distribution of Important Insect Pests

The bean pest complex varies greatly throughout Latin America and is not well documented. However, Gutierrez *et al.* (43) reported that the leafhopper is the most widely distributed insect in Latin America, with chrysomelids (mainly *Diabrotica balteata*), cutworms, crickets, pod damaging insects (especially *Apion godmani*) and storage insects listed in decreasing levels of importance (Table 2). The authors gave no estimates of the economic importance of these pests. The leafhopper is the most important bean insect in Central America (6), followed in importance by the chrysomelids (Table 3).

A simplified distribution of the principal bean pests is shown in Figure 1. For example, the Mexican bean beetle occurs in Mexico, the Guatemalan highlands and Nicaragua. The bean-pod weevil (*Apion* spp.) still is a problem as far south as northern Nicaragua. Snails, not shown, are a severe problem to bean culture in El Salvador and Honduras.

Stored grain insects, *Acanthoscelides obtectus* and *Zabrotes subfasciatus,* are found in all areas of Latin America. *A. obtectus* occurs primarily in higher altitudes in both fields and warehouses in Chile, Argentina, Peru and Colombian mountains, while *Z. subfasciatus* is found primarily in beans stored at lower elevations.

Economic Losses

Potential loss from insect damage varies greatly between and among regions, due to differences in planting dates, cultivars and cultural practices. Miranda (81) reported insect losses of 33-83% when non-treated plots were compared to treated plots. Losses from *Apion* in El Salvador were 94% (67), although average losses are lower. In 16 insecticidal trials in Central America, controls yielded an average of 47% less than the highest yielding insecticidal treatment, with greatest losses inflicted by leafhoppers (Table 4). These figures probably over-estimate the importance of insects in bean culture, since such insecticidal trials normally are planted to coincide with the highest levels of insect attack. This was apparent in studies with Diacol-Calima, which is susceptible to leafhopper attack and which sustained losses of 14-23% (average 22%) during the rainy season, while dry season losses were 73-95%. The average loss was 76% (Fig. 2). Studies by

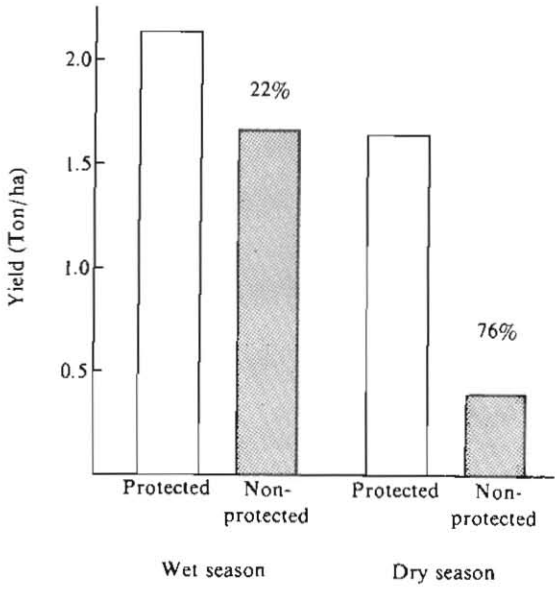


Fig. 2- Average yield of Diacol-Calima of best insecticidal treatment compared with nonprotected plots in wet and dry season (Ave. 3 trials in each season).

Pinstrup-Andersen *et al.* (87) in the Cauca Valley in Colombia estimated that *Empoasca kraemeri* caused an average 10.8% crop loss on 12,000 ha of beans grown in 1974, resulting in a loss of $749,000 in U.S. currency for that growing season.

Economic Threshold Populations

An important aspect of pest management is the level of damage that can be tolerated economically. Greene and Minnick (39) obtained a 37% yield reduction due to 25% defoliation one week before flowering, while 25-33% defoliation during flowering did not reduce yield. Results have shown that defoliations between 30 and 45 days after planting (beginning of flowering to end of flowering) were most damaging to yield (15). Yield losses greater than 35% occurred only when more than 60% of the foliage was removed. Leafhopper studies at CIAT (15) indicated a 6.4% yield loss occurred for each additional nymph present per leaf (Fig. 3). These data indicate that beans can withstand certain levels of defoliation before yield losses occur.

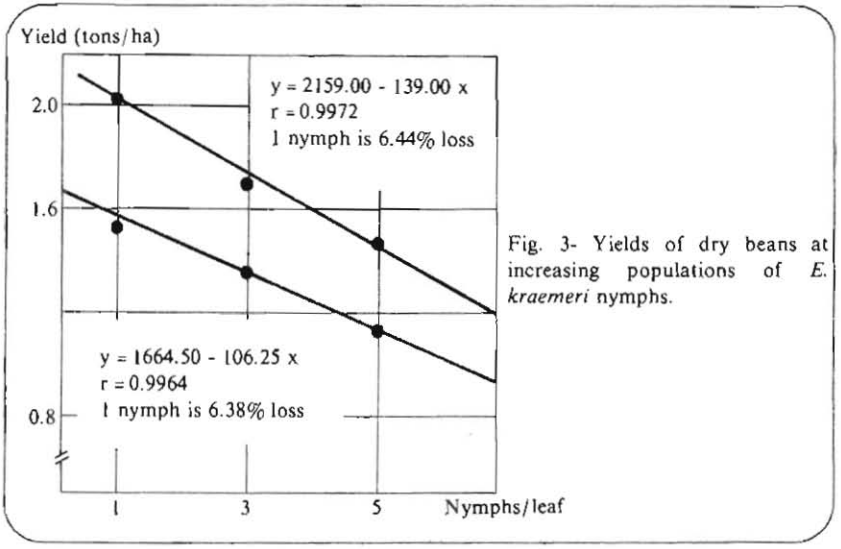


Fig. 3- Yields of dry beans at increasing populations of *E. kraemeri* nymphs.

Seedling-Attacking Insects

Seed Corn Maggot

Hylemya cilicrura (Rondani) (Diptera: Anthomyiidae).

The seed corn maggot is a bean pest in Chile, Mexico and areas of the United States and Canada. The genus has been named *Delia, Phorbia* and *Hylemya*. Other species reported on beans include *H. platura* and *H. liturata*. *H. cilicrura* and *H. liturata* are closely related (79), although McLeod (76) separated them by differences in nutritional requirements and infertility of interspecific hybrids.

Common names frequently used for the seed corn maggot in Latin America include mosca de la semilla, mosca de la raíz and gusano de la semilla.

Oviposition takes place near seeds or plants in the soil. Larvae feed on bean seeds (Fig. 4) or seedlings (Fig. 5) and pupate in the soil (79). Harris *et al.* (46) reported an incubation period of two days, a larval stage of nine days and a pupal stage of eight to 12 days at 21° - 23°C. Crops susceptible to larval attack include beans, maize, potatoes, beets, pepper, tobacco and other vegetables (79). The scientists also found evidence that above 24°C, pupae enter estivation. The average female produced 268 eggs.

Fig. 4- (above) Larvae of seed corn maggot, *Hylemya cilicrura* feeding on a bean seedling.

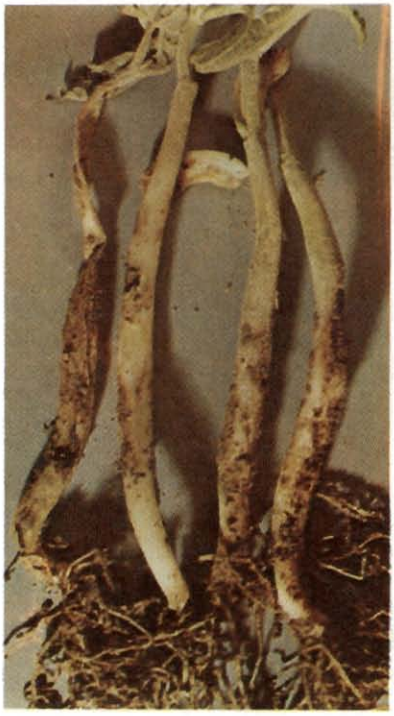


Fig. 5- (right) Damage caused by the seed corn maggot on bean seedlings.

Adult females (the adult fly resembles the housefly) were abundant on dandelion and aphid honeydew and were less active at temperatures higher than 32°C. Adults are attracted to newly disturbed soil and organic matter in which their larvae can develop, for example, in decaying spinach. Size of the adult population is not necessarily related to severity of seed damage.

Hertveldt and Vulsteke (50) report 20-30% germination loss when one or two larvae were present per bean seed, while two or three larvae reduced germination 50%. Damage includes poor germination and production of deformed seedlings (baldheads) and occurs when larvae feed between the

cotyledons, thereby injuring the embryo. Larvae also can penetrate the stem of germinating seeds and damage young plants.

Late planting in Chile causes rapid seed germination and reduces exposure time to *Hylemya* spp. In three spring plantings at one month intervals the percentage of plants which germinated and were damaged by *Hylemya* spp. was reduced from 27 to 9 to 2%, respectively (C. Quiroz, personal communication). Humid soils with high organic matter were more likely to attract ovipositing females, especially if the field was recently plowed.

Biological control is reported to operate only at low levels and does not provide effective control (79).

Plant resistance to seed corn maggots is reported by Vea and Eckenrode (120). To insure the high larval population needed for screening, they planted during periods of high fly population and increased natural infestation by band-applying meat and bone meal. The bean lines C-2114-12 and P.I. 165426 showed 0 and 4% stand loss, respectively, while the susceptible cultivar Sprite had an 88% loss. The percentage of emerged seedling damage also was lowest for P.I. 165426 and C-2114-12. White-seeded cultivars were susceptible. Rapid emergence and hard seed coats contributed to resistance. Guevara (40) also reported differences in level of attack by *Hylemya* spp., and black-seeded cultivars were less damaged than yellow-seeded cultivars.

For many years, a combined Dieldrin + fungicide seed dressing was the standard treatment for control of *Hylemya* spp. (36). Repeated exposure of the maggot to chlorinated hydrocarbons has led to development of insect resistance to the chemical. Insecticides such as Diazinon, Carbofuran and Chlorpyrifos applied as granules in the furrow or as a seed slurry can control the larvae effectively (24). C. Quiroz (personal communication) obtained better control with Carbofuran than with Aldrin when applied as a granule at planting time in Chile.

Cutworms, Whitegrubs, Crickets

Many species of cutworms damage beans by causing stand losses as larvae sever the stems of young seedlings (Fig. 6). Older plants can be damaged by stem girdling (Fig. 7), which predisposes plants to wind breakage. Common cutworm genera include *Agrotis, Feltia* and *Spodoptera*. General biology and control of cutworms are discussed by Metcalf and Flint (78).

Fig. 6- Bean plant severed by a cutworm larva.

Fig. 7- Cutworm damage on an older bean plant.

Common names frequently used for cutworms in Latin America include trozadores, cortadores, nocheros, rosquillas, lagarta militar and lagarta rosca. Common names frequently used for whitegrubs include gallinaciegas, chizas and mojojoys. Common names frequently used for crickets include grillos and grillotopos.

