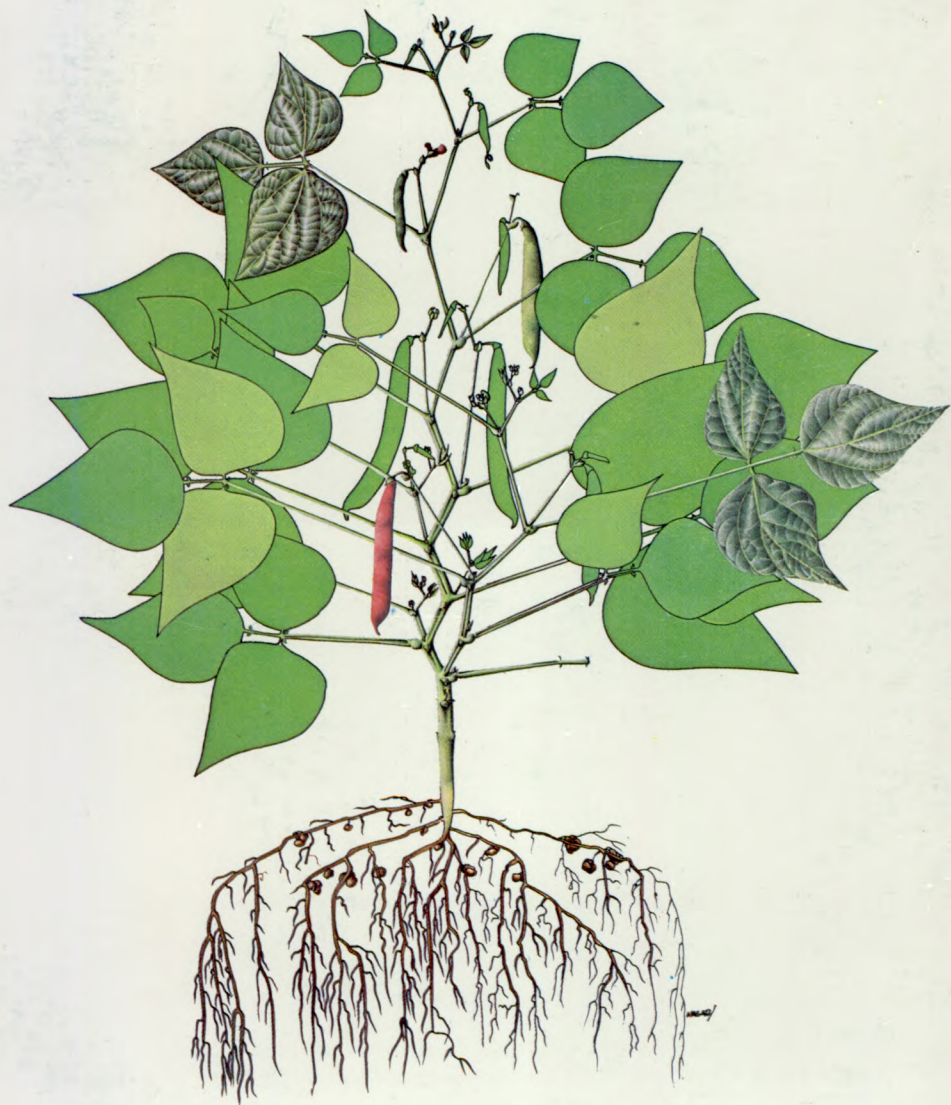


Bean Production Problems in the Tropics



The Centro Internacional de Agricultura Tropical (CIAT) is a development-oriented, agricultural research institution dedicated to the application of science toward lasting alleviation of hunger and poverty in developing countries.

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Information and conclusions reported herein do not necessarily reflect the position of any of the aforementioned entities.



Cover drawing:

An artist's impression of a *Phaseolus vulgaris* bean plant. There are about 35,000 varieties of *P. vulgaris* which differ from each other in their morphological, physiological, biochemical, and genetic characteristics. Drawn by Julio Martínez at CIAT's Graphic Arts Unit.

Bean Production Problems in the Tropics

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Edited by **Howard F. Schwartz** and
Marcial A. Pastor-Corrales

 **Centro Internacional de Agricultura Tropical**

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FOREWORD

Beans are grown in more than 12 million ha and constitute the most important food legume for more than 500 million people in Latin America and Africa. Beans are the leading source of protein and are an important source of calories for many of the poorest in these two continents. Despite their nutritional importance, however, production growth rates have been declining in Brazil, the Andean region, and throughout Africa. In most low-input systems where the majority of beans are produced, the principal factors responsible for bean yield and quality losses are diseases, insect pests, plant nutritional deficiencies, and drought.

The Centro Internacional de Agricultura Tropical is proud to present to bean researchers and to the world of agricultural science a second book on bean production problems, covering the most important production constraints of beans in Latin America and Africa.

Because a considerable amount of important information has become available since the publication of **Bean Production Problems: Disease, Insect, Soil and Climatic Constraints of *Phaseolus vulgaris***, a new, completely revised, version was needed. In addition to completely rewriting each section of the first book, new sections have been added and other bean researchers have joined the list of contributors. Thus, this second version represents the combined efforts of many internationally recognized bean research authorities who have contributed their knowledge and experience to this very comprehensive review of bean production constraints. We sincerely hope and trust that this book will be a significant contribution to the solution of these very important constraints.

We gratefully acknowledge the valuable support provided by the International Development Research Centre of Canada. Through a cooperative project with CIAT's Training and Communications Support Program, this center contributed by funding the costs of

technically revising and editing the manuscript, and the development and preparation of the manuscript for publication. CIAT, in keeping with its continuing devotion to the agricultural and economic growth of developing regions and the improvement of living standards for people of the tropical world, publishes this book with pleasure.

John L. Nickel
Director General, CIAT

PREFACE

The common dry bean, *Phaseolus vulgaris*, is the most important food legume for direct human consumption in the world. Production occurs in a wide range of cropping systems and environments spanning regions as diverse as Latin America, Africa, the Middle East, China, Europe, the United States, and Canada. In Latin America, the leading bean producer and consumer, beans are a traditional and very important food for the lower income strata, particularly in Brazil, the Andean Zone, Central America, and some Caribbean countries. However, the highest per capita consumption in the world occurs in eastern Africa, especially in the Great Lakes Region. Beans are also an important source of dietary protein in Kenya, Tanzania, Malawi, Uganda, and Zambia.

Beans in Latin America and Africa are primarily a small-farmer crop, grown with few purchased inputs, and besieged by an array of biological, edaphic, and climatic problems, making beans notoriously low in yield, particularly when compared with the average yields obtained in temperate regions of North America and Europe. In tropical bean production regions, diseases, insect pests, and low soil fertility are the most important production constraints. Most of the landraces and improved varieties grown in these areas are susceptible to one or more of these production constraints, preventing the realization of their full yield potential and causing production instability from one year to the next.

In most tropical bean production regions, diseases are often the most important constraint to bean production, particularly in Latin America. More plant pathogens, greater pathogenic variation, and more virulent isolates of these pathogens are found attacking beans in Latin America and Africa than in temperate regions. The prevalence and importance of each disease varies considerably with locality, season, year, and cultivar; however, some pathogens such as those that cause anthracnose, angular leaf spot, common bacterial blight, rust, and bean common mosaic virus, are widespread and economically important. Usually, one or more of these

pathogens are found to cause yield losses in most bean-producing areas of Latin America and Africa. Other pathogens are also significant economically but are restricted to growing regions with specific environmental conditions that favor their survival and spread. This group includes bean golden mosaic virus, web blight, and ascochyta blight. Some are widespread but not economically important such as root rots, and the rest are not widespread and not economically important.

Insects pests are also very important in Latin America and Africa and cause considerable damage to production before and after harvest. Some significant pests are restricted to one continent. Bean fly, for example, is extremely important in Africa but is not present in Latin America. Bean pod weevil is economically important and present only in Mexico and some countries of Central America. Other insect pests such as bruchids and leafhoppers, are widespread in most tropical bean-producing regions.

In Latin America and Africa, beans are grown on many different soil types, which often limit plant growth and yields because of nutritional deficiencies or toxicities. Edaphic problems have been extensively reported for large bean production areas of Brazil, the Andean Zone, Central America, and Africa.

To overcome the major production constraints in beans, research is a must. This book intends to bring together the most current knowledge available about each of the most important bean production constraints. The authors of the different chapters are bean researchers with acknowledged broad experience in bean research. We hope, therefore, that this book will provide the type of information usually needed by bean scientists and policy makers.

This book can be seen as having six general sections, each containing chapters on specific bean constraints by one or more of the 29 contributing authors. The first section reviews trends of bean production and constraints in Latin America and Africa. The second section covers fungal diseases; the third, bacterial diseases; the fourth, viral and mycoplasma diseases; the fifth, insect pests; and the last, other bean production constraints, that is, nutritional disorders, nematodes, seed pathology, and additional problems.

We express our sincere gratitude and appreciation to Drs. William J. Zaumeyer and Guillermo E. Gálvez who, with the support of the Rockefeller Foundation and the Centro Internacional de Agricultura Tropical, laid the foundation for creating the 1980 bean review book, entitled "Bean Production Problems: Disease, Insect, Soil and Climatic Constraints of *Phaseolus vulgaris*."

We would also like to extend our grateful thanks to the 12 scientists who contributed their expertise to the 1980 edition and who generously allowed us to build upon their creativity and insight for this revised and expanded version:

| | |
|-----------------------------------|---|
| John H. Sanders | (Bean Production and Pest Constraints in Latin America) |
| Edgar Vargas | (Rust) |
| G. Chaves | (Anthracnose) |
| Silamar Ferraz | (Angular Leaf Spot) |
| H. A. Bolkan | (Root Rots) |
| Pablo Guzmán and Mauricio Castaño | (Web Blight) |
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| Michael A. Ellis and G. E. Gálvez | (Seed Pathology) |
| Reinhart H. Howeler | (Nutritional Disorders) |

We also express our gratitude to the authors who contributed to the 1989 version and to the following scientists who donated illustrations, reviewed various chapters, and aided in the development of the book:

| | |
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Chapter 1

TRENDS IN WORLD COMMON BEAN PRODUCTION

Douglas Pachico*

The common or dry bean (*Phaseolus vulgaris* L.) is produced primarily in tropical low-income countries which account for over three-quarters of the annual world production of 8.5 million metric tons (Table 1). The common bean is the most important food legume in the developing world and in North America where nearly one million tons of beans are produced annually. European bean production is only slightly less than that of North America, although other pulses are of greater importance.

Table 1. Average world production of common beans during 1982-84.

| Region' | Percentage of world production | Production (t in thousands) |
|----------------------------|--------------------------------------|--------------------------------|
| Developing countries in: | | |
| Latin America | 46.7 | 3983 |
| Sub-Saharan Africa | 24.1 | 2056 |
| West Asia and North Africa | 3.5 | 299 |
| East and South Asia | 3.0 | 256 |
| Total developing countries | 77.3 | 6594 |
| Developed countries in: | | |
| North America | 11.6 | 988 |
| Europe | 10.4 | 887 |
| Pacific | 0.7 | 65 |
| Total developed countries | 22.7 | 1940 |
| World | 100.0 | 8534 |

SOURCE: Compiled by author from FAO, 1983, 1984a, and 1985.

* Agricultural economist and Head, Bean Program, Centro Internacional de Agricultura Tropical (CIAT), Cali, Colombia.

In the developing world, small farmers are the principal producers of beans, often as a secondary crop in association with maize. A high proportion of beans in these countries is consumed on the farm or traded only in local markets. Thus, with limited resources and other pressing demands on the administrative capacity of agricultural ministries of many developing countries, the difficulties of collecting accurate data on common beans are immense. Consequently, data for many countries constitute little better than an informed guess. Nor is it only for developing countries that common bean data are problematic. The Food and Agriculture Organization of the United Nations (FAO) notes that some European data on area for common beans are overestimated and, consequently, yields are underestimated because of the combination of data from mixed cropping and monoculture (FAO, 1984a, p. 6).

World common-bean production can be conveniently grouped into twelve regions (Table 2), the most important of which are Brazil, Mexico, and eastern African highlands. Beans are a major staple in these regions which together contribute to half of the world's production. The USA and the Southern Cone of South America are major producers for export markets. Eastern and Western Europe are also significant producers, although Western Europe is also a major importer. In the African Great Lakes Region and Central America, beans are an important staple. West Asian production is concentrated in Turkey and Iran.

Per capita consumption of the common bean and its contribution to nutrition is highest in the African Great Lakes Region where beans provide one-third of total protein intake and one-eighth of total calories (Table 3). Beans are also very important in the eastern African highlands where one-sixth of proteins come from beans. Among the poor and middle classes in Brazil, Mexico, and Central America, the nutritional importance of beans is almost as high as in eastern Africa.

Latin America, the center of origin for the common bean, is the leading bean producer in the world. It contributes more than two-fifths of the total world production with an annual output of about four million metric tons. Beans are by far the most important pulse crop in Latin America, accounting for nearly 80% of total pulse production. The common bean is also the most important food

Table 2. Average production and yield of common beans in major production regions during 1982-84.

| Region^a | Production (t in thousands) | Yield (kg/ha) |
|-------------------------------|--|--------------------------|
| Brazil | 1801 | 458 |
| Mexico | 1215 | 623 |
| Eastern Africa | 1157 | 597 |
| North America | 988 | 1583 |
| Eastern Europe | 606 | 904 |
| African Great Lakes | 571 | 766 |
| Southern Cone | 411 | 1038 |
| Central America and Caribbean | 375 | 704 |
| West Asia | 299 | 1103 |
| Western Europe | 281 | 627 |
| Southern Africa | 256 | 631 |
| Andean | 181 | 611 |

a. Regions are defined as:

| | |
|--------------------------------|---|
| Eastern Africa: | Ethiopia, Kenya, Somalia, Tanzania, Uganda |
| Eastern Europe: | Albania, Bulgaria, Czechoslovakia, German Democratic Republic, Hungary, Poland, Romania, USSR, Yugoslavia |
| African Great Lakes: | Burundi, Rwanda, Zaire |
| Southern Cone: | Argentina, Chile, Paraguay, Uruguay |
| Central America and Caribbean: | Costa Rica, Cuba, Dominican Republic, El Salvador, Guatemala, Haiti, Honduras, Nicaragua, Panama |
| West Asia: | Iran, Turkey |
| Western Europe: | Austria, Belgium, France, Greece, Italy, Netherlands, Portugal, Spain, Sweden, United Kingdom |
| Southern Africa: | Angola, Lesotho, Madagascar, Malawi, South Africa, Swaziland, Zimbabwe |
| Andean: | Bolivia, Colombia, Ecuador, Peru, Venezuela |

SOURCE: Compiled by author from FAO, 1983, 1984a, and 1985.

legume in sub-Saharan Africa which is the second leading bean-producing region with an annual production of two million tons. The combined production of beans in North Africa, West Asia, and East Asia is slightly over half a million tons per year. However, in these regions the common bean is less important than other pulses.

Bean productivity is highest in North America where yields reach about 1.5 t/ha (Table 2). In the Southern Cone, West Asia, and

Table 3. Average consumption of the common bean (*Phaseolus vulgaris*) in major producing regions.

| Region ^a | Annual apparent consumption 1982-84 (kg per capita) | Share of total protein intake 1979-81 (%) | Share of total calorie intake 1979-81 (%) |
|----------------------------------|--|---|---|
| Brazil | 14.0 | 12.0 | 4.2 |
| Mexico | 16.5 | 10.6 | 5.1 |
| Eastern Africa | 19.3 | 16.9 | 7.3 |
| North America | 2.5 | 1.1 | 0.5 |
| Eastern Europe | 1.5 | 0.5 | 0.2 |
| African Great Lakes | 47.7 | 34.0 | 13.1 |
| Southern Cone | 4.1 | 2.1 | 0.9 |
| Central America and Caribbean | 9.8 | 7.6 | 2.9 |
| West Asia | 3.3 | 2.1 | 0.9 |
| Western Europe | 1.8 | 1.0 | 0.4 |
| Southern Africa | 4.6 | 3.0 | 1.2 |
| Andean | 3.2 | 3.2 | 1.2 |

a. Regions are defined in footnote of Table 2.

SOURCE: Compiled by author from FAO, 1983, 1984a, 1984b, and 1985.

Eastern Europe, yields are around 1 t/ha. Elsewhere, yields typically average 0.6 t/ha, except in Brazil where productivity is slightly lower.

Production growth has been highly variable among bean-producing regions over the last two decades (Table 4). Notable growth has occurred in high-yield regions of the Southern Cone and West Asia. Propelled by export opportunities, Southern Cone bean production increased at an annual rate of 8.4% during 1972-74 to 1982-84. It has surpassed the production of Central America, Western Europe, southern Africa, and the Andean region.

The largest absolute gain in bean production occurred in eastern Africa and the African Great Lakes Region where output increased nearly a billion tons over the last two decades (Table 5). Production in eastern Africa grew very rapidly during 1962-64 to 1972-74 at 6.1% per year and output continued to expand from 1972-74 to

Table 4. Average growth rates in production of common beans in major producing regions during the periods of 1962-64 to 1982-84.

| Region ^a | Annual percentage | |
|-------------------------------|--------------------------|--------------------------|
| | 1962-64 to 1972-74 | 1972-74 to 1982-84 |
| Brazil | 2.2 | 0.5 |
| Mexico | 2.2 | 3.3 |
| Eastern Africa | 6.1 | 2.8 |
| North America | 0.4 | 0.9 |
| Eastern Europe | -0.4 | 3.1 |
| African Great Lakes | 6.0 | 3.3 |
| Southern Cone | 5.1 | 8.4 |
| Central America and Caribbean | 1.0 | 2.5 |
| West Asia | 4.1 | 3.9 |
| Western Europe | -3.3 | -3.2 |
| Southern Africa | 2.5 | 1.2 |
| Andean | 1.4 | 0.5 |

a. Regions are defined in footnote of Table 2.

SOURCE: Compiled by author.

Table 5. Average common bean production (t in thousands) in major producing regions during the periods of 1962-64 to 1982-84.

| Region ^a | 1962-64 | 1972-74 | 1982-84 |
|-------------------------------|---------|---------|---------|
| Brazil | 1420 | 1726 | 1801 |
| Mexico | 742 | 905 | 1215 |
| Eastern Africa | 523 | 903 | 1157 |
| North America | 885 | 917 | 988 |
| Eastern Europe | 476 | 459 | 606 |
| African Great Lakes | 246 | 423 | 571 |
| Southern Cone | 120 | 192 | 411 |
| Central America and Caribbean | 273 | 299 | 375 |
| West Asia | 145 | 210 | 299 |
| Western Europe | 507 | 374 | 281 |
| Southern Africa | 184 | 230 | 256 |
| Andean | 152 | 173 | 181 |

a. Regions are defined in footnote of Table 2.

SOURCE: Compiled by author.

1982-84 at 2.8% per year. The African Great Lakes Region shows a similar pattern of rapid growth in the 1960s, followed by much slower growth in the 1970s.

Mexico has achieved significant advances in bean production over the last two decades, but production has been highly variable, particularly in recent years, and production trends are consequently less consistent than they may appear at first glance.

In most regions of the developing world, growth in bean production has tailed off in the last decade. Brazil, eastern Africa, the African Great Lakes Region, southern Africa, and the Andean zone all experienced slower growth during 1972-74 to 1982-84 than during the previous ten-year period. In the present decade, population growth has outstripped that of bean production in all four regions. Western European production has declined consistently to about half of 1962-64 levels and has dropped from fifth to tenth among bean-producing regions.

Comparison of annual growth rates in yields and area sown provide insights on the causes of declining growth among many bean producers (Table 6). In general, there has been little improvement in yields. This is true both for slow-growth regions such as the Andes and southern Africa, and for rapid growth regions such as the Southern Cone and West Asia. Area expansion in marginal agricultural lands has been the major source of production growth in Brazil, the African Great Lakes Region, eastern and southern Africa, the Southern Cone, and Central America. Where area expansion has slowed as land became scarcer, as in eastern Africa, the African Great Lakes Region, or the Andes, production growth rates have also fallen.

International trade in common beans is of relatively minor importance for countries where beans are a major staple such as Brazil, Mexico, eastern Africa, or the African Great Lakes Region (Table 7). However, bean imports can be critically important to Brazil and Mexico in order to supplement periodic production shortfalls. For example, Mexico imported an average of 400,000 t/yr in both 1980 and 1981. Other "production shortfall" importers are Cuba (73,000 t/yr) and Venezuela (65,000 t/yr). The biggest market for beans is

Table 6. Average growth rates for yield and area of common beans in major producing regions during 1962-64 to 1982-84.

| Region ^a | Yield | | Area | |
|----------------------------------|--------------------------|--------------------------|--------------------------|--------------------------|
| | (annual percentage) | | (annual percentage) | |
| | 1962-64 to 1972-74 | 1972-74 to 1982-84 | 1962-64 to 1972-74 | 1972-74 to 1982-84 |
| Brazil | -0.7 | -2.8 | 3.0 | 3.1 |
| Mexico | 3.9 | 0.9 | -1.5 | 2.3 |
| Eastern Africa | -0.7 | 0.8 | 6.7 | 1.9 |
| North America | -0.3 | 1.1 | 0.7 | -0.3 |
| African Great Lakes | 0.4 | 1.7 | 5.6 | 1.6 |
| Southern Cone | 0.0 | 1.2 | 5.2 | 7.3 |
| Central America and Caribbean | 1.1 | -0.4 | -0.1 | 2.9 |
| West Asia | 1.1 | -1.8 | 3.1 | 5.7 |
| Western Europe | 2.3 | 0.1 | -5.7 | -3.3 |
| Southern Africa | 0.7 | -0.7 | 1.8 | 1.9 |
| Andean | -0.3 | 0.8 | 1.7 | -0.3 |

a. Regions are defined in footnote to Table 2.

SOURCE: Compiled by author.

Table 7. Average international trade in common beans during 1982-84.

| Region ^a | Net trade balance ^b | Net value of balance | Trade as share of production |
|----------------------------------|-----------------------------------|-------------------------|---------------------------------|
| | (t in thousands) | (US\$ in millions) | (%) |
| Brazil | -18 | -14 | 1.0 |
| Mexico | -22 | -26 | 1.8 |
| Eastern Africa | +26 | +8 | 2.2 |
| North America | +349 | +181 | 35.4 |
| Eastern Europe | +5 | +0.2 | 0.8 |
| African Great Lakes | 0 | 0 | 0 |
| Southern Cone | +215 | +75 | 52.3 |
| Central America and Caribbean | -85 | n.a. ^c | 22.7 |
| West Asia | +6 | +4 | 2.6 |
| Western Europe | -350 | -158 | 124.6 |
| Southern Africa | -32 | -22 | 12.5 |
| Andean | -80 | -34 | 44.2 |

a. Regions are defined in footnote to Table 2.

b. Negative numbers indicate imports and positive numbers indicate exports.

c. n.a.: Data not available.

SOURCE: Compiled by author from FAO unpublished data.

Western Europe which imports over half of its consumption requirements. The principal exporters are United States (311,000 t/yr), Argentina (177,000 t/yr), and Chile (38,000 t/yr).

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Chapter 2

COMMON BEANS IN AFRICA AND THEIR CONSTRAINTS

D. J. Allen, M. Dessert, P. Trutmann, and J. Voss*

Introduction

The common bean (*Phaseolus vulgaris* L.) is an ancient New World domesticate. Beans spread widely in post-Columbian times and reached Africa from Brazil with the slave trade. They had reached Europe by the sixteenth century and probably spread to coastal parts of Africa not long afterward through the Portuguese. *Phaseolus vulgaris* became established as a food crop in Africa before the colonial era. The wealth of local names given to distinctive cultivars is evidence of the long establishment of beans as a food crop in East Africa (Greenway, 1945; Leakey, 1970a).

The total annual production of common beans in Africa is estimated at two million tons of dry seed. This is about 25% of world production (Table 1).

The Production Environment

The common bean is adapted to temperate and cool tropical climates. In Africa, production is concentrated in the cool highlands of central and tropical eastern Africa where beans are the most important pulse crop. However, beans are also grown as a winter irrigated crop in North Africa and parts of southern Africa. Within the highland areas, the production environment is diverse; the altitude ranges from 800 to 2300 m above sea level, although the higher elevation zones (1900-2300 m) are largely confined to the

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Table 1. Estimated annual production (t in thousands) of common beans in Africa, according to region.

| Region | Proportion of total production | |
|--------------------|--------------------------------|-------|
| | (t in thousands) | (%) |
| Great Lakes Region | | |
| Rwanda | 282 | 12.8 |
| Burundi | 193 | 8.8 |
| Zaire | 96 | 4.4 |
| Eastern Africa | | |
| Ethiopia | 33 | 1.5 |
| Kenya | 567 | 25.8 |
| Uganda | 259 | 11.8 |
| Somalia | 1 | > 0.1 |
| Southern Africa | | |
| Tanzania | 350 | 15.9 |
| Zambia | 35 | 1.6 |
| Malawi | 67 | 3.0 |
| Mozambique | 15 | 0.7 |
| Zimbabwe | 46 | 2.1 |
| Angola | 40 | 1.8 |
| Lesotho | 10 | 0.5 |
| Swaziland | 1 | > 0.1 |
| Other regions | 205 | 9.3 |
| Total Africa | 2200 | 100.0 |

SOURCES: CIAT, 1985 and 1986; FAO, 1986.

volcanic slopes of the Virunga region of central Africa. In contrast to Latin America, production of *P. vulgaris* in Africa gives way to *P. coccineus* L. above 2300 m. Most production is found on plateaus between 1200 and 1700 m.

Soil type also varies considerably between regions of production. Beans in the Ruhengeri district of northern Rwanda and to the west of Arusha in northern Tanzania, enjoy excellent fertile volcanic soils. Elsewhere, production can be seriously constrained by soil infertility, including acidity. Highly acid soils, with a pH as low as 4.2, are found in the bean-producing areas of Mbala district of northern Zambia, in the Usambara Mountains near Lushoto in Tanzania, and on the Nile Zaire Crest of Rwanda.

Mean temperature in the principal areas of bean production ranges from 16 to 24 °C. Annual precipitation is in the range of 500-2000 mm, with a bimodal distribution in eastern Africa (usually between latitudes 6° N and S) as a result of movements of the intertropical convergence zone. Average annual rainfall varies substantially with location and, in some places, particularly in the drier regions at the unstable frontiers of rainfall systems, rainfall is markedly variable from year to year (Bunting, 1961). A valuable method is available for calculating the confidence limits for seasonal variation in rainfall in East Africa (Manning, 1956). However, in bean-producing areas, mean precipitation during a single season varies relatively little: 400 mm (about the minimum rainfall required for a bean crop) to 800 mm. Seasonal length, from sowing to harvest, varies from about 70 days in drier lowlands to about 150 days in humid highlands, although obviously seasonal length depends also on latitude of the site and growth habit of the predominant bean cultivar.

The wide variability of production environments results in a wealth of diversity in cropping systems as well as in agronomic constraints to bean production.

Crop Production Systems

Beans are produced in a wide range of production systems in Africa. Large-scale monoculture production of navy beans for canning and export still occurs in some areas, although this industry has collapsed in northern Tanzania, Uganda, and Ethiopia where canning-bean production was once substantial. For example, in Tanzania, the production of navy beans for export started in 1937 and expanded to more than 2500 tons in 1952. Rising interest in the crop attracted inexperienced producers; quality therefore declined rapidly just when canners became increasingly demanding. In an effort to keep the industry alive, the cultivar Michigan Pea Bean was introduced into East Africa without careful testing for adaptation. Unlike the cultivar Comptesse de Chambord which was the principal cultivar grown in the early years, Michigan Pea Bean was especially susceptible to rust and, as a result, was almost totally destroyed.

Subsequent work focused on screening a collection of white-seeded types for suitability for local production for canning. The cultivar Mexico 142 was among those selected and is now one of the most widely grown navy beans in eastern Africa (Leakey, 1970a; Macartney, 1966; Robertson, 1955). In the Arusha region of Tanzania, about 25,000 ha of beans are grown on a large scale on contract to European seed firms. The cultivars grown are bush types selected for their acceptability in Europe as snap beans and are produced in monoculture. They receive more inputs, including aerial application of insecticide, than do food bean crops.

In the Great Lakes Region of central Africa, beans are grown primarily for home consumption and usually in association with other crops. In Burundi, although as much as 20% of the crop may finally be marketed, farmers almost never initially intend to market them (Bergen, 1986). The same situation arises in Rwanda where available data (SESA, 1984; J. Voss, unpublished data) reveal a home consumption rate of more than 80%. The north Kivu region of Zaire has a much higher degree of marketing with sales to Kinshasa and, in times of shortage, to Rwanda and Burundi. Although reliable statistics are not available, estimates suggest that market-oriented production may be as high as 70%.

The cultivation of staked climbing beans predominates in those parts of the Great Lakes Region which have high rainfall, high population density, and fertile soils. This includes the Ruhengeri and Gisenyi regions of Rwanda, most of north Kivu in Zaire, and parts of the west flank of the Nile Zaire Crest in Burundi. The main reasons for growing climbing beans in these areas are their greater resistance to pathogens (because of their physiological escape mechanism) and the need to intensify production (because of high population density).

Climbing beans are grown in a number of systems. At high altitudes, between 2000 and 2300 m, monoculture predominates, but relay cropping and associated cropping with maize are also practiced. At lower altitudes, 1200-2000 m, complex associations become more common. In Rwanda and Burundi the most common associations are with bananas (Figure 1)¹, maize (most commonly

1. This and all other numbered figures are collected together as a separate booklet at the end of the book. Lettered figures are found within the text.

staked between maize plants), and sweet potatoes. In north Kivu, staked climbing beans are most often grown in monoculture, perhaps because of the more market-oriented production. However, associations with maize, bananas, and coffee are also practiced.

Landraces of mixed seed type are common in Uganda (Leakey, 1970a), Malawi (Martin and Adams, 1985), southern Tanzania, and, especially, in the Great Lakes Region. Here, varietal mixtures (Figure 2) provide small farmers with a more reliable seed yield under low-input conditions, apparently by buffering against environmental stress, including disease. Work carried out by the International Service for National Agricultural Research (ISNAR) has demonstrated that most exotic varieties were less well adapted and more affected by diseases than the mixtures of local varieties used by farmers (ISNAR, 1983). The shift to cultivation of pure varieties is associated with market production. Consumer preferences for certain grain types apparently govern traders' demand for greater grain uniformity and price premiums, so accounting for this shift. Pure lines receive a market price premium over mixtures at about 20% in Burundi, as much as 100% in Zaire, and at over 900% in Uganda where uniformity and the need to meet consumer preferences are of paramount importance.

Food beans for subsistence are typically produced on a small scale, usually in association with other crops. In Uganda, an estimated 75% of all beans are grown in association on small farms. Similarly complex cropping systems are found in Kenya, the southern highlands of Tanzania, northern Zambia, and Malawi (Edje et al., 1981; Leakey, 1970a; Spurling, 1973). The crop most commonly associated with beans is maize, although the bean-banana-coffee association predominates in some areas. Other companion crops include sweet potatoes, peas, cassava, yams, cocoyams, potatoes, and peanuts (groundnuts).

In Malawi, more than 94% of cultivated land is under associated cropping (Edje et al., 1981) as in other densely populated areas, including the Kagera Region of Tanzania (Tibaijuka, 1984) and the Great Lakes Region. Associated cropping is more common in areas where land is scarcer (because of denser human population) and less common in areas where production is more market oriented (as in

Kenya). However, monoculture seldom accounts for more than 40%. Associated cropping offers several advantages to the small farmer: it enables greater productivity where land is restricted (Neumann et al., 1986), it decreases the risk of complete crop failure, and it often decreases disease severity (Msuku and Edje, 1982; van Rheenen et al., 1981). The banana-bean association is common in Rwanda, Burundi, Uganda, and the Kagera Region of Tanzania. In Rwanda, 60% of bean production is estimated as being in association with banana (Nyabyenda et al., 1981). The situation is similar in Burundi. The banana association plays an important role in reducing drought stress for the associated bean crop and thus improves the stability of the system. However, the water and nutrient relations of the banana-bean association have not received sufficient attention (Osiru and Mukiibi, 1984). In the coffee-growing areas of north Kivu, Zaire, coffee is always associated with beans.

Crop Production Constraints

The main production constraints reported in the literature are poor agronomic practices, soil infertility, lack of improved cultivars, moisture stress, weed competition, and damage caused by pests and diseases. However, in systems involving complex associations, the claim often made by researchers that farmers' practices are sub-optimal is difficult to evaluate objectively because research designs become almost impossibly complex. Too often, assumed priorities reflect prejudices on part of the scientist rather than the true constraints to crop productivity. Indeed, some systems of subsistence agriculture are balanced, self-supporting, tropical agroecosystems (Igbozurike, 1971; Janzen, 1973) in which coevolved crops have achieved an equilibrium, not only with one another and with their environment (Bunting, 1975), but also with their parasites. Consequently, the farmer always has a stable source of food for himself and his family, rather than risk hunger for the sake of high productivity. The poorer the farmer and the less fertile the soil, the more important yield stability becomes. His decision to grow beans in complex associations and often in varietal mixtures therefore stems from the need to maximize stability of performance rather than productivity per se. The determination, then, of the relative

importance of production constraints can and must be performed with diagnostic exploratory trials onfarm. This will set realistic priorities for future research in each agroecological zone in which beans are produced. For example, in those parts of Rwanda where beans have been cultivated for several centuries, onfarm trials have yet to show significant yield advantages of new varieties over traditional ones. Conversely, in areas of recent immigration, new varieties have shown yield advantages of as much as 35% superior to farmer mixtures (Graf and Trutmann, 1987).

The Centro Internacional de Agricultura Tropical (CIAT) team in the Great Lakes Region has been using a multitiered approach to identify the main production constraints. This consists of a combination of farmer surveys, informal interviews, trials to determine limiting factors, and onfarm varietal trial evaluations. Farmer surveys in Ruhengeri, Rwanda, show that insect attack, drought, excess rain and associated diseases, low soil fertility and insufficient compost and manure, and lack of land were all considered by farmers as significant production constraints (Table 2).

Table 2. The importance of varietal characteristics, according to 120 farmers interviewed in Ruhengeri, Rwanda, 1985-86.

| Importance | Characteristic | Score ^a |
|----------------------|----------------------|--------------------|
| High importance | Yield | 92 |
| | Rain tolerance | 85 |
| | Earliness | 78 |
| | Drought tolerance | 76 |
| Medium importance | Taste | 60 |
| | Upright architecture | 48 |
| Low importance | Storability | 36 |
| | Fast cooking | 31 |
| | Green bean quality | 29 |
| | Leaf quality | 20 |
| | Color | 6 |

a. Scoring is based on a scale of 0 to 100 where 100 signifies that all farmers identify the characteristic as very important.

SOURCE: J. Voss and K. Dessert, unpublished data.

Trials in the Great Lakes Region to determine limiting factors have shown soil fertility and diseases to be the two most limiting factors under most production conditions. A clear negative interaction between soil fertility and disease is often found. Gains made through increasing soil fertility are offset by losses from increasing disease pressure if diseases are not controlled. If a farmer is forced by economic or labor considerations to choose between increasing soil fertility or controlling diseases, the latter is more likely to bring about significant yield increases (Graf and Trutmann, 1987; Trutmann and Graf, 1987).

At lower altitudes in the Great Lakes Region, and elsewhere in eastern and southern Africa, insect pests are also significant limiting factors. Bean fly (*Ophiomyia* spp.) can cause substantial damage, especially on less fertile land. Recent work in northern Zambia suggests that application of fertilizer onfarm may effectively suppress the damage resulting from bean-fly infestation.

Disease as a Production Constraint

The common bean was introduced to the highlands of eastern Africa about 400 years ago and the highlands are now a secondary center of genetic diversity. It appears that accompanying the crop were many of the seed-borne pathogens that plague the crop in its primary center of origin in the New World. The principal diseases of beans are, therefore, essentially the same in the two centers. Nevertheless, there are a few important dissimilarities in the pathogen spectra of the two continents.

Literature on bean diseases in Africa is fragmentary. Most major reviews have not dealt extensively with African literature, although Allen (1983) has attempted to redress the imbalance. Notable gaps in knowledge of the importance of bean pathogens include Angola, Cameroun, Chad, and Togo, each of which is a significant producer of the crop.

In comparison to fungi and bacteria, whose distributions are relatively well cataloged in territorial checklists of pathogens (CMI, 1970, 1971, and 1979), virus distribution is poorly known. Because viruses are difficult to identify, maps of their distribution in Africa

are prone to inaccuracy, especially when identification has been based on symptomatology alone.

The most important virus pathogen of beans in Africa is the bean common mosaic virus (BCMV). It is reliably identified from central and eastern Africa where necrotic strains are common and damaging (CIAT 1987; Kulkarni, 1973; Mink, 1985; Omunyin, 1979; Silbernagel et al., 1986). Peanut stunt virus has been identified recently in beans in the Sudan (Ahmed and Mills, 1985) but cucumoviruses are not known from beans in East Africa (Bock et al., 1975). Similarly, southern bean mosaic virus (SBMV) has not yet been detected in beans in eastern Africa, although it is known in legumes in western Africa (Givord, 1981; Lamptey and Hamilton, 1974). Bean golden mosaic virus (BGMV) has not been found, although a closely related virus occurs in lima beans (*Phaseolus lunatus* L.) in Nigeria (Vetten and Allen, 1983; Williams, 1976). Cowpea mild mottle virus, known in various legumes in West Africa, has recently been found in natural infections of bean in Tanzania (Mink, 1985). Alfalfa mosaic virus is recorded in beans in South Africa (Neveling, 1956). Both tobacco mosaic virus (Hollings et al., 1981) and bean yellow mosaic virus (BYMV) have been recorded in beans in Kenya, although BYMV is now thought as eradicated. Peanut mottle virus is also known from *Phaseolus* spp. in East Africa (Bock, 1973).

Among the bacterial diseases, the only one of uncertain status is bacterial wilt caused by *Curtobacterium flaccumfaciens* (syn. *Corynebacterium*) which is thought to occur in Kenya (Hubbeling, 1973). Bacterial brown spot, incited by *Pseudomonas syringae* van Hall pv. *syringae*, is also known from beans in Kenya and Burundi (Duveillier and D. Perreux, personal communication, 1986; Kaiser and Ramos, 1980). Both common bacterial blight and halo blight are widespread and important.

The major fungal diseases of beans in Africa, as in Latin America, are angular leaf spot, anthracnose, and rust. Ascochyta blight is very damaging in the highlands of the Great Lakes Region, and floury leaf spot, caused by *Mycovellosiella phaseoli* (Drummond) Deighton, is locally important. Web blight is probably of little importance (unlike in Central America where it is severe). Certain fungal pathogens have not been reported from Africa, including

white leaf spot caused by *Pseudocercospora albida* (Matta *et* Belliard) Yoshii *et* Aamodt, gray leaf spots (*Cercospora vanderysti* P. Henn. and *C. castellanii* Matta *et* Belliard), and the round leaf spot, *Chaetoseptoria wellmanii* Stevenson. Conversely, scab (Figure 3), caused by *Elsinoë phaseoli* Jenkins is known from beans only in Africa, although it is a pathogen of lima bean and cowpea in the New World (Allen, 1983; Jenkins, 1931).

There is evidence, in some cases, of substantial diversity among pathogens in Africa. Studies of anthracnose (Ayonoadu, 1974; Leakey and Simbwa-Bunnya, 1972), rust (Allen, 1975a; Howland and Macartney, 1966; Mmbaga and Stavely, 1986), and angular leaf spot (Hocking, 1967) have each revealed new variants that do not correspond exactly with races described in the New World. Preliminary evidence from studies on ascochyta blight in Africa suggest that the most important causal agent is *Phoma exigua* var. *diversispora* (Bub.) Boerema and not *P. exigua* var. *exigua* Desmazieres, the latter being a synonym of *Ascochyta phaseolorum* Saccardo (Boerema, 1972; Boerema *et al.*, 1981; M. Gerlagh and G. H. Boerema, personal communication, 1986).

Recent collaborative studies on halo blight by J. D. Taylor from the National Vegetable Research Station in England and scientists at CIAT have identified new races of *Pseudomonas syringae* pv. *phaseolicola* not known to occur outside Africa. Similarly, the predominance of necrotic strains of BCMV in eastern Africa contrasts with known strain spectra elsewhere. This raises the question of the origin of some of these variants. It is no longer certain that they all have necessarily coevolved with *P. vulgaris* and have been transported with its seed.

Estimates of the relative importance of bean diseases in Africa (Table 3) have been obtained chiefly from studies conducted on research stations where artificial inoculation can be relied upon. While such estimates can give some indication of potential loss, they do not always accurately reflect the relative importance of a particular disease among other agronomic constraints experienced on the farm.

Table 3. Estimates of crop losses induced by pathogens in beans in Africa.

| Disease | Cultivar | Crop loss (%) | Source |
|-----------------------------|--------------------|---------------------|-----------------------------|
| Anthracnose | — | 92 | Peregrine, 1971 |
| | T 8 | 86 | Shao and Teri, 1985 |
| | Mexico 142 | 27 | Shao and Teri, 1985 |
| | T 3 | 4 | Shao and Teri, 1985 |
| Angular leaf spot | Selian Wonder | 25 | Swai and Keswani, 1984 |
| | Kabanima | 8 | Swai and Keswani, 1984 |
| Rust | White-seeded types | 100 | Howland and Macartney, 1966 |
| | Selian Wonder | 11 | Mbowe and Keswani, 1984 |
| | Canadian Wonder | 14 | Mbowe and Keswani, 1984 |
| Scab | — | 43-76 | Mutitu, 1979 |
| Bean common mosaic virus | Kabanima | 14-18 | Meketo and Keswani, 1984 |

Recent results from diagnostic onfarm trials in Rwanda have recorded grain yield increases of 400-1000 kg/ha in beans from the chemical control of fungal and bacterial pathogens. In the highlands, above 1900 m, there are demonstrable advantages in using combined resistance to anthracnose, angular leaf spot, and ascochyta blight, as well as controlling root diseases. At intermediate altitudes, anthracnose and angular leaf spot resistance is required, and BCMV resistance is necessary for climbing cultivars (Trutmann and Graf, 1987).

In Zambia, Greenberg et al. (1987) have used multiple regression analysis of disease scores against seed yield of beans to estimate yield loss caused by pathogens and to set priorities among diseases at any given location. Ohlander (1980) took a similar approach to bean diseases in Ethiopia, demonstrating that similar studies are required elsewhere, because priorities change from location to location.

More work is also needed on the possible interactions between pathogens and the diseases they cause (Allen and Russell, 1987). Casual observations in the field suggest that interactions may

sometimes lead to misidentification of diseases and perhaps also to alteration of host responses in resistance screening.

Disease Management

Current practices

Surveys in Rwanda demonstrate that farmers' conceptual knowledge of "disease" is very scanty: "disease" is almost always equated with "too much sun" or "too much rain" (CIAT, 1985). Chemical control of disease in beans is almost nonexistent because of the scarcity of agrochemicals, limited access to equipment with which to apply pesticides, and the meager capital available to smallholders for buying them. Nevertheless, there is evidence that current cultural practices adopted by many bean farmers do limit disease severity and spread. Traditional practices such as shifting cultivation, with its intervening periods of bush fallow; the burial of crop debris in mounds² in the chitemene farming system of northern Zambia (Richards, 1939); and the cultivation of crop mixtures, provide some measure of disease management. Recent studies (CIAT, 1986 and 1987) show that roguing of diseased seedlings and removal of diseased basal leaves at weeding can decrease disease incidence. The chosen time of sowing and plant population may also, in some instances, aid escape from disease. Studies in the southern highlands of Tanzania suggest that the selection of unblemished seed by farmers is also likely to lessen disease severity in a subsequent crop (F. M. Shao, unpublished data, 1983).

Various studies on the effect of crop association on disease severity have shown that diseases of beans are usually, but not invariably, less severe in a maize intercrop (Msuku and Edje, 1982; van Rheenen et al., 1981). Various factors have been suggested such as impeded spore dispersal, altered microclimate, and various biotic effects (Allen, 1975b; Allen and Skipp, 1982; Moreno, 1977).

Similarly, varietal mixtures of beans are more stable and better buffered against disease than are pure lines (Ishabairu and Teri,

2. The mounds are made when clearing the cropping land. Crop debris and residues, grasses, and weeds are piled up and covered with earth. The mounds are then left until they convert to compost when they are used as fertilizer for the cropping land.

1983; A. Panse and J. H. C. Davis, unpublished data, 1986). This is in keeping with similar studies done on mixtures of cereals (Jeger et al., 1981; Wolfe et al., 1981).

Prospects for improved systems of integrated disease management

Existing systems of crop production in Africa tend to be stable, being adapted to the environment and current needs and resources of the small-farming family. However, they may not be sufficiently productive to meet the needs of the future. In order to increase their productivity, we must understand how existing cropping systems work. The next step is to devise means of changing those systems, albeit without recourse to heavy input. Bunting (1983) has suggested that the first gift agricultural science has to offer to a crop producer is a range of improved varieties that are adapted to the local environment and that have some built-in resistance to as many as possible of the pests and diseases which are locally important. Indeed, among the control strategies available, host-plant resistance has become widely recognized as the pivot of integrated disease management, to which both chemical and cultural control measures may contribute. Resistant cultivars cost the farmer nothing, nor does their adoption necessarily disrupt his farming system.

Very little attention was given to the genetic improvement of beans for local consumption in Africa before independence. In eastern Africa, for example, breeding efforts were directed at the selection of navy bean cultivars for canning and export (Macartney, 1966; Robertson, 1955). Work on beans as a subsistence crop has been confined, in effect, to the last 25 years. A breeding program, begun by S. K. Mukasa and continued by C. L. A. Leakey in Uganda, was the first and, perhaps, most successful (Leakey, 1970a). Subsequent programs have been established in many other countries, notably Malawi (Edje et al., 1981; Mughogho et al., 1972), Kenya (Njugunah et al., 1981; van Rheenen, 1979), Tanzania (Karel et al., 1981), Rwanda (Nyabyenda et al., 1981), Ethiopia (Assefa, 1985; Ohlander, 1980), and Zambia (Grain Legume Research..., 1986?; Sarmezey, 1977).

Improved cultivars have been released by many of these national programs. In Uganda, during the mid 1960s, selections made for

resistance to anthracnose among local cultivars led to the naming of Banja 2 which was subsequently used as a parent in hybridization. Banja 2, in turn, led to the K series of lines, notably K 20, many of which outyielded Banja 2. Some also possessed resistance to angular leaf spot in addition to anthracnose. Crosses made during the sixties in Uganda formed the nucleus for further improvement. Lines such as K 20 and Kabanima, are now found in many African countries (Leakey, 1970a). K 20 was later released as GLP 2 in Kenya in the early 1980s and Kabanima was released in Tanzania in 1978 (Karel et al., 1981). Releases made recently in Tanzania include P 304 (a climbing type with large cream-colored seed of Colombian origin, renamed Uyole 84) and T 23 (like Kabanima, a large-seeded sugar bean, renamed Lyamungu 85).

The contribution of breeding and selection to improvement in productivity is most spectacular in Zambia, where Carioca was released as a new bean variety in 1985. Under experimental conditions, Carioca has shown an average improvement in seed yield of 450% over the previously recommended variety, Misamfu Speckled Sugar. In onfarm trials it has given almost double the yield of local cultivars without added inputs. The superiority of Carioca appears to depend on its combined resistance to scab (in Zambia), angular leaf spot, and anthracnose, as well as tolerance to soil acidity (Grain Legume Research..., 1986?).

Similar improvements are expected to occur elsewhere, as further advances in disease-resistance breeding are made. The bases for further improvements are more effective use of the very extensive germplasm collection of *Phaseolus* held at CIAT, more reliable methods of field screening against disease, more precise definition of agroecological zones to more accurately deploy in the environment combined resistance and the cultivars that possess it, and further development of regional networks for the effective exchange of superior genotypes, information, and ideas (Allen and Ndunguru, 1984). Since 1983, three regional programs have been based in Rwanda, Ethiopia, and Tanzania to serve the Great Lakes Region of central Africa, eastern Africa, and southern Africa, respectively.

It has long been appreciated that there is no premium on genetic uniformity in tropical subsistence farming and there is no need to develop pure lines of beans in Africa (Leakey, 1970b). In fact, it is

important to retain enough genetic diversity for cultivar improvement, particularly as future systems of bean production are likely to be more intensive in terms of time and space, especially in areas already densely populated. Such intensity in turn will lead to concomitant changes in disease pressure. Host-plant resistance has to be supported by higher standards of seed health (through selection and safer seed dressings) and by diversified systems of farming that provide some measure of protection from disease. It may be possible to alter the components of varietal mixtures without impairing their intrinsic balance.

In systems where varietal mixtures predominate, methods of disease control other than host-plant resistance remain an important component of disease management strategy. Time must be allocated to investigate farmers' current practices to identify areas where simple improvements to the system can be made. Cultural practices are important because of their intrinsic bias toward small farming where the land to labor ratio is low. Better cultural practices can improve the quality of farmers' seed (CIAT, 1987; Trutmann and Kaytare, 1986). The use of specific crop associations, rotations, or composts may reduce foliar and soil-borne diseases.

Although available technologies have been recently reviewed by Palti (1981) and Hoitink and Fahy (1986), little is known about technologies currently used by African farmers. Certain chemical seed treatments may find a place where specific problems such as root rots and seed-borne pathogens, are severe (Trutmann, 1987). Similarly, cheap phytosanitary products have an important role in the production of high quality seed of improved varieties.

The challenge that now confronts Africa is to devise means of bringing about significant improvements in productivity without placing heavy reliance on added inputs and without adversely disrupting existing systems of cropping. Development of sustainable cropping systems with beans is likely to rest substantially upon effective disease management. New materials and methods are now being developed through cooperation between CIAT, other international agencies, and the national bean programs. If they are used effectively in the environments to which they are adapted, then a significant impact can be made on bean production in Africa.

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Chapter 3

COMMON BEANS IN LATIN AMERICA AND THEIR CONSTRAINTS

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Introduction

Statistical information in Chapter 1 shows that Latin America ranks first in bean production and consumption among the tropical regions of the world. Beans are grown throughout the continent from the northern states of Mexico (30° N) down to regions as far south as the Chiloé Island in Chile (43° S). In Brazil, beans are grown in the Amazon basin where it is warm and humid, in the northeast where it is warm and dry, and in the subtropical highlands in the south. In Argentina, beans are grown in the northwestern provinces, from 150 km N to 600 km S of the Tropic of Capricorn, at 300 to 1000 m.a.s.l., and with 45 to 1000 mm of annual rainfall. In Chile, they are produced in the dry and warm central lowlands under irrigation. In Peru, beans are grown in the arid coastal valleys, the eastern and western valleys of the Andean highlands, and the Amazon basin. In Colombia, Ecuador, and Bolivia, beans are produced in the Andean valleys during two rainy and two dry seasons annually. In Venezuela, bean production takes place in the north coast at sea level where it is hot and humid, and in mountain valleys and tablelands which are subtropical. In Central America, they are grown on the dry and warm Pacific slopes, on mountain sides and cooler high valleys, and in the warm, moderately dry, interior lowlands.

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In Mexico, they are produced in the north which has a continental climate, in the warm central tablelands under irregular rainfall patterns, and in most areas at sea level.

Beans are not widely grown on the Atlantic side of Central America and the Caribbean area where rainfall is heavy and high humidities prevail. Neither are they grown above 3000 m.a.s.l. in Peru, Ecuador, or Bolivia. Considering the wide diversity of climates, soils, and socioeconomic environments found between the Tropics of Cancer and Capricorn, it is not surprising that bean production in Latin America is subject to numerous constraints that vary from region to region; nor that beans are produced under widely differing cropping systems (Andrews and Kassam, 1976), with different plant types, and seeds of varying colors and sizes (Voysest, 1983).

Beans as a Domestic and Export Product

Common beans marketed as dry beans are used entirely for consumption by humans in Latin America. However, consumption patterns show wide variation (Table 1). Argentinian or Chilean annual consumption is low compared with that of Brazil or Mexico but this does not prevent the former countries from devoting a considerable area to beans for export.

Latin American countries can be grouped into three categories:

Net exporters. Argentina is a typical case: the land area cultivated under common beans increased to 200,000 ha in the eighties and Argentina is the leading bean exporter in Latin America. Beans are grown in the northwestern provinces (Salta, Tucumán, Santiago del Estero, and Jujuy). About 5000 ha of beans are grown for local consumption in Misiones, a province neighboring Brazil and Paraguay.

Exporters and consumers. Chile is the most representative country in this category. Although figures vary annually, usually half of the Chilean bean production is for export (FAO, 1982). It consists mainly of pea, black, Red Mexican, Red Kidney, and pinto bean types. The locals, however, prefer other colors and sizes such as gray or light tan, and medium- to large-sized grains. For the other

Table 1. Annual per capita bean consumption (kg) in Latin America.

| Country ^a | Annual per capita consumption (kg) | | | | | | | | | |
|-----------------------|------------------------------------|-----|-----|-----|-----|------|-------|-------|------|---|
| | 0-1 | 1-2 | 2-3 | 3-5 | 5-7 | 7-10 | 10-13 | 13-16 | > 16 | |
| Chile | | | | X | | | | | | |
| Argentina | | X | | | | | | | | |
| Uruguay | X | | | | | | | | | |
| Paraguay ^b | | | | | | | | | | X |
| Brazil | | | | | | | | X | | |
| Bolivia | X | | | | | | | | | |
| Peru | | | X | | | | | | | |
| Ecuador | | | | X | | | | | | |
| Colombia | | | | X | | | | | | |
| Venezuela | | | | | X | | | | | |
| Panama | | | X | | | | | | | |
| Costa Rica | | | | | | | X | | | |
| Nicaragua | | | | | | | | | | X |
| Honduras | | | | | | X | | | | |
| El Salvador | | | | | | X | | | | |
| Guatemala | | | | | | | X | | | |
| Mexico | | | | | | | | X | | |
| Dominican Republic | | | | | | X | | | | |
| Haiti | | | | | | X | | | | |
| Cuba | | | X | | | | | | | |

a. Countries are listed from south to north.

b. Possibly includes cowpea (*Vigna unguiculata* (L.) Walp.).

SOURCE: FAO (Food and Agriculture Organization of the United Nations). Various issues. Food balance sheets. Rome, Italy.

Latin American countries of this category, export sales are more sporadic and not as significant.

Net consumers. This category embraces most Latin American countries among which there are large differences in annual consumption per capita. In Brazil and Mexico, during 1979-81, the average per capita consumption was between 14.0 and 16.5 kg of beans per year, while in Argentina and Uruguay, it was less than a kilogram. Table 1 shows that per capita bean consumption in Latin America declines as one moves south from Mexico to Chile, with Brazil and Paraguay being exceptions. In some countries such as Paraguay and Bolivia, the urban population consumes more beans in comparison with the rural population, particularly in Paraguay.

Usually, however, urban populations consume fewer beans than rural populations.

Classes of Beans Grown in Latin America

The types of beans grown in Latin America are listed in Table 2. The class most widely distributed is the black bean. The high daily consumption of black beans in Mexico, Guatemala, Cuba, Venezuela, parts of Brazil, Central America and the Caribbean, Misiones Province in Argentina, and Santa Cruz Department in Bolivia makes this class of bean attractive to countries such as Argentina and Chile, which grow black beans exclusively for export.

Small reds form another important bean class. These beans are grown in El Salvador, Honduras, Nicaragua, Costa Rica, Jamaica, Cuba, and Brazil. Although the small red beans have an attractive appearance, suitable for export markets, the diversity of preferences in color intensity, shape, size, and brightness means that they are rarely grown for export.

For the same reasons neither are red-mottled beans commonly exported. For example, in the Caribbean there is strong preference for the round, medium-sized, variegated beans (Miss Kelly in Jamaica, Pompadour in Dominican Republic), whereas in the Andean zone, particularly Colombia, the elongated large-sized grains such as Diacol Calima are preferred. Variation of consumer preference in this class is largely governed by the tones of colors involved, their patterns, and base colors. Other classes of red beans include the solid-red, large beans that are grown in the Caribbean, Colombia, and Ecuador and the Red Kidney types that are planted in the Caribbean and southern highlands of Peru for local use, and in Chile and Argentina for export.

The "bayo" class, a generalized name for a type of beans with a seed color ranging from cream to light tan, is also widely distributed—in Mexico, Brazil (where they are known as *Mulatinhos*), Ecuador, Peru, and Chile.

The sulfur-yellow class of beans are grown in coastal areas of Peru where they are known as *Canarios* and in Mexico where they are known as "*Azufrados*" or "*Peruanos*." Other types of yellow

Table 2. Classes of beans grown in Latin America.

| Color | Country | Class | Equivalent U.S. class |
|--------|-----------------|---------------------------|-----------------------|
| White | Chile | Arroz | Navy |
| | Chile | Cristal Blanco | White Marrow |
| | Peru, Ecuador | Panamito | Small White |
| | Peru | Caballero | White Marrow |
| | Argentina | Alubia | |
| Cream | Brazil | Mulatinho | |
| | Brazil | Carioca ^a | |
| | Mexico | Bayo Gordo | |
| | Mexico | Bayo Blanco | |
| | Mexico | Ojo de Cabra ^a | |
| | Peru | Bayo Chimú | |
| | Peru | Cocacho | |
| | Chile | Bayo Titán | |
| | Chile | Cristal Bayo | |
| | Chile, Ecuador | Bayo Bolón | |
| | Chile | Hallados Alemanes 114 | Pinto |
| | Mexico | Pinto Nacional | Pinto |
| | Colombia | Cargamanto ^a | Cranberry |
| | Uruguay | Frutilla ^a | Cranberry |
| Yellow | Brazil | Jalo and Jalinho | |
| | Brazil | Enxofre | |
| | Mexico | Azufrado | |
| | Mexico, Peru | Peruano | |
| | Mexico | Canario | |
| | Mexico | Garbancillo | |
| | Peru | Canario | |
| | Peru | Amarillo Gigante | |
| | Peru | Ucayalino | |
| | Ecuador | Canario Bolón | |
| Brown | Brazil | Chumbinho | |
| Pink | Brazil | Rosinha | |
| | Mexico | Rosita | Pink |
| | Mexico | Flor de Mayo ^a | |
| | Colombia | Andino ^a | |
| | Belize, Jamaica | Miss Kelly ^a | |
| | Argentina | Chaucha Colorada | |
| | Cuba | Mulangrí | |
| | Peru | Rojo Mollepetá | Red Kidney |
| | Chile | Red Kloud | Red Kidney |
| | Belize, Jamaica | Red Kidney | Red Kidney |
| | Cuba | Velasco Largo | Red Kidney |

(Continued)

Table 2. (Continued).

| Color | Country | Class | Equivalent U.S. class |
|--------|---|-----------------------------------|--------------------------|
| Red | Central America | Small reds | Red Mexican |
| | Brazil | Roxinho, Roxão | |
| | Colombia | Calima, Nima | |
| | Colombia | Gualí, Catfo ^a | |
| | Colombia | Radical, Sangretoro, Bola Rojo | |
| | Ecuador | Cargabello ^a | |
| | Dominican Republic | Pompadour ^a | |
| Purple | Colombia | Mortiño ^a | |
| Black | Central America, Mexico, Caribbean, Venezuela | Negro | Black Turtle Soup |
| | Brazil | Preto | Black Turtle Soup |
| Gray | Chile | Tórtola | |

a. The color is not solid.

SOURCE: Voysey, 1983.

beans are also grown in the highlands of these countries—one of them, known in Mexico as “Canario,” is also grown in Panama, Ecuador, Bolivia (under the name of “Manteca” or “Mantequilla”), and in Brazil where it is called “Jalo.”

The white-seeded beans, large and small, are grown in Peru and Ecuador. Chile grows mainly the small white beans and Argentina the large ones. Brazil, in addition to black (Pretos), cream (Mulatinhos), and yellow (Jalo) beans, also grows a type of small-seeded beans known as Rosinha (pink), Roxinho (red), Chumbinho (brown), and the widely grown Carioca (cream with dark stripes). The production and consumption pattern of beans in Latin America is complicated by strong traditional consumer preferences for color and grain size. To further complicate the picture, farmers have their own preferences, especially with regard to plant types that most suit their particular production system.

Bean Production Structure

A large part of bean production in Latin America takes place on small farms ranging from 1-10 ha in size, often on sloping land of limited fertility. Some estimates suggest that perhaps 80% of the area planted with common beans in Latin America is found on hill sides. Moreover, these small holdings are dispersed and, in contrast to other crops, a main production area can seldom be determined (Aguirre and Miranda-M., 1973; Hernández-Bravo, 1973).

In Brazil, one of the largest bean producers of Latin America and which accounts for about half of the Latin American production, an estimated 34% of production is on farms of less than 10 ha. In Mexico, which contributes one-fourth of the Latin American bean production, an estimated 67% of its production comes from farms of less than 5 ha (Pachico, 1982). Even in Chile, an important bean exporter in the region, beans are produced by small to medium growers whose farms vary from 20-40 ha (Fassbender, 1967). Except for Argentina where beans are usually produced on large holdings with considerable technical input, Latin American beans are usually produced by small landholders. More than half the production occurs on farms smaller than 20 ha and more than 20% on farms of less than 5 ha (Pachico, 1984). The extreme cases are represented by countries such as Haiti, the Lesser Antilles, and Paraguay where production is almost exclusively done by small-farm families. In the remaining countries, production is usually done by small-farm families and small-scale commercial producers. In Mexico, Brazil, Chile, and Cuba, it is possible to find the three types of beans producers. Colombia, Venezuela, Dominican Republic, Peru, Guatemala, and Costa Rica have limited areas where large-scale, highly mechanized production occurs.

D. Pachico (unpublished data) classified bean-producing regions based on economic resources such as land, availability of labor, fertilizers, and pesticides. This gives a useful idea of the diversity in the structure of bean production in Latin America. These classes are:

Frontier, extensive: Land is plentiful relative to labor; large farms are mechanized; low investment put in fertilizers and

pesticides. Examples include Argentina (northwest), Costa Rica (Upala), Guatemala (Petén), and Brazil (Mato Grosso, Goiás).

Small farm, intensive: Labor is plentiful relative to land; moderate to favored environmental conditions; may invest in fertilizers and pesticides. Examples include Colombia (Antioquia, southern Nariño), Costa Rica (San Isidro del General), and Brazil (highlands of Espírito Santo, Paraná, Santa Catarina).

Small farm, extensive: Moderate to high ratio of labor to land; little capital investment; less favorable growing conditions (drought, poor soils). Examples include Peru (Chota), Mexico (arid highlands), and Brazil (Bahia).

Large farm, mechanized: Agrochemicals used in moderately favorable conditions. Examples include Brazil and Mexico.

Irrigated: Moderate to high labor and capital inputs. Examples include Chile (central valley), Peru (coastal regions), Mexico (Sinaloa), and Brazil (coastal Espírito Santo).

Another criterion can be used to classify bean-production regions, based on the cropping systems. Without attempting to establish a definitive classification, it is apparent that Latin American beans are grown under five main production systems:

Bush beans in monoculture: This system is common in low-to-medium altitude areas, chiefly in Brazil, Argentina, Mexico, Chile, Peru, Cuba, and the Dominican Republic.

Bush, semiclimbing, and climbing beans in relay systems with maize: The relay system is mainly found in low to intermediate altitudes of Colombia (Antioquia) and Central America.

Bush beans intercalated with maize: This system, where maize and beans are usually sown at the same time, is common in intermediate altitudes in Colombia, Venezuela, Brazil, and Central America.

Climbing beans in direct association with maize: The system is found in the higher altitudes (2000 m.a.s.l.) of Colombia, Ecuador, Guatemala, and Peru.

Covered bean ("tapado" system): This system is found in lower and intermediate areas with high precipitation such as Costa Rica, El Salvador, and Nicaragua.

The system of bush beans in monoculture can be used by both small and large farmers while the other four systems are used only by small farmers.

In Latin America beans are often grown in association, principally with maize, but also with cassava, coffee, potatoes, and other crops (de Andrade et al., 1974; Hernández-Bravo, 1973; Moreno-R. et al., 1973; Ruiz de Londoño et al., 1978). About 60% to 80% of Latin American bean production is in association with other crops (Gutiérrez-P., et al., 1975; Pinchinat et al., 1976). Whether relay or simultaneous planting system is adopted depends mostly on precipitation patterns. Where there is a unimodal rainfall distribution the relay system is usually employed: maize is planted in the first, more rainy, season; climbing beans are planted in the second season; the beans use the maize as a support. In Central America and in some areas of the Andean zone such as Antioquia in Colombia, this is the most common production system (Bastidas-Ramos, 1977).

In high, cool areas where the growth period of beans and maize is long during the single rainy period, associate cropping is the predominant system. This is the case in the highlands of southern Colombia, Ecuador, and Peru where maize and beans are planted simultaneously. Beans intercalated with maize is a system that is used in almost all bean-producing zones of Central America and Brazil.

The "covered bean" ("tapado") system is a primitive production system which predominates in regions of very high precipitation in Costa Rica and Nicaragua. Seed is broadcast over a plot covered by certain weeds. The weeds are then cut down by hand with machete and thrown over the seeds to cover them (Aguirre and Miranda-M., 1973). This system, primitive and low producing as it may be, is excellent on erosion-prone slopes and in the management of the splash-dispersed inoculum of web blight¹ (*Rhizoctonia solani*

1. Also caused by *Thanatephorus cucumeris* (Frank) Donk. which is the perfect stage of *Rhizoctonia solani*.

Kühn) which causes a serious foliar disease. The cut-down weeds form a mulch that covers the blight and prevents its dispersal. However, this system may favor slug survival and crop damage in some production regions in Central America.

Constraints to Production

Of the major world crops, beans are probably one of the most susceptible to diseases and insect attacks. In most production areas, diseases and pests constitute the major factor that significantly lowers onfarm yields. More than 200 diseases and 200-450 insects can affect bean productivity (CIAT, 1981b).

Bean production in Latin America suffers from many edaphic, climatic, and biotic stresses. However, the main factors responsible for low yields are high disease-and-insect pressure, drought, low plant density (to avoid high disease pressure) and farmer's economic inability or reluctance to use inputs.

Web blight is a disease, the importance of which has been underestimated. Previous reports (Costa, 1972; Crispín-Medina and Gallegos, 1963; Echandi, 1966 and 1976) mention it only as a devastating disease in the warm, humid areas of Mexico and Central America and lowlands of Colombia. However, recent reports have confirmed that this disease is widespread in many bean-producing regions of Latin America (Gálvez et al., 1980).

In some years and locations, bean golden mosaic virus (BGMV) is also severe. This virus has become a serious problem in many regions of southern and central Brazil (Minas Gerais, Goiás, north Paraná) (Costa, 1972; Costa and Cupertino, 1976); Central America (Gálvez, 1982; Gámez, 1971), the Caribbean, and the lowlands and eastern coast of Mexico (CIAT, 1981b). Recently, BGMV has also been observed attacking beans in Argentina.

In cooler regions, anthracnose is important, as are other fungal diseases, root rots, and halo blight (Cardona-Alvarez and Skiles, 1954; Echandi, 1966; Shands et al., 1964). Each of these diseases can cause yield losses as high as 80%-100%. Losses to bean common mosaic virus (BCMV) can range from 53%-96% (Crispín-Medina

and Campos-Avila, 1976; Echandi, 1966; Laborde-C., 1967); to bean rust from 18%-85% (Carrijo, 1975; CIAT, 1976); and to anthracnose as high as 95% (CIAT, 1976). Seed transmission of pathogens responsible for BCMV, anthracnose, angular leaf spot, halo blight, and common bacterial blight complicate the disease picture. Table 3 shows the major disease problems in different bean-producing regions in Latin America.

The most important insect pests in Latin America are the leafhoppers (*Empoasca* spp.) (van Schoonhoven and Cardona, 1980). Cutworms are also important in most Latin American bean-production zones (Bonnefil, 1965; Gutiérrez-P. et al., 1975). The pod weevil (*Apion godmani* Wagner), is a major pest in Mexico, Guatemala, El Salvador, and northern Nicaragua. The Mexican bean beetle (*Epilachna varivestis* Mulsant) is an important pest in Mexico, Guatemala, and El Salvador. Slugs (*Vaginulus plebeius* (Fisher) and *Limax maximus* L.) are particularly important in Central America (Bonnefil, 1965; Enkerlin-S., 1957; van Schoonhoven and Cardona, 1980). Leafhoppers have reduced yields of susceptible cultivars by as much as 90%; and reductions of 20%-50% are common on many farms even when insecticides are used (CIAT, 1985). Storage insects such as *Acanthoscelides obtectus* (Say) and *Zabrotes subfasciatus* (Boheman) inflict heavy losses on stored beans, forcing rapid sale of grain. This contributes to postharvest price declines and marked seasonal price fluctuations (van Schoonhoven, 1976). At least 28 other insects are reported to occur on stored beans but are of minor importance or migrate from nearby stored produce to beans (van Schoonhoven and Cardona, 1980).