Cutworm attack in beans occurs erratically and is difficult to predict. Therefore, it is better to control cutworms with baits applied in the late afternoon near the plants than to use the common preventive chemical control with Aldrin. A formulation of 25 kg sawdust (or maize flour), 3 liters molasses and 1 kg Trichlorfon per hectare also is effective in controlling crickets and millipedes.

In preliminary trials at CIAT, it appeared that beans were not a preferred host for *Spodoptera frugiperda,* which is one of the most important cutworm species. In associated cropping of beans with maize, cutworm damage in beans was nearly zero. Likewise, cutworm damage was significantly greater (71%) in maize monoculture than in maize associated with beans.

Whitegrubs (Fig. 8), mainly a problem in crops following pasture, can be controlled by proper land preparation. Chemical control is possible with Carbofuran or Disulfoton band-applied (0.9 kg a.i./ha) and with Aldrin incorporated into the soil.

Fig. 8- Whitegrub larvae extracted from the base of infected plants.

Crickets and molecrickets also are listed as pests of beans (Fig. 9) in some countries (90), but they seldom cause significant economic losses.

Lesser Corn Stalk Borer

Elasmopalpus lignosellus (Zeller) (Lepidoptera: Pyralidae).

E. lignosellus is a serious bean pest in parts of Peru (F. Avalos, personal communication), Brazil (18) and other countries in Latin America. It attacks a variety of weeds and cultivated plants including maize, sugar cane, cereals, legumes and nutgrass.

Common names frequently used for the lesser corn stalk borer in Latin America include coralillo, barrenador del tallo, elasmo and lagarta elasmo.

Larvae (Fig. 10) enter the stem just below the soil surface and tunnel upwards (Fig. 11), causing plant mortality and subsequent stand loss. The adult oviposits eggs singly on the leaves or stems, or in the soil. The six larval instars are passed in 13-24 days, after which they pupate in the soil (59). Dupree (23) found little evidence of stem boring activity prior to the third instar.

Control is achieved with clean fallowing for prolonged periods or with heavy irrigation (11, 124). Leuck and Dupree (60) observed egg and larval parasitism by species of Tachinidae, Braconidae and Ichneumonidae on larvae collected from cowpeas. Chemical control should be started at planting time and granular insecticides should be directed near the seeds to kill larvae present in the soil.

Leaf-Feeding Insects

Chrysomelids

Many species of Chrysomelids attack beans in Latin America, the most prevalent genera (Fig. 12) being *Diabrotica, Neobrotica, Cerotoma* and *Andrector* (6). *D. balteata* LeConte probably is the most abundant species. Ruppel and Idrobo (100) list 36 species of Chrysomelids, including the additional genera *Epitrix, Chalepus, Colaspis, Maecolaspis, Systena* and others. This review will concentrate mostly on *D. balteata* (Fig. 13), the banded cucumber beetle.

Common names frequently used for chrysomelids in Latin America include crisomelidos, cucarroncitos de las hojas, diabroticas, doradillas, tortuguillas, vaguitas and vaguinhas.

Fig. 9- Typical cricket damage on a bean plant.

Fig. 10- Mature larvae of the lesser corn stalk borer, *Elasmopalpus lignosellus*.

Fig. 11- (right) Damage caused by lesser corn stalk borer.

Fig. 12- (below) Color variation in adults of Chrysomelids.

Fig. 13- (lower right) Adult *Diabrotica balteata*.

Most damage by Chrysomelids occurs during the seedling stage (Fig. 14) when the insect consumes a relatively high percentage of foliage. Boonekamp (7) concluded that feeding by adult Chrysomelids has little effect on bean yield except when attack occurs during the first two weeks after planting or, to a lesser extent, during the flowering stage of the plants. Larvae also may damage bean roots and root nodules containing *Rhizobium* (nitrogen-fixing bacteria). Sometimes adults feed on young pods. Chrysomelids also are known to transmit bean rugose mosaic virus (29).

Females (one to two weeks old) oviposit eggs singly or in clusters of up to 12 eggs in soil cracks or beneath plant debris. An adult may lay more than 800 eggs during a lifespan of 17-44 days (average 26 days). Oviposition usually occurs at intervals of a few days. Eggs hatch in eight days at 21°C and six days at 27°C. The three larval stages are passed in 11 days on soybean roots at 27°C. Pupae form in a pupal cell in the ground, and this stage lasts seven days at 27°C (88). Young and Candia (130) reported an incubation period of five to nine days, a larval period of 17 days, and a prepupal-pupal stage of nine to 17 days. The maximum egg production by adults that fed on bean leaves was 144 per female. Pulido and López (91) found an average of 326 eggs produced when adults were fed only soybean leaves and 975 eggs when adults were fed soybean leaves, flowers and young pods. When fed soybean leaves, adults lived for 69-112 days. Harris (48) observed adult color variation within *D. balteata* and especially within *Cerotoma facialis* (Erichson).

While adults feed on many plants including maize (silk and pollen) and beans (leaves), the larvae may develop on roots of maize, beans (Fig. 15) and other crops. Pulido and López (91) list 32 host plants. Of these, maize and beans with five other plant species are listed as hosts for adults and larvae. Harris (48) reported that common bean-field weeds in the Cauca Valley serve as larval hosts and include *Amaranthus dubius, Leptochloa*

Fig. 14- Severe damage caused by adult Chrysomelids.

Fig. 15- Larval damage of *Cerotoma facialis* on bean hypocotyl.

filiformis, Echinochloa colonum and *Rottboellia exaltata*. He found *D. balteata* and *C. facialis* adults preferred beans rather than soybeans, peanuts, cotton or maize. Larvae of *D. balteata* can be reared on maize but not on bean roots, while those of *C. facialis* can be reared on beans but not on maize roots (7). Young (129) reported that in Mexico *D. balteata* adults have a feeding preference for young bean plants and an oviposition preference for young maize plants. When bean and maize were grown in association, *C. facialis* larvae had a high preference for bean roots and *D. balteata* larvae for maize roots (7).

Fig. 16- Adult Reduviid preying on an adult Chrysomelid.

Predation of adult chrysomelids by Reduviids (Fig. 16) often is observed in the field. Young and Candia (130) reported a Tachinid occurred as an adult parasite. Chemical control often is recommended with Carbaryl, Malathion or Dimethoate.

Lepidopterous Leaf Feeders

Several species of Lepidoptera develop on beans. Although larvae commonly are found on beans, populations usually are too low to cause economic damage.

Bean Leafroller

Urbanus (= Eudamus) proteus (L.) (Lepidoptera: Hesperiidae).

The bean leafroller is distributed widely on beans from the United States to Brazil. Greene (37) calculated that yield reduction occurred when more than 725 cm^2 leaf area per plant was destroyed.

Common names frequently used for the bean leafroller in Latin America are gusano fósforo and gusano cabezón.

Although the first three larval stages of the leafroller do not cause appreciable damage, the fourth can reduce yield when more than 26 larvae

Fig. 17- Bean leaf folded by young larva of the bean leafroller.

occur per plant. The fifth instar consumes about 162 cm^2 of leaf area, and economic losses occur when an average of four larvae eat 33% of the total leaf area. Assuming 50% mortality per instar, 141 eggs per plant (a population level seldom observed) would be required to cause significant damage.

The butterfly lays one to six eggs per lower leaf surface. Young larvae then fold and tie a small section of the leaf margins together (Fig. 17) within which they live and pupate. However, often they may feed elsewhere. Larvae are easily recognized by their three dorsal longitudinal lines and larger red-brown head capsule (Fig. 18) (92). Greene (38) reported that in the field only 4% of the eggs reached the fifth instar. At 29.5°C eggs hatched in three days, the larval stage was passed in 15 days and the pupal stage passed in nine days. He observed large numbers of adults on *Lantana camara* flowers and in flowering bean fields. Van Dam and Wilde (119) studied its life cycle in Colombia and found that the egg stage lasted an average of four days while the larval and pupal stages required 23 and 11 days, respectively, to develop. Larvae have been found frequently on beggar weed *(Desmodium tortuosum)* and other *Desmodium* species (92).

Chemical control seldom is justified and natural control by parasites and predators is commonly observed. In Colombia, for example, larval parasitism ranged from 21 to 40% during a one-year study (119).

Fig. 18- Mature larva of bean leafroller, *Eudamus proteus.*

Saltmarsh Caterpillar

Estigmene acrea (Drury) (Lepidoptera: Arctiidae).

The saltmarsh caterpillar, although commonly found on beans, usually is recognized as a pest of cotton, lettuce and sugarbeets (110). Young and Sifuentes (131) report preferred natural hosts include *Amaranthus palmeri* and *Physalis angulata*. The pest also occurs on maize, horticultural crops, soybean, sesame, tobacco, cotton and several weed hosts.

The common name frequently used for the saltmarsh caterpillar in Latin America is gusano peludo.

The adult moth places egg masses of up to 1000 eggs on *A. palmeri*, and larvae develop in 17-19 days. The young larvae aggregate (Fig. 19) and can skeletonize isolated bean plants. Older larvae are solitary, their bodies are covered with setae (Fig. 20), and they pupate on the soil in plant debris. The adult is a white moth with black dots on its wings (131).

Individual plants on which the gregarious stages are passed may be damaged severely, although beans seldom suffer economic damage. In the Cauca Valley in Colombia, 12 Dipterous species caused an average 31% parasitism on larvae (96). Young and Sifuentes (131) reported that coccinellids and malachiids are egg predators, and reduviids are larval predators. Several hymenopterous parasites of larvae also have been reported. Chemical control is seldom justified.

Fig. 20- (above) Mature larva of *Estigmene acrea*.

Fig. 19- (left) Young larvae of the saltmarsh caterpillar aggregated on a bean leaf. Older larvae are solitary.

Hedylepta

Hedylepta (= Lamprosema) indicata (Fabr.) (Lepidoptera: Pyralidae).

H. indicata is a pest of beans, soybeans and other legumes in South America (32, 100). The common name frequently used for *Hedylepta indicata* in Latin America is Hedylepta.

Fig. 22- (above) Mature larva of *Hedylepta indicata*.

Fig. 21- (left) Leaf-feeding damage by *Hedylepta indicata* larva.

Adult moths oviposit on the lower surface of leaves, where a female lays an average of 330 eggs. The eggs hatch in four days, the green larvae (Figs. 21 and 22) develop in 11 days, pupate (Fig. 23), and five days later the adult emerges (52). Larvae feed on the parenchyma of leaves which they weave together (Fig. 24). Therefore, they are protected from exposure to insecticides.

The level of biological control is high. García (32) found more than 85% larval parasitism by *Toxophroides apicalis* (Hymenoptera: Ichneumonidae). A carabid predator of *H. indicata* larvae passes its entire life cycle between the leaves woven together by *Hedylepta* (57). Chemical control is most effective with Methamidophos and Dicrotophos (30), but their use is seldom justified.

Fig. 23- Pupa of *Hedylepta indicata* among leaves woven together by the larva.

Fig. 24- Typical damage caused by *Hedylepta indicata*.

Mexican Bean Beetle

Epilachna varivestis Mulsant (Coleoptera: Coccinellidae).

The Mexican bean beetle is mainly a soybean pest (118), but beans have been damaged in the United States, Mexico, Guatemala and El Salvador (in the latter during the wet season). It differs in behavior from most coccinellids in that larvae and adults feed on foliage, stems and young pods, whereas the family is more commonly predaceous. Synonyms include *Epilachna corrupta* Mulsant and *E. maculiventris* Bland.

The common name frequently used for the Mexican bean beetle in Latin America is conchuela.

In El Salvador, *Phaseolus vulgaris, P. lunatus, P. atropurpureus, Vigna sinensis* and *Glycine max* are hosts (65) while beggarweed also is reported to be a host. Turner (116) reared the beetle on *P. vulgaris, P. coccineus, P. lunatus, V. sinensis* and *Dolichos lablab;* high larval mortality occurred on the latter. He classified *P. aureus* and *Vicia fabae* as immune. *P. aureus, P. mungo* and *P. radiatus* are less preferred hosts than *P. vulgaris* (4, 127). This preference is attributed mainly to the sucrose concentration which serves as an arrestant combined with differences in olfactory action of the foliage (4). LaPidus *et al.* (54) confirmed these results in studies of seeds from resistant and susceptible plants.

Young larvae feed on the lower leaf surface and usually leave the upper epidermis undamaged, while older larvae (Fig. 25) and adults (Fig. 26) often feed over the entire leaf. Third and fourth instar larvae consume more than adults. Stems and pods often are eaten if high population densities exist. The larvae do not chew the leaf tissue, but scrap the tissue, compress it and then swallow only the juices. De la Paz *et al.* (21) concluded that most

Fig. 25- Mature larva of Mexican bean beetle.

Fig. 26- Adult Mexican bean beetle on lower surface of a bean leaf.

damage occurred when young plants were infested. Infestation of 41-day old plants with 25 larvae each, reduced yield 93% more than delaying infestation to 71 days after planting.

The adult female beetle begins oviposition seven to 15 days after copulation and lays yellow to orange-colored eggs on the lower leaf surface in groups of four to 76 (average 52) (100). Mancía and Roman (66) obtained an average of 10 egg batches with 36-54 eggs per batch (average 43). Eggs hatch in six days, the four larval instars are passed in 15-16 days, the prepupal stage in two days and the pupal stage in six or seven days. The yellow larvae are covered with branched spines. Pupation occurs with larvae attached to the lower leaf surface. Adults are copper colored with 16 black spots and live four to six weeks. In El Salvador, the beetle passes four generations on beans from May to November. In the United States, adults hibernate in woodlands and bean debris and are often gregarious (25).

Predators of eggs and the first larval instar include *Coleomegilla maculata* De Geer and *Hippodamia convergens* Guenée. Adults are attacked by the mite, *Coccipolipus macfarlanei* Husband (66), and *C. epilachnae* Smiley also is observed in El Salvador (108). *Pediobius faveolatus* (Crawford) (Hymenoptera: Eulophidae) reduced Mexican bean beetle populations on soybeans (109).

Removal of plant debris and deep plowing are recommended to control the insect. Reduced plant density decreases beetle injury, as egg mass numbers per plant decreased from 1.07 to 0.15 when plant spacing was increased from 5 to 12 cm. Yield reduction was decreased from 23 to 11%, and pod damage also declined (117).

Plant resistance to the Mexican bean beetle has been studied in some countries. In free-choice cage studies on 60 bean and lima bean cultivars, Idaho Refugee and Wade were resistant, losing only 25% foliage, while Bountiful had 62% of the foliage destroyed. The number of eggs and egg masses and adult weights were reduced more than 50% when beetles were reared on resistant versus susceptible lines (10). Wolfenbarger and Sleesman (127) did not observe resistance in *P. vulgaris* material they investigated. They tested Idaho Refugee and Wade and rated them susceptible (8.5 on a 1-9 scale, with 9 most susceptible). Based on leaf feeding damage, the highest level of resistance was found in *Phaseolus aureus*. Nayar and Fraenkel (82) hypothesized that phaseolunatin (a cyanogenic glycoside) attracts beetles when present in low concentrations but may be responsible for resistance in germplasm containing high concentrations of this compound. The entries Puebla 84 *(P. coccineus),*

Guanajuato 18 and Zacatecas 48 *(P. vulgaris)* were resistant (31). Fewer eggs were laid on Gto. 18 and Oax. 61-A. They concluded that antibiosis and non-preference were responsible. More recently, Raina *et al.* (93) found that the cultivars Regal (snapbean), Baby Fordhook (lima) and Baby White (lima) had less than 40% leaf damage and suffered significantly less from attacks than other cultivars tested. Raina *et al.* (93), Thomas (113), Wolfenbarger and Sleesman (127), and Campbell and Brett (10) concluded that lima beans as a group were less preferred than snapbeans.

Cadena and Sifuentes (9) obtained effective chemical control with Carbaryl. Malathion and Methyl Parathion were much less effective. They suggested the first application be made when 25 adults/ha were present, the second application be combined with *Apion* spp. control and a third application be made only if needed. Recommendations in the United States are that farmers spray when one beetle or egg mass is found per 6 foot (1.8 m) row. The beetles are counted on the ground after shaking the plant. Hagen (44) obtained an effective 10-week control with a planting application of insecticides such as Disulfoton, Carbofuran, Phorate, Aldicarb and Fensulfothion.

Piercing Insects

Leafhoppers

Empoasca kraemeri Ross and Moore (Homoptera: Cicadellidae).

E. kraemeri is the most important insect pest of beans. It occurs from Florida and Mexico south to Ecuador and Peru. *E. fabae* and *E. solanae* occur in the United States and Canada but not in South America (97). Other *Empoasca* species in South America include *E. prona, E. aratos* and *E. phaseoli* (6).

Common names frequently used for leafhoppers in Latin America include Empoasca, chicharritas, lorito verde, cigarra, saltahojas and cigarrinha verde.

E. kraemeri does not transmit virus diseases, the only *Empoasca* species known to have this attribute being *E. papayae,* which transmits bunchy top virus of papaya. The only leafhopper known to transmit a bean virus (bean curly top) is the beet leafhopper, *Circulifer tenellus.* The brown leafhopper, *Scaphytopius fuliginous* Osborn, transmits a mycoplasma-like organism to beans and soybeans in Colombia (Refer to Chapter 11).

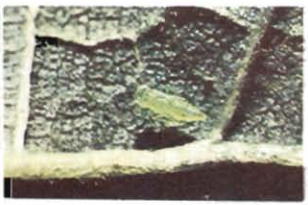


Fig. 28- (above) Nymph of *Empoasca kraemeri.*

Fig. 27- (left) Adults of *Empoasca kraemeri.*

Eggs of *E. kraemeri* hatch in eight or nine days, and the five nymphal instars are passed in eight to 11 days (123). Females and males (Fig. 27) live for 65 and 58 days, respectively. Oviposition ranged from 13-168 eggs (average of 107) per female. The eggs are commonly laid singly on leaf blades, petioles, leaf tissue or stems of bean plants; 50-82% of the eggs laid per plant may be located in the petioles (34). Leafhoppers breed on many cultivated and non-cultivated plants. *Empoasca* spp. nymphs (Fig. 28) have been collected from more than 80 plant species in Colombia.

Plant damage may be caused by physical feeding injury in phloem tissue, although a toxin also may be involved. Plant damage appears as leaf curling and chlorosis, stunted growth (Fig. 29), greatly reduced yield (Fig.

Fig. 29- Typical leaf curling and yellowing damage caused by leafhopper feeding.

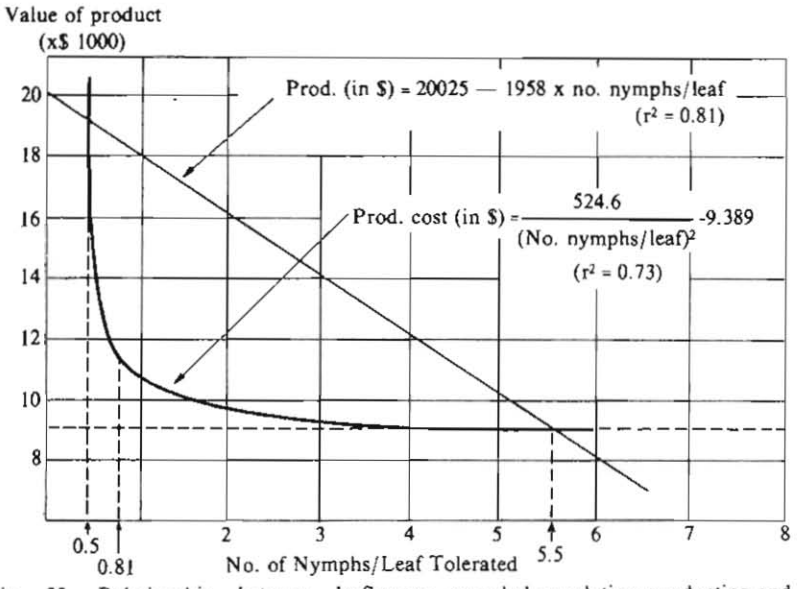


Fig. 30- Relationship between leafhopper nymphal population, production and production costs (in Col. $).

30) or complete crop loss. Leafhopper attack is more severe during hot dry weather associated with insufficient soil moisture. Furthermore, damage by a uniform number of leafhoppers *(E. fabae)* is less during humid weather than during periods of moisture stress (5). Miranda (80) obtained yields of 1182 kg/ha when dry beans were planted December 21, but only 121 kg/ha when beans were planted January 21 in El Salvador. It is assumed that high temperatures and water stress aggravate *Empoasca* spp. damage, especially in areas of Colombia at elevations of 1000-1500 m (99). Screening at CIAT for *Empoasca kraemeri* resistance usually is made during dry or semi-dry seasons when insect populations are highest (14). However, plantings during the late part of the dry season sometimes remain relatively free of damage, and leafhoppers collected at this time caused less damage than those collected earlier.

Various cultural practices often can be manipulated to reduce leafhopper populations and damage. Maize has reduced populations of *Empoasca kraemeri* when beans were planted in association. Leafhopper populations were reduced significantly in plots where maize was planted 20 days prior to beans (72 adults per 90 bean plants) as compared to fields where maize and beans were planted on the same day (133 adults per 90 bean plants). Corn whorl worm *(Spodoptera frugiperda)* populations also were significantly reduced in fields where beans were planted 20 days before maize (eight larvae per 40 maize plants), compared to fields where maize and beans were planted on the same day (26 larvae per 40 maize plants).