Soil-related constraints become important as bean production is increasingly concentrated on more marginal land, with low pH and high phosphorus fixation. Associated aluminum toxicity reduces root development and increases sensitivity to water deficits (CIAT, 1985). Nitrogen deficiency is also a limiting factor in many soils where beans are grown. This is complicated by a low capacity for nitrogen fixation in most currently used cultivars (Graham and Halliday, 1977). Analysis of 110 Central American soils showed that 20% had a pH of less than 6.0 (Müller et al., 1968), 66% were highly deficient in phosphorus (FAO, 1982), and 75% were nitrogen deficient (Díaz-Romeu et al., 1970). A similar situation was demonstrated in Brazil (Malavolta, 1972) when 232 bean fertiliza-

Table 3. Major disease problems in different bean-producing regions of Latin America.

| Country | Diseases ^a | | | | | | | | | | | |
|---|-----------------------|----|-----|-----|-----|----|-------------------|----|-----|---------|------|-------|
| | Fungi | | | | | | Bacterial blights | | | Viruses | | |
| | Rust | WB | ANT | ALS | ASC | RR | CBB | HB | BCM | BGMV | BYMV | BCIMV |
| Argentina | | | | | | | | | | | | |
| Warm zone (Salta, Tucumán, Stgo. del Estero) | x | x | x | x | | x | | x | | | | x |
| Temperate zone: Humid (Rosario de la F., Metán) | | | x | x | | | | x | | | | |
| Temperate zone: Dry (Trancas) | | | | x | | | | x | | | | |
| Temperate zone: (Sta. Isabel in Salta, Candelaria) | | | | x | | | | | | | | |
| Belize | | x | | | | | | | | | | |
| Bolivia | | | | | | | | | | | | |
| Santa Cruz | x | | | | | | | | x | | | |
| Brazil | | | | | | | | | | | | |
| Parts of Amazonas, Pará, Acre, and Rondônia | | x | | | | | | | | | | |
| Pernambuco (mata), Bahia, Sergipe, Alagoas | x | | x | x | | x | | x | | | | |
| Parts of Minas Gerais, Espírito Santo, Rio de Janeiro | x | | x | x | | | | x | | | x | |
| Parts of Minas Gerais, Goiás | x | | x | x | | | | x | | | x | |

(Continued)

Table 3. (Continued).

| Country | Diseases ^a | | | | | | | | | | | |
|--|-----------------------|----|-----|-----|-----|----|-------------------|----|-----|---------|------|-------|
| | Fungi | | | | | | Bacterial blights | | | Viruses | | |
| | Rust | WB | ANT | ALS | ASC | RR | CBB | HB | BCM | BGMV | BYMV | BCIMV |
| São Paulo, Mato Grosso, parts of Paraná | x | | x | x | | | x | | x | | x | |
| Rio Grande do Sul, Santa Catarina, parts of Paraná | x | | x | | | | x | | x | | x | |
| Colombia | | | | | | | | | | | | |
| Warm (800-1300 m.a.s.l.) | x | x | | | | | x | | x | | | |
| Medium (1300-1500 m.a.s.l.) | | | x | x | | | x | | | | | |
| Moderately cool (1700-2400 m.a.s.l.) | | | x | x | x | x | | | x | | | |
| Costa Rica | | | | | | | | | | | | |
| Brunca Region (Perez Zeledon) | | x | x | | | | | | | | | |
| Central Region (Valle Central) | x | | | x | | | | | x | | | |
| Cuba | x | | | | | | x | | x | | | |
| Chile | | | | | | | | | x | | x | |
| Dominican Republic | x | x | x | | | | | | x | | x | |
| Ecuador | | | | | | | | | | | | |
| Coast | x | | | | | | | | | | | |
| Highlands | | | x | x | x | | | | | | | |

(Continued)

Table 3. (Continued).

| Country | Diseases ^a | | | | | | | | | | | |
|--|-----------------------|----|-----|-----|-----|----|-------------------|----|-----|---------|------|-------|
| | Fungi | | | | | | Bacterial blights | | | Viruses | | |
| | Rust | WB | ANT | ALS | ASC | RR | CBB | HB | BCM | VM | BYMV | BCIMV |
| El Salvador | | | | | | | | | | | | |
| Balstein (Sta. Ana, Aluachapán, Sonsonate) | | | | | | | x | | | | | |
| Central (La Libertad, San Salvador, Cuscatlán) | | | | | | | x | | x | | | |
| Guatemala | | | | | | | | | | | | |
| Oriente (Jutiapa) | x | | | | | | | | x | | | |
| Altiplano (Chimaltenango) | x | | x | | | | | | | | | |
| Central coastal region (Escuintla) | | x | | | | x | | | | | | |
| North (Petén) | | x | | | | | | | | | | |
| Jamaica | x | | x | | | | x | | x | x | | |
| Mexico | | | | | | | | | | | | |
| Warm, with dry winter (Sinaloa) | x | | | | | x | | | | x | | x |
| Warm, humid (Veracruz) | x | x | | | | | | x | | x | x | |
| Temperate, humid (Jalisco) | x | | x | x | | x | x | | | | | |
| Temperate, semiarid (Durango) | x | | x | | | x | | | | | | |
| Warm, arid (Chihuahua) | x | | | | | x | | | x | | | |

(Continued)

Table 3. (Continued).

| Country | Diseases ^a | | | | | | | | | | | |
|--|-----------------------|----|-----|-----|-----|----|-------------------|----|-----|------|------|-------|
| | Fungi | | | | | | Bacterial blights | | | | | |
| | Rust | WB | ANT | ALS | ASC | RR | CBB | HB | BCM | BGMV | BYMV | BCIMV |
| Nicaragua | | | | | | | | | | | | |
| Region 1 | x | x | | | | | x | | | x | | |
| Region 4 | | x | | x | | | x | | | x | | |
| Region 5 | | x | | x | | | x | | | x | | |
| Region 6 | x | x | x | x | | | x | | | x | | |
| Panama | x | x | | | | | x | | | x | | |
| Paraguay | x | | | | | | x | | | x | | |
| Peru | | | | | | | | | | | | |
| Coastal region (Lambayeque, Chincha, Camaná) | x | | | | | x | | | | | x | |
| Highlands (Cajamarca, Cusco) | | | x | x | x | | | | | x | | |
| Jungle (Pucallpa, Tarapoto) | | x | | | | | | | | | x | |

a. WB = Web blight; ANT = anthracnose; ALS = angular leaf spot; ASC = Ascochyta blight; RR = root rot; CBB = common bacterial blight; HB = halo blight; BCMV = bean common mosaic virus; BGMV = bean golden mosaic virus; BYMV = bean yellow mosaic virus; BCIMV = bean chlorotic mottle virus.

SOURCE: CIAT Bean Team trip reports, unpublished data.

tion trials, covering eight states, reported responses to nitrogen (67 times), phosphorus (103 times), potassium (15 times), lime (31 times), and microelement combinations (17 times). Aluminum (Buol et al., 1975) and manganese toxicities, associated with the low soil pH (Döbereiner, 1966) and molybdenum deficiency (Franco, 1977), complicated fertilizer recommendations.

Drought is a serious threat to bean production in many areas of Latin America, rivaled in importance by soil fertility problems (White and Singh, n.d. In semiarid regions, large areas of beans are grown, exclusively dependent on irregular rains. North central Mexico, including the States of Chihuahua, Durango, Zacatecas, and Aguascalientes, and northeast Brazil, including the States of Pernambuco, Alagoas, Paraíba, Ceará, Rio Grande do Norte, and part of Bahia, represent almost 2 million hectares of beans and are the best examples of semiarid regions threatened yearly with severe droughts. Deserts may not constitute an important drought area in quantitative terms, but often support large areas of bean production. For example, the rainless coast of Peru where irrigation costs often limit farmers to a single irrigation, supports 50% of the country's bean production. Drought stress is even enhanced when farmers plant late in the rainy season to avoid disease pressure.

Besides these extreme examples of bean production in drought situations, most bean-producing regions experience periods of dryness with varying differences in frequency and severity of stress. Throughout the tropics, areas with apparently adequate mean precipitation frequently suffer from water deficits because of seasonal fluctuations in rainfall. Consequently, bean production is impaired. According to data so far obtained by the CIAT Agroecological Studies Unit (ASU) (CIAT, 1985), 73% of the total Latin American bean production occurs in microregions that have moderate to severe mean water deficits at some time during the cropping season. Little of this production is irrigated (Table 4).

Although serious water deficits are a major production constraint, high temperature is not. According to data from ASU, most beans (76%) in Latin America are produced at temperatures close to the optimum (20-23 °C) for *Phaseolus* species.

Table 4. Climatic classification of bean-production zones in Latin America.

| Climatic type | General description of climatic type | Growing season mean temperature (°C) | Growing season daily water balance (WB) ^a (+ mm/day) | Latin American production zone | |
|---------------|---|--------------------------------------|---|--------------------------------|-----------|
| | | | | (t in thousands) | (total %) |
| A | Average temperatures and adequate mean seasonal WB | 22 | -1.5 to 0.4 | 661 | 17 |
| B | Average temperatures and slight excess in WB | 23 | 0.4 to 4.0 | 118 | 3 |
| C | Average temperatures and large deficits in WB (irrigated areas) | 23 | -5.6 to -5.1 | 528 | 14 |
| D | Average to moderately low temperatures with possible deficit in WB toward end of the growing season | 20 | -2.7 to -1.6 | 1672 | 42 |
| E | High temperatures with possible deficit in WB toward end of growing season | 26 | -4.1 to -0.3 | 262 | 6 |
| F | Moderately low temperatures and moderate water stress | 16 | -2.3 to -1.9 | 451 | 11 |
| G | Low temperatures and adequate mean seasonal WB | 13 | -0.09 to -0.05 | 45 | 1 |

a. Mean of conditions in the microregions constituting each production zone. Overall, 110 microregions have been defined.

SOURCE: CIAT, 1981a.

Low and unstable bean yields are, in some cases, caused by the use of cultivars whose physiological characteristics are not suitable for the production environments in which they grow. Cultivars with a determinate, erect, bush growth habit can be planted in areas well suited to intensive cultivation with a degree of mechanization. These types are characterized by early and intense flowering, which contributes to low and unstable yields, and by a reduced ability to compensate for low planting densities, which is common on most small farms. These cultivars do not have a mechanism for renewed flowering when stress is relieved (CIAT, 1985). They are grown extensively because farmers like their erectness, earliness, and large seed size. In contrast to mechanized production systems, most common bean producers in Latin America cultivate indeterminate types in complex multiple cropping systems (Andrews and Kassam, 1976). Many of these have prostrate plant types and, in monoculture, pods come in contact with soil at maturity. Some cultivars are too late, or are poorly adapted to row and relay intercropping with maize. Type II cultivars are the least competitive, whereas types IIIb, IVa, and IVb are progressively more competitive (Laing et al., 1984). Type IV is most favorably grown with maize (Adams et al., 1985).

Growth habit instability has been related to a phytochrome response to differences in spectral quality (Kretchmer et al., 1977 and 1979) and photoperiod (Kretchmer et al., 1977). Common beans are grown in the tropics under daylengths that vary from 11-15 hours (Masaya and White, 1986). In subtropical areas, as days become shorter, beans are often planted in relay cropping, using stalks of the preceding maize crop as physical support for the long and flexible bean stems. Photoperiod-insensitive types originate mainly from extreme latitudes and occur primarily in growth habits I and II, while large-seeded climbing types, mainly from the Andean zone, are rarely insensitive (CIAT, 1976 and 1977).

Equally important as the biotic and abiotic environmental stresses that affect crop production are socioeconomic constraints. A high proportion of Latin American bean production occurs on small farms and in associated cropping systems. This, in itself, imposes constraints to increased bean production. Although associated cropping usually is more efficient in the total exploitation

of environmental resources than beans grown by themselves, bean yields are reduced 30%-50% (Francis et al., 1978). The task of extending new technologies is likely to be more costly among many small farmers than among few large farmers. Development of an integrated system for the supply of agricultural inputs and marketing of the harvested products are therefore impeded. Furthermore, the costs of individual technical assistance will be prohibitively high. Statistics show that a substantial proportion of bean output is consumed by the producer. As much as 30% of Latin American bean production is estimated as subsistence (Pachico, 1982). When a crop is produced primarily for subsistence, cash is not generated from the production process, thereby making it less likely for growers to use bought inputs in production.

Conclusions

In Latin America, bean yields are low and the bean production environment complex. Efforts to increase bean yields must therefore be done at a regional level and aim to improve local production systems, understand local grain-type requirements, and research local production problems. Beans, being often a subsistence or small-farmer crop, do not receive the research attention that cash crops such as coffee or cotton, enjoy. Collaboration among bean research institutes among countries of an ecologically uniform region must therefore be encouraged.

Although the average bean yield is low, because of competition from associated crops, attacking the beans' disease susceptibility may be the most profitable venue for researchers aiming to increase yields. Because beans are disease susceptible, farmers consider them as a high-risk crop that does not merit good agronomy. With a multiple-pest-resistant variety farmers may find crop risk reduced and so respond with improved agronomy and thus obtaining higher yields. This concept has borne out in Costa Rica and Argentina where improved varieties have prompted farmers to improve their production agronomy.

Bean research is a challenge to scientists trying to improve the crop. The variability of cropping systems and of grain-type requirements, the difficulty to improving the potential yield of any

legume crop, and the need to improve the beans' digestibility are all challenges which need to be met, if the lives of millions of small farmers are to improve. This has to be achieved even though beans receive low priority in local government agricultural research financing.

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Chapter 4

ANGULAR LEAF SPOT

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Introduction

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

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

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

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

Taxonomy

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

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

Etiology

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

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

Epidemiology

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

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

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

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

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

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

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

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

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

Symptomatology

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

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

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

Control by Cultural Practices

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

Control by Chemicals

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

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

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

Control by Plant Resistance

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

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

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

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

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

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

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

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

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

Preliminary studies were conducted at CIAT (unpublished data) on a series of 21 bean cultivars to examine the pathogenicity, virulence, and aggressiveness of 17 *P. griseola* isolates from Argentina, Brazil, Colombia, Costa Rica, Guatemala, Mexico, and Nicaragua. Differences in pathogenicity were observed among all the isolates, and within isolates from the same country. Quantitative differences (in percentages) between the cultivars were observed for disease, number of lesions, lesion size, number of spores/mm², and the number of days required to induce the same level of disease. Differences in the date of disease initiation, lesion size, disease progress, and severity were also observed between cultivars under field conditions. Many lines with broad resistance in several

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

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

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Chapter 5

ANTHRACNOSE

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Introduction

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

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

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

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

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

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

Etiology

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

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

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

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

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

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

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

Epidemiology and Plant Infection

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

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

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

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

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

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

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

Symptomatology

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

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

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

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

Control by Cultural Practices

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

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

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

Control by Chemicals

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

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

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

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

Control by Plant Resistance

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

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

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

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

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

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

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

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

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

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

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

Physiology of the Host-Parasite Interaction

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

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

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

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

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

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

Inheritance and Sources of Resistance

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

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

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

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

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

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

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

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

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

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

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

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Chapter 6

ROOT ROTS

George S. Abawi*

Introduction

There are many root diseases of beans and several occur throughout many bean-growing areas of the world (Abawi et al., 1985; Sherf and MacNab, 1986; Walker, 1952; Zaumeyer and Thomas, 1957). Continuous bean production, improper crop rotation, and increased soil compaction are some of the factors that contribute to the prevalence and severity of root diseases. Root rots have caused considerable damage to beans in northeast Brazil, the highlands of Mexico, Nicaragua, coastal Peru, United States, and many other countries. Detailed information on bean yield losses from root diseases in Latin America and other bean-growing regions is limited. However, yield losses can be considerable and often vary among fields of the same area, as well as in the same field from season to season. This variability is affected by prevailing environmental and soil conditions at planting time, midseason stresses, and the type and number of root pathogens present and active during disease initiation and development. Root diseases also indirectly affect beans by reducing their efficiency in using soil nutrients. They make roots susceptible to an increased range of stresses such as temperature variation, drought, and many biological stresses.

Bean-root diseases can be incited by species of several plant pathogenic fungi. The major ones are species of *Fusarium*, *Rhizoctonia*, *Pythium*, *Thielaviopsis*, *Sclerotium*, *Aphanomyces*, *Phymatotrichum*, and *Macrophomina*. These pathogens may each infect beans, causing a characteristic disease, or may, if occurring together, infect in any possible combination, resulting in disease

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complexes. The major root pathogens that predominate and become a limiting production factor differ from one bean-growing region to another (Abawi and Pastor-Corrales, 1986).

For example, Fusarium wilt caused by *Fusarium oxysporum* f. sp. *phaseoli* Kendrick *et* Snyder is the major disease in northeast Brazil, whereas *Rhizoctonia solani* Kühn and *Fusarium solani* f. sp. *phaseoli* (Burkholder) Snyder *et* Hansen are the major pathogens in the coastal areas of Peru. In Colombia, *Macrophomina phaseolina* (Tassi) Goid. is the most important in the Quilichao area, whereas *Rhizoctonia solani* is prevalent in the Popayán area, and *Fusarium oxysporum* f. sp. *phaseoli*, and, to a lesser extent, *Sclerotium rolfsii* Saccardo, dominate in the Pasto-Ipiales area. In contrast, *Pythium ultimum* Trow, *Thielaviopsis basicola* (Berkely *et* Broome) Ferraris, *Rhizoctonia solani*, and *Fusarium solani* f. sp. *phaseoli* are all important and often occur as disease complexes in New York State, USA (Abawi *et al.*, 1985). It is therefore important to determine the etiology of bean root-diseases where cultivar development is in progress or root-disease management strategies are warranted.

Aboveground symptoms in a field with severe incidence of root diseases include poor seedling establishment, uneven growth, chlorosis, and premature defoliation of severely infected plants (Figure 14). Poor seedling establishment and reduced plant density are the result of seed rot and damping-off. The latter occurs when germinating seeds and young seedlings are attacked during the first two to three weeks after planting. Root-rot infection of older plants usually results in reduced vigor, discoloration, and slow rotting of stem and root tissues. Roots of severely infected plants are reduced in size and may exhibit different degrees of decay. Tap roots of severely infected plants often die, although coarse adventitious roots may develop from the hypocotyl areas above infected tissues. These roots also become infected later, but their production continues during moist soil conditions and helps the plant survive. The shape and color of lesions on stem and root tissues are specific and characteristic for each attacking pathogen. To properly examine bean roots, plants must be dug up carefully and the soil removed without disturbing the fibrous root system.

The use of highly resistant bean cultivars is the most effective control strategy for root diseases. It is especially appropriate for

farmers in developing countries with low inputs. However, until an adapted cultivar resistant to all pathogenic organisms in the region becomes available, a combination of compatible and effective measures for controlling root diseases must be used (Burke and Miller, 1983; Papavizas and Lewis, 1979; Sumner et al., 1986a and 1986b). A cultivar that is susceptible to a component of the root disease complex may be managed with an economical control measure that is chemical (seed or soil treatment), cultural (crop rotation, organic mulch, adjusting planting time, fertilizer or herbicide use, land preparation), biological (addition or enhancement of beneficial soil-borne organisms), or a combination of these measures.

A soil-indexing procedure is available that can effectively determine the root-rot potential of bean fields (Kobriger and Hagedorn, 1983). Used as part of an integrated program, such a procedure can aid growers in avoiding problem fields where possible and thus avert a loss. A similar test differentiated relatively clean fields from those with severe root-rot problems in New York State (Abawi et al., 1985; G. S. Abawi, unpublished data). The test involved growing beans for five weeks in representative soil samples from the fields in question under greenhouse conditions that were favorable for root-rot development. Root-rot potential was determined from the root-rot ratings obtained and the percentage of reduction in plant weight as compared with those of plants grown in pasteurized samples of the same soil.

Rhizoctonia Root Rot

Introduction

Rhizoctonia root rot, caused by *Rhizoctonia solani* Kühn (teleomorph is *Thanatephorus cucumeris* (Frank) Donk), is a common root-rot disease of beans in Latin America and the world (Parmeter, 1970; van Bruggen et al., 1986; Zaumeyer and Thomas, 1957). The fungus is distributed throughout most agricultural soils at various levels of infestation and can infect many plant species. Losses of more than 10% have occurred in the United States. The author has observed nearly 100% infection and almost complete

losses in bean plantings near Popayán, Colombia, the coastal areas of Peru, and central and western areas of New York State. It should be noted that *Rhizoctonia solani*, and its telemorph, is the pathogen of web blight, a foliar disease (Chapter 8).

Common names frequently used for rhizoctonia root rot in Latin America include “pudrición radical por Rhizoctonia,” “chancro,” “tizón,” “pudrición del tallo,” “tombamento,” “podredumbre del tallo,” and “podridão radicular.”

Etiology

In nature, *Rhizoctonia solani* and its telemorph (Tu and Kimbrough, 1978) exist as many strains, differing in cultural appearance, physiology, and pathogenicity (Parmeter, 1970). The naturally occurring strains or isolates differ in mycelium color, zonation, type and number of sclerotia, size of aerial mycelium, growth rate, saprophytic behavior, and enzyme production (Galindo et al., 1982; Papavizas, 1964 and 1965; Papavizas and Ayers, 1965; Parmeter, 1970). However, all isolates have the mycelial characteristics of *R. solani* (Parmeter, 1970), consisting of a constriction at the base of hyphal branches, formation of a prominent dolipore septum at the branch near the point of origin, multinucleate condition of young hyphal tip cells, and typically brown mycelium.

Anastomosis among *R. solani* isolates demonstrates relationships among fungal isolates. Most *R. solani* isolates fall within one of four main anastomosis groups (AG) that are different morphologically, physiologically, and pathogenically (Adams and Butler, 1979; Ogoshi and Ui, 1979; Sherwood, 1969). However, several other AGs have been reported recently (Ogoshi and Ui, 1979). Most *R. solani* isolates associated with bean root rot belong to AG4, but isolates of AG2 and a few of AG1 have also been found to be pathogenic to beans (Galindo et al., 1982). Generally, good correlation has been found between the growth rate of isolates and their pathogenicity to beans.

The telemorph, *Thanatephorus cucumeris*, may occur and form a hymenial layer at the base of plants and/or on the underside of soil

aggregates during periods of high humidity and rainfall (Parmeter and Whitney, 1970). Basidia are short and barrel shaped with stout straight sterigmata while basidiospores are smooth, thin walled, and hyaline (Tu and Kimbrough, 1978). Some *R. solani* isolates may be induced to produce the basidial stage in vitro (Adams and Butler, 1983). *Rhizoctonia solani* uses carbon and mineral sources with a high efficiency (Parmeter, 1970; Sherwood, 1969). *Rhizoctonia solani* isolates are usually auxotrophic. However, no specific carbon source consistently supports the growth of all isolates and some require specific growth factors. The optimal temperature for growth is 23-28 °C, although lower and higher optima have been reported for various isolates. Specific isolates may also respond differently to varying pH levels, but most isolates attain optimal growth at pH 5-7 (Sherwood, 1970).

Epidemiology

Rhizoctonia solani contains a wide array of pathogenic isolates (Talbot, 1970). Some isolates are specific for one crop such as beans, while others attack a wide range of hosts (Garza-Chapa and Anderson, 1966; Papavizas and Ayers, 1965; Papavizas et al., 1975; Sherwood, 1969). Isolates vary in the degree of virulence expressed toward a single host (Bolkan and Butler, 1974; Díaz-Polanco, 1968). Disease severity is influenced by soil moisture, soil temperature, nutritional status of the inoculum (Shephard and Wood, 1963; Weinhold et al., 1969), and the plant and root exudates which stimulate mycelial growth (Dodman and Flentje, 1970; van Gundy et al., 1977). Pathogenic variants may arise during basidiospore production or more commonly by hyphal anastomosis between different field isolates (Bolkan and Butler, 1974). Activities of *R. solani* are most abundant in the top 10 cm of soil. Population densities are highest shortly after harvest and before incorporation of bean residue into the soil (Papavizas et al., 1975). However, the fungus is unevenly distributed in soil, hence the clumped distribution of lesions on hypocotyl tissue and clustered pattern of infected plants in a field (Campbell and Pennypacker, 1980).

Inoculum sources of *R. solani* consist of sclerotia, hyphae, and basidiospores. However, the importance of basidiospores as an

inoculum source for bean root-rot is unknown. Inocula may survive in soil as sclerotia or thick-walled hyphae associated with plant debris, and/or as saprophytic growth on organic matter (Parmeter, 1970). The fungus can penetrate the intact cuticle and epidermis by infection pegs produced from infection cushions (Christou, 1962a), or by individual hyphae (Dodman and Flentje, 1970), and through natural openings and wounds. Penetration is believed to occur by mechanical pressure and enzymatic degradation of host cells (Bateman, 1970). The optimal soil temperature for development of hypocotyl cankers is 18 °C. Relatively few cankers develop at temperatures above 21 °C. The disease is more severe during the first two to three weeks and particularly under wet conditions and somewhat cool weather. As plants age they become less susceptible to severe damage by *R. solani*. Apparently, at high temperatures plants emerge more rapidly and thus escape infection (Bolkan et al., 1974; Leach, 1947; Zaumeyer and Thomas, 1957). The field population density of *R. solani* is dependent upon the presence of a susceptible crop. The pathogen can be disseminated into new areas by irrigation water, transplanted material, aerially disseminated sclerotia or basidiospores, and infected or contaminated seeds. The fungus may be internally and externally seed-borne (Bolkan et al., 1976; Díaz-Polanco, 1968; Ellis et al., 1975; Kramer et al., 1975). *Rhizoctonia solani* can survive in association with dry soil aggregates and thus be disseminated by wind-blown particles.

Symptomatology

Rhizoctonia solani may induce seed rot, damping-off, stem canker, root rot, and pod rot. *Rhizoctonia* can infect seeds before germination, resulting in seed decay. Lesions on a young seedling expand rapidly and result in damping-off. Seed and seedling infections reduce seedling establishment and therefore lower plant densities often severely enough to be visually observed. The characteristic symptoms on infected plants are reddish brown, sunken lesions on the stem and taproot (Figure 15). As infection progresses, sunken cankers enlarge (Figure 16) and those that are close together may coalesce and girdle the stem (Figure 17), retard growth, and eventually kill the plant.

Rhizoctonia solani can also infect pods in contact with the soil surface, causing water-soaking, the characteristic reddish brown sunken lesions, and distinct margins around the lesions. Minute brown sclerotia may develop on the surface of, or be embedded in, these cankers. These lesions may serve as an inoculum source for infection of beans in transit and ensure fungus dissemination as well as causing seed discoloration. The fungus can be seed transmitted in beans. Infection of bean with *R. solani* may interact with other root-rot fungi (Pieczarka and Abawi, 1978a) and plant parasitic nematodes (Reddy et al., 1979).

Control by cultural practices

Because *R. solani* has a worldwide distribution (Leach and Garber, 1970), including in uncultivated soils (Baker and Martinson, 1970), exclusion and eradication usually are not effective field control measures. Nevertheless, the local pathogenic potential is increased by introducing infested soil and infected or contaminated plants and seeds from other regions. *Rhizoctonia solani* can be eradicated from infected greenhouse soil by steaming at 60 °C for 30 minutes (Leach and Garber, 1970).

Rhizoctonia solani infection may be reduced by various cultural practices. In Popayán, Colombia, *R. solani* is less severe during the wet rainy season if beans are planted on raised beds that facilitate good drainage. Seedling injury is minimized by shallow planting so that less seedling tissue is exposed to inoculum. However, increased plant lodging may occur. Manning et al. (1967) reported that seeds planted 7.5 cm deep developed more root rot and hypocotyl injury than seeds planted only 2.5 cm deep. In the San Joaquin Valley of California, shallow plantings (1.5-2.5 cm deep) apparently reduced disease severity to a level where there was no need for fungicidal application (Leach and Garber, 1970). In addition, planting should be delayed until the soil has warmed sufficiently to reduce *R. solani* infections (Bolkan et al., 1974; Zaumeyer and Thomas, 1957).

Continuous planting of beans in the same field increases the inoculum density of *R. solani*. However, crop rotation with nonhost crops reduces the incidence of bean root rot even though it does not completely eradicate the pathogen (Burke and Kraft, 1974). *Rhizoc-*

tonia solani populations rapidly decline in soil planted with wheat, oats, barley, or maize. Population levels remain relatively high in soil planted with susceptible bean, pea, or potato plants.

A suggested, but yet unproven, alternative to crop rotation is soil amendment with decomposable material (Leach and Garber, 1970) or the incorporation of selected residue (Manning and Crossan, 1969; Papavizas et al., 1975). Snyder et al. (1959) demonstrated that bean infection was significantly reduced in greenhouse studies by adding a barley, wheat, or maize amendment. Similarly, Manning and Crossan (1969) showed that a maize amendment significantly reduced hypocotyl rot under greenhouse and field conditions, the inhibitory effect lasting nearly a year. Also, many antagonists or mycoparasites such as *Trichoderma* species, have effectively reduced activities of *R. solani* when incorporated with organic amendments (as carriers) or directly on seed (Bell and Sumner, 1984; Chet and Baker, 1981; Chet et al., 1981; Marshall, 1982; Tu and Vaartaja, 1981).

Another cultural practice that is effective in reducing surface inoculum of *R. solani* and thus disease incidence, is deep plowing (Papavizas and Lewis, 1979). Turning under soil and crop residue to a depth of 20-25 cm has reduced *Rhizoctonia* root rot on beans for three years.

Control by chemicals

Fungicides that are effective against *R. solani* include PCNB, benomyl, carboxin, Busan 30A, thiram, zineb, chloroneb, and others. These fungicides are commonly applied as seed treatments (1-3 g a.i./kg seed) before or during planting (Bolkan et al., 1976; Ellis et al., 1975; Peterson and Edgington, 1970). The most commonly used fungicide to control *R. solani* is PCNB. Bristow et al. (1973) and Crossan et al. (1963) report that PCNB, applied as an in-furrow low-volume spray (5.8 kg in 378 L of water/ha), provides excellent control of *R. solani*. Chloroneb and PCNB are highly specific toward *R. solani* and should be mixed with metalaxyl or pyroxychlor where *Pythium* spp. also are a problem (Leach and Garber, 1970; Lewis et al., 1983; Locke et al., 1983). In New York State, combinations of fungicides that included captan, metalaxyl,

and chloroneb were most effective when applied as slurry seed treatments (Abawi et al., 1985). Fungicide seed treatments for the control of *R. solani* often are effective for enhancing seedling emergence (van Bruggen et al., 1986) and establishment. However, they seldom provide protection to the expanding root zone of older plants and are therefore ineffective for controlling the root-rot phase of the pathogen.

Herbicides have been reported to both increase and decrease root-rot severity (Campbell and Altman, 1977; Grinstein et al., 1976; Hagedorn and Binning, 1982; Johal and Rahe, 1984). Hagedorn and Binning (1982) showed that root-and-hypocotyl rot of bean was suppressed significantly by preplant incorporation into the soil of dinoseb at 6.7 kg a.i./ha. Campbell and Altman (1977) reported that the herbicide cycloate reduced the colonization of bean segments by *R. solani*, probably by inhibiting fungal growth. In contrast, Grinstein et al. (1976) reported that dinitramine herbicide reduced plant resistance to infection by *R. solani*. Similarly, the number and size of hypocotyl lesions caused by *R. solani* were increased by preplant application of trifluralin (Wrona et al., 1981).

Control by plant resistance

Older plants often become more resistant to *R. solani* infection, possibly because of increased calcium content in the plant tissue (Bateman and Lumsden, 1965), induction of phytoalexins (Pierre and Bateman, 1967; Smith et al., 1975; VanEtten and Bateman, 1970), and/or decline in hypocotyl and root exudates which stimulate infection-cushion formation by the fungus (de Silva and Wood, 1964; Stockwell and Hanchey, 1983). 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 this resistance was inherited as a single dominant factor (Zaumeyer and Thomas, 1957). The dry bean cultivar Uribe Redondo was reported by Cardona-Alvarez (1954) to be highly resistant to rhizoctonia root rot in Colombia. Prasad and Weigle (1969 and 1970) reported that Venezuela 54 and P.I. 165426 are highly resistant to *R. solani* infection and suggested that resistance may be linked to dark seed-coat color.

Extracts from black-seeded lines contained phenolic substances inhibitory to the growth of *R. solani* (Prasad and Weigle, 1976). Several investigators (Beebe et al., 1981; Dickson and Boettger, 1977; Silva and Hartmann, 1982) have previously observed a close relation between black-seeded materials with resistance to *R. solani*. However, white-seeded cultivars with resistance to this fungus have also been identified recently. Two dry bean breeding lines, B 3088 and B 3787, and a wax bean cultivar were highly resistant to rhizoctonia root rot (Zaumeyer and Meiners, 1975). In addition, the CIAT bean accessions A 300, BAT 1753, EMP 81, RIZ 21, and RIZ 30 were highly tolerant to *R. solani* in Colombia (Pastor-Corrales and Abawi, 1986). Sumner (1985) demonstrated the differential responses of bean cultivars and accessions to the different anastomosis groups of *R. solani* and suggested it is important to adequately characterize the local fungus isolates in order to develop resistant bean cultivars.

Fusarium Root Rot

Introduction

Fusarium root rot of beans is caused by *Fusarium solani* (Martius) Appel and W. f. sp. *phaseoli* (Burk.) Snyder and Hans. It was first reported in 1916 by Burkholder in New York State (Kraft et al., 1981; Zaumeyer and Thomas, 1957). The pathogen is prevalent and causes varying degrees of damage in most bean-growing areas of the world.

In United States, fusarium root rot has caused serious losses in the states of New York, Idaho, Colorado, Washington, and Nebraska (Burke and Miller, 1983; Burke and Nelson, 1967; Keenan et al., 1974; Sherf and MacNab, 1986; Steadman et al., 1975). It has been reported also in Spain, Bulgaria, England, and other areas in Europe. In Latin America, fusarium root rot has been identified in Brazil (Costa, 1972; Vieira, 1967), Colombia (Barros-N., 1966), Peru (Dongo-D. and Osorio-D., 1961), Venezuela (Casanova and Díaz-Polanco, 1966), Costa Rica (Echandi, 1966), and Mexico (Crispín-Medina et al., 1964). Keenan et al. (1974) reported that an unusually high yield loss of 86% occurred in

Colorado because of a drastic decrease in the number of pods per plant.

Burke and Nelson (1967) found that yield losses under severe disease pressure ranged from 6%-53%, depending upon the bean cultivar and other stress factors. Pieczarka and Abawi (1978a) demonstrated that a synergistic interaction exists between *F. solani* f. sp. *phaseoli* and *Pythium ultimum*, resulting in higher disease severity ratings and increased damage to bean.

In addition to the common bean (*Phaseolus vulgaris* L.), the fusarium root-rot pathogen attacks lima bean (*P. lunatus* L.), scarlet runner bean (*P. coccineus* L.), *Vigna angularis* (Willd.) Ohwi et Ohasi, and *V. aconitifolia* (Jacq.) Maréchal. *Fusarium solani* f. sp. *phaseoli* has also been reported to be pathogenic on pea (*Pisum sativum* L.), cowpea (*Vigna unguiculata* (L.) Walpers subsp. *unguiculata*), *Onobrychis viciifolia* Scop., and *Pueraria lobata* (Willd.) Ohwi (Auld et al., 1976; Zaumeyer and Thomas, 1957).

Common names frequently used for fusarium root rot in Latin America are "pudrición radical por Fusarium," "pudrición seca," and "podridão radicular seca."

Etiology

Most isolates of *F. solani* f. sp. *phaseoli* produce appressed mycelial growth (pseudopionnotes) on artificial agar media (Kraft et al., 1981). Fungal colonies are usually blue to blue-green, but occasionally are white to buff in color. Three types of asexual spores are produced by all isolates: microconidia, macroconidia, and chlamydospores. Macroconidia are sickle shaped, multiseptate, and are usually produced on sporodochia. Microconidia are usually produced on simple short conidiophores. The dark and thick-walled chlamydospores are produced abundantly on or in infected host tissues and are long-term survival structures. Conidia and hyphae in soil, and even on agar media, are often converted to chlamydospores (Kraft et al., 1981; Nash et al., 1961). Chlamydospores are round to subglobular or pear shaped and 6-16 μm in diameter. They are formed terminally, on short branches, or intercalary in the hyphae. Chlamydospores are often produced

singly, but can be found in pairs or clumped together in higher numbers.

The interspecific taxon (forma specialis) *phaseoli* is distinguished from all other members of *F. solani* on the basis of its physiological and pathological adaptation to beans. Differences in pathogenicity among isolates of *F. solani* f. sp. *phaseoli* have not been clearly demonstrated. However, considerable differences among isolates of this pathogen have been documented on artificial agar media.

Epidemiology

Chlamydospores of *F. solani* f. sp. *phaseoli*, either associated with infected bean tissue or free in soil, are often under the influence of soil fungistasis. They can therefore remain dormant in soil with little mobility for a long time (Burke, 1965; Kraft et al., 1981; Nash et al., 1961). When soil fungistasis is reversed, chlamydospores germinate where bean seed and root exudates are available (Cook and Snyder, 1965; Kraft et al., 1981; Schroth and Cook, 1964). Chlamydospores of *F. solani* f. sp. *phaseoli* can be stimulated to germinate by exudations from nonhost plants or when they are close to fresh organic matter (Barros-N., 1966; Cook and Snyder, 1965; Kraft et al., 1981; Zaumeyer and Thomas, 1957). The pathogen was reported to directly penetrate bean tissue or enter through stomata and wounds. After penetration, the fungus grows intercellularly throughout cortical tissues, but is stopped by the endodermis layer (Kraft et al., 1981). Growth and sporulation (of macro- and microconidia) may be seen on stem tissues, above the soil line under moist soil conditions. Chlamydospores are also produced on and in root and hypocotyl tissues.

The pathogen is disseminated within and between bean fields by such means as movement of infected soil, infected host tissues, colonized debris, drainage and irrigation water, contaminated bean seed, (Burke, 1965; Kraft et al., 1981). Once introduced into a field, this pathogen becomes uniformly distributed at high densities after two or three bean crops (Kraft et al., 1981). The pathogen is also capable of colonizing roots of nonhost crops without causing disease symptoms, and colonizing organic matter under certain environmental conditions, therefore maintaining or increasing its

population in the absence of beans (Barros-N., 1966; Kraft et al., 1981; Schroth and Cook, 1964).

Growth and yield losses inflicted by *F. solani* f. sp. *phaseoli* to vigorously growing beans are minimal (Burke and Miller, 1983). Tests conducted in field microplots showed that as high as 4000 propagules per gram of soil did not cause yield loss to nonstressed plants even though it caused severe discoloration of cortical tissues of roots and hypocotyls (Abawi and Cobb, 1984). However, this pathogen causes severe rotting of the entire root system with high yield losses on stressed bean plants, as demonstrated by Burke and others (Burke and Miller, 1983; Kraft et al., 1981).

Stress factors that aggravate fusarium root rot and its damage to beans include soil compaction, excess soil moisture, drought, high-density plantings, herbicide damage, the ammonium form of nitrogen fertilizers, toxic metabolites of decomposing crop residue, and soil temperatures unfavorable for bean seed germination and growth (Diehl and Steadman, 1981; Dryden and Van Alfen, 1984; Kraft et al., 1981; Miller and Burke, 1985a and 1985b; Singh et al., 1981). In addition, parasitism of roots by plant parasitic nematodes such as *Meloidogyne* spp. and *Pratylenchus* spp., and other pathogenic fungi such as *Pythium ultimum* or *Rhizoctonia solani*, may also increase fusarium root-rot severity and damage (Hutton et al., 1973; Pieczarka and Abawi, 1978a). Growth of the pathogen on agar media is optimal at 29-32 °C, but disease severity and damage under field conditions is greater at 22 °C than at 32 °C.

Symptomatology

Initial symptoms of fusarium root rot appear as longitudinal, narrow, reddish lesions or streaks on the hypocotyl and primary root (Figure 18) about one to two weeks after seedling emergence. As infection progresses, lesions become numerous, coalesce, and the entire underground stem and root systems may become covered with reddish brown superficial lesions (Figure 19). The discoloration 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 are frequently killed by the fungus and may remain attached as decomposed and dried rem-

nants. When the primary root is killed, the lower stem may become pithy or hollow. There is no pronounced wilting symptom although severely infected plants are stunted, chlorotic, and exhibit premature defoliation. Lateral adventitious roots often develop above the initial lesion areas and support plant growth so that a crop yield is still produced, provided soil moisture is adequate. However, pod number per plant and seed size may be reduced. Adventitious roots may later become similarly infected and sometimes are killed by the pathogen.

Control by cultural practices

When virgin soils are to be used for bean production, all measures must be employed to prevent the introduction of the pathogen into these soils such as the exclusion of infected bean residue, infected seeds, contaminated irrigation water, or soil adhering to agricultural implements. Eradication on a large scale is uneconomical and impossible once the pathogen becomes established within the field. Well-drained and well-fertilized soils promote vigorous plant growth. Shallow cultivation prunes lateral roots, which usually form above infected hypocotyl tissues, and must be avoided in heavily infected plantings. Hilling up soil around the stem of infected plants will promote adventitious root formation and thus will reduce root-rot damage. Excessively high plant populations may increase disease incidence because of root competition and concentration of root exudates, and ought to be avoided in heavily infested fields.

Long-term crop rotation with nonsusceptible plants such as wheat and barley, lowers soil populations of *F. solani* f. sp. *phaseoli* and reduces damage to beans (Maloy and Burkholder, 1959). However, a crop rotation of two- to three-year duration is rarely effective. Soil amendment with various crop residues with high carbon to nitrogen ratios such as small grains and maize, may reduce root-rot damage. Natural biological control by resident soil microorganisms is enhanced (Adams et al., 1968; Kraft et al., 1981; Maier, 1961; Olivas-E. and Romero-C., 1972), but only if adequate nitrogen fertility is available.

Cultural practices that reduce soil compaction and loosen hard pans are most effective in reducing root-rot damage to beans (Burke and Miller, 1983). Secondary tillage that encourages soil compaction decreases colonization of beans by symbiotic vesicular-arbuscular mycorrhizal fungi (Mulligan et al., 1985). Loosening the soil by chisels allows deep rooting, reduces water stress, and counteracts the adverse effect of the pathogen which is concentrated in the top soil zone (that is, the plow layer).

Control by chemicals

Various chemicals used as seed or soil treatments reduce fusarium root-rot severity on hypocotyls and roots of young seedlings. These chemicals are thiram, PCNB, benomyl, captafol, and Busan 30A. Seed treatment with effective fungicides, especially when applied as a slurry, will protect against seed rot and seedling damping-off and thus will ensure good seedling establishment in infested fields. Abdel-Rahman (1976) obtained good control by applying benomyl as an over-the-row spray (0.56 kg/ha) immediately after planting. Busan 30A (2.4 L/ha) and captafol (4.7 L/ha) also provided adequate control. However, most chemical soil treatments are not completely effective, are expensive, and do not last long enough to prevent infection of adventitious roots at later stages in the growing season.

Mussa and Russell (1977) report that the herbicides trifluralin, bentazon, and Avadex and the insecticides Metasystox and nicotine stimulate growth of *F. solani* f. sp. *phaseoli* and may increase root-rot damage. Eptam, dinoseb, glyphosate, and others also may increase root-rot incidence (Johal and Rahe, 1984; Wyse et al., 1976a and 1976b). However, Hagedorn and Binning (1982) showed that preplant incorporation into the soil of dinoseb increases bean yield and reduces root rot incited by several pathogens, including *F. solani* f. sp. *phaseoli*.

Control by plant resistance

Many bean genotypes reportedly have a high level of resistance to *F. solani* f. sp. *phaseoli* (Beebe et al., 1981; Boomstra and Bliss,

1977; Boomstra et al., 1977; Burke and Miller, 1983; Dickson and Boettger, 1977; Kraft et al., 1981; Statler, 1970; Wallace and Wilkinson, 1965 and 1975). However, many of these genotypes are late maturing, small seeded, and have other undesirable agronomic characteristics. Early maturing cultivars with resistance to *Fusarium* have been found amongst some pink cultivars such as Sutter Pink, Viva, Roza, and Gloria (Burke and Miller, 1983; Kraft et al., 1981). Although progress is being made, commercial cultivars with high levels of resistance to fusarium root rot that are early maturing and bush type beans are not yet available. Burke and Miller (1983) reported that *Fusarium*-resistant genotypes are also more tolerant to cold soil, drought, and soil compaction than susceptible cultivars. They suggested that combining tolerances to stress factors with *Fusarium* resistance would be most effective in controlling fusarium root rot of beans.

Resistance to fusarium root rot derives mainly from New York 2114-12 and P.I. 203958. P.I. 203958 is also resistant to pythium blight caused by five species of *Pythium* and to black root rot. It is controlled by three to seven dominant genes (Bravo et al., 1969; Wallace and Wilkinson, 1965). Hassan et al. (1971a) 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 (Boomstra and Bliss, 1977). They also stated that recurrent selection would be the most suitable breeding method to improve the recovery of this quantitative trait.

Boomstra et al. (1977) tested 800 accessions and identified 18 plant introductions (mostly 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 or African countries. Several reports (Beebe et al., 1981; Kistler and VanEtten, 1981; Pierre, 1971; Pierre and Bateman, 1967; Smith et al., 1981 and 1982) have shown that phaseolin and other phytoalexins are involved in the resistance mechanism operating in bean genotypes against *F. solani* f. sp. *phaseoli*.

Fusarium Yellows

Introduction

Fusarium yellows of beans is caused by *Fusarium oxysporum* Schlecht. f. sp. *phaseoli* Kendrick and Snyder (Kendrick and Snyder, 1942). The disease was first reported in California in 1928 and later in other regions of United States, including Colorado, Idaho, Montana, Rhode Island, and South Carolina. Serious outbreaks of this disease in Latin America have been reported from Colombia, Brazil, Panama, Costa Rica, and other countries of Central America (Cruz et al., 1974; Kraft et al., 1981; Sherf and MacNab, 1986; Weber, 1973; Wellman, 1977). Detailed information on the etiology, epidemiology, physiology, and management of fusarium wilt diseases, including bean yellows, can be found in Mace et al. (1981).

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

Etiology

The fusarium yellows pathogen is morphologically similar to all the members of the species *F. oxysporum*. However, it is recognized by its physiological and pathological adaptation to beans, hence the interspecific taxa designation f. sp. (forma specialis) *phaseoli* (Mace et al., 1981). Recently, Ribeiro and Hagedorn (1979b) documented the occurrence of two pathogenic races of *F. oxysporum* f. sp. *phaseoli* based on the differential reaction of bean germplasm to isolates obtained from Brazil, Netherlands, and United States. This pathogen produces microconidia, macroconidia, and chlamydospores. Dissemination, survival, and germination in soil are essentially similar to those described for *F. solani* f. sp. *phaseoli* (Kraft et al., 1981; Mace et al., 1981). This pathogen has been associated with seed as a surface contaminant (Weber, 1973; Zaumeyer and Thomas, 1957).

Epidemiology

The pathogen is capable of penetrating intact root tissue, usually near the root tip and just behind the root cap. After penetration, hyphae of the pathogen move inter- and intracellularly and invade the developing xylem vessels (Mace et al., 1981). Penetration of older parts of root and hypocotyl tissue also occurs, usually through wounds or natural openings (Dongo-D. and Müller, 1969; López-Duque and Müller, 1969). The fungus is confined to xylem vessels until the later stages of disease development, although limited invasion of xylem parenchyma tissue may occur. Infection appears to proceed between xylem vessels in susceptible cultivars, through hyphal growth, and through the transport of newly formed microconidia by the transpirational stream. Conidia are eventually trapped on the perforation plates and end walls of xylem vessels. The trapped conidia germinate, penetrate the cell walls, and produce microconidia in the adjoining vessel which then repeat the growth cycle until the whole vascular system is colonized. Progress between vessels is rapidly stopped in resistant cultivars, probably as a result of chemical and structural alterations in host tissue (Mace et al., 1981). The latter include vascular occlusion by the formation of gel plugs, tyloses, deposition of additional wall layers, and infusion of these structures with phenols and other metabolites (López-Duque and Müller, 1969; Mace et al., 1981). At later stages of disease development, pathogens grow into adjacent cortical tissue, producing large numbers of chlamydospores. The fungus may also emerge on the surface of infected plant tissue, producing abundant pink mycelial growth and conidia. Optimal temperature for growth on agar media is about 28 °C, but the most severe disease development occurs at 20 °C (Ribeiro and Hagedorn, 1979a). It was also reported that *Fusarium* yellow severity was increased in the presence of root-knot nematodes (*Meloidogyne javanica* (Treub) Chitwood and *M. incognita* (Kofoid et White) Chitwood) (Ribeiro and Ferraz, 1983; Singh et al., 1981).

Symptomatology

Aboveground symptoms on susceptible cultivars will appear seven to nine days after inoculation and severely infected plants may

die within 21 days (Thomas and Wood, 1981). However, disease severity was proportional to the incubation temperature and inoculum density (Ribeiro and Hagedorn, 1979b). Initial symptoms appear on lower leaves which exhibit yellowing and wilting (Figure 20). These symptoms may be confused with those caused by phosphorus deficiency. This yellowing and wilting becomes more pronounced and progresses upward into younger leaves. Stunting may also become evident, especially if plant infection occurred during the seedling stage. The margin of infected leaves may become necrotic and diseased plants become progressively more chlorotic. The fungus also can cause water-soaked lesions on pods (Goth, 1966). Severely infected plants may exhibit permanent wilting and premature defoliation. The characteristic pink-orange spore masses of the fungus may appear on stem and petiole tissue (Figure 21). Vascular discoloration is the diagnostic symptom (Figure 22) and is usually evident after the initial appearance of foliar symptoms. However, the reddish brown vascular discoloration of root, stem, and petiole tissue of infected plants may vary considerably in intensity, depending on cultivar reaction, severity of infection, and environmental conditions.

Control

Cultural and chemical control measures reported for *F. solani* f. sp. *phaseoli*, especially crop rotation and fungicide seed treatments, are also applicable for fusarium yellows on beans (Costa, 1972; Kendrick and Snyder, 1942; Mace et al., 1981; Sherf and MacNab, 1986). However, the most effective control measure against fusarium yellows is the use of resistant cultivars. Echandi (1967) reported that all commercial bean cultivars in Costa Rica that were evaluated under artificial conditions were susceptible to fusarium yellows. In the state of Santa Catarina, Brazil, the newly released bean variety, EMPASC 201, is very susceptible (R. Balardin, personal communication). Nevertheless, the cultivars Manteigão Preto, Manteigão Lustroso, Manteigão 41, Pintado, Roxinho Precoce, Carioca, Pintadinho Precoce, Suieu, Cherokee Wax, Processor, Contender, and Rosinha Sem Cipo were resistant in Brazil (Costa, 1972; Cruz et al., 1974; Echandi, 1967; Ribeiro and Hagedorn, 1979a; Zaumeyer and Meiners, 1975). However, given

that the pathogen is variable, these varieties may not be resistant elsewhere. Dongo-D. and Müller (1969) reported that their resistant cultivars usually are red-seeded and produce many strong lateral roots after inoculation.

Recently, Ribeiro and Hagedorn (1979a) showed that a single gene controlled resistance to each of the two known races of *F. oxysporum* f. sp. *phaseoli*. The dominant gene controlling resistance to the Brazilian race was named FOP 1 and was present in the cultivars Tenderette, Pintado, and, possibly, Early Gallatin. Resistance to the European and North American race was controlled by an incompletely dominant gene, named FOP 2, which was found in the cultivar Prato Ubershinla. Pastor-Corrales and Abawi (1987) evaluated large numbers of bean accessions for resistance to a Brazilian isolate of *F. oxysporum* f. sp. *phaseoli* under controlled greenhouse conditions. Several accessions were highly resistant, including BAT 336, BAT 477, BAT 1385, BAT 1400, G 4000, A 300, A 301, LM 21525, WAF 4, Cacahuete, Mortiño, Ecuador 605, XAN 112, AND 323, AND 357, AND 286, AND 313, XAN 195, Calima, Ecuador 1056, and HF 665-63-1 (a breeding line selected by Dr. Paulo Miranda, Recife, Pernambuco, Brazil).

Pythium Root Rot

Introduction

Pythium root rot is caused by several *Pythium* species such as *P. ultimum* Trow, *P. irregulare* Buisman, *P. aphanidermatum* (Edson) Fitzpatrick, and *P. myriotylum* Drechsler (Casanova and Díaz-Polanco, 1966; Gay, 1969; Hoch et al., 1975; Kraft and Burke, 1971; Lumsden et al., 1976; Pieczarka and Abawi, 1978c; Stanghellini and Hancock, 1971; Walker, 1952; Zaumeyer and Thomas, 1957). Less common species are cited by Zaumeyer and Thomas (1957) and Lumsden et al. (1976). In Latin America, *P. aphanidermatum* appears to be a common species (Casanova and Díaz-Polanco, 1966).

Pythium-incited diseases have been reported from United States (Adegbola and Hagedorn, 1969; Dickson and Abawi, 1974; Hendrix and Campbell, 1973; Hoch et al., 1975; Kobriger and Hagedorn, 1974).

1984; Kraft and Burke, 1971; Pieczarka and Abawi, 1978c), Canada (Chew and Hall, 1984; Sippell and Hall, 1982a and 1982b), Brazil (de Carvalho, 1965), El Salvador (Acuña and Waite, 1975), Mexico (Crispín-Medina and Campos-Avila, 1976; Crispín-Medina et al., 1964), Venezuela (Casanova and Díaz-Polanco, 1966), and many other countries. These diseases are major production problems of beans and especially of snap bean cultivars grown in United States (Dickson and Abawi, 1974; Pieczarka and Abawi, 1978c). However, their importance in Latin America and Africa has not yet been clearly established.

Common names frequently used for pythium root rot in Latin America are “marchitamiento por *Pythium*,” “murcha de *Pythium*,” and “pudrición radical por *Pythium*.”

Etiology

Pythium species grow well on artificial media, producing the characteristic coenocytic hyphae, sporangia, and oospores. The asexual reproductive structure (sporangium) can be filamentous, globose, lobate, or oval in shape, depending on the species. Sporangia may germinate directly by a germ tube, as is the case with *P. ultimum*, or through the production of zoospores, as in *P. aphanidermatum* and *P. myriotylum*. Zoospores are kidney shaped with two lateral flagella. Zoospore production is preceded by formation of a vesicle at the tip of a discharge tube which arises from the sporangium. The sexual stage is characterized by production of the oogonium and antheridium, and eventual oospore production after successful fertilization of mature oogonia.

Depending on the species, oogonia are either smooth walled or spiny. The antheridium also varies between species in shape, origin, and number per oogonium. Oospores are thick walled, smooth, plerotic (fill the oogonial cavity) or aplerotic (partially fill the oogonial cavity). They germinate after they are converted to thin-walled structures (Lumsden and Ayers, 1975) by germ tubes, which function as infection hyphae, or by the production of zoospores. *Pythium* spp. are natural soil inhabitants and can survive for a long time through active saprophytic growth or in the form of resistant structures such as oospores (Stanghellini, 1974; Walker, 1952;

Wellman, 1972). However, *Pythium* spp. are considered poor competitors (Hendrix and Papa, 1974) and their saprophytic activities are usually restricted (Barton, 1961). Activities of *Pythium* spp. are especially favored by high soil moisture (Hendrix and Papa, 1974; Stanghellini, 1974). Sporangia of *P. ultimum* can survive for 11 months in soil, whereas zoospores of *P. aphanidermatum* survive only up to seven days in field soil (Hendrix and Papa, 1974). Hoppe (1966) reported that *P. ultimum* survived in air-dried soil for 12 years, but survived for only two years at temperatures below -18 °C.

Species of *Pythium* vary greatly in their temperature requirements. *Pythium ultimum* and *P. debaryanum* Hesse are commonly active at low soil temperatures and thus are considered as low-temperature species. *Pythium aphanidermatum* and *P. myriotylum*, however, are encountered at higher soil temperatures and are considered as high-temperature species (Hendrix and Campbell, 1973; Hendrix and Papa, 1974; Zaumeyer and Thomas, 1957). Hoch et al. (1975) reported that *P. ultimum* is highly pathogenic at 16 °C and 28 °C, whereas *P. aphanidermatum* is only slightly pathogenic at 16 °C but highly pathogenic at 28 °C. However, Pieczarka and Abawi (1978b) found that a low-temperature species such as *P. ultimum*, was always more damaging at 15 °C than at higher temperatures. Optimal pH and temperature for germination of *P. aphanidermatum* oospores in sterilized soil were 7.5 and 30 °C, respectively (Adams, 1971).

Various workers have studied and enumerated the soil population densities of *Pythium* spp., but these data usually have included the total densities of pathogenic and nonpathogenic species. Pieczarka and Abawi (1978b) reported that soil populations of *Pythium* species varied considerably between and within bean fields. Average densities of the low-temperature species (principally *P. ultimum*) ranged from 133-1560 propagules/g of oven-dry soil. Subsequent greenhouse tests revealed that one propagule/g of oven-dry pasteurized soil caused a 31% reduction in plant growth and 85% reduction in stand count. However, much higher population densities are required for serious damage to occur on plants grown in natural soils.

Short-distance dispersal of *Pythium* species within fields may occur by zoospore movement in soil and water, or by wind and

water splashing of soil infested with oospores, sporangia, chlamydospores, or mycelial fragments. Long-distance dispersal may occur through movement of plant debris or infested soil in irrigation water or on equipment, and possibly by wind-blown soil particles (Hendrix and Campbell, 1973).

Epidemiology

Penetration of bean tissue by *Pythium* spp. usually occurs directly through the intact root and stem epidermal layer after formation of infection pegs (Dow and Lumsden, 1975; Endo and Colt, 1974). Penetration may also occur through natural openings with or without appressorial formation, and directly through wounds by individual hyphae (Endo and Colt, 1974). Severity of infection is affected by root exudates, inoculum density, soil moisture, soil temperature, and soil pH (Kraft and Erwin, 1967; Pieczarka and Abawi, 1978b). Soil temperature and moisture, however, are the most important factors since *Pythium* spp. are most active as pathogens in soils with high moisture levels (Hendrix and Campbell, 1973).

In general, *Pythium* species contribute to the complex involving other root-rot pathogens such as *Rhizoctonia solani*, *Fusarium solani* f. sp. *phaseoli*, and nematodes (Dickson and Abawi, 1974; Pieczarka and Abawi, 1978a). Pieczarka and Abawi (1978c) reported that *P. ultimum* acts synergistically with *F. solani* f. sp. *phaseoli* to cause increased root-rot damage on beans, but *R. solani* apparently is antagonistic to *P. ultimum* and reduces root-rot severity.

Symptomatology

Depending on the time of attack, species of *Pythium* cause seed rot, pre- and postemergence damping-off, root rot, foliar blight, and pod rot diseases (Abawi et al., 1985; Adegbola and Hagedorn, 1969; Hoch et al., 1975; Pieczarka and Abawi, 1978b). Seeds may be invaded (Figure 23) and killed by the fungus very shortly after planting and before germination. The fungus can attack all parts of seedlings up to about eight days old, resulting in preemergence and

postemergence damping-off. On older plants, *Pythium* causes a reduction and discoloration of the root system (Figure 24) and a complete rotting and decay of fibrous rootlets (Figure 25). Elongated, water-soaked areas also appear on the stem. The cortical region of both root and stem tissues of severely infected plants become very soft, brownish, somewhat sunken, and eventually collapse (Figure 26).

During continual wet weather the fungus spreads upward, infecting stem branches, petioles, leaves, and, at times, may reach the growing tip, resulting in wilt and plant death. Also, during cool and prolonged moist conditions, pods in contact with the soil often will become infected, exhibiting water-soaking and fluffy white fungal growth that resembles a brush. This phase of the disease may be mistaken for the early stages of the white mold disease caused by *Sclerotinia sclerotiorum* (Libert) de Bary.

Infection by *Pythium* spp. may also begin on foliage of young or mature bean plants under moist conditions (Adegbola and Hagedorn, 1969). Although infection points may appear on any above-ground tissue, they are most commonly found on axillary buds. Infection results in the death of buds and spreads rapidly to other plant tissue. Infected tissue initially exhibits water-soaking, brownish discoloration, and eventually becomes covered with fluffy white mycelial growth. Severely infected plants (Figure 27) prematurely defoliate and eventually die.

Control by cultural practices

Since *Pythium* spp. are indigenous to most soils (Stanghellini, 1974), exclusion is not a practical control measure. *Pythium* root rot may be minimized by cultural practices that reduce soil moisture and soil compaction as well as increase plant vigor. Wide plant spacing provides better soil aeration, less soil shading, and less pathogen spread between plants. Nitrogenous compounds can be toxic to and may suppress *Pythium* species such as *P. aphanidermatum*, when incorporated into the soil (Grover and Sidhu, 1966). 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. and improving soil tilth.

Disease incidence and severity are affected by root damage from other soil-borne pathogens (Piecarka and Abawi, 1978b and 1978c) and cultural practices such as soil cultivation, that result in root pruning. Piecarka and Abawi (1978a) suggested that pythium root rot incidence would be less if beans were planted in well-drained soils and on raised beds or ridges.

Control by chemicals

Various chemicals reduce the severity of infections caused by *Pythium* spp. These include the fungicides fenaminosulf, chloroneb, pyroxychlor, captan, thiram, zineb, and metalaxyl applied singly or in combinations. Fumigants such as chloropicrin, methyl bromide, and dichloropropene also have been highly effective, but are expensive and difficult to apply (Hendrix and Campbell, 1973). Seed treatments with prothiocarb and Terrazole were also effective (Papavizas et al., 1977). Metalaxyl is the most recently available fungicide that is highly effective against *Pythium*-incited diseases on a variety of crops, including beans. The seed treatment formulation of metalaxyl is used at a rate of 1.4 g/kg, preferably as a slurry seed treatment. Metalaxyl can also be used as an in-furrow or over-the-row band-incorporated treatment at planting time, using 12 ml, diluted in water, per 100 m of linear row.

Control by plant resistance

Bean cultivars and accessions with resistance to infection by *Pythium* spp. have been identified (Adegbola and Hagedorn, 1970; Dickson and Abawi, 1974; Reeleder and Hagedorn, 1981; York et al., 1977; Zaumeyer and Meiners, 1975). Adegbola and Hagedorn (1970) reported that P.I. 203958 (also resistant to fusarium root rot and to black root rot) and Bush Green Pod are resistant to pythium blight caused by five species of *Pythium*. The white-seeded snap bean breeding line 1273 from Cornell University, New York State, was highly tolerant to seed decay and pre-emergence damping-off diseases incited by *P. ultimum* under artificial soil infestations and growth chamber conditions (Dickson and Abawi, 1974; York et al., 1977). This resistance was polygenic and recessive in nature.

Specific parental combinations did yield a higher proportion of resistant F_3 progeny with colored seed coats (York et al., 1977).

Dickson and Boettger (1977) found an association between seed-coat color and resistance to *Pythium* species, but this association can be broken. However, line 1273, Black Turtle Soup, and P.I. 203958 (although all are resistant to the seed decay phase) were susceptible to root rot incited by *Pythium* species. Thus, bean germplasm may have to be evaluated separately for resistance to each stage of infection of the disease incited by these pathogens (Pieczarka and Abawi, 1978b). Recently, Reeleder and Hagedorn (1981) reported that P.I. 203958, Oregon 70-169-1, and Wisconsin 46 were resistant to hypocotyl rot, but not to root rot incited by *P. myriotylum*.

Southern Blight

Introduction

Southern blight or sclerotium root rot of bean is caused by *Sclerotium rolfsii* Sacc. The disease occurs in many warm and humid bean-growing areas located between the northern and southern 38° latitudes (Sherf and MacNab, 1986). Sclerotium root rot has been reported as an important disease of beans in many Latin American countries, including Brazil (Costa, 1972; Kimati and Mascarenhas, 1967; Shands et al., 1964; Vieira, 1967), Mexico (Crispín-Medina and Campos-Avila, 1976), Costa Rica (Echandi, 1976), and Venezuela (Casanova and Díaz-Polanco, 1966). The author has also observed severe incidence of this disease in Colombia and Peru. Direct estimates of yield losses caused by this pathogen in beans are not available.

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

Etiology

Sclerotium rolfsii has a wide host range of more than 200 species of plants, involving most vegetable crops and including beans (Sherf and MacNab, 1986). The fungus grows readily on a variety of artificial agar media and on host residue on the soil surface under favorable environmental conditions. It produces white and coarse mycelium and numerous characteristic sclerotia that are smooth walled, round (0.5-1.5 mm in diameter), and brown. *Sclerotium rolfsii* does not produce asexual spores and the basidial state, *Aetholia rolfsii* (Curzi) Tu and Kimbr., is rarely produced in culture or in the field (Walker, 1952).

Sclerotia of *Sclerotium rolfsii* survive in soil for at least one year. The fungus can also survive in infected host tissue (Singh and Mathur, 1974) and saprophytically by colonizing available organic residue. High moisture and temperature are required for optimal growth and reproduction of the fungus in soil. This pathogen is sensitive to low temperature and rarely occurs in bean-growing areas with cold periods. In culture media, it grows at temperatures between 13-37 °C, with an optimum of 30-35 °C. Sclerotia germinate at temperatures between 10-35 °C, but require high relative humidity of above 99%. Sclerotial germination in soil decreases with increased depth due to reduced aeration (Abeygunawarena and Wood, 1957). Germination occurs at a soil pH range of 2.6-7.7, with an optimum of 2.6-4.4 (Coley-Smith and Cooke, 1971). Sclerotial germination is induced by volatiles which emanate from crop residue in the soil and is enhanced by wet and dry conditions (Beute and Rodríguez-Kabana, 1979; Linderman and Gilbert, 1975).

Dispersal of the pathogen may occur through contaminated irrigation water, infested soil adhering to agricultural tools and animals, or contaminated seed (Bolkan et al., 1976; Sherf and MacNab, 1986; Walker, 1952; York et al., 1977). Sclerotia can pass through the digestive tract of animals without losing viability and, therefore, can be transported relatively long distances by animals fed with infected host material (Leach and Davey, 1942).

Epidemiology

Southern blight of beans is most destructive at high temperature and moisture conditions which favor sclerotial germination and optimal mycelial growth. Maximum disease severity occurs at 25-35 °C which is also the optimal range for mycelial growth and sclerotial germination of *S. rolf sii*. Serious disease outbreaks often accompany unusual wet seasons. Southern blight usually occurs in epidemic proportions when rainy periods follow dry periods. The disease is not a problem on calcareous soils with a high pH. However, sclerotial production and germination are greater under acidic conditions.

The pathogen is strongly aerobic and, thus is prevalent in light well-aerated soils. Deeply buried sclerotia are prevented from germinating (Jenkins and Averre, 1986). Mycelial strands, originating from infected debris or germinating sclerotia, penetrate bean tissue through natural openings, wounds, or by direct penetration of intact tissue (Sherf and MacNab, 1986; Walker, 1952). Before penetration can occur, there has to be an appreciable mycelial growth of *S. rolf sii* on the plant surface to be invaded (Abeygunawarena and Wood, 1957; Coley-Smith and Cooke, 1971). After penetration, the fungus ramifies very rapidly in stem and root tissues, resulting in hydrolysis and death of tissue in advance of invasion. Several hydrolytic enzymes and phytotoxins are produced by *S. rolf sii* and are present in infected tissue (Bateman, 1969; Bateman and Beer, 1965; VanEtten and Bateman, 1969). Bateman and Beer (1965) suggested that a synergistic interaction exists between oxalic acid and polygalacturonase and that this synergism plays a major role in the penetration and rapid destruction of host tissue by *S. rolf sii*.

Symptomatology

Infection of beans by *S. rolf sii* can result in damping-off, stem blight, and root rot. Initial symptoms on infected plants appear as dark-brown, water-soaked lesions on the lower stem surface area just below the soil line (Figure 28). These lesions extend downward, through stem tissue into the tap root, and may destroy the cortical tissue and so start root-rot symptoms. Under moist conditions,

lesions on the stem tissue continue to progress downward and eventually may kill the entire root system. Aboveground symptoms consist of leaf yellowing and defoliation of the upper plant branches which may be followed by a sudden wilt condition. Abundant, white, coarse mycelium and sclerotia and soil particles are often found attached to stem tissue near the soil line. Bean pods in contact with the soil may also become infected and rot. Fungal growth on the soil surface will continue, especially under wet conditions, and may result in plant-to-plant infections.

Control by cultural practices

Control measures that exclude introduction of *S. rolfsii* into clean fields such as avoiding the use of contaminated seeds or infected plant material, should be practiced. Eradication of susceptible weed hosts and destruction of infected host residue by burning or deep plowing will reduce soil population densities of *S. rolfsii* and, therefore, disease potential. Buildup of inoculum can also be reduced by avoiding low-pH soils, improving soil drainage, using wide plant spacing, applying lime to increase soil pH, and using a long crop rotation with nonhost crops such as sorghum, maize, or other cereals. Soil application of nitrogenous amendments such as ammonia, ammonium nitrate, urea, and others have reduced infection of host tissue by *S. rolfsii* (Henis and Chet, 1968; Leach and Davey, 1942). Reynolds (1970) reported that a soil amendment with coconut mulch reduced infection and increased bean yield considerably.