Leafhopper adult and nymphal populations were decreased 43 and 70%, respectively, in bean plots with nearly 100% weed cover (16). This reduction in *Empoasca kraemeri* populations was not ascribed to increased parasite or predator populations. Bean yields were comparable in weed-free and weedy plots, the decrease in leafhopper populations being counter-balanced by the increased weed competition (17). Leafhopper populations also were significantly reduced in bean plots surrounded by borders (1 m wide) of grassy weeds such as *Eleusine indica* and *Leptochloa filiformis*.

Mulching and shading also reduced initial *Empoasca kraemeri* populations. Only 18 insects were collected from mulched plots at 20 days after planting, whereas non-mulched plots yielded 103 adults. By 45 days after planting, the beans in the mulched plots were more vigorous than those in the non-mulched plots wherein the leafhopper populations were then highest (16).

Varietal resistance to leafhoppers in beans was reported in the United States for Wells Red Kidney (5) and other materials (71). Idaho Refugee and U.S. Refugee No. 5 are resistant to leafhopper damage by *E. fabae* and *E. kraemeri* (15, 33). Tissot (114) observed equal leafhopper population levels on resistant and susceptible cultivars, which is consistent with results obtained at CIAT.

In the United States, Wolfenbarger and Sleesman (125, 126) evaluated 1619 lines for resistance to *E. fabae* and found that P.I. 151014 had 0.3 nymphs per leaf (lowest count), while Dutch Brown had 19.7 nymphs per leaf (highest count). They found no correlation between number of epidermal hairs and nymphal population per cultivar but reported a 90-96% correlation between nymphal counts and damage estimates (125). A relationship did exist between leafhopper resistance and plant characteristics such as tallness, resistance to BCMV, pink or mottled-colored seed and intermediate maturity (125). The lowest nymphal counts were obtained on *Phaseolus lunatus, Phaseolus aureus* and *V. mungo*. There are barriers to crossing these species with *P. vulgaris*. However, results from interspecific crosses between *P. vulgaris* and *P. coccineus* suggest that resistance may be recessively inherited (128). Chalfant (12) reported a 50% yield reduction when protected and unprotected plots were compared, regardless of the degree of varietal susceptibility.

A major screening program for varietal resistance to *Empoasca kraemeri* has been inititated at CIAT (Fig. 31) where more than 8000 *P. vulgaris* accessions have been tested to date. The selection scheme is based on elimination of highly susceptible materials. Ten test cultivars are planted between rows of ICA-Tui (standard tolerant cultivar). Diacol-Calima or

Fig. 31- Susceptible (left) and resistant (right) entries after exposure to *Empoasca kraemeri*.

ICA-Bunsi are planted around the plot as a susceptible border. ICA-Tui always is rated as grade 2 in a 0-5 damage scale. In wet season plantings, the most resistant bean materials identified yield equally with or without insecticidal protection, while susceptible cultivars suffer losses of up to 40%. Such resistance levels have given adequate protection against *Empoasca* in Peru. However, in the dry season at CIAT, even these materials require insecticidal protection. A breeding program is underway to increase resistance levels within commercially acceptable cultivars.

Correlations have not been obtained at CIAT between nymphal counts and damage scores as reported by Wolfenbarger and Sleesman (125) and Chalfant (12). Populations of the insect are much higher at CIAT than in the United States and susceptible cultivars receive so much damage that leafhoppers avoid them for oviposition (15).

The resistance mechanism is not clearly understood, but tolerance is probably responsible. ICA-Tui has a low degree of non-preference which is lost during no-choice tests. Antibiosis has not been found to be present (122). Hooked trichomes can capture nymphs and may be another resistance mechanism (86). Nymphal mortality of *E. kraemeri* was low on hooked trichomes in studies at CIAT and may be due to decreased trichome density on expanded leaves. By the time leafhopper eggs hatched, the leaves in which they were laid were fully expanded and the trichomes were less dense.

Two egg parasites (*Anagrus* sp. and *Gonatocerus* sp.) and a diyinid nymphal parasite have been reported as natural enemies of *E. kraemeri*, but they do not seem to be very effective. Thus, Gómez and Schoonhoven (34) concluded that in spite of high levels of parasitism (60-80%), *Anagrus* sp. was unable to keep the pest populations below acceptable levels.

Chemical control of leafhoppers is obtained by a variety of products. Foliar sprays of Carbaryl (1 kg a.i./ha) and Monocrotophos (0.5 kg a.i./ha) are effective. Granular soil-applied Carbofuran (placed under but

not in contact with the seed) at 0.7 - 1.0 kg a.i./ha protected plants for 30-40 days, while 0.6 - 0.7 kg a.i./ha of Carbofuran seedcoated also gave excellent control (14, 16).

Whiteflies

Five species of Aleyrodids live on beans in the Americas. They are *Bemisia tabaci, B. tuberculata, Tetraleurodes acaciae, Trialeurodes abutilonae* and *T. vaporiarorum*. These species also have other leguminous and non-leguminous hosts.

Common names frequently used for whiteflies in Latin America are mosca blanca and mosca branca.

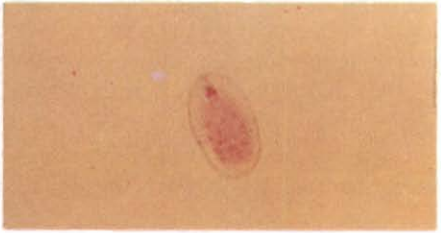
B. tabaci (Gennadius) is a vector of bean virus diseases such as bean golden mosaic (BGMV) and bean chlorotic mottle. The insect species has a wide range of synonyms. Some race identifications are based upon their virus transmission characteristics. Whitefly feeding does not damage bean plant development directly but does so indirectly when a virus is transmitted.

Eggs are laid singly or in groups on the lower leaf surface where the egg pedicel is inserted into the epidermis. The egg to adult stage requires about three weeks. Oviposition ranges from 25-32 eggs per female. The three immature stages and pupal stage occur on the lower leaf surface (Figs. 32 and 33). Identification is made on the immature stage (101).

In Guatemala, large differences exist according to geographical zone and planting date (3) for intensity of attack by whiteflies. Chemical control is

Fig. 32- (left) Eggs of whiteflies.

Fig. 33- (below) Pupa of *Trialeurodes* species.



most effective (measured as reduction of percent BGMV infested plants) with Metasystox or Oxydemeton-methyl and Monocrotophos (foliar application 15 and 30 days after planting), or Thimet or Phorate and Carbofuran granular application during planting (3). In El Salvador, Mancía *et al.* (68) report good control was obtained with the systemic granular insecticides Aldicarb, Carbofuran and Phorate.

Aphids

Several aphid species attack bean plants. Their direct damage is assumed to be of little importance, but their ability to transmit bean common mosaic virus makes them important pests economically. Further details are related by Zaumeyer and Thomas (133) and elsewhere in this book.

Common names frequently used for aphids in Latin America include afidios, pulgones, afidios and pulgao do feijoeiro.

Zaumeyer and Thomas reported the following aphids capable of transmitting bean common mosaic virus: *Aphis gossypii, A. medicaginis, A. rumicis, A. spiraecola, Brevicoryne brassicae, Hyalopterus atripilicis, Rhopalosiphum pseudobrassicae, Macrosiphum ambrosiae, M. solanifolii, M. pisi* and *Myzus persicae*. Costa and Rossetto (18) report aphids occur on bean foliage and roots in Brazil. In CIAT, control of bean common mosaic is sought by incorporation of genes which are resistant to the virus.

High aphid mortality occurs when insects are captured by hooked hairs on bean leaves. Capture percentage and number of hooked hairs increased when plants were grown under dry conditions, compared to when they were grown under ample moisture (28). A similar relationship was reported by McKinney (75) for *Myzus persicae* and thrips.

Thrips

Thrips have been found as pests of beans in several Latin American countries, but their attacks may not have much economic importance. *Frankliniella* sp., *Sericothrips* sp. and *Caliothrips braziliensis* (Morgan) have been reported in Brazil (98) and Colombia (90), where *C. braziliensis* is the most abundant species. Common names frequently used for thrips in Latin America are trips and bicho candela.

Larvae and adults feed on the undersurface of the cotyledonary leaves of seedlings. In older plants they also can be found feeding on leaves, flowers and petioles. When populations are high, thrips cause reduction in the size

Fig. 34- Damage caused by thrips on young bean plant.

and development of young plants(Fig. 34). In general, they seldom become an economic pest. Most attacks are localized towards the borders of the field and usually occur in hot, dry weather.

Females insert their eggs in the leaves, petioles and stems. In laboratory studies at CIAT, the eggs of *C. braziliensis* hatched in five to six days. The first larval instar lasted one or two days and the second instar four or five days. Pupation occurs in the soil and debris. The pupal stage took from two to three days to develop. Longevity and fecundity of the adults of this species have not been studied.

Chemical control is seldom justified. Adults and nymphs of *Orius tristicolor* are common predators of *Sericothrips* sp. and *C. braziliensis*.

Pod-Attacking Insects

Bean Pod Weevil

Apion godmani Wagner (Coleoptera: Curculionidae).

A. godmani is a serious bean pest in Central America where Mancía *et al.* (67) report up to 94% bean loss in El Salvador, especially during the rainy season. The bean pod weevil is considered the most serious bean pest in certain regions of El Salvador. The weevil also is of importance in Mexico, Guatemala, Honduras and Nicaragua and has been reported on beans in Colombia (1).

Common names frequently used for the bean pod weevil in Latin America are picudo de la vaina and picudo del ejote.

The weevil is prevalent especially in the highland, central and southern regions of Mexico during the rainy season (74), where up to 90% of the crop may be destroyed (26). In Mexico, *A. aurichalceum* is second in importance to *A. godmani*. The oviposition behavior of the former species

is different since the female lays about 35 eggs in the distal portion of a pod, allowing the other seeds of the pod to escape attack (74).

Several other less important *Apion* species also attack beans and include *A. aurichalceum, A. perpilosum, A. calcaratipes, A. germanum, A. griseum* and *Chalrodenus aenerus. A. godmani* also has been called *Trichapion godmani* (62, 74). Other host plants include *Dalea, Desmodium, Rhynchosia* and *Tephrosia* spp. (73).

The adult weevil is black and about 3 mm long. During the wet season, two generations may be formed, with possibly a third occurring during the dry season. Overwintering sites could not be located in Mexico (74). Under laboratory conditions of 20.8°C and an average 75% relative humidity, Mancía (62) stated that the egg stage of the weevil lasts five days. The three larval instars are passed in six days, while the prepupal and pupal stage last two and nine days, respectively. The adult insect can remain three or four days in the pupal chamber but usually emerges immediately after pupation. Adult longevity may extend from 10 days to nearly a year (62), and adults may mate several times. Mancía (62) reported a maximum of 392 eggs were laid by each female, with four to six eggs laid per day. The preoviposition period lasted 10 days with a 12-day incubation period, 22-34 day larval stage, two-day prepupal stage, six to 10-day pupal stage and a two to three-month adult stage.

Adults appear when bean plants are still small and occasionally cause light feeding damage to leaves, pods and flowers. Oviposition damage occurs in the newly formed pods. During the daytime the female adult chews a small hole in the mesocarp of 1-4 cm long pods, usually above the developing seed, and deposits an egg. These spots are visible as white hyperplastic deformations (Fig. 35), and later the adult exit-holes from the pod wall also can be found (73, 74). Young pods which are attacked may abort (26).

Fig. 35- Hyperplastic deformations caused by ovipositing females of *Apion*.

Fig. 36- Damage caused by larva of *Apion* in bean pod.

Larvae in the second instar stage bore into the mesocarp of the pod wall (Fig. 36) and begin feeding on the developing seed, leaving the hylum intact. One larva per seed is normal. However, three to five larvae per seed have been found during heavy infestations, with a maximum of 22 larvae present in a pod (62). McKelvey *et al.* (73) normally found one larva per seed and a maximum of seven per seed and 28 per pod. Larvae live in a feeding chamber and cannot feed on mature seed (73).

Mancía (62) found two Braconid parasites of *Apion* larvae, one of which belongs to the genus *Triaspis*. McKelvey *et al.* (73) found no influence of planting date on level of infestation, although there was a tendency for lower infestations in early and late plantings.

Guevara (41) tested six cultivars for resistance and found that 4% of Pinto 168 bean seed was infested, while 67% of Negro Mecentral bean seed was infested. Puebla 152 (17% infestation) and Mexico 228-7 (12% infestation) were intermediate in resistance. Pinto 168 yielded equally well with or without chemical protection, Puebla 152 and Mexico 228-7 required two sprays, and the susceptible test cultivar Negro Mecentral required three or four applications to control the weevil.

Ramírez *et al.* (95) tested 14 cultivars and found Negro 151 was the most resistant with 84 *Apion godmani* larvae per 60 pods. Resistant Bayo 164 and Pinto 168 had 90 and 108 larvae per 60 pods, respectively. Canocel was the most susceptible cultivar with 806 larvae per 60 pods and the highest adult count per pod. Ranked in descending order, Negro 151, Chapingo 55-111-7, Pinto 168 and Amarillo 154 had fewer adults. Mancía (61) tested 2004 *P. vulgaris* entries for resistance to *Apion* spp. and obtained nine highly resistant cultivars and two less resistant but did not identify them. Highly resistant entries had 1-5% seed damage, while the most susceptible entry had 43-94% seed damage.

After four years of testing, McKelvey *et al.* (74) report the cultivars Puebla 152, Hidalgo 6, Puebla 2, and Hidalgo 24 consistently had lower infestations than others tested. Other resistant cultivars included Puebla 32-A-2 and 20-B-2; Hidalgo 33-A-1, 28-A-2, 38-A-1 and 14-A-3; and Gto. 3-A-2 and 10-A-5. Guevara (40) evaluated *Apion* spp. resistance in Mexico and resistant sources (based upon percent seed infested in 100 pods) included Pinto 162 and 168; Amarillo 153, 154 and 155; EAP 88B and Negro 151. Later, Hidalgo 15A and 24; Puebla 2 and 57-B-3; Tlax. 2-1-C; Amarillo 156 and 164; and Negro 157 were added (42). Resistance to *Apion* spp. was incorporated in crosses involving Hidalgo 6 and Puebla 32. Although no details are given on the resistance mechanism or mode of inheritance, highly resistant lines were obtained in crosses between Puebla 2 x Hidalgo 12-A-1, Hidalgo 12-A-1 x Puebla 32 and Zacatecas 4A-2 x Hidalgo 6-1. Medina and Guerra (77) tested 14 cultivars and found Negro 66, Jamapa, Canario 101 and 107 were resistant to *Apion* spp., *Empoasca* spp. and the Mexican bean beetle. Ojo de Cabra and Negro Criollo were resistant to *Apion* spp. and *Empoasca* spp. Bayomex, Delicia 71 and Querétaro 183-1 were resistant only to *Apion* spp. Mancía (61) states that immunity to *Apion* spp. exists in *Phaseolus coccineus (= P. multiflorus).* However, in a recent study, Yoshii (132) did not find a significant difference in Apion attack between *P. vulgaris* and *P. coccineus.*

Although future use of resistant cultivars holds great promise, chemical control still remains important. Several products have been tested and Monocrotophos, Methomyl, Methyl Parathion and Carbaryl give effective control. Granular Carbofuran applied at planting (2.5 kg a.i./ha) gave the best control (63). Methyl Parathion gave adequate and economic control when applied as a spray six days after flower inititation and again seven days later. A single spray was effective if applied 13 days after flower initiation (69).

Corn Ear Worm

Damage by the *Heliothis* complex, *H. zea* (Boddie) and *H. virescens* (F.) (Fig. 37), is sporadic but can be severe. Common names frequently used for the corn ear worm in Latin America include Heliothis, helotero, bellotero and yojota.

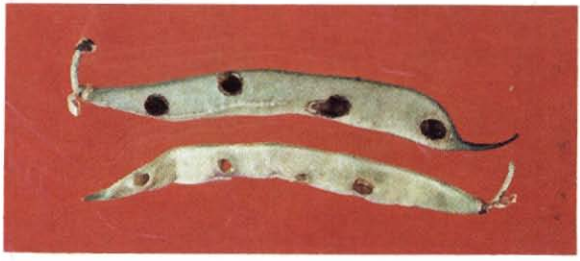


Fig. 37- Severe damage caused by *Heliothis* species.

The adult oviposits on young leaves, and larvae (Fig. 38) feed on seeds by perforating the podwall above the seed. Several seeds per pod may be destroyed, and secondary rotting can destroy the remaining seeds. It is not clear which of the two species is most common in beans. However, during a severe attack at CIAT only *H. virescens* was found.

Chemical control of older larvae is difficult, but high levels of parasitism usually occur. Posada and García (89) list 26 different parasite or predator species of *Heliothis* spp. in Colombia. In a CIAT study, 89% of field collected larvae were parasitized by a Tachinid fly. Recent findings also indicate that pyrethrins at low dosages effectively control *Heliothis virescens* larvae.

Other Pod-Boring Insects

Epinotia

Epinotia opposita Heinrich (Lepidoptera: Olethreutidae).

E. opposita is an important insect pest in Peru and Chile (124). Common names frequently used for *Epinotia opposita* in Latin America include polilla del fríjol and barrenador de la vaina.

Its larvae feed on or in the terminal buds, and/or perforate the stems and pods. Larvae weave their excrement together and push it out of the feeding canals. The insect also may cause flower damage and abortion. Bud and stem deformations occur after larval attack (Fig. 39), and pod damage can result in rotting by secondary organisms (2). In alfalfa, young larvae web leaves together and live therein. Other host plants include soybeans, peanuts, peas, cowpeas, lentils and clover (124).

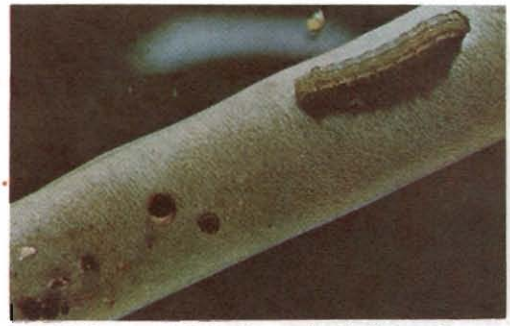


Fig. 38- Larva of *Heliothis* species feeding on bean pod.

Fig. 39- Bud deformation caused by larval feeding of *Epinotia opposita*.

About four days after copulation, females oviposit an average of 110 eggs in four to eight egg masses during a period of one or two weeks. Eggs are laid on young plant tissue. The egg stage lasts four and seven days during summer and winter, respectively, and during these corresponding seasons the five larval stages are passed in 14 and 23 days. Pupation occurs in a cocoon on the leaves or the ground (124). Adults live 15-22 days and are active at night.

Wille (124) observed a Tachinid larval parasite *(Eucelatoria australis)* which pupates in the host pupal skin. Avalos (personal communication) tested nearly 200 cultivars for *Epinotia opposita* resistance and found large differences in percentage of terminal buds and pods attacked. Adequate chemical control was obtained with Aminocarb, Toxaphene + Methyl Parathion or Omethoate (115). Early spring plantings reduced percentage of pod damage by *Epinotia* to 4%, as compared with 72% damage in late spring plantings (C. Quiroz, personal communication).

Laspeyresia leguminis

Laspeyresia leguminis Heinrich. (Lepidoptera: Olethreutidae).

L. leguminis attacks beans, soybeans, broad beans and lima beans (1, 124). The common name frequently used for *Laspeyresia leguminis* in Latin America is Laspeyresia.

Its damage often is confused with that caused by *Epinotia opposita.* However, unlike *Epinotia opposita,* it may web pods together (Avalos, personal communication). Adults oviposit on pods where young larvae bore into them and destroy the seeds. The larva pupates in the pod (124). Control is similar to that of *Epinotia opposita.*

Maruca

Maruca testulalis (Geyer) (Lepidoptera: Pyralidae).

M. testulalis is reported to occur in Brazil (100), Colombia (90), Cuba, Puerto Rico (58) and Africa (112). Like most of the other podborers, *M. testulalis* oviposits near or on flower buds, flowers, young leaves and pods. The common name frequently used for *Maruca testulalis* in Latin America is gusano perforador de la vaina.

Damage to leaves and flowers occurs prior to podboring-type feeding (106). The insect may attack several species of legumes (58). According to Broadley (8) larvae pass through five instars in eight to 13 days at 25° - 29°C. Pupation occurs in the soil.

M. testulalis is distinguished from *Etiella zinckenella* (the lima bean podborer) by larval and adult coloring. *Maruca testulalis* larvae have four

black or dark gray spots on each segment and adults rest with wings outspread. Larvae of *M. testulalis* expulse frass from the pods, while those of *E. zinckenella* leave it in the pod (111).

Storage Insects

Bruchids

The principal pests of stored beans are *Acanthoscelides obtectus* (Say) and *Zabrotes subfasciatus* (Boheman). Synonyms of *A. obtectus* include *Mylabris obtectus* and *Bruchus obtectus,* while synonyms of *Z. subfasciatus* are *Z. pectoralis, Z. dorsopictus* and *Spermatophagus subfasciatus.* Both pests are widely distributed from Chile to the United States. Common names frequently used for bruchids in Latin America include gorgojo, gorgojo pintado, gorgojo común del fríjol, caruncho and gorgulho de feijao.

At least 28 other insects are reported to occur on stored beans but are of minor importance or migrate from nearby stored produce onto beans.

The life history of the two most important bean storage pests, *A. obtectus* and *Z. subfasciatus,* is basically similar and was studied in detail by Howe and Currie (51). The main difference is in oviposition behavior. *A. obtectus* females scatter eggs among stored seeds or infest beans in the field where they lay eggs in cracks or cuts of growing pods. The newly-hatched larvae of *A. obtectus* later penetrate the seed. In contrast, *Z. subfasciatus* eggs are firmly attached to the seed and after hatching, the young larvae bore through their eggshell and the seedcoat in one process (51).