Díaz-Polanco and Castro (1977) isolated a *Penicillium* sp. which gave good biological control of *S. rolfsii* under greenhouse conditions. Backman and Rodríguez-Kabana (1975) demonstrated the effectiveness of the antagonist *Trichoderma harzianum* Rifai in controlling *S. rolfsii* under field conditions on peanuts.

Control by chemicals

Sclerotia are difficult to destroy with fungicides. However, various fungicides are effective against *S. rolfsii*, including PCNB, captafol, fentin acetate, and tridemorph, when applied as soil

treatments (Endo and Colt, 1974; Mukhopadhyay and Upadhyay, 1976; Sherf and MacNab, 1986; Sturgeon and Jackson, 1976). The herbicide Eptam, however, aggravated the damage caused by *S. rolfsii* to ladino clover and cotton. It reduced the biocontrol activity of *Trichoderma viride* Persoon ex Fries against *S. rolfsii* (Peeples et al., 1976).

Control by plant resistance

Only limited information is available on the reaction of bean germplasm to infection by *S. rolfsii*. However, Mexico 348-2 and Blanco are moderately tolerant to *S. rolfsii*.

Black Root Rot

Introduction

Black root rot of beans is caused by *Thielaviopsis basicola* (Berk. and Br.) Ferr. (syn. *Chalara elegans* Nag Raj and Kendrick). The distribution and importance of this pathogen to beans in Latin American and African countries are not known. It causes damage to beans in United States, Italy, and Germany (Abawi et al., 1985; Walker, 1952; Zaumeyer and Thomas, 1957). However, this pathogen is widespread in Latin America and Africa and causes severe black root-rot diseases on many susceptible crops, including alfalfa, beet, carrot, celery, cotton, maize, peanuts, peas, squash, sweet potatoes, tobacco and tomato (Yarwood and Levkina, 1976).

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

Etiology

The fungus grows and sporulates readily on artificial agar media. It exhibits considerable variation in colony appearance, zonation, growth rate, and the shape and number of spores produced (Huang and Patrick, 1971; Specht and Griffin, 1985). Asexual spores produced by *T. basicola* are endoconidia and chlamydospores. The hyaline, small, and cylindrical endoconidia are produced within the

conidiophores (phialides) and are extruded singly or in chains. Chlamydospores are thick walled, dark brown, multicellular, and are produced laterally or terminally on the mycelium. Individual cells of the chlamydospores eventually separate, each having the ability to germinate and therefore infect. The long-term survival structures of *T. basicola* in soil are chlamydospores because endoconidia are short-lived under natural conditions.

The fungus can be easily isolated from soil on fresh carrot discs or selective agar media (Specht and Griffin, 1985). *Thielaviopsis basicola* is widely distributed in bean fields in New York, but its density is variable among fields, ranging from 39-516 propagules/g of soil. The overall average for all fields sampled was 223, with individual samples ranging from 0-1213 propagules/g of soil. In field microplot tests, the initial population densities of *T. basicola* correlated significantly with reduced weight of bean roots, total foliage and pods, and also with increased root-rot severity (Abawi and Cobb, 1984). Means of dispersal for this pathogen among fields are similar to those reported for *Rhizoctonia* or *Fusarium* species. It appears that the growth and sporulation of *T. basicola* are favored by relatively high temperatures, but its damage to beans is more severe at low temperatures (15-20 °C) which are not optimal for plant growth (Maier, 1961). Activities of the fungus are also favored by high moisture, neutral to alkaline soil conditions, and nitrogen fertilizers (Papavizas et al., 1970; Smiley, 1975; Wilcox, 1965).

Epidemiology

Hyphae, originating from chlamydospores of *T. basicola*, penetrate intact bean tissue directly, without forming appressoria (Christou, 1962b). However, it may also penetrate bean tissue through wounds or become established in lesions produced by other pathogens such as *Fusarium solani* f. sp. *phaseoli* (Walker, 1952). Lumsden and Bateman (1968) reported that phosphatidase enzymes may play a major role during penetration of bean epidermal cells and later phases of pathogenesis of *T. basicola*. The fungus ramifies intra- and intercellularly by producing constricted and nonconstricted hyphae, respectively. Chlamydospores are produced by nonconstricted hyphae throughout infected tissues. Under moist conditions, reproductive hyphae protrude through the epidermis

layer, resulting in the production of masses of chlamydospores and endoconidia.

Symptomatology

The main symptom of this disease on beans is the production of numerous elongated lesions on stem and root tissues. Lesions are initially reddish purple, but later become dark charcoal to black in color. As infection progresses, the lesions often coalesce to form large black areas on the hypocotyl and roots (Figure 29). Superficial lesions cause limited damage, but deep and severe infections cause plant stunting, premature defoliation, and eventual plant death.

Control by cultural practices

Selection of well-drained soils, crop rotation with nonhost crops, and maintaining relatively low soil pH will reduce soil populations of this pathogen and may lower disease severity. Incorporation of several plant residues have suppressed black root rot on beans (Papavizas and Lewis, 1971; Papavizas et al., 1970). The most effective amendments were alfalfa hay, cabbage, and oil-seed meals which also reduced population density and viability of chlamydospores of *T. basicola* in the field.

Control by chemicals

Soil treatments with fungicides such as benomyl, thiabendazole, and captan or fumigants such as Vorlex and dazomet are highly effective against black root rot of beans (Papavizas and Lewis, 1971; Papavizas et al., 1970). However, it is doubtful that the use of these chemicals on beans is economical or feasible.

Control by plant resistance

Hassan et al. (1971b) reported that the breeding line 2114-12 and P.I. 203958 (which is also resistant to fusarium root rot and pythium blight) are resistant to the black root-rot fungus. They concluded that these two accessions have the same genes for resistance to *T.*

basicola. The resistance was controlled by, perhaps, three partially recessive genes. Since then, these lines have been used in many breeding programs as sources of resistance. Pierre (1971) suggested that, in beans, resistance to *T. basicola* results from the formation of two phytoalexins which restrict the size and development of lesions.

Texas Root Rot

Introduction

Texas root rot, or phymatotrichum root rot, is caused by *Phymatotrichum omnivorum* (Shear) Duggar. The fungus has a wide host range, attacking more than 2000 species of dicotyledonous plants, but not monocotyledonous plants (Streets and Bloss, 1973). However, this pathogen is largely confined to the alkaline soils of southwestern United States, and northern and central regions of Mexico (Lyda and Burnett, 1975; Streets and Bloss, 1973). In these areas, it is an important disease of cotton and alfalfa. Crispín-Medina and Campos-Avila (1976) reported that *P. omnivorum* is a minor disease of beans in Mexico. Texas root rot has not been reported on beans in other Latin American countries. Streets and Bloss (1973) provide detailed information on the ecology, biology, and diseases caused by *P. omnivorum*.

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

Etiology

P. omnivorum has a brown mycelium, consisting of large fine cells, and strands produced by many intertwined hyphae. Slender, acicular hyphae are produced by cells on the outer layer of the strands. The strands branch in a cross-shaped manner which is a diagnostic feature of this fungus. Under moist conditions, brown spore mats are produced on the soil surface and contain masses of conidia that are hyaline, single celled, globose to ovate, and borne on the swollen tip of vegetative hyphae. The function of these

conidia are unknown since their germination is erratic. Sclerotia are dark, vary in size and shape, and are produced singly or in chains. The basidial stage appears to occur rarely in soil or on agar media during relatively cool periods (15-20 °C). Basidia are formed in clusters and basidiospores are strongly curved. 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 for up to 12 years.

Epidemiology

Phymatotrichum root rot is usually found in localized spots within a field and occurs primarily in soils with a pH of 8 or slightly higher (Lyda and Burnett, 1975; Streets and Bloss, 1973). Hyphae from germinating sclerotia or infected host tissue grow on the root surface, producing coarse strands that envelop the root, and then penetrate the host tissue. Host penetration always occurs below the soil line on roots or stem tissues. Progress of hyphae in host tissue is both inter- and intracellular and host cells appear to die before penetration by hyphae. Disease development is favored by relatively dry soil and high temperature.

Symptomatology

Underground symptoms induced by *P. omnivorum* are dark, sunken lesions that often become covered with coarse whitish to yellowish mycelium. A pinkish-buff color may be present on lightly infected young rootlets. The aboveground symptoms consist of stunting and sudden wilting which usually appear during blossom initiation.

Control

Specific information for the control of Texas root rot on beans is very limited. Long crop rotation with nonhost crops such as maize, small cereals, and sorghum; eradication of susceptible weeds; choice of soils with relatively low pH; deep plowing; and soil application of the ammonium form of nitrogenous fertilizer will reduce soil populations of the fungus and suppress disease development. Bean

germplasm should be screened to identify available sources of resistance, if any, for use in breeding programs.

Aphanomyces Root-and-Hypocotyl Rot

Introduction

This disease is caused by two formae specialis of *Aphanomyces euteiches* Drechs., that is, *A. euteiches* f. sp. *phaseoli* which infects only beans and *A. euteiches* f. sp. *pisi* which infects beans and, particularly, peas (Pfender and Hagedorn, 1982a and 1982b). Beans have long been known to be susceptible to infection by *A. euteiches* (Papavizas and Ayers, 1974). However, the first documentation of serious damage to beans by *A. euteiches* under field conditions was that of Pfender and Hagedorn (1982a and 1982b). *Aphanomyces* damage to beans was also observed in two bean fields in western New York for the first time during 1986 (G. S. Abawi, unpublished data). Reports of damage to beans by this pathogen in Latin American countries or other bean-growing areas are not available. Papavizas and Ayers (1974) provide detailed information on the ecology, biology, and diseases of *Aphanomyces* species on peas and sugar beets.

Epidemiology

Mycelium and zoospores of *Aphanomyces* are believed to survive for only a short time in soil (Papavizas and Ayers, 1974). However, in the absence of susceptible hosts, they may survive by colonizing nonhost plants or organic debris in soil, resulting in the production of new spores. Oospores can survive for more than 10 years. The fungus can be disseminated between fields by wind-blown infected debris or infested soil, contaminated seed, or on agricultural implements. These bean pathogens have an optimal growth temperature of 28 °C on agar media. No growth occurs at 35 °C (Pfender and Hagedorn, 1982a). They cause the most severe damage at 24-28 °C, less damage at 20 °C, and only slight damage at 16 °C (Pfender and Hagedorn, 1982b). High soil moisture is essential for the activities of these pathogens, signifying that soil moisture content affects the severity of their diseases.

Symptomatology

Symptoms (Figure 30) on severely infected plants may become confused with those incited by *Pythium* spp. Initial symptoms on root and hypocotyl tissues appear as water-soaked and straw-colored lesions. Under favorable conditions, these lesions expand rapidly through the cortical tissues, resulting in soft rotting of the tissues which then become brown. Cortical tissues of the roots may become completely destroyed and slough off. The necrotic streaking on the hypocotyl may extend well above the soil line and infected areas may become sunken. Severely infected plants are stunted, show chlorosis, and suffer premature defoliation. *Aphanomyces* may interact synergistically with *Pythium* spp., increasing damage to beans and causing higher mortality (Pfender and Hagedorn, 1982b).

Control

Very limited information is available for control of this disease on beans. However, avoidance of heavily infested soil, use of crop rotation, improvement of soil drainage, and the application of organic and inorganic soil amendments have reduced *Aphanomyces* root-rot severity on peas (Papavizas and Ayers, 1974). Interestingly, the fungicide metalaxyl, although highly effective against *Pythium* species, is ineffective against species of *Aphanomyces*.

Pfender and Hagedorn (1982a) reported that all bean cultivars and breeding lines evaluated in their tests were susceptible to infection by *A. euteiches* f. sp. *phaseoli*. Only the Wisconsin breeding line 46 showed slight damage. Resistances to *A. euteiches* f. sp. *phaseoli* and *Pythium* species were also reported (Rand et al., 1983) in the Red Kidney type Plant Introductions: 209488, 313454, 309758, 209492, and 312068.

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Chapter 7

RUST

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Introduction

Bean rust is caused by *Uromyces appendiculatus* (Pers.) Unger var. *appendiculatus* (syn. *U. phaseoli* (Reben) Wint.). The disease has a worldwide distribution (Zaumeyer and Thomas, 1957). It consistently causes major production problems in humid tropical and subtropical areas and periodic severe epidemics in humid temperate regions (Ballantyne, 1974; Vargas-G., 1980; Zaumeyer and Thomas, 1957).

In Latin America, major losses occur in Argentina, Bolivia, Brazil, Colombia, Costa Rica, Cuba, Dominican Republic, Ecuador, El Salvador, the Chimaltenango district of Guatemala, Haiti, Honduras, Jamaica, Mexico, northern Nicaragua, and coastal Peru (Crispín-Medina et al., 1976; Dongo-D., 1971; González-Avila, 1976; Guerra and Dongo-D., 1973; López-G., 1976; Rodríguez-Alvarado, 1976; Shaik, 1985b; Vargas-G., 1970, 1971, and 1980).

Major losses occur in Burundi, Ethiopia, Kenya, Malawi, Rwanda, South Africa, Tanzania, Uganda, and Zimbabwe (Assefa, 1985; CIAT, 1981). Severe epidemics occur in Australia, China, United States, and some areas of Europe (Ballantyne, 1978; Kelly, 1982; Teng, 1932; Yeh, 1983; Zaumeyer and Thomas, 1957). A major rust epidemic occurs in many areas of Mexico every four to

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five years, although in the valley of Mexico, other valleys, and some Gulf states rust is endemic and causes substantial losses every year (Crispín-Medina et al., 1976).

Yield losses are most severe when plants are infected during the preflowering and flowering stages of development (Almeida et al., 1977a; Costa, 1972; Crispín-Medina et al., 1976; Nasser, 1976; Wimalajeewa and Thavam, 1973; Yoshii and Gálvez, 1975). Disease loss estimates in the greenhouse and field include 40%-50% plant dry weight reduction (Almeida et al., 1977a). Yield losses are estimated at 18%-28% (Dongo-D., 1971; Venette and Jones, 1982b; Wimalajeewa and Thavam, 1973; Zulu and Wheeler, 1982), 36%-45% (Kelly, 1982; Nasser, 1976; Venette and Jones, 1982b), and 40%-100% (Hilty and Mullins, 1975; Kelly 1982; Schwartz, 1984; Solís, 1977; Venette and Jones, 1982b; Zaumeyer and Thomas, 1957).

Uromyces appendiculatus infects many species of *Phaseolus*, including tepary bean (*P. acutifolius* A. Gray var. *acutifolius*), scarlet runner bean (*P. coccineus* L.), lima bean (*P. lunatus* L.), *P. coccineus* subsp. *obvallatus* (Schlecht.) M.M.S., *P. polystachyus* (L.) B.S.P., *P. maculatus* Scheele, *P. polystachyus* var. *sinuatus* (Nutt) M.M.S., and common bean (*P. vulgaris* L.). It also infects siratro (*Macroptilium atropurpureum* (DC.) Urb.), cowpea (*Vigna unguiculata* (L.) Walp. ssp. *unguiculata*), (Arthur, 1915; Rey-G. and Lozano-T., 1961; Zaumeyer and Thomas, 1957), *V. luteola* (Jacq.) Benth., *V. adenantha* (G. F. Meyer) M.M.S., and *V. vexillata* (L.) A. Rich. (Almeida et al., 1977c). The prevalent host is *P. vulgaris*. Its natural occurrence on *P. lunatus* in United States is rare, and differs from the primary rust pathogen of *Vigna* species which is the cowpea rust fungus (*Uromyces vignae*) (Cummins, 1978).

Common names frequently used for rust in Latin America include "roya" and "chahuixtle" in Spanish and "ferrugem" in Portuguese.

Almeida (R. T. Almeida, 1977) reported the existence of a variety of bean rust collected in 1945 from *Macroptilium longepedunculatum* (Benth.) Urban (then known as *Phaseolus longepedunculatus* ex Benth.) by Viegas, who named the rust *Uromyces*

phaseoli longepedunculati Viegas. Almeida studied herbarium samples of the original collection, confirmed that it differs morphologically from *U. appendiculatus* var. *appendiculatus*, and, according to current nomenclature rules, named it *Uromyces appendiculatus* (Pers.) Ung. var. *brasiliensis* R. Almeida var. nov.

Phaseolus vulgaris, although susceptible to the soybean rust fungus (*Phakopsora pachyrhizi* Sydow), is, apparently, an uncommon host of that pathogen (Cummins, 1978; Stavely et al., 1985; Vakili and Bromfield, 1976). This fungus is not known to produce pycnia or aecia and produces uredia and teliosori very different from those of *U. appendiculatus* (Cummins, 1978; Stavely et al., 1985). Several uredia, each less than 0.3 mm in diameter, are produced in a necrotic lesion 0.2 to 4 mm in diameter. Uredia and spores are lighter in color and spores are smaller than those of *U. appendiculatus*. In Popayán, Colombia, *Phakopsora pachyrhizi* occurs on *Phaseolus lunatus*, and *P. lunatus* x *P. vulgaris* hybrids, but not on *P. vulgaris* (M. A. Pastor-Corrales, unpublished data).

Etiology

Uromyces appendiculatus is an obligate parasite which belongs to the Basidiomycotina subdivision of fungi. It has an autoecious, macrocyclic life cycle which is completed entirely on the bean host (Andrus, 1931; Cummins, 1978). Overwintering, resting teliospores germinate to produce basidia and basidiospores that infect the host leaf, producing pycnia. Upon cross fertilization with pycniospores, an aecium is produced and aeciospores develop, infecting the leaf and producing uredia pustules. The uredia in turn, produce uredospores that infect the plant, producing more uredia and giving rise to repeated infections over most of the growing season. As uredia age, if conditions are appropriate, they produce thick-walled teliospores.

Pycnia and aecia are rarely observed under field conditions although aecia have been found in regions of Oregon (Zaumeyer and Thomas, 1957), New York (Jones, 1960), North Dakota (Venette et al., 1978), and southern Germany (Heinze, 1974). In North Dakota, the aecia were observed on volunteer bean plants within a canopy of wheat in a field that had contained rusted beans

the previous year. Aecia have been studied in detail in the greenhouse by Andrus (1931) and, more recently, by Groth and Mogen (1978).

When the basidiospores infect bean leaves, it takes about six days at 22-26 °C for a small chlorotic fleck containing the pycnium to develop (Figure 31). About seven days later, the pycnium produces droplets of cloudy white nectar containing spermatia (+ or - mating type) and receptive hyphae (Andrus, 1931; Gold and Mendgen 1984a; Groth and Mogen, 1978). Cross fertilization of a pycnium by pycniospores of the opposite mating type will begin aecium formation, usually on the lower leaf surface (Figure 32), within 9-12 days at 22-26 °C. 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. Eight to ten days later each aeciospore infection produces a uredium with uredospores (Andrus, 1931; Groth and Mogen, 1978).

Subsequent cycles of infection rely solely upon the uredospore stage. These uredospores are capable of germinating to provide infectious hyphae that infect the plant and form new uredia in which new uredospores, and eventually teliospores, will develop (Andrus, 1931). Fusion of dikaryotic nuclei occurs in the teliospores immediately after they are formed (Gold and Mendgen, 1984b). Teliospores are produced by many but not all races (Groth and Mogen, 1978; Groth and Shrum, 1977; Harter and Zaumeyer, 1941; Stavely, 1984b).

Teliospores of *U. appendiculatus* require a dormant period before they will germinate (Gold, 1983; Gold and Mendgen, 1983a; Harter et al., 1935). Gold and Mendgen (1983a) found that teliospores, removed from bean leaves, will germinate after 9-48 months of storage in a refrigerator at 4 °C and 70% relative humidity (r.h.) upon incubation in the proper environment. Storage at the extremes of -18 °C or 20 °C severely reduces germinability (Gold, 1983; Groth and Mogen, 1978). For teliospores exposed to the winter environment in Germany, the dormant period lasts three or four months and maximum germination occurs in seven to eight months. High summer temperatures kill ungerminated teliospores. When teliospores are exposed to favorable conditions following sufficient overwintering outdoors or proper storage indoors, a

three- to four-day lag precedes germination (Gold, 1983; Gold and Mendgen, 1983a; Groth and Mogen, 1978). Optimal laboratory temperature and light intensity for teliospore germination on 2%-distilled-water agar in a petri dish are 18 °C and 17,000 lux (Gold, 1983; Gold and Mendgen, 1984a and 1984c).

Alternating light and dark periods are essential for teliospore germination and release of the basidiospores. Peak basidiospore release occurs after about 7 hours of dark (Gold, 1983; Gold and Mendgen, 1984a). The minimal dark period is three to four hours and the minimal light period is 0.5 hr with 1000 lux. Groth and Mogen (1978) found that prewashing teliospores in cold running water for three hours to eight days had no noticeable effect on teliospore germination. However, some teliospores germinated on water agar three to four weeks after a brief washing. Exposing teliospores to unidentified volatile substances from germinating bean seedlings for 8-12 days stimulates germination in the presence of alternating light and dark periods and also overcomes the requirement for dormancy (Gold, 1983; Gold and Mendgen, 1983b).

The teliospore germinates to produce a basidium in which meiosis occurs and on which haploid basidiospores develop (Gold and Mendgen, 1984b). Mature basidiospores are reniform to ovate-elliptical in shape, smooth surfaced, and measure 9 µm by 16 µm. If supplied with 100% r.h. in darkness, basidiospores begin to germinate on agar or bean leaves in about two hours (Gold, 1983; Gold and Mendgen, 1984a). On a susceptible cultivar, an appressorium is formed, penetration is direct (Gold, 1983), and inter- and intracellular hyphae develop (Gold and Mendgen, 1984c). Pycnial formation is favored by temperatures of 22-26 °C (Gold and Mendgen, 1984c; Groth and Mogen, 1978).

The most commonly observed spore forms are the uredospore (summer or vegetative spore) and teliospore (winter or resting spore). Uredospores are produced in rows within the cinnamon-brown uredium (sorus, pustule) on the upper or lower leaf surface. Uredospores are light brown, one celled, spiny, thin walled, and globoid to ellipsoid in shape. They may have two equatorial or superequatorial pores and measure 20-27 µm by 24-30 µm (Cummins, 1978; Zaumeyer and Thomas, 1957). Near the end of the

growing season, teliospores may form within the pustule in response to changes in light intensity, temperature, moisture, cultivar response, leaf age, or plant maturity. Teliospores have a hyaline pedicel and are blackish brown, one celled, have few to numerous verrucae (wart-like projections), are rarely smooth, thick walled, and are globose to broadly ellipsoid in shape. They may have a hyaline papilla over the pore and measure 24 μm by 30 μm . Some races of *U. appendiculatus* do not produce teliospores (Groth and Mogen, 1978; Groth and Shrum, 1977; Harter and Zaumeyer, 1941; Stavely, 1984a), apparently surviving solely by uredospores.

Although *U. appendiculatus* does not grow in culture, viable spores can be preserved for varying time periods in the laboratory. Uredia and uredospores on dried leaves have been successfully stored at -20 °C for two years (Harter and Zaumeyer, 1941). Dundas (1948) reported that storage at -18 °C for five to seven months could reduce spore germination markedly and induce pathogenic mutations. Uredospore germinability is higher if spores are collected from young, rather than old, uredia and leaves, and if they are produced at 16-21 °C rather than at 24-27 °C (Imhoff et al., 1981). Uredospores can be conveniently stored at -18 °C for about one to three years if removed from uredia, placed in a vial, and dried over a desiccant for a few hours to remove excess moisture before freezing (Bromfield, 1964; Davison and Vaughan, 1963b; Stavely, 1983). Uredospores stored at 7 °C for 26 weeks were still capable of infecting plants in the greenhouse (Harter and Zaumeyer, 1941). Viable spores (40% germination) have been recovered after storage for nearly two years in a special freezer at -60 °C (Schein, 1962) and after storage for at least seven years in liquid nitrogen (Cunningham, 1973). Frozen uredospores of some rust fungi are dormant upon thawing, but not those of *U. appendiculatus* (Bromfield, 1964).

Epidemiology

Infection by *Uromyces appendiculatus* uredospores is favored by prolonged periods (10-18 hours) of moisture, greater than 95% r.h., and moderate temperatures between 17-27 °C (Augustin et al., 1972; González-Avila, 1976; Harter et al., 1935; Schein, 1961a; Zaumeyer and Thomas, 1957). The optimal temperature for uredospore

germination is 16-24 °C. Germination occurs in the first six to eight hours in the presence of moisture (Imhoff et al., 1981; von Alten, 1983). Temperatures greater than 32 °C may kill the fungus (Crispín-Medina et al., 1976; Imhoff et al., 1982; Schein, 1961a and 1961b; Zaumeyer and Thomas, 1957). Temperatures less than 15 °C retard fungal development (Crispín-Medina et al., 1976; Imhoff et al., 1981 and 1982; Zaumeyer and Thomas, 1957). Daylength and light intensity are important factors (Harter and Zaumeyer, 1941). Augustin et al. (1972) reported that infection is favored by incubation in low light intensity ($2 \times 10^{-5} \mu\text{E cm}^{-2} \text{s}^{-1}$) for 18 hours.

The latent period for uredium development (measured as time from inoculation until 50% of the uredia on the adaxial leaf surface open), varies from seven days at 24 °C to nine days at 16 °C constant canopy-level air temperatures (Imhoff et al., 1982). Leaf temperatures in this study were 1-3 °C higher than air temperatures. At 27 °C constant air temperature, lesions do not develop to the sporulation stage.

Uredospore production and release also are influenced by moisture and temperature. Spore production increases when infected plants are exposed to high humidity conditions for limited or prolonged periods (Imhoff et al., 1982; Yarwood, 1961). Cohen and Rotem (1970) reported that sporulation increased when infected plants received at least a 12-hour photoperiod. *Uromyces appendiculatus* can produce one million uredospores per square centimeter on leaves bearing two to 100 uredia per square centimeter (Yarwood, 1961). This spore production occurs in waves, peaking every three to four days. Efficiency of sporulation per unit of leaf area varies inversely with uredium density (Imhoff et al., 1982). Dense infection also reduces uredium size (Harter and Zaumeyer, 1941; Stavely, 1984c). Nasser (1976) reported that the largest number of spores are released during temperate (higher than 21 °C), dry (less than 60% r.h.) days which are preceded by a long dew period or rain the previous night. Uredospores can survive nearly 60 days under field conditions (Zambolim and Chaves, 1974). They contain a water-soluble germination self-inhibitor, methyl cis-3,4 dimethoxycinnamate (Allen, 1972; Macko et al., 1970 and 1976). This inhibitor is removed by washing spores with water and is counteracted by a water-soluble substance in bean leaves (Thomas and

Meiners, 1977), as well as by several defined compounds (Macko et al., 1976).

Uredospores and teliospores can overwinter in bean debris and on wooden supports used for climbing beans (Davison and Vaughan, 1963b). Uredospores can be transported long distances by wind currents. They may provide primary, as well as secondary, inoculum during epidemics in Latin America, Africa, and other places where multiple cropping and/or staggered planting dates provide a continuum of susceptible host tissue during favorable environmental conditions.

Bean rust incidence may be influenced by different cropping systems. For example, in one study, rust incidence was lower when beans were grown in monoculture than in association with maize (GLP, 1976). However, in another study, rust incidence was significantly higher under monoculture than in multiple cropping of beans with maize (Moreno and Mora, 1984). Apparently several factors such as resistance induced by incomplete infection of the beans by pathogens of the companion crop and microclimatic effects, may influence such situations (Allen, 1976; Moreno and Mora, 1984).

Infection by Uredospores

Uromyces appendiculatus uredospores will germinate in the absence of the host if the germination inhibitor is removed by washing with water (Macko et al., 1970). Germination is enhanced by supplying certain divalent cations (Baker et al., 1983a). The appressorium is induced by certain contact stimuli such as the stomatal outer lip (Wynn, 1976) or a scratch on a hydrophobic membrane (Staples et al., 1985). Under artificial conditions, this signal may be replaced by potassium (Staples et al., 1983), glucose and sucrose (Kaminskyj and Day, 1984), or inhibitors of cyclic nucleotide phosphodiesterase (Hoch and Staples, 1984).

The infection process for a uredospore begins as a germ tube develops an appressorium upon physical contact with the edges of a stoma (Pring, 1980; Wynn, 1976). Infection is most efficient on young leaves which are less than 70% of their final size (Groth and

Urs, 1982; Harter and Zaumeyer, 1941; Schein, 1965; Stavelly, 1983; von Alten, 1983). In contrast, on older leaves, fewer appressoria (von Alten, 1983), less necrosis in the necrotic small-uredium reaction (Shaik and Steadman, 1986), and fewer and smaller uredia occur (Kolmer et al., 1984; von Alten, 1983; Zulu and Wheeler, 1982). An infection peg develops from the appressorium and pushes between the guard cells until the fungal cytoplasm is transferred into the substomatal vesicle. The substomatal vesicle contains numerous glyoxysomes, lipid bodies, and glycogen particles (Mendgen, 1973). In most instances, only one infection hypha emerges from the substomatal vesicle. At the tip of the infection hypha, haustorial mother cell development is induced upon contact with a parenchymatous cell (Mendgen, 1978a). The host cell is penetrated, a haustorium differentiates, and nutrients are transferred from the host to the haustorium and intercellular hypha (Mendgen, 1979). Intercellular ramification proceeds throughout the host tissue, eventually forming a young uredium (Pring, 1980; Sziráki et al., 1984).

Host physiology and biochemistry are affected during the infection and sporulation processes. Respiration increases and photosynthesis decreases during infection, especially after the sixth day (Raggi, 1980). Initially, reducing sugars, sucrose, starches, and free amino acids increase in infected tissue. Later, certain amino acids and sugars decrease as sporulation begins (Inman, 1962; Raggi, 1974). Various enzymes such as peroxidase, catecholoxidase, glycolate-oxidase, and glyoxalate reductase, increase their activity during infection (Montalbini and Cappelli, 1973; Raggi, 1974; Sempio et al., 1975). Quinones such as vitamin K, plastoquinones A, C, and O, and ubiquinone, also increase during rust infection and development (Montalbini, 1973). In hypersensitive, necrotic-resistant reactions, deposition of tannins and death of affected host cells occur soon after infection (de la Torre-Almaraz et al., 1985).

Infection reduces the transfer of metabolic byproducts from leaves to roots and developing seeds (Zaki and Durbin, 1965). Stomatal transpiration decreases two days after infection (Duniway and Durbin, 1971b; Sempio et al., 1966) because stomatal opening is inhibited (Duniway and Durbin, 1971b). Transpiration and water vapor loss through the damaged cuticle then increases as infection

proceeds (Duniway and Durbin, 1971a; Sempio et al., 1966). Infected plants become more sensitive to moisture stress as sporulation occurs (Duniway and Durbin, 1971a).

Symptomatology

Uromyces appendiculatus may infect leaves (Figure 33), pods (Figure 34), and, rarely, stems and branches (Figure 35). 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 (Figure 36) about five or six days after inoculation. These spots enlarge to form mature reddish brown uredial pustules which rupture the epidermis about two days later. Sporulation begins and the uredium may attain a diameter of 1-2 mm within 10-12 days after inoculation. Secondary and tertiary uredia may develop around the perimeter of this primary uredium (Harter and Zaumeyer, 1941; Zaumeyer and Thomas, 1957). The entire infection cycle occurs within 10-15 days. Uredospores are released passively from open uredia and scattered by farm implements, insects, animals, and wind currents (Yarwood, 1961; Zaumeyer and Thomas, 1957). Later, black teliospores may form in the uredium. The teliosori become dark brown to black as teliospores replace uredospores (Figure 37). The bean rust fungus is not seed transmitted (Zaumeyer and Thomas, 1957).

Various interactions have been observed between infections by *Uromyces appendiculatus* and other bean pathogens or nonpathogens, usually under controlled conditions. Rust infection may predispose plants to subsequent infection by bean pathogens such as the halo blight bacterium (*Pseudomonas syringae* pv. *phaseolicola* (Burk.) Young et al.), anthracnose fungus (*Colletotrichum lindemuthianum* (Saccardo et Magnus) Briosi et Cavara) (Figure 38), and the root-rot fungus (*Thielaviopsis basicola* (Berkely et Broome) Ferraris), and by nonpathogens such as cucumber powdery mildew (*Sphaerotheca fuliginea*) and tobacco mosaic virus (TMV) (Yarwood, 1969 and 1977).

A high incidence of rust infection may suppress the appearance of halo blight symptoms (Yarwood, 1969). Necrotic rings can occur on the perimeter of rust uredia when rust-infected plants are inoculated

with TMV (Gill, 1965; Wilson, 1958), and possibly other viruses (Figure 39), or with cucumber downy mildew (*Pseudoperonospora cubensis* (Berk. et Curtis) Rostovzev) (Yarwood, 1977). 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 rust and virus are inoculated simultaneously onto plants (Gill, 1965; Wilson, 1958).

Control by Cultural Practices

Cultural controls include crop rotation and removal of old plant debris which may bear viable uredospores and teliospores (Vieira, 1967; Zaumeyer and Thomas, 1957). However, such sanitation measures may have only limited value in controlling rust (Plaut and Berger, 1981). Reduced plant density also may decrease rust incidence. Planting dates may be adjusted in certain production areas to avoid or reduce the incidence of rust infection. Such adjustment will minimize exposure to moderate to cool temperatures and long dew periods during the critical preflowering to flowering stage of plant development.

Biological Control

Biological control is not intentionally used for bean rust, but it may have some potential for the future. The fungus (*Verticillium lecanii* (Zimm.) Viegas) penetrates, invades, and kills uredospores and teliospores, and colonizes uredia of *U. appendiculatus* (Allen, 1982; Grabski and Mendgen, 1986). This pathogen of the rust fungus is easily found in some seasons in the subtropics and tropics (R. T. McMillan, personal communication) and may have a role in the cyclic nature of rust epidemics. It has given 68% control of bean rust in the greenhouse, but gave little control in the field in Germany (Grabski and Mendgen, 1985). *Bacillus subtilis* (Ehrenberg) Cohn, and other *Bacillus* spp. to a lesser degree, gave excellent control of bean rust when applied before inoculation of plants with uredospores in the greenhouse (Baker et al., 1983b). When sprayed on

field-grown beans three times per week, *B. subtilis* caused a 75% reduction in rust severity (Baker et al., 1985).

Results from recent experimental greenhouse and field tests suggest that inoculation of specific bean cultivars with specific races of *U. appendiculatus* to which they are not susceptible will protect against other races to which they are susceptible (M. A. Pastor-Corrales, unpublished data).

Control by Chemicals

Bean rust reduces yields more severely when infection occurs before, rather than after, flowering. Therefore, chemical control is most effective during early plant development (Yoshii and Gálvez, 1975). Bean rust has been controlled by dusting plants every 7-10 days with sulfur at a rate of 25-30 kg/ha (Crispín-Medina et al., 1976; Harter et al., 1935; Zaumeyer and Thomas, 1957), after uredia first appear. However, sulfur can cause leaf burning if applied at higher rates at temperatures above 30 °C.

A seven- to fourteen-day spray schedule is recommended for other preventive chemicals such as chlorothalonil (225 g/100 L), or maneb (4 kg/ha), and/or mancozeb (3-4 kg/ha) (Costa, 1972; Crispín-Medina et al., 1976; Frenhani et al., 1971; González et al., 1977; Hilty and Mullins, 1975; Steadman and Lindgren, 1983; Tompkins et al., 1983; Venette and Jones, 1982a; Vieira, 1967; Wimalajeewa and Thavam, 1973).

Other effective chemicals but which have not yet been approved for use in the United States are bitertanol, triadimefon, and Propiconazole (Mullins and Hilty, 1985; Nieuwoudt, 1984; Venette and Jones, 1982a). Phytotoxicity can be a problem with this last group of fungicides (Mullins and Hilty, 1985).

Uredospores germinate on beans treated with triphenylphosphite, a chemical that is not commercially available as a fungicide. Although the uredospores infect the host plant and form haustorial mother cells, haustoria and uredia do not develop (Rusuku et al., 1984).

Oxycarboxin can be somewhat therapeutic. It is effective when sprayed at 1.8-2.5 kg/ha 20 and 40 days after planting or every two weeks until the end of flowering (Costa, 1972; Crispín-Medina et al., 1976; Frenhani et al., 1971; González et al., 1977; Yoshii and Granada, 1976). Dongo-D. (1971) reported that one preflower application of oxycarboxin (0.9 kg/ha) reduced rust infection by 40% and increased yields by 26%. However, seed treatment with oxycarboxin did not give satisfactory control (Frenhani et al., 1971). Oxycarboxin (4000 ppm) is therapeutic when applied up to three days after inoculation and preventive when applied less than seven days before inoculation (Almeida et al., 1977b and 1977c). Although Issa and de Arruda (1964) concluded that chemical control was not economically practical in parts of Brazil, this is not true in epidemic years in many other areas of the world.

In the absence of rust, yields of beans sprayed with some fungicides may still exceed that of unsprayed beans because of improved micronutrient nutrition or other benefits.

Pathogen Variation

Uromyces appendiculatus is among the most pathogenically variable of all plant pathogens. This variability was first reported by Harter et al. in 1935. The first 20 races were defined in United States in 1941 (Harter and Zaumeyer, 1941) by differential reactions (immune to susceptible) of seven bean cultivars after inoculation with different isolates of the fungus. Host cultivars or lines, the reactions of which are used to differentiate among pathogenic races, are called "differentials."

Variability in *U. appendiculatus* has occurred in many regions of the world, including Australia (Ballantyne, 1978; Ogle and Johnson, 1974), Brazil (Augustin and da Costa, 1971; Carrijo et al., 1980; Coelho and Chaves, 1975; Dias-F. and da Costa, 1968; Junqueira-Netto et al., 1969), Central America (Christen and Echandi, 1967; Vargas-G., 1970, 1971, and 1972), Colombia (Zúñiga de Rodríguez and Victoria-K., 1975), eastern Africa (Howland and Macartney, 1966), Mexico (Crispín-Medina and Dongo-D., 1962), New Zealand (Yen and Brien, 1960), Peru (Guerra and Dongo-D., 1973), Portugal (Rodríguez, 1955), and Taiwan (Yeh, 1983). Intensive

studies have identified at least 80 races in Brazil (Augustin and da Costa, 1971; Carrijo et al., 1980; Coelho and Chaves, 1975; Dias-F. and da Costa, 1968; Junqueira-Netto et al., 1969; Vieira, 1983), 65 in United States (Fisher, 1952; Groth and Shrum, 1977; Harter and Zaumeyer, 1941; Stavely, 1984c; Zúñiga de Rodríguez and Victoria-K., 1975), 31 in Mexico (Crispín-Medina and Dongo-D., 1962), 25 in Australia (Ballantyne, 1978; Ogle and Johnson, 1974), 21 in Jamaica (Shaik, 1985b), 18 in Puerto Rico (López-G., 1976; Ruiz et al., 1982), 15 in Taiwan (Yeh, 1983), and 2-8 in other countries (Christen and Echandi, 1967; Guerra and Dongo-D., 1973; Howland and Macartney, 1966; Rodríguez, 1955; Vargas-G., 1970 and 1971). Two to eight races are frequently found in single field collections from a susceptible cultivar (Ballantyne, 1978; Coelho and Chaves, 1975; Groth and Roelfs, 1982b; Stavely, 1984c). Isolation and increase of spores from a single uredosorus is usually necessary to obtain a pure culture that will give a uniform reaction on each differential. Sometimes several successive such isolations are required to achieve purity.

Most authors have assigned successive numbers to each new race. Thus, races 1-57 are now identified in the first series (Fisher, 1952; Harter and Zaumeyer, 1941; Stavely, 1984c; Zúñiga de Rodríguez and Victoria-K., 1975), of which 55 are from United States and two are from Colombia (Zúñiga de Rodríguez and Victoria-K., 1975). In Brazil, race numbers are preceded by capital letters that symbolize the place of origin. Thus, there are 16 B races from Rio Grande do Sul (Augustin and da Costa, 1971; Dias-F. and da Costa, 1968), 26 FM (Ferrugem, Minas Gerais) races (Junqueira-Netto et al., 1969), and 39 V races from Viçosa (Carrijo et al., 1980; Coelho and Chaves, 1975). In Australia, Ballantyne (1978) assigned lower case letters, a through i, to each of ten differentials and named races by letters of the differentials upon which they were virulent. Her race designations are therefore abbreviated virulence/avirulence formulae. Because of the occurrence of intermediate host reactions with bean rust, an arbitrarily assigned level must be used to separate virulence from avirulence. Otherwise an additional designation has to be used for the intermediate reaction. Differential lines, containing one of each of a number of single resistance genes backcrossed separately into a single recurrent parent to create a nearly isogenic set of differentials, are used for some cereal rust

fungi. However, much more genetic research is needed before such a set can be developed for bean rust.

An International Bean Rust Workshop, held in Puerto Rico in 1983, developed a standard list of 20 differential cultivars (Table 1) and adopted a standard grading scale for rating host reaction (Table 2). Such standardization aimed to overcome the inconsistencies that had developed over the years in the differentials and grading scales that were employed (Stavely et al., 1983). Most race identifications from 1941 to 1983 used most of the original Harter and Zaumeyer (1941) differentials, but often some were deleted and other cultivars added (Augustin and da Costa, 1971; Ballantyne, 1978; Dias-F. and da Costa, 1968; Fisher, 1952; Pereira and Chaves, 1977). A unique set of differential cultivars was used in Mexico (Crispín-Medina and Dongo-D., 1962). Some cultivars used as differentials from 1941 to 1983 were or had become genetically mixed or heterozygous (segregating for reaction to some races). Hence, the new international set of 20, which has now been reduced to 19 (Stavely, 1984c), has been single-plant selected for several generations to obtain homozygosity (Stavely, 1984c; Stavely et al., 1983). Limited quantities of seed of these differentials are available from the authors of this chapter. Most of the other differential cultivars used from 1941 to 1983 are available in the International Bean Rust Nursery, distributed by the Centro Internacional de Agricultura Tropical (CIAT), Colombia (CIAT, 1979; CIAT, 1985).

Table 1. Cultivars adopted at the 1983 International Bean Rust Workshop, USA, as standard differentials for defining races of *Uromyces appendiculatus*.^a

| | |
|----------------------------|-------------------------------|
| U.S. 3 | Mexico 235 |
| California Small White 643 | Mexico 309 |
| Pinto 650 | Brown Beauty |
| Kennedy Wonder 765 | Olathe Pinto |
| Kennedy Wonder 780 | AXS 37 |
| Kennedy Wonder 814 | NEP 2 |
| Golden Gate Wax | Aurora |
| Early Gallatin | 51051 |
| Redlands Pioneer | Compuesto Negro Chimaltenango |
| Ecuador 299 | |

a. Mountaineer White Half Runner was in the original list but has been deleted because of its similarity to Kennedy Wonder 780 (Stavely, 1984c).

SOURCE: Stavely et al., 1983.

Table 2. The uniform bean rust grading scale adopted at the 1983 International Bean Rust Workshop, USA, with the addition of interpretative symbols for degree of resistance or susceptibility suggested by these reaction grades.

| Grade ^a | Definition | Symbol ^b |
|-------------------------------|--|---------------------|
| 1 | Immune, having no visible symptoms | I |
| 2 | Necrotic or chlorotic spots, without sporulation, and less than 0.3 mm in diameter | HR |
| 2+ | Spots, without sporulation, 0.3-1.0 mm diameter | HR |
| 2++ | Spots, without sporulation, 1.0-3.0 mm diameter | HR |
| 2+++ | Spots, without sporulation, greater than 3.0 mm diameter | HR |
| 3 | Uredia less than 0.3 mm diameter | R |
| 4 | Uredia 0.3-0.5 mm diameter | MR |
| 5 | Uredia 0.5-0.8 mm diameter | MS |
| 6 | Uredia larger than 0.8 mm diameter | S |
| 2+, 2++, etc. -3, -4, etc. | Necrotic spot of appropriate size surrounding uredosori of appropriate size | R, MR ^c |

- a. When several reaction grades are present, they are recorded in order of predominance, the most prevalent being listed first and least prevalent, last. Intensity is recorded separately, using the modified Cobb Scale (Stavely, 1985).
- b. These symbols have been used at Beltsville for at least 15 years (J.P. Meiners and J.R. Stavely, unpublished data) and the categories resemble Ballantyne's categories (Ballantyne, 1978). Their precise definitions are: I = immune; HR = hypersensitive or highly resistant; R = resistant, reactions having any of the grades 2 with grade 3 present or predominant with some grade 4; MR = moderately resistant, grade 4 predominant and no grade 5 uredia; MS = moderately susceptible, uredia larger than grade 4, but none larger than grade 5; S = susceptible, grade 6 uredia. Another category is VS = very susceptible, grade 6 uredia predominant.
- c. This reaction first described by Harter and Zaumeyer (1941) occurs on Kentucky Wonder 780 with many races. It is characterized by a uredium in the center of a necrotic spot. Whether R, MR, or other is determined by the size of uredium as described in footnote b.

SOURCE: Stavely et al., 1983.

By using appropriate inoculation methods (Ballantyne, 1978; Coelho and Chaves, 1975; Davison and Vaughan, 1964; Stavely, 1983 and 1984b) and grading scales, it is possible to determine whether an isolate is already a described race or unique by comparing it with reported races (Stavely, 1984c). When making comparisons with earlier race descriptions, care must be taken since several changes were made in the grading scale from 1941 to 1983

(Ballantyne, 1978; Crispín-Medina and Dongo-D., 1962; Davison and Vaughan, 1963a; Harter and Zaumeyer, 1941). However, these scales have been well enough defined to often permit separation of new isolates from previously described races (Stavely, 1984a).

Control by Plant Resistance

Resistance to bean rust is expressed in many ways (Figure 40). Resistant reactions range from immunity, through various consistent types of hypersensitive, nonsporulating, or sporulating necrotic reactions (necrotic spot with a small, central uredium), to very small, small, or intermediate uredia (Table 2) (Ballantyne, 1978; Harter and Zaumeyer, 1941; Stavely et al., 1983). Different types of cell reactions also occur within the leaf (Mendgen, 1978b). Smaller uredia produce fewer uredospores and, if sufficiently small, have no effect on host yield (Pastor-Corrales and Correa-Victoria, 1983). Genetic studies require use of pathogenically uniform, single uredium isolates (cultures) of defined races (Ballantyne, 1978; Stavely, 1984b and 1984c).

Genetic studies of resistance have shown that reaction grade is controlled by single dominant genes and that there are many such genes in beans (Ballantyne, 1978; Christ and Groth, 1982a; de Carvalho et al., 1978; Grafton et al., 1985; Kolmer and Groth, 1984; Meiners, 1981; Stavely, 1984a and 1984b; Stavely and Grafton, 1985; Zaumeyer and Harter, 1941). *P. vulgaris* has only $n=11$ chromosomes and *U. appendiculatus*, if it is similar to cereal rust fungus, *Puccinia graminis* (McGinnis, 1953), has only about $n=6$ chromosomes. The gene-for-gene relationship has been shown to occur in the *U. appendiculatus*-*P. vulgaris* host-pathogen interaction (Christ and Groth, 1982a and 1982b). Monogenic, dominant resistance-genes have been identified that are effective against multiple pathogen races (Kardin and Groth, 1985; Stavely and Grafton, 1985). They occur in linkage groups (complex loci) in which there is a single gene for each of many races (Stavely, 1984a and 1984b; Stavely and Grafton, 1985). Some genes are epistatic to other single resistance genes (Kolmer and Groth, 1984; Stavely 1984a and 1984b).

In pedigree and backcross breeding resistance is screened by using several appropriate individual races simultaneously on single plants (Stavely, 1983). In this way, it is possible to "pyramid" two, three, or more such genes or complex loci that are effective against multiple races. Thus, it is possible, by identifying and carefully deploying resistance genes, to develop cultivars with several known genes for resistance to available races and significantly reduce the likelihood of resistance-breaking races developing (Coyne and Schuster, 1975; Schafer and Roelfs, 1985). If virulence and avirulence genes be tightly linked in the pathogen, then resistance may be stabilized by combining as few as two appropriate host resistance genes or linkage groups of such genes (Van der Plank, 1968). However, this is not yet a useful hypothesis, because among the avirulence/virulence genes that have so far been identified in rust fungi no such linkages have been found. A multiline, in which each component line contains a different broadly effective gene or linkage group backcrossed into the same recurrent parent, may also stabilize resistance (Coyne and Schuster, 1975; Van der Plank, 1968).

Should virulence in basidiospores and uredospores be under independent genetic control in *U. appendiculatus*, pathogen variability may be reduced and resistance better stabilized by separately breeding for resistance to the basidiospore stage (Groth and Roelfs, 1982a). However, the same pathogen genes appear to condition virulence or avirulence in both basidiospores and uredospores (Kolmer et al., 1984).

Nearly 70 years ago, a reduced intensity of uredia per unit of leaf area and decreased spore production were recognized as potentially useful forms of resistance to bean rust (Fromme and Wingard, 1921). Of course, if a line has a necrotic, nonsporulating reaction or immunity to a portion of the races present in an area, the uredium intensity will also be reduced. So, a critical first step in assessing any suspected reduced intensity-type resistance is to determine the line's reaction to each race. Some cultivars such as Royal Red Kidney (Groth and Urs, 1982) and Jamaica Red (Shaik, 1985a), have a kind of resistance in which uredial intensity has been reduced with all races tested thus far. This is called "low receptivity" and can be assessed under carefully measured and controlled inoculum con-

centration, host growth rate, and leaf age (Groth and Urs, 1982). Stomatal density is directly proportional to the number of uredia that develop. However, the sparseness of stomata is apparently not the only cause of low receptivity (Groth and Urs, 1982; Shaik, 1985a). Recent evidence suggests that increased leaf-hair density also reduces the number of uredia by preventing a portion of the uredospores from reaching the leaf surface (Shaik, 1985a). Analysis of the genetic control of stomatal and leaf-hair density may reveal a polygenic mechanism and it may be possible to enhance low receptivity through intensive, careful selection for transgressive segregants.

A longer latent period from infection to sporulation, an important component of so-called "slow rusting," may not be associated with the reduced uredium-intensity type of resistance (Shaik, 1985a), although it is associated with monogenic, small-uredium resistance (Stavely, 1984b). Certain Cuban cultivars are apparently late or slow rusting (González-Avila, 1974).

Vieira (1972) has suggested that in Brazil, where diverse cultivars have been developed locally, there is substantial "horizontal" resistance (equally effective against all races). Eight Brazilian bean lines varied in incubation period, latent period, infection frequency, infection type, and infection intensity against different isolates of *U. appendiculatus*. This suggests that so-called "vertical" (probably single) resistance genes play at least some role in expression of these reactions (Menten and Bergamin-Filho, 1981).

There are several other potentially useful types of resistance to bean rust. Germplasm may vary in length of dew or drying periods and increase in resistance with plant development (Ballantyne, 1974; Berger, 1977). Some cultivars are more heavily infected in lower than upper foliage (Canessa-Mora and Vargas-G., 1977). Rodríguez-Medina (1976) reported that Mexico 309, which has a series of linked monogenic factors for resistance to many races (Stavely, 1984b), is susceptible to race CR-29, but yields as well as cultivars resistant to this race. Tolerance, in which fully susceptible type uredosori occur, but yield is not reduced, would be a most desirable character if methods were found to identify it in the process of developing new cultivars.

Alexander et al. (1985) measured virulence changes in a polymorphic *U. appendiculatus* population over five asexual generations. He found that changes in virulence may be independent of pathogen exposure to host resistance. *U. appendiculatus* frequently carries virulence at a level much higher than the minimum needed for pathogenicity.

Many bean cultivars and lines have been bred for resistance to rust (CIAT, 1979 and 1985; Stavely and Steinke, 1985; Wood and Keenan, 1982; Zaumeyer and Thomas, 1957); for example, such popular cultivars as Olathe, Fleetwood, Aurora, and the CIAT cultivars BAT 48, 73, 76, 93, 308, and 520. Although these cultivars are not resistant to all races of rust, they comprise a significant factor in reducing yield losses from rust.

Table 3. The most rust-resistant cultivars in the International Bean Rust Nurseries from 1975 to 1984; and the percentage of their reactions, according to reaction class across all locations and years.

| Cultivar tested in years | Reaction and percentage of occurrence ^a | | | |
|---------------------------|--|------|------|-------------------|
| | I | HR | R | MR-S |
| 1975-1984 | | | | |
| Redlands Greenleaf B | 19.6 | 45.8 | 30.8 | 3.7 |
| Redlands Greenleaf C | 16.1 | 40.4 | 39.4 | 4.0 |
| Cocacho | 15.9 | 44.7 | 33.0 | 6.4 |
| Mexico 309 | 40.2 | 41.1 | 12.1 | 6.5 |
| Cuilapa 72-1 | 29.9 | 37.4 | 25.2 | 7.5 ^b |
| Ecuador 299 | 18.7 | 37.4 | 35.5 | 8.4 ^b |
| Mexico 235 | 26.8 | 35.0 | 28.9 | 9.3 ^b |
| Turrialba 4 | 29.6 | 27.8 | 31.5 | 11.1 |
| Puerto Rico 5 | 23.4 | 38.3 | 26.2 | 12.1 |
| Compuesto Chimaltenango 3 | 22.3 | 50.5 | 22.3 | 11.6 |
| Compuesto Chimaltenango 2 | 31.1 | 32.0 | 22.3 | 14.6 ^b |
| Redlands Autumn Crop | 10.3 | 39.2 | 35.0 | 15.5 |
| Turrialba 1 | 17.9 | 29.2 | 34.9 | 17.9 ^b |
| 1976-1984 | | | | |
| Redlands Pioneer | 13.0 | 54.3 | 29.3 | 3.3 |
| Mexico 6 | 11.9 | 34.5 | 41.7 | 11.9 ^b |

a. Reactions are described in Table 2, footnote b. Percentages are calculated by using only those locations where readings were obtained.

b. Uredia larger than 0.5 mm at one or more locations in 1981 to 1984.

SOURCE: CIAT, 1985.

The International Bean Rust Nursery was established in 1974 and is coordinated by CIAT pathologists (G. E. Gálvez, H. F. Schwartz, and M. A. Pastor-Corrales). It has tested differential cultivars and resistant germplasm worldwide since 1975 (CIAT, 1979 and 1985; Meiners, 1974). No cultivar or line has yet been resistant for all years at all locations in this nursery. The most resistant of the standard entries are listed in Table 3. The most resistant CIAT lines have been the BAT cultivars listed above, which have been tested continuously since 1979. As more is learned about pathogen virulence, pathogen race dynamics, and genetics of host resistance, the potential for developing effective deployment strategies for resistances will lead to more effective control of bean rust.

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Chapter 8

WEB BLIGHT

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Introduction

Web blight is caused by the fungus *Rhizoctonia solani* Kühn—the sclerotial, or asexual, stage of the basidiomycete fungus *Thanatephorus cucumeris* (Frank) Donk. *Rhizoctonia solani* is a soil-borne fungus that is widely distributed throughout the world. Both the sclerotial and basidial stages can initiate the disease, although they cause different symptoms. In most areas of Latin America where blight occurs, the sclerotial stage is significant for the initiation and epidemiology of the disease (Galindo, 1982, Galindo et al., 1982c, 1983a, and 1983b).

Rhizoctonia solani is a pathogen of a large number of host species including bean, beet (Abawi and Martin, 1985), cabbage, carrot, cucumber, eggplant, melon, soybean (O'Neill et al., 1977), tobacco, tomato, watermelon, and many uncultivated plants (Daniels, 1963; Vargas-G., 1973). It also causes a diversity of diseases such as seed decay, root-and-hypocotyl rot, and foliar blight. Although diverse in host range and disease symptomatology, the isolates demonstrate specialization according to their mode of attack. Even though morphologically similar, some isolates cause aerial infection such as web blight of beans, while others attack only roots and hypocotyls (see Chapter 6, p. 107-114).

Web blight is a very important bean-production problem in the humid lowland tropics of Latin America and the Caribbean, where warm to high temperatures and abundant rainfall prevail. The disease also occurs, and can cause severe damage, in middle altitude areas (1200-1600 m.a.s.l.), particularly during rainy weather and

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high humidity. Under field conditions web blight can occur at any stage of the bean-crop cycle and cause severe blight, resulting in rapid defoliation and often complete crop failure (Crispín-Medina and Gallegos, 1963; Galindo, 1982). In the Guanacaste region of northern Costa Rica, a web blight epidemic caused up to 90% reduction of bean yields in 1980 (Se perdió la cosecha de frijol veranero en Guanacaste, 1980).

In Latin America, web blight occurs in the warm, humid, southern, bean-producing areas of Mexico (Crispín-Medina and Gallegos, 1963), all countries of Central America and the Caribbean (Echandi, 1966; Galindo, 1982; Manzano, 1973), and in South America in the Amazon region of Peru and Brazil (Deslandes, 1944; Müller, 1934), the coffee zone of Colombia, and the northwestern region of Argentina (Costa, 1972; Ploper, 1981). Web blight has also been reported in United States, Japan, Philippines, Burma, and Sri Lanka (Weber, 1939; Zaumeyer and Thomas, 1957). The lack of reports of web blight occurrence from African countries suggests that this disease is, currently, of minor importance (CIAT, 1981), although it has been reported from Kenya (Mukunya, 1974) and Malawi (Msuku and Edje, 1982).

Common names used for web blight in Latin America in Spanish include "mustia," "mustia hilachosa," "telaraña," "chasparría," "Rhizoctonia del follaje," and "pringue." In Portuguese, common names include "mela," "mela do feijoeiro," "murcha da teia micélica," and "podridão das vagens."

Etiology

The asexual stage of the web blight fungus, *Rhizoctonia solani*, is distributed worldwide (Baker et al., 1967; Hawn and Vanterpool, 1953; Papavizas and Davey, 1962). This pathogen was originally described as *R. microsclerotia* Matz, although this designation is no longer accepted (Parmeter et al., 1967; Zaumeyer and Thomas, 1957). The current accepted designation for the basidial stage is *Thanatephorus cucumeris* (Flentje et al., 1963b).

Isolates of *R. solani* are highly variable in cultural characteristics, response to environmental changes, and pathogenicity. However,

they can be classified into different groups, according to the anastomosis grouping (AG) concept: that is, hyphal fusion occurs only between isolates of the same AG. Earlier researchers showed that the majority of *R. solani* isolates fall into one of four anastomosis groups: AG-1, AG-2, AG-3, and AG-4 (Parmeter et al., 1969). Three more groups, AG-5, AG-6, and AGB1 have since been discovered and AG-2 was recently subdivided into AG2-1 and AG2-2 (Kuninaga et al., 1978).

Hyphal anastomosis groups are not, according to most authors, host specific, although some tendencies are evident (Bolkan and Ribeiro, 1985; Parmeter et al., 1967). Except for AGB1, the anastomosis groups are genetically unique and differ in pathological and cultural characteristics (Bolkan and Ribeiro, 1985; Kuninaga et al., 1978).

Galindo et al. (1982b) characterized 71 isolates of *R. solani* that were obtained from naturally infected bean leaves in different bean-growing areas of Costa Rica. All isolates were pathogenic to leaf and hypocotyl tissues of the bean cultivar Mexico 27, but varied significantly in virulence which was positively correlated to growth rate in culture. Twenty-six isolates belonged to AG-1, 38 to AG-2, and 9 did not anastomose with any of the four AG-4 testers used.

Similarly, Bolkan and Ribeiro (1985) reported that two Brazilian isolates of *R. solani*, obtained from kidney bean leaves, belonged to AG-1, while seven isolates from kidney bean hypocotyls belonged to AG-4. Most of the *R. solani* isolates associated with bean hypocotyls and soils in New York belonged to AG-4. However, some isolates belonged to AG-1 and AG-2, but not to AG-3 (Galindo et al., 1982a). All six aerial isolates of *R. solani* associated with web blight in Colombia were AG-1 (Galindo et al., 1982a).

In addition, the *R. solani* isolates associated with web blight are characteristically fast growing, produce abundant sclerotia, and are intolerant of carbon dioxide. Those associated with seed decay and root-and-hypocotyl rot are characteristically fast growing, produce fewer sclerotia, and are more tolerant of carbon dioxide (Flentje and Stretton, 1964). Parmeter et al. (1967) established that *Rhizoctonia* isolates which possess multimediate hyphae have *Thanatephorus cucumeris* as their perfect stage and those which possess binuclear hyphae have *Ceratobasidium* as the perfect stage.

The following description of *Rhizoctonia solani* is from Holliday (1980). Colonies on potato dextrose agar (PDA) are at first colorless, rapidly becoming brown. Aerial mycelium is variable, giving a felted or mealy surface on which long, sparsely branched hyphae are frequently present. Some isolates show diurnal zonation. Sclerotia develop as a crust, radiating out from the inoculum center or scattered over the colony surface. Hyphae are usually 5-12 μm wide and up to 250 μm long, with cells at the advancing edge of a colony. Branches form near the distal end of cells, are constricted at the point of origin, and are septate above this constriction. Phase contrast microscopy shows cells are multinucleate (2-25, mostly 4-8), with conspicuous dolipore septa. An older mycelium shows large variation in hyphal dimensions and has shorter cells because of the formation of secondary septa. The branching angle is nearly 90° and branches may arise at various points along the cell length. Some hyphae differentiate into swollen moniliform cells which are 30 μm or more in width. Small (0.2-0.5 mm diameter), immature, superficial, white sclerotia also form and become brown to dark brown, rough, and subglobose with maturity (Weber, 1939). Isolates grown in the laboratory on PDA may differ for growth rate, sclerotial production (Flentje and Stretton, 1964), mycelium color, amount of aerial mycelium, saprophytic behavior, and enzyme production (Papavizas, 1964 and 1965; Papavizas and Ayers, 1965).

The basidial stage, *Thanatephorus cucumeris*, was first discovered in beans in the USA by Weber (1939) who reported that mycelia and sclerotia from both asexual and sexual sources were indistinguishable. Basidial fructifications appear whitish and form on top of a hymenium which is a thin sheet or collar commonly found on stems or leaves just above the soil surface or on soil particles. It is discontinuous and composed of barrel-shaped subcylindrical basidia, 10-25 μm long x 16-19 μm wide, arranged in imperfect cymes or racemes. The short basidia bear stout, slightly divergent sterigmata, usually four in number, but can have two to seven per basidium. They are 5.5-36.5 μm long and occasionally have adventitious septa. Hyaline basidiospores, produced on the sterigmata, are oblong to broadly ellipsoid, unilaterally flattened, prominently apiculate, smooth, and thin walled. They measure 6-14 μm x 4-8 μm and germinate by repetition.

The fungus grows rapidly in continuous, indirect, or intermittent light. Within 24-36 hours it can cover the surface of a 9-cm petri dish containing artificial media incubated at 26-29 °C. Sclerotia form in culture but differ from those produced on host plants which are brown to dark brown, and more irregular in form and size (as large as 1 cm in diameter), and more or less flattened (Weber, 1939). Heterokaryosis occurs in *T. cucumeris* and may alter its ability to form sclerotia on minimal media or to form isolate pathogenicity and variants (Flentje and Saksensa, 1957; Flentje et al., 1963a and 1967; Gálvez and Cardona-Alvarez, 1960; McKenzie et al., 1969; Meyer and Parmeter, 1968).

The perfect stage of the web blight fungus can be induced in vitro (Flentje, 1956; Stretton et al., 1964; Tu and Kimbrough, 1975) with 12-16 hours of light (Flentje et al., 1963b; Stretton et al., 1964; Weber, 1939; Whitney, 1964), adequate aeration (Whitney, 1964), 20-30 °C, and 40%-60% relative humidity (Stretton et al., 1964; Weber, 1939). Self-sterile mutants frequently appear in progenies of basidiospores (Stretton et al., 1967; Whitney, 1964). Isolates of *Rhizoctonia solani* vary for their cultural characteristics and ability to fruit on artificial media or sterilized soil (Houston, 1945; Olsen et al., 1967; Stretton et al., 1964). For example, pathogenic isolates of *T. cucumeris* fruit only on sterilized soil, while nonpathogenic isolates fruit on either substrate (Stretton et al., 1964).

Epidemiology

Web blight epidemics are favored by rainy weather, high (30 °C) to moderate (20 °C) air temperature (average 25-26 °C), high to moderate soil temperature, and high relative humidity of at least 80% (Galindo, 1982; Galindo et al., 1983b; Weber, 1939; Zaumeyer and Thomas, 1957). The main sources of inocula that can initiate infection are sclerotia and mycelium fragments, either free in the soil or present on colonized debris. Bean plants are inoculated by the web blight pathogen when raindrops splash soil particles, infested with sclerotia or mycelium, onto plants (Galindo et al., 1983b; Prabhu et al., 1982).

Basidiospores can also cause infection (Echandi, 1965). However, in most locations with abundant rain and endemic web blight,

basidiospores do not contribute significantly to epidemic development, particularly when lesions from basidiospore infection appear late in the crop cycle (Galindo et al., 1983b). Infected bean seed can disseminate the pathogen over long distances, introduce it into new fields, or act as a source of primary inoculum. When rain-splashed sclerotia and mycelium are the main source of inoculum, initial symptoms of web blight always appear on primary leaves two weeks after planting.

The mycelium of the fungus first grows on the soil particles splashed onto bean leaves and then advances to adjacent healthy tissue, causing primary or initial infections. Trifoliolate leaves are usually infected by hyphal strands growing from infected primary leaves, but can also be infected by rain-splashed soil. Infected leaves rapidly become covered by small sclerotia of the fungus. New sclerotia also form, beneath the canopy, on fallen leaves and the soil surface within 24 hours. After trifoliolate leaves are infected, plant-to-plant infection occurs through direct hyphal growth from previously infected leaves (Galindo et al., 1983b).

Basidiospores are dispersed during the night (Echandi, 1965) and remain viable for only a few hours. Sclerotia can remain viable in soil for several years and can survive as vegetative mycelium within plant residue (Weber, 1939).

Symptomatology

Web blight symptoms initiated by rain-splashed sclerotia or mycelium fragments differ from those elicited by basidiospores. Sclerotia germinate during periods of favorable environmental conditions by producing hyphae, a few mm in length, that branch profusely until they reach host tissue. An infection cushion then develops and penetration occurs directly or through stomata (Dodman et al., 1968; Weber, 1939). Subepidermal hyphae develop inter- and intracellularly. Lesions first appear on the primary leaves as small necrotic spots (5-10 mm in diameter) with brown centers and olive-green margins. These lesions resemble hot-water scalds. Under favorable environmental conditions, high humidity, and warm temperature, they progress very rapidly but appear irregular and somewhat zonate (Figure 41). Under dry conditions, their

development stops. Often these lesions coalesce and affect the entire leaf. Infected leaves rapidly become covered by small sclerotia and mycelium.

The light-brown superficial hyphae spread in a fan-shaped manner on either leaf surface. Hyphae may grow rapidly over healthy leaves, petioles, flowers, and pods (Figure 42), eventually killing plant parts or covering the entire plant with a web of mycelium (Figure 43). Small brown sclerotia (Figure 44) form three to six days after infection (Galindo, 1982; Weber, 1939; Zaumeyer and Thomas, 1957). The many lesions produced by basidiospores are distinct, small, necrotic, circular, and measure 2-3 mm in diameter (Figure 45). They are light brown or brick red with a lighter center. Under humid and rainy conditions, these round spots fall from the leaf surface, resulting in a symptom known as "cock's eye." These lesions usually do not enlarge much, nor coalesce to form large lesions, and seldom cause defoliation. Pod lesions caused by sclerotia, mycelium, or basidiospores are similar to foliage lesions. Pod lesions initiated by basidiospores are also small, circular, and have light-brown centers surrounded by a reddish brown darker border.

Bean pods may become infected during the grain-filling stage. Young pod infections appear as light-brown, irregular-shaped lesions which frequently coalesce and kill the pod.

Seeds can become infected in the endosperm and radicular end of the embryo and on the seed-coat surface (Baker, 1947; Cardoso et al., 1980; Leach and Pierpoint, 1956; Le Clerg, 1953).

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 nonhosts such as tobacco, maize, and grasses. A most effective cultural practice is mulching. Mulch forms a barrier and impedes the splashing of pathogen propagules from the soil to plant tissues. Under experimentation, effective mulches are rice husks, maize leaves, sugarcane leaves, or standing weeds killed by herbicides 15 days after planting (Galindo et al., 1982c and 1983b;

Rosado-May, 1982; Rosado-May and García-Espinosa, 1985). Examples of preemergent herbicides used to kill weeds for mulching are paraquat or glyphosate (1 kg/ha) (Galindo et al., 1983a). Postemergent herbicides such as fluazifop-butyl (1 kg/ha) and bentazone (0.75 kg/ha), can be used for broad-leaved weeds. Obando (1983) and Sancho (1984) established that, for an effective, integrated, control of the pathogen, preemergent applications of paraquat, pendimethalin, and glyphosate can be used in association with foliar applications of the fungicide benomyl.

Small subsistence bean farmers in Costa Rica and Nicaragua rely upon a similar practice known as “frijol tapado” (covered beans). This practice consists of broadcasting bean seeds into plots with established weeds and cutting the weeds down to cover the seeds as a plant mulch. By using herbicides, a standing weed mulch can be created (Galindo et al., 1982c). Indeterminate cultivars grow through the mulch and eventually cover it, effectively preventing new weed growth and conserving soil moisture. In addition, the mulch prevents the splashing of infected soil. This practice is effective, even in areas where the climate is optimal for web blight development (Galindo, 1982; Galindo et al., 1982a, 1982b, and 1982c, 1983a, and 1983b). However, mulches may create more favorable conditions for slug infestation and resulting crop loss in some production regions.

Where farmers have more resources, beans should be planted in spaced furrows (Corrêa, 1982; Weber, 1939, Zaumeyer and Thomas, 1957) which will maximize air circulation and improve micro-climatic conditions. Intercropping beans in relay or in association with maize will also reduce disease severity (Msuku and Edje, 1982; Rosado-May, 1982).

Control by Chemicals

Benomyl (0.25-0.5 kg/ha) helps manage the pathogen when it is applied at first-symptom appearance and then every 15 days (Cardoso, 1980; Cardoso and de Oliveira, 1982; Manzano, 1973; Oliveira et al., 1983). The chemicals protect plant foliage from infection by inoculum from nearby infested soil. Fentin acetate (0.16 kg/ha) or fentin hydroxide (0.20 kg/ha) applied after benomyl

(Cardoso and de Oliveira, 1982), gives good control. Thiophanate-methyl (0.5 kg/ha), carbendazim (0.5-1.0 kg/ha), and captafol (1.0-3.5 kg/ha) (CIAT, 1975; Manzano, 1973) are also useful. The use of systemic fungicides is important where rains prevail. However, expense may limit their use, even though recent work has shown that two or three applications are sufficient to control mild infections (Villalobos-Pacheco, 1985).

Control by Plant Resistance

Cultivars differ in their reaction to the web blight pathogen under field conditions. Susceptible cultivars exude chemicals which stimulate the formation of infection cushions whereas resistant or tolerant cultivars do not exude these chemicals (Flentje et al., 1963a). Although various cultivars have low levels of resistance to the web blight pathogen (Manzano, 1973; Weber, 1939), there are no reports of cultivars with high resistance or immunity. The Centro Internacional de Agricultura Tropical (CIAT), in collaboration with the national bean programs of Colombia and Costa Rica, has identified bean cultivars with some resistance to web blight. These are: Turrialba 1, Porrillo 70, Porrillo Sintético, S-630-B, and Talamanca (Mora and Gálvez, 1979). Crosses with these cultivars have produced progenies exhibiting resistance such as Negro Huasteco 81, Huetar, HT 7716, and HT 7719, which are superior to the resistant parents.

Integrated Control

The most practical approach to manage this very serious and damaging disease is by using an integrated management strategy. Such strategy is based upon cultural practices, complemented by judicious use of chemicals, and, where possible, use of resistant cultivars. This involves using clean seed, eliminating pathogen-infested crop debris at harvest, wide row-spacing (Corrêa, 1982; Weber, 1939; Zaumeyer and Thomas, 1957), planting resistant cultivars with erect architecture to permit greater air circulation, mulching and minimum tillage, applying fungicides, and rotating with nonhost crops such as cereals and vegetables. Such practices can offer an economic, efficient, and practical control of web blight.

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Chapter 9

WHITE MOLD

H. F. Schwartz and J. R. Steadman*

Introduction

The white mold fungus, *Sclerotinia sclerotiorum* (Lib.) de Bary, is distributed worldwide. It is most important in the temperate zones of the northern and southern hemispheres. However, it is also a problem in areas with tropical or arid climates, especially during cool seasons or under favorable microclimatic conditions (Reichert and Palti, 1967). The fungus has therefore been reported in the common bean and vegetable fields of Argentina (Hauman-Merck, 1915), Brazil (Shands et al., 1964), Mexico (Crispín-Medina and Campos-Avila, 1976), Peru (Christen, 1969), Colombia, Venezuela (Pons et al., 1979), other areas of Latin America (Echandi, 1976), Asia, Africa (Allen, 1983), Europe, Australia, and North America.

Sclerotinia sclerotiorum is pathogenic to a wide range of host plants. Purdy (1979) listed 64 families as being hosts to *S. sclerotiorum*, Schwartz listed 399 hosts (unconfirmed reports in some instances), and the world literature mentions 374 species of 237 genera. Diseases caused by *S. sclerotiorum* include blossom end rot, stem rot, watery soft rot, pink rot, cottony rot, drop, flower rot, fruit rot, root rot, timber rot, and white mold. Hosts are as diverse as ornamentals, tree fruits, vegetables, oil-seed crops, and legumes.

Purdy presented an extensive list of crop production losses which underscored the impact that this fungus can have on crop production. For example, snap bean production in the seventies was reduced greatly in New York State (Abawi and Grogan, 1975; Natti, 1971). Zaumeyer and Thomas (1957) reported bean losses of 30% in Virginia during 1916. Yield losses averaged 30% in Nebraska during

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1970-73, although in individual fields losses were as high as 92% (Kerr et al., 1978). Yield losses in Canada have varied from 15%-60%, depending upon the cultivar infected (Beversdorf and Hume, 1981).

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 (Kohn, 1979). Because of taxonomic nomenclature considerations, a new name, *Whetzelinia sclerotiorum* (Lib.) Korf et al., was proposed (Korf and Dumont, 1972) and appeared in the literature for a brief period. However, it is now correct to use *S. sclerotiorum* (Kohn, 1979).

The fungus produces large (one to several millimeters in diameter or length), black, and irregularly-shaped resting structures called sclerotia (Figure 46). The sclerotia germinate to form hyphae or mycelium. A normal sclerotium has an outer black rind that is three cells deep, a two- to four-cell deep cortex, and a large inner medulla from which hyphae develop during germination (Huang, 1983). A sclerotium, after undergoing a conditioning period, can also germinate carpogenically to produce one or more apothecia (Figure 47). The apothecia represent the sexual stage of the fungus. They average 3 mm in diameter and protrude 3-6 mm above the soil surface (Ramsey, 1925).

Each apothecium contains thousands of cylindrically shaped asci, each of which contains eight ascospores (Walker, 1969). An ascus measures 7-10 μm in diameter by 112-156 μm in length (Coe, 1944; Kosasih and Willetts, 1975; Ramsey, 1925). Over a period of days an apothecium may discharge more than 2 million ascospores (Schwartz and Steadman, 1978). The ascospores are ovoid and vary 4-10 μm in width and 9-16 μm in length (Coe, 1944; Kosasih and Willetts, 1975; Ramsey, 1925; Walker, 1969). *Sclerotinia sclerotiorum* can produce asexual spores, called microconidia (3-4 μm

diameter), during any stage of its life cycle. However, they do not function during sexual fertilization or in host infection (Kosasih and Willetts, 1975; Ramsey, 1925).

Epidemiology

Fields used repeatedly for bean production, even in short crop rotations, will often contain many sclerotia. Sclerotia formed on or within diseased tissue may be dislodged onto the soil surface by wind or harvesting operations. Subsequent land preparation redistributes them within the soil profile and over the field (Cook et al., 1975). Sclerotia also can be distributed by furrow irrigation within fields (Schwartz and Steadman, 1978) and by reuse of irrigation runoff water between fields (Brown and Butler, 1936; Steadman et al., 1975). They can survive in sandy loam soils for at least three years (Cook et al., 1975) and are capable of producing secondary sclerotia (Adams, 1975; Cook et al., 1975; Williams and Western, 1965).

The minimal quantity of soil-borne sclerotia needed to induce significant plant infection has not been intensively studied. However, populations of 0.2 sclerotia per 30 cm² (Abawi and Grogan, 1975) and less than 1-10 sclerotia per kg of soil (Adams and Ayers, 1979; Lloyd, 1975; Schwartz and Steadman, 1978) are known to exist in fields planted to snap, Great Northern, and Pinto beans. Schwartz and Steadman (1978) determined that 1 sclerotium per 5 kg soil was sufficient to cause 46% disease severity in Nebraska. Suzui and Kobayashi (1972b) reported that 3.2 sclerotia per m² caused 60%-95% plant infection in a kidney bean field in Japan. Sclerotia are persistent and the availability of primary inoculum from outside bean fields apparently explains why there is no correlation between white-mold incidence and severity, and previous cropping history (Abawi and Grogan, 1979). Herbicide practices may also influence carpogenic germination in host and nonhost fields (Radke and Grau, 1986): some herbicides enhance, while others inhibit, germination.

Apothecia formation (carpogenic germination) is greatest after 10-14 days, at 15-18 °C, with soil moisture at 50% of field capacity (wet soil) (J. M. Duniway, G. S. Abawi, and J. R. Steadman,

unpublished data), or in a soil with a matrix potential of -80 to -240 mb (-8 to -24 kPa) (Abawi and Grogan, 1979). Carpogenic germination occurs in fields of common bean, maize, sugar beet (Schwartz and Steadman, 1978), snap bean (Abawi and Grogan, 1975), cauliflower, tomato (Letham et al., 1976), lettuce (Hawthorne, 1976; Newton and Sequeira, 1972), and table beet. It occurs in grassland (Suzui and Kobayashi, 1972b) and in lemon, orange (Smith, 1916), and other fruit orchards (Abawi and Grogan, 1975). In a sandy loam soil, studied by Schwartz and Steadman (1978), many sclerotia germinated and formed apothecia in common bean (11-14 apothecia per m²) and sugar beet (7-11 apothecia per m²) 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 furrow of the irrigated row.

Most ascospores discharged by a germinated sclerotium are deposited close to the release point (Suzui and Kobayashi, 1972a). However, Williams and Stelfox (1979) reported crop infection in fields 150 m to as far as several kilometers away (Abawi and Grogan, 1979; Bardin, 1951; Burke et al., 1957). Mature asci forcibly discharge their ascospores for more than 1 cm into the air, after being exposed to a slight decrease in moisture tension and change in relative humidity. (Abawi and Grogan, 1979). Ascospores have been trapped between 30 and 147 cm above the soil surface in barley and rapeseed fields, respectively. This suggests that crops differ in their ability to restrict spore movement (Williams and Stelfox, 1979). The bean canopy traps a large percentage of ascospores, saturating the available infection sites and promoting a high local infection (Steadman, 1983).

A mucilaginous material that can cement the spores to host tissue is discharged along with ascospores (Abawi and Grogan, 1979). In one study, more than 30% of blossoms, randomly collected from a bean field containing apothecia, exhibited evidence of *Sclerotinia sclerotiorum* after plating on acidified potato dextrose agar (PDA) (Muckel and Steadman, 1981). Honeybees may have disseminated the fungus propagules to blossoms. The fungus clearly survives periods of unfavorable microclimatic conditions. Ascospores on bean leaves remain viable for 12 days in the field. Mycelium, found

in or on dry colonized bean blossoms, remains viable for 25 days in the laboratory (Abawi and Grogan, 1975) and 33 days in the field (Muckel and Steadman, 1981). Viable ascospores (90% germination) have been stored frozen (-19°C) for 24 months on Millipore membrane (type HA, $0.45\text{ }\mu\text{m}$) filters placed over calcium chloride. They also keep in the refrigerator at 2°C (Hunter et al., 1982b). Ascospores, found on shaded bean leaves at 12-15 cm above soil level and within a dense canopy, averaged 20% greater survival than on topmost leaves. Ultraviolet light, high relative humidity, and high temperatures are detrimental to ascospore survival (Caesar and Pearson, 1983).

Sclerotinia sclerotiorum is a cosmopolitan fungus and occurs in regions where conditions are favorable such as moisture and low temperature. (Reichert and Palti, 1967). Brooks (1940) and Moore (1955) report that white-mold epidemics occur when mean temperatures are less than 21°C and humidity or moisture levels are high. About 48-72 hours of continuous wetness on leaves within the canopy or on dry colonized blossoms are required for infection by ascospores. However, only 16-24 hours of wetness are required to infect moist blossoms (Abawi and Grogan, 1979). Secondary spread of the fungus occurs at 18°C and 100% relative humidity (Starr et al., 1953; van den Berg and Lentz, 1968). Abawi and Grogan (1975) suggest that a film of surface moisture is necessary if the fungus is to develop and spread.

The rate of spread is also influenced by temperature. Gupta (1963) reported that coriander plants infected with *S. sclerotiorum* died within 4-10 days at $19-24^{\circ}\text{C}$, but did not die at 29°C —apparently because the plants outgrew the fungus. Microclimatic conditions may be as important as macroclimatic conditions for infection and pathogen development. For example, irrigation practices significantly alter microclimatic parameters, often encouraging the development of *S. sclerotiorum*. Frequent furrow irrigation reduces day air and leaf temperatures by $3-4^{\circ}\text{C}$ and soil temperatures by 10°C , and increases soil moisture content by 10% (Weiss et al., 1980a and 1980b).

Symptomatology

Sclerotinia sclerotiorum infects bean plants by colonizing senescent and dead plant organs such as blossoms (Figure 48), cotyledons, seeds, leaves, or injured plant tissue (Abawi and Grogan, 1975; Abawi et al., 1975a; Cook et al., 1975; McLean, 1958; Natti, 1971; Purdy and Bardin, 1953). Blodgett (1946) observed cotyledonary rot on bean seedlings which developed from mycelia- or sclerotia-infested seed lots planted in the greenhouse. Verdugo-G. and Fucikovsky-Zak (1980) report that *S. sclerotiorum* was transmitted by bean seed. However, Steadman (1975) showed that infected seeds were completely colonized by the fungus before germination and/or plant emergence. No plant infection arose from apparently healthy seed even though they came from infected seed lots. Colonization of senescent tissue usually results from germinated ascospores, but mycelial colonization can occur directly from sclerotia (Abawi and Grogan, 1975; Cook et al., 1975).

After colonizing a senescent plant organ, the fungus enters the host by mechanically disrupting the cuticle. It uses a dome-shaped infection cushion which had developed from an appressorium. Large vesicles form between the cuticle and epidermal layers and infection hyphae develop intercellularly. Hyphae branch from the infection hyphae and ramify inter- and intra-cellularly (Lumsden and Dow, 1973; Purdy, 1958), causing a watery soft rot. The fungus produces many enzymes and other products, including endo- and exopolysaccharuronase, pectin methyl esterase (Lumsden, 1976), and oxalic acid (Maxwell and Lumsden, 1970), all of which are important to pathogenesis.

Symptoms of infection first appear as a water-soaked lesion (Figure 49), followed by a white moldy growth on the affected organ (Figure 50). 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 (Figure 51) (Blodgett, 1946; Zaumeyer and Thomas, 1957). Although many bean plant types such as great northern, pinto, and kidney, exhibit this characteristic bleaching, in some navies and small whites it is more difficult to distinguish white-mold infection. Plant wilting may also be seen within the plant canopy after plant stems and/or vines are infected (Figure 52).

Biological Control

Many soil microorganisms associate with sclerotia of *S. sclerotiorum* and may cause sclerotia to degrade or not germinate. Such organisms include the fungi *Coniothyrium minitans* Campbell, *Trichoderma* sp., *Aspergillus* sp., *Penicillium* sp., *Fusarium* sp., *Mucor* sp. (Huang and Hoes, 1976; Merriman, 1976; Rai and Saxena, 1975; Trutmann et al., 1982; Turner and Tribe, 1976), *Sporidesmium sclerotivorum* Uecker et al. (Ayers and Adams, 1979), and *Teratosperma oligocladium* Uecker et al. (Ayers and Adams, 1981). *Sclerotinia sclerotiorum* also is inhibited by various antibiotic substances produced by the fungus *Gibberella baccata* (Wallroth) Saccardo (Guerillot-Vinet et al., 1950), actinomycetes such as *Streptomyces* sp. (Leben and Keitt, 1948; Lindenfelser et al., 1958), and bacteria (Darpoux and Faivre-Amiot, 1949). The fungi *Coniothyrium minitans* (Trutmann et al., 1982) and *Gliocladium virens* Miller et al. (Tu, 1980) inhibit sclerotia formation and germination myceliogenically and carpogenically.

However, none of these biological agents has been used effectively in controlling *S. sclerotiorum* incidence or in protecting bean plants from infection under field conditions. Nevertheless, research is continuing in Australia, Canada, and United States on developing some of these mycoparasites as biological control agents.

Ginger rhizome peelings have inhibited ascospore germination on chickpea (*Cicer arietinum* L.) and indicate a new approach to the control of *Sclerotinia sclerotiorum* (Singh and Singh, 1984).

Control by Cultural Practices

For controlling the pathogen, Zaumeyer and Thomas (1957) recommend cultural practices such as crop rotation, flooding, reduced seeding rates, fewer irrigations, and destruction of those bean-cull screenings which contain sclerotia. Similar recommendations have been made in Brazil (Costa, 1972). Deep plowing also has been advocated (Merriman, 1976), and disputed (Brooks, 1940; Gabrielson et al., 1971; Partyka and Mai, 1962), as a control measure. Crop rotation is not likely to be effective because sclerotia survive in soil and tillage operations, ensuring the presence of

sclerotia at or near the soil surface (Cook et al., 1975). However, this practice does help reduce the number of sclerotia within the field and hence controls yield-loss potential. Flooding has limitations and may not be practical in many situations. Planting density depends on the cultivar and its growth potential. For example, reduced planting rates for vigorous vine types can result in large dense canopies which would promote white-mold development.

Irrigation frequency can influence disease incidence on cultivars with indeterminate plant growth habits and dense plant canopies (Weiss et al., 1980a and 1980b). Growers should not irrigate if white-mold infection is prevalent within their bean fields (Steadman et al., 1976) or, at least, should reduce late-season irrigations (Weiss et al., 1980b). Reuse of irrigation water should be avoided or the water treated to remove sclerotial and/or ascospore contamination (Steadman et al., 1975.)

A survey of bean fields in Canada revealed that infected and uninfected crops grew on soils with a pH of 7.5 and 7.0, respectively. However, the authors did not determine the nature nor the applicability of this association (Haas and Bolwyn, 1972). Heavy fertilizer rates are not recommended because they increase disease incidence (Andersen, 1951) by, presumably, stimulating canopy density. Planting beans after alfalfa, similarly, can stimulate canopy density and lead to severe white-mold incidence.

Chemical Control

Applying benomyl, DCNA or dicloran, dichlone, PCNB, or thia-bendazole around early- to mid-bloom controls *S. sclerotiorum* infection on snap and common beans, particularly under dryland conditions (Beckman and Parsons, 1965; Campbell, 1956; Costa, 1972; Forster, 1980; Gabrielson et al., 1971; Lloyd, 1975; McMillan, 1973; Natti, 1971; Verdugo-G. and Fucikovsky-Zak, 1980). However, Partyka and Mai (1958) 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 common bean cultivars grown under irrigation (Steadman, 1979).

Sporadic results also have occurred in Canada, California, Colorado (Schwartz et al., 1987b), Montana, Washington and Wyoming. Other fungicides such as vinclozolin, procymidone (Vulsteke and Meeus, 1982), and iprodione, are being tested for their effectiveness in controlling white mold. Timing of the chemical application and thoroughness of coverage are critical to successful control (Steadman, 1983). Because of the expense of fungicide applications, forecasting systems such as that proposed for snap bean by Hunter et al. (1984), need to be developed.

Radke and Grau (1986) report that herbicides can influence carpogenic germination in the laboratory. Trifluralin, pendimethalin, metribuzin, simazine, and atrazine stimulate the germination of sclerotia and increase the number of stipites and apothecia per sclerotium. Although simazine and atrazine enhance stipes formation, the stipites and apothecia that formed were malformed. Linuron and DNBP inhibit germination and apothecial development, and alachlor causes variable responses.

Control by Plant Resistance

An association between canopy development and white-mold incidence and disease severity has been observed in various crops, including peanuts (Coffelt and Porter, 1982) and beans. Row spacing, growth habit, plant density, daylength, temperature, and fertilizer application can influence canopy development and therefore disease incidence, especially with indeterminate bean types (Blodgett, 1946; Coyne et al., 1974, 1977, and 1978; Gaxiola-L., 1977; Haas and Bolwyn, 1972; Natti, 1971; Schwartz et al., 1978 and 1987b; Steadman et al., 1973; Zaumeyer and Thomas, 1957). An open canopy facilitates air circulation and light penetration within the canopy. As a result, moist leaf and soil surfaces dry more rapidly, reducing or preventing infection. Some indeterminate cultivars produce a distinct tunnel above the open furrow as opposed to a dense and intertwined canopy. This architectural trait helps prevent contact between foliage and pods with moist debris on the soil surface (Fuller et al., 1984c). Selecting for disease avoidance, however, can be accomplished on a single-plant or single-row basis only if intergenotypic interference is reduced (Fuller et al., 1984b).

An example of the interaction between row spacing and cultivar is with the cultivar Aurora. Because of its upright, open growth habit it escapes infection when it is planted at a within-row spacing of 4-5 cm (Coyne et al., 1977). However, when it is planted 30.5 cm apart within the row it sprawls and is more severely infected. Orienting bean rows parallel with the prevailing wind direction may also reduce disease incidence by providing improved air circulation and better light penetration (Haas and Bolwyn, 1972).

Resistance to *S. sclerotiorum* in the field has been observed in *Phaseolus vulgaris* germplasm (Anderson et al., 1974; Blodgett, 1946; de Bary, 1887; McClintock, 1916; Ramsey, 1925; Yerkes, 1955). Resistant materials include Black Turtle Soup (BTS-3), Black Valentine, Tacaragua, Cacahuatate, Ex Rico 23, and P.I. 169787 (Anderson et al., 1974; Beversdorf and Hume, 1981; Fuller et al., 1984a; Schwartz et al., 1987a). Disease incidence and rate of disease development are slower in Ex Rico 23 in Canada under field conditions (Beversdorf and Hume, 1981; Tu and Beversdorf, 1982). However, plants with field resistance and entries which escaped disease can be infected in controlled environment chambers where they are exposed to colonized tissue for 18-36 hours under high humidity (Hunter et al., 1981 and 1982a). This test is known as the limited term inoculation test and is sensitive. It is useful for screening germplasm for partial (field) or higher degrees of resistance such as identified in P.I. 415965, P.I. 169787, P.I. 204717, and P.I. 417603 (*Phaseolus coccineus*) (Hunter et al., 1982a).

Resistance also has been identified in *P. coccineus* (Adams et al., 1973; Hunter et al., 1981; Steadman et al., 1974; Verdugo-G. and Fucikovsky-Zak, 1980) and *P. coccineus* x *P. vulgaris* hybrids (Abawi et al., 1975b). This type of physiological resistance is necessary in areas such as New York State, where bush beans are grown and escape or where plant architecture plays a minor role in resistance.

The resistance of *P. vulgaris* lines such as Tacaragua, BTS-3, A 51, A 55, 83 VEF MXA 222, Rabia de Gato, and Porrillo Sintético, is quantitatively inherited and due primarily to additive gene action (Fuller et al., 1984a). Repeated selection (recurrent selection schemes) should accumulate genes for resistance and help identify

the highest level of resistance possible (Dickson et al., 1982; Fuller et al., 1984a; Lyons et al., 1985).

Attempts are being made to develop stable resistance by using a plant structure which maximizes disease avoidance and also has physiological resistance to *S. sclerotiorum* (Coyne et al., 1977; Hunter et al., 1982a; Schwartz et al., 1987b). Such cultivars should be part of an integrated control program that includes the use of fungicides, disease forecasting, and practice of appropriate cultural practices.

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Chapter 10

ADDITIONAL FUNGAL PATHOGENS

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Introduction

Beans are exposed to many pathogenic fungi at various stages of their plant development. Infection may occur on seedlings and mature plants throughout the growing season or postharvest. Some of the more prevalent and economically important plant pathogenic fungi have already been described in this book. Unfortunately, very little information exists concerning the epidemiology and control of many other fungi considered to be of minor importance to bean production. However, in the tropics many of these pathogens can become very important in specific regions of bean production. Likewise, many of today's minor pathogens may become tomorrow's major pathogens as agricultural practices change. This chapter briefly describes some of these fungi and lists others.

Alternaria Leaf-and-Pod Spot

Alternaria leaf-and-pod spot is caused by various fungi of the *Alternaria* species, including *A. alternata* (Fr.) Keissler (syn. *A. tenuis* Nees); *A. brassicae* f. *phaseoli* Brun.; *A. fasciculata* (Cke. et Ell.) L. R. Jones et Grout; *A. tenuissima* (Nees ex Fries) Wiltshire; *A. macrospora* Zimm.; and *A. brassicicola* (Schw.) Wiltsh. (Abawi et al., 1977; Allen, 1983; Bera, 1983; Russell and Brown, 1977; Saad and Hagedorn, 1969; Weber, 1973; Zaumeyer and Thomas, 1957). These fungi are reported from East Africa (Angus, 1962; Ebbels and Allen, 1979), Brazil (Gomes and Dhingra, 1983; Shands et al., 1964), Costa Rica (González, 1973), Colombia (Ellis et al., 1976a),

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Chile, Mexico, Venezuela (Wellman, 1977), England (Russell and Brown, 1977), Canada (Tu, 1982), and United States (Abawi et al., 1977; Saad and Hagedorn, 1969; Zaumeyer and Thomas, 1957). Severe epidemics may cause premature defoliation but yield losses are not usually significant. However, snap bean losses of 12% occurred in New York since infected pods were unacceptable for processing (Abawi et al., 1977).

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

Alternaria brassicae (Berkeley) Saccardo produces greenish brown, septate, and branched hyphae with erect conidiophores in culture. Conidia are smooth, long 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 by 9-33 μ m (Weber, 1973).

Alternaria spp. are wound parasites. They usually form lesions only on older or senescent plant tissue during periods of high humidity that last for three or four days (Abawi et al., 1977; Saad and Hagedorn, 1969) and are relatively cool (16-20 °C). However, *A. tenuis* can also penetrate the leaf directly or through stomata (Saad and Hagedorn, 1969). *A. alternata* can also enter through stomata (O'Donnell and Dickinson, 1980). *A. tenuis* produces a toxin (tentoxin) in culture which induces plant chlorosis when applied to roots (Durbin et al., 1973; Saad et al., 1970). However, the fungus does not produce detectable quantities of tentoxin during natural infection of leaves or pods.

Leaf symptoms appears as small, gray to reddish brown, irregular-shaped spots or flecks which may be water-soaked and surrounded by a darker brown border. These lesions gradually enlarge and develop as concentric rings that become brittle and fall out, producing a shot-hole appearance (Figure 53). 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 on the plant or reduce plant vigor. The fungus also can blemish leaves (Figure 54) and pods (Figure 55) by producing a brown discoloration on the surface; it also damages

developing seeds (Abawi et al., 1977; Gomes and Dhingra, 1981; González, 1973; Russell and Brown, 1977; Saad and Hagedorn, 1969; Tu, 1982; Weber, 1973; Zaumeyer and Thomas, 1957). The reddish to dark brown or black flecks may coalesce and produce streaks on infected pods (Abawi et al., 1977). *Alternaria* spp. can be seed-borne (Ellis et al., 1976a; Tu, 1982). Seed transmission can be high if infection occurs near maturity (Gomes and Dhingra, 1981).

Control measures are seldom necessary but consist of wider plant and row spacing, use of chemicals, development of resistant cultivars (Abawi et al., 1977), and crop rotation. Chemical control uses chlorothalonil (1200 µg a.i./L) (Abawi et al., 1977), thiophanate (2 g/L), and zineb (2.4 g/L). Iprodione (2.4 g a.i./L) reduces disease severity and increases yield in the susceptible cultivar Fleetwoodd in Canada (Tu, 1983). *A. alternata* may be insensitive to, or favored by, spray applications of benomyl (Abawi et al., 1977; Russell and Brown, 1977; Tu, 1983) and chlorothalonil (Tu, 1982 and 1983). Tu (1983) urges that effective products like iprodione must be used judiciously to avoid or delay the development of resistant *Alternaria* strains.

Ascochyta Blight

Ascochyta blight of beans, also known as ascochyta leaf-and-pod spot, is a fungal disease of economic importance only in regions with cool humid conditions such as those found at elevations above 1000 m in the Andean region of South America. The disease is therefore of economic importance in most of the middle- (1200-1600 m.a.s.l.) to high-altitude (1600-2600 m.a.s.l.) bean-growing regions of Colombia, Ecuador, and Peru. It is also important in the high-altitude valleys of Guatemala (Echandi, 1976). The disease has also been reported in Brazil (Costa, 1972), Venezuela (Wellman, 1977), Costa Rica (Echandi, 1976), United States, and other regions of the world (Zaumeyer and Thomas, 1957).

In Africa, ascochyta blight is also important in the high-altitude, humid, cool, bean-growing valleys of Burundi, Rwanda, Zaire, Kenya, and Zambia (CIAT, 1981).

The taxonomy and etiology of the causal agent of the ascochyta blight pathogen is not well understood. However, the fungus causing ascochyta blight is usually recognized as *Ascochyta bolts-hauseri* Saccardo. However, according to Boerema, it should be called *Phoma exigua* var. *diversispora* (Bub.) Boerema (Boerema, 1982). It is a serious pathogen, causing ascochyta blight of beans in Western Europe and Africa (Boerema et al., 1981; Stoetzer et al., 1984). *Phoma exigua* var. *exigua* Desmazieres (Boerema et al., 1981), formerly known as *Ascochyta phaseolorum* Saccardo, has been also reported as a less important pathogen associated with ascochyta blight.

Yield losses greater than 40% were measured in Colombia under moderate disease pressure (Schwartz et al., 1981b). *Ascochyta pisi* Libert occurs in Venezuela (Wellman, 1977). The common names frequently used for ascochyta blight (leaf spot) in Latin America are "ascochyta" and "mancha de ascochyta."

Phoma exigua isolates produce hyaline, septate, submerged mycelium in culture. Spores are usually two-celled and 20 by 5 μm in size (Zaumeyer and Thomas, 1957). Sporulation and germination are greatest at 21 °C, while mycelial growth is greatest at 24 °C. The fungus is inactivated by temperatures above 30 °C (Namekata and Figueiredo, 1975). The fungus produces pycnidia which measure 60-150 μm in diameter (Zaumeyer and Thomas, 1957). *Phoma exigua* var. *diversispora* pycnidia measure 160 by 120 μm and conidia measure 6.8 by 2.7 μm . Most conidia are one-celled (Boerema et al., 1981).

Infection by *Phoma exigua* var. *diversispora* is favored by high humidity, continuous rains accompanied by winds, and cool to moderate temperatures (Boerema et al., 1981; Echandi, 1976). Symptoms first appear on leaves. They are black, concentric, zonate lesions (Figure 56), 1-3 cm in diameter, and may later contain small black pycnidia (Boerema et al., 1981). These dark to black lesions also may appear on the peduncle, petiole (Figure 57), node, and pod (Figure 58), and can cause stem girdle and plant death. The fungus may also spread systemically throughout the plant. Premature leaf drop may occur during severe epidemics (Weber, 1973) and the fungus is seed-borne (Boerema et al., 1981).

Control measures are crop rotation, wide plant spacing, planting clean seed, chemical treatment of seed, and foliar application of sulfur fungicides (Schwartz et al., 1981b; Teranishi, 1970). Chemical control measures include benomyl (0.55 g/L), zineb (2.4 g/L), chlorothalonil (2.24 kg/ha), and carbendazim (M. A. Pastor-Corrales, personal communication). Common bean germplasm is being screened to identify sources of resistance which may contribute to disease control. Although there are germplasm differences in reaction to the ascochyta blight pathogen, most *P. vulgaris* L. accessions so far evaluated are either susceptible or have low levels of resistance. However, high levels of resistance and immunity are present in accessions of *P. coccineus* L., particularly in the subspecies *polyanthus* such as Guate 1076 (G 35182), and in interspecific hybrids obtained by crossing these two species (CIAT, 1987).

Ashy Stem Blight

Ashy stem blight of bean is caused by the fungus *Macrophomina phaseolina* (Tassi) Goid. (Dhingra and Sinclair, 1977; Weber, 1973; Zaumeyer and Thomas, 1957). The fungus is a warm-temperature pathogen of the beans *Phaseolus vulgaris* and *P. lunatus* L., soybean, maize, sorghum, and many other crops (Watanabe et al., 1970). It occurs mainly in Latin America: Brazil (Díaz-Polanco and Casanova, 1966; Shands et al., 1964), Mexico, Cuba, Chile (M. A. Pastor-Corrales, personal communication), Peru, Colombia, Venezuela, Central America (Wellman, 1977); but also in other parts of the world such as Kenya, Zambia, and Egypt (CIAT, 1981; Stoetzer et al., 1984; Zaumeyer and Thomas, 1957). Ashy stem blight is more prevalent and damaging to beans that are exposed to drought and warm temperatures. Losses of 65% have occurred in beans grown in the United States (Zaumeyer and Thomas, 1957). However, no loss estimates are available for Latin America.

Common names frequently used for ashy stem blight (charcoal rot) in Latin America include “macrofomina,” “pudrición gris de la raíz,” “pudrición carbonosa de la raíz,” “tizón cenizo del tallo,” “podredumbre carbonosa,” and “podridão cinzenta do caule.”

The fungus produces black globose pycnidia that contain large, colorless, 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 μm long and 5-8 μm wide and are produced on nearly straight conidiophores which may have a truncate tip and measure 12-20 μm in width and 6-25 μm in length (Zaumeyer and Thomas, 1957). 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 (Figure 59). The fungus produces black, sunken cankers which have a sharp margin and often contain concentric rings. The plant's growing tip may be killed or the stem broken where it is weakened by the canker. Infection may continue 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 (Figure 60) (Dhingra and Sinclair, 1977; Weber, 1973; Zaumeyer and Thomas, 1957).

A few days after infection the fungus produces small, smooth, black sclerotia (50-150 μm in diameter) in infected tissue (Figure 61) 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 (Figure 62). The fungus may produce air-borne conidia which cause leaf spots on mature plants (Díaz-Polanco and Casanova, 1966). *Macrophomina phaseolina* can be seed-borne (Ellis et al., 1976a; Weber, 1973; Zaumeyer and Thomas, 1957).

Control measures are planting clean seed, treating seed with chemicals such as Ceresan and benomyl (Abawi and Pastor-Corrales, n.d.b), and sanitation or deep-plowing plant debris containing pycnidia and sclerotia. Organic soil amendments (carbon to nitrogen ratio of 10:20) and high soil temperatures (30 °C) and moisture (60% moisture-holding capacity) may reduce sclerotia levels (Dhingra and Sinclair, 1977). Sclerotia survival in soil can be reduced further by applying benomyl (1 kg/ha) and thiophanate-methyl (Ilyas et al., 1976), by fumigating the soil with methyl bromide and chloropicrin (Watanabe et al., 1970), and by using herbicides such as Eptam, dinoseb, alachlor, fluorodifen, and

fluometuron (Filho and Dhingra, 1980). There are resistant cultivars such as Negrito (Dhingra and Sinclair, 1977; Vieira, 1983; Zaumeyer and Thomas, 1957). More recently, bean lines BAT 85, BAT 477, San Cristóbal 83, G 5059, and BAT 336 have shown resistance under field and greenhouse conditions (Abawi and Pastor-Corrales, n.d.a; Pastor-Corrales and Abawi, 1988).

Cercospora Leaf Blotch

Cercospora leaf spot and blotch of beans are caused by *Cercospora canescens* Ellis Martin and *C. cruenta* Saccardo (syn. *Pseudocercospora cruenta* (Sacc.) Deighton). The latter fungus is the imperfect state of *Mycosphaerella cruenta* Latham. *C. phaseoli* Dearness et Bartholomew and *C. caracallae* (Speg.) Chupp also cause leaf spots of bean (Skiles and Cardona-Alvarez, 1959; Weber, 1973; Zaumeyer and Thomas, 1957). These fungi, primarily *C. canescens* and *C. cruenta*, occur in Brazil (Shands et al., 1964), Colombia (Skiles and Cardona-Alvarez, 1959), Puerto Rico, Trinidad, Jamaica, Venezuela, Argentina (Wellman, 1977), and United States (Zaumeyer and Thomas, 1957). Yield losses are slight in United States but can be serious in the Philippines on mung bean (*Vigna radiata* (L.) Wilczek). There are no reports of serious losses in Latin America, although defoliation has occurred in Colombia (Orozco-Sarria, 1958).

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 shaped, curved, or straight. *C. cruenta* spores measure 50-150 μm in length and 6-9 μm in width, while *C. canescens* spores measure 50-100 μm in length and 3-4.5 μm in width (Zaumeyer and Thomas, 1957).

Symptoms include brown or rust-colored lesions (Figure 63) which may coalesce and vary in shape (circular to angular) and size (2-10 mm). *C. canescens* produces irregularly shaped, light brown lesions with a gray center in leaves, pods, stems, and branches (Orozco-Sarria, 1958). These lesions may have a grayish center with

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 trifoliolates. Blemishes may occur on stems and pods and the fungi can become seed-borne (Dhingra and Asmus, 1983; Orozco-Sarria, 1958; Weber, 1973; Zaumeyer and Thomas, 1957). A pink to purple discoloration occurred on bean seed inoculated with *Cercospora kikuchii* isolated from infected soybeans (Kilpatrick and Johnson, 1956).

Control measures are seldom necessary. However, copper fungicides applied to foliage are effective (Zaumeyer and Thomas, 1957). Orozco-Sarria (1958) 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 the fungus *Chaetoseptoria wellmanii* Stevenson. It occurs in Mexico, Panama, Central America, Venezuela, and the West Indies (Wellman, 1977). The fungus has a wide host range within the Leguminosae. It may cause complete defoliation of beans and 50% yield loss in regions of high humidity and moderate temperatures (Wellman, 1972). The common name frequently used for chaetoseptoria leaf spot in Latin America is "mancha redonda."

Chaetoseptoria wellmanii produces medium to large, circular lesions (Figure 64) which may have a gray surface with black pycnidia in the center and may be surrounded by a dark border (Wellman, 1972). Infection is more common in primary leaves in Mexico and defoliation also may occur. The pathogen can be seed-borne (Crispín-Medina et al., 1976).

The main control measure is to develop resistant or tolerant cultivars (Crispín-Medina et al., 1976). Benomyl (0.55 g/L) may be a sufficient chemical control.

Diaporthe Pod Blight

Diaporthe pod blight of beans is caused by the fungus *Diaporthe phaseolorum* (Cooke et Ellis) Saccardo (Weber, 1973). *D. arctii* (Lasch) Nits. is pathogenic to bean stems (Zaumeyer and Thomas, 1957). *D. phaseolorum* has a conidial stage known as *Phomopsis subcircinata* Ell. et Ev. (USDA, 1960). No estimates of its prevalence or importance are currently available, although Wellman (1977) reports that it is a weak parasite in Honduras. Common names frequently used for diaporthe pod blight in Latin America are "añublo de la vaina" and "tizón de la vaina."

Diaporthe phaseolorum produces hyaline, oblong ascospores measuring 10-12 by 2-4 μm and having one septation. The ascospores are produced inside black perithecia, which measure 300 μm in diameter. Pycnidiospores are produced in the black pycnidia and the oval spores measure 6-9 by 2-5 μm (Weber, 1973).

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 the lesions. Pod infections may then occur and pods become discolored with pycnidia present in the lesions (Weber, 1973). The fungus can be seed-borne in soybeans and in beans (Ellis et al., 1976a).

Control measures include crop rotation, planting clean seed, and use of foliar fungicides such as benomyl (0.55 g/L). Resistant soybean cultivars have been developed. If available and practical as a control measure, common bean germplasm should be screened to identify sources of resistance.

Downy Mildew

Downy mildew, a fungal bean disease that usually occurs under low temperatures, is caused by *Phytophthora nicotianae* Breda de Haan var. *parasitica* (Dastur) Waterh. (Holliday, 1980; Zaumeyer and Thomas, 1957) and *P. phaseoli* Thaxter (Crispín-Medina et al., 1976). The pathogen has caused yield losses in Mexico, Puerto Rico (Crispín-Medina et al., 1976; Zaumeyer and Thomas, 1957), El Salvador, Costa Rica (M. A. Pastor-Corrales, personal communication), Venezuela, Peru, and Panama (Wellman, 1977). Common

names frequently used for downy mildew in Latin America are “mildeu veloso” and “míldio veloso.”

Symptoms first appear on the leaves and 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, bordered by reddish brown, are visible on green pods, especially those in contact with the soil surface (Figure 65). If low temperatures and high humidity persist the entire pod may be infected, shrivel, and dry up (Crispín-Medina et al., 1976).

Control measures are included crop rotation for three years; use of chemicals such as zineb, maneb, nabam, or captan (Crispín-Medina et al., 1976); production of pods free from soil contact (Zaumeyer and Thomas, 1957); and development of cultivars with an upright plant architecture and open canopy to improve air circulation. If available and practical as a control measure, common bean germplasm should also be screened to identify sources of resistance.

Entyloma Leaf Smut

Entyloma leaf smut of beans is caused by a species of the fungus *Entyloma* (Schieber and Zentmeyer, 1971; Vakili, 1972; Wellman, 1972). *Entyloma* leaf smut occurs in the bean-production regions of Costa Rica, Dominican Republic, El Salvador, Guatemala, Honduras, and Nicaragua (Schieber and Zentmeyer, 1971; Vakili, 1972 and 1978). *Entyloma petuniae* Speg. occurs on beans in Argentina (Wellman, 1977). 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 teleutospores (Wellman, 1972). Lesions are round or oval and first appear as water-soaked but become gray-brown in color on the upper leaf surface and gray-blue on the lower leaf surface (Figure 66). Lesions may coalesce and be delimited by leaf veinlets (Schieber and Zentmeyer, 1971). Infection usually occurs only on the primary leaves or first and second sets of trifoliolate

leaves. Severe foliage infection of 40%-60% may occur (Vakili, 1972).

The fungus can be controlled chemically by either treating seed with carboxin (5 g/kg seed) or using a foliar spray of benomyl (0.55 g/L). If available and practical as a control measure, common bean germplasm should be screened to identify sources of resistance.

Floury Leaf Spot

Floury leaf spot of beans is caused by the fungus *Mycovellosiella phaseoli* (Drummond) Deighton (200) (syn. *Ramularia phaseoli* (Drummond) Deighton) (Weber, 1973). The fungus occurs in eastern and central Africa (Rwanda, Burundi, and Zaire), Malaysia, Papua New Guinea (Holliday, 1980), Brazil (Minas Gerais and Espírito Santo), Nicaragua, Colombia, Venezuela (Cardona-Alvarez and Renaud, 1967; Cardona-Alvarez and Skiles, 1958; Vieira, 1983; Vieira and Shands, 1965b; Vieira et al., 1977), Ecuador, Honduras, Panama, Guatemala, and Dominican Republic (Schieber, 1969; Wellman, 1977). The disease occurs at elevations between 1500 and 2000 meters in Colombia and Guatemala (Cardona-Alvarez and Skiles, 1958; Schieber, 1969). No estimates of yield losses caused by it are available.

The common names frequently used for floury leaf spot in Latin America are “mancha harinosa” and “mancha farinhosa.”

Ramularia phaseoli produces hyaline, usually nonseptate, conidia which are oval to lemon shaped and measure 7-18 by 4-6 μm (Weber, 1973). It produces a white growth (1-1.5 cm in diameter) of conidiospores and conidia on the lower surface of leaves (Figure 67), in contrast to powdery mildew (*Erysiphe polygoni* DC.) which usually infects only the upper leaf surface. Chlorosis usually occurs on the upper leaf surface and corresponds to the lower leaf lesions. Spots are angular at first. Infection begins on older leaves and then progresses to new foliage. Severe infections can cause considerable premature defoliation (Cardona-Alvarez and Skiles, 1958; Weber, 1973), although this is not usual.

Chemical control is obtained by applying benomyl (0.55 g/L) or thiophanate (2 g/L). If available and practical as a control measure,

common bean germplasm should be screened to identify sources of resistance.

Gray Leaf Spot

Gray leaf spot of beans is caused by *Cercospora vanderysti* P. Henn.—now reclassified as *C. castellanii* Matta et Belliard—and occurs in Venezuela, Central America (Wellman, 1977), Brazil (Minas Gerais and Espírito Santo) (Shands et al., 1964; Vieira, 1983; Vieira and Shands, 1965a; Vieira et al., 1977), and Colombia, usually at elevations greater than 1000 m where high moisture and low to moderate temperatures persist (Skiles and Cardona-Alvarez, 1959). No estimates of yield losses are available and the pathogens are apparently confined to tropical America. 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 (Figure 68). 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 later forms on the lower leaf surface (Figure 69) and is diagnostic of pathogen (Skiles and Cardona-Alvarez, 1959; Vieira, 1983). Severe infections (Figure 70) 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 the use of resistant cultivars such as Rico Pardo 896, Cornell 49-242, Carioca, and Caraota 260 (Asmus, 1981).

Gray Mold

Gray mold of beans is caused by *Botrytis cinerea* Pers. ex Fries which has as its perfect stage *Botryotinia fuckeliana* (de Bary) Fuckel (Polach and Abawi, 1975). The fungus can be a serious problem during periods of high moisture and low temperatures in various regions of United States and Europe (Johnson and

Powelson, 1983b; Polach and Abawi, 1975; Zaumeyer and Thomas, 1957). It is a minor pathogen in Brazil and seldom causes any significant damage (Costa, 1972). It also is reported in Peru, Trinidad, El Salvador (Wellman, 1977), and Colombia (Ellis et al., 1976a).

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 by 8-12 μm in size (Weber, 1973). Apothecia (Figure 71) and ascospores are formed by the perfect stage of the fungus which demonstrates variability in virulence according to strain and mating type (Polach and Abawi, 1975).

Infection usually starts from senescent blossoms colonized by the fungus or at wounds on plant parts such as leaves, stems, or pods (Figure 72). Penetration occurs from an infection cushion (García-Arenal and Sagasta, 1977). Symptoms appear as a water-soaked, greenish gray area on the affected tissue which subsequently wilts and dies. Phytoalexins (phaseolin, phaseolidin, phaseolinisoflavan) form inside and outside the lesions (Fraile et al., 1980; García-Arenal and Sagasta, 1977; van den Heuvel and Grootveld, 1980). These compounds and kievitone inhibited growth of two *B. cinerea* isolates differing in pathogenicity to bean (Fraile et al., 1982). Seedlings also may become wilted and die, although damage usually consists of a watery soft rot of pods (Johnson and Powelson, 1983a and 1983b; Weber, 1973; Zaumeyer and Thomas, 1957). Black stromata and sclerotia (as large as 4 mm in diameter) may be produced in infected tissue (Polach and Abawi, 1975) and resemble those formed by the white mold (*Sclerotinia sclerotiorum*). The fungus can be seed-borne (Ellis et al., 1976a).

Control measures are reduced plant density, increased row width, reduced irrigation frequency (Johnson and Powelson, 1983a and 1983b), and application of foliar fungicides (Vulsteke and Meeus, 1982). However, some strains of the fungus are resistant to fungicides, including benomyl (Hisada et al., 1979; Johnson and Powelson, 1983a; Pearson et al., 1980; Polach and Abawi, 1975). If available and practical as a control measure, common bean germplasm should be screened to identify sources of resistance.

Phyllosticta Leaf Spot

Phyllosticta leaf spot is caused by the fungus *Phyllosticta phaseolina* Saccardo which is favored by high moisture and moderate temperatures (Goth and Zaumeyer, 1963; Shands et al., 1964; Vieira, 1983). The fungus occurs in Brazil (Shands et al., 1964), Costa Rica, Nicaragua, El Salvador, Guatemala, Peru, Argentina, Puerto Rico (Wellman, 1977), and United States (Goth and Zaumeyer, 1963; Zaumeyer and Thomas, 1957). No reports are available concerning yield losses. The common name frequently used for phyllosticta leaf spot in Latin America is “mancha de phyllosticta.”

Phyllosticta phaseolina produces hyaline, one-celled pycnidiospores which are 4-6 by 2-3 μm in diameter. Pycnidia are 90 μm in diameter (Wellman, 1972).

Symptoms usually 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 with 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 (Goth and Zaumeyer, 1963; Zaumeyer and Thomas, 1957).

Control measures consist of foliar fungicides (Zaumeyer and Thomas, 1957). If available and practical as a control measure, common bean germplasm should be screened to identify sources of resistance.

Powdery Mildew

Powdery mildew of beans is caused by *Erysiphe polygoni* DC. and is distributed worldwide. Infection is favored by moderate temperatures and humidity. However, it can be prevalent within a wide range of environmental conditions (Zaumeyer and Thomas, 1957). The pathogen seldom causes extensive damage in Brazil and Costa Rica (Echandi, 1976; Shands et al., 1964; Vieira, 1983), but can

seriously reduce yields in Peru (Echandi, 1976). Yield losses varied from 17% to 69% in Colombia when different cultivars became severely infected before flowering (Schwartz et al., 1981a).

Common names frequently used for powdery mildew in Latin America include "oídio," "oidio," "mildeu polvoso," "cinza," "ceniza," and "míldio pulverulento."

The fungus produces hyaline conidia in chains on the leaf surface. The spores are ellipsoid, one-celled, and measure 26-52 by 15-23 μm in size. In Europe and North America, spherical black perithecia (120 μm in diameter), uncommon in the tropics, may form and contain asci and ascospores which measure 24-28 by 11-13 μm (Weber, 1973).

Symptoms first appear as slightly darkened mottled spots on the upper leaf surface which later become covered by a circular growth of white, powdery mycelium (Figure 73). The entire leaf and plant may become covered by mycelium (Figure 74), become malformed and yellow, and senesce prematurely. Stems and pods can be infected (Figure 75), resulting in yield loss and seed transmission. Pods may be stunted, malformed, or killed during severe epidemics. The fungus can be seed-borne (Zaumeyer and Thomas, 1957), probably as spores on the seed-coat surface.

Control measures are planting clean seed and using foliar chemicals such as sulfur, dinocap (1.2 g/L), or lime sulfur (10 ml/L). Concepción-T. (1977) did not observe significant yield increases with chemicals such as benomyl. However, Schwartz et al. (1981a) obtained effective control with benomyl (1 kg/ha). Resistant cultivars exist, but resistance can be overcome by the existence of different physiologic races (Schwartz et al., 1981a; Zaumeyer and Meiners, 1975; Zaumeyer and Thomas, 1957). Sources of resistance not specific to race must be sought and used where practical.

Scab

Scab of beans is caused by species of the fungus *Elsinoë* such as *E. phaseoli* Jenkins (Allen, 1983; Chupp and Sherf, 1960; Weber,

1973). It has a conidial stage known as *Sphaceloma phaseoli* (Holliday, 1980). The fungus occurs in Mexico, Central America, and the West Indies on lima beans (Chupp and Sherf, 1960), but has not been reported on *Phaseolus vulgaris*. However, in African countries such as Kenya and Zambia, the disease is important on common beans (CIAT, 1981; Holliday, 1980; Mutitu, 1979; Mutitu and Mukunya, 1979; Stoetzer et al., 1984). Yield losses have reached 70%.

The hyaline conidia of *E. dolichi* are produced on conidiophores on a hyaline to yellowish stromatic rind. Conidia are spherical to elliptical and measure 3-8 by 1-3 μm . Ascomata may also form on the leaf surface and cover the lesions as dark punctate bodies, measuring 100-600 μm . Asci are subglobose to ellipsoid, measure 20-32 by 15-22 μm , and contain septate ascospores. *Elsinoë phaseoli* conidia are hyaline to pale colored and measure 10 by 4 μm . Ascomata measure 30-40 μm , and ascospores measure 13-15 by 5-6 μm (Weber, 1973).

Symptoms may appear on leaves, stems, or pods as raised, wartlike protuberances (as large as 1 cm in diameter) which are tan to red or brown in color. Leaf spots may follow the venation on either leaf surface, become yellow, and have slightly raised margins. Stem lesions are brown to gray with yellow to black borders. Pod lesions are brown to purple-black, circular, punctate, and about 5 mm in diameter. Pods may become malformed. Conidia are abundantly produced in dark-colored pycnidia in the dark lesions (Weber, 1973).

Control measures are use of clean seed (Chupp and Sherf, 1960; CIAT, 1981) and crop rotation. Although limited screening of common bean germplasm has been conducted in Kenya (Stoetzer et al., 1984), additional work is needed.

White Leaf Spot

White leaf spot of beans is caused by the fungus *Pseudocercospora albida* (Matta et Belliard) Yoshii et Aamodt and is found in

Guatemala (Yoshii and Aamodt, 1978) and Colombia (Schwartz et al., 1981b) in sites higher than 1500 m. In Colombia, yield losses have exceeded 40% (Schwartz et al., 1981b). 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. Angular white spots (Figure 76) also may occur on the upper leaf surface and eventually enlarge and coalesce. Leaf necrosis may occur (Yoshii and Aamodt, 1978). 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 (Figure 77).

Benomyl (0.6 g/L) and mancozeb (2.4 g/L) can control white leaf spot (Schwartz et al., 1981b). Yoshii and Aamodt (1978) 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, Aguas Calientes 79, Michoacán 31, Arrox I-565, and R20 Antioquia 18.

Yeast Spot

Yeast spot or seed pitting of beans is caused by *Nematospora coryli* Peglion, *N. gossypii* Ashby et Nowell, and *Eremothecium cymbalariae* Borzi. It can be a seed production problem in Brazil (Costa, 1972; Menten et al., 1979a, 1979b, and 1980; Paradela-Filho et al., 1972; Vieira, 1983), Costa Rica, Ecuador, Peru, the West Indies (Wellman, 1977), and United States (Zaumeyer and Thomas, 1957). It can cause yield losses varying from 10%-100%, depending on its effect on seed quality and commercial appeal, especially in lima-bean production (Zaumeyer and Thomas, 1957). Menten et al. (1979b) report that common-bean seed weight can be reduced by as much as 28% and that seed quality and viability are also reduced. The common name frequently used for yeast spot in Latin America is "mancha de levedura."

Insects such as the *Megalotomus parvus* Westwood (Paradela-Filho et al., 1972), southern green stink bug (*Nezara viridula* (L.)), and lygus bugs (*Lygus hesperus* Knegt. and *L. elisus* van Duzee), transmit the causal organism and also may damage seeds directly from toxins secreted during the feeding process (Zaumeyer and Thomas, 1957). Galli et al. (1968) report that *Nematospora coryli* also persists in weeds such as *Cassia occidentalis* L., *Momordica charantia* L., *Bauhinia purpurea* L., and *Crotalaria* sp.

These yeast organisms belong to the Nematosporaceae family (Menten et al., 1980). *Nematospora coryli* produces a variable morphology in culture. First, it develops elliptical cells 6-10 μm wide by 8-14 μm long, followed by mature spherical cells of 20 μm in diameter and mycelium-like strands which measure 2.5-3.5 μm in width by 90-140 μm in length. *Nematospora coryli* grows in culture at temperatures between 15 and 40 $^{\circ}\text{C}$, but 25-30 $^{\circ}\text{C}$ is more favorable for infection (Zaumeyer and Thomas, 1957). *Ashbya gossypii* (Ashby et Nowell) Guillierm. has a faster growth rate than *N. coryli* when grown on potato dextrose agar or yeast extract malt agar at 25 $^{\circ}\text{C}$ in darkness (Menten et al., 1979a). These species and *E. cymbalariae* differ for cultural and morphological but not pathogenic characteristics (Menten et al., 1980).

Symptoms appear after insects have fed upon the pods. During feeding, the insects puncture the developing seeds and transfer fungal propagules to the wound sites. The spores germinate and infect the seeds (including the embryonic cotyledonary leaves), producing irregular, slightly sunken lesions about 1 mm in diameter. The lesions may be rose colored, tan, or brown (Costa, 1972); Vieira, 1983; Weber, 1973). *Nematospora coryli* has been recovered from infected seeds (Menten et al., 1979b).

Control measures consist of eliminating weed hosts, controlling insect populations, and selecting clean seed (Zaumeyer and Thomas, 1957).

Additional Pathogens

Some of the many other fungi reported as pathogens of beans (*Phaseolus* species) are listed in Table 1.

Table 1. Additional fungal pathogens of beans.

| Pathogen | Plant symptom or disease | Lit. cited |
|--|-----------------------------|------------|
| <i>Acrostalagmus</i> spp. | | 27 |
| <i>Aristostoma oeconomicum</i> Sacc. | Leaf spot | 89 |
| <i>Asteroma phaseoli</i> Brun. | Leaf, Pod spots | 89 |
| <i>Botryodiplodia theobromae</i> | Seed decay | 27 |
| <i>Brachysporium pisi</i> Oud. (perhaps a <i>Curvularia</i> sp.) | Leaf spot | 73 |
| <i>Cephalosporium gregatum</i> Allington et Chamberlain | Stem rot | 89 |
| <i>Ceratophorum setosum</i> Kirchn. | | 89 |
| <i>Chaetomium indicum</i> Cda. | | 89 |
| <i>Choanephora cucurbitarum</i> (Berk. et Rav.) Thaxter | Leaf spot, Pod rot | 45 |
| <i>Cladosporium album</i> Dows. | | 89 |
| <i>Cladosporium cladosporioides</i> (Fresen.) de Vries | Leaf spot | 52 |
| <i>Cladosporium herbarum</i> Pers. ex Fr. | Pod, Seed, Leaf spots | 73 |
| <i>Colletotrichum truncatum</i> (Schw.) Andrus et Moore | Pod, Stem spots | 84 |
| <i>Corticium salmonicolor</i> Berk. et Broome | Plant rot | 85 |
| <i>Corynespora cassicola</i> (Berk. et Curt.) Wei. | Leaf spot | 7, 62, 77 |
| <i>Cristulariella pyramidalis</i> Waterman et Marshall | Leaf spot | 44 |
| <i>Curvularia</i> spp. | Leaf spot, secondary | 85 |
| <i>Dendrophoma</i> spp. | | 11 |
| <i>Dimerium grammodes</i> (Kze.) Garman (<i>Parodiella perisporioides</i> (Berk. et Curt.) Speg.) | Leaf spot, secondary | 84 |
| <i>Diplodia natalensis</i> P. Evans | Seed contaminant | 89 |
| <i>Diplodia phaseolina</i> Sacc. | Pod spot | 89 |
| <i>Epicoccum neglectum</i> Desm. | Leaf spot | 89 |
| <i>Fusarium culmorum</i> (W.G. Sm.) Sacc. | Stem rot | 85 |
| <i>Fusarium equiseti</i> (Cda.) Sacc. | Damping-off | 85 |
| <i>Fusarium lateritium</i> Nees | Stem canker | 85 |
| <i>Fusarium macroceras</i> Wr. et Reinking | Pod decay | 89 |
| <i>Fusarium roseum</i> Lk. | | 89 |

(Continued)

Table 1. (Continued).

| Pathogen | Plant symptom or disease | Lit. cited |
|---|-----------------------------|------------|
| <i>Fusarium semitectum</i> Berk. et Rav. | Pod decay | 21, 85 |
| <i>Fusarium vasinfectum</i> Atk. | | 89 |
| <i>Gloeosporium corallinum</i> (Peyl.) Sacc. et Trav. | | 89 |
| <i>Glomerella cingulata</i> (Ston.) Spauld. et Schrenk. | | 89 |
| <i>Helminthosporium victoriae</i> Meehan et Murphy | Pod spot | 89 |
| <i>Heterosporium</i> spp. | Sooty leaf spot | 89 |
| <i>Hypochnus centrifugus</i> (Lev.) Tul. | | 89 |
| <i>Hypochnus cucumeris</i> Frank. | Damping-off | 89 |
| <i>Leptosphaeria phaseolorum</i> Ell. et Ev. | Stem disease | 89 |
| <i>Macrosporium communae</i> Rab. | | 89 |
| <i>Macrosporium consortiale</i> Theum. | | 89 |
| (<i>Stemphylium consortiale</i> Theum.) | | 89 |
| <i>Macrosporium leguminis phaseoli</i> P. Henn. | | 89 |
| <i>Macrosporium phaseoli</i> Faut. | | 89 |
| <i>Microsphaera diffusa</i> Cke. et Pk. | Leaf spot | 73 |
| <i>Microsphaera euphorbiae</i> (Pk.) Berk. et Curt. | Leaf spot | 89 |
| <i>Monilia</i> spp. | | 27 |
| <i>Mycena citricolor</i> (Berk. et Curt.) Sacc. | Leaf spot | 85 |
| Mycorrhizal fungi | Root parasitism | 89 |
| <i>Mycosphaerella phaseolicola</i> (Desm.) Ideta. | Leaf spot | 89 |
| <i>Myrmaecium roridum</i> Tode | Pod disease | 85 |
| <i>Nectria</i> spp. | | 85 |
| <i>Nigrospora</i> spp. | Pod decay | 28 |
| <i>Periconia pycnospora</i> Fr. | Pod disease | 85 |
| <i>Pestalotiopsis</i> spp. | | 27 |
| <i>Peyronellaea</i> spp. | | 27 |
| <i>Phakopsora vignae</i> (Bres.) Arth. (<i>Phakopsora pachyrhizi</i> Sydow) (<i>Physopella concors</i> Arth.) | Leaf rust (Soybean rust) | 76, 89 |
| <i>Phoma terrestris</i> Hans. | Root rot, secondary | 89 |

(Continued)

Table 1. (Continued).

| Pathogen | Plant symptom or disease | Lit. cited |
|--|-----------------------------------|------------|
| <i>Phyllachora phaseoli</i> (P. Henn.) Th. et Syd. | Leaf spot (Tar spot) | 73 |
| <i>Phyllosticta noackiana</i> All. | Leaf spot | 85 |
| <i>Phyllosticta phaseolorum</i> Sacc. et Speg. | Leaf spot (Ochraceous spot) | 89 |
| <i>Physarum cinereum</i> (Batsch) Pers. | | |
| <i>Phytophthora cactorum</i> (Leb. et Cohn) Schroet. | | 89 |
| <i>Phytophthora capsici</i> Leon. | | 89 |
| <i>Pleiochaeta setosa</i> (Kirchn.) Hughes | Leaf-and-pod spot (Brown spot) | 57 |
| <i>Pleospora herbarum</i> (Ders. et Fr.) Rab. (<i>Stemphylium botryosum</i> Wallr.) | Leaf spot | 73 |
| <i>Pullularia pullulans</i> (de By.) Berkhout. | Seed spot | 73 |
| <i>Pythium anadrum</i> Drechs. | | 73 |
| <i>Pythium arrhenomanes</i> Drechs. | Root rot | 73 |
| <i>Pythium helicoides</i> Drechs. | Root rot | 73 |
| <i>Pythium oligandrum</i> Drechs. | Root, Pod rots | 73 |
| <i>Pythium rostratum</i> Butl. | Root rot | 73 |
| <i>Pythium vexans</i> de By. | | 73 |
| <i>Rhizoctonia dimorpha</i> Matz. | Plant rot | 85 |
| <i>Rhizoctonia ferrugena</i> Matz. | | 89 |
| <i>Rhizopus nigricans</i> Ehrenberg | Pod rot | 84 |
| <i>Rhizopus stolonifer</i> (Ehr. ex Fr.) Lind | Soft rot | 73 |
| <i>Rhizopus tritici</i> K. Saito | Soft rot | 73 |
| <i>Sclerophoma phaseoli</i> Karak | Pod spot | 89 |
| <i>Septoria phaseoli</i> Maubl. | Leaf spot | 85 |
| <i>Sphaerotheca humuli</i> var. <i>fuliginea</i> (Schlecht.) Salmon. | Leaf spot | 89 |
| <i>Stagonospora hortensis</i> Sacc. | Leaf spot | 73 |
| <i>Stagonospora phaseoli</i> Dearn. et Malbr. | | 73 |
| <i>Stemphylium botryosum</i> Wallr. | Leaf spot | 85 |
| <i>Uromyces fabae</i> (Pers.) de Bary | Rust | 89 |
| <i>Vermicularia polytricha</i> Cke. | | 89 |
| <i>Verticillium albo-atrum</i> Reinke et Berth. | Root, Shoot diseases | 85 |

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Chapter 11

COMMON BACTERIAL BLIGHT

A. W. Saettler*

Introduction

Common bacterial blight is caused by the bacterium *Xanthomonas phaseoli* (Erw. Smith) Dowson and its brown pigment-producing fuscous variant, *X. phaseoli* var. *fuscans* (Burk.) Starr *et* Burk. Both bacteria are now recognized as *X. campestris* pv. *phaseoli* (Smith) Dye (Andersen, 1985) and will be referred to collectively as XCP throughout this chapter. Common blight is distributed worldwide (Costa, 1972; Crispín-Medina and Campos-Avila, 1976; Crispín-Medina *et al.*, 1976; Mukunya *et al.*, 1981; Orozco-Sarria, 1971; Pinto de Torres, 1968; Schieber, 1970; Vieira, 1967; Wallen and Galway, 1979). Common names frequently used for common bacterial blight in Latin America include “bacteriosis,” “añublo bacterial común,” “tizón común,” and “crestamento bacteriano.”

Yield losses caused by either of the two strains of XCP are difficult to estimate because the two bacteria frequently occur together in the same field, on the same plant, and causing identical symptoms. However, in 1967, XCP together damaged at least 75% of Michigan's 265,000 hectares of navy beans, with 10%-20% yield reductions (Focus on Michigan's bean industry, 1971). In two years of field trials, Wallen and Jackson (1975) reported a 38% yield loss in Ontario, Canada, because of XCP. Aerial infrared photographic surveys showed that these losses ranged from 1252 tons in 1970 to 218 tons in 1972 (Jackson and Wallen, 1975; Wallen and Jackson, 1975). Yield losses estimated at 22% and 45% have been obtained by natural and artificial infections, respectively, in Colombia (Yoshii *et al.*, 1976a). Economic surveys, based upon field observations in the same region, estimated yield losses of 13% (Pinstруп-Andersen *et al.*, 1976).

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Reported hosts of XCP are common bean (*Phaseolus vulgaris* L.), scarlet runner bean (*P. coccineus* L.), urd bean (*Vigna mungo* (L.) Hepper), mung bean (*V. radiata* (L.) Wilczek var. *radiata*), tepary bean (*P. acutifolius* A. Gray var. *acutifolius*), *V. aconitifolia* (Jacq.) Maréchal, *V. angularis* (Willd.) Ohwi et Ohasi, *Lablab purpureus* (L.) Sweet, *Strophostyles helvola* (L.) Elliott, soybean (*Glycine max* (L.) Merrill), *Mucuna deeringiana* (Bort.) Merrill, *Lupinus polyphyllus* Lindl., and cowpea (*V. unguiculata* (L.) Walp. ssp. *unguiculata*) (Vakili et al., 1975; Zaumeyer and Thomas, 1957).

Etiology

Laboratory isolations and purifications are necessary to distinguish the two strains of XCP; the *fuscans* strain produces a diffusible brown pigment (melanin) on media containing tyrosine (Hayward and Waterston, 1965a and 1965b). Pigment-producing strains are more virulent than those not producing pigment (Basu and Wallen, 1967). However, the pigment may not be essential for pathogenicity and its production in *Xanthomonas* species not pathogenic to beans indicate that this is not a stable taxonomic character (Basu, 1974; Dye, 1962).

The XCP bacterium is a gram-negative straight rod that is strictly aerobic and motile by a polar flagellum. It produces a yellow water-insoluble carotenoid and mucoid growth on nutrient glucose agar. It produces acid on media containing arabinose, glucose, mannose, galactose, trehalose, or cellobiose. It also causes proteolysis of milk (Dye and Lelliott, 1974) and starch hydrolysis. The XCP grows well on potato dextrose, nutrient, and yeast-extract-dextrose calcium carbonate (YDC) agars. The YDC media is the most commonly used. It consists of 10 g of yeast extract, 10 g of dextrose, 2.5 g of calcium carbonate, and 20 g of agar in 1 liter of distilled water (Saettler, 1971). When glucose is deleted from YDC, the colonies of XCP are not mucoid.

Several general (Kado and Heskett, 1970; Schaad and White, 1974) or relatively selective (Claflin et al., 1985; Trujillo and Saettler, 1980) media for XCP are available which allow for rapid isolation of the pathogen and are useful for epidemiological studies. The XCP can be stored on silica gel for long periods (Leben and

Sleesman, 1982). Many bacteria are tolerant to desiccation and can survive extended dry conditions (Leben and Sleesman, 1982; Trujillo and Saettler, 1981). The XCP produces an extracellular polysaccharide in culture and in the host plant (Leach et al., 1957). The polysaccharide aids survival for prolonged periods under varied environmental conditions (Wilson et al., 1965).

Epidemiology

The XCP bacteria are warm-temperature pathogens, causing greater damage to plants at 28 °C than at lower temperatures (Goss, 1940; Mack and Wallen, 1974; Patel and Walker, 1963). They grow optimally *in vitro* from 28 to 32 °C and growth declines gradually as temperature is lowered until growth stops at 16 °C. Detailed meteorological and microclimatological data are not available to determine specifically which factors influence the development of bacterial blight epidemics. In general, however, common blight epidemics are favored by high temperature and humidity (Sutton and Wallen, 1970).

Infection of bean seed is the most effective means of survival for XCP. Bacteria have been recovered from bean seed that were 3, 10, and 30 years old (Basu and Wallen, 1966; Zaumeyer and Thomas, 1957; and Trujillo and Saettler, 1980, respectively). Seed-borne strains normally are virulent when recovered (Alvarez-C. et al., 1979; Saettler, 1971 and 1974; Saettler and Perry, 1972; Schuster and Coyne, 1977). Contamination by XCP is both internal and external; external contamination can be eliminated by applying bactericides such as streptomycin, to the seed.