Larvae of both species molt four times before pupating. During the last larval instar, the feeding and pupation cell becomes externally visible as a circular window in the seed where larvae feed on the lower testa surface. After pupation the adult may remain in the cell for several days before pushing out the window. It also has the ability to emerge by eating away the exit. Adults normally do not eat but may consume water or nectar. Oviposition starts rapidly after emergence as adults are short-lived (51).

The optimum conditions for rapid development of *A. obtectus* eggs are 70% RH and 30°C, when the insects spend 22-23 days inside the beans. Mortality during development occurs mainly when larvae penetrate the seed or when the exit hole is not large enough for adult emergence. Adults live 12 days at 30°C and 70% relative humidity. A female may lay an average of 63 eggs (51).

The optimum development period for *Z. subfasciatus,* including the egg stage, is about 25 days at 70% RH and 32.5°C. In this species, 7% of adults were unable to escape from the pupal cell (Fig. 40) and died. *Zabrotes subfasciatus* adults exhibit strong sexual dimorphism. The female usually weighs 1.5 times as much as the male. Adults live eight days at 30°C and 70% RH. A female may lay and average of 36 eggs (51).

Acanthoscelides obtectus (Fig. 41) is distributed throughout higher latitudes and altitudes, while *Zabrotes subfasciatus* (Fig. 42) is found predominantly in warmer areas (103). In studies by Giles in Nicaragua (Giles, personal communication), beans were infested inititally with *A. obtectus* (99.7%) and *Z. subfasciatus* (0.3%) at different elevations above sea level. After 16 weeks the ratio became 0: 100% at 56 m; 5: 95% at 450 m; and 27: 73% at 680 m. Average temperatures at these three elevations were 28.2°C, 25.2°C and 24.3°C, respectively. These data suggest that *A. obtectus* is a stronger competitor at lower temperatures.

No precise information was found in the literature concerning economic losses caused by insects in stored beans (Fig. 43). McGuire and Crandall (72) estimate that storage losses may reach 35% in Mexico, Central

Fig. 40- Pupal cells of *Zabrotes subfasciatus;* note the eggs firmly attached to the seed.

Fig. 41- Adult *Acanthoscelides obtectus.*

Fig. 42- Adults of *Zabrotes subfasciatus.*

Fig. 43- Beans destroyed by a serious attack of *Zabrotes subfasciatus.*

America and Panama, but they do not specify if losses are caused by insects or other factors. A marketing survey in the Recife area of Brazil revealed that the average storage and handling losses incurred during the marketing process amounted to 13% (107). A survey of farms in bean-growing areas and 30 warehouses in Colombia revealed that the average storage period is short and that only an estimated 7% loss occurred (103).

Farmers control weevils by applying ashes from fireplaces to beans stored for future planting. This method appeared to be effective (15) as a physical barrier to weevils. Storing beans in undamaged pods is a safe control measure against *Zabrotes subfasciatus* attack. Eggs deposited on the podwalls hatch and larvae penetrate the podwalls but die inside the pods without penetrating the seed. However, this method cannot be used to control *Acanthoscelides obtectus*, since this insect is able to attack beans in the pods. Labeyrie (53) showed that storing beans unshelled or delaying the harvest greatly enhanced *Acanthoscelides obtectus* attack. Another non-chemical method for controlling weevils is the use of black pepper. One gram of ground pepper per 385 g of beans reduced infestations of *A. obtectus* by 78% after four months storage when compared to untreated lots (55). Inert dusts, such as crystalline silica, bentonite and magnesium carbonate effectively kill *A. obtectus*. Apparently the fraction of fine particles determines the efficiency of control. Adult death rates of 50% in 12 hours by bentonite has been ascribed to water loss (13).

Vegetable oils, applied at the rate of 1 ml oil/kg seed, reduced progeny production on bean seed treated with cotton seed oil to five Bruchids, compared to 265 on non-treated samples. The treated seed retained its germination ability (17). Total control was obtained with 5 ml oil/kg seed. No adults emerged from material infested 75 days after treatment (104).

Chemical control of weevils is readily obtained with a variety of products. Pyrethrins are highly effective (70, 102). Pyrethrins with bases of marc gave long-lasting control and provided more acceptable seed appearance than Pyrethrins with talc as carrier (15). Synthetic Pyrethrins also gave excellent control. Most warehouses in Colombia used few products to control storage insects. In 33% of the warehouses, owners used aluminium phosphide, 40% used methyl bromide, 27% used carbon bisulfide and 13% used Pyrethrin. One warehouse owner confessed he used Aldrin to control bruchids (103).

Much of the *Phaseolus vulgaris* germplasm collection of CIAT has been tested for resistance to *Z. subfasciatus*. Several entries were rated inititally resistant but were susceptible when retested. Seed should show resistance during at least two seed generations before it can be considered resistant

and useful for further studies. Varietal resistance to the bruchids also has been reported by Lefebre (56), Pabón *et al.* (84) and Ramalho *et al.* (94).

Other Pests

Mites

Spider Mites

Tetranychus desertorum Banks (Acarina: Tetranychidae).

Spider mites usually attack beans (Fig. 44) near physiological maturity and rarely affect yield. Common species are *T. desertorum* and *T. telarius*. *T. desertorum* has a wide host range as Nickel (83) observed 13 hosts in Paraguay. Common names frequently used for the red spider mite in Latin America include acaros, arañita roja and ácaro rajado.

Fig. 44- Leaf damage and webs produced by spider mites.

The biology of *T. desertorum* was studied by Nickel (83) who concluded that low temperatures limit geographical distribution of the pest. In laboratory studies on beans in Colombia, the incubation period lasted five days, the immature stages six days, and the female oviposited an average of four eggs per day during 15 days (85). This is a slightly slower development rate and also a lower oviposition rate than cited by Nickel.

The cultivars Oregón 58 R (J.G. Rodríguez, personal communication) and CRIA - 1-1, are resistant in Peru. Under CIAT greenhouse conditions, both were more resistant than ICA-Pijao and Diacol-Calima, but in the field Oregón 58 R was as susceptible as Diacol-Calima and ICA-Pijao. CRIA-1-1 exhibited an intermediate level of resistance. Biological control by several predator mites has been effective in detailed studies. However,

chemical control is used mostly. Mites can become resistant to pesticides, thereby requiring the application of different combinations of chemicals. Gonzalez (35) recommends the use of uniform restricted planting dates and chemical control with Omethoate mixed with Oxydemetonmethyl or Tetradifon with Monocrotophos. Wilcox and Howland (121) recommend Phorate and Disulfoton as granular soil-applied insecticides for lima beans.

Tropical Mites

Polyphagotarsonemus latus (Banks) (Acarina. Tarsonemidae).

P. latus, sometimes called the tropical mite, can attack beans and cause post-flowering damage especially during humid and warm weather. The mite genus is synonymous with *Tarsonemus*, *Neotarsonemus* and *Hemitarsonemus*. It is a small pale green mite, difficult to see without magnification and little known on beans. Common names frequently used for the tropical spider mite in Latin America include acaro blanco, acaro branco and acaro tropical.

The mite is a bean pest in Brazil (18) and in the Cauca Valley of Colombia. It also has been observed in Peru and Central America. Many other hosts beside beans are known and include potato (22), tomato, *Centrosema* spp., *Dolichos* spp. (20), green pepper, dahlia and cotton (45). The mite also attacks several common weeds in bean fields. Measurements on individual plants have revealed 56% yield loss in beans grown at CIAT (15).

The tropical mite has a short life-cycle which is composed of the egg, larva, pseudopupa (developmental stages) and adult stage. The developmental stages last one to three, two, and two days respectively at 27°C (27). Under laboratory conditions of 22° - 28°C at CIAT (105), the duration of these periods was two, one, and one day, respectively. Males lived for 12 days, while females lived 15 days and laid an average of 48 eggs.

Symptoms of mite damage become evident as leaf edges roll upwards and have a shiny appearance (Fig. 45). Depending on the cultivar, the lower leaf surface may turn purple. Young leaves do not develop normally and remain stunted, often turning yellow to gold (Fig. 46). The pods can be attacked and become covered with a brown wound tissue (Fig. 47) which may resemble sunscald damage. Some cultivars show a downward curling of leaf edges and a darkening of the leafblade. Symptoms are commonly confused with those induced by virus or mineral deficiencies.

Endosulfan, Monocrotophos, Carbaryl, Dicofol, Triazophos and Omethoate provide good chemical control at CIAT (105). Costa (19)

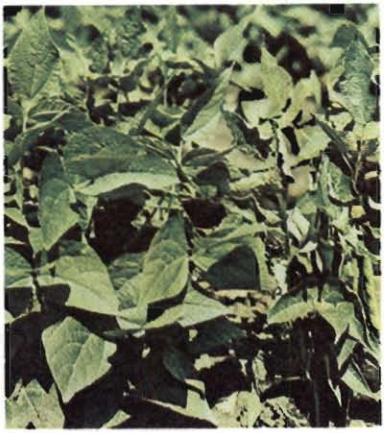


Fig. 46- (above) Discoloration of lower leaf surface due to tropical mite.

Fig. 45- (left) Leaf rolling symptoms caused by tropical mite damage.

Fig. 47- Discoloration of bean pods due to tropical mite.

Fig. 48- Adult slug on bean plant with pod and leaf-feeding damage.

recommends Carbophenothion, Chlorobenzilate, Chlorfensulphide and Endosulfan for control on cotton. Mite populations apparently are stimulated by Dimethoate (47).

Slugs

Slugs (Fig. 48), like mites, do not belong to the class of insects, however, occasionally are serious bean pests in El Salvador and Honduras. The reported species belong to the family Limacidae, and include *Vaginulus plebeius* Fisher, *Limax maximus* L. and *Deroceras agreste* L. (49, 64). Common names frequently used for slugs in Latin America are babosas and lesmas.

Although hermaphroditic, after copulation females lay up to 800 eggs in egg masses under plant debris or in soil cracks. At 27°C they hatch in 24 days and reach sexual maturity three or four months later. Slugs are nocturnal but may be active during wet, cloudy days. Young slug damage is apparent when whole leaves, with the exception of the veins, are consumed

Fig. 49- Leaf damage due to slug feeding.

(Fig. 49). Older slugs consume entire leaves. Entire seedlings also may be consumed, and pod damage may occur. Most damage occurs along borders of fields and progresses inwards, especially if vegetation and debris provide ample protection for the slugs during the day.

Control is best achieved by cleaning fields and borders of weeds and plant debris. Curative control is obtained with baits, such as Methaldehyde or Carbaryl applied in bands along borders or within affected areas in the late afternoon. Some formulations are (per ha): Methaldehyde 99% (65 g) mixed with wheatbran (25 kg) and molasses (20 l). Carbaryl 80% (0.5 kg) or Thrichlorfon (0.5 kg) may be used to replace Methaldehyde (64).