Seed lots can be assayed for the presence of XCP by incubating seeds in water or a liquid medium and then inoculating susceptible plants with the suspension by injection, water-soaking (Schuster and Coyne, 1975a), or vacuum infiltration (Lahman and Schaad, 1985; Venette and Naves, 1978). The most recent techniques of detection include enzyme-linked immunosorbent assay (ELISA), immunofluorescence, and a combined semiselective media and serology test (Afanador and Victoria, 1981; Malin et al., 1983; Trujillo and Saettler, 1979). Saettler and Perry (1972) assayed 101 navy bean seed lots for internal seed contamination with XCP and

about 35% of the lots were contaminated: 13% with the *fuscans* variant and 52% with both strains. Wallen et al. (1963) sampled 23 seed lots from Ontario, Canada, and isolated virulent cultures of the *fuscans* strains from more than 50% of the samples. The minimum number of infected seeds required to incite an epidemic is not known but must be determined for various cultural and environmental conditions.

Short-term survival within or on healthy-appearing bean plants occurs during the growing season (Thomas and Graham, 1952) and bacteria multiply on symptomless leaves (Weller and Saettler, 1978 and 1980a). XCP grows epiphytically on leaves of nonhost crop species such as soybean (*Glycine max*), maize (*Zea mays* L.), beet (*Beta vulgaris* L.), and cowpea (*Vigna unguiculata* ssp. *unguiculata*), and weeds (*Chenopodium album* L., *Amaranthus retroflexus* L., *Solanum nigrum* L., *Ambrosia artemisiifolia* L., and *Echinochloa crus-galli* (L.) Beauvois). Viable populations were recovered up to 21 days after bacteria were placed on leaf surfaces. Spread of XCP from *C. album* and *A. retroflexus* to bean plants occurred within 12 days after the weeds were inoculated (Cafati and Saettler, 1980b).

Overwinter survival of XCP in infested plant debris has been reported from some temperate regions (Burkholder, 1930). In Nebraska XCP survived in bean debris placed on top of the soil surface, but not when buried 20 cm below. Survival was greater under dry than under moist environmental conditions. Bacteria were recovered from the soil up to six weeks after burial. However, Schuster (1967) speculated that survival occurred in infested plant debris. In contrast, Sutton and Wallen (1970) could not isolate XCP from soil in which infected plants had grown. Saettler et al. (1986) concluded from a 10-year study in Michigan that XCP did not survive in association with residue. Several reports mention that blight symptoms failed to develop when pathogen-free seed was planted in soil infested with XCP from the previous season (Burkholder, 1930; Hedges, 1946; Wimalajeewa and Nancarrow, 1980). However, it is believed that, under some conditions, blight organisms can survive in soil for 18 months or more.

In general, then, in temperate bean-growing regions, infested bean residue is not always an important primary inoculum source of

XCP. However, in tropical bean-growing regions, infested residue is probably important in bean blight epidemiology because of the opportunities for bacteria to multiply and survive as epiphytes on perennial hosts and because of the practice of intercropping. However, van Rheenen et al. (1981) observed a decreased incidence of XCP spread throughout beans grown in association with maize compared with monoculture. Apparently, the maize provided a biological barrier to the physical movement (e.g., by wind or rain) of bacteria between bean plants. Further research is therefore needed to study the factors that affect the survival and longevity of XCP under tropical and temperate conditions.

The XCP bacteria are disseminated effectively on and within bean seed. Seed transmission of XCP has been known since 1872 (Schuster and Coyne, 1974 and 1975c). Plants grown from infected seed frequently bear lesions on cotyledons, nodes, or primary leaves. These lesions serve as secondary sources of inoculum during favorable environmental conditions (Burkholder, 1930). Infected seed or infested plant debris may be present within bean cull piles which then act as initial sources of inoculum (Burke, 1957). Volunteer plants present in fields provide another locus from which bacteria may be disseminated to susceptible plants.

Secondary spread of common and fuscous blight bacteria is effected by rain accompanied by wind (Zaumeyer and Thomas, 1957), windblown soils (Claflin et al., 1973), irrigation water (Steadman et al., 1975), people and animals, and insects such as the whitefly (Sabet and Ishag, 1969). XCP survives on insects. Leaf-feeding insects such as the borer *Diaprepes abbreviatus* (Boh.) and the beetle *Cerotoma ruficornis* (Ol.), can transmit the bacteria to wounds caused during feeding (Kaiser and Vakili, 1978). Spread of XCP by aerosols (Venette and Kennedy, 1975) has not been reported but other bacterial pathogens are spread this way.

Symptomatology

Both strains of XCP induce identical symptoms on leaves, stems, pods, and seeds. Leaf symptoms initially appear as water-soaked spots (Figure 78) which enlarge and frequently coalesce with adjacent lesions. Infected tissues appear flaccid and lesions are often

encircled by a narrow zone of lemon-yellow tissue. Necrosis then develops (Figure 79) and may become extensive enough (Figure 80) to cause defoliation or stem girdle (Zaumeyer and Thomas, 1957).

Blight bacteria enter leaves through natural openings such as stomata and hydathodes, and wounds (Zaumeyer and Thomas, 1957). They then invade intercellular spaces, causing a gradual dissolution of the middle lamella. Bacteria enter the stem through stomata of the hypocotyl and epicotyl and reach vascular elements from infected leaves or cotyledons. Colonization of xylem tissue may cause plant wilting by plugging vessels or disintegrating cell walls. The XCP does not systemically infect all *Phaseolus vulgaris* cultivars (Haas, 1972). Stem girdle or joint rot may develop at the cotyledonary node, especially in plants that grew from infected seed, and cause the plant to break at the node (Zaumeyer and Thomas, 1957) (Figure 81).

Pod lesions appear as water-soaked spots which may enlarge and become dark, red, and slightly sunken. If infection occurs during pod and seed development, infected seed may rot or shrivel (Figure 82). Seed infection occurs when the bacteria enter pod sutures via the pedicel or pod vascular system 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. If bacteria enter through the funiculus, only the hilum may become discolored. Studies have shown that infected seed can be found even in symptomless pods (Cafati and Saettler, 1980c; Weller and Saettler, 1980b). Symptoms on seed manifest as butter-yellow spots on white or light-colored seeds (Saettler and Perry, 1972; Zaumeyer and Thomas, 1957), but are difficult to see on medium to dark-colored seeds. Seedlings which develop from severely infected seed may have damaged growing tips, be stunted, or killed (snakehead) (Zaumeyer and Thomas, 1957).

There are several reports that other bean diseases can affect the severity of common blight. Panzer and Nickeson (1959) demonstrated that common blight is more severe in the presence of bean common mosaic virus, particularly late in the season. Hedges (1944) found that the common mosaic virus persisted in cultures of *X. phaseoli* for six weeks. Díaz-Polanco (1972) also showed that in the

infection of bean leaves a synergistic effect existed between *X. phaseoli* and the ashy stem blight fungus (*Macrophomina phaseolina* (Tassi) Goid.)

Zaumeyer and Thomas (1957) suggested that the *fuscans* variant caused a slight hypertrophy and darkening of the stem at the point of artificial inoculation of young seedlings. Moreover, several authors report severe plant symptoms following inoculation with the *fuscans* strain (Ekpo and Saettler, 1976; Zaumeyer and Thomas, 1957). Inoculation with mixtures of the two strains can induce severer symptoms than inoculation with a single strain (Ekpo, 1975).

Control by Cultural Practices

Cultural practices used to control common blight are planting pathogen-free seed (Webster et al., 1983a; Weller and Saettler, 1980b), crop rotation, and deep-plowing (Zaumeyer and Thomas, 1957). Clean or certified seed must be produced in regions free of pathogen or where environmental conditions discourage disease development. All seed must be tested for internal XCP contamination because studies have shown that symptomless bean plants can still produce contaminated seed (Cafați and Saettler, 1980c). Crop rotation with resistant crops gives time for the XCP population in bean debris within a field to decline.

Chemical Control

Various chemicals are used to protect foliage against XCP. Although some chemicals are effective in controlling foliage infection, yield increases have usually been minimal. Effective compounds include basic copper sulfate (Dickens and Oshima, 1969), copper hydroxide, and potassium *N*-hydroxymethyl-*N*-methylthiocarbamate (Bunema) (Weller and Saettler, 1976). Streptomycin provided marginal control in laboratory and field tests; it is translocated within the plant but not into the developing seeds (Mitchell et al., 1952; 1953; and 1954). However, antibiotics should not be applied to leaves because resistant mutants of the pathogen may develop. A new approach to seed treatment, still in experimen-

tal stage, is to use organic solvents to infuse antibiotics into bean seed.

Control by Plant Resistance

Strains of XCP differ in pathogenicity and virulence within and between geographical locations (Jindal and Patel, 1984; Schuster and Coyne, 1975b; Yoshii et al., 1976b). Schuster and Coyne (1971) obtained isolates from Colombia that were more virulent than several North American strains. Strains from Uganda were as virulent as those from Colombia (Schuster et al., 1973). Isolates with even greater virulence have since been identified (Ekpo and Saettler, 1976; Jindal and Patel, 1984). Differences in pathogenicity can also exist between colonies taken from individual stock cultures of XCP (Corey and Starr, 1957; Smale and Worley, 1956). However, documenting these differences has been complicated by variation in inoculation methods, age of isolates, and other factors.

Several different methods of plant inoculation have been tested:

pricking the cotyledon or cotyledonary node with a needle or scalpel dipped in inoculum (Arp et al., 1971; Burkholder and Bullard, 1946);

rubbing the second trifoliolate leaves with a cotton swab soaked with a carborundum-inoculum mixture (Corey and Starr, 1957);

soaking leaves with inoculum at high pressure (Arp et al., 1971; Schuster, 1955);

vacuum infiltrating into leaves (Venette and Naves, 1978);

pricking leaves with a multiple needle cushion (Andrus, 1948; Pastor-Corrales et al., 1981; Pompeu and Crowder, 1972); and

clipping leaves with scissors or razor blades dipped in inoculum (Ekpo, 1975; Webster, 1978; Webster et al., 1980).

Inoculum concentrations can influence the disease reaction. Optimal concentrations for uniform infection are between 10 million to 100 million cells/ml (Coyne et al., 1973; Ekpo, 1975; Pompeu and Crowder, 1973).

Phaseolus vulgaris cultivars and breeding materials vary in their reaction to infection by XCP (Mohan, 1981; Webster et al., 1980 and 1983b) (Figure 83). Immunity to infection has not been found, but many genotypes are resistant to infection, with little, if any, yield loss (Allen, 1983). However, bacteria can survive in tissue of resistant lines without causing symptoms (Cafati and Saettler, 1980a; Scharen, 1959). Phytoalexins, apparently, are not involved in resistance (Wyman and VanEtten, 1982). In general, beans are more susceptible to infection after the start of blossoming, that is, during the reproductive stage (Coyne and Schuster, 1973, 1974a, and 1974d; Coyne et al., 1973). Many workers, therefore, inoculate plants during flowering and evaluate reactions three to four weeks later. However, in the tropics, inoculations at three to four weeks after planting may be more useful, particularly if germplasm is variable in maturity, growth habit, and adaptation (CIAT, 1978; Webster, 1978). Coyne and Schuster (1974b) observed differential leaf and pod reactions to infection by XCP. The reactions were conditioned by different genes (Schuster et al., 1983; Valladares-Sánchez et al., 1983). Thus, the time of evaluation and design of disease rating scales must carefully account for these factors (Saettler, 1977).

Schuster (1955) first reported that *Phaseolus acutifolius* A. Gray (tepary bean) was resistant to XCP. Honma (1956) transferred genes from this resistant source into *Phaseolus vulgaris*, using embryo rescue to produce F₁ hybrid plants. Coyne and co-workers (1963 and 1973) surveyed more than 1000 plant introduction (P.I.) lines for resistance to XCP in the field. They found seven highly resistant *P. vulgaris* genotypes: 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 Gualí (accessions from Colombia), and Great Northern (G.N.) Nebraska No. 1 selection 27. Yoshii et al. (1978) reported that P.I. 282086 and P.I. 313343 exhibited resistant foliage reactions, but that the former also exhibited a susceptible pod reaction.

Phaseolus acutifolius "Tepary Buff" (Coyne and Schuster, 1974a) and P.I. 169932 (Yoshii et al., 1978) had high degrees of resistance with no symptoms observed. Several lines of *P. coccineus* were also resistant, but less so than tepary (Coyne and Schuster, 1974a). McElroy (1985) showed that three major genes determined the

reaction to a Colombian isolate of XCP of a cross of resistant with susceptible tepary beans. He successfully transferred resistance derived from the resistant source (Thomas and Waines, 1984) in a backcross program to different susceptible *P. vulgaris* cultivars.

Several of these resistant materials have been tested at various locations and exposed to bacterial isolates more virulent than those originally used. Although G.N. Nebraska No. 1 selection 27 and P.I. 207262 were also resistant to Brazilian isolates of XCP *fuscans* (Cafati and Kimati, 1972), the former was susceptible to a Colombian XCP isolate (Coyne et al., 1973). Poor plant adaptation to tropical growing conditions in Colombia apparently prevented the expression of resistance by G.N. Jules and P.I. 207262 (CIAT, 1978; Webster, 1978), until the plants became agronomically adapted through breeding and selection. Arnaud-Santana (1985) observed that *P. vulgaris* cv. Pompadour Checa is susceptible in the Dominican Republic (short days), but was moderately resistant in Nebraska (long days). However, susceptibility was expressed again when crossed to resistant adapted germplasm. Coyne et al. (1965 and 1973) found an association between delayed flowering and common blight resistance in Nebraska (long photoperiods), while Mohan (1981) found no association in Brazil (short photoperiods).

Inheritance of resistance to XCP recently has been reviewed (Coyne and Schuster, 1974a; Leakey, 1973; Schuster and Coyne, 1981; Zaumeyer and Meiners, 1975). Honma (1956) made the original interspecific cross between resistant *P. acutifolius* "Tepary 4" and susceptible *P. vulgaris* and found that resistance was quantitatively inherited. Coyne et al. (1965) further studied the inheritance of resistance in crosses 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 (Coyne et al., 1973).

The late-maturing G.N. Tara and G.N. Jules (Coyne and Schuster, 1969 and 1970) and early maturing G.N. Valley (Coyne and Schuster, 1974c) cultivars, derived from the cross with G.N. 1140, are resistant to XCP in most temperate regions of United States. G.N. Starr is an early maturing cultivar in which genes for resistance in P.I. 165078 (also tolerant to the bacterial wilt (*Corynebacterium flaccumfaciens* ssp. *flaccumfaciens*)) were trans-

ferred through six backcrosses to the recurrent parent G.N. Nebraska No. 1 selection 27 (tolerant to *X. phaseoli*) (Coyne and Schuster, 1976).

Coyne et al. (1966 and 1973) report that the cross between G.N. 1140 and G.N. Nebraska No. 1 selection 27 exhibited partial dominance for susceptibility. Similar inheritance patterns also were reported by Pompeu and Crowder (1972) for crosses between G.N. Nebraska No. 1 selection 27 and local 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 generation (Coyne and Schuster, 1974d). Transgressive segregation has been observed in these crosses (Coyne et al., 1966 and 1973; Pompeu and Crowder, 1972; Valladares-Sánchez et al., 1979 and 1983). Breeders should therefore be able to increase the levels of resistance within promising germplasm.

Suggestions for the Integrated Control of XCP

There are a number of practices which bean growers can use to minimize losses from XCP. These practices are described in the form of instructions:

Plant high-quality disease-free seed. Use the highest quality seed that is free of internal XCP infection. Discard all seed showing spotting or discoloration characteristic of XCP.

Treat seed with a bactericide. Treat all bean seed prior to planting with a slurry containing a bactericide that will kill bacteria infesting the seed surface.

Avoid cropping beans after beans. Practice a 2- to 3-year crop rotation to protect seed from blight organisms and other soil-borne pathogens that build up when beans follow beans too closely in rotation.

Deep-plow all bean refuse after harvest. Deep-plow fields with infected bean straw as soon as possible after harvest. This will prevent infested leaf tissue and straw from being transported to those parts of the farm where beans may be planted in the following year. This practice is especially important if a 2- to 3-year crop

rotation cannot be followed. If necessary, infected debris must be removed manually and destroyed by burning.

Isolate infected fields. Do not plant beans grown for seed next to commercial bean fields. This will avoid the spread of XCP from adjacent fields by wind, water, man, or animals. Do not grow beans where the water runoff from last year's contaminated bean fields can contaminate the new (unused) fields. The more isolated the field, the greater the chances are of avoiding infection. Avoid unnecessary activity in bean fields.

Use good herbicides to control weeds. Weed-free fields permit aeration around the plants so that they dry off more quickly. The shorter the exposure to continual wetness, the shorter the blight infection periods and so the lesser the infection in plants. In addition, some weeds may actually harbor bean blight bacteria.

Stay out of the fields as much as possible. Never work in the fields while the plants are wet with dew or rain because bacteria spread and infection takes place most readily under these conditions. Remember that every time you enter a field there is a chance of spreading pathogens by animals, humans, or equipment.

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Chapter 12

HALO BLIGHT

H. F. Schwartz*

Introduction

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

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

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Etiology

Pseudomonas syringae pv. *phaseolicola* cells are single straight rods and move by using multitrichous polar flagellae. The cells are gram-negative, strictly aerobic, and do not require growth factors. Poly- β -hydroxybutyrate is not accumulated as an intracellular carbon reserve. Cultures produce diffusible fluorescent pigments, particularly in iron-deficient media. Arginine dihydrolase is absent (Doudoroff and Pallerozin, 1974). The bacterium does not use glutarate, meso-tartrate, DL-glycerate, isoascorbate, betaine, erythritol, sorbitol, meso-inositol, nor N-caproate. It does use D-glucuronate, L(+)-arabinose, sucrose, succinate, DL- β -hydroxybutyrate, transaconitate, L-serine, L-alanine, and phydroxybenzoate (Misaghi and Grogan, 1969; Sands et al., 1970). It is oxidase-negative (Kovacs, 1956).

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

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

Epidemiology

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

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

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

Halo blight symptoms develop in six to ten days at 24-28 °C and may be delayed two or three days at higher temperatures (Zaumeyer and Thomas, 1957). Populations of one million colony-forming units per 30 square centimetres of leaf tissue (10^6 c.f.u./30 cm²) are apparently required for symptom development (Stadt and Saettler, 1981). Halo expression is more common at 16-20 °C than at 24-28 °C (Patel and Walker, 1963). Halo symptoms usually do not develop above 28 °C, although small and numerous water-soaked lesions may still be present (Zaumeyer and Thomas, 1957).

Symptomatology

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

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

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

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

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

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

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

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

Control by Cultural Practices

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

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

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

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

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

Chemical Control

Ralph (1976) reported that soaking bean seed in a 0.2% streptomycin solution for two hours prevented the transmission of halo blight bacteria by contaminated seed. However, the solution also reduced plant emergence by more than 20% compared with water-soaked controls. Hagedorn (1967) found that although streptomycin seed treatment was not always beneficial, it provided some residual protection against later plant infection. Taylor and Dudley (1977b) reduced primary infection from infected seed by 98% when it was slurry-treated with streptomycin (2.5 g a.i./kg seed) or kasugamycin (0.25 g a.i./kg seed). Streptomycin-resistant mutants have been obtained in vitro but often were not pathogenic nor survived in bean tissue (Russell, 1975).

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

cin sulfate, and dihydrostreptomycin sulfate (Hagedorn et al., 1969; Ralph, 1976; Saettler and Potter, 1970; Taylor and Dudley, 1977a; Zaumeyer and Thomas, 1957). Such chemicals are applied 7 to 10 days with ground or aerial spray equipment at rates of 200–400 g/1000 m². They are also applied at first flower and pod set at the rate of 0.1% a.i. per 675 litres per hectare to prevent the spread and development of halo blight on leaves and pods (Hagedorn et al., 1969; Saettler and Potter, 1970; Taylor and Dudley, 1977a). The application of antibiotics to the foliage may induce the development of resistant mutants. Their use should therefore be reduced or avoided. Legard and Schwartz (1987) demonstrated that timely copper hydroxide sprays significantly reduce or limit the establishment of syringae-type pseudomonads on bean foliage.

Control by Plant Resistance

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

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

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

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

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

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

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

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

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

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

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Chapter 13

ADDITIONAL BACTERIAL DISEASES

S. K. Mohan and D. J. Hagedorn*

Bacterial Wilt

Introduction

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

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

Etiology

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

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some straight and some wedge shaped. The bacterium is gram-positive, strictly aerobic, and motile by one, or rarely two or three, polar or subpolar flagella. The bacterium also causes hydrolysis of esculin (Cummins et al., 1974).

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

Epidemiology

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

The pathogen is seed-borne. It can survive up to 24 years in infected seed which may be discolored yellow, orange, or blue (Schuster and Christiansen, 1957; Schuster and Coyne, 1975; Schuster and Sayre, 1967; Zaumeyer and Thomas, 1957) (Figure 89). The bacterium does not overwinter well in soil but can survive between growing seasons in plant debris or on weeds. More virulent strains are better adapted for survival (Schuster and Coyne, 1974).

Symptomatology

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

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

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

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

Control by cultural practices

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

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

Control by plant resistance

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

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

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

Coyne and co-workers studied the inheritance of bacterial wilt resistance (Coyne et al., 1965 and 1966b). The resistant G.N. Star derives from the cross between P.I. 165078 (resistant accession from Turkey) and susceptible Great Northern Nebraska No. 1 selection 27 (Coyne and Schuster, 1976). Two complementary dominant genes conferred susceptibility and the absence of either one or both resulted in resistance. Susceptibility was dominant in a cross between P.I. 136725 (resistant accession from Canada) and susceptible G.N. 1140. In a cross between P.I. 165078 and G.N. 1140, resistance was quantitatively inherited. The degree of resistance varies among germplasm sources: for example, P.I. 136725 is less resistant than P.I. 165078, especially at high temperatures. P.I. 165078 was crossed with G.N. 1140 to produce the resistant cultivar Emerson (Coyne and Schuster, 1971) which has since been used for the commercial production of Great Northern beans.

Bacterial Brown Spot

Introduction

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

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

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

Etiology

The cells of *Pseudomonas syringae* pv. *syringae* are single straight rods and are motile by multitrichous flagella. The bacterium is gram-negative, strictly aerobic, and does not require growth factors. Poly- β -hydroxybutyrate is not accumulated as an intracellular carbon reserve. Cultures produce diffusible fluorescent pigments, particularly in iron-deficient media. Thus, the bacterium is a typical fluorescent pseudomonad of the *P. syringae* group. Arginine dihydrolase is absent (Doudoroff and Palleroni, 1974). The bacterium uses D-gluconate, glutarate, meso-tartrate, DL-glycerate, isoascorbate, betaine, sorbitol, meso-inositol, sucrose, N-caproate, N-caprylate, N-caprate, DL- β -hydroxybutyrate, citrate, glycerol, and L-proline (Misaghi and Grogan, 1969; Sands et al., 1970).

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

Epidemiology

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

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

Symptomatology

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

Control by chemicals

Hagedorn et al. (1969) report that various chemicals such as copper sulfate or copper hydroxide (86% cupric hydroxide with 56% metallic copper), can be applied at 200-400 g/1000 m² to control foliage and pod lesions. This control required weekly sprays after the emergence of the first trifoliolate leaf and resulted in a significant yield response only during severe epidemics. Detailed studies on epiphyte development (Legard and Schwartz, 1987) and disease incidence and severity on foliage revealed significantly less disease in sprayed irrigated beans (Morris et al., 1981).

Control by plant resistance

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

Inoculation methods are dusting seeds with pulverized infected tissue (Hagedorn et al., 1972) and spraying bacterial suspensions at 15 psi in the greenhouse and 150 psi in the field (Coyne and Schuster, 1969; Saad and Hagedorn, 1971). Injection of inoculum into very small seedlings in the crook neck stage of development has also been successful (Antonius and Hagedorn, 1981). Inoculations (1000-10,000 c.f.u./ml) identified lines with high levels of resistance (for example, WBR 133 and Wisconsin BBSR 130) more effectively than lines with moderate field resistance (for example, Wisconsin BBSR 17 and 28). Seedlings became increasingly susceptible during 3-4 days after emergence. Best results were obtained when seedling development was uniform (Antonius, 1982; Antonius and Hagedorn, 1981). Inoculum concentrations as high as 10⁵-10⁶ c.f.u./ml have been used in the greenhouse (Coyne and Schuster, 1969; Saad and Hagedorn, 1971).

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

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

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

Pod resistance of WBR 133 to low inoculum concentrations was higher than its pod resistance to high concentrations. Resistance was adversely affected by increased soil moisture (Daub and Hagedorn, 1976). Symptom expression in susceptible (Tender White) and resistant (WBR 133) beans was different at all inoculum concentrations tested. However, there were almost no differences in bacterial growth rates and final bacterial populations in the two hosts at high inoculum levels (Daub and Hagedorn, 1980). In the field, about one million cells/g of fresh weight were isolated from leaves of susceptible Eagle beans compared with the 1000 cells/g isolated from leaves of resistant WBR 133. Epiphytic populations on resistant bean-breeding lines were intermediate (Daub and Hagedorn, 1981). Wisconsin BBSR 130 was derived from a cross between a resistant selection, WBR 133 (from P.I. 313537), and susceptible Slimgreen. It is resistant to bacterial brown spot, common bacterial blight, halo blight, bean common mosaic virus, race gamma of the anthracnose pathogen, two rust races, and *Fusarium* yellows (Hagedorn and Rand, 1977). These and other germplasm sources should provide useful levels of resistance that can be incorporated effectively into commercially acceptable cultivars.

Wildfire

Introduction

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

Etiology

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

Epidemiology

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

Symptomatology

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

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

Control

No specific control measures are known.

Miscellaneous Bacterial Pathogens

There are other bacteria which are pathogenic to beans (*Phaseolus* spp.), but are not discussed in this book. Instead, they are listed in Table 1. Little, if any, information exists in bean literature, concerning their economic importance, distribution, symptomatology, epidemiology, and control measures.

Table 1. Miscellaneous bacterial pathogens of beans.

| Pathogen | Symptom | Literature cited |
|---|----------------|------------------|
| <i>Agrobacterium tumefaciens</i> (E.F. Smith et Towns.) Conn. | Crown gall | a |
| <i>Azotobacter chroococcum</i> Beijerinck | Overgrowth | b |
| <i>Azotobacter indicus</i> Starkey et De | Overgrowth | b |
| <i>Bacillus lathyri</i> Manns. et Taub. | Streak | c |
| <i>Bacillus megaterium</i> de Bary | Overgrowth | b |
| <i>Bacillus pumilis</i> Meyer et Gottheil | Overgrowth | b |
| <i>Bacillus subtilis</i> (Ehrenberg) Cohn | Overgrowth | b |
| <i>Corynebacterium fascians</i> (Tilford) Dows. | Gall | c |
| <i>Erwinia carotovora</i> (L.R. Jones) Holland | Market disease | a |
| <i>Erwinia nulandii</i> | Pink seed | d |
| <i>Escherichia coli</i> (Migula) Castellani et Chalmers | Overgrowth | b |
| <i>Micrococcus luteus</i> (Schroeter) Cohn | Overgrowth | b |
| <i>Pseudomonas adzukicola</i> | | c |

(Continued)

Table 1. (Continued).

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

a. USDA, 1970.

b. Serrada *et al.*, 1982.

c. Zaumeyer and Thomas, 1957.

d. Schuster *et al.*, 1981.

e. Tani and Baba, 1979.

f. Sirry *et al.*, 1981.g. Schuster *et al.*, 1980.

h. Johnson, 1956.

i. Schuster and Coyne, 1977.

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Chapter 14

MYCOPLASMA-LIKE DISEASES

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Introduction

Some plant diseases, known as “yellows,” were believed to have a viral etiology. However, in 1967, various workers (Doi et al., 1967b; Ishiie et al., 1967), through the use of electron microscopy and antibiotics, have demonstrated that “yellows” are actually caused by mycoplasma-like microorganisms (MLOs). Many diseases have since been associated with MLOs. Symptoms are characterized by plant chlorosis, stunting, excessive proliferation of branches (witches’ broom), bud proliferation (Derrick and Newsom, 1984), and disorders of floral organs (phyllody and virescence) (Davis, 1974; Davis and Whitcomb, 1970; de Lourds, 1975; Kitajima and Costa, 1972; Maramorosch, 1974; Maramorosch et al., 1974; Whitcomb, 1973). Many of the causal agents are transmitted by leafhoppers (Homoptera) to various hosts, including cultivated crops of the Leguminosae family (Bowyer and Atherton, 1970 and 1971; Bowyer et al., 1969; Derrick and Newsom, 1984; Granada, 1976 and 1979b; Iwaki, 1975; Kaloostian et al., 1976; Murayama, 1966; Nielson, 1968; Shinkai, 1965).

Mycoplasma organisms, including MLOs and spiroplasmas, are prokaryotes, lack a cell wall but possess a membrane, are highly pleomorphic, measure 0.2-1.0 μm in diameter, and contain ribosomes, RNA, and DNA (Murayama, 1966). Using electron microscopy, MLOs can be seen normally within plant sieve elements, but also within phloem parenchyma. MLOs are very difficult to multiply in vitro. However, Sugiura et al. (1977) maintained, and apparently multiplied, MLOs associated with Peach-X-disease by placing them in the dead cells of salivary glands of its leafhopper

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vector, *Colladonus montanus* (van Duzee). MLOs are resistant to penicillin but are susceptible to other antibiotics such as tetracycline.

Spiroplasmas infect various hosts but have not been detected in beans. Spiroplasmas are motile, have a definitive helicoid morphology, and measure 0.25 by 3.25 μm . Spiroplasmas have been cultured in vitro (Chen and Liao, 1975; Fudl-Allah et al., 1972; Saglio et al., 1971; Williamson and Whitcomb, 1975). They are transmitted by leafhoppers (Chen and Liao, 1975; Markham et al., 1974; Williamson and Whitcomb, 1975). Corn stunt (Davis et al., 1972) and stubborn disease of citrus (Fudl-Allah et al., 1972) are caused by spiroplasma organisms.

Pathogenic MLOs Associated with Legumes

Various MLOs infect beans and other leguminous crops. They cause diseases such as legume little-leaf, witches' broom, phyllody, and virescence.

Legume little-leaf. Hutton and Grylls (1956) described the legume little-leaf disease associated with forage legumes in Australia as being transmitted by the leafhopper *Orosius argentatus* (Evans) which is also a vector of tomato big bud. Electron microscopic studies have revealed the presence of MLOs in the sieve tubes and phloem parenchyma of naturally infected siratro (*Macroptilium atropurpureum* (DC.) Urb.), alfalfa (*Medicago sativa* L.), tomato (*Lycopersicon esculentum* Mill.), and cowpea (*Vigna unguiculata* (L.) Walpers ssp. *unguiculata*). They also appeared in experimentally infected plants of *Nicotiana glutinosa* L., *Datura stramonium* L., periwinkle (*Vinca rosea* L.), and common bean (*Phaseolus vulgaris* L.). MLOs were also detected in the sieve tubes of dodder (*Cuscuta australis* R. Br.) that was experimentally used for little-leaf transmission and in the salivary glands of those leafhoppers (*O. argentatus*) that had fed on the infected plants (Bowyer and Atherton, 1970 and 1971; Bowyer et al., 1969).

Trials showed that tetracycline, applied as spray (100 $\mu\text{g}/\text{ml}$) every two or three days for four to eight weeks, caused remission of little-leaf symptoms on the new growth of *N. glutinosa*, *Callistephus*

chinensis (L.) Nees, and *Lycopersicon esculentum*. However, the symptoms reappeared when treatment was suspended. Electron microscopic examinations revealed that there were no pleomorphic corpuscles present in the phloem of plants exhibiting a decreased symptom severity. Moreover, leafhoppers were unable to transmit the pathogen from these plants (Bowyer and Atherton, 1972).

Witches' broom and phyllody. Witches' broom has been known to occur on sweet potato (*Ipomoea batatas* (L.) Lamk.), soybean (*Glycine max* (L.) Merrill), peanut (*Arachis hypogaea* L.), pea (*Pisum sativum* L.), bean, and cowpea for many decades in Japan (Murayama, 1966; Shinkai, 1965). Shinkai (1972) found that the leafhopper vector of sweet potato witches' broom differed from that transmitting the pathogen to legumes. However, both vector species belonged to the genus *Nesophrosyne* (later reclassified as *Orosius*). The sweet potato vector transmitted the pathogen only to species in the Convolvulaceae family and to *Vinca rosea*. The legume vector transmitted the pathogen to members of the Leguminosae and several species of Compositae, Amaranthaceae, Cruciferae, and Chenopodiaceae (Murayama, 1966; Shinkai, 1965). The vectors of MLOs causing witches' broom in legumes and sweet potato are now classified as *Orosius orientalis* and *O. ryukyuensis*, respectively (Shinkai, 1972).

The latent period of the causal agent in the legume vector is about one month. This can be shortened by raising the temperature, for example, 17 days at 30 °C. Diseased bean plants exhibit typical symptoms of witches' broom such as yellowing, reduced leaflets, shoot proliferation, and phyllod-like disorders of floral organs (Murayama, 1966; Shinkai, 1965). Mycoplasma-like corpuscles are found in the phloem of diseased legume plants (Doi et al., 1967a) in different parts of the world.

Although *Phaseolus vulgaris* was not included in the list presented by Iwaki (1975), the occurrence of witches' broom and phyllody in Indonesia was reported in several legume crops, including soybean, peanut, urd bean (*Vigna mungo* (L.) Hepper), cowpea, and *Crotalaria* sp. The MLO has a latent period of nearly three weeks in the vector *Orosius argentatus*. Transmission trials have shown that the causal agent of witches' broom in legumes can infect other plant species. Histological examination using the electron microscope confirmed the presence of MLOs in plant tissues.

Witches' broom and phyllody have caused economic damage to cowpea in the Philippines (Benigno, 1977) and Thailand (Deema, 1977). Electron microscopy revealed the presence of MLOs in the phloem of infected plants. However, no additional information exists concerning the transmission and vectors of these diseases. In a revision of virus and plant problems associated with MLOs, Mishra (1977) described witches' broom in *Vigna radiata* (L.) Wilczek var. *radiata* and *V. mungo* in India but gave no information concerning the pathogen.

Kitajima and co-workers (Kitajima and Costa, 1972 and 1979; Kitajima et al., 1974) reported the occurrence of witches' broom in several legumes such as *Crotalaria juncea* L., *C. paulina*, *Desmodium* sp., soybean, and siratro. Electron microscopic observations demonstrated that there was a consistent association between the presence of MLOs and the disease. No work has yet been conducted on its transmission or the identification of its vector.

A low (1%-3%) incidence of witches' broom and phyllody has been observed in the green belt of the Federal District in Brazil. The infectious nature of this disease was demonstrated by grafting. Mycoplasma-like corpuscles were found in sieve tubes of the vascular region of naturally or experimentally infected plants (Figures 96 and 97). The vector remains unknown.

Maramorosch et al. (1974) detected MLOs in sieve tubes of pigeonpea (*Cajanus cajan* (L.) Millsp.) exhibiting witches' broom symptoms. However, no details were given for its pathology or transmission.

Virescence. In Zagora and Morocco, Cousin et al. (1970) identified mycoplasma-like corpuscles in the cortical parenchyma of beans exhibiting symptoms of virescence. However, they did not furnish economical or pathological data concerning the disease or its pathogen.

Unfortunately, little data are available which identify the MLOs associated with virescence or witches' broom of legumes in different parts of the world. In the three cases studied in most detail—Australia, Japan, and Indonesia—the similarity of host range and vector (Hutton and Grylls, 1956; Iwaki, 1975; Shinkai, 1965) suggests that the etiological agent may be similar. There is not

enough information to conclude that virescence and witches' broom are caused by the same or different mycoplasma species. Host and vector specialization may explain why certain MLOs are associated with diseases that have restricted host ranges.

Machismo. A mycoplasma-like disease was first detected in 1968 in infected soybean plants growing in the Cauca Valley of Colombia (Baeza, 1970; Granada, 1976). Since then it has increased in cultivated soybean crops and its incidence in individual fields varied from 0.4%-80%, with corresponding yield losses of 8-1600 kg/ha (Granada, 1979b). After 1980, a disease with similar symptoms was observed in commercial bean fields grown in the Cauca Valley with a disease incidence of 8%-15% (Granada, 1978b). During 1981-1985, incidence of the disease in both beans and soybeans has been less than 1% (Granada, 1984).

This mycoplasma-like organism can infect the following hosts: soybean (*Glycine max*), common bean (*Phaseolus vulgaris*), *Vigna angularis* (Willd.) Ohwi et Ohasi, *V. umbellata* (Thunb.) Ohwi et Ohasi, lima bean (*P. lunatus* L.), *Crotalaria spectabilis* Roth., *C. juncea*, *Desmodium* sp., periwinkle (*Vinca rosea*), pigeonpea (*Cajanus cajan*), *Rhynchosia minima* (L.) DC., and *Galactia glaucescens* Kunth. (Granada, 1978a). Common names frequently used for bean mycoplasma in Latin America are "machismo" and "amachamiento."

Electron microscopic evaluation of infected bean or soybean (Fletcher et al., 1984) tissue revealed the presence of mycoplasma-like corpuscles in phloem cells. The mycoplasma-like etiology also has been confirmed by symptom expression and Dienes' staining with tetracycline (Fletcher et al., 1984; Granada, 1979c).

The mycoplasma-like organism is transmitted by the brown leafhopper *Scaphytopius fuliginosus* Osborn (Figure 98) (Granada, 1976 and 1979b). High population levels of this insect have been detected in infected soybean fields in Colombia (García et al., 1975). This vector has been shown to transmit the mycoplasma-like organism to bean plants grown under controlled conditions (Granada, 1979a). The same vector has been recently reported in association with the machismo-like disease of soybeans in south-western Mexico (Fletcher et al., 1984).

When one- to six-day-old bean seedlings were exposed to infective adults of *S. fuliginosus* for five days, the average incubation time of the pathogen was 37 days (range of 31-43 days) (Granada, 1979a). This is similar to the 39-day incubation period obtained in soybeans tested under the same conditions (Granada, n.d.). The organism is not transmitted mechanically or by seed, but can be by grafting (Granada, 1979a). Legume little-leaf disease has an incubation period of only 19-23 days (Bowyer and Atherton, 1971).

Symptoms of mycoplasma infection usually become apparent during flowering and pod development when reproductive structures are converted into vegetative structures. Early infection turns flower petals a light to dark green (virescence) and flowers are smaller but have longer sepals than normal. A corrugated structure emerges from the unopened floral apex which is filiform at the upper end and resembles a rolled leaf when dissected (phyllody) (Figure 99). Later infections may cause pods to be rigid, thin, erect, twisted, corrugated, oriented upward, and shaped like a half-moon (Figure 100). These pods form few, if any, seeds. Severe symptoms are characterized by flowers being reduced to small buds and supported on a large petiole from which additional small leaves and petioles may proliferate (Figure 101). The plant as a whole resembles a typical witches' broom (Figure 102). Late infection of plants bearing healthy appearing pods may stimulate premature germination of seeds still in the pod (Figure 103). Germinated seeds can be transplanted and develop into normal plants free of MLOs (G.A. Granada, unpublished data).

This MLO induces similar symptoms during flowering in other hosts such as lima bean (*P. lunatus*), soybean (Figure 104), *Vigna angularis*, *V. umbellata*, *Galactia glaucescens*, and *Desmodium* sp. (Granada, 1978a). Infected *Crotalaria spectabilis* plants demonstrate abundant vegetative ramification before flowering, which does not occur in *C. juncea* (Figure 105) (G.A. Granada, unpublished data). The pumpkin (*Cucurbita maxima* Duchesne) has recently been found to also be a host of machismo (Varón de Agudelo, 1984).

Control measures are the observation of normal planting dates, maintenance of adequate crop rotation, and not planting continuous or simultaneous cycles of susceptible crops such as beans and

soybeans. This will reduce the buildup and the continued survival of insect vector populations and sources of inoculum from infected plants. Ideally, when it is economically feasible, infected plants are removed from the field and destroyed. In addition, weed hosts are also eradicated from fields and surrounding borders or irrigation canals. When dealing with a relatively high incidence (5%-10%) of machismo and the vector, insecticides such as those used to control the green leafhopper (*Empoasca kraemeri* Ross *et* Moore), may also reduce brown leafhopper populations.

Under greenhouse conditions the vector has shown sensitivity to all insecticides used on beans. Spraying of oxytetracycline at 100 ppm, every five days, starting 20-30 days before flowering, is recommended in Mexico for plant mycoplasma control (de la Rosa-García, 1981). However, this measure is not considered practical for machismo of either beans or soybeans in Colombia.

Although plant resistance would provide an ideal control measure, the screening of bush type materials from both the Instituto Colombiano Agropecuario (ICA) and the Centro Internacional de Agricultura Tropical (CIAT) bean programs to date has not detected a resistance level that is commercially acceptable to Colombian markets (G.A. Granada, unpublished data).

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Chapter 15

APHID-TRANSMITTED VIRUSES

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General Introduction

Various aphid-borne viruses infect beans and include bean common mosaic virus (BCMV), bean yellow mosaic virus (BYMV), cucumber mosaic virus (CMV), soybean mosaic virus (SMV), and alfalfa mosaic virus (AMV). This chapter will review the geographical distribution, economic importance, host range, physiochemical properties, purification, transmission, epidemiology, symptomatology, and control of these viruses.

Bean Common Mosaic Virus

Introduction

Bean common mosaic was one of the first virus diseases reported in the world when Iwanoski (1894) observed it in the Soviet Union. Since then the 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 (Cafati-K. and Alvarez-A., 1975; Costa et al., 1971; Crispín-Medina and Campos-Avila, 1976; Dean and Wilson, 1959; El-Shamy et al., 1972; Gámez, 1973; Hampton et al., 1983; Inouye, 1969; Joshi et al., 1981; Kaiser et al., 1968; Klessner, 1961; Kulkarni, 1973; Lockhart and Fischer, 1974; Moreno et al., 1968; Provvidenti et al., 1982; Schieber, 1970; Yerkes and Crispín-Medina, 1956; Zaumeyer and Thomas, 1957).

Plant infection may reach 100% in fields and yield losses range from 35% to 98% (Gálvez and Cárdenas-A., 1974; Hampton, 1975;

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Zaumeyer and Thomas, 1957). Hampton (1975) 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-A. (1974) reported that yield losses varied from 6% to 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 common bean (*Phaseolus vulgaris* L.), lima bean (*P. lunatus* L.), tepary bean (*P. acutifolius* var. *acutifolius*), *Vigna angularis* (Willd.) Ohwi et Ohashi, *V. aconitifolia* (Jacq.) Maréchal, *V. umbellata* (Thunb.) Ohwi et Ohashi, urd bean (*V. mungo* (L.) Hepper), scarlet runner bean (*P. coccineus* L.), siratro (*Macroptilium atropurpureum* (DC.) Urb.), *V. radiata* (L.) Wilczek var. *radiata*, *P. polyanthus* Greenman, *Vigna unguiculata* spp. *unguiculata* var. *sesquipedalis* (L.) Verdc., cowpea (*V. unguiculata* (L.) Walp. ssp. *unguiculata*), broad bean (*Vicia faba* L.), *Crotalaria spectabilis* Roth., *Canavalia ensiformis* (L.) DC., *Lupinus albus* L., *Nicotiana clevelandii*, *Macroptilium lathyroides* (L.) Urb., pea (*Pisum sativum* L.), alfalfa (*Medicago sativa* L.), *Lablab purpureus* (L.) Sweet, common clover (*Trifolium pratense* L.), and *Rhynchosia minima* (L.) DC. (Bos, 1971; Kaiser and Mossahebi, 1974; Kaiser et al., 1971; Meiners et al., 1978; Ordosgoitty, 1972; Zaumeyer and Thomas, 1957). *Sesbania exaltata* (Raf.) V.L. Cory and siratro (*Macroptilium atropurpureum* (DC.) Urb.) are reported as symptomless hosts (Meiners et al., 1978). R. O. Hampton (personal communication) has pointed out that additional research is needed to confirm that *Vicia faba* and *Vigna* species are true hosts, particularly with regard to seed transmission.

Chenopodium quinoa (Willd.), *Gomphrena globosa* L., *Tetragonia expansa* J. Murr., and cultivars of *Phaseolus vulgaris* serve as local-lesion indicators to various strains of BCMV (Alvarez-A. and Sepúlveda-R., 1982; Bos, 1971; Castaño-J. et al., 1982; Polak and Chod, 1972; Saettler and Trujillo, 1972; Schneider and Worley, 1962; Trujillo and Saettler, 1972a and 1973; Zaumeyer and Goth, 1963). In nature, however, BCMV is primarily restricted to *Phaseolus* spp., particularly *P. vulgaris*. It is possible that some susceptible hosts reported above were infected by serologically related viruses and not by BCMV strains.

Bean common mosaic virus was called bean virus 1 and *Marmor phaseoli* Holmes (Zaumeyer and Thomas, 1957). The name given to

bean common mosaic virus in Latin America is “mosaico común” in Spanish and “mosaico comum” in Portuguese.

Symptomatology

Bean common mosaic virus may incite three types of symptoms: mosaic, systemic necrosis (black root), or local lesions or malformations, depending upon the cultivar, time of infection, strain, and environmental conditions. Mosaic symptoms appear in systemically infected cultivars and may cause mottling, curling, stunting, and malformation of primary leaves (Figure 106), especially if primary infection occurred through infected seed. The trifoliolate leaves may exhibit leaf malformation and mosaic (Figure 107). Infected leaves may appear narrower and longer than uninfected leaves (Figure 108).

Systemically infected plants may have smaller and fewer pods than infected plants. Infected pods occasionally may be covered with small dark green spots and mature later than uninfected pods (Zaumeyer and Goth, 1964; Zaumeyer and Thomas, 1957).

Systemic necrosis (black root) symptoms may appear in cultivars having hypersensitive resistance (I gene) to systemic mosaic upon infection by necrosis-inducing strains, especially at high temperatures (26-32 °C). However, some necrosis-inducing strains are temperature independent (Drijfhout, 1978). The incidence of black root in Latin America is usually negligible but may reach 100% in Africa.

Black-root symptoms initially appear as a progressive vein necrosis (Figure 109) of the young trifoliolates which then die. The older leaves start to wilt and, eventually, the entire plant dies. Characteristic reddish brown to black streaks appear on the stems, roots, and pods (Figure 110). The entire vascular system soon becomes necrotic (Figure 111) (Drijfhout, 1978; Hubbeling, 1972; Zaumeyer and Thomas, 1957).

Local lesions may appear on the leaves of some cultivars. These lesions may be induced by mechanical inoculation or aphid transmission. They manifest as reddish to dark brown necrotic ring-shaped lesions or spots (Figure 112), 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 (Polak and Chod, 1972; Saettler and Trujillo, 1972; Schneider and Worley, 1962; Trujillo and Saettler, 1972a, 1972b, and 1973; Zaumeyer and Goth, 1963).

Physical properties

Bean common mosaic virus particles can be observed easily with the electron microscope in crude sap or partially purified preparations. The filamentous flexuous virus particles are 730-750 nm in length and 12-15 nm in width (de Camargo et al., 1968; Morales, 1979). Cytoplasmic inclusions are also induced by the virus and readily appear in the light or electron microscope as cylindrical pinwheels (Figure 113) (de Camargo et al., 1968; Hoch and Provvidenti, 1978; Valdés et al., 1982). Virus particles are transported throughout the phloem. They can be detected in upper plant parts within 24-48 hours and in the root system within 60 hours after inoculation (Ekpo and Saettler, 1974 and 1975).

Bean common mosaic virus particles are inactivated in sap at 56-65 °C, have a dilution end point of 10^{-3} to 10^{-4} , and are infectious for one to four days (Bos, 1971; Gámez, 1973).

Morales (1979) developed a purification method which isolates BCMV with a high degree of purity and in adequate amounts to produce a specific antiserum.

Epidemiology

Bean common mosaic virus can be transmitted mechanically, in pollen and seed, 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 (Morales, 1979). Many workers have also added abrasives such as Carborundum powder to inoculum to help introduce virus particles into plant cells (Cafati-K., 1968; Zaumeyer and Thomas, 1957).

An inoculation efficiency of nearly 100% can be achieved in the greenhouse, while in the field efficiency is lower because adverse environmental factors affect both viruses and plants.

Virus particles can be transmitted in pollen grains, ovules, and flowers of infected plants (Ekpo and Saettler, 1974; Wilson and Dean, 1964; Zaumeyer and Thomas, 1957). Seed transmission likewise can occur in susceptible cultivars of *Phaseolus vulgaris*, *P. acutifolius*, *P. coccineus*, *P. polyanthus*, *Macroptilium lathyroides*, *Rhynchosia minima*, and in *Vigna* species (Kaiser and Mossahebi, 1974; Meiners et al., 1978; Noble and Richardson, 1968; Phatak, 1974; Provvidenti and Braverman, 1976; Provvidenti and Cobb, 1975; Robertson, 1962; Skotland and Burke, 1961). The percentage of seed transmission varies from 3% to 95%, according to cultivar and time of infection, especially before flowering (Alconero and Meiners, 1974; Alvarez-A., 1977; Crispín-Medina and Grogan, 1961; Gálvez and Cárdenas-A. 1974; Gálvez et al., 1977; Kulkarni, 1973; Montenegro-B. and Galindo-A., 1974; Ordosgoitty, 1972; Schippers, 1963; Zaumeyer and Thomas, 1957). BCMV particles are reported to survive in bean seed for at least 30 years (Zaumeyer and Thomas, 1957).

Insect vectors such as aphids (Figure 114) can transmit BCMV effectively from infected plants to healthy plants. Reported aphid vectors include *Macrosiphum solanifolii* (Ashmead), *M. pisi* (Kalt.), *M. ambrosiae* (Thomas), *Myzus persicae* (Sulzer), *Aphis rumicis* L., *A. gossypii* Glover, *A. medicaginis* Koch, *Hyalopterus atriplicis*, and *Rhopalosiphum pseudobrassicae* Davis (Zaumeyer and Thomas, 1957; Zettler and Wilkinson, 1966). Studies have determined that aphid populations are often lower than those of other insect species in bean fields, but that the aphids are responsible for transmission of BCMV. The efficiency of transmission depends upon the source of inoculum, but usually virus acquisition and transmission (Zettler, 1969) occurs within one minute.

In the tropics and other regions, infected seeds and plants of susceptible bean cultivars serve as sources of primary inoculum for BCMV (Hampton, 1967; Robertson and Klostermeyer, 1961 and 1962). Aphids are responsible for the secondary transmission of the virus. In Colombia, CIAT studies determined that relatively high aphid populations were able to incite 100% plant infection from a seed source that was only 2%-6% infected.

Control by cultural practices

Various cultural practices such as planting date and clean-seed production, minimize BCMV incidence in susceptible cultivars. Burke (1964) found a correlation between planting date and virus incidence which was associated with aphid population levels. Bean plantings, therefore, must be adjusted to minimize the period during which susceptible cultivars are exposed to infection by aphids migrating from other crops to beans during the growing season.

Planting BCMV-free seed can effectively reduce the initial inoculum. However, to reduce transmission of BCMV from other infected bean plants or weed hosts, it may also be necessary to control aphids with insecticides (Sánchez and Pinchinat, 1974). No chemicals or other treatments are available to remove or destroy BCMV particles present within infected seed (Zaumeyer and Thomas, 1957).

Control by plant resistance

Plant resistance to bean common mosaic virus has been available for nearly 60 years after the cultivar Robust was discovered to be resistant. The resistance of Robust is conferred by a single recessive gene (Baggett et al., 1966; Cafati-K. and Alvarez-A., 1975; Guerra et al., 1971; Hernández-Bravo and Gálvez, 1976; Zaumeyer and Thomas, 1957). Cultivars that were subsequently developed, having Robust resistance, include Great Northern U.I. 1, 59, 81, and 123; Red Mexican U.I. 3 and 34; Royal Red; and Pinto U.I. 72, 78, and 111 (Burke et al., 1969; Smith, 1962a and 1962b; Zaumeyer and Thomas, 1957). These cultivars have been resistant to the type strain of BCMV for more than 50 years (Zaumeyer and Meiners, 1975).

Nearly 50 years ago another source of resistance was identified in Corbett Refugee. This resistance is conferred by a dominant hypersensitive gene which conditions the black-root reaction. The majority of snap bean cultivars and some of the common bean cultivars developed in United States have derived their resistance from Corbett Refugee. They include Wisconsin Refugee, Idaho Refugee, and Refugee U.S. 5 (Zaumeyer and Thomas, 1957). This resistance has been effective for nearly 50 years. Burke and

Silbernagel (1974) and van Rheenen and Muigai (1984) 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, Titán in Chile, Peru 257 in Peru, Tacarigua in Venezuela, and Jamapa and Sataya 425 in Mexico (Cafati-K. and Alvarez-A., 1975; Drijfhout, 1978; Montenegro-B. and Galindo-A., 1974; Ortega-Y. and Barrios-G., 1972; Trujillo and Saettler, 1972b; Ziver-M. and Cafati-K., 1968).

Hagel et al. (1972) 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 application 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 (Alvarez-A., 1977; Alvarez-A. and Ziver-M., 1965; Bercks, 1960; Drijfhout, 1978; Drijfhout and Bos, 1977; Drijfhout et al., 1978; Innes and Walkey, 1980; Silbernagel, 1969). Drijfhout (1978) assigned 22 cultivars to 11 resistance groups and divided the 15 known viral strains in seven pathogenicity groups (Table 1).

Cultivars in resistance groups one to six do not express systemic necrosis to any viral strains. However, they do express systemic mosaic symptoms to one or more of the BCMV strains. These cultivars have recessive genes only. The experimental line IVT 7214 (resistance group 7) does not exhibit systemic mosaic nor necrosis upon inoculation with any known viral strain. It possesses a recessive gene *bc 3* which is effective against all known strains at this time. Cultivars in resistance groups 8 to 10 may exhibit only systemic necrosis to one or more of the necrosis-inducing strains of BCMV. These cultivars, therefore, have the dominant *I* gene. The IVT 7233 line has the dominant *I* gene, together with a recessive gene of cultivar group 6 which protects against systemic necrosis. This line exhibits only local necrotic lesions when inoculated with a

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 | Vla | Vlb | VII | | | |
| | | West-landia NL 1 | Type US 1 | Puerto Rico PR 1 | NL 7 | NL 8 | Florida US 5 | Western US 4 | Idaho or B US 3 | Colona NL 6 | NY 15 US 2 | Imuna NL 2 | Michelite NL 3 | Jo-landa NL 5 | Mexico US 6 | Great North. NL 4 |
| Cultivars with recessive alleles (I*1*) of the necrosis gene | | | | | | | | | | | | | | | | |
| 1 | Dubbele Witte Str. Gr. Ref | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + |
| 2 | Redl. Gr. C Puregold Wax Imuna | — | — | — | + | — | + | + | + | + | + | + | + | + | + | + |
| 3 | Redl. Gr. B Gr. North. 123 | — | — | — | — | — | + | + | + | — | — | + | + | + | + | + |
| 4 | Sanilac Michelite 62 Red Mex. 34 | — | — | — | — | + | — | — | — | + | + | + | + | + | — | — |
| 5 | Pinto 114 | — | — | — | — | — | — | — | — | + | + | + | + | + | — | — |
| 6 | Monroe Gr. North. 31 Red. Mex. 35 | — | — | — | — | — | — | — | — | — | — | — | — | + | + | + |
| 7 | IVT 7214 | — | — | — | — | — | — | — | — | — | — | — | — | — | + | + |

(Continued)

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 | Vla | Vlb | VII | |
| | | West-landia NL 1 | Puerto Rico PR 1 | NL 7 | NL 8 | Florida US 5 | Western US 4 | Idaho or B US 3 | Colona NL 6 | NY 15 US 2 | Imuna NL 2 | Miche-lite NL 3 | Jo-landa NL 5 | Mexico US 6 |
| Cultivars with dominant alleles (II) of the necrosis gene | | | | | | | | | | | | | | |
| 8 | Widusa Bl. Turtle S.I | — | — | — | + | — | ±n | ±n | ±n | — | — | ±n | ±n | — |
| 9a | Jubila | — | — | — | — | — | ±n | ±n | ±n | — | ±n | ±n | ±n | — |
| 9b | Top Crop 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.

SOURCES: Drijfhout, 1978; Drijfhout et al., 1978.

necrotic BCMV strain. These genes have been successfully incorporated to produce mosaic and black-root resistant, commercial cultivars (Drijfhout, 1978).

Bean Yellow Mosaic Virus

Introduction

Bean yellow mosaic virus (BYMV) is widely distributed throughout the world. However, it usually occurs in legumes other than beans. The virus occurs in North America, Europe, East Africa, Japan (Bos, 1970; Inouye, 1969, Vanderveken, 1963; Zaumeyer and Thomas, 1957), Chile (Cafati-K. et al., 1976), Argentina (von der Phalen, 1962), Brazil (Costa et al., 1971; Kitajima and Costa, 1974), Uruguay, and possibly northern Mexico.

BYMV infected up to 100% of the plants grown in a field in United States (Zaumeyer and Thomas, 1957). Hampton (1975) reported that BYMV could cause serious yield losses with a 33% and 41% reduction in pod number and seed yield, respectively.

Bean yellow mosaic virus has been called *Phaseolus virus 2*, *Gladiolus mosaic virus*, *pea mosaic virus*, and *bean virus 2* by earlier workers (Zaumeyer and Thomas, 1957). Common names for BYMV in Latin America include “mosaico amarillo” and “moteado amarillo” in Spanish, and “mosaico amarelo” in Portuguese.

Bean yellow mosaic virus strains have a wide host range which includes common bean (*Phaseolus vulgaris*), mung bean (*Vigna radiata* var. *radiata*), lima bean (*P. lunatus*), pigeonpea (*Cajanus cajan* (L.) Millsp.), chickpea (*Cicer arietinum* L.), sweet pea (*Lathyrus odoratus* L.), lentil (*Lens culinaris* Med.), *Melilotus albus* Med., *Cucurbita sativum*, pea (*Pisum sativum*), broad bean (*Vicia faba*), *V. americana*, *V. monanthos* Desf., hairy vetch (*V. villosa* Roth.), *V. sativa* L., *V. atropurpurea* Desf., *Vigna unguiculata* ssp. *unguiculata* var. *sesquipedalis*, cowpea (*Vigna unguiculata* ssp. *unguiculata*), common clover (*Trifolium pratense*) *T. incarnatum* L., *T. hybridum* L., alfalfa (*Medicago sativa*), *M. lupulina* L., soybean (*Glycine max* (L.) Merrill), *Gladiolus* spp., *Trigonella foenum-graecum* L., *Crotalaria spectabilis*, *Lupinus*

densiflorus Benth., *Proboscidea jussieui* J.C. Keller, *Cladrastis lutea* (Michx. f.) C. Koch, *Robinia pseudoacacia* L., *Freesia* Eckl. ex Klatt sp., *Babiana* Ker-Gawl sp., *Ixia* L. sp., *Sparaxis* Ker-Gawl sp., *Tritonia* Ker-Gawl sp., *Viola* L. sp., tobacco (*Nicotiana tabacum* L.), *N. sylvestris* Speg. et Comes, and *N. rustica* L. (Bos, 1970; Jones and Diachun, 1977; Provvidenti and Hunter, 1975; Provvidenti and Schroeder, 1972; Zaumeyer and Thomas, 1957; Zettler and Abo-El-Nil, 1977). Not all BYMV strains infect or induce symptoms in these hosts.

Symptomatology

BYMV-induced infection and symptoms vary considerably, depending on the strain, host, environmental conditions, and time of infection. Initial symptoms of BYMV systemic infection appear as small chlorotic spots which gradually enlarge and coalesce to produce a general chlorosis on affected leaves (Figure 115). Young leaves may become malformed (Figure 116). Yellow and green mottling becomes more intense on leaves as they age. Infection causes shortened internodes, proliferation of branches, epinasty, and plant stunting. It also may delay maturity (Zaumeyer and Thomas, 1957).

Systemic necrosis symptoms can be induced by specific strains of BYMV. Other BYMV strains are able to incite local necrotic lesions on leaves. The typical chlorotic leaf symptoms also may be present (Cafati-K. et al., 1976; Zaumeyer and Thomas, 1957). Epinasty and early plant death may also occur (Tatchell et al., 1985). Reddish brown spots may form on infected pods which can be malformed, depending upon the specific virus strain (Zaumeyer and Thomas, 1957).

Physical properties and purification

Particles of BYMV are indistinguishable from those of BCMV because they belong to the same virus group. BYMV particles are flexuous rods (Figure 117), 750 nm in length and 15 nm in width (Varma et al., 1968). BYMV induces crystalline inclusions in both cytoplasm and nuclei; the cytoplasmic cylindrical inclusions, or

pinwheels, are typical of the potyvirus group (Bos, 1969 and 1970; de Camargo et al., 1968; Inouye, 1973; Kitajima and Costa, 1974; Tapio, 1972) (Figure 113).

Bean yellow mosaic virus has a thermal inactivation point between 50 and 60 °C and a dilution end point between 10^{-3} and 10^{-4} . Particles retain their infectiousness for one to two days and occasionally up to seven days in sap at room temperature. These properties depend upon the virus source, host plant, and experimental conditions (Bos, 1970; Musil et al., 1975; Zaumeyer and Thomas, 1957).

Purification of BYMV was difficult in early work because particles aggregate easily and also agglutinate to plant chloroplasts. Various workers have developed methods to partially purify BYMV (Bancroft and Kaesberg, 1959; Huttinga, 1973; Huttinga and Mosch, 1974). Morales (1979) developed a procedure which yields highly purified and yet natural BYMV preparations. Jones and Diachun (1977) also developed a reliable purification procedure.

Bean yellow mosaic virus and its various strains are serologically distinguishable (Beczner et al., 1976; Bercks, 1960 and 1961; Bos, 1970; Bos et al., 1974; Granett and Provvidenti, 1975; Jones and Diachun, 1977; Musil et al., 1975; Uyemoto et al., 1972; Zaumeyer and Thomas, 1957). Jones and Diachun (1977) 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.

Epidemiology

Bean yellow mosaic virus is easily transmitted mechanically and by aphids, but it is not transmitted in the seed of *P. vulgaris*. However, it can have a low transmission in the seed of *Vicia faba* and other legumes (Bos, 1970).

Aphid vectors include *Acyrtosiphon pisum* (Harris), *Macrosiphum euphorbiae* (Thomas), *Myzus persicae*, and *Aphis fabae* Scopoli (Bos, 1970; Grylls, 1972; Hagel and Hampton, 1970; Sohi,

1964; Swenson and Welton, 1966; Thottappilly et al., 1972). 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 (Evans and Zettler, 1970; Sohi, 1964; Thottappilly et al., 1972). Some BYMV strains may lose aphid transmissibility during storage or maintenance by mechanical inoculation.

Control

Plant resistance is the most reliable control measure available (Zaumeyer and Meiners, 1975). Resistance to specific strains is conditioned by plant genes such as By-2 (Dickson and Natti, 1968; Schroeder and Provvidenti, 1968). Sources of resistance to the BYMV strain inducing pod malformation have been identified in various Great Northern lines such as G.N. U.I. 31, 59, 123, and 1140. This resistance is conferred by three recessive genes with modifiers (Baggett, 1957; Baggett and Frazier, 1957; Cafati-K. et al., 1976; Guglielmetti, 1974; Provvidenti and Schroeder, 1973; Zaumeyer and Meiners, 1975). G.N. U.I. 31 also contains two recessive genes for resistance to the severe strain. Breeding for combined resistance to type and severe strains is best done by testing large F_2 populations with one strain, followed by testing progeny with the alternate strain (Tatchell et al., 1985). Resistance to BYMV strains has been found in interspecific crosses between *Phaseolus vulgaris* and *P. coccineus* (Baggett, 1956; Baggett et al., 1966; Zaumeyer and Thomas, 1957).

Cucumber Mosaic Virus

Introduction

Cucumber mosaic virus (CMV) is widely distributed throughout the world (Bird et al., 1974; Bos and Maat, 1974; Jayasinghe, 1982; Marchoux et al., 1977; Meiners et al., 1977; Milbrath et al., 1975; Zaumeyer and Thomas, 1957), affecting over 750 susceptible species in more than 80 plant families (Doine et al., 1979; Price, 1940). *Phaseolus vulgaris* is naturally infected by CMV and some commercial plantings have been noticeably affected by this virus (Bird et

al., 1975; Bos and Maat, 1974; Marchoux et al., 1977; Provvidenti, 1976; Whipple and Walker, 1941). No cultivar or germplasm accession is immune, although good levels of tolerance exist.

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

Cucumber mosaic virus can be propagated in *Nicotiana* species such as *N. clevelandii*, and assayed in local-lesion hosts such as cowpea (*Vigna unguiculata* ssp. *unguiculata*), *Chenopodium amaranticolor* Coste et Reynier, and *C. quinoa* (Francki et al., 1979).

Symptomatology

Symptoms of CMV infection may consist of a mild mosaic, vein clearing, vein banding, leaf rolling or distortion, epinasty, and/or apical necrosis. Both local and systemic symptoms are usually observed in *P. vulgaris* (Jayasinghe, 1982). 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 may also occur (Bird et al., 1974 and 1975; Milbrath et al., 1975; Provvidenti, 1976).

Physical properties

Cucumber mosaic virus is the type strain of the cucumovirus group whose isometric particles (about 28 nm in diameter) encapsidate three functional molecules of single-stranded RNA (Francki et al., 1979). CMV has a thermal inactivation point of 70 °C, a dilution end point between 10^{-4} and 10^{-5} , and is infectious in vitro for three to six days at 23 °C (Milbrath et al., 1975).

Various purification procedures have been developed (Bock et al., 1975; Bos and Maat, 1974; Francki et al., 1979; Gibbs and Harrison, 1970; Meiners et al., 1977; Murant, 1965; Scott, 1963). These procedures have enabled researchers to develop antisera to study CMV and its strains.

Transmission

Cucumber mosaic virus is transmitted mechanically, in seed, and by insect vectors such as aphids. It can be transmitted mechanically from infected beans, tobacco, cucumbers (Figure 118), and other hosts (Bird et al., 1974; Marchoux et al., 1977; Meiners et al., 1977). Seed transmission varies from less than 1% to 40%, depending upon the bean cultivar (Bird et al., 1974; Bos and Maat, 1974; Jayasinghe, 1982; Marchoux et al., 1977; Meiners et al., 1977; Provvidenti, 1976). Bos and Maat (1974) reported that CMV retained its infectiousness in stored bean seeds for 27 months.

More than 60 species of aphids may transmit CMV. They include *Aphis gossypii* and *Myzus persicae* (Meiners et al., 1977; Provvidenti, 1976). Meiners et al. (1977) report that aphids retained CMV for as long as 40 minutes after a 10-minute accession feeding period.

Control

Control measures include planting seed free of CMV and crop rotation to reduce the number of hosts for the virus and its insect vector. Chemical control may be used to reduce aphid populations in other host crops. Bean cultivars differ in their resistance, but none are highly resistant.

Soybean Mosaic Virus

The rapid expansion of soybean plantings in traditional common-bean-producing areas has increased the frequency of soybean mosaic virus infection of susceptible bean cultivars (Costa et al., 1978; Provvidenti et al., 1982).

Soybean mosaic virus is another potyvirus widely distributed because it is easily transmitted by seed and aphids (Bos, 1972). Bean cultivars can be systemically infected, showing local lesions only or systemic mosaic or necrosis. Black-seeded cultivars usually exhibit local or systemic hypersensitivity (Costa et al., 1978). Systemic symptoms in beans are usually more severe than those induced by bean common mosaic virus.

Soybean mosaic virus is mechanically transmissible and can be transmitted by several aphid species, notably *Acyrtosiphon pisum*, *Aphis fabae*, and *Myzus persicae*. The thermal inactivation point is between 55-60 °C, its dilution end point around 10^{-3} , and sap may still be infectious after three days at room temperature (Bos, 1972). The virus can be seed-transmitted in *Phaseolus vulgaris* (Castaño-J. and Morales, 1983; Provvidenti et al., 1982).

Soybean mosaic virus is best propagated in susceptible soybean (*Glycine max*) cultivars. It can be isolated by using the purification methods used for bean common or yellow mosaic viruses. Some bean cultivars such as Top Crop and Monroe, are local-lesion assay hosts (Castaño-J. et al., 1982).

Because of the lack of information on the present distribution and incidence of SMV in the main bean-growing areas, the epidemiology and control of this virus have not been investigated. However, genetic resistance will be the main control measure in the future, using the resistant bean genotypes identified so far (Costa et al., 1978; Provvidenti et al., 1982).

Alfalfa Mosaic Virus

Alfalfa mosaic virus (AMV) is an aphid-transmitted virus that was first detected on beans in United States (Zaumeyer and Thomas, 1957). The virus consists of various strains, including yellow dot, alfalfa yellow mosaic, vein necrosis, and spot mosaic (Zaumeyer, 1963; Zaumeyer and Goth, 1963; Zaumeyer and Patiño, 1960; Zaumeyer and Thomas, 1957). None of these strains of AMV is economically important (Zaumeyer and Thomas, 1957).

Alfalfa mosaic virus has been known as lucerne mosaic virus, alfalfa virus 1, alfalfa virus 2, Medicago virus 2, and *Marmor medicaginis* Holmes (Bos and Jaspars, 1971; Zaumeyer and Thomas, 1957). Although it occurs on other legumes, alfalfa mosaic virus has not been found on beans in Latin America. In Spanish, the virus and its strains are called “mosaico de la alfalfa,” “punto amarillo,” “mosaico amarillo de la alfalfa,” “necrosis venal,” “mosaico de la mancha,” and “calico.”

The virus and its strains produce a systemic mottling of leaves, necrosis of leaves or stems, and dieback of the growing point (Costa et al., 1971b). However, the most common symptom consists of local necrotic lesions which have a diameter of 0.5-3.0 mm (Zaumeyer and Thomas, 1957).

The alfalfa mosaic virus is transmitted mechanically, but apparently not in bean seed. However, it is transmitted in the seed of alfalfa (6%) and pepper (1%-5%). The virus is a bacilliform, multicomponent RNA virus (Bos and Jaspars, 1971).

Because AMV is not an economically important virus disease of beans, there are no specific control measures.

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Chapter 16

BEETLE-TRANSMITTED VIRUSES

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The beetle-borne viruses of common beans have become widely distributed in the major bean-production areas of the world. The abundance of insect vectors, the high concentration of these mechanically transmissible viruses in infected plants, and seed transmission of some of these viruses are the main epidemiological factors. Although beetle-borne viruses belong to different virus groups, they all have isometric particles, are 25-30 nm in diameter, and their beetle vectors belong to the families of Chrysomelidae, Coccinellidae, and Meloidae.

Bean Southern Mosaic Virus

Bean southern mosaic virus (BSMV) is undoubtedly the most widely distributed of the beetle-borne viruses which infect beans. This virus was first observed in southern United States (hence its name) and now is present in all the main bean-production nations of the world (Costa, 1972; Cupertino et al., 1982; Ferault et al., 1969; Jayasinghe, 1982; Murillo, 1967; Yerkes and Patiño, 1960; Zaumeyer and Thomas, 1957). BSMV can cause significant yield losses of over 50% by reducing the amount and weight of seed produced by infected bean plants. The virus has a host range restricted to legumes with the possible exception of cucumber (*Cucumis sativus* L.) (Jayasinghe, 1982). Susceptible legumes include soybean (*Glycine max* (L.) Merrill), common bean (*Phaseolus vulgaris* L.), tepary bean (*P. acutifolius* A. Gray var. *acutifolius*), lima bean (*P. lunatus* L.), pea (*Pisum sativum* L.), *Trifolium alexandrinum* L., *Cyamopsis* sp., *Melilotus indica* (L.) All., and cowpea (*Vigna unguiculata* (L.) Walp. ssp. *unguiculata*)

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(Boswell and Gibbs, 1983; Jayasinghe, 1982). The type (bean) strain infects bean, soybean, and lima bean, but not cowpea, while the cowpea strain infects cowpea, soybean, pea, and *Cyamopsis* sp., but not bean (Shepherd and Fulton, 1962). In Latin America, BSMV is known as "mosaico sureño" (Spanish) or "mosaico-do-sul" (Portuguese).

In *Phaseolus vulgaris* BSMV can induce diverse symptoms such as mosaic or mottle, rugosity, epinasty, vein yellowing, stunting, and necrotic local lesions, depending on the variety inoculated (Boswell and Gibbs, 1983; Jayasinghe, 1982; Tremaine and Hamilton, 1983). Most Pinto lines such as Pinto U.I. 114, are good local-lesion assay hosts. The cultivar Bountiful is recommended for maintaining the virus and as a propagation host. *P. acutifolius* is particularly sensitive to BSMV, exhibiting various necrotic reactions upon inoculation with this virus. Several accessions of *P. coccineus* L. (scarlet runner bean), on the contrary, proved to be resistant to BSMV (Jayasinghe, 1982). In nature, however, BSMV is often isolated from bean plants that show mild leaf mottling and moderate leaf curling (Figure 119). Southern bean mosaic virus is often encountered in a mixture with other viruses such as bean rugose mosaic virus (BRMV) or bean yellow stipple virus (BYSV).

Bean southern mosaic virus is the type member of the sobemovirus group which characteristically have isometric particles 28-30 nm in diameter and contain one molecule of positive-sense single-stranded RNA (Boswell and Gibbs, 1983; Tremaine and Hamilton, 1983) (Figure 120). These virus particles are often present inside vacuoles of an infected mesophyll cell (Jayasinghe, 1982). BSMV has a thermal inactivation point between 90 and 95 °C, a dilution end point of 10^{-5} to 10^{-6} , and longevity in vitro of over three months at room temperature. There are several purification methods for virus isolation (Boswell and Gibbs, 1983; Jayasinghe, 1982; Tremaine and Hamilton, 1983).

The virus is seed-borne and can be carried both in the embryo (Uyemoto and Grogan, 1977) or as a contaminant on the seed coat (McDonald and Hamilton, 1972 and 1973). This virus, however, becomes inactivated upon the dehydration or storage of contaminated seeds (Cheo, 1955). Secondary transmission occurs naturally by several species of chrysomelid beetles such as *Cerotoma facialis*

Erickson, *C. trifurcata* Forster, *Diabrotica adelpha* Harold, *D. balteata* Le Conte, and *Epilachna varivestis* Mulsant (Boswell and Gibbs, 1983; Fulton and Scott, 1974 and 1977; Murillo, 1967; Tremaine and Hamilton, 1983; Walters, 1964b and 1965). These insect vectors acquire the virus after feeding on infected plants for periods of less than a day and can retain it for several days afterward (Walters and Henry, 1970). The virus is also readily transmitted by mechanical means (Tremaine and Hamilton, 1983).

Bean southern mosaic virus is best controlled by planting resistant cultivars. Resistance to BSMV in *P. vulgaris* is expressed mainly as hypersensitivity rather than as immunity (Jayasinghe, 1982; Yerkes and Patiño, 1960; Zaumeyer and Thomas, 1957).

Because few existing bean cultivars are resistant, the virus is managed directly by planting virus-free seed and indirectly by chemically controlling the insect vector. Because maize is one of the preferred hosts of some chrysomelid vectors of BSMV, the common association of maize with beans sometimes aggravates the incidence of bean southern mosaic virus.

Bean Mild Mosaic Virus

Bean mild mosaic virus (BMMV) has been isolated from infected bean plants in El Salvador (Waterworth et al., 1977) and Colombia (Jayasinghe, 1982; Waterworth, 1981). This virus probably has a wider geographical range since the mild symptoms it induces are not easily recognized. In Spanish, the name of the virus is “virus del mosaico suave del frijol.”

Although BMMV alone does not seem to affect bean plants significantly, in mixed infection the virus acts synergistically, enhancing symptom expression (Jayasinghe, 1982; Waterworth et al., 1977). The bean cultivars 27 R, Top Crop, and Widusa are diagnostic hosts (Boswell and Gibbs, 1983; Waterworth, 1981).

BMMV infects several legumes: soybean (*Glycine max*), *Lablab purpureus* (L.) Sweet, *Canavalia gladiata* (Jacq.) DC., *C. ensiformis* (L.) DC., siratro (*Macroptilium atropurpureum* (DC.) Urb.), *M. lathyroides* (L.) Urb., tepary bean (*Phaseolus acutifolius* var. *acutifolius*), scarlet runner bean (*P. coccineus* L.), common bean (*P.*

vulgaris), *Rhynchosia minima* (L.) DC., and *Sesbania exaltata* (Raf.) V.L. Cory (Boswell and Gibbs, 1983; Waterworth, 1981). *Gomphrena globosa* L. and *Chenopodium quinoa* (Willd.) are susceptible to the Central American isolate of BMMV but not to the Colombian isolate (Boswell and Gibbs, 1983; Jayasinghe, 1982; Waterworth, 1981).

The symptoms induced by BMMV in *P. vulgaris* are expressed as vein yellowing and mild mosaic (Figure 121). Systemically infected plants tend to recover and latent infections are common (Boswell and Gibbs, 1983; Jayasinghe, 1982).

The bean mild mosaic virus consists of isometric particles of about 28 nm in diameter and containing single-stranded RNA. This virus is not serologically related to other viruses of similar morphology and physicochemical properties and, therefore, is still ungrouped. It has a thermal inactivation point of 84 °C, dilution end point of 10^{-8} , and longevity in vitro of 42 and 65 days for the Central American and Colombian isolates, respectively (Boswell and Gibbs, 1983; Jayasinghe, 1982; Waterworth, 1981). Crystalline virus aggregates have been observed in root phloem of infected *P. acutifolius* cells (Jayasinghe, 1982).

The bean cultivars Nep-2, Pinto, and Top Crop have been used as propagative hosts in different purification procedures (Jayasinghe, 1982; Waterworth et al., 1977). The purified virus is a good immunogen (Boswell and Gibbs, 1983; Waterworth, 1981).

The bean mild mosaic virus is readily transmitted by mechanical means, especially by contaminated tools. The virus is also transmitted by the chrysomelids *Cerotoma ruficornis* Olivier, *Diabrotica undecimpunctata howardii* Barber, *D. balteata*, *Epilachna varivestis* Mulsant, and *Gynandrobrotica variabilis* (Boswell and Gibbs, 1983; Hobbs, 1981; Waterworth, 1981; Waterworth et al., 1977). It can also be seed-borne in *P. vulgaris* (Jayasinghe, 1982).

Resistance to BMMV has been found only in *Phaseolus leptostachyus* Benth, *P. filiformis* Benth (immunity), and *P. lunatus* (hypersensitive resistance) (Boswell and Gibbs, 1983; Jayasinghe, 1982). Consequently, the current recommendations for bean mild mosaic virus control aim to reduce chrysomelid vector populations in the field.

Bean Rugose Mosaic Virus

Bean rugose mosaic virus (BRMV) was first detected in Costa Rica in 1964 (Gámez, 1972a) and, later, in Guatemala (Gámez, 1971), El Salvador (Granillo et al., 1975), Colombia, and Brazil (Kim, 1977). The economic importance of this virus is not yet known. The virus causes systemic infection in common bean (*Phaseolus vulgaris*), tepary bean (*P. acutifolius* var. *acutifolius*), *Macroptilium lathyroides*, lima bean (*P. lunatus*), broad bean (*Vicia faba* L.), *Trifolium incarnatum* L., soybean (*Glycine max*), chickpea (*Cicer arietinum* L.), and pea (*Pisum sativum*) (Gámez, 1972a). The cowpea (*Vigna unguiculata* ssp. *unguiculata*) also has been reported as susceptible to BRMV (Cartín-González, 1973).

Common names frequently used for bean rugose mosaic virus in Latin America include “mosaico rugoso,” “ampollado,” “arrugamiento,” “encarrugamiento,” and “mosaico em desenho.”

The bean rugose mosaic virus reactions in beans include systemic infection, local lesions, or immunity (Gámez, 1972a; Zaumeyer and Thomas, 1957). Severity of the systemic infection depends upon the virus strain and plant cultivar infected. In general, plants infected by BRMV exhibit a severe mosaic, rugosity, malformation, and leaf puckering (Figure 122). Pods of infected plants exhibit varying degrees of malformation and mottling, although in some cultivars mottling is not present (Cartín-González, 1973; Gámez, 1972a; Granillo et al., 1975).

Bean cultivars used as diagnostic species for BRMV are Stringless Green Refugee, Kentucky Wonder, Sure Crop Wax, Michelite, Sanilac, Potomac, Tender Green, Top Crop, Great Northern U.I. 60, Plentiful, ICA Pijao, and 27 R. Cowpea cultivars such as Monarch and Early Ramshorn, and soybean cultivars such as Lee, Hill, Hood, Improved Pelican, Hampton, Beinville, and Biloxi, have also been used. *Chenopodium amaranticolor* Coste et Reynier is a local lesion host. Many bean cultivars produce local lesions after inoculation with BRMV. The bean cultivars Colección 109 R, 27 R, and ICA Gualí have been used to propagate BRMV (Cartín-González, 1973; Gámez, 1972a).

The bean rugose mosaic virus is a comovirus with isometric particles 28-29 nm in diameter. It has three component particles,

two of which contain single-stranded RNA. The thermal inactivation point of BRMV is between 65 and 79 °C. It has a dilution end point between 10^{-4} and 10^{-5} . It remains infectious in crude extracts for 48-96 hours at 22 °C (Gámez, 1972a; Zaumeyer and Thomas, 1948). Virus particles can be found in the cytoplasm of infected cells, forming vacuolate and cytoplasmic crystalline diagnostic inclusions (de Camargo et al., 1976; Gálvez et al., 1977; Kitajima et al., 1974).

The bean rugose mosaic virus can be mechanically transmitted. However, it is disseminated in the field by insect vectors of the subfamily Galerucinae, family Chrysomelidae (Fulton et al., 1975a). Bean rugose mosaic virus is transmitted by *Cerotoma ruficornis*, *Diabrotica balteata* (Figure 123), and *D. adelpha* (Cartín-González, 1973; Fulton and Scott, 1977; Gámez, 1972a). The virus can be acquired by its vectors during feeding periods of less than 24 hours. As with many virus-vector associations, a high percentage of insects transmits the virus for as long as two days. The transmission rate then drops markedly, although occasionally some insects transmit the virus for longer periods (Fulton et al., 1975a; Selman, 1973; Walters, 1969). *Cerotoma ruficornis* can transmit the virus for as long as seven to nine days, but *D. balteata* and *D. adelpha* transmit it for only one to three days (Cartín-González, 1973; Gámez, 1972a).

Several cultivars which react with local lesions can be used as resistance sources. 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. The third determines susceptibility to systemic infection (Machado, 1973; Machado and Pinchinat, 1975). Chemical control of vectors, as for all other beetle-transmitted viruses, is possible.

Bean Pod Mottle Virus

Bean pod mottle virus (BPMV) is known to occur in North America. The bean cultivars Pinto, Black Valentine, and Bountiful have been suggested as diagnostic hosts (Boswell and Gibbs, 1983). Other susceptible plant species are *Chenopodium quinoa*, pea (*Pisum sativum*), *Sesbania exaltata*, *Canavalia ensiformis*, lentil (*Lens culinaris* Med.), and lima bean (*Phaseolus lunatus*) (Boswell and Gibbs, 1983; Moore and Scott, 1971).

The bean pod mottle virus significantly affects yield because it characteristically induces malformation of pods and seed abortion (Zaumeyer and Thomas, 1948 and 1957). Leaf blistering and puckering are not diagnostic of BPMV infections. Systemic mottling, stunting, and leaf and pod distortion are symptoms commonly associated with BPMV-infected natural hosts such as common bean (*Phaseolus vulgaris*), soybean (*Glycine max*), and *Desmodium paniculatum*.

The bean pod mottle virus belongs to the comovirus group whose members possess isometric particles 28 nm in diameter and two genome segments of single-stranded RNA, encapsidated in different particles. BPMV has a thermal inactivation point around 70 °C, a longevity in vitro of 62-93 days, and a dilution end point of 10^{-4} . *Glycine max*, Black Valentine, Cherokee Wax, and Bountiful have been used as propagative hosts to isolate the virus (Bancroft, 1962; Boswell and Gibbs, 1983; Moore and Scott, 1971; Zaumeyer and Thomas, 1948). Diagnostic virus-induced inclusions in infected cells have been found only in myelinic bodies and osmiophilic globules, and then only as a few virus particles (Kim and Fulton, 1971 and 1972; Kim et al., 1974).

The virus is transmitted by mechanical means and by beetle vectors such as *Cerotoma trifurcata*, *Diabrotica balteata*, *D. undecimpunctata howardii*, *Colaspis flavida*, *C. lata*, *Epicauta vittata*, and *Epilachna varivestis* (Boswell and Gibbs, 1983; Fulton and Scott, 1974; Fulton et al., 1975a; Horn et al., 1970; Moore and Scott, 1971; Patel and Pitre, 1971; Ross, 1963; Walters, 1964a). BPMV is not seed-borne (Boswell and Gibbs, 1983).

Several sources of resistance are available in *P. vulgaris* which confer immunity or resistance to BPMV (Thomas and Zaumeyer, 1950). Chemical control of the beetle vectors is also recommended in cases where this measure is economically feasible.

Bean Curly Dwarf Mosaic Virus

Bean curly dwarf mosaic (BCDMV) was first isolated from beans in El Salvador in 1971 and detected in Guatemala in 1985. No estimates of yield losses are available but BCDMV reportedly occurred in 1%-15% of plants in bean fields in El Salvador. The host

range of BCDMV includes common bean (*Phaseolus vulgaris*), tepary bean (*P. acutifolius* var. *acutifolius*), lima bean (*P. lunatus*), pigeonpea (*Cajanus cajan* (L.) Millsp.), chickpea (*Cicer arietinum*), *Crotalaria juncea* L., soybean (*Glycine max*), *Lathyrus sativus* L., lentil (*Lens culinaris* Med.), *Macroptilium lathyroides*, pea (*Pisum sativum*), *Sesbania exaltata*, broad bean (*Vicia faba*), and mung bean (*Vigna radiata* (L.) Wilczek var. *radiata*) (Meiners et al., 1977).

Susceptible hosts show a range of symptoms, depending upon the cultivar (Figure 124) and stage of plant development. Plants infected at an early stage of development are extremely stunted and produce no yield. Older plants 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 (Meiners et al., 1977).

The bean curly dwarf mosaic virus is a comovirus serologically related to quail pea mosaic virus but not to bean rugose mosaic virus (Waterworth et al., 1974). BCDMV particles are 25-28 nm in diameter and infectious in dilutions as weak as 1×10^{-5} in 0.025 M phosphate buffer. Dilutions are still infectious after incubation at room temperature for three weeks or after heating at 50 °C for 10 minutes (Meiners et al., 1977). A purification method is available (Walters, 1958).

The bean curly dwarf mosaic virus may be transmitted by the spotted cucumber beetle (*Diabrotica undecimpunctata howardii*), Mexican bean beetle (*Epilachna varivestis*), banded cucumber beetle (*D. balteata*), and flea beetle (*Cerotoma ruficornis*) (Meiners et al., 1977; Waterworth et al., 1977). Recently, two other genera, *Gynandrobrotica* and *Paranapiacaba* have also been shown to transmit BCDMV (Hobbs, 1981). The spotted cucumber beetle and Mexican bean beetle retained BCDMV infectiousness for two and three days, respectively, after a 24-hour accession feeding. BCDMV is also transmitted mechanically and by seed (Meiners et al., 1977).

Studies in El Salvador suggest that insect vectors transmit the viruses to beans from infected wild plant species growing on the

edge of fields: the incidence of virus-infected plants is less in the center of bean fields than in the outer edges (Meiners et al., 1977). BMMV commonly occurs in mixture with BCDMV (Figure 125). Its economic importance depends on the combined infection with other viruses (Waterworth et al., 1977) or on the susceptibility of certain bean genotypes which react to BCDMV with systemic necrosis. No control measures are reported for bean curly dwarf mosaic virus but chemical control of vectors should be effective.

Bean Yellow Stipple Virus

Bean yellow stipple virus (BYSV) was first isolated in Illinois in 1948 (Zaumeyer and Thomas, 1950) and later in Costa Rica and Cuba in 1972 and 1978, respectively (Gámez, 1972b and 1976). BYSV is synonymous with cowpea chlorotic mottle virus (CCMV) which occurs in southern United States, Mexico, and probably in Central America (Fulton et al., 1975b). There are no studies of its economic importance in beans.

Only leguminous species have been reported susceptible to systemic infection by BYSV. Susceptible plants include common bean (*Phaseolus vulgaris*), tepary bean (*P. acutifolius* var. *acutifolius*), lima bean (*P. lunatus*), *Vigna umbellata* (Thunb.) Ohwi et Ohashi, *V. aconitifolia* (Jacq.) Maréchal, *Macroptilium lathyroides* (L.) Urb., cowpea (*Vigna unguiculata* (L.) Walp. ssp. *unguiculata*), *V. unguiculata* ssp. *unguiculata* var. *sesquipedalis* (L.) Verdc., *V. hirta*, soybean (*Glycine max*), *G. javanica*, and pigeonpea *Cajanus cajan* (L.) Millsp. (Gámez, 1976; Kuhn, 1964; Walters, 1958). In other studies, *Cyamopsis tetragonoloba* (L.) Taub., urd bean (*Vigna mungo* (L.) Hepper), and pea (*Pisum sativum*) also were susceptible (Zaumeyer and Thomas, 1950).

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

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 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 (Gámez, 1972b and 1976; Zaumeyer and Thomas, 1950).

Bean cultivars susceptible to BYSV include Stringless Green Refugee, Pinto U.I. 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 produce local necrotic lesions and include *Lablab purpureus* (L.) Sweet, soybean (*Glycine max*), *Crotalaria juncea*, and *C. paulina*. *Lablab purpureus* has been used in studies on virus infectiousness. *Chenopodium amaranticolor* and *C. album* L. react with whitish local lesions. The bean cultivars Colección 109 R and Pinto U.I. 78 have been used to multiply the virus (Gámez, 1976; Zaumeyer and Thomas, 1950).

Bean yellow stipple virus is a member of the bromovirus group (Harrison et al., 1971; Lane, 1974) with isometric particles 26-30 nm in diameter (Gámez, 1972b and 1976). The virus has a thermal inactivation point of 76 °C, a dilution end point between 10^{-5} x 10^{-4} , and a longevity in vitro of five days at 18 °C and one day at 20 °C (Gámez, 1976; Zaumeyer and Thomas, 1950). Purification procedures have been described (Gámez, 1971). BYSV induces amorphous and filamentous inclusions as well as membranous vesicles which contain virus particles (Kim, 1977).

Bean yellow stipple virus is not seed transmitted (Gámez, 1976; Zaumeyer and Thomas, 1957), but is easily transmitted mechanically. Dissemination occurs principally through beetle vectors such as *Cerotoma ruficornis* and *Diabrotica balteata*. 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* retains it 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 (Gámez, 1976).

All bean cultivars tested experimentally are susceptible (Gámez, 1976; Zaumeyer and Thomas, 1950). Control of insect vectors is an

effective method of reducing virus incidence when it becomes economically important.

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Chapter 17

WHITEFLY-TRANSMITTED VIRUSES

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Introduction

Whiteflies belong to the order Homoptera, family Aleyrodidae, and are currently reported to transmit 28 different plant viruses (Gibbs and Harrison, 1976; Varma, 1963). The whitefly species that are vectors of plant viruses include *Bemisia tabaci* Gennadius (*B. inconspicua* (Quaintance)), *B. lonicerae* Takahashi, *B. manihotis* Frappa, *B. tuberculata* Bandar, *B. vayssieri* Frappa, *Aleurotrachelus socialis* Bondar, *Aleurothrixus floccosus* Maskell, *Trialeurodes abutiloneus* (Haldeman), *T. natalensis* Corbett, and *T. vaporariorum* (Westwood) (Bird and Maramorosch, 1978; Costa, 1969 and 1976b; Mound, 1973; Russell, 1957). However, only the whiteflies *B. tabaci*, *T. abutiloneus*, and *T. vaporariorum* are confirmed as vectors of plant viruses (Harris, 1981).

Bemisia tabaci, the common whitefly, is the most prevalent whitefly vector of plant viruses. It exhibits considerable variability in its feeding and reproductive habits on different plant species. Flores and Silberschmidt (1958) and Russell (1975) attribute this variation to the existence of biotypes, while Bird (1957, 1958, and 1962) and Bird and Sánchez (1971) refer to them as races: *B. tabaci* race *jatrophae* and race *sidae*. However, the strong host preference behavior of *B. tabaci* must be taken into account (Mound, 1973).

Very few whitefly-transmitted agents have been isolated and proved to be viruses. Bird et al. (1975a) suggested that the diseases associated with whitefly-transmitted agents should be considered as rugaceous diseases.

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In beans, two important, apparently related but different, diseases have been consistently associated with the common whitefly vector *B. tabaci*: bean golden mosaic and bean dwarf mosaic.

Bean Golden Mosaic Virus

Introduction

Bean golden mosaic was first reported in 1961, in Brazil (Costa, 1965), as a minor disease in the State of São Paulo. It has since been recorded in the major bean-production areas of Brazil, including Minas Gerais, Paraná, and Goiás. The disease also occurs in other bean-production regions of Latin America such as El Salvador, Guatemala, Nicaragua, Costa Rica, Panama (Gámez, 1969 and 1970), Puerto Rico (Bird and López-Rosa, 1973; Bird et al. 1972 and 1973), Jamaica, Dominican Republic (Abreu-Ramírez, 1978; Pierre, 1975; Schieber, 1970), Colombia (Gálvez et al., 1975), Cuba (Blanco-Sánchez and Bencomo-Pérez, 1978 and 1981), Belize, Mexico (Yoshii, 1981), Honduras, and Venezuela.

Bean golden mosaic is also known as bean yellow mottle, bean golden-yellow mosaic, bean yellow mosaic, and bean double-yellow mosaic (Bird and López-Rosa, 1973; Bird et al., 1972 and 1973; Crispín-Medina and Campos-Avila, 1976; Crispín-Medina et al., 1976; Schieber, 1970; Zaumeyer and Smith, 1964 and 1966). The Spanish and Portuguese names for bean golden mosaic are “mosaico dorado del frijol” and “mosaico dourado do feijoeiro,” respectively.

Bean golden mosaic is now an economically important disease in Latin America, especially Brazil, parts of Central America, and the Caribbean. Brazilian bean production has been severely reduced by the disease since 1972. Its increasing seriousness has been attributed to increasing whitefly populations associated with the expanding soybean production in bean-growing areas. Soybean is a preferred host of the vector (Costa, 1975a; Costa et al., 1975b).

Various workers (Caner et al., 1981; Costa and Cupertino, 1976; de Almeida et al., 1984; Ferraz et al., 1980; Gámez, 1972; Menten et al., 1980; Pierre, 1972 and 1975) report that infection by

BGMV reduces the number of pods, number of seeds per pod, and seed weight. Reported yield losses were 57% in Jamaica (Pierre, 1972 and 1975), 48%-85% in Brazil (Costa and Cupertino, 1976; Menten et al., 1979), 40%-100% in Guatemala (Ordoñez-Matzer and Yoshii, 1978), and 52%-100% in El Salvador (Cortez and Díaz, personal communication). Yield losses vary considerably, depending on plant age at the time of infection, varietal differences, and, possibly, viral strain (Costa, 1975a).

The host range of BGMV includes common bean (*Phaseolus vulgaris* L.), lima bean (*P. lunatus* L.), tepary bean (*P. acutifolius* A. Gray var. *acutifolius*), *P. polystachyus* (L.) B.S.P., *Macroptilium longepedunculatum* (Benth.) Urban, the ancestral form of common bean (*P. vulgaris* var. *aborigeneus* (Burk.) Baudet), scarlet runner bean (*P. coccineus* L.), *Macroptilium erythroloma* (Benth.) Urb., *M. lathyroides* (L.) Urb., *Teramnus uncinatus* (L.) Sw., mung bean (*Vigna radiata* (L.) Wilczek var. *radiata*), cowpea (*V. unguiculata* (L.) Walp. ssp. *unguiculata*), and *Calopogonium mucunoides* Desv. (Abreu-Ramírez and Gálvez, 1979; Agudelo-S., 1978; Bird and López-Rosa, 1973; Bird and Maramorosch, 1978; Bird et al., 1972 and 1975a; Chagas et al., 1981; CIAT, 1973, 1975, 1976, 1978 to 1981, and 1983 to 1985; ; Costa, 1965, 1975a, 1975b, 1976a, and 1976b; Díaz-Chávez, 1972; Flores and Silberschmidt, 1966; Gámez, 1971; ICTA, 1976; Pierre, 1975; Williams, 1976; Yoshii et al., 1979a).

Symptomatology

Most susceptible bean genotypes exhibit a brilliant yellow coloring, starting in leaf veins (Figure 126). Symptoms may appear in the first trifoliolate leaves within 14 days after planting. Bird et al. (1975a) 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.

Susceptible cultivars exhibit a marked rugosity and distortion of leaves, many of which may be completely yellowed or, at times, almost bleached (Figure 127). Some cultivars present symptoms that are less intense and may exhibit some recuperation at a later stage of development.

Pods of infected plants are considerably malformed (Figure 128). Seeds may be discolored, malformed, and reduced in size and weight (Costa, 1975a; Gámez, 1969 and 1970). Some plants infected at an early stage may be severely stunted and often do not produce any pods.

The symptomatology of BGMV is similar to that of lima bean golden mosaic virus in Africa (Williams, 1976) and lima bean yellow mosaic in India. However, the Indian virus differs in its host range (Nene et al., 1972; Rath and Nene, 1974). Mung bean yellow mosaic, urd bean yellow mosaic, and yellow mosaic of *Lablab purpureus* (L.) Sweet likewise have a similar symptomatology (Nair et al., 1974; Nariani, 1960; Nene et al., 1972; Ramakrishnan et al., 1973; Zaumeyer and Thomas, 1957). However, they are not able to infect the majority of *Phaseolus vulgaris* cultivars (Ramakrishnan et al., 1973).

Electron microscopic evaluations of infected bean tissue reveal that the principal cellular symptom is a dramatic change in chloroplast morphology, particularly in the lamellar system (Kitajima and Costa, 1974). Recently Kim et al. (1978) 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 nucleic of infected cells. Distinct changes in the nucleoli also occur—evident as a segregation of granular complexes and fibrils which may fill as much as 75% of the nuclear volume (Goodman and Bird, 1978).

Physical properties

The viral etiology of bean golden mosaic was demonstrated recently by Gálvez and Castaño (1976) and Goodman (1977b). They observed that fixed BGMV consisted of icosahedral particles united in pairs (dimer particles or geminates). The bonded particles are flattened at their point of union (Figure 129) and measure 19 by 32 nm, while individual particles have a diameter of 15-20 nm. Matyis et al. (1976) reported individual particles measured 12-13 nm in diameter. A similar particle morphology was found for viruses causing tomato golden mosaic, euphorbia mosaic (Matyis et al., 1975 and 1976), BGMV of beans in Brazil, Colombia, El Salvador,

Dominican Republic, Guatemala, Mexico, and BGMV of *P. lunatus* in Nigeria (Gálvez et al., 1977).

BGMV particles have a thermal inactivation point of 50-55 °C (Gálvez and Castaño, 1976), a final dilution end point of 10^{-1} (Gálvez and Castaño, 1976) to 10^{-2} (Bird et al., 1977a and 1977b), and an in vitro longevity of 48 hours at room temperature (Gálvez and Castaño, 1976). Goodman and co-workers (1977a and 1977b; Goodman and Bird, 1978; Goodman et al., 1977) determined that the particles have a sedimentation coefficient value of 69 S, a particle mass of 2.6×10^6 daltons, a 260 nm absorbance value of 7.7, and a 260/280 absorbance ratio of 1.4. The genome of BGMV consists of two circular molecules of single-stranded DNA, each of which has a molecular weight of about 7.5×10^5 (Goodman, 1977a and 1977b; Goodman and Bird, 1978; Goodman et al., 1980; Haber et al., 1981; Harrison, 1985). BGMV contains a predominant protein species with a molecular weight of 27,400 (Goodman et al., 1980).

Matthews (1979) included BGMV in a new virus group called the geminivirus, based upon its particle characterization, physiochemical properties, and single-stranded DNA.

Transmission and epidemiology

Most BGMV isolates can be transmitted artificially by mechanical inoculation (Costa, 1969 and 1976b; Meiners et al., 1975), the exception being the Brazilian isolates of BGMV (Matyis et al., 1976). Successful inoculation required a high temperature of 30 °C. At 24-28 °C the transmission rate was only 30%; no transmission occurred below 21 °C.

Nearly 100% transmission can be obtained under greenhouse conditions at 27 °C with BGMV inoculum extracted from plants infected 12-20 days earlier in cold 0.1M phosphate buffer at pH 7.5. Transmission is significantly reduced when older plants are used as inoculum. Bird et al. (1977b) used a similar buffer at pH 7.0 to obtain 100% transmission by inoculation with an airbrush at 80 lb/sq. in.

BGMV is not transmissible in seed from infected bean plants, for example, Pierre (1975) tested seed from 300 infected bean plants,

and Costa (1965, 1975a, 1975b, and 1976b) tested seed from 350 infected lima bean plants. None of these seeds was infected by BGMV.

The natural mode of BGMV transmission is through the vector, the common whitefly (*Bemisia tabaci*). Nene (1973) studied the biology of whiteflies in relation to legumes such as mung bean (*Vigna radiata* (L.) Wilczek var. *radiata*), urd (*Vigna mungo* (L.) Hepper), and soybean (*Glycine max* (L.) Merrill). The insect can produce 15 generations a year during which time populations may be restricted to a single crop species or may migrate to other plant species. A whitefly lays 30-150 eggs (Figure 130) during its life cycle which, in India, lasts 13-20 days during March to October (monsoon season) or 24-72 days during November to March (dry season). Populations of whiteflies are reduced as the urd bean crop matures and may migrate to other plants such as crucifers, lentils, and peas.

The life cycle on cotton in India (Russell, 1975) varies from 14 to 107 days. It is shortest during April to September (14-21 days), and is longer during November to February (69-72 days). Most oviposition occurred at temperatures higher than 26.5 °C and none 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 (Costa, 1969 and 1976b; Nene et al., 1972).

Costa (1969) states that whitefly-transmitted viruses are not acquired as rapidly as aphid-transmitted viruses and that inoculation efficiency increases with prolonged virus acquisition periods. Whitefly-transmitted viruses have a defined but short incubation period and are sometimes retained for life in the insect vector. Whitefly adults can acquire and transmit BGMV within 5 minutes (Arévalo-R. and Díaz-Ch., 1966; Bird et al., 1972; Gámez, 1971). The inoculation efficiency increases as population size increases per infected plant (Arévalo-R. and Díaz-Ch., 1966; Bird and Maramorosch, 1978; Costa, 1969 and 1976b; Gámez, 1971; Varma, 1963). Gámez (1971) found an average acquisition and incubation period of three hours for each vector. The retention period varies according

to the acquisition period but may last 21 days or the entire life of the whitefly (Arévalo-R. and Díaz-Ch., 1966; Bird et al., 1975a; Costa, 1969 and 1976b; Gámez, 1971; Varma, 1963). The insects occasionally have been observed to lose their transmission capacity (Gámez, 1971).

Immature forms (Figure 131) can acquire the mung bean yellow mosaic virus which then persists through pupation and can be transmitted during the adult stage. In one study at least 50% of transmission occurred from adults (Figure 132) which in immature form had fed on infected plants (Nene et al., 1972; Rathi and Nene, 1974). Costa (1976b) reported that female whiteflies were more efficient than males as vectors of BGMV to *Phaseolus vulgaris*, *P. acutifolius*, and *P. polystachyus*. However, males were more efficient vectors for *P. lunatus* and *Macroptilium longepedunculatum*.

BGMV is not seed-transmitted and probably persists in wild and cultivated hosts, particularly legumes (Costa, 1975b and 1976b; Díaz-Ch., 1972; Gámez, 1971; Pierre, 1975). Pierre (1975) considers that, in Jamaica, lima beans, *Macroptilium lathyroides* and poinsettias (*Euphorbia pulcherrima* Willd. ex Klotzsch) are natural hosts for BGMV. In Brazil, the increased production of soybeans has greatly increased whitefly populations and therefore BGMV incidence in beans (Costa, 1975a; Costa et al., 1975b). Tobacco, tomato, and cotton plantings in El Salvador and Guatemala are responsible for the high whitefly populations in those countries (Alonzo-Padilla, 1975 and 1976; CIAT, 1973, 1975, 1976, 1978 to 1981, and 1983 to 1985; Granillo et al., 1975).

In Latin America, bean golden mosaic virus is usually prevalent in elevations below 1500 m (Bird and Maramorosch, 1978; Costa, 1975a). At these altitudes whitefly populations and temperatures are higher and inoculum sources are more numerous. In Jamaica, Cuba, and the Dominican Republic, BGMV incidence is less during November to March when temperatures and insect vector populations are lower. In Brazil, BGMV is more common and severe at elevations between 400-800 m and toward 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 are growing (Costa, 1965 and 1975a; Vetten and Allen, 1983).

Control by cultural practices

The incidence of bean golden mosaic virus is reduced considerably when beans are planted far from crops such as soybean (Menten and Roston, 1980), cotton, and tobacco. These crops, although not susceptible to BGMV, produce large whitefly populations which transmit the virus.

Changing the date of planting where possible, so that young bean plants develop during periods of lower temperatures and higher moisture, will reduce the presence of the whitefly vector of BGMV (Alonzo-Padilla, 1975 and 1976; Blanco-Sánchez and Bencomo-Pérez, 1978; Costa, 1965 and 1975a; Costa et al., 1975b; Granillo et al., 1975; Pierre, 1975).

There are no economical and practical biological control measures currently available (Nene et al., 1972; Sifuentes-A., 1978). Plant mulches can reduce whitefly populations (Avidov, 1957) but are not practical.

Control by chemicals

Bean golden mosaic virus can be controlled by applying insecticides to reduce the number of viruliferous whiteflies.

Systemic insecticides such as carbofuran and aldicarb, effectively control whitefly populations when applied at planting time (Alonzo-Padilla, 1976). Substantial yield increases were obtained in the Dominican Republic by applying carbofuran (2.5 g/m row) at planting, followed by 0.15% monocrotophos applied at 6, 15, and 30 days after plant emergence (Abreu-Ramírez and Gálvez, 1979; Abreu-Ramírez et al., 1979; Méndez et al., 1976; Peña and Agudelo-S., 1978; Peña et al., 1976). Ideally, chemical control is combined with other measures such as cultural practices, to be economically feasible and to achieve a higher level of protection.

Control by plant resistance

Plant resistance can provide an economical method of disease control. However, of more than 10,000 accessions of *Phaseolus vulgaris* and some accessions of *P. lunatus*, *P. acutifolius*, and *P. coccineus* evaluated under field and laboratory conditions, not one single accession proved immune to BGMV (Abreu-Ramírez et al., 1979; CIAT, 1973, 1975, 1976, 1978 to 1981, and 1983 to 1985; Costa, 1965 and 1975a; Costa et al., 1975a; Gámez, 1969, 1970, and 1971; Pierre, 1975; Yoshii et al., 1979a). However, some accessions exhibited a low to moderate level of disease resistance or tolerance. These were, among others, Porrillo Sintético and Porrillo 70, Turrialba 1, ICA Pijao, ICA Tui, Venezuela 36, and Venezuela 40. Various *P. coccineus* accessions from the Instituto de Ciencia y Tecnología Agrícolas (ICTA) germplasm bank are tolerant in Guatemala. They include Guatemala 1278, 1279, 1288, 1291, 1296, 1299, M 7689-A, and M 7719 (CIAT, 1973, 1975, 1976, 1978 to 1981, and 1983 to 1985; ICTA, 1976; Yoshii et al., 1979a and 1979b).

Pompeu and Kranz (1977) 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. Tulmann-Neto et al. (1976, 1977a, and 1977b) obtained a 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 Turrialba 1, but it is not as agronomically acceptable.

The tolerance of Turrialba 1, Porrillo 1, and ICA Pijao has been confirmed in Guatemala, El Salvador, and in the Dominican Republic, under moderate to high disease pressure in bean nurseries interplanted between tomatoes, tobacco, cotton, and soybeans to favor high whitefly populations (Figure 133).

These tolerant materials have been successfully used in breeding programs which have already produced black-seeded cultivars such as ICTA Quetzal in Guatemala and Negro Huasteco in Mexico (CIAT, 1973, 1975 to 1978, and 1984). These cultivars can produce as much as 1500 kg/ha under moderate disease pressure.

Bean Dwarf Mosaic Virus

Introduction

The name "bean dwarf mosaic" (BDMV) is given here to a disease previously known as "bean chlorotic mottle." This disease is widespread in tropical bean-growing areas where the whitefly vector exists (Agudelo-S., 1978; Bird, 1958; Bird and López-Rosa, 1973; Bird and Maramorosch, 1978; Bird and Sánchez, 1971; Bird et al., 1970; Costa, 1976b; Costa and Bennett, 1953; Crandall, 1954; Granillo et al., 1975; Jayasinghe, 1982). However, its incidence has been low in most regions, with notable exceptions such as Argentina where thousands of hectares have been affected. Infected bean plants produce severely malformed pods or, often, no pods at all (Costa, 1975a).

The causal virus (BDMV) is believed to be a variant of abutilon mosaic virus (AbMV) that adapted to beans. It is possible that more than one variant or strain of AbMV can affect beans. The reputed host range includes common bean (*Phaseolus vulgaris*), lima bean (*P. lunatus*), *Abutilon hirtum* Sweet, hollyhock (*Althaea rosea* (L.) Cav.), *Bastardia viscosa* HBK., *Corchorus aestruans* L., *Gossypium barbadense* L., *G. esculentum* Mill., *Hibiscus brasiliensis* L., okra (*H. esculentus* L.), *Malva parviflora* L., *Malva sylvestris* L., *Malvaviscus* Adans. sp., *Sida acuminata* DC., *S. aggregata* Presl., *S. bradei* Ulbricht, *S. carpinifolia* Mast., *S. cordifolia* 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 physalodes* (L.) Gaertn., *Nicotiana glutinosa* L., tobacco (*N. tabacum* L.), potato (*Solanum tuberosum* L.), peanut (*Arachis hypogaea* L.), *Canavalia ensiformis* (L.) DC., *Cyamopsis tetragonolobus* (L.) Taub., soybean (*Glycine max* (L.) Merr.), lentil (*Lens culinaris* Med.), *Lupinus albus* L., and pea (*Pisum sativum* L.) (Bird, 1958; Bird and López-Rosa, 1973; Bird and Maramorosch, 1978; Bird and Sánchez, 1971; Bird et al., 1970 and 1975a; Costa, 1954, 1955, and 1965; Costa and Carvalho, 1960a and 1960b; Crandall, 1954; Debrot-C. and Ordosgoitti-F., 1975; Flores and Silberschmidt, 1963; Flores et al., 1960; Granillo et al., 1975; Kitajima and Costa, 1974; Owen, 1946; Silberschmidt and Flores, 1962; Silberschmidt and Tomasi, 1955 and 1956).

Symptomatology

BDMV can cause a severe dwarfing characterized by proliferation of buds and a bunchy or rosette type of plant development. In some plants a witches' broom is produced besides the characteristic chlorotic mottling (Figure 134). Chlorotic spots or mottled areas may be produced on leaves of tolerant cultivars or older susceptible plants (Figure 135). These spots may be accompanied by a rugosing of leaves (Figure 136). Severely affected plants produce few or no pods. Figure 137 illustrates AbMV symptoms produced in an infected *Pavonia sidaefolia* plant, and Figure 138 illustrates symptoms of infectious chlorosis of Malvaceae in *Malva* sp.

Physical properties

Since BDMV has not been isolated yet, its physicochemical properties are not completely known. Kitajima and Costa (1974) observed isometric particles 20-25 nm in diameter in infected tissue of *Sida micrantha*. Costa and Carvalho (1960a and 1960b) determined that AbMV has a thermal inactivation point of 55-60 °C, a final dilution end point of 5-6, and retains its infectiousness for 48-72 hours in vitro.

Transmission and epidemiology

Mechanical transmission of AbMV is very difficult but has been accomplished by Costa and Carvalho (1960a and 1960b) 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. (1975a) were unable to transmit AbMV mechanically and had difficulties with its natural vector, the common whitefly (*Bemisia tabaci* race *sidae*).

Whiteflies have been demonstrated to transmit BCMV and AbMV to beans (Bird, 1958; Bird et al., 1975a; Costa, 1954, 1955, 1965, 1975a, and 1976b; Costa and Bennett, 1953; Flores and Silberschmidt, 1958; Orlando and Silberschmidt, 1946; Silberschmidt and Ulson, 1954; Silberschmidt et al., 1957). Bird et al. (1975a) showed that whiteflies can acquire the virus during a 15- to

20-minute feeding period and retain their ability to transmit AbMV for seven days. Costa (1975a) showed that, via the whitefly, AbMV is easily transmitted from *Sida* sp. to beans but with difficulty from beans to beans.

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 BCMV also may occur in beans when large plantings of other susceptible crops such as soybeans and cotton, are planted nearby (CIAT, 1973, 1975, 1976, 1978 to 1981, and 1983 to 1985; Costa, 1965; Yoshii, 1975).

Control

The epidemiology of BCMV is similar in all respects to that of BGMV. The same integrated control approach is therefore recommended, including chemical control of the common whitefly (*B. tabaci*). Although Costa (1965 and 1976b) could not identify any resistance within *Phaseolus vulgaris* in Brazil, several bean genotypes have shown field resistance in Argentina and at the Centro Internacional de Agricultura Tropical (CIAT) in Colombia. Resistance was also found in other species such as *Vigna angularis* (Willd.) Ohwi et Ohasi, mung bean (*V. radiata* (L.) Wilczek var. *radiata*, *V. umbellata* (Thunb.) Ohwi et Ohashi, *V. radiata* var. *sublobata* (Roxb.) Verdc. (Costa, 1965). Much additional research is required to verify the resistance of these materials and characterize the virus.

Euphorbia Mosaic Virus

Introduction

Euphorbia mosaic virus (EMV) was isolated in 1950 from *Euphorbia prunifolia* Jacq. (Costa and Bennett, 1950) and has since been observed in many species of *Euphorbia*. The virus has been detected in beans in Brazil but is not economically important. Common names frequently used for EMV in Latin America include "mosaico de las euforbiáceas" and "encarquilhamento da folha."

The host range of EMV includes *Euphorbia prunifolia*, *Datura stramonium*, tomato (*Lycopersicon esculentum* Mill.), *Nicandra physalodes*, *Nicotiana glutinosa*, *Canavalia ensiformis*, soybean (*Glycine max*), lentil (*Lens culinaris*), and common bean (*Phaseolus vulgaris*) (Bird et al., 1975a, 1975b, and 1977a; Costa, 1965, 1975a, and 1976b; Costa and Carvalho, 1960a; Meiners et al., 1975).

Symptomatology

The euphorbia mosaic virus usually 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 as green tissue grows asymmetrically around the initial necrotic lesions (Figure 139). Abnormal development of axillary buds also may occur and plants are commonly stunted.

Physical properties

Matyis et al. (1975 and 1976) partially purified EMV and reported that it consists of identically paired particles that are 25 nm in diameter and individual isometric particles that are about 12-13 nm in diameter. They suggested that EMV belongs to the gemini-virus group.

Costa and Carvalho (1960a and 1960b) reported that EMV in sap has a thermal inactivation point of 55-60 °C and retains its infectiousness in vitro for more than 48 hours. Bird et al. (1977a) also reported that EMV has a thermal inactivation point of 55-60 °C but retains its infectiousness in vitro for less than 24 hours and has a dilution end point of 10^{-3} . Infectiousness 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. to *Datura* sp. at a rate of 31% and easily between *Datura* sp. (Bird et al., 1975b and 1977a; Costa and Carvalho, 1960a and 1960b). The virus was also transmitted between two bean varieties (Meiners et al., 1975). EMV is not seed transmitted (Bird et al., 1975a; Costa, 1975a).

The common whitefly (*Bemisia tabaci*) can acquire the virus after a 10-minute feeding period, but requires a 20-minute incubation period for transmission. The whitefly vectors can retain their infectiousness for 20 days (Bird et al., 1975a; Costa, 1965 and 1976b; Costa and Bennett, 1950).

Euphorbia mosaic virus is seldom 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 BCMV or AbMV (Costa, 1965, 1975a, and 1976b). However, plant resistance has been identified in accessions of *Vigna angularis*, *V. radiata* var. *radiata*, *V. umbellata*, and *V. radiata* var. *sublobata*.

Rhynchosia Mosaic Virus

Introduction

Rhynchosia mosaic virus (RMV) was isolated in Puerto Rico. It produces symptoms similar to those reported for infected *Rhynchosia minima* (L.) DC. in other tropical countries (Bird, 1962; Bird and López-Rosa, 1973; Bird and Maramorosch, 1978; Bird and Sánchez, 1971; Bird et al., 1975a; Maramorosch, 1975). Symptoms of RMV are similar to those caused by BDMV 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 splendens* F. Sellow ex Roem. et Schult., pigeonpea (*Cajanus cajan* (L.) Millsp.), *Canavalia ensiformis*, *C. maritima* (Aubl.) Thou., *Crotalaria juncea* L., soybean (*Glycine max*), *Macroptilium lathyroides*, *Pachyrhizus erosus* (L.) Urban, ancestral form of common bean (*Phaseolus*

vulgaris var. *aborigeneus*), tepary bean (*P. acutifolius*) cv. P.I. Wright and variety *acutifolius*, scarlet runner bean (*P. coccineus*), lima bean (*P. lunatus*), *Vigna longifolia* (Benth.) Verdcourt, common bean (*P. vulgaris*), *Rhynchosia minima*, *R. reticulata* (Sw.) DC., *Vigna aconitifolia* (Jacq.) Maréchal, *V. angularis* (Willd.) Ohwi et Ohashi, okra (*Hibiscus esculentus* L.), cotton (*Gossypium hirsutum* L.), *Malachra capitata* L., *Oxalis berrelieri* L., *Nicotiana acuminata* (R.C. Grah.) Hook, *N. alata* Link and Otto, *N. bonariensis* Lehmann, *N. glutinosa*, *N. nightiana* Goodspeed, *N. maritima* H.M. Wheeler, *N. paniculata* L., and tobacco (*N. tabacum*) (Bird, 1962; Bird et al., 1975a).

Symptomatology

Rhynchosia mosaic virus infection of beans causes symptoms such as leaf malformation, yellowing (Figure 140), witches' broom, and plant stunting. When infection occurs in young plants, symptoms are proliferation of flowers and branches and little, if any, seed production (Bird and Sánchez, 1971).

The virus has not yet been isolated to study its physical properties.

Transmission and epidemiology

Mechanical transmission (18%) has been demonstrated by using the tobacco cultivar, Virginia 12, as source of inoculum (Bird and López-Rosa, 1973; Bird et al., 1975a). Rhynchosia mosaic virus has not been found to be seed transmitted (Bird et al., 1975a).

The virus is easily transmitted by the common whitefly (*Bemisia tabaci*) (Bird, 1962; Bird et al., 1975a). Transmission can be achieved in less than 24 hours and the insect retains its infectiousness 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. Greenhouse investigations in Puerto Rico (Bird et al.,

1975a), revealed that the bean cultivars La Vega (R 19) and Santa Ana (selection from Masaya, Nicaragua) were tolerant to the virus and had a good level of resistance in the field.

Other Potentially Pathogenic Whitefly-Transmitted Viruses of Beans

Bird (1957) and co-workers (1975a) report that in Puerto Rico there are three other viruses capable of infecting beans under controlled conditions. They are *Jatropha* mosaic virus, isolated from *Jatropha gossypifolia* L. and transmitted by the common whitefly, *Bemisia tabaci* race (biotype) *jatrophae*; *Merremia* mosaic virus, isolated from *Merremia quinquefolia* Hall and transmitted by *Bemisia tabaci* race *sidae*; and *Jacquemontia* mosaic virus, isolated from *Jacquemontia tamnifolia* Griseb and transmitted by *Bemisia tabaci* race *sidae*.

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Chapter 18

ADDITIONAL VIRUSES

F. J. Morales and G. E. Gálvez*

Introduction

At least 70 different viruses infect *Phaseolus vulgaris* L. under experimental or natural conditions. This observation clearly shows the potential susceptibility of this species to those legume viruses and their strains which can adapt to beans under a mixed-cropping system. This chapter describes some of the viruses that have occasionally infected beans under natural conditions.

Beet Curly Top Virus

Curly top of beans is caused by a geminivirus (BCTV) transmitted by the beet leafhopper, *Circulifer tenellus* (Baker). This virus can cause economic losses to beans and other cultivated crops, mainly to beets (*Beta vulgaris* L.) (Bennett, 1971; Zaumeyer and Thomas, 1957). Curly top, reportedly, has 10 strains which differ in their virulence (Zaumeyer and Thomas, 1957). The Spanish name of beet curly top in Latin America is “ápice rizado de la remolacha.”

Infected young bean plants commonly exhibit leaf puckering, downward curling, cupping, and yellowing (Figure 141). Primary leaves of infected plants may be thicker and more brittle than those of uninfected plants. Younger leaves are usually more curled and cupped than older leaves (Nuland et al., 1983). The leaf curling and yellowing symptoms may resemble feeding damage induced by the green leafhopper (*Empoasca* sp.).

The main control measure is the use of resistant or tolerant cultivars. The resistance of some bean cultivars is temperature-sensitive and can be destroyed at high temperatures, regardless of

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plant age at the time of inoculation (Silbernagel and Jafri, 1974). However, there are some breeding lines which are highly resistant to the virus (Silbernagel, 1979). New infections depend on the movement of leafhoppers which may overwinter on some weed species such as mustards (Nuland et al., 1983).

Tobacco Yellow Dwarf Virus

Bean summer death apparently occurs only in Australia (Ballantyne, 1968; Ballantyne et al., 1969; Bowyer and Atherton, 1971) and is transmitted by the brown leafhopper, *Orosius argentatus* (Evans). Bean summer death was originally suspected to have a mycoplasma-like etiology, but was discovered (Bowyer and Atherton, 1971) to be caused by a geminivirus similar to the beet (bean) curly top virus. The name of the causal geminivirus has now been changed to tobacco yellow dwarf virus (Thomas and Bowyer, 1984).

The host range of bean summer death includes *Phaseolus vulgaris*, *Datura stramonium* L., the beets *Beta vulgaris* var. *vulgaris* and *B. vulgaris* var. *cicla*, and *Callistephus chinensis* (L.) Nees (Bowyer and Atherton, 1971). The Spanish translation for bean summer death is “muerte de verano del frijol.”

The symptoms of this disease are stunting, leaf curling, vascular necrosis, epinasty, interveinal chlorosis, wilting, and death of the plant. Symptom development is more rapid after a period of high temperature (Ballantyne, 1968; Ballantyne et al., 1969). The insect vector has a minimum latent period of 24-48 hours. It remains infectious for at least 21 days after acquiring the virus during the nymphal or adult stage (Thomas and Bowyer, 1984).

Ballantyne et al. (1969) report that various materials resistant to curly top in the United States were resistant to bean summer death in Australia.

Tomato Spotted Wilt Virus

Tomato spotted wilt virus (TSWV) occurs in Brazil and Canada on various plant species. Although it does not cause economic damage to beans, it induces severe malformation and stunting in infected

bean plants. The appearance of chlorotic or necrotic spots on affected leaves is also a diagnostic feature (Costa and Foster, 1941; Costa et al., 1971).

The virus is transmitted by various thrips such as *Thrips tabaci* Lindeman, *Frankliniella schultzei* (Trybom), *F. fusca* (Hinds), and *F. occidentalis* (Pergande) (Costa, 1957; Costa and Foster, 1941; Costa et al., 1971; Paliwal, 1974).

Tomato spotted wilt virus is also known as Kromnek virus, *Lycopersicum* virus 3, pineapple yellow spot virus, and tomato bronze leaf virus. In Latin America, it is known as “marchitamiento manchado del tomate” (Spanish) and “vira-cabeça” (Portuguese).

The virus particles are round, 80-120 nm in diameter, surrounded by a lipid membrane, and contain RNA. Its identification and characterization are reported by Best (1968) and Ie (1970). There are no specific control measures because it is limited in distribution and importance.

Tobacco Streak Virus, Red Node Strain

Red node occurs in the United States (Zaumeyer and Thomas, 1957) and Latin America (Costa et al., 1971; Silberschmidt and Nobrega, 1943). This disease is caused by a strain of tobacco streak virus (Zaumeyer and Thomas, 1957). The common Latin American names of red node are “nudo rojo” (Spanish) and “novermelho” (Portuguese), and of tobacco streak virus “mosaico rayado del tabaco” (Spanish).

Symptoms include a reddish discoloration at the nodes of stems and pulvini of leaves (Figure 142), as well as reddish concentric rings on pods. In severe cases, infected plants will bend over or break at a discolored node. Veins and veinlets of leaves may exhibit a red to reddish brown streaking (Nuland et al., 1983). Pods may shrivel and not produce seed. Plants also may be stunted or killed (Zaumeyer and Thomas, 1957).

The virus is transmitted mechanically, apparently in bean seed (Fulton, 1971; Zaumeyer and Thomas, 1957), and by thrips (Nuland et al., 1983). However, R.O. Hampton has never detected seed

transmission in thousands of field-infected seedlings of susceptible cultivars, but has recovered the virus from nearby weeds and other crop hosts (personal communication). The virus particles are isometric and about 28 nm in diameter (Mink et al., 1966).

Miscellaneous Bean Viruses

In Brazil, Costa et al. (1983) studied the transmission, by the common whitefly, *Bemisia tabaci*, of carlavirus-like particles. The particles are 650 nm in length and 13 nm in diameter and are characteristically transmitted to the bean cv. Jalo by aphids.

This virus infected more than 80 of the bean varieties tested, inducing very mild or no symptoms in most of them. In the bean cv. Jalo the virus induces a mild mottle, vein chlorosis, and a yellow angular mosaic in older leaves. The virus does not appreciably stunt the plant. However, a slight reduction in the number of pods per plant and seeds per pod is apparent in infected bean plants. The virus is not seed-borne. There are no specific measures of control.

Other virus diseases of beans include peanut stunt (Allen, 1983; Quiot et al., 1979), cowpea severe mosaic, tobacco ringspot, and tobacco necrosis (Allen, 1983).

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Chapter 19

SEED PATHOLOGY

H. F. Schwartz and F. J. Morales*

Introduction

Dry or common beans (*Phaseolus vulgaris* L.) are not vegetatively propagated and therefore depend on seed production for the perpetuation of the crop. The quality of common bean seeds used for planting by farmers in developing countries is usually low, especially among smallholders. Farmers in developed regions usually give priority to high-quality seeds and use them for production.

Seeds provide an efficient method for the transfer of plant pathogenic microorganisms between locations and seasons. More than 50% of the major bean diseases can be seed-borne (Ellis et al., 1977; Hampton, 1983). As a farmer plants infested seeds, he also sows the potential for future disease problems. Seed transmission of plant pathogens is of concern in developing countries because most farmers plant seeds saved from previous harvests (Gutiérrez-P. et al., 1975), thereby perpetuating diseases. The effect of seed-borne organisms upon germination of bean seeds is not well documented. However, many internally borne fungi are known to decrease seed germination (Dhingra, 1978; Ellis et al., 1976d) and field emergence (Figures 143-146). The halo-blight bacterium (*Pseudomonas syringae* pv. *phaseolicola* (Burk.) Young et al.) is seed-borne. Severely infected seeds germinate at a low rate, producing deformed seedlings (Katherman et al., 1980; Saettler et al., 1981; Weller and Saettler, 1980). Seed viability, germination, and contamination by microorganisms also can be affected by mechanical damage which may occur during harvesting, threshing, and/or planting (Dickson and Boettger, 1976; Schweitzer, 1972; Weller and Saettler, 1980).

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The extent of transmission from seed to crop or of development of seed-borne disease depends on various factors such as the amount or rate of seed-borne inoculum; extent or rate of transmission of this inoculum to the seedling at any stage of its plant development; subsequent rate of inoculum or disease increase until harvest; and rate of re-establishment of seed-borne inoculum during the next seed generation. Seed pathology programs must also consider those biological factors which influence pathogen development, detection, and management. These are inoculum potential, infection probability, other means of transmission, variation in pathogen virulence and host susceptibility, accuracy and reliability of testing methods, and efficacy of seed disinfection (Neergaard, 1977).

Seed-borne Fungi

Many fungi can be borne internally or as surface contaminants in seeds of *Phaseolus vulgaris* (Table 1). Many of these microorganisms are also seed-borne in other members of the Leguminosae such as soybean (*Glycine max* (L.) Merr.), pigeonpea (*Cajanus cajan* (L.) Millsp.), and cowpea (*Vigna unguiculata* (L.) Walpers ssp. *unguiculata*) (Ellis et al., 1976d). Most internally borne fungi are located inside the seed coat and some infection may occur in the cotyledon or embryo (Bolkan et al., 1976; Dhingra and Asmus, 1983; Ellis et al., 1976a; Menten et al., 1979). The anthracnose fungus (*Colletotrichum lindemuthianum* (Saccardo et Magnus) Briosi et Cavara) can become seed-borne after penetrating pod walls (Figure 147). Angular leaf spot (*Isariopsis griseola* Sacc.) is usually found in the hilum area of the seed coat (Correa-Victoria, 1984).

Date of harvest is important in producing high-quality and pathogen-free seeds (Ellis et al., 1976b; Rena and Vieira, 1971). Weed management also reduces seed infection by some pathogens such as web blight (*Rhizoctonia solani* Kühn) and pod decay (*Fusarium semitectum* Berk. et Rav.) (Chagas and Dhingra, 1979). Seed infection by fungi increases (Gomes and Dhingra, 1981) and seed germination decreases if harvesting is delayed (Figures 148 and 149) (Ellis et al., 1976b). It is, therefore, important that seed be

Table 1. Examples of seed-borne and seed-contaminating microorganisms associated with common beans (*Phaseolus vulgaris* L.).

| Microorganism | Common name | Source ^a |
|--|---------------------|---------------------|
| FUNGI | | |
| <i>Acrostalagmus</i> spp. | | 26 |
| <i>Alternaria</i> spp. | Leaf-and-pod spot | 61 |
| <i>Ascochyta bolishaueri</i> Saccardo | Leaf-and-pod spot | 38 |
| <i>Ascochyta phaseolorum</i> Saccardo | Leaf-and-pod spot | 38 |
| <i>Aspergillus candidus</i> Link ex Fries | Storage rot | 43 |
| <i>Aspergillus glaucus</i> Link ex S.F. Gray | Storage rot | 43 |
| <i>Aspergillus niger</i> van Tieghem | Storage rot | 26 |
| <i>Aspergillus repens</i> de Bary | Storage rot | 43 |
| <i>Aspergillus restrictus</i> Smith | Storage rot | 43 |
| <i>Botryodiplodia theobromae</i> Patonillard | Seed decay | 26 |
| <i>Botrytis cinerea</i> Persoon ex Fries | Gray mold | 26 |
| <i>Cercospora canescens</i> Ellis et Martin | Leaf spot | 26 |
| <i>Cercospora cruenta</i> Saccardo | Leaf blotch | 76 |
| <i>Chaetoseptoria wellmanii</i> Stevenson | Leaf spot | 13 |
| <i>Cladosporium herbarum</i> (Persoon) Link | Cladosporium spot | 68 |
| <i>Colletotrichum dematium</i> (Persoon ex Fries) Grove | | 26 |
| <i>Colletotrichum lindemuthianum</i> (Saccardo et Magnus) Briosi et Cavara | Anthraxnose | 76 |
| <i>Colletotrichum truncatum</i> (Schweinitz) Andrus et Moore | Stem anthracnose | 41 |
| <i>Curvularia</i> spp. | Leaf spot | 18 |
| <i>Dendrophoma</i> spp. | | 3 |
| <i>Diaporthe phaseolorum</i> (Cooke et Ellis) Saccardo | Pod-and-stem blight | 26 |
| <i>Diplodia natalensis</i> Pole-Evans | Seed contaminant | 76 |

(Continued)

Table 1. (Continued).

| Microorganism | Common name | Source ^a |
|---|-------------------|---------------------|
| <i>Erysiphe polygoni</i> DC. | Powdery mildew | 76 |
| <i>Fusarium equiseti</i> (Corda) Saccardo | Damping-off | 26 |
| <i>Fusarium moniliforme</i> Sheldon | | 54 |
| <i>Fusarium oxysporum</i> f.sp. <i>phaseoli</i> Kendrick <i>et</i> Snyder | Fusarium yellows | 76 |
| <i>Fusarium roseum</i> Link | | 18 |
| <i>Fusarium semitectum</i> Berkeley <i>et</i> Ravenel | Pod decay | 74 |
| <i>Fusarium solani</i> f.sp. <i>phaseoli</i> (Burkholder) Snyder <i>et</i> Hansen | Root rot | 52 |
| <i>Fusarium sulphureum</i> Schlechtendahl | | 26 |
| <i>Isariopsis griseola</i> Saccardo | Angular leaf spot | 55 |
| <i>Macrophomina phaseolina</i> (Tassi) Goid. | Ashy stem blight | 76 |
| <i>Monilia</i> spp. | | 26 |
| <i>Mucor</i> spp. | | 18 |
| <i>Nematospora coryli</i> Peglion | Yeast spot | 74 |
| <i>Nigrospora</i> spp. | | 22 |
| <i>Penicillium</i> spp. | Storage rot | 43 |
| <i>Pestalotiopsis</i> spp. | | 26 |
| <i>Peyronellaea</i> spp. | | 26 |
| <i>Phomopsis phaseolina</i> | Leaf-and-pod spot | 26 |
| <i>Rhizoctonia solani</i> Kühn | Root rot | 42 |
| <i>Rhizopus</i> spp. | Soft rot | 3 |

(Continued)

Table 1. (Continued).

| Microorganism | Common name | Source ^a |
|---|-----------------|---------------------|
| <i>Sclerotinia sclerotiorum</i> (Libert) de Bary | White mold | 6 |
| <i>Sclerotium rofsii</i> Saccardo | Southern blight | 3 |
| <i>Sporotrichum</i> spp. | | 61 |
| <i>Stemphylium</i> spp. | Leaf spot | 61 |
| <i>Thanatephorus cucumeris</i> (Frank) Donk | Web blight | 76 |
| BACTERIA | | |
| <i>Achromobacter</i> spp. | | 61 |
| <i>Aerobacter aerogenes</i> (Kruse) Beijerinck | | 61 |
| <i>Agrobacterium radiobacter</i> (Beijerinck et van Delden) Conn | | 61 |
| <i>Alcaligenes viscosus</i> Weldin | | 61 |
| <i>Bacillus cereus</i> Frankland et Frankland | | 61 |
| <i>Bacillus megatherium</i> Schroeter | | 61 |
| <i>Bacillus polymyxa</i> (Prazmowski) Macé | | 61 |
| <i>Bacillus sphaericus</i> Neide | | 61 |
| <i>Bacillus subtilis</i> (Ehrenberg) Cohn | | 61 |
| <i>Corynebacterium flaccumfaciens</i> pv. <i>flaccumfaciens</i> (Hedges) Dowson | Bacterial wilt | 76 |
| <i>Corynebacterium helvolum</i> (Zimmermann) Kisskalt et Berend | | 61 |

(Continued)

Table 1. (Continued).

| Microorganism | Common name | Source ^a |
|--|--------------------------------------|---------------------|
| <i>Micrococcus</i> spp. | | 61 |
| <i>Pseudomonas fluorescens</i> (Trevisan) Migula | | 61 |
| <i>Pseudomonas syringae</i> pv. <i>phaseolicola</i> (Burk.) Young et al. | Halo blight | 76 |
| <i>Pseudomonas syringae</i> pv. <i>syringae</i> van Hall | Bacterial brown spot | 76 |
| <i>Xanthomonas campestris</i> pv. <i>phaseoli</i> (Smith) Dye | Common and fuscous bacterial blights | 76 |
| VIRUSES | | |
| Bean common mosaic virus | BCM | 76 |
| Bean southern mosaic virus | BSM | 76 |
| Bean western mosaic virus | Strain of BCM | 76 |
| Cherry leafroll virus | | 31 |
| Cucumber mosaic virus | CMV | 48 |
| Tobacco streak virus | Red node strain | 76 |

a. Numbers refer to sources' order in list of references.

harvested immediately after plant maturity. In some cultivars, pod contact with the soil may cause significantly higher levels of seed infection by various soil-borne fungi such as web blight (*Rhizoctonia solani*), southern blight (*Sclerotium rolfsii* Saccardo) (Figure 150), and ashy stem blight (*Macrophomina phaseolina* (Tassi) Goid.) (Figure 151). This may result in a significantly lower seed germination than in seeds collected from pods of the same plant but free from soil contact (Ellis et al., 1976c; Zaumeyer and Thomas, 1957). When harvesting seed-production fields care must be taken to prevent pods coming into contact with the soil. Subsistence farmers, in particular, must take care when handpicking desirable pods to supply seeds for future plantings.

Seed treatment is relatively inexpensive and can improve germination and field emergence of seed lots that are moderately infected. Protective fungicides such as captan, Ceresan (now discontinued), and thiram, diffuse into the seed coat where many seed-borne fungi are found, without entering the cotyledons (Ellis et al., 1976a and 1977). The recommended application rate for most seed treatment is 1-2 g/kg of seed. Systemic fungicides such as metalaxyl and benomyl, penetrate both seed coat and cotyledons, providing a degree of control (Bolkan et al., 1976; Dhingra and Muchovej, 1980; Ellis et al., 1976b and 1977; Muchovej and Dhingra, 1980).