Future of Insect Control in Latin America

Cultivars are available which possess genetic resistance to insect pests such as *Empoasca kraemeri, Apion godmani, Epilachna varivestis,* and *Epinotia opposita.* The main objective in bean entomology research should be to incorporate resistance to key insect pests into commercially acceptable cultivars which already posses resistance to plant diseases such as bean common mosaic virus and rust.

Development of varietal resistance will take time, during which most national programs are improving current chemical control recommendations. Recent studies with systemic granular insecticides such as Carbofuran or Phorate have reduced bean golden mosaic virus incidence greatly and may preserve natural biological control. Several bean programs still recommend application of chlorinated hydrocarbons to control insect pests.

Future emphasis must be placed on development of a pest management system within which biological, cultural and other control strategies are an

integral part. However, the short growing season of beans and fallow periods may reduce the effectiveness of biological control in these systems. The increasing use of resistant cultivars should reduce the need for pesticides and assure the survival of agents contributing to biological control. It may be desirable to locate and release more efficient natural enemies. However, national programs may be restricted by lack of funds and trained personnel. Biological control by other agents, such as parasitic fungi or bacteria, also must be investigated further.

Cultural practices should play an important role in a pest management system. Shifting of planting dates may be a powerful tool in controlling insects. However, it has limited application where rainfall distribution primarily governs planting dates. *Empoasca kraemeri* control is favored by planting at the beginning of the rainy season when leafhopper populations are low. *Hylemya* spp. control is favored by a late planting date, and a preplant plowing may also be useful. However, the biology and ecology of most insect pests has not been studied sufficiently to allow valid recommendations.

As discussed before, the distribution of principal bean insects varies greatly within Latin America. Proper quarantine measures also should continue to be enforced to limit pest distribution.

The most important aspect of crop pest management will be elimination of unnecessary pesticidal applications in a practical and economical manner. Accurate knowledge must be obtained between the relationship of insect pest populations and yield reductions. Most entomologists involved with bean research expect that a certain amount of feeding damage can be sustained by the plant before economically significant yield reduction occurs. Leafhopper research indicates that the first insect present on a plant causes more damage than those which follow (16). This indicates that the decision to spray is not only based upon expected yield loss, but also upon the cost of insecticidal spray and the consequences of this spray to later pest development, such as lepidopterous insects and their biological enemies. The curve of population level versus *Empoasca kraemeri* damage is different from that of foliage feeders where part of the foliage can be removed without adversely affecting yield.

Associated cropping is a system in which an estimated 80% of the beans in Latin America are grown. This system demands more attention. It is possible that abandoning this system may reduce the stability of the ecosystem and increase specific insect pest populations and their importance.

Finally, excellent work has been accomplished by Latin American entomologists. However, lack of funds often prohibits publication of this work, so others cannot profit from their knowledge and experience. The

vacuum thus created has hindered more rapid progress in bean entomological research to reduce bean yield losses due to insects in Latin America.

Table 1. Major insect pests of beans in Latin America.

SEEDLING-ATTACKING INSECTS	
Seed Corn Maggot	*Hylemya* spp.
Cutworm	*Spodoptera frugiperda*
Whitegrub	
Cricket	
Lesser Corn Stalk Borer	*Elasmopalpus lignosellus*
LEAF-FEEDING INSECTS	
Chrysomelids	*Diabrotica balteata*
	Cerotoma spp.
Lepidoptera-Saltmarsh Caterpillar	*Estigmene acrea*
-Bean Leafroller	*Urbanus proteus*
	Hedylepta indicata
Mexican Bean Beetle	*Epilachna varivestis*
SUCKING INSECTS	
Leafhopper	*Empoasca kraemeri*
White Fly	*Bemisia tabaci*
Aphids	*Aphis* spp.
Thrips	*Caliothrips braziliensis*
POD-ATTACKING INSECTS	
Bean Pod Weevil	*Apion godmani*
Pod Borers	*Epinotia opposita*
	Laspeyresia leguminis
	Maruca testulalis
	Heliothis spp.
STORAGE INSECTS	
Bruchids	*Zabrotes subfasciatus*
	Acanthoscelides obtectus
OTHER PESTS	
Mites - Spider Mites	*Tetranychus* spp.
- Tropical Mites	*Polyphagotarsonemus latus*
Slugs	*Vaginulus plebeius*

Table 2. Most important insect pests in 12 Latin American countries (43)*.

Pest damage group	Principal species	Number of countries in which insect is important
Piercing Insects	*Empoasca* spp.	12
Leaf-feeding Insects	*Diabrotica* spp.	10
(not Lepidoptera)	*Epilachna* spp.	10
Cutworms, Crickets	—	8
Pod-attacking Insects	*Apion godmani*	5
Stored Grain Insects	—	5

Brazil, Colombia, Costa Rica, El Salvador, Guatemala, Haiti, Honduras, Nicaragua, Panama, Paraguay, Peru and Dominican Republic

Table 3. Relative importance* of bean insects in Central America (6).

Country	Leafhoppers	Chrysomelids	Bean pod weevil	Whitefly	Mexican bean beetle
Costa Rica	4	4	1	2	1
Nicaragua	3	3	1	3	3
El Salvador	4	3	3	2	1
Honduras	4	3	4	3	1
Guatemala	4	2	3	2	4

* Relative importance measured on a 0-4 scale: 0 = insects absent; 4 = insects very numerous.

Table 4. Average percent yield loss (highest yielding insecticidal treatment compared with untreated plots) from 16 insecticidal trials reported in bean literature.

Area	Number of experiments	Principal insect involved	Average % yield loss
Mexico, El Salvador	5	*Apion godmani*	54.2
Mexico	3	*Empoasca kraemeri*	64.0
Mexico	2	*Epilachna varivestis*	55.0
El Salvador, Mexico, Puerto Rico	6	Unspecified	30.5
Total	16	Weighted average	47.25

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Appendices

Appendix I. Official common name and formula of chemicals cited in text.

The chemical compounds listed below were cited by authors in various chapters of this book. The list is intended as an aid in the proper identification of these chemicals and does not constitute an endorsement of them by CIAT.

Official Common Name	Chemical Formula*
FUNGICIDES	
Benomyl	Methyl 1-(butylcarbamoyl)-2-benzimidazolecarbamate
Bordeaux Mixture	Mixture of copper sulfate and calcium hydroxide
Bunema	Potassium N-hydroxymethyl-N-methyldithiocarbamate
Busan	2-(Thiocyanomethylthio) benzothiazole
Captafol	cis-N-(1,1,2,2 - Tetrachloroethylthio) 4-cyclohexene-1,2-dicarboximide
Captan	N-(Trichloromethylthio)-4-cyclohexene-1,2-dicarboximide
Carbendazim	Methyl-1H-benzemidazol-2-ylcarbamate
Carboxin	5,6-Dihydro-2-methyl-1,4-oxathiin-3-carboxanilide
Ceresan	Phenyl mercuric acetate
Cloroneb	1,4-Dichloro-2,5-dimethoxybenzene
Chlorothalonil	Tetrachloroisophthalonitrile
Dichlone	2,3-Dichloro-1,4-naphthoquinone
Dicloran	2,6-Dichloro-4-nitroaniline
Dinocap	Mixture of 2,4-Dinitro-6-octylphenyl crotonate and 2,6-Dinitro-4-ocylylphenyl crotonate
Fenaminosulf	Sodium p-(dimethylamino) benzenediazo sulfonate
Fentinacetate	Triphenyltin acetate
Fentin Chloride	Triphenyltin chloride
Fentin Hydroxide	Triphenyltin hydroxide
Ferbam	Ferric dimethyldithiocarbamate
Mancozeb	Manganese ethylenebisdithiocarbamate plus zinc ion
Maneb	Manganous ethylenebisdithiocarbamate

* Thomson, W.T. 1977. Agricultural Chemicals, Books I-IV. Thomson Publications, Fresno, California.

Official Common Name	Chemical Formula*
Metiram	Mixture of ammoniates of ethylene (dithiocarbamate)-zinc and ethylenebis-dithiocarbamic acid bimolecular and trimolecular cyclic anhydrosulfides and disulfides
Nabam	Disodium ethylenebisdithiocarbamate plus metallic sulfates
NF-44	2-(3-methoxycarbonyl-2-thioureido) aniline
Oxycarboxin	5,6-Dihydro-2-methyl-1,4-oxathiin-3-carboxanilide-4,4-dioxide
PCNB	Pentachloronitrobenzene
Prothiocarb	S-ethyl-N-(3-dimethylaminopropyl)-thiol carbamate hydrochloride
Pyroxychlor	2-chloro-6 methoxy-4-(trichloromethyl)pyridine
Thiabendazole	2-(4-Thiazolyl) benzimidazole
Thiophanate	Diethyl 4,4,-o-phenylenebis 3-thioallophanate
Thiophanate-methyl	Dimethyl 4,4-o-phenylenebis (3-thioallophanate)
Thiram	Tetramethylthiuram disulfide
Tridemorph	N-Tridecyl-2,6-dimethylmorpholine
Zineb	Zinc ethylenebisdithiocarbamate
Ziram	Zinc dimethyldithiocarbamate

FUMIGANTS

Chloropicrin	Trichloronitromethane
DD	Mixture of 1,3-Dichloropropene and 1,2-Dichloropropane
EDB	1,2-Dibromoethane
Ethylene Oxide	Epoxyethane
Methyl Bromide	Bromomethane
Nemagon	1,2-dibromo-3-chloropropane
Phenamiphos	Ethyl-3-methyl-4-(methyl thio) phenyl (1-methyl ethyl) phosphoramidate

HERBICIDES

2,4-D	2,4-Dichlorophenoxyacetic acid
Bentazon	3-isopropyl-1H-2,1,3-benzothiadiazin-(4) 3H-one 2,2-dioxide
Cycloate	S-Ethyl cyclohexylethylthiocarbamate
Dinitramine	N^3,N^3-Diethyl 2,4-dinitro-6-trifluromethyl-1, 3 phenylenediamine
Eptam	S-Ethyl dipropylthiocarbamate
Paraquat	1:1-Dimethyl-4,4'-Bipyridinium (cation) dichloride
Trifluralin	Alpha, Alpha, Alpha, Trifluoro-2,6-dinitro-N,N-dipropyl-p-toluidine