The most efficient method of producing seeds free of a specific pathogen is to use a cultivar that is immune or resistant to that pathogen. Variation exists among cultivars for susceptibility to specific pathogens (Asmus and Dhingra, 1985). Cultivars which are tolerant to a specific pathogen may still allow limited development of the pathogen and therefore potential seed transmission. Seed from such cultivars must be assayed carefully to determine whether seed-borne fungi are present.

Seed-borne Bacteria

At least 95 species and varieties of bacteria are seed-borne in crops (Coyne and Schuster, 1974). Various bacterial pathogens are internally seed-borne in *Phaseolus vulgaris* (Table 1). Common bacterial blight (*Xanthomonas campestris* pv. *phaseoli* (Smith) Dye) and bacterial wilt (*Corynebacterium flaccumfaciens* pv.

flaccumfaciens (Hedges) Dows.) can remain viable for 2-10 and 5-24 years, respectively, in seeds (Schuster and Coyne, 1974).

Seeds with visible symptoms of *Xanthomonas campestris* pv. *phaseoli* infection are found in visibly infected pods. However, symptomless seeds can still be internally contaminated and so provide inoculum for disease outbreaks. Infected seed symptoms vary from a slightly darkened spot in the hilum region to discoloration and shrivelling of the seed coat. Weller and Saettler (1980) reported that seed-surface populations can exceed 40,000 bacteria per seed and that a minimum population of 1,000-10,000 per seed was needed to produce an infected plant under field conditions. External infection of seeds occurs during threshing when bacteria from dried bean tissue (especially stems and pods) become air-borne in bean dust (Weller and Saettler, 1980).

There are no satisfactory methods of seed treatment that completely control internally borne bacteria of common beans. Several methods and compounds have been tested with varying results. External seed contamination can be reduced by application of streptomycin (Taylor and Dudley, 1977).

The most reliable method of producing seeds free from bacterial pathogens is to select production areas where environmental conditions and cultural practices do not favor bacterial growth and development (Guthrie et al., 1975). Copeland et al. (1975) state that additional control can be achieved by long rotations of nonhost crops, planting different cultivars in alternating seasons, and sequential planting of adjacent fields to reduce large acreages of susceptible plants uniformly mature at one point during a growing season.

Most certification programs rely upon laboratory tests for cleanliness or as a routine complement of field inspections for bacterial diseases. Traditional seed tests rely upon seed-soak bioassays and usually require large quantities of seeds and testing resources to detect a minimal threshold of infection in any given seed lot (Sheppard, 1983a; Webster et al., 1983; Weller and Saettler, 1980). Many programs are investigating newer procedures and combinations which may be more precise and efficient such as ELISA (enzyme-linked immunosorbent assay) and other serological procedures; immunosorbence; immunofluorescence; electron

microscopy; selective growth media; and dilution plating (Klement, 1983; Kulik, 1984a and 1984b; Kulik and Stanwood, 1984; Lahman and Schaad, 1985; Sheppard, 1983a; van Vuurde and van Henten, 1983; van Vuurde et al., 1983). Halo blight and common bacterial blight detection varies from 100-1,000 to 10,000-100,000 colony forming units/ml, according to the method used. For example, immunofluorescence (Malin et al., 1983 and 1985; van Vuurde and van Henten, 1983; van Vuurde et al., 1983) is more sensitive than other methods such as ELISA (Barzic and Trigalet, 1982). However, low levels of seed-borne pathogenic bacteria cannot yet be reliably detected by any method (Malin et al., 1985). The sensitivity, specificity, reliability, and expense of each method varies considerably. Seed pathology laboratories have not yet standardized testing procedures or threshold levels for certification.

At present, no commercial cultivar is immune to infection by the common bacterial blight (Cafati-K. and Saettler, 1980) or halo blight pathogens. However, resistance to infection occurs and differential pod susceptibility can be used to further reduce seed contamination by the common bacterial blight pathogen and others (Coyne and Schuster, 1974; Webster et al., 1983).

Seed-borne Viruses

Of the 70 or more viruses which infect *Phaseolus vulgaris*, only seven are known to be transmitted in bean seed (Table 1). Bean common mosaic and bean southern mosaic viruses are considered as the most significant economically. The seed transmission properties of bean common mosaic virus have been the subject of various studies since 1919 (Ekpo and Saettler, 1974; Hampton, 1983; Reddick and Stewart, 1919). In general, the virus is transmitted in a high but variable proportion (often more than 50%) of seeds produced by susceptible plants. Seed transmission varies according to the cultivar infected, time of infection (for example, little seed transmission occurs after flowering), and virus strain involved (Hamilton, 1983; Zaumeyer and Thomas, 1957). There are also susceptible bean genotypes which restrict seed transmission of bean common mosaic virus to less than 1% (F.J. Morales and M. Castaño-J., unpublished data).

Bean southern mosaic virus can be internally transmitted through infected bean embryos (Uyemoto and Grogan, 1977). However, the virus is mainly a seed-coat contaminant since seed transmission is low and, furthermore, considerably reduced by dehydration associated with seed maturity (Cheo, 1955). Nevertheless, bean southern mosaic virus can be efficiently transmitted (10%-20%) in seeds of some cultivars and cause economically significant yield losses (Hamilton, 1983; Morales and Castaño-J., 1985).

Other seed-transmitted viruses are currently considered of minor economic significance in the tropics and other regions. Cucumber mosaic virus is perhaps internally seed-borne (1%-30%) in *P. vulgaris* (Bos and Maat, 1974; Davis et al., 1981; Hamilton, 1983), because it is stable and survives seed storage periods of more than two years. Soybean mosaic virus infects *P. vulgaris*, including seeds, under natural conditions (Castaño-J. and Morales, 1983). Seed transmission, however, is low and many bean cultivars are not susceptible to infection. Bean mild mosaic virus is apparently seed-borne as a seed-coat contaminant (Jayasinghe, 1982). However, the virus is highly infectious and not easily inactivated by desiccation. Tobacco streak virus transmission reportedly varies from 1%-26% (Hamilton, 1983), but neither it nor the cherry leafroll virus are significant problems in tropical bean-producing regions.

The main recommendation for virus-free seed production is field multiplication of seeds obtained from virus-free plants grown under greenhouse conditions. Multiplication fields need to be planted in areas free of seed-borne virus and, if possible, of insect vectors. Roguing seed-infected seedlings or plants in the field is recommended only in the absence of insect vectors. Chemical control of insect vectors is not worthwhile in the case of aphid-borne viruses such as the bean common mosaic, soybean mosaic, or cucumber mosaic, because they are acquired and transmitted by aphids in a few seconds. Insecticides can reduce seed transmission of beetle-borne viruses such as bean southern mosaic and bean mild mosaic.

Virus detection must be simple, rapid, specific, sensitive, and inexpensive (Carroll, 1979; Hamilton, 1983; Kulik and Stanwood, 1984). Bean seed-transmitted viruses are most effectively detected by ELISA because other conventional serological techniques are

affected by nonspecific reactions. A polyclonal antiserum containing antibodies to several seed-borne viruses is desirable.

In the absence of antisera, the "growing on" test is recommended. That is, a representative seed sample (at least 100 seeds for advanced lines or cultivars, or 50 seeds for segregating materials) is sown in trays or pots. Fifteen to 30 days after sowing the health of the seedlings is visually assessed. Since some viruses may not induce visible symptoms in all genotypes or under certain environmental conditions, the "indexing" of bean seedlings with indicator plants is necessary.

Seed Certification

Benefits derived from the use of clean seeds have been demonstrated in temperate regions such as the United States (Copeland et al., 1975; Guthrie et al., 1975), Canada (Sheppard, 1983b), and Australia (Lovelady, 1974), and in tropical regions such as Africa and Latin America (Douglas, 1980; Issa et al., 1964; Sánchez-M. and Pinchinat, 1974). Clean-seed production has been difficult in Brazil (Issa et al., 1964; Wetzel et al., 1972), but programs are being developed. Clean-seed production fields must be located in areas where the environment is unfavorable for the survival of, infection by, and spread of pathogenic microorganisms. An ideal production site has an annual rainfall of less than 30 cm, a daily relative humidity of less than 60%, a daily temperature regime between 25-35 °C, and gravity-irrigation facilities. Production sites also must be located in regions where common beans or other legumes are not grown commercially in order to avoid contamination by insect-transmitted viruses that have wide host ranges. Ideally, a seed-production program is coordinated by a national seed policy (Douglas, 1980) that requires a form of inspection and certification that will ensure seed cleanliness and purity.

Plants must be inspected weekly during their growth to detect and eliminate infected plants. Critical evaluation times after germination are 30-45 days to detect bean common mosaic virus, and 30-60 days to detect common bacterial blight, angular leaf spot, anthracnose, and web blight. The ideal tolerance is 0% infection by any bean pathogen which may be transmitted by seed. However, this

tolerance may have to be raised when seed is produced under those tropical conditions which are marginal for successful clean-seed production.

Successful production of clean seeds also needs proper field management during maturation and harvest. Chemical applications may be required to prevent or reduce plant infection by pathogens or the buildup of insect vectors. Foliar applications of chemicals 7-10 days after flowering and again before plant maturity, will reduce pod infection by plant pathogens and/or saprophytes, and improve seed viability. Mature pods which are not in contact with the soil must be harvested immediately.

A windrow inspection is necessary if beans are not harvested and threshed immediately. Pods must be carefully threshed and cleaned to avoid mechanical damage and cracking. They should be stored under proper conditions. Subsequent laboratory (serology or other detection procedures) and greenhouse tests are carried out to verify that the seeds are indeed pathogen-free or within established standards.

It is not possible to determine if a seed lot is free from infected or infested seeds, but it is possible to certify that a seed lot contains less than a specified level of infection. Seed testing must use controlled conditions (especially for temperature and moisture) and detailed procedures which maximize the probability of recovering the pathogen of interest. Tests vary from simple seed grow outs on media or in pots to complicated laboratory schemes which involve washing, soaking, grinding, infiltration, and state-of-the-art physical and chemical techniques (Schaad, 1982).

Proper seed storage conditions are vital for maximizing the survival of high-quality seeds for long periods and for minimizing storage losses inducted by various seed-borne saprophytes and pathogens (Table 1). Proper storage conditions are also critical for minimizing health threats from fungal byproducts such as aflatoxin which has been recovered from beans inoculated with storage rot (*Aspergillus parasiticus* Speare) (Seenappa et al., 1981). López-F. and Christensen (1962) report that the seed moisture content must be less than 15%, preferably 13%, and seed must be stored in conditions of less than 75% relative humidity. López-F. and

Crispín-Medina (1971) report that cultivars vary in their resistance to seed-storage-disease microorganisms. Also, storage temperatures lower than 10 °C will extend the viability of bean seeds.

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Chapter 20

NEMATODES

George S. Abawi and F. Varón de Agudelo*

Introduction

Numerous plant-parasitic nematodes (eelworms) are associated with roots and soils of beans and other plants throughout the world (Table 1). Many of these nematodes have been reported to cause considerable damage to many crops, including beans (Abawi and Jacobsen, 1984; Costa, 1972; Keplinger and Abawi, 1976; Mai et al., 1977; Manzano et al., 1972; McSorley, 1980; McSorley et al., 1981; Melton et al., 1985; Navarro-A. and Barriga-O., 1974; Freire, 1976; Freire and Ferraz, 1977a; Renaud and Thomason, 1973; Rhoades, 1983; Riedel, 1978; Sen and Jensen, 1969; Taylor, 1965; Taylor and Sasser, 1978; Taylor et al., 1970; Zaumeyer and Thomas, 1957). However, only the species of the *Meloidogyne* and *Pratylenchus* genera are frequently and consistently found on beans in relatively high densities in Latin and North America.

Nematode infestations at high initial population densities cause significant yield losses. For example, yield losses may reach 10% to 80% with lesion nematodes (Elliott and Bird, 1985; Robbins et al., 1972), and 50% to 90% with root-knot nematodes (Freire and Ferraz, 1977a; Varón de Agudelo and Gálvez 1974; Varón de Agudelo and Riedel, 1982; Zaumeyer and Thomas, 1957). In addition, plant-parasitic nematodes, particularly the root-knot nematodes, are known to predispose many crop plants to various soil-borne microorganisms that induce root rot and wilt diseases (Elliott et al., 1984b; Powell, 1979; Ribeiro and Ferraz, 1983; Schuster, 1959; Singh et al., 1981b; Walker and Wallace, 1975).

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Table 1. Nematodes frequently found in association with roots of common beans and other plants.

| Scientific name | Common name |
|---|------------------------|
| <i>Aphelenchoides</i> spp. | Bud-and-leaf nematode |
| <i>Belonolaimus gracilis</i> Steiner | Sting nematode |
| <i>Belonolaimus longicaudatus</i> Rau | Sting nematode |
| <i>Criconemella</i> spp. | Ring nematode |
| <i>Ditylenchus destructor</i> Thorne | Potato-rot nematode |
| <i>Ditylenchus dipsaci</i> (Kühn) Filipjev | Stem-and-bulb nematode |
| <i>Helicotylenchus</i> spp. | Spiral nematode |
| <i>Heterodera glycines</i> Ichinohe | Soybean-cyst nematode |
| <i>Heterodera humuli</i> Filipjev | Hop-cyst nematode |
| <i>Heterodera schachtii</i> Schmidt | Sugarbeet nematode |
| <i>Heterodera trifolii</i> Goffart | Clover-cyst nematode |
| <i>Meloidogyne arenaria</i> (Neal) Chitwood | Root-knot nematode |
| <i>Meloidogyne hapla</i> Chitwood | Root-knot nematode |
| <i>Meloidogyne incognita</i> (Kofoid <i>et</i> White) Chitwood | Root-knot nematode |
| <i>Meloidogyne javanica</i> (Treub) Chitwood | Root-knot nematode |
| <i>Pratylenchus brachyurus</i> (Godfrey) Filipjev <i>et</i> Schuurmans Stekhoven | Root-lesion nematode |
| <i>Pratylenchus penetrans</i> (Cobb) Filipjev <i>et</i> Schuurmans Stekhoven | Root-lesion nematode |
| <i>Pratylenchus scribneri</i> Steiner | Root-lesion nematode |
| <i>Rotylenchulus reniformis</i> Linford <i>et</i> Oliveira | Reniform nematode |
| <i>Trichodorus</i> spp. | Stubby-root nematode |
| <i>Tylenchorhynchus</i> spp. | Stunt nematode |
| <i>Xiphinema elongatum</i> Schuurmans Stekhoven <i>et</i> Teunissen | Dagger nematode |
| <i>Xiphinema krugi</i> Lordello | Dagger nematode |
| <i>Xiphinema setariae</i> Luc | Dagger nematode |

This chapter will only summarize available information on root-knot nematodes (*Meloidogyne* spp.) and root-lesion nematodes (*Pratylenchus* spp.) found on beans. For general information on plant-parasitic nematodes, see Mai and Lyon (1975) for taxonomic treatments with an easy-to-use pictorial key for the identification of

plant-parasitic nematodes; Zuckerman et al. (1971) for the principles of plant nematology and the ecology, biology, and management of nematodes as plant pathogens; Varón de Agudelo and Riedel (1982) for the main nematodes found on beans and their control (an auditorial prepared at the Centro Internacional de Agricultura Tropical (CIAT) for training programs); and Sasser and Kirby (1979), Taylor and Sasser (1978), and Taylor et al. (1970) for detailed information dealing with the worldwide distribution, ecology, epidemiology, and management of root-knot nematodes (International Meloidogyne Project publications).

Common names frequently used for *Meloidogyne* species in Latin America include “nematodos de las nudosidades radicales” and “galhas das raizes.” Names commonly used for *Pratylenchus* species include “nematodos de las lesiones radicales,” “lesiones por nematodos,” and “definhamento de nematoide.”

Epidemiology and Life Cycle

Root-knot nematodes

Although there are about 50 reported species of root-knot nematodes, four major species (*M. arenaria*, *M. hapla*, *M. incognita*, and *M. javanica*) have accounted for about 99% of all populations collected from cultivated crop species, including beans. Differential host tests and cytogenetical analysis have identified four races of *M. incognita*, two races of *M. arenaria* populations, and one race each of *M. javanica* and *M. hapla*. Populations of *M. hapla* occur in relatively cold areas since they tolerate temperatures as low as -15 °C. The other three species are adapted to and occur in high-temperature areas. *Meloidogyne incognita* and *M. javanica* are the most prevalent root-knot species in tropical and subtropical regions.

Root-knot nematodes are obligate, endoparasites with a wide host range, including agronomic crops and weeds that belong to many plant families. These nematodes are most abundant and cause serious damage in coarse-textured soil with good drainage (Crispín-Medina et al., 1976; Taylor et al., 1982) such as the coastal soils of Peru. Very few populations of *Meloidogyne* spp. have been found in

soils with more than 40% clay or 50% silt fractions (Taylor et al., 1982). Root-knot nematodes survive in soil as eggs and larvae. Length of survival in soil varies with the nematode species, stage of development, soil texture, soil moisture, and soil aeration (Taylor and Sasser, 1978). Dissemination of nematodes among fields and growing regions can be by irrigation water, vegetative plant parts, and soil infested with eggs or larvae which adhere to farm implements, animals, or man (Crispín-Medina et al., 1976; Vieira, 1967).

The life cycle of *Meloidogyne* spp., as is the case with other plant-parasitic nematodes, involves five developmental stages. Eggs are deposited by mature females in an egg sac consisting of a gelatinous matrix (glycoprotein-type substance) secreted by the female. This sac protects the eggs from dehydration (Figure 152) (Bird and Soeffky, 1972) and may contain as many as 1000 eggs. Eggs are oval to ellipsoidal and slightly concave (Figure 153). They are 30-52 μm by 67-128 μm in size (Thorne, 1961). The vermiform first-stage larvae and, later after the first molt, the second-stage larvae develop in the egg. The second-stage juvenile hatches by breaking the egg shell with repeated thrusting of its well-developed stylet (about 10 μm long). These juveniles (Figure 154) are 375-500 μm long and 15 μm in width.

Second-stage, infective juveniles of *Meloidogyne* spp. move through the soil in search of host roots. Usually, they penetrate roots just behind the root cap and migrate inter- and intracellularly upwards through cortical tissue toward the stele (Ngundo and Taylor, 1975c). The juvenile head is inserted into the vascular system near the region of elongation to obtain plant nutrients. Plant cells in the vicinity of the juvenile increase in number (hyperplasia) and size (hypertrophy) as a result of nematode secretions. Giant cells form near the juvenile head by the fusion and enlargement of plant cells in response to nematode feeding. These giant cells (syncytia) produce root swellings called galls or knots.

Sedentary juveniles continue to enlarge during the formation of giant cells and galls, completing the second and third molts after which the sexes can be differentiated. Males and females are mature after the fourth molt. Adult males are vermiform, measure 0.03-0.36 by 1.20-1.50 mm, lack a bursa, and have a well-developed stylet.

Males are not essential for reproduction. Adult females are pyriform (Figure 155), pearly white, visible on roots without magnification, have a soft cuticle, and measure 0.27-0.75 by 0.40-1.30 mm (Southey, 1965).

Depending upon the host and soil temperature (Tyler, 1933), the entire life cycle (Figure A) may be completed in 17-57 days (Ngundo and Taylor, 1975a). Slight plant injury is apparent 10 days after penetration, but within 40 days epidermal cells often collapse, particularly if females had deposited eggs near the outer root surface (Ngundo and Taylor 1975b). Penetration by and pathogenicity of *Meloidogyne* spp. are affected by plant age, susceptibility, size of nematode populations, and the environment (Gilvonio-Vera and Ravines, 1971; McClure et al., 1974; Ngundo and Taylor, 1975c; Sosa-Moss and Torres, 1973).

Infection of beans by root-knot nematodes results in the reduction and malformation of the root system. There are accompanying physiological changes and a decreased efficiency in the absorption of water and nutrients (Melakeberhan et al., 1985; Wilcox and Loria, 1986). In addition, root-knot nematodes interact with other plant pathogens, resulting in increased plant damage caused by other diseases such as fusarium wilt (Ribeiro and Ferraz, 1983; Singh and Reddy, 1981b), rhizoctonia root rot (Reddy et al., 1979), bean rust (Bookbinder and Bloom, 1980), bacterial wilt (Schuster, 1959), and tobacco ring spot virus (Walker and Wallace, 1975). Infection by nonhost nematodes also reduces rhizobium nodulation (Singh and Reddy, 1981a).

Root-lesion nematodes

Species of *Pratylenchus* are migratory endoparasites and are vermiform during all five developmental stages (Thorne, 1961). Although there are about 40 reported species of *Pratylenchus*, only *P. brachyurus*, *P. penetrans*, and *P. scribneri* are frequently found on beans. These three species are widely distributed and have numerous host crops in many plant families. Eggs, juveniles, and adults survive in infected roots or free in soil. Juveniles and adults can penetrate unsubsided plant roots and move through and between root cells causing cell breakdown and necrosis. Breakdown

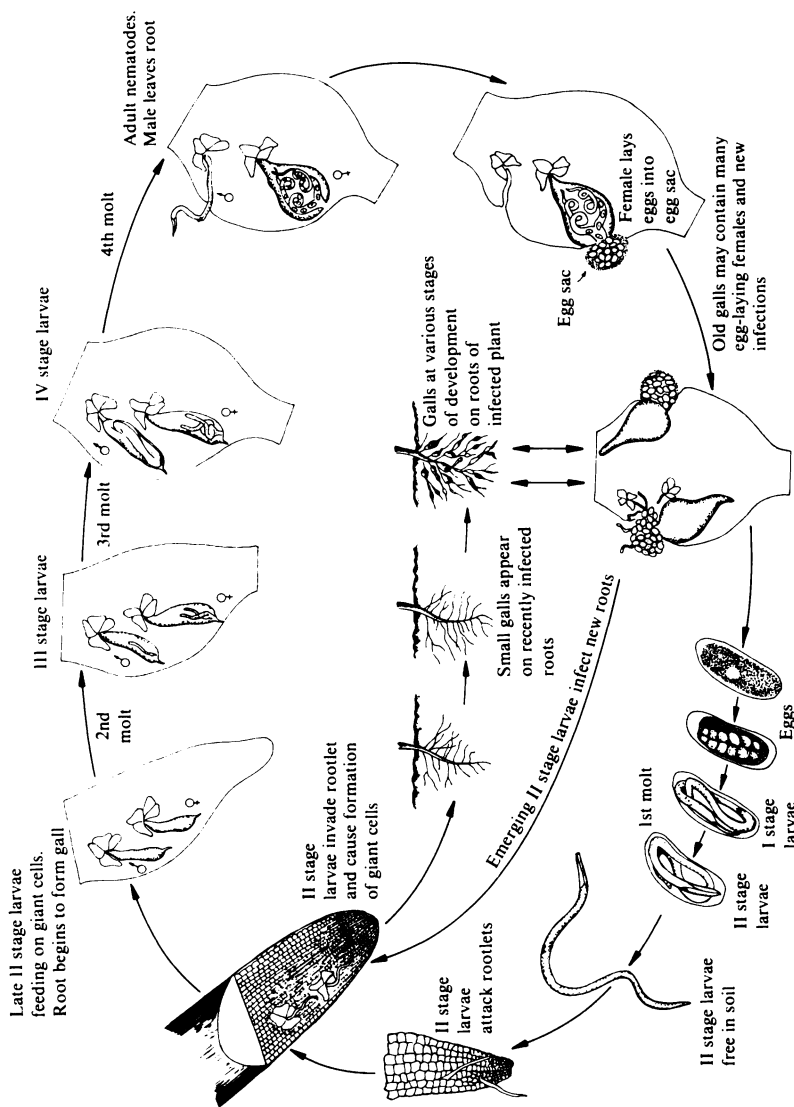


Figure A. Life cycle of *Meloidogyne* sp. (from Agrios, 1978).

of cell walls results, in part, from the mechanical action of nematode spears (stylets), pressure of their body movements in roots, and from enzymes and other substances secreted by the nematodes. Root-lesion nematodes are restricted to the root cortex (Thomason et al., 1976; Thorne, 1961).

Females lay eggs in clusters in root tissues. First-stage larvae and, after the first molt, second-stage juveniles form within the egg. After hatching, second-stage juveniles begin to feed in or migrate outside root tissues in search of other roots to parasitize (Figure 156). Except for the sexual organs, males and females of *Pratylenchus* spp. are similar. They are about 20-25 μm long and 0.4-0.7 μm wide. In some species males are numerous and are required for the reproduction of the species (Mai et al., 1977). Length of the life cycle (Figure B) is variable, depending on nematode species, host crop, and environmental conditions. It ranges from 25-50 days.

Damage to crops, including beans, depends on initial nematode density in soil. A recent greenhouse study (Elliott and Bird, 1985) showed that the growth of susceptible beans was reduced by an initial soil population density of 50 or more *P. penetrans* per 100 cm^3 soil. Yield of susceptible bean cultivars was reduced 43%-47% at densities of 150 *P. penetrans* per 100 cm^3 soil. Species of *Pratylenchus* interact with other soil-borne organisms infecting bean roots. For example, infection by *P. penetrans* increases the incidence and severity of fusarium root rot (Hutton et al., 1973) and of the mycorrhizal fungus *Glomus fasciculatus* (Thaxter *sensu* Gerdemann) Gerdemann et Trappe (Elliott et al., 1984b).

Symptomatology

Plants infected with species of *Meloidogyne* or *Pratylenchus* do not necessarily exhibit characteristic foliar symptoms. Severely infected plants may show chlorosis, stunting, necrosis of leaf margins, and wilting during periods of moisture stress (Figure 157). Distribution of infected plants within a field depends on the history of nematode infestation and the cropping system practiced. In a newly infested field, infected plants showing foliar symptoms may be restricted to one or a few small areas. If a susceptible crop is grown repeatedly in an infested field, the small areas in which growth is poor will

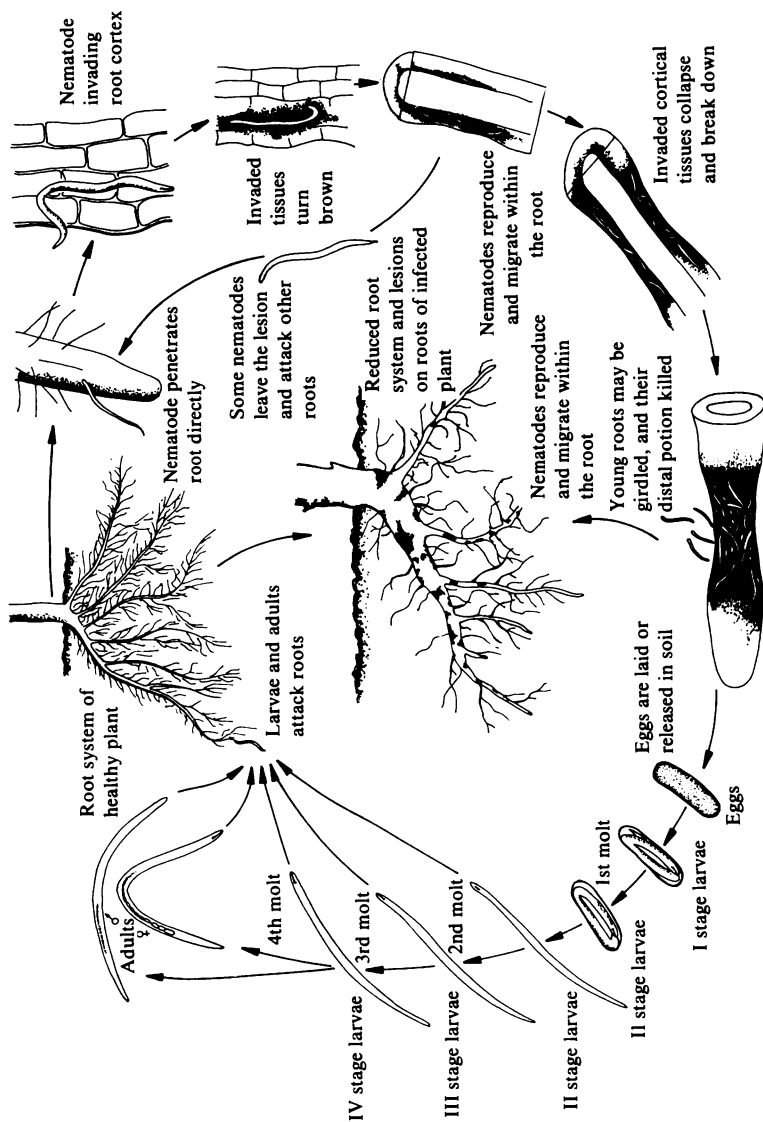


Figure B. Life cycle of *Pratylenchus* sp. (from Agrios, 1978).

gradually enlarge and affected areas with stunted and chlorotic plants will appear throughout the field.

Diagnostic symptoms for nematode infection can, however, be found more clearly on the root system. For proper examination of bean roots, plants must be dug up carefully and the soil removed with as little disturbance to fibrous roots as possible. Roots of bean plants infected by *Meloidogyne* spp. exhibit galls or root knots, usually on the primary and secondary roots (Figure 158). Depending on the species involved, galls may range in size from as small as a pinhead to 12 mm or more in diameter. In addition, the root system becomes malformed with shortened and thickened individual roots which may appear as a mass of galls. Intensive galling interferes seriously with normal root functions, often causing premature defoliation and plant stunting, but rarely death. Stem and hypocotyl tissues may become infected and also exhibit galls, especially when bean seeds are planted too deep (Fassuliotis and Deakin, 1973). Galls induced by root-knot nematodes cannot be detached from the root system without breaking the root. In comparison, nodules formed by nitrogen-fixing *Rhizobium* bacteria are loosely attached to the sides of roots (Renaud and Thomason, 1973).

Bean plants heavily infected by root-lesion nematodes have a reduced root system and, depending on the cultivar, may exhibit brown or black small lesions on the roots (Figure 159). These lesions result from penetration and feeding activities of nematodes in epidermal and cortical tissues (Ngundo and Taylor, 1975b; Thomason et al., 1976). However, diagnostic proof of damage by these nematodes requires extraction of larval and adult stages from roots and adjacent soil. Parasitic nematodes can also be observed directly inside roots by using a compound microscope. However, they can be confused with bacterial feeders unless staining techniques are used by trained observers.

Under natural field conditions infections of bean roots by species of *Meloidogyne* and *Pratylenchus* occur in the presence of many pathogenic and saprophytic soil microorganisms in the rhizosphere. Thus, these nematodes play an important role as a component of the microbial complexes that cause discoloration, necrosis, and eventually decay of plant roots. Decay results from various interactions that can occur among nematodes and soil microorganisms, as well

as from the ability of the nematodes to affect the physiology of plant roots and so predispose them to the detrimental activities of rhizosphere fauna and flora (Elliott et al., 1984b; Powell, 1979; Ribeiro and Ferraz, 1983; Schuster, 1959; Singh et al., 1981b; Walker and Wallace, 1975).

Control by Cultural Practices and Biological Agents

Crop rotation can reduce population levels of root-knot nematodes when beans are planted once every two or three years in rotation with nonhosts such as maize. Growing crops antagonistic to nematodes such as *Tagetes minuta* L. (marigolds), *Crotalaria spectabilis* Roth. (rattlebox) (Hackney and Dickerson, 1975; Navarro-A. and Barriga-O., 1970; Zaumeyer and Thomas, 1957), or *Indigofera hirsuta* L. (hairy indigo) can reduce populations of both root-knot and root-lesion nematodes (Rhoades, 1976). However, many plant-parasitic nematodes such as *Meloidogyne* and *Pratylenchus* species have a wide host range which make crop rotation at times hard to formulate or impractical.

Other cultural practices which reduce nematode populations include long fallow periods, deep plowing, weed control, and, where practical, flooding for one or two weeks (Crispín-Medina et al., 1976; Taylor and Sasser, 1978; Vieira, 1967). Several parasitic and antagonistic microorganisms of eggs and adult stages of plant-parasitic nematodes have been described (Barron, 1977; Kerry, 1980; Mankau, 1980; Sayre, 1980). However, the field effectiveness of these organisms and their economic commercial use are not encouraging.

Control by Chemicals

Chemical control of plant-parasitic nematodes with nematicides is very effective and used widely on annual agronomic crops. However, use of nematicides is expensive for a crop like beans and requires care in handling and often the use of special equipment for application. Fumigant nematicides such as D-D soil fumigant (1,3-dichloropropene and related hydrocarbons), methyl bromide, chloropicrin, and Vorlex, have been used successfully on beans and other

crops (Hartmann, 1968a; Jiménez, 1976; Johnson et al., 1979; McSorley and Parrado, 1983; Powell, 1974; Reddy, 1984; Rhoades, 1976 and 1983; Robbins et al., 1972).

In addition, control of nematodes and increase of bean yield have been obtained with the use of nonfumigant nematicides such as aldicarb, phenamiphos, carbofuran, and oxamyl, applied as a broadcast or band and incorporated into the soil (Abawi and Crosier, 1985; Elliott et al., 1984a; Jiménez, 1976; Rhoades, 1983; Singh and Reddy, 1981b). The application of the nematicide oxamyl to beans as a foliar spray has been effective against many nematodes (Abawi and Mai, 1975; McSorley, 1980; Smittle and Johnson, 1982). However, its activity against the root-knot nematode is limited and a combination of a soil treatment with foliar sprays of oxamyl is recommended (Starr et al., 1978). There have been some encouraging results from the application of nematicides such as oxamyl, as seed treatments to beans (Carvalho et al., 1981; Ngundo and Taylor, 1974; Parisi et al., 1972; Sosa-Moss and Camacho-Guerrero, 1973; Truelove et al., 1977).

Control by Plant Resistance

The use of bean cultivars highly tolerant to plant-parasitic nematodes is the most efficient control strategy, especially for small farmers with limited production inputs. Numerous reports are available that describe the evaluation and identification of bean germplasm with tolerance to plant-parasitic nematodes, especially the *Meloidogyne* spp. (Arias and Ranaud, 1982; Blazey et al., 1964; Cabanillas, 1982; Dickerson and Franz, 1974; Elliott and Bird, 1985; Fassuliotis et al., 1970; Ginoux et al., 1979; Hadisoeganda and Sasser, 1982; Hartmann, 1968a, 1968b, and 1971; López, 1980; Ngundo, 1977; Reddy et al., 1979; Sasser and Kirby, 1979; Singh et al., 1981a; Taha et al., 1977; Varón de Agudelo and Gálvez, 1974; Vieira, 1967; Wilcox and Loria, 1986; Wyatt and Fassuliotis, 1979; Wyatt et al., 1980a, 1980b, and 1983; Zaumeyer and Meiners, 1975; Zaumeyer and Thomas, 1957). The cultivars and breeding lines that are reported as tolerant to root-knot nematodes are Alabama 1, 2, 8, and 19, Spartan, State, P.I. 165426, Rico 23, Manteigão Fosco 11, Porto-Alegre-Vagem-Roxa, Coffee Wonder, Manão Wonder,

Spring Water Half Runner, Wingard Wonder, P.I. 165435, P.I. 313709, Nyakahuti, Red Haricot, Rono, Saginaw, Kibu, Bountiful, Tender Pod, Brittle Wax, My Finca, E.E.U.U. 1-263, Contender, Tender Green, Nema Snap, B 4175, and Strike.

Saginaw, Seafarer, Tuscola, and others are reported as tolerant to the root-lesion nematode (*P. penetrans*). Resistant lima bean cultivars include Hopi, L 5980, Nema Green, Westan, and White Ventura (Allard, 1954; Wester et al., 1958).

Root-knot resistant germplasm is stable (Taylor and Sasser, 1978), but resistance to one race or species of root-knot nematodes is often independent of other races or species. For example, the bean cultivar Contender was highly resistant to races 2, 3, and 4 of *M. incognita*, but only moderately resistant to race 1 (Hadi-soeganda and Sasser, 1982). P.I. 165426 is resistant to *M. incognita* (Fassuliotis et al., 1970), but is susceptible to simultaneous infection by *M. incognita* and *M. javanica* (Ngundo, 1977).

Resistance to gall formation and resistance to the buildup of nematode populations in root systems are characters independent of tolerance to yield reduction. They are probably governed by separate genetic mechanisms (Hadi-soeganda and Sasser, 1982; Wyatt, 1976). Selection of tolerant bean germplasm is often based upon root galling, egg-mass formation, and number of eggs produced per gram of root tissue. However, the galling index does not always correlate with yield (Ngundo, 1977). Galling, female development, and egg-mass production increase as temperature is raised from 16 to 28 °C (Fassuliotis et al., 1970; Freire and Ferraz, 1977b). A hypersensitive necrotic (resistant) response may appear about four days after inoculation (Fassuliotis et al., 1970). A recent report has suggested that cultivar tolerance in beans to root-knot nematodes is related to the effects of nematodes on plant-water relations (Wilcox and Loria, 1986).

Only limited information is available on the inheritance of resistance to plant-parasitic nematodes in beans. Resistance to *M. incognita* is governed by two or three dominant (Hartmann, 1971) and two recessive genes (Ginoux et al., 1979).

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Chapter 21

INSECTS AND OTHER PESTS IN AFRICA

A. K. Karel and A. Autrique*

Introduction

One of the most important bean-production constraints in tropical and subtropical Africa is the wide range of insect pests. Insects attack every part of the bean plant from roots to pods and seeds and cause heavy losses (Karel et al., 1981). Pests infest beans not only in the field, but also in storage. However, for various reasons, few subsistence farmers control insect pests with chemicals; nor do they use insect-resistant cultivars or clean seed.

A substantial proportion of common beans are lost to pest damage every year in Africa. The losses in beans vary from slight to 100%, depending on area, season, cultivar, planting date, and cultural practices. Although accurate and reliable data on bean losses from insect pests are not available in various parts of Africa, estimates are available of losses from some pests (Table 1). Karel (1984a) and A.K. Karel and Ashimogo (unpublished data) recorded as much as 70% seed yield loss in Tanzania. Storage bean losses in eastern Africa are estimated to be between 30% and 73% (Karel, n.d.; Khamala, 1978).

Mixed cropping is practiced by 75%-90% of farmers in Africa (Leakey, 1970). There are many advantages in associated cropping such as reduced pest incidence and damage, erosion control, lower economic risk, and optimization of crop productivity (Desir and Pinchinat, 1976). Although mixed cropping reduces the pest population of some species, it must be combined with other protective

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Table 1. Yield losses in common beans from insect pests in Africa.

| Pest | Country | Yield loss (%) | Source |
|-----------------------|-----------------------|---------------------------|----------------------------------|
| Foliage beetle | Tanzania | 18-31 | Karel and Rweyemamu, 1984 |
| Aphid | Uganda | 90 | Nyiira, 1978 |
| | Tanzania | 37 | Swaine, 1969 |
| | Burundi | 50 | Autrique et al., 1985 |
| Bean fly | Kenya | 30-100 | De Lima, 1983 |
| | Tanzania | 33-100 | Karel and Matee, 1986 |
| | | | Wallace, 1939 |
| | Burundi | 50 | Autrique, 1985 |
| | Central Africa | 50 | Autrique, 1985 |
| | Uganda | 100 | Greathhead, 1968 |
| | Zimbabwe | 50-100 | Taylor, 1958 |
| Thrips | Uganda | 27 | Ingram, 1969b |
| Pod borers | Kenya | 15-25 | De Lima, 1983 |
| | Tanzania | 33-53 | Karel, 1985d |
| Bruchids | Kenya | 73 | Khamala, 1978 |
| | Tanzania | 30 | Karel, n.d. |
| | Uganda | 23 | Rubaihayo et al., 1981 |

measures to optimize yields. Literature from many studies in several African countries suggest that large yield increases can be obtained with effective insect control (Karel and Ndunguru, 1980). Use of cultural control methods and resistant cultivars will further reduce losses caused by insects.

Insect pests are often found in complexes (Figure A) and such complexes are often responsible for severe damage and reduction in bean yields. However, insect complexes vary greatly throughout Africa (Table 2) and in most cases are not well documented. So far, only listings have been made: Hill (1975) listed over 60 insect species that attack beans; more recently, Karel (1984b) identified more than 80 insect species associated with beans in eastern Africa. These attack every part of the bean plant (Figure B) from the root to the pods and seeds, and seeds in storage (Table 3) (Karel et al., 1981).

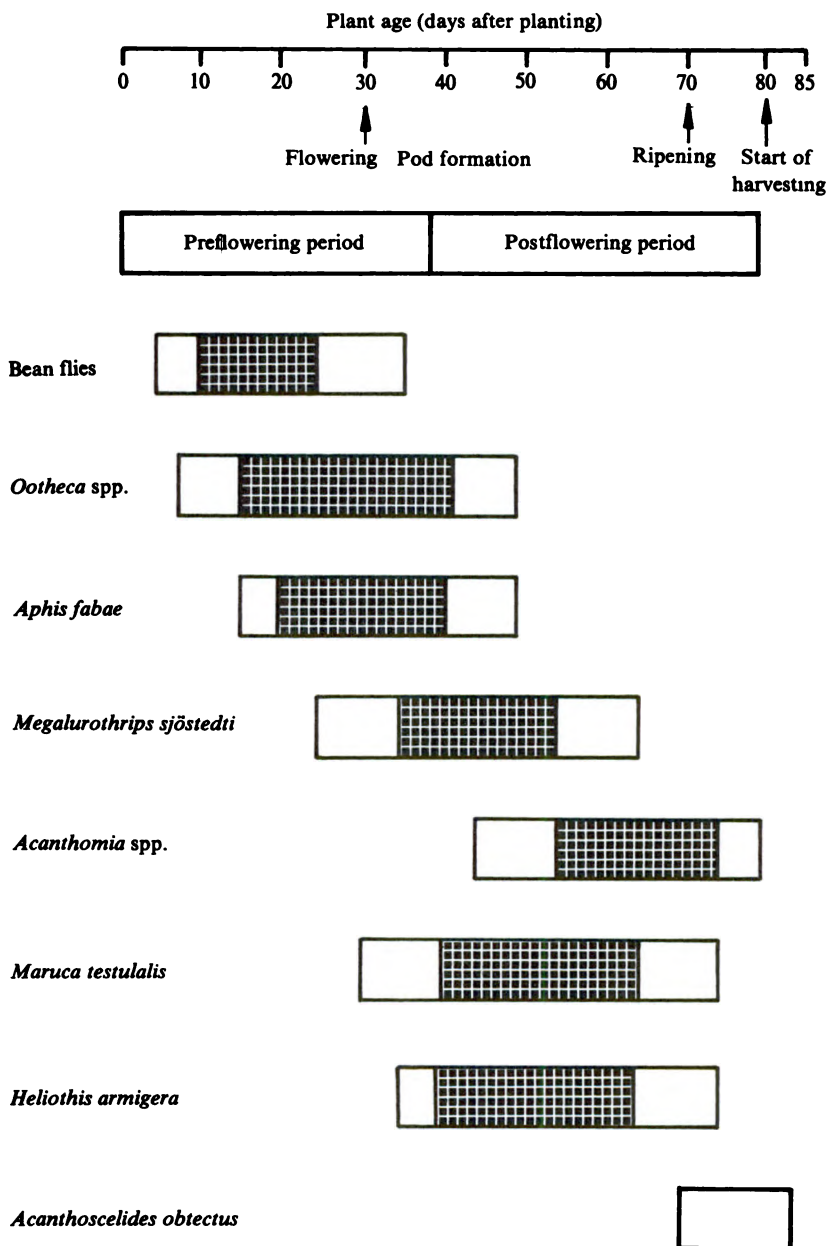


Figure A. The time of occurrence and peak activity of important insect pests of common bean (*Phaseolus vulgaris* L.) in Tanzania (Karel, 1982). = Peak activity; = Occurrence.

Table 2. Economic importance^a of bean insect pests in major bean-producing countries of Africa.

| Insect species | North Africa | | | East Africa | | | | Southern Africa | | |
|---|--------------|----------|-------|-------------|--------|---------|--------|-----------------|--------|----------|
| | Egypt | Ethiopia | Kenya | Tanzania | Uganda | Burundi | Rwanda | Malawi | Zambia | Zimbabwe |
| <i>Ophiomyia phaseoli</i> | I | I | I | I | I | I | I | I | I | I |
| <i>Aphis fabae</i> | M, L | M, L | I, M | M | I | I, M | M | M | L | L |
| <i>Empoasca</i> spp. | M | A | M, L | L | L | L | L | A | A | L |
| <i>Ootheca</i> spp. | A | A | M, L | I, M | L | M, L | A | M | A | A |
| <i>Megalurothrips sjöstedii</i> | A | A | M, L | M | M, L | L | L | A | M | L |
| <i>Maruca testulalis</i> | A | A | M, L | I, M | I | M | L | A | M, L | L |
| <i>Heliothis armigera</i> | M | I | I | I, M | M | L | M, L | M | M, L | M |
| <i>Acanthoscelides obtectus</i> and <i>Zabrotes subfasciatus</i> | A | A | I | I | I | I, M | I | I | M, L | M |

a. Economic importance of pest: I = important; M = moderately important; L = less important; A = absent or not reported.

SOURCES: Adams, 1984; Dieudonné, 1981; Edje et al., 1981; Karel et al., 1981; Nyabenda et al., 1981; Rubaihayo et al., 1981.

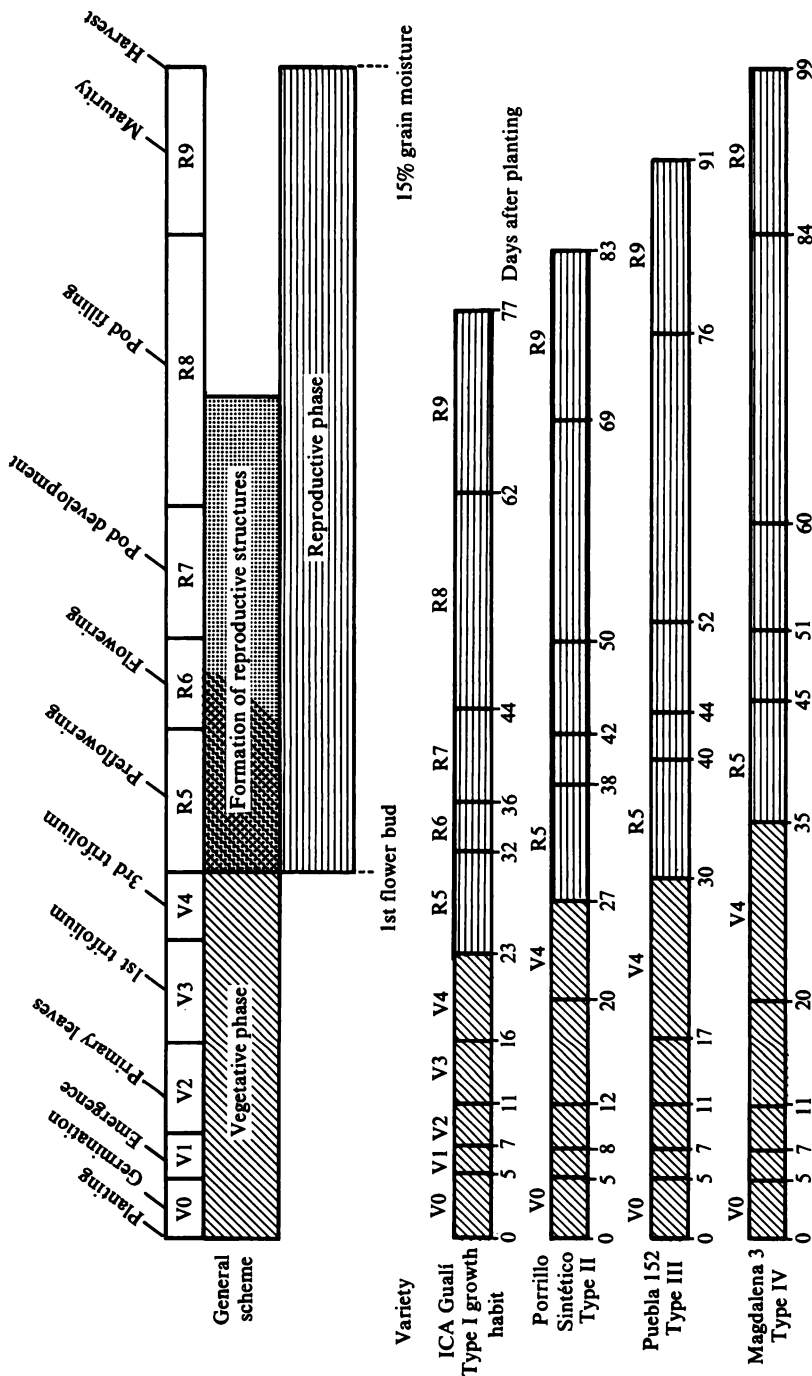


Figure B. Phases and stages of development of the common bean at the Centro Internacional de Agricultura Tropical (CIAT), Palmira, Colombia, at 24 °C and 1000 m.a.s.l. (Adapted from Fernández et al., 1982.)

Table 3. Major insect pests of common beans in Africa.

| Common name | Scientific name | Pest status ^a | Damage |
|---------------------|--|--------------------------|--|
| Bean fly | <i>Ophiomyia phaseoli</i> Tryon <i>O. centrosemais</i> de Meijere <i>Melanagromyza spencerella</i> Greathead | 1 | Feed on stem during preflowering period, especially at seedling stage |
| Leafminer | <i>Liriomyza trifolii</i> Burgess | 2, 3 | Maggots damage leaves by making serpentine tunnels while feeding on leaf palisade tissues |
| Black bean aphid | <i>Aphis fabae</i> Scopoli | 1 | Sucks plant sap from leaves and stem at seedling stage and from pods; virus vector |
| Cowpea aphid | <i>Aphis craccivora</i> Koch | 2 | Sucks plant sap from leaves and stem at seedling stage and from pods; virus vector |
| Leafhopper | <i>Empoasca lybica</i> Le Berg <i>E. dolichi</i> Paoli | 2 2 | Suck sap from leaves during preflowering period |
| Common whitefly | <i>Bemisia tabaci</i> (Gennadius) | 2 | Sucks plant sap from the underside of leaves |
| Foliage beetle | <i>Ootheca mutabilis</i> Sahlberg <i>O. bennigseni</i> Weise | 1, 3 1, 3 | Feed on leaves during preflowering period; virus vector |
| Blister beetle | <i>Coryna kersteni</i> Gerstaecker <i>C. apicicornis</i> Guerin | 3 3 | Feed on pollen (and destroy anthers) and other flower parts |
| Blister beetle | <i>Mylabris amplexans</i> Gerstaecker <i>M. dinctincta</i> Bertoloni <i>M. tristigma</i> Gerstaecker | 3 3 3 | Feed on flower parts, destroying them |
| Striped bean weevil | <i>Acidodes leucogrammus</i> Erichson | 2 | Larvae feed inside stem, causing cankerous swellings; adults make holes in leaves during feeding |

(Continued)

Table 3. (Continued).

| Common name | Scientific name | Pest status ^a | Damage |
|------------------------|---|--------------------------|--|
| Striped foliage beetle | <i>Luperodes quaternus</i> Fairmaire | 2 | Feeds on leaves at seedling stage |
| Flower thrips | <i>Megalurothrips sjöstedti</i> Trybom | 1, 3 | Damages flower buds and flowers by sucking sap |
| Legume pod borer | <i>Maruca testulalis</i> (Geyer) | 1, 2 | Feeds on flower buds, flowers, and green pods |
| American bollworm | <i>Heliothis armigera</i> Hubner | 3 | Feeds on flowers, pods, and sometimes foliage |
| Spiny bug | <i>Clavigralla schadabi</i> Dolling <i>C. tomentosicollis</i> Stål <i>C. hystricodes</i> Germar | 2, 3 | Suck sap from green pods and cause their premature drying and shrivelling |
| Giant coreid bug | <i>Anoplocnemis curvipes</i> Fabricius | 2, 3 | Sucks sap from green pods and cause their premature drying and shrivelling |
| Coreid bug | <i>Riptortus dentipes</i> Fabricius | 2 | Sucks sap from green pods and cause their premature drying and shrivelling |
| Green stink bug | <i>Nezara viridula</i> (Linnaeus) | 2 | Sucks sap from green pods and cause their premature drying and shrivelling; feeding punctures cause necrosis |
| Bean weevil | <i>Acanthoscelides obtectus</i> (Say) | 1 | Damages seeds in storage; infests dry seeds in field |
| Mexican bean weevil | <i>Zabrotes subfasciatus</i> (Boheman) | 1 | Damages seeds in storage |

a. Pest status: 1 = major pest; 2 = minor (secondary) pest; and 3 = sporadic in occurrence.

Bean Fly (Diptera: Agromyzidae)

Bean fly, *Ophiomyia phaseoli* Tryon (earlier described as *Melanagromyza phaseoli*) is a widely distributed pest of seedling beans in eastern, central, and southern Africa, Asia, and Australia. It has not yet been recorded on beans in the Americas. It is the most important pest of common beans in Africa (Dieudonné, 1981; Edje et al., 1981; Greathead, 1968; Hassan, 1947; Jack, 1913; Karel, 1985a; Le Pelley, 1959; Moutia, 1944; Nyabenda et al., 1981; Ohlander, 1980; Wallace, 1939). It was recently reported on beans in Nigeria (Deeming, 1979). Two other species of bean fly, *Ophiomyia centrosematis* de Meijere and *Melanagromyza spencerella* Greathead, have also been recorded in eastern Africa (Greathead, 1968; N. S. Irving, unpublished data; Karel, 1985a). Spencer (1973) considers the *M. sojae* reported from Uganda to be synonymous with *O. phaseoli*. Species of *Ophiomyia* and *Melanagromyza* such as *O. centrosematis*, *M. spencerella*, and *M. dolichostigma* de Meijere, may have been considered as *O. phaseoli* in some literature. For example, the cases of bean-fly oviposition on stems reported by Walker (1960) were probably of *M. spencerella* (described in 1968 by Greathead) and not of *O. phaseoli*.

Bean flies are known by several common names such as stem fly, bean stem maggot, stemborer, pea stemborer, stem miner, bean stem miner, snap bean fly, and soybean leafminer. Karel (1985a) has summarized the literature on bean flies on beans with a detailed bibliography.

Life cycle

Bean flies are minute insects measuring 1.9 to 2.2 mm in length, with a wing span of 4.9 mm. The flies are shiny metallic black (Figure 160). The female is usually a little bigger than the male and can be recognized by her bluntly pointed abdominal tip. *Ophiomyia phaseoli* can be distinguished from all other species, except *M. spencerella*, by its unusually elongated shiny ocellar triangle that reaches to or beyond the lower orbital setulae. It is readily distinguished from *M. spencerella* in males by the form of the aedeagus; and in females, although with more difficulty, by the

shape and serration of the ovipositor blade. *Ophiomyia centrosematis* can be distinguished from the other two species by its ocellar triangle and genitalia (Greathead, 1968).

Oviposition in *O. phaseoli* is peculiar. It consists of a series of actions carried out by the female fly: after alighting on a leaf, the female walks about on the leaf surface for a while. Once she has located a suitable site she raises her abdomen so that the ovipositor is perpendicular to the leaf surface. She then makes a series of downward movements with her abdomen to pierce the leaf surface with the ovipositor. She makes several elliptical cavities (ovipunctures) (Karel, 1985a), after which she moves backward and feeds on the exudate that has oozed from the ovipunctures. For oviposition, the female aligns the ovipositor, at an ovipuncture, with the leaf axis so that an opening leads toward the base of the leaf. This has the effect of directing the larva, when hatched, down the stem.

The female *O. phaseoli* oviposits on the upper surface of the leaves (Karel 1985a), although a few eggs are also laid on the lower leaf surface (Abul-Nasr, 1977; van der Goot, 1930; Greathead, 1968). However, Agarwal and Pandey (1961), Ali (1957), and Manohar and Balsubramanian (1980) observed greater oviposition on the lower leaf surface in beans. Davis (1969) reported that oviposition on the lower surface of the leaves usually occurs during rainy weather. The favorite site for oviposition is near the midrib, at the base of recently unfolded trifoliolate leaves (Davis, 1969; van der Goot, 1930; Greathead, 1968; Ho, 1967; Rogers, 1979). Karel (1985a) reported that the majority of ovipunctures are made in the basal one-third of the leaf. Many more ovipunctures are usually made than are used for oviposition and some are used for adult feeding only (Davis, 1969; Greathead, 1968; Ho, 1967; Swaine, 1969). Karel (1985a) reported that eggs are laid in only 10%-15% of the ovipunctures made.

Although the female of *M. spencerella* scarifies leaf tissue in the same way as does *O. phaseoli*, presumably for feeding purposes, it rarely oviposits on leaves. *Ophiomyia centrosematis*, unlike the other two species, causes no damage to leaves. Eggs of *Ophiomyia centrosematis* and *M. spencerella* are laid in the stem and hypocotyl. The bulk of the oviposition by *M. spencerella* occurs on the hy-

hypocotyl at ground level, two to three days after seedling emergence, whereas *O. centrosematis* does not prefer hypocotyl oviposition as much. However, because *M. spencerella* also deposits eggs in young stem tissue above the cotyledon, the ovipositional sites of the two species are indistinguishable (Greathead, 1968). *Melanagromyza spencerella* also lays its eggs in pockets beneath the epidermis, as does *O. phaseoli*. In *O. centrosematis* and *M. spencerella* stem oviposition is usually oriented with the opening on the lower side and egg situated above the opening (Greathead, 1968). Eggs are not visible from the outside, but can be seen if the leaf or hypocotyl is held up against light or is cleared with alcohol (van der Goot, 1930).

The eggs of the three species of bean fly are smooth, white, oval, and measure about 0.3 mm in length and 0.1 mm in diameter. They are laid singly in the ovipunctures. In her life time, a female lays about 70 eggs (Karel, 1985a). Agarwal and Pandey (1961) found an average of 33 eggs, while Otanes y Quesales (1918) recorded an average of 200 eggs.

The larva hatches from its egg in two to four days. The newly emerged larva, transparent to yellowish white in color, can be easily seen among the green leaves because of its black mouth hooks and body movement. Soon after hatching, the larva tunnels through the leaf tissue, beneath the epidermis, to a nearby main vein or directly to the midrib. The larval tunnels can be seen on the underside of the leaf as silvery mines. The larva then feeds and tunnels through the midrib to the petiole (leaf stalk) where it molts into a second instar. The larva then mines a branch or upper part of the stem and molts again. The third-instar larva bores down the stem of the plant.

The mines of *O. phaseoli* and *O. centrosematis* can be seen below the epidermis with the help of a hand lens. However, the *M. spencerella* larva feeds and tunnels within the stem and therefore its tunnels are not apparent from the outside. The larva continues to feed down the stem into the root. It returns to pupate in the stem just above the soil surface (Greathead, 1968) or sometimes it pupates in the root (Ho, 1967). The larva changes its direction if it meets necrotic or previously mined tissue and progresses farther up the stem before pupating. The *O. centrosematis* and *M. spencerella* larvae mine downward, feeding extensively in the hypocotyl and

taproot before returning to ground level or above to the nearest healthy tissue to pupate.

Fully grown larvae are 2.5 mm long with black rasping hooks (mouth parts), and yellow-white prothoracic and posterior (anal) spiracles (Ho, 1967; Karel, 1985a). The average number of pores in posterior spiracles of the larva of *O. phaseoli* is 8 ± 1 , while *M. spencerella* has an average of 10 ± 1 . The number of pores in the posterior spiracle of *O. centrosematis* larva average only three (Greathead, 1968). The total larval period lasts eight to ten days in warm climate (Karel, 1985a).

The fully grown larva pupates below the stem epidermis (Figure 161), although in older plants pupation may also occur at the base of a petiole. The puparium is found beneath the epidermis, with the head pointed upward and the ventral surface toward the axis of the stem. Before pupation, the area at the front end of the puparium is thinned to a semitransparent window which aids the emergence of the adult. The *M. spencerella* larva pupates in the same position as does *O. phaseoli* after preparing a window. The *O. centrosematis* larva pupates in the same way as *O. phaseoli*, but a window is not prepared. Instead, the anterior spiracles pierce the dry epidermis and project from it (Greathead, 1968).

The pupae of bean flies are barrel-shaped, about 5.5 mm by 2.2 mm in size. The pupae of *O. phaseoli* are usually translucent yellow-brown, while those of *O. centrosematis* are translucent red and yellow-brown. The pupae of *M. spencerella*, however, are opaque and shiny black (Greathead, 1968; Karel 1985a). The number of openings (pores) on posterior spiracles average 8 ± 1 and 10 ± 1 for *O. phaseoli* and *M. spencerella*, respectively. However, the posterior spiracles of *O. centrosematis* have only three openings (Figure C) (Greathead, 1968). The pupal period lasts seven to nine days in warm climates (Karel, 1985a; Swaine, 1969).

After emergence, adults are light brown before they turn shiny black. Adults usually emerge from the puparium in the morning. The total life cycle from egg to adult emergence varies with environmental conditions: in warm weather, it averages 20 days (17-23 days); in cool weather, it averages 42 days (Karel, 1985a). Greathead (1968) reported, for *O. phaseoli*, a life cycle from egg to



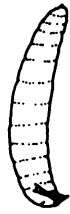
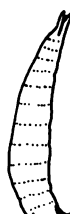



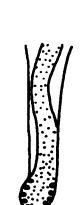










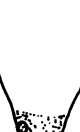

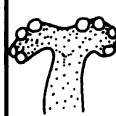
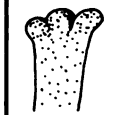


| | <i>Ophiomyia phaseoli</i> | <i>Ophiomyia centrosematis</i> | <i>Melanagromyza sojae</i> | <i>Melanagromyza dolichostigma</i> |
|----------------------|---|--|---|---|
| Larva Last instar |  |  |  |  |
| Anterior spiracles |  |  |  |  |
| Posterior spiracles |  |  |  |  |
| Pupa |  |  |  |  |
| Anterior spiracles |  |  |  |  |
| Posterior spiracles |  |  |  |  |

Figure C. Morphological characteristics of four bean fly species, including *Ophiomyia centrosematis*.
(Taken from Talekar and Chen, 1986.)

adult emergence of 27-31 days on potted plants at 21 °C. The life cycles of *M. spencerella* and *O. centrosematis* from egg to adult emergence on potted plants at 21 °C were 28-35 and 35 days, respectively (Greathead, 1968). The development period is longer at higher altitudes, where temperatures are lower, than at lower altitudes (Davis, 1969). Agarwal and Pandey (1961) reported that eight to nine generations occur per year in India, while van der Goot (1930) reported 14 generations per year in Java, Indonesia.

Adult flies copulate two to six days after emergence. However, Greathead (1968) and Babu (1978) reported a pre-mating period of three days. Mating lasts only a few minutes and takes place only once in the fly's life. Lall (1959) observed a mating period of two to three minutes. The copulating males live for eleven days, while the ovipositing females live for 8-12 days. The female starts laying eggs two to four days after copulation.

Damage

Damage caused by bean flies is most devastating during the seedling stage of the bean plant. *Ophiomyia phaseoli* attacks the bean plant as soon as the first pair of leaves begin to unfold. It continues to attack as other new leaves unfold. *Melanagromyza spencerella* scarifies leaf tissue in the same way. *Ophiomyia centrosematis* does not damage bean leaves to any economic significance.

The main damage is caused by larval feeding and tunnelling in stem tissue. With *O. phaseoli* and *O. centrosematis*, most damage is done by larvae to the first pair of leaves. Later in the life of the plant, the larvae do little damage. Both species of bean fly are external stemborers and feed beneath the stem epidermis where pupation also takes place. A considerable portion of the stem tissue is eaten by larvae and the stem epidermis is later ruptured by puparia. Consequently, with heavy infestation, young plants are considerably weakened and growth is stunted. According to Greathead (1968), attack by *O. centrosematis* is less concentrated on young plants and is the rarest of the three species. It is of negligible economic importance in Uganda.

The larvae of *M. spencerella* bore internally in the stem pith. They also feed extensively in the hypocotyl and tap roots of the bean plant (Figure 162). Pupation occurs deep within the stem tissue. *Ophiomyia phaseoli* has been reported to cause heavy damage and high bean-plant mortality (Greathead, 1968; Ho, 1967; Karel and Matee, 1986; Otanes y Quesales, 1918; Swaine, 1969; Wallace, 1939). According to Greathead (1968), it is a serious pest of beans. Where both *O. phaseoli* and *M. spencerella* occur together, it is probable that the economic damage caused is by *M. spencerella*—the larvae of this species reach and destroy the root system before those of *O. phaseoli*.

A concentration of puparia results in the swelling, splitting open, and rotting of the base of the affected plant. If seedling bean plants are seriously affected, they suffer premature leaf fall and are either killed or severely stunted. Older plants are similarly affected but are not usually killed by the attack. Plant damage is more pronounced in dry conditions than in wet. The bean fly is more destructive when planting is delayed. Greathead (1968) reported that the overall effect of bean fly on the crop depends on each plant's powers of recovery, specifically, an ability to produce adventitious roots (Figure 163). Plants that do not rapidly recover from root damage by developing adventitious roots wilt (Figure 164) and die. They are also liable to break at ground level during windy periods or storms. Plants that produce adventitious roots soon recover from the initial heavy infestation and are sufficiently robust to survive later damage. However, as much as 100% yield losses (Table 1) have been recorded from attack by bean flies in eastern Africa (Wallace, 1939).

Control

Several methods have been used for the control of bean fly with varying degrees of success. Cultural practices such as adjustment of planting time, crop rotation, and associated cropping, can reduce bean-fly populations and damage (Karel and Matary, 1983; Karel et al., 1981; Mohamed and Karel, 1986). Earthing-up (hilling) is often recommended as a cultural control practice because the bean plant produces adventitious roots above the damaged stem part and so recovers from bean-fly damage. Several insecticides, including

dimethoate, endosulfan, monocrotophos, cypermethrin, and pyrethrum, are effective against bean fly (Karel and Matee, 1986; Karel et al., 1981; Matee and Karel, 1984; Swaine, 1969; Walker, 1960). Mansuetus and Karel (1985) have effectively reduced bean-fly damage by using neem (*Azadirachta indica* A. Juss) extract, an insecticide of plant origin. Many parasites of bean-fly have been reported (Greathead, 1968; Hassan, 1947; Oree and Hallman, 1987; Taylor, 1958).

Development of resistant cultivars offers a promising means for bean-fly control. Varietal resistance to *O. phaseoli* in common beans has been reported from Mauritius (Moutia, 1945), Australia (Rogers, 1974 and 1979), and Taiwan (CIAT, 1981; Lin, 1981). In Ethiopia, Abate (1983a and 1983b) screened about 200 bean accessions under a moderate bean-fly attack. Resistant bean lines have also been found in Malawi (Edje et al., 1981).

A screening program for varietal resistance to *O. phaseoli* has begun at the Sokoine University of Agriculture, Morogoro, Tanzania, with several hundred exotic and local *Phaseolus vulgaris* accessions. The selection scheme is based on eliminating highly susceptible materials. Test cultivars are planted, using the Canadian Selian Wonder cultivar as a susceptible border plant. Plants are rated according to number of ovipunctures, larval and pupal counts, and stem damage. Several cultivars have shown low to moderate resistance to bean fly (Karel, 1985c; Karel and Maerere, 1985; Msangi and Karel, 1985; Mushebezy and Karel, 1986; Rwamugira and Karel, 1984). These are A 62, A 63, A 83; BAT 1210, BAT 1275, BAT 1570, CB 137 (CIAT materials), T 8, TMO 75, TMO 91, TMO 117, Chipulupulu, Kablanketi, Sumbawanga B, and YC-2 (improved lines from Uyole Agriculture Centre, Tanzania). Morphological and anatomical parameters such as trichome density on leaf surface, leaf thickness, leaf area, stem diameter, internode length, and adventitious roots, are being assessed on all promising materials to identify potential resistance mechanisms. Preliminary investigations suggest that resistance in some accessions is manifested as tolerance and antibiosis (Mushebezy and Karel, 1986; Rwamugira and Karel, 1984).

Leafminer (Diptera: Agromyzidae)

The leafminer, *Liriomyza trifolii* Burgess, is a minor pest of beans and other legumes in Africa. It is a sporadic pest in Kenya and Tanzania (De Lima, 1979; Katundu, 1980). It is an important pest of beans in Egypt and Mauritius (Fagoonee and Toory, 1983; Hammad, 1978).

Life cycle

The adult leafminer is a small agromyzid fly, about 2 mm long. The dorsal side of the body is dark, but has a bright yellow scutellum. The abdomen is barred with yellow bands. The head, legs, and ventral part of the body are also yellow. Females have well-developed ovipositors, which distinguish them from males.

The female fly makes several ovipunctures, like the bean fly, on the upper leaf surface. However, it makes them near the margins, especially in the apical half of the leaf, whereas the bean fly makes them near the basal region of the leaf. Eggs are laid in only some ovipunctures, while others are used for feeding (A.K. Karel, unpublished data). Feeding punctures and ovipunctures with eggs are clearly visible as white spots, unlike the bean fly which makes elliptical cavities on the upper surface of the leaf.

After hatching, the maggot tunnels through the palisade tissue. There are three larval instars. Fully grown larvae measure 2-3 mm long and are yellow. Mature larvae fall to the ground and pupate in plant debris. Adult flies emerge from the yellowish brown pupae. The entire life cycle, from egg to adult emergence, lasts about 21 days on beans (Katundu, 1980). Several generations can therefore develop in one season.

Damage

Damage is caused by the maggot which destroys the palisade tissue of leaves by making serpentine tunnels (Figures 165 and 166). These leafminer tunnels make leaves unacceptable for consumption as a green vegetable. Larval feeding and tunnelling also reduce the photosynthetic area, thereby resulting in yield losses if damage is

severe. However, precise figures for losses on beans from leafminers are not available.

Control

Leafminers can be controlled with one or two applications of diazinon, monocrotophos, or dimethoate. The most promising approach, however, is the use of resistant cultivars. Some work on bean-plant resistance to leafminer has recently been started in Mauritius. Distribution and density of leaf trichomes, as well as nutritional status, are important selection criteria. High trichome density acts as a physical deterrent to leafminers, and senescing primary bean leaves are not preferred (Fagoonee and Toory, 1983).

Aphids (Hemiptera: Homoptera: Aphididae)

The bean aphid, *Aphis fabae* Scopoli, is the main aphid pest of common beans in Africa (Figures 167). It is widespread, especially in the higher altitudes of Sudan, Ethiopia, Kenya, Uganda, Rwanda, Burundi, Tanzania, Zaire, Malawi, Zimbabwe, Angola, Cameroun, and Nigeria (Remaudière et al., 1985b). The cowpea aphid, *Aphis craccivora* Koch, the major aphid problem of cowpea in Africa, may also damage beans, especially at lower altitudes (Figure 168).

Life cycle

Aphis fabae is a dull black aphid with black siphunculi and cauda. The third antennal segment bears 9-20 irregularly arranged sensoria, whereas in *A. craccivora* there are three to eight arranged in a row. The femora bears many fine hairs on all surfaces and the cauda has 10-19 hairs (Eastop and van Emden, 1972). The adult is 2 mm long with a powdery white secretion on abdominal segments (Karel, n.d.). Usually, only females are found and they reproduce parthenogenetically. Apterous forms are produced when food is abundant and climatic conditions are optimal. When food is in short supply or there is overcrowding in the colonies, alate (winged)

aphids develop. Winged adults may invade bean fields soon after crop emergence.

Aphis fabae has a wide host range (Remaudière et al., 1985a), but the source of primary flights to beans is unknown. There are four nymphal instars. The entire life cycle from egg to adult emergence requires 11-13 days and adults live for 6-15 days. The biology of *A. craccivora* has been extensively studied (Singh and Allen, 1980) and is similar to that of *A. fabae*.

Damage

Apterous bean aphids are found in colonies around the stem, growing points, and leaves (Figure 167). Infested leaves are destroyed and yellowed by the aphids' feeding (sucking) activities. Plants become desiccated and may eventually die (Karel et al., 1981). Sometimes the infestation continues during postflowering. However, the direct damage by bean aphids is usually minimal. An indirect and usually more harmful effect of aphid attack is the transmission and spread of bean common mosaic virus (BCMV). This disease severely reduces the seed yield of susceptible cultivars (Karel, n.d.). Aphid infestation is often particularly severe during a dry spell or late in the season. However, in humid weather, large aphid infestations can be wiped out by entomophagous fungi (Autrique et al., 1985).

Control

Insecticide control of aphids on common beans is effective (Karel et al., 1981; Swaine, 1969). However, there is always a danger of aggravating aphid problems by eradicating their parasites and predators (Ingram, 1969a) (Figure 169). For example, in Burundi, *Aphidius colemani* Viereck (Aphididae) naturally reduces the populations of *A. fabae* (Starý et al., 1985). Pirimicarb is the safest aphicide for beneficial insects.

Bean cultivars resistant to aphids offer a good possibility for control. Rose et al. (1978) identified sources of resistance at the Asian Vegetable Research and Development Center (AVRDC) in Taiwan. Although there are no studies on bean resistance to aphids

in Africa, the high mortality of bean aphids on resistant cultivars occurs because they are caught by the hooked trichomes on bean leaves. de Fluiter and Ankersmit (1948) reported that increased trichome density on bean leaves increased aphid capture. More aphids were trapped by plants grown under dry conditions than by those grown under ample moisture. Farrell (1976) reported that beans intercropped with peanuts in Malawi reduced the spread of peanut rosette virus because their leaf trichomes trap *A. craccivora*, the virus vectors.

Leafhoppers (Hemiptera: Homoptera: Cicadellidae)

Leafhoppers of the genus *Empoasca* are widely distributed in tropical and subtropical Africa. *Empoasca lybica* Le Berg is a minor pest of beans and other legumes in many parts of Africa. *E. dolichi* Paoli is a minor pest of beans in eastern Africa. Leafhoppers are serious bean pests in Egypt. Eight species of leafhoppers, *E. signata*, *E. lybica*, *Asymmetrasca decedens*, *Orosius albicinctus*, *Neolinmus aegypticus*, *Balclutha hebe*, *B. rosea*, and *B. saltuella* have been identified on beans in Egypt (Hammad, 1978). *Empoasca kraemeri* Ross *et* Moore is one of the most important insect pest of beans in Latin America (Wilde and van Schoonhoven, 1976), but apparently does not occur in Africa.

Life cycle

Adult leafhoppers are elongate, light green to yellowish green, and measure about 2.5 mm long. Females lay eggs in leaf veins on the lower surface of young leaves, on petioles, or sometimes within stems of young seedlings. The number of eggs laid varies with the species. A female *E. lybica* lays 80-140 eggs which hatch in six to nine days, depending on the temperature. Five nymphal instars occur over a period of about seven to ten days. Adult longevity is 30-50 days. The biology of *E. kraemeri* on beans in Latin America (Wilde and van Schoonhoven, 1976) is similar to that of *E. lybica*.

Damage

Leafhoppers infest beans during the seedling stage. Frequently, severe leaf damage occurs without reducing the bean yield. Both adults and nymphs infest the lower surface of leaves and suck plant sap. Symptoms of damage, often described collectively as "hopper-burn," comprise a characteristic yellow discoloration of leaf margins, followed by a downward cupping of leaves. The downward cupping results from losing plant sap and possibly from injection of toxic saliva. Infested plants lose vigor and become increasingly susceptible to diseases and other insects. Infestation is favored by hot dry conditions.

Control

Leafhoppers on beans can be controlled with one or two applications of dimethoate, methomyl, monocrotophos, and permethrin. The most promising approach, however, is the use of resistant cultivars. Bean cultivars with low to moderate resistance to *E. kraemeri* have been identified in Colombia (Wilde and van Schoonhoven, 1976).

Whitefly (Hemiptera: Heteroptera: Aleyrodidae)

Common whitefly, or tobacco whitefly, *Bemisia tabaci* Genn., is a minor pest of common beans in Africa. It occurs in northeastern, eastern, central, and western Africa.

Life cycle

The adult common whitefly is an active insect about 1 mm long and males are slightly smaller than females. The light yellow body is covered with a white mealy secretion. The wings are white and similar in size. The third segment of the antennae is much longer than other segments. Eggs are elliptical and measure 0.2-0.3 mm. They are laid singly on a short pedicel which is inserted into the stomata on the lower surface of the leaf. Eggs are white when laid but later turn brown before hatching in about seven days. A female

lays 25-32 eggs. Nymphs, except for first instars, are immobile. They cluster on the underside of leaves and resemble tiny scale insects. There are three nymphal stages. The pupa (puparium) is oval, whitish to yellowish, and measures about 0.6-0.8 mm. The entire life cycle from egg to adult emergence requires about 21 days.

Damage

Both adults and nymphs of whitefly suck sap from leaves. When infestation is severe, the upper surface of leaves becomes mottled with light yellowish spots. However, direct feeding damage is minor compared with the possible indirect effect of virus transmission: *B. tabaci* is the vector of the bean golden mosaic virus (BGMV). However, this virus has not yet been identified on beans in Africa. *B. tabaci* also transmits cowpea mild mottle virus (CMMV), long known in various hosts in Africa, including peanuts. CMMV has recently been found in beans in Tanzania (G. I. Minks, personal communication). However, *B. tabaci*'s role as vector of CMMV in beans has not yet been confirmed.

Control

Chemical control is most effective with one or two applications of carbofuran, dimethoate, or monocrotophos. Carbofuran and phorate granule application at planting time is also effective.

Beetles (Coleoptera: Chrysomelidae)

A number of coleopterous beetles feed on foliage and flowers of common beans. They are very diverse in habits and distribution. Some of the more economically important species of beetles are described here.

Foliage beetles (*Ootheca* spp.)

Ootheca mutabilis Sahlberg and *O. bennigseni* Weise are the two most important foliage-feeding chrysomelid beetles on seedling and adult bean plants in Kenya, Tanzania, Uganda, Burundi, Zambia,

and Malawi. *Ootheca mutabilis* is also an important foliage feeder of cowpea (Singh and Allen, 1980; Singh and van Emden, 1979). Bean seed yield losses from *O. bennigseni* range from 18% to 31% in Tanzania (Karel and Rweyemamu, 1984). They are also vectors of some cowpea viruses, including cowpea mosaic and cowpea mottle (Singh and van Emden, 1979). *Ootheca* spp. may be potential vectors of viruses in beans in Africa, but research is needed to confirm this.

Life cycle. The adult *O. bennigseni* is about 6 mm long, oval, and shiny light brown or orange in color (Figure 170). However, the color varies considerably and light black or brown adults are not uncommon. Eggs are elliptical, yellow and translucent, and are laid in the soil. Eggs are held together in masses of 40-60 by a sticky substance secreted by the female. The total number of eggs laid by a single female varies from 200 to 400. Eggs hatch in 11 to 14 days. Larvae develop in the soil and there are three larval instars that together last 40-45 days. Pupation requires 14 to 20 days. The development period from larva to adult varies considerably and ranges from 65 to 180 days, depending on climatic conditions. Diapause during the dry season ensures the beetles' survival, thereby synchronizing adult emergence with the onset of rains and crop emergence. The life cycle of *O. mutabilis* is similar to that of *O. bennigseni* (Ochieng, 1977).

Damage. Adults feed on leaves by making holes in the interveinal regions (Figure 171). Heavy infestation reduces leaves to a skeleton, thus seriously impairing photosynthetic activity (Karel et al., 1981). Severe damage can result in seedling death. Sometimes, the beetle continues to feed on plants even after flowering and occasionally feeds on floral parts (Karel and Rweyemamu, 1984).

Control. Infestation of *Ootheca* beetles can be avoided to some extent by late planting. Several insecticides, including cypermethrin and endosulfan, are effective against this pest (Karel and Rweyemamu, 1984). Recent studies on natural insecticides from plants demonstrated that *O. bennigseni* can be effectively controlled with two or three sprays of 2% neem kernel extract (Hongo and Karel, 1986; Mansuetus and Karel, 1985).

The most promising approach to controlling *Ootheca* beetles is developing resistant cultivars. Recently, some bean cultivars resistant to *O. bennigseni* have been developed in Tanzania. Bean cultivars A 62, A 67, A 87, BAT 1252 (CIAT materials), Kabanima, Mexican 142, T 8, UAC 116, and YC-2 (Uyole Agriculture Center materials) are moderately to highly resistant to *O. bennigseni* (Karel, 1985b; Karel and Rweyemanu, 1984).

Striped foliage beetle (Coleoptera: Chrysomelidae)

The striped foliage beetle, *Luperodes quaternus* Fairmaire (syn. *Medythia quaterna* Fairmaire), is widely distributed from eastern to western Africa and also occurs in Sudan (Schmutterer, 1969). It is a minor pest of beans.

Life cycle. The adult is a small beetle, about 4 mm long, with white and light brown longitudinal stripes on the elytra. The biology of this beetle is not fully known. Adults lay eggs in the soil where larval and pupal development takes place.

Damage. The striped foliage beetle feeds on the margins of newly emerged leaves of bean seedlings. The beetle sometimes also damages developing pods (Figure 172). Although cowpea mosaic virus is transmitted by this beetle to cowpeas (Whitney and Gilmer, 1974), it is not known if it transmits bean viruses.

Control. Insecticide control is seldom required as populations of striped beetles on beans are usually low. However, several insecticides, including dimethoate and endosulfan, are effective, should insect populations warrant control measures.

Striped bean weevil (Coleoptera: Curculionidae)

The striped bean weevil, *Acidodes leucogrammus* Erichson, is a sporadic pest of beans in eastern, central, and western Africa.

Life cycle and damage. The adult weevil is 7-9 mm long and reddish brown to dark brown with three white markings on the elytra (Figure 173). The adult female lays its eggs on the stem. After the larvae hatch, they tunnel and feed inside the stem, causing

cankorous swellings (Figure 174). The damage results in stunted growth of bean plants. In severe infestations, the stem may break and the plant often dies. Fully grown larvae are about 10 mm in length, legless, C-shaped, and white. Adult weevils cut round holes out of leaf blades during their feeding activity.

Control. Usually, control of this pest is not required. However, if infestation is heavy, several insecticides, including cypermethrin, dimethoate, and endosulfan, are effective.

Blister beetles (Coleoptera: Meloidae)

A number of blister beetles, or flower beetles, belong to two genera, *Mylabris* and *Coryna*, and are important pests of bean flowers. They are commonly found in most of sub-Saharan Africa from eastern to western Africa and down to South Africa. Some common species of *Mylabris* are *M. amplexans*, *M. aperta*, *M. bifasciata*, *M. bipartita*, *M. dicincta*, *M. dilloni*, *M. escherichi*, *M. farquharsoni*, *M. hypolachna*, *M. ligata*, *M. severeni*, *M. sjöstedti* Borchm, *M. temporalis*, *M. tristigma* (Figure 175), and *M. tristis* (Buyckx, 1962; Forsyth, 1966; Hall, 1985; Le Pelley, 1959; Schmutterer, 1969). *Coryna kersteni* Gerstaecker (Figure 176) and *C. apicicornis* Guérin are two important flower-feeding beetles of beans in eastern Africa (Le Pelley, 1959).

Life cycle. Flower beetles are easily recognized by the characteristic brightly colored elytra with broad black, yellow, or red bands (Figures 175 and 176). They are about 15 to 35 mm long and are strong fliers. Eggs are laid in the soil where larvae and pupae are usually found. Larvae undergo hypermetamorphosis and each larval instar is different. Pupation takes place in the soil.

Damage. Beetles cause serious damage to beans by devouring recently opened flowers. They often appear in larger numbers on beans intercropped with maize, sorghum, and other cereals (A. K. Karel and A. Autrique, unpublished data).

Control. Because adult beetles are strong fliers, controlling them with insecticide is difficult. Repeated sprays of endosulfan can control the pest to some extent. However, the most practical means of control is to handpick the beetles.

Flower Thrips (Thysanoptera: Thripidae)

Flower thrips, *Megalurothrips sjöstedti* Trybom (syn. *Taeniothrips sjöstedti*) is an important bean pest in Nigeria, Rwanda, Burundi, Zaire, Zimbabwe, Botswana, and South Africa (Anneck and Moran, 1982; Ingram, 1969b; Nyiira, 1973; Taylor, 1969). Another species of flower thrips, *T. nigrocarnis* (syn. *T. distalis*), has also been recorded feeding on flower buds and flowers of beans in Egypt and Tanzania (Hammad, 1978; Karel et al., 1981; Schmutterer, 1969). *Frankliniella dampfi* Priesner has occurred on beans in Uganda (Ingram, 1969b).

Life cycle

Flower thrips, *M. sjöstedti*, is a shiny black insect that measures about 1 mm in length (Figure 177). Males have not been observed and it is assumed that breeding is parthenogenetic (Ingram, 1969b). Eggs are probably laid in flower buds and are difficult to detect. Two nymphal instars have been recorded. Pupation occurs in the soil. The entire life cycle from egg to adult emergence probably requires 10 to 14 days (Ingram, 1969b). However, Singh and Allen (1979) reported that the life cycle took 14 to 18 days on cowpeas. The biology of the insect is, however, not completely known.

Damage

Both nymphs and adult thrips damage bean flower buds and flowers. It is a more serious pest in drier areas (Karel, n.d.). In severe infestations, flower buds do not open and no flowers, and hence pods, are produced. Feeding punctures on the base of flower petals and stigma can be observed with a hand lens. Feeding injury is characterized by distortion, malformation, and discoloration of flowers. Heavy infestations sometimes lead to flower abortion (Karel et al., 1981).

Control

Spraying with cypermethrin and monocrotophos effectively controls flower thrips (Karel, 1984a; Karel and Mghogho, 1985;

Karel et al., 1981). However, Ingram (1969b) reported that insecticides reduce thrips populations without improving seed yield.

The use of resistant bean cultivars offers a more promising approach to flower thrips control. Screening for resistance to flower thrips in common beans has recently begun at the Sokoine University of Agriculture, Morogoro, Tanzania. Some cultivars show a low level of resistance (A. K. Karel, unpublished data). When thrips infest cowpea peduncles ethylene is produced (Wien and Roesingh, 1980). This fact has been used to develop a screening technique with a synthetic growth regulator, ethephon [(2-chloro-ethyl) phosphonic acid]. Cowpea cultivars susceptible to abscission caused by thrips also show increased abscission after ethephon treatment. The technique may also be useful in identifying sources of resistance in common beans to abscission from flower thrips.

Legume Pod Borer (Lepidoptera: Pyralidae)

The legume pod borer, *Maruca testulalis* (Geyer), occurs throughout the tropics and subtropics, including all of sub-Saharan Africa. It is an important pest of common beans and other legumes, especially cowpea, in many parts of Africa. *Maruca testulalis* is one of the most important post-flowering pests of beans in Tanzania and other eastern African countries (Karel, 1985d; Karel et al., 1981). Losses in seed yield of common beans in Tanzania from *M. testulalis* has been estimated to be over 30% (Karel, 1985d).

Life cycle

The biology of *M. testulalis* has been studied extensively in Africa, especially in relation to cowpeas (Akinfenwa, 1975; Jackai, 1981; Taylor, 1967 and 1978). Eggs are laid singly on flower buds, flowers, and young leaves of bean plants. Eggs are round to oval, measure 0.65 by 0.45 mm, are light yellow, translucent, and have reticulate sculptures on the thin and delicate chorion (Taylor, 1978). The number of eggs laid is 10-100 per female (Singh and van Emden, 1979). Eggs hatch in two to three days (Taylor, 1967).

Caterpillars are whitish with dark spots on each side of the body segment, forming dorsal longitudinal rows. There are five larval

instars, which together last eight to 14 days (Jackai, 1981; Karel, n.d.). The mature caterpillar is about 16 mm long. A prepupal stage of one to two days exists before pupation occurs in a double-walled pupal cell under leaf debris. The pupa is initially green or pale yellow but later darkens to grayish brown. The pupal period lasts five to 15 days. The complete life cycle from egg to adult emergence varies from 18 to 35 days (Taylor, 1978). Adult moths are active during the rainy season and survive for five to seven days. Adult moths have brown forewings with three white spots and grayish white hind wings (Figure 178).

Damage

The most serious damage from caterpillars is their feeding on flower buds and flowers. They also cause extensive damage to green pods (Figure 179). The early instars also infest peduncles or tender parts of stems. The characteristic larval feeding symptom is the webbing together of flowers, pods, and leaves. Frass is often present on pods (Figure 180) (Singh and van Emden, 1979).

Control

Several insecticides, including cypermethrin, carbaryl, endosulfan, fenitrothion, and monocrotophos, are effective against *Maruca* larvae (Karel, n.d.; Karel, 1985d; Karel et al., 1981; Singh and Allen, 1980). Although host-plant resistance to *M. testulalis* offers great potential in the control of legume pod borer, screening for pod-borer resistance in beans has not been done.

American Bollworm (Lepidoptera: Noctuidae)

The American bollworm, *Heliothis armigera* Hubner, is distributed widely in the tropics and subtropics, including most of the African continent. The common name is a misnomer as *H. armigera* does not occur in the Americas, although the closely related *H. zea* (Boddie) and *H. virescens* (F.) do occur. *H. armigera* is a major pest of common beans and other legumes in Africa, especially in eastern Africa (Karel, n.d. and 1985d; Karel et al., 1981; Nyiira, 1973;

Roberts and Chipeta, 1973; Swaine, 1969). It is a polyphagous pest, attacking several other cultivated crops besides grain legumes (Karel, n.d.).

Life cycle

The adult is a stout-bodied, brown, nocturnal moth with a wingspan of about 40 mm. Eggs are spherical, 0.5 mm in diameter, and yellow but turn brownish before hatching. They are laid singly, usually on growing points and leaves. Each female moth may lay as many as 1000 eggs. The incubation period varies from three to five days on beans. There are six larval instars and the larval period lasts from 14 to 24 days (Hill, 1975). Larvae have a characteristic pale white longitudinal band against an almost black band on each side of the body (Figure 181). Larvae often appear green or brown on beans, although their color varies considerably on other crops (Karel, n.d.). Fully grown larvae are about 40 mm long. Pupation occurs in the soil at a depth of about 40 mm. Pupae are shiny black and measure 16 mm long. The pupal period may vary from 10 to 14 days on beans. The life cycle can be completed in 28 to 42 days. Two generations of larvae are recorded in Tanzania—the first generation on early season beans and the second generation on beans sown later in the season (Swaine, 1969).

Damage

Larvae cause serious damage to the bean crop as they feed on pods. The early instar larvae feed on flowers and young pods by making clean circular holes. The main damage is caused by older larvae burrowing into green pods and eating developing seeds (Figure 182) (Karel, n.d.). Infested pods shrivel as a result of seed damage. Infestation is generally heavier during the long rainy season than during the short rainy season in eastern Africa. Losses in seed yield as heavy as 20% have been recorded on beans (Karel, 1985d).

Control

Several insecticides, including carbaryl, endosulfan, monocrotophos, and cypermethrin, effectively control young larvae (early

instars) of *Heliothis* (Karel, 1984a, 1985d, and n.d.; Karel et al., 1981; Swaine, 1969). Several larval parasites of *Heliothis armigera* have been recorded (Karel, 1981; Reed, 1965). No host-plant resistance studies have yet been undertaken.

Pod-sucking Bugs (Hemiptera)

Various species of pod-sucking bugs infest beans during pod production and cause considerable damage and yield losses. Among the major pests are spiny bugs (*Clavigralla* spp.), giant coreid bug (*Anoplocnemis curvipes* F.), coreid bug (*Riptortus dentipes* F.) (Coreidae), and green stink bug (*Nezara viridula* (L.) (Pentatomidae). These insects suck sap from developing pods, thereby shrivelling pods and seeds. Affected pods turn yellow, dry prematurely, seeds do not develop, and, in severe infestation, pods fall off the plants. The bugs not only cause loss of seed yield but also reduce the germination rate of surviving seeds.

Spiny bugs (Hemiptera: Coreidae)

Spiny bugs, *Clavigralla schadabi* Dolling (syn. *Acanthomia horrida* Germar) and *C. tomentosicollis* Stål (syn. *Acanthomia tomentosicollis* Stål), constitute two common species of coreid bugs that infest beans and other legumes in eastern and western Africa. A third species of *Clavigralla*, *C. hystricodes* (syn. *A. hystricodes*) occurs in Tanzania (Bohlen, 1978).

Life cycle. The biology of the three species of *Clavigralla* is similar. Materu (1968) described the biology and population dynamics of *C. schadabi* and *C. tomentosicollis* in Tanzania. Adult bugs measure 7-10 mm in length. The body of these bugs is covered with conspicuous short hair and the prothorax has two spines. The prothoracic spines project anteriorly in *C. schadabi* and *C. hystricodes*. In *C. tomentosicollis*, the spines are smaller and project from the lateral sides of the prothorax (Figure 183). *Clavigralla schadabi* is grayish and smaller than *C. tomentosicollis* which is hairy and brownish. *Clavigralla hystricodes* is black and has a shorter body than the other two species (Karel, n.d.).

Females lay eggs in batches of 10-70. A female may lay as many as 200 eggs which hatch in about six days. There are five nymphal instars over a total period of 28-35 days (Materu, 1968). Nymphs and adults are sluggish and are not easily disturbed. The bugs often feed together on a single pod.

Damage and control. Bugs suck sap from developing seeds and cause dimpling in the seed coat and browning and shrivelling of seeds and pods. Insecticides such as dimethoate, endosulfan, and monocrotophos, provide good control (Karel, n.d.; Nyiira, 1978; Swaine, 1969). However, Matteson (1982) reported that in northern Nigeria spraying cowpeas grown in association with cereals increased pod-sucking bug populations, especially those of *C. tomentosicollis*, and reduced yields considerably. The increase in pod-sucking bug populations was attributed to the insecticide having killed the pest's natural enemies.

Giant coreid bug (Hemiptera: Coreidae)

The giant coreid bug, *Anoplocnemeis curvipes* F., is a minor pest of beans, and a major pest of cowpeas and pigeonpeas, in tropical Africa.

Life cycle. The adult *Anoplocnemeis* is dull black, about three cm long, and is a strong flier. Male and female bugs can be easily distinguished by the shape of their hind legs. In males, these are abnormally broad and each bear a large spine. The gray eggs are laid in chains on leguminous plants other than beans—eggs are rarely laid on bean plants. A single female lays 6-12 chains of 10-40 eggs each. The eggs hatch in about 7-11 days. There are five nymphal instars. The early instar nymphs resemble ants. The total nymphal period requires 30-60 days, depending upon climatic conditions. The adult life span varies from 24 to 84 days.

Damage and control. Damage to beans is caused mainly by adult bugs feeding on young pods. The bugs also feed on tender shoot tips, causing dieback-like symptoms. Insecticides used for the control of *Clavigralla* are also effective in controlling this bug. Ochieng (1977) has identified several egg parasites of *Anoplocnemeis* in Nigeria.

Coreid bug (Hemiptera: Coreidae)

Several species of *Riptortus*, known as coreid bugs, have been recorded feeding on common beans in Africa. *Riptortus dentipes* F. is the most common of these species. Other species are *R. tenuicornis* Dall. and *R. longipes* Dall. (Forsyth, 1966; Le Pelley, 1959).

Life cycle. Adult bugs are slender, about 17 mm in length, and light brown with white or yellow lines on the sides of the body. They are strong fliers. One female lays about 50 eggs in small batches. Eggs are rarely laid on the bean plant and are more commonly laid on other leguminous plants and weeds. The eggs hatch in about six days. Five nymphal instars develop over an 18-day period.

Damage and control. The bugs cause considerable damage to bean plants by sucking sap from green pods. Because the bugs are strong fliers and constantly visit bean plants from alternative host plants, the control of *Riptortus* spp. is difficult. However, insecticides used for the control of *Clavigralla* are also effective against *Riptortus* spp., if repeated applications are made. Some egg parasites also keep the bug population in check. The development of resistant bean cultivars offers good potential for future control.

Green stink bug (Hemiptera: Pentatomidae)

The green stink bug, *Nezara viridula* (L.), is a minor pest of beans. It has a wide range of hosts in tropical and subtropical Africa (Karel et al., 1981; Nyiira, 1978; Swaine, 1969).

Life cycle. The biology of the bug varies considerably according to climatic conditions. Because these insects breed very little on beans, the damage is caused by adults which fly from alternative host plants into the bean field during flowering. A female bug lays 150-400 eggs in four to six batches of 30-80. Eggs are laid on the underside of young leaves. There are five nymphal instars. The early instar nymphs are brightly colored and spotty (Figure 184). They are usually found in clusters. The entire life cycle from egg to adult emergence requires 40 to 80 days. Adults are always green and are strong fliers. The adult life span is 30-60 days. The bugs breed throughout the year if food sources are available (Figure 185).

Damage. Damage to bean pods is caused primarily by adults sucking sap from young pods. Feeding punctures cause necrosis, resulting in pod spotting and deformation. Typical damage symptoms are yellowing, premature drying of pods, and lack of seed formation. Affected pods may wither and sometimes fall off. The bugs also inject a fungus, *Nematospora coryli* Peglion, into developing seeds, and cause additional damage (Wallace, 1939; Chapter 10, this volume, p. 247-248).

Control. Several chemicals, including dimethoate, diazinon, endosulfan, fenitrothion, and monocrotophos, are effective against *N. viridula* (Karel et al., 1981; Swaine, 1969). Certain cultural practices such as adjustment of planting time, also reduce damage from the bugs. Several egg parasites also keep the pest population in check.

Storage Insects

Several species of bruchids (Coleoptera: Bruchidae) infest and damage stored beans in Africa. However, two species, *Acanthoscelides obtectus* (Say) (bean weevil) and *Zabrotes subfasciatus* (Boheman) (Mexican bean weevil), are the most important stored-bean pests in Africa and Latin America. In addition, *Callosobruchus chinensis* (L.) and *C. maculatus* (Fabricius) also cause some damage to beans in Africa.

Bean weevil (Coleoptera: Bruchidae)

The bean weevil, *Acanthoscelides obtectus* (Say), is a widely distributed pest of stored beans. It occurs in Africa, Latin America (Chapter 22, this volume), southern USA, and southern Europe. It is the most important pest of stored beans in the cool highlands of Africa, ranging from Ethiopia in the north to South Africa.

No precise information on losses in stored beans by bruchids is available. However, farm storage for six months is accompanied by about 40% loss in weight with as much as 80% of the seed being infested and unfit for human consumption. Losses vary between 7% in Colombia to 73% in Kenya (Khamala, 1978; van Schoonhoven, 1976).

The biology and life cycle of bruchids have been extensively studied in Latin America (Chapter 22, this volume).

Bruchids can be controlled, with little trouble and expense, by cleaning storage containers and surrounding area. Growing beans at least one kilometer from farm stores (the primary sources of bruchid infestation) effectively controls bruchids in the fields.

Other control methods used in Africa are similar to those used in Latin America. However, in Burundi, good results are obtained with laterite dust (Standaert et al., 1985). Neem-seed oil effectively controls the Mexican bean weevil and could be equally effective on *A. obtectus* (Kiula and Karel, 1985). In eastern Africa, bruchids are commonly controlled by dusting with pyrethrins (McFarlane, 1970). As yet, little work has been done on varietal resistance in beans to this pest in Africa, although some work has started recently in Rwanda.

Mexican bean weevil (Coleoptera: Bruchidae)

The Mexican bean weevil, *Zabrotes subfasciatus* (Boheman) (syn. *Z. pectoralis*, *Z. dorsopictus*, and *Spermatophagus subfasciatus*) is the most important pest of stored beans in warmer regions. It usually occurs at altitudes below 1000 m above sea level in tropical Africa and Madagascar (Davies, 1972; Southgate, 1978). However, no documented information on losses caused by Mexican bean weevil is available from Africa.

As with *Acanthoscelides obtectus*, the biology and life cycle of the Mexican bean weevil have been extensively studied in Latin America (Chapter 22, this volume).

The control measures described for *A. obtectus* are equally effective against *Z. subfasciatus*.

Other Pests

Red spider mite (Acarina: Tetranychidae)

The red spider mite or two-spotted spider mite, *Tetranychus cinnabarinus* Boisdu. (syn. *T. telarius* L.), is widely distributed in

tropical and subtropical parts of the world. In Africa, it is found on beans, cotton, and other plants (Hill, 1975; Khamala, 1978; Nyiira, 1978). A closely related species, *T. urticae*, occasionally infests bean leaves in Uganda (Nyiira, 1978).

Life cycle. Adult females are oval, red or green, and measure 0.4-0.5 mm long. Males are slightly smaller. Immature forms and adults have two spots on their dorsa. The female mite lays spherical, white eggs, about 0.1 mm in diameter. They are laid singly on the underside of leaves. A single female lays as many as 200 eggs. Eggs hatch in four to seven days. Nymphs are six-legged, pinkish, and slightly larger than the eggs. There are two nymphal stages, the protonymph and deutonymph (Hill, 1975), each lasting three to five days. They are green or red and have four pairs of legs. The total nymphal period lasts six to ten days. Adult females live for three weeks.

Damage. Both nymphs and adults feed on the lower side of leaves between the main veins. Yellow spots appear where a group of mites have been feeding together. Clusters of yellow spots are visible on the upperside of leaves, especially between main veins near the leaf stalk. Mite feeding causes a silvering of bean leaves. Later, the affected area spreads, the leaf reddens, withers, and falls off. Since mites usually attack beans near plant maturity, they rarely influence seed yield. The mites cause more damage when there is moisture stress (Nyiira, 1978).

Control. Usually, the mite population is very small on beans and control measures are not required. However, if damage is appreciable, control is achieved by spraying with carbaryl, dicofol, endosulfan, malathion, or monocrotophos. A predacious mite, *Phytoseiulus riegeli* (Phytoseiidae) has controlled *T. cinnabarinus* on cotton in Kenya and Uganda (Hill, 1975). This predator can also be used for controlling red spider mite on beans.

Tropical spider mite (Acarina: Tarsonemidae)

The tropical or broad spider mite, *Polyphagotarsonemus latus* (Banks), is a minor pest of beans, cotton, coffee, potato, and tomato in some parts of Africa. It has occurred in Kenya, Tanzania,

Uganda, Burundi, Central African Republic, Sudan, and Nigeria (Hill, 1975).

Life cycle. The adult mite is yellow or pale green. It is about 1.5 mm long and, because of its color and size, is sometimes very difficult to see without a magnifying glass. Eggs are laid singly on the underside of young leaves. They are oval shaped but flattened on the lower side. The upperside of the egg is covered with five or six rows of white tubercles. Eggs are 0.7 mm long and hatch in two to three days. The larva turns into a pseudopupa and remains in this stage for two to three days. Adult males usually pick up the female pseudopupae and carry them to newly opened leaves. Male pupae are not usually moved but when the adult males emerge, they migrate to new leaves. A female mite lives for about 14 days, laying two to four eggs per day (Hill, 1975).

Damage and control. The broad spider mite damages bean plants after flowering, especially during humid and warm weather. The sucking activity of the mite causes leaf edges to roll upwards with a shiny appearance. The lower leaf surface may turn purplish. Young leaves do not develop normally and remain stunted, turning yellow. Sometimes pods are also attacked (Hill, 1975).

Insecticides used for *T. cinnabarinus* effectively control this mite. Dimethoate is not effective.

Snails and slugs (Molluscs)

Snails and slugs are minor pests of beans in some parts of Africa, slugs being more important.

Limicolaria kambeul (Achatinidae). *Limicolaria kambeul* Burgess is a snail found in humid parts of Africa south of the Sahara. It has occurred in eastern Africa, Sudan, and Congo (Schmutterer, 1969), in wet places and in areas with high relative humidity.

The snail possesses a calcium salt spiral shell in which the visceral hump is coiled (Figure 186). The head and foot of immature and adult snails are grayish brown to brown. Juvenile and adult snails measure 1.2-1.6 cm and 6-10 cm, respectively. The shell is usually

yellow or yellow-brown and sometimes has longitudinal brown stripes. The adult snail lays white, spherical eggs in a nest prepared in damp soil. After hatching, young snails remain within the soil for some time before surfacing to feed on organic matter. The pest is nocturnal and rests during the day on either plants or soil.

The snail attacks a variety of crops, including beans, during the rainy season. However, maize and peanuts are preferred hosts. Immature snails do the most damage by making large holes in bean leaves during the night. They usually appear in large numbers.

Handpicking is the easiest way to control the pest in small bean fields as populations of the snail are usually low. However, molluscide baits, consisting of metaldehyde added to wheat or sorghum bran, can also be placed under attacked plants.

Slugs. The most commonly found slugs on beans are *Limax maximus* L., *Deroceras agreste* L., and *Vaginulus plebeius* (Fisher). These species also occur in tropical countries of Asia and Latin America. Slugs, unlike snails, are streamlined and have no spirally wound shell. The biology of slugs is not well known, but see Chapter 22, this volume, for a description.

To control slugs, it is important to keep bean fields clean of plant debris and weeds which act as shelter for slugs. Because infestation by slugs often starts from field borders, control can be achieved by spraying border plants with carbaryl or dimethoate in late afternoon or early evening.

Future of Pest Control in Africa

Chemical control is perhaps the most common method of controlling bean pests. Although the use of insecticides such as dimethoate, endosulfan, fenitrothion, and monocrotophos, has been highly successful, it has sometimes caused adverse effects, especially in developed countries. For example, insecticides kill the natural enemies of pests and encourage the development of resistant strains of economically important pests. Moreover, insecticides are often too expensive or unavailable to subsistence farmers in many developing countries, including those of Africa. Hence, a high

research priority in bean entomology in Africa must be to conserve the natural biological control of existing and potential pests.

That insecticides are applied only when pest infestation warrants it and not on a routine basis, must be stressed as part of effective and economical control of bean pests. More attention must also be given to nonsynthetic chemical insecticides. The use of plant extracts such as neem, offers a new dimension for future chemical control of insects on beans (Hongo and Karel, 1986).

Sources of resistance to important insect pests must be incorporated into agronomically acceptable cultivars such as those which are already resistant to important plant diseases. The development of varietal resistance to bean pests, however, will take time. Moreover, as with other crops, resistance to insect pests will not, by itself, prevent yield losses caused by the whole disease and pest complex. However, the use of resistant cultivars will reduce the need for repeated insecticide applications and favor the survival of natural enemies, allowing for a more effective, natural, biological control of pests.

The use of natural enemies (parasites, predators, and pathogens) as a method of controlling bean pests has not yet been adopted in Africa, even though it is effective. Yet, many pests such as bean aphids, are controlled, without human intervention, by their parasites in many bean-growing countries of Africa. It is only recently that exotic aphidiid parasites (Hymenoptera) were introduced into Burundi to assist the indigenous *Aphidius colemani* Viereck (Autrique et al., 1985) that was partly regulating bean aphid populations. The short growing season of beans and fallow periods may hinder the implementation of an effective and deliberate biological control strategy for bean pests in traditional African farming systems.

Various cultural practices such as optimal plant populations, appropriate time of planting, species diversity, use of trap crops, crop rotation, intercropping, and removal of crop residues, have shown potential for controlling bean pests (Karel et al., 1983). Cultural practices are readily available to the subsistence farmer and, in most cases, do not require extra investment. Future control

methods must emphasize the implementation of cultural practices that support biological control and host-plant resistance strategies.

The integration of various control methods requires the development of an "integrated pest management (IPM)" strategy. IPM approaches the control of crop pests from an ecological viewpoint and must be based on an adequate knowledge of the agroecosystem. It offers a framework for developing a system of pest control which combines all suitable control methods such as host-plant resistance, cultural practices, biological control, and chemical control. The core of this approach lies in applying the concept of "economic damage threshold." This threshold is defined as the density of a pest population at which it does not cause enough injury to justify the economic costs of control efforts (Karel, 1983; Karel et al., 1983; Matteson, 1984). When the pest density surpasses the economic threshold, control measures must be taken. Because IPM is dynamic, its improvement requires constant feedback from field experiences. Hopefully, some progress will be quickly made to develop and implement IPM programs for beans in Africa. However, the needs of subsistence farmers are complex and require a total production package. IPM programs must therefore be developed as part of that package.

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Chapter 22

INSECTS AND OTHER INVERTEBRATE BEAN PESTS IN LATIN AMERICA

César Cardona*

Introduction

As with other crops, insects and other pests affect common or dry bean production before and after harvest. Many species have been listed as pests of common beans (King and Saunders, 1984; Mancía and Cortez, 1975; Ruppel and Idrobo, 1962). The few that are recognized as economically important pests are listed in Table 1 according to their main feeding habits. The given division cannot be maintained strictly because the Mexican bean beetle and chrysomelids may also attack young pods while pod borers such as *Epinotia* and *Heliothis*, may also feed on leaves and buds. Slugs and spider mites are not insects but are listed because of their economic importance in certain areas.

This chapter updates pertinent literature available on bean pests in Latin America, with emphasis on bean-pest ecology and non-chemical control methods. Emphasis is also given to those insects or pest situations for which valuable, new information has been published since 1980 (van Schoonhoven and Cardona, 1980).

Geographical Distribution of Important Bean Pests

A simplified distribution of the principal bean pests in Latin America is shown in Figure A. Documentation on the bean-pest complex has improved since 1980. New authoritative descriptive reviews have been published. Table 2 lists general references on the insect fauna registered on beans in Latin America.

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Table 1. Major insect and invertebrate bean pests found in Latin America.

| Feeding norm and common name | Scientific name |
|-------------------------------------|---|
| Seedling-attacking insects | |
| Seedcorn maggot | <i>Delia platura</i> (Meigen) |
| Cutworms | <i>Agrotis ipsilon</i> , <i>Spodoptera exigua</i> (Hübner) |
| White grubs, crickets | <i>Phyllophaga mentriesi</i> (Blanchard), <i>Gryllus assimilis</i> F. |
| Lesser cornstalk borer | <i>Elasmopalpus lignosellus</i> (Zeller) |
| Leaf-feeding insects | |
| Chrysomelids | <i>Diabrotica</i> spp., <i>Cerotoma</i> spp. |
| Saltmarsh caterpillar | <i>Estigmene acrea</i> (Drury) |
| Bean leafroller | <i>Urbanus proteus</i> (L.) |
| Webworm (Hedylepta) | <i>Omiodes indicata</i> (F.) |
| Mexican bean beetle | <i>Epilachna varivestis</i> Mulsant |
| Leafminers | <i>Liriomyza</i> spp. |
| Piercing and sucking insects | |
| Leafhopper | <i>Empoasca kraemeri</i> Ross & Moore |
| Common whitefly | <i>Bemisia tabaci</i> (Gennadius) |
| Aphids | <i>Aphis</i> spp., and others |
| Thrips | <i>Caliothrips braziliensis</i> (Morgan) |
| Stink bugs | <i>Acrosternum marginatum</i> (Palisot de Beauvois), and others |
| Pod-attacking insects | |
| Bean-pod weevil | <i>Apion godmani</i> Wagner |
| Pod borers | <i>Heliothis</i> spp., <i>Epinotia opposita</i> Hein., <i>E. aporema</i> (Walsingham), <i>Etiella</i> <i>zinkenella</i> (Treitschke), <i>Maruca testulalis</i> (Geyer) |
| Storage insects | |
| Bruchids | <i>Acanthoscelides obtectus</i> (Say), <i>Zabrotes subfasciatus</i> (Boheman) |
| Other pests | |
| Spider mites | <i>Tetranychus desertorum</i> Banks, <i>Tetranychus</i> <i>urticae</i> Koch |
| Tropical spider mites | <i>Polyphagotarsonemus latus</i> (Banks) |
| Slugs | <i>Sarasinula plebeia</i> (Fisher) ^a |

a. Identification needs further confirmation (K. L. Andrews, personal communication).

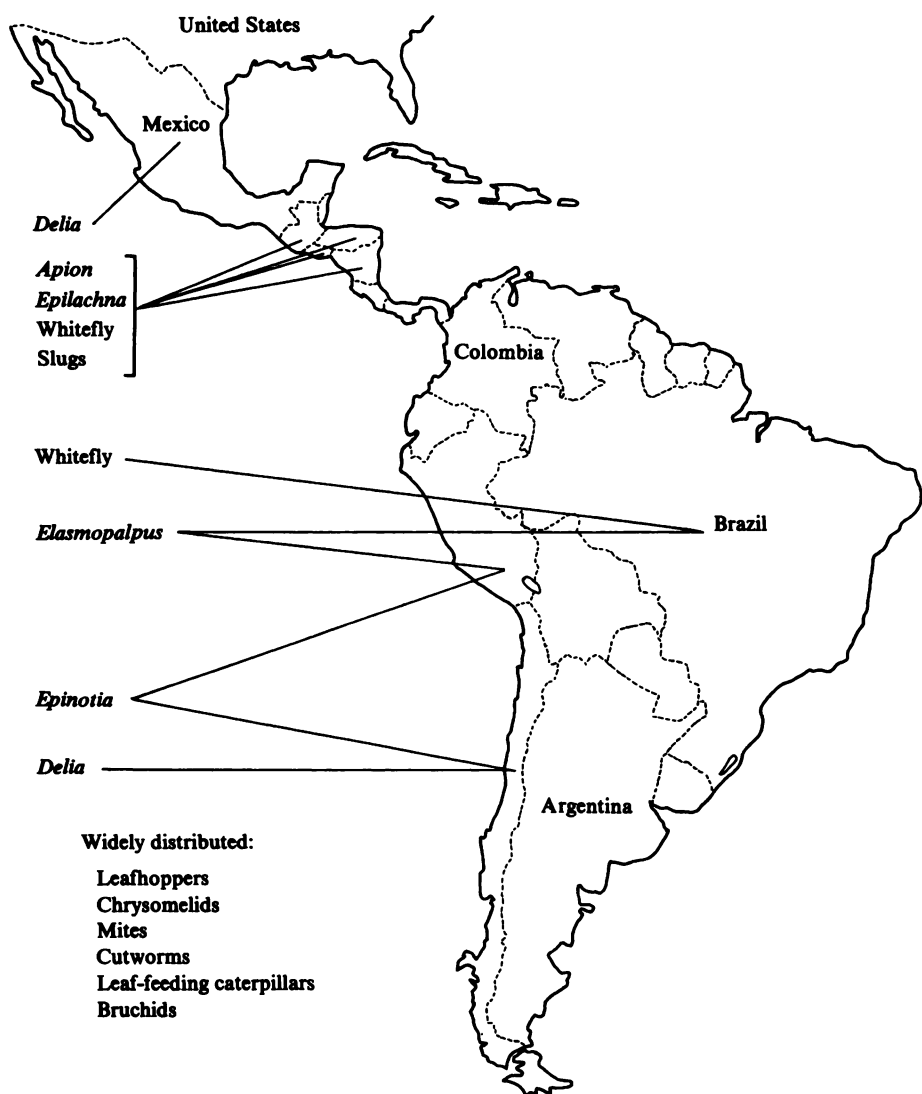


Figure A. Geographic distribution of bean pests in Latin America.

Table 2. Selected general references on bean pests in Latin America.

| Country or region | Source |
|--------------------------|--|
| Argentina | Costilla (1983) |
| Brazil | Costa and Rossetto (1972); de Carvalho et al. (1982) |
| Central America | Bonnefil (1965); King and Saunders (1984); Andrews (1984) |
| Chile | Olalquiaga-Fauré (1953); Ripa-Schaul (1981) |
| Colombia | Posada-O. et al. (1970); Posada-O. and García (1976) |
| Cuba | Pendás-Martínez (1983) |
| Guatemala | Salguero (1981) |
| Haiti | Kaiser and Meléndez (1976) |
| Honduras | Peairs (1980); Passoa (1983); Andrews (1984) |
| Latin America | Ruppel and Idrobo (1962); van Schoonhoven and Cardona (1980); Cardona et al. (1982b) |
| Mexico | Miranda (1971); Sifuentes-A. (1981); Armenta-Cárdenas (1983) |
| Nicaragua | Sequeira et al. (1978) |
| Peru | Wille-T. (1943); Avalos-Q. (1977); Avalos-Q. (1982) |
| El Salvador | Mancía and Cortez (1972); Mancía and Cortez (1975) |
| Caribbean region | Parasram (1973) |

Leafhoppers, chrysomelids, cutworms, spider mites, leaf-feeding caterpillars, and storage insects (bruchids) are the most widely distributed pests of beans in Latin America. Of regional importance in Mexico and parts of Central America are the bean-pod weevil, the common whitefly, and, to a lesser extent, the Mexican bean beetle. The seedcorn maggot is more common and important in Mexico and Chile than elsewhere, while *Epinotia* species (pod borers) continue to be major pests in Chile and Peru.

The most important recent change in pest status is the rise of the slug (*Sarasinula plebeia* (Fisher)) to a key pest position in Central

America. This phenomenon has been well documented (Andrews, 1983a; Andrews and Dundee, 1986). Interestingly, leafminers (*Liriomyza* species) have become more troublesome in Peru and Ecuador than before, possibly as a result of insecticide abuse and other factors.

Economic Losses

Insect losses vary widely between and within regions. Estimates based upon yield reductions in insecticidal trials tend to overestimate the importance of insects. Thus, yield losses resulting from leafhopper damage during dry seasons are estimated as high as 80%, while losses during wet seasons averaged 22% (CIAT, 1975). A more realistic estimate of the economic importance of the leaf-hopper was obtained by Pinstup-Andersen et al. (1976) who calculated an 11% crop loss in commercial fields in Colombia.

Losses from the bean-pod weevil (*Apion* spp.) in Central America are variable. Sifuentes-A. (1981) estimated 50% losses occurred in Mexico, while Guevara-Calderón (1961) reported as much as 80% damage. Salguero (1983b) found an average of 17% damage in central, and 9%-60% damage in southeastern, Guatemala.

Losses can be expressed in other terms and not necessarily as percentage of yield reductions. In Central America, slugs affect half a million farmers' crops per year (Andrews, 1983a). Since there are few crop alternatives for the subsistence farmer to grow, the pest becomes a serious socioeconomic problem. Bruchid damage is another example of a pest problem which affects small farmers' economies. Fear of bruchid damage forces farmers to sell their produce as soon as possible, even when supply is high and prices are low (van Schoonhoven, 1976).

A recent survey of bean scientists (CIAT, 1984b) revealed that, at least in qualitative terms, the leafhopper is regarded as the most important insect pest of beans in Latin America (Table 3), followed by chrysomelids, bruchids, whitefly, and soil insects. *Apion godmani* Wagner and slugs were not considered as important, possibly because the sample size among Central American scientists was small. Chrysomelids, leafhoppers, whitefly, and soil insects were

Table 3. Insect pests of beans in Latin America ranked by 35 bean scientists, according to their importance in terms of incidence and need to be controlled by chemical means.

| Pest | Times insect mentioned as: | | | Weighted rank for importance | Chemical control required | |
|---------------------------|----------------------------|----------|------------|------------------------------|---------------------------|------|
| | Severe | Moderate | Occasional | | Mentions | Rank |
| Leafhoppers | 13 | 14 | 7 | 1 | 20 | 2 |
| Chrysomelids | 10 | 19 | 6 | 2 | 24 | 1 |
| Bruchids | 13 | 8 | 6 | 3 | 9 | 5 |
| Whiteflies | 9 | 11 | 2 | 4 | 13 | 3 |
| Soil insects | 6 | 9 | 14 | 5 | 10 | 4 |
| Aphids | 1 | 9 | 14 | 6 | 9 | 5 |
| Slugs | 6 | 3 | 6 | 7 | 7 | 6 |
| Spider mites | 1 | 7 | 11 | 8 | 3 | 9 |
| <i>Heliothis</i> spp. | 3 | 3 | 10 | 9 | 6 | 7 |
| Leaf-feeding caterpillars | 0 | 7 | 10 | 10 | 6 | 7 |
| Other pod borers | 0 | 7 | 6 | 11 | 4 | 8 |
| Stink bugs | 2 | 1 | 13 | 12 | 2 | 10 |
| <i>Apion</i> spp. | 2 | 0 | 2 | 13 | 2 | 10 |
| <i>Epilachna</i> sp. | 1 | 1 | 3 | 14 | 2 | 10 |

SOURCE: CIAT, 1984b.

regarded as those pests for which chemical controls were more frequently needed.

Progress has been made in establishing initial action thresholds and/or economic injury levels for controlling identified pests (Table 4). These may change as research on new or refined techniques continues.

Table 4. Action thresholds for some bean pests, according to their economic injury level.

| Pest | Country | Economic injury level | Source |
|-------------------------|-------------|---|--|
| <i>Apion</i> spp. | Guatemala | 4-6 adults/4 m of row | Salguero (1983b) |
| <i>Acrosternum</i> spp. | Colombia | 1 late-instar nymph/ 0.6 m ² | Hallman et al. (1986) |
| Chrysomelids | Colombia | 2-4 adults/plant | Cardona et al. (1982a) |
| Cutworms | General | 10% of plants cut | Hallman (1985) |
| <i>Epilachna</i> sp. | Mexico | 25 adults/ha | Cadena-L. and Sifuentes-A. (1969) |
| | USA | 1-1.5 larvae/plant | Michels and Burckhardt (1981) |
| <i>Heliothis</i> spp. | Colombia | 8 larvae/m ² | Hallman (1985) |
| Leafhoppers | Colombia | 2-3 nymphs/leaf | CIAT (1976) |
| | Honduras | 2 nymphs/leaf or 2 adults/plant | Andrews (1984) |
| Leafminers | Peru | 1-2 larvae/leaf | Espinosa-G. and Sánchez-V. (1982) |
| Leafrollers | USA | 26 fourth-instar or 4-5 fifth-instar larvae/plant | Greene(1971a) |
| <i>Omiodes</i> sp. | Brazil | 33% defoliation | de Bortoli (1980) |
| Slugs | El Salvador | 0.2 active slugs/m ² or 0.4 slugs/ traps/night | Andrews and Huezo de Mira (1983) |
| | Honduras | 1 slug/m ² or 1 slug/ trap/night | Andrews and Barletta (1985) |

Control Methods

Bean-cropping systems in Latin America are variable. So are bean-pest control tactics. These vary from sophisticated, large-scale applications of granular insecticides (to control whiteflies in

Argentina) to occasional insecticidal applications by small farmers, or even to complete reliance on natural mortality factors to suppress insect populations.

The short growing season of beans and frequent fallow periods reduce the effectiveness of biological control. Apart from the introduction of larval parasites of Mexican bean beetle in Mexico, there have been no attempts to mass-rear, mass-release, or manipulate parasites or predators of bean pests in Latin America. However, research in this area and in the potential use of pathogenic fungi or bacteria, must continue, if only to know which beneficial organisms must be preserved.

Cultural control practices are important in some cases. Shifting the planting date can reduce pressure from leafhoppers, bean-pod weevils, and seedcorn maggots. However, it has limited applications where rainfall distributions govern planting dates. Common agronomic practices such as weeding, land preparation, and burning of residues, are useful for controlling slugs, cutworms, white grubs, and other soil pests. The common practice of planting associated crops must not be discouraged among small farmers. Research has shown that this system regulates populations of leafhoppers, Mexican bean beetles, *Apion* spp., and chrysomelids.

Host-plant resistance studies have identified cultivars with genetic resistance to leafhoppers, bruchids, bean-pod weevil, Mexican bean beetle, and pod borers. Such studies must continue as a major objective in research, together with studies on minimizing pesticide applications. A decision to spray must not only be based upon expected yield losses, but also upon treatment costs and upon the consequences this spray will have on later pest development. Most national programs have updated their chemical control recommendations. Recently, valuable information has been obtained on action threshold populations and critical crop-growth periods for control of several species. Progress in establishing action thresholds (Table 4) will help formulate recommendations to meet the objective of pest management. Pohronezny et al. (1981) and Andrews (1984) provide recent examples of how to carry out integrated pest management programs.

Seedling-attacking Insects

White grubs, cutworms, and crickets

White grubs, cutworms, and crickets are minor pests of beans in Latin America. Damage from these insects is usually confined to small scattered areas of bean-producing regions and plant losses are not high. Outbreaks, however, can be locally devastating.

Common names frequently used for white grubs in Latin America include "gallinas ciegas," "chizas," "mayates," and "mojojoys." Cutworms are called "tierreros," "trozadores," "cortadores," "nocheros," "rosquillas," "lagarta militar," and "lagarta rosca." Common names for crickets and mole crickets are "grillos" and "grillotopos," respectively.

White grubs (Figure 187) feed on roots and show a characteristic patchy distribution. Damaged plants wilt and exhibit yellowing of leaves. Plant losses from white-grub attack usually occur in crops that follow pasture. Losses can be reduced by proper land preparation and weed control or, if there is a history of previous attacks, by incorporation of granular insecticides. *Phyllophaga menetriesi* (Blanchard) is described by King and Saunders (1984) as an important species in Central and South America.

Cutworms damage beans by cutting stems of young seedlings (Figure 188). Older plants can be damaged by stem girdling, although this damage is less common. *Agrotis*, *Feltia*, and *Spodoptera* are common cutworm genera and *Agrotis ipsilon* (Hufnagel) is the most important species. The biology and control of cutworms are discussed by Metcalf et al. (1962).

Cutworm attacks in beans are sporadic and difficult to predict. Therefore, it is better to control cutworms with baits placed, in late afternoon, near plants rather than with preventive insecticide treatments such as granular formulations of various insecticides. A mixture of sawdust, molasses, and trichlorfon or carbaryl is effective, and controls crickets and millipedes as well.

Crickets and mole crickets have been listed as bean pests in some areas (Posada-O. et al., 1970). However, they seldom cause significant economic losses (Figure 189).

Seedcorn maggot (Diptera: Anthomyiidae)

The seedcorn maggot, *Delia platura* (Meigen) (syn. *Hylemya cilicrura* Rond.), is a bean pest in Chile, Mexico, and parts of United States and Canada. It has also been reported from Central America (King and Saunders, 1984) and Brazil (Hohmann, 1980). There has been some confusion about the taxonomy of this group: the genus has been named *Delia*, *Phorbia*, and *Hylemya*. McLeod (1965) separated species on the basis of their nutritional requirements and infertility of interspecific hybrids. Maize, beans, potatoes, beets, tobacco, vegetables, and peas have been listed as host plants. Damage is more serious in Mexico and Chile than elsewhere in Latin America.

Common names for the seedcorn maggot in Latin America are "mosca de la semilla," "mosca de la raíz," "gusano de la semilla," and "mosca de semente." The biology of this species has been studied by Harris et al. (1966), Hohmann (1980), and Miller and McClanahan (1960). Adults resemble houseflies and females lay eggs near seeds or plants in the soil. Larvae feed on bean seeds (Figure 190) or seedlings (Figure 191), and pupate in the soil. Eggs are white and hatch in two (Harris et al., 1966) or four to eight days, depending on the temperature (Sandsted et al., 1971). The pupal stage lasts 9-12 days and there can be as many as three generations per crop. The first generation is the most damaging.

Leaf damage by *D. platura* ranges from a few holes in the first true leaves to complete destruction of the growing point. In laboratory experiments, 5-10 maggots per seed were required to significantly reduce stands of kidney, lima, and snap beans (Vea et al., 1975). Subsequently, Vea and Eckenrode (1976b) determined that a 25% loss of the first pair of unifoliate leaves significantly reduced yield in snap beans by 11%-48%. In common beans, a loss as large as 70% of the first pair of unifoliate leaves did not affect final yields. When the maggot feeds on the growing point, the resulting damaged plant is stunted, incurring the name "baldhead." Most of such plants shrivel and die, resulting in high plant stand losses.

Cultural practices help reduce seedcorn maggot damage. Shallow planting in warm, moist soil can hasten emergence and thus reduce

the susceptible period (Sandsted et al., 1971). Montecinos-Urbina (1982) recommended late planting, especially in areas with soils rich in organic matter which may attract ovipositing females. Biological control was not effective (Miller and McClanahan, 1960).

Resistance to seedcorn maggot was found by Vea and Eckenrode (1976a) in two breeding lines which had significantly lower levels of stand losses than did susceptible commercial cultivars. White-seeded beans were more susceptible. Hagel et al. (1981) found some variability for seedcorn maggot resistance in 160 common bean accessions, but concluded that resistant materials benefited from the additional protection provided by treatment with chlorpyrifos. Black, pink, and dark Red Kidney types were less susceptible. Guevara-Calderón (1969) in Mexico also found less damage in black-seeded cultivars than in yellow.

For many years, a dieldrin seed-dressing was used to control *D. platura*. As this product is prohibited in many countries, and as the insect developed resistance to chlorinated hydrocarbons, recent research has focused on identifying alternatives. Chlorpyrifos was recommended by Gould and Mayor (1975), Crowell (1976), and Ruppel (1982) who also recommended seed-dressing with diazinon. Granular formulations of carbofuran, fonofos, and phorate have also been effective (Eckenrode et al., 1973; Ruppel, 1982).

Lesser cornstalk borer (Lepidoptera: Pyralidae)

The lesser cornstalk borer (*Elasmopalpus lignosellus* (Zeller)) is a widespread pest of beans in Central and South America, but is most serious in Brazil (Costa and Rossetto, 1972) and Peru (Avalos-Q. and Lozano-V., 1976). This polyphagous insect attacks beans, sugarcane, cotton, sorghum, rice, peanuts, cowpea, and several graminaceous weeds. Common names are "coralillo," "barrenador menor del tallo," "gusano saltarín," and "elasmó."

Females lay eggs singly on leaves, stems, or in the soil. The larval stage lasts 13-26 days, and there are six instars. Pupation occurs in the soil (Leuck, 1966). Dupree (1965) found little evidence of stem-boring activity before the third larval instar.

Damage is caused by the larvae (Figure 192) which enter the stem just below the soil surface and tunnel upwards (Figure 193). Attacks

usually occur when plants are 10-12 cm high with two leaves. Damaged plants look flaccid and wilt or lodge. Attacks usually occur in irregular patterns (Salinas, 1976). These symptoms are similar to those caused by the scolytid *Hypothenemus* sp. and the root borer *Conotrachelus phaseoli* Marshall (Calil and Chandler, 1982; Calil et al., 1982).

Avalos-Q. and Lozano-V. (1976) evaluated 93 bean cultivars for lesser cornstalk borer resistance but did not find variability. Some species of Braconidae, Ichneumonidae, and Tachinidae have been identified as larval parasites (Leuck and Dupree, 1965; Salinas, 1976). However, their efficacy in suppressing lesser cornstalk borer populations has not yet been evaluated.

Seed dressings with insecticides were evaluated by Campos-P. (1972) with variable results. Granular insecticides placed near the seeds must be applied before planting. Campos-P. (1972) and Wille-T. (1943) recommend clean fallowing for prolonged periods and heavy irrigation to achieve control.

Leaf-Feeding Insects

Chrysomelids (Coleoptera: Chrysomelidae)

Chrysomelid beetles are among the most widely distributed pests of beans in Latin America (Bonnefil, 1965; King and Saunders, 1984; Passoa, 1983; Ruppel and Idrobo, 1962). Prevalent genera are *Diabrotica*, *Neobrotica*, and *Cerotoma*. Other genera listed by Grillo-Ravelo (1979), Popov et al. (1975), Ruppel and Idrobo (1962), Valverde et al. (1978), and Yépez-Gil and Montagire-A. (1985) include *Epitrix*, *Systema*, *Colaspis*, *Gynandrobrotica*, *Chalepus*, *Nodonota*, *Chaetocnema*, and *Maecolaspis*. *Cerotoma* and *Diabrotica* are the most important, and this review will concentrate on the banded cucumber beetle (*Diabrotica balteata* LeConte) (Figure 194) and the bean beetle (*Cerotoma facialis* Erickson) (Figure 195).

Some common names for chrysomelids in Latin America are "crisomélidos," "doradillas," "diabrotica," "tortuguillas," "mayas," "vaquitas," "vaquinhas," and "cucarroncitos de las hojas."

Chrysomelids can affect beans in three ways: larvae damage roots and root nodules; adults feed on foliage at all stages of crop growth; and adults act as vectors of important viral diseases (Gámez, 1972). Sometimes adults also feed on flowers and young pods.

The biology of the banded cucumber beetle (*D. balteata*) as a polyphagous species was studied by Pulido-F. and López de Pulido (1973). They listed 32 host plants for this species. Of these, beans and maize were hosts for larvae and adults. González et al. (1982) demonstrated that *D. balteata* does not survive on bean roots and the bean beetle (*C. facialis*) does not feed on maize roots. This confirmed previous findings by Young and Candia (1962) that *D. balteata* adults have a feeding preference for young bean plants and oviposition preference for soil in which young maize plants are growing.

Females undergo a preoviposition period which varies from 5-12 days in Colombia (González et al., 1982) to 4-8 days in Mexico (Young and Candia, 1962). Oviposition takes place singly or in clusters of as many as 12 eggs in soil cracks or beneath plant debris. A female can lay as many as 800 eggs and has an average life cycle of 37 days. Eggs hatch in five to six days, and the three larval instars together last 14 days. Pupation takes place in a cell in the ground (Pitre and Kantack, 1962) and lasts six to seven days. The sex ratio is usually 1:1. Pulido-F. and López de Pulido (1973) found that nutrition has a significant effect on female fecundity. Females fed with soybean leaves laid an average of 326 eggs, while those fed with soybean leaves, flowers, and young pods laid 975 eggs. Maximum egg production by females fed with bean leaves was 144 per individual.

The biology of *C. facialis* is similar. Females live 52 days, undergo a 5-12 day preoviposition period, and lay an average of 532 eggs per female. The egg stage lasts six days, there are three larval instars which together last 10-11 days, and pupation lasts six to seven days. The sex ratio is 1:1 (González et al., 1982).

Most damage by chrysomelids occurs during the seedling stage. Adult (Figure 196) and larval (Figure 197) damage at different population levels and crop-growth stages was evaluated by Cardona et al. (1982b). Second- and third-instar larvae were more damaging than first instars and could cause as much as 100% loss under

greenhouse conditions. Significant damage and reduction in leaf area were detected when plants were infested one, four, and seven days after planting. Fourteen-day-old and older plants did not show a significant reduction in leaf area. Under field conditions, mixed and pure populations of *C. facialis* and *D. balteata* caused yield losses when infestation levels were two to four adults per plant during early growth stages and, to a lesser extent, during flowering. No significant damage occurred at other growth stages.

Intercropping of beans with banana in Costa Rica significantly reduced populations of *D. balteata* and *C. ruficornis* (Olivier) (Risch, 1982). Predation of adults by reduviids has been observed (Hallman, 1985). Young and Candia (1962) identified a tachinid adult parasite. When natural control is not effective and populations reach critical levels, sprays with carbaryl, methomyl, or malathion are useful. Insecticide applications are usually not justified when average natural populations are 0.6-1.0 adults per plant. Cardona et al. (1982b) recommend limiting sprays to early growth stages or the initial flowering period when populations are higher than two adults per plant.

Mexican bean beetle (Coleoptera: Coccinellidae)

The Mexican bean beetle, *Epilachna varivestis* Mulsant, called "conchuela" in Latin America, is basically a pest of soybeans (Turnipseed and Kogan, 1976). However, it attacks common beans in United States, Mexico, parts of Guatemala, Honduras, and El Salvador. It is also a pest of cowpea and lima beans in El Salvador (Mancía and Román-Cortez, 1973). Beggarweed, scarlet runner bean (*Phaseolus coccineus* L.), and *Lablab purpureus* (L.) Sweet are also host plants (Turner, 1932). Augustine et al. (1964) and Wolfen-barger and Slesman (1961c) found that mung bean (*Vigna radiata* (L.) Wilczek) and urd bean (*V. mungo* (L.) Hepper) were less preferred hosts than common bean (*Phaseolus vulgaris* L.).

Damage is caused by both larvae (Figure 198) and adults (Figure 199) which feed on leaves. Stems and pods can also be damaged when populations are high. Larvae do not chew leaves but scrape the tissue, compress it, and then swallow the juices. Damage is more

serious at early crop-growth stages and mature larvae are more damaging than adults (Turner, 1935).

The preoviposition period lasts 7-15 days. Females lay yellow to orange-colored eggs on the undersurface of leaves. The eggs are laid in groups of 36-54 per batch with an average of 43 (Mancía and Román-Cortez, 1973). Hatching occurs six days later and the four larval instars are completed in 15-16 days. The prepupal stage lasts two days and the pupal stage six to seven days. Pupation occurs on leaves and pupae attach to the lower leaf surface. Adults are copper-colored, with 16 black spots on the elytra, and live four to six weeks. In United States, adults hibernate, often gregariously, in woodlands and bean debris (Elmore, 1949). In El Salvador, the beetle passes through four generations from May to November (Mancía and Román-Cortez, 1973).

Mellors and Bassow (1983) compared the life cycles on beans and soybeans and did not find differences in developmental periods. Hammond (1984) later reported that development on common beans took 16% less time than on soybeans.

There have been several studies on host-plant resistance to the Mexican bean beetle, with varying results. For example, Wolfenbarger and Slessman (1961c) did not observe resistance in the *P. vulgaris* accessions they investigated. They rated the cultivars Idaho Refugee and Wade as very susceptible. However, Campbell and Brett (1966) reported them as resistant. These authors found more variability among *P. vulgaris* cultivars. They also showed that egg number, egg masses, and adult weights were significantly reduced when beetles were reared on resistant cultivars. In Mexico, Montalvo and Sosa (1973) classified the cultivars Guanajuato 18 and Zacatecas 48 (*P. vulgaris*) and Puebla 86 (*P. coccineus*) as resistant. Egg numbers and adult weights were reduced—nonpreference and antibiosis were the mechanisms apparently responsible. More recently, cultivars Regal (snap beans) and Baby Fordhook and Baby White (lima beans) were reported as resistant (Raina et al., 1978).

The mechanisms of resistance to the Mexican bean beetle need further clarification. Augustine et al. (1964) suggested that high sucrose concentrations act as an arrestant. This hypothesis is

contrary to findings by Jones et al. (1981) and LaPidus et al. (1963) who concluded that sugar acts as a phagostimulant and that phenolic compounds reduce feeding rates. Experimental data by Arévalo-Aponte (1977) supports the hypothesis of the importance of sugar concentration as a phagostimulant. Resistant cultivars Puebla 84 and Zacatecas 48 had lower concentrations of saccharose, fructose, and galactose than susceptible cultivars. An earlier hypothesis on the importance of phaseolunatin (a cyanogenic glycoside) as an attractant (Nayar and Fraenkel, 1963) also needs further experimental support.

Recent work on resistance to Mexican bean beetle has concentrated on improving screening methodologies and knowledge of host plant-insect interactions (Raina et al., 1980; Wilson, 1981).

The role of natural enemies in suppressing beetle populations is an active area of research. Predators