Official Common Name	Chemical Formula*
INSECTICIDES	
Aldicarb	2-Methyl-2-(methylthio)propionaldehyde O-(methylcarbamoyl) oxime
Aldrin	Hexachlorohexahydro-endo, exo-dimethanonaphthalene
Aminocarb	(4-dimethylaminophenyl-3-methyl-phenyl)-N-methylcarbamate
Carbaryl	1-Naphthyl methylcarbamate
Carbofuran	2,3-Dihydro-2,2-dimethyl-7-benzofuranyl methylcarbamate
Carbophenothion	S-((p-chlorophenylthio)methyl)0,0-diethyl phosphorodithioate
Chlorfensulphide	4-Chlorophenyl 2,4,5-trichlorophenyl azosulfide and 1,1-Bis-(4 chlorophenyl)ethanol
Chlorobenzilate	Ethyl 4,4'-dichlorobenzilate
Chlorpyrifos	0,0-Diethyl-0-(3,5,6-trichloro-2-pyridyl) phosphorothioate
Diazinon	0-0-Diethyl-0-(2-isopropyl-6-methyl-5-pyrimidinyl) phosphorothiate
Dicofol	1,1-Bis(p-Chlorophenyl)-2 2,2-trichloroethanol
Dicrotophos	Dimethyl phosphate ester with 3-hydroxy-N,N-dimethyl-cis-crotonamide
Dieldrin	Hexachloroepoxyoctahydro-endo, exo-dimethanonaphthalene
Dimethoate	0,0-Dimethyl S-(N-methylcarbamoylmethyl) phosphorodithioate
Disulfoton	0,0-Diethyl-(s-2-(ethylthio)ethyl) phosphorodithioate
Endosulfan	6,7,8,9,10,10-Hexachloro-1,5,5a,6,9,9a-hexahydro-6,9-methano-2,4,3-benzodioxathiepin-3-oxide
Fensulfothion	0,0-Diethyl 0-(4-(methylsulfinyl)phenyl) phosphorothioate
Malathion	0,0-Dimethyl phosphorodithioate ester of diethyl mercaptosuccinate
Methaldehyde	Metacetaldehyde
Methamidophos	0,S-Dimethyl phosphoramidothioate
Methomyl	S-Methyl N-(methylcarbamoyl)oxy) thioacetimidate
Methyl Parathion	0,0-Dimethyl-o-p-nitrophenyl phosphorothioate
Monocrotophos	Dimethyl phosphate of 3-hydroxy-N-methyl-cis-crotonamide
Omethoate	0,0-Dimethyl S-(N-methylcarbamoylmethyl) phosphorothioate
Oxydemeton-methyl	S-(2-ethylsulfinyl)ethyl)0,0-dimethyl phosphorothioate
Phorate	0,0-Diethyl-S-((ethylthio)methyl)phosphorodithioate
Pyrethrins	dl-2-Allyl-4-hydroxy-3-methyl-2-cyclopenten-1-one ester of di cistranschrysanthemum monocarboxylic acid
Tetradifon	p-chlorophenyl 2,4,5-trichlorophenyl sulphone
Toxaphene	Octachlorocamphene
Triazophos	1-Phenyl-3-(0,0-diethyl-thionophosphoryl)-1,2,4-triazole
Trichlorfon	Dimethyl (2,2,2,-trichloro-1-hydroxy ethyl) phosphonate

Appendix II. Conversion values for measurement units commonly referred to in text.

U.S. TO METRIC UNITS*		METRIC TO U.S. UNITS*	
Temperature			
Degrees Farenheit	= (C° x 1.8) + 32°F	Degrees Centigrade =	(F° - 32)/1.8
Length and Area			
1 inch	= 2.54 centimeters	1 centimeter	= 0.39 inches
1 foot	= 0.31 meters	1 meter	= 3.28 feet
1 square foot	= 0.09 square meters	1 square meter	= 10.76 square feet
1 acre	= 0.41 hectares	1 hectare	= 2.47 acres
Weight			
1 ounce	= 28.35 grams	1 gram	= 0.04 ounces
1 pound	= 0.45 kilograms	1 kilogram	= 2.21 pounds
1 ton	= 0.91 metric ton	1 metric ton	= 1.10 tons
Volume			
1 fluid once	= 29.57 cubic centimeters (ml.)	1 cubic centimeter (ml.)	= 0.03 fluid ounces
1 gallon	= 3.79 liters	1 liter	= 0.26 gallons
1 ounce/gallon	= 7.49 grams/liter	1 gram/liter	= 0.13 ounces/gallon
1 ounce (fl.)/gallon	= 7.81 milliliters/liter	1 milliliter/liter	= 0.13 fl. ounces/gallon
1 pound/acre	= 1.12 kilograms/hectare	1 kilogram/hectare	= 0.89 pounds/acre
1 gallon/acre	= 9.35 liters/hectare	1 liter/hectare	= 0 11 gallons/acre

Other Useful Conversions

1 gallon = 4 quarts = 8 pints = 16 cups = 128 fluid ounces

1 fluid ounce = 2 tablespoons = 6 teaspoons

1 part per million (ppm) = 1 milligram/liter = 0.0001% = 0.013 fluid ounces/100 gallons

1% = 10,000 ppm = 10 grams/liter = 1.33 ounces/gallon

1 micron (μ) = 1×10^{-4} centimeter = 3.94×10^{-5} inch

* Conversion values adapted from: (1) Agricultural Chemicals, Book IV - Fungicides. 1976/77 Revision by W T. Thomson, Thomson Publications; (2) ISCO Tables, a Handbook of Data for Biological and Physical Scientists, 4th Ed 1972. Instrumentation Specialties Company; (3) Fungicide and Nematocide Tests, Vol 33. Results of 1977, American Phytopathological Society

Appendix III. Taxonomic clarification of various host scientific names cited in text.

Cited Name	New Classification*
Dolichos lablab (L.)	*Lablab purpureus* (L.) Sweet
Lablab niger Medik.	*L. purpureus* (L.) Sweet
Phaseolus aconitifolius Jacq.	*Vigna aconitifolia* (Jacq.) Marechal
P. adenanthus G. F. Meyer	*V. adenantha* (G. F. Meyer) Marechal, Mascherpa and Stainier
P. angularis (Willd.) W.F. Wight	*V. angularis* (Willd.) Ohwi and Ohashi
P. atropurpureus DC.	*Macroptilium atropurpureum* (DC.) Urban
P. aureus Roxb.	*V. radiata* (L.) R. Wilczek
P. bracteatus Nees and Mart	*M. bracteatum* (Nees and Mart.) Marechal and Baudet
P. calcaratus Roxb.	*V. umbellata* (Thunb.) Ohwi and Ohashi
P. dysophyllus Bentham	*M. atropurpureum* (DC.) Urban
P. lathyroides L.	*M. lathyroides* (L.) Urban
P. limensis Macfadyen	*Phaseolus lunatus* L.
P. multiflorus Lam.	*P. coccineus* L.
P. mungo L.	*V. mungo* (L.) Hepper
P. obvallatus Schlecht	*P. coccineus* subsp. *obvallatus* (Schlecht.) Marechal, Mascherpa and Stainier
P. polyanthus Greenman	*P. coccineus* subsp. *polyanthus* (Greenman) Marechal, Mascherpa and Stainier
P. radiatus L.	*V. radiata* (L.) R. Wilczek
P. retusus Bentham	*P. ritensis* Jones
P. riccardianus Tenore	*V. umbellata* (Thunb.) Ohwi and Ohashi
P. sinuatus Nutt. ex Torr. and Gray	*P. polystachyus* var. *sinuatus* (Nutt.) Marechal, Mascherpa and Stainier
Vigna hirta Hooker	*V. vexillata* (L.) A. Richard
V. repens (L.) Kuntze	*V. luteola* (Jacq.) Bentham in Mart.
V. sesquipedalis (L.) Fruhw.	*V. unguiculata subsp. unguiculata* cv.-gr.*sesquipedalis* E. Westphal
V. sinensis (L.) Savi ex Hassk	*V. unguiculata* (L.) Walpers

* According to Marechal, R., J.M. Mascherpa and F. Stainier. 1978. Etude taxonomique d'un groupe complexe d'especes des genres Phaseolus et Vigna (Papilionaceae) sur la base de donnees morphologiques et polliniques, traites par l'analyse informatique. Memoires des Conservatoire et Jardin Botaniques de la Ville de Geneve, Boissiera Vol 28, 273 p.

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Code listing of photographers

Chapter	Figure Number	Photographer Code
2	1	23
	2	23
	3	41
	4	41
	5	41
	6	41
	7	41
	8	41
	9	41
	10	30
3	1	41
	2	41
	3	41
	4	41
	5	41
	6	41
4	1	41
	2	4
	3	41
	4	41
5	1	41
	2	41
	3	5
	4	1
	5	5
	6	41
	7	41
	8	1
	9	41
	10	41
	11	1
	12	41
	13	1
6	1	41
	2	41
	3	34
	4	39
	5	34
7	1	41
	2	41
	3	41
	4	41
	5	36
	6	41
	7	41
8	1	1
	2	1
	3	1
	4	41
	5	41
	6	41
	7	41
	8	41
	9	41
	10	41
	11	41
	12	18
	13	41
	14	18
	15	41
	16	1
	17	41
	18	41
	19	41
	20	41
	21	33
	22	41
	23	41
	24	41
	25	41

* Refer to list of contributing authors and photographers for code identity.

Code listing of photographers (continued)

Chapter	Figure Number	Photographer Code
9	1	41
	2	41
	3	41
	4	41
	5	41
	6	41
10	1	7
	2	25
	3	25
	4	41
	5	41
	6	40
	7	25
	8	25
	9	25
11	1	28
	2	28
	3	22
	4	36
	5	41
	6	22
	7	41
	8	22
	9	22
12	1	18
	2	18
	3	41
	4	14
	5	41
	6	18
	7	14
	8	18
	9	14
	10	41
	11	41
	12	35
	13	41
13	1	18
	2	14
	3	35
	4	18
	5	32
	6	32
	7	32
14	1	18
	2	14
	3	14
	4	28
	5	18
	6	18
	7	14
	8	18
	9	41
	10	41
	11	35
	12	31
	13	31
	14	18
	15	31
16	1	14
	2	14
	3	14
	4	14
	5	41
	6	15
	7	15
	8	15
	9	15
	10	41
17	1	3
	2	3
	3	46
	4	9
	5	30

Code listing of photographers (continued)

Chapter	Figure Number	Photographer Code
17	6	9
	7	1
18	1	41
	2	39
	3	41
	4	41
	5	35
	6	41
	7	39
	8	41
	9	41
	10	35
	11	43
	12	29
	13	45
19	1	14
	2	17
	3	17
	4	17
	5	14
	6	14
	7	27
	8	36
	9	27
	10	17
	11	36
	12	27
	13	21
	14	27
	15	27
	16	17
	17	37
	18	14
	19	14
	20	17
20	1	14
	2	14
	3	14
	4	2
	5	14
	6	14
	7	39
	8	14
	9	39
	10	14
	11	14
	12	20
	13	14
	14	14
	15	14
	16	14
	17	14
	18	14
	19	14
	20	14
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	22	14
	23	14
	24	14
	25	26
	26	26
	27	14
	28	14
	29	39
	30	14
	31	39
	32	6
	33	14
	34	41
	35	39
	36	39
	37	14
	38	14
	39	39
	40	14

Code listing of photographers (continued)

Chapter	Figure Number	Photographer Code	Chapter	Figure Number	Photographer Code
20	41	14	20	46	41
	42	14		47	14
	43	14		48	14
	44	14		49	14
	45	14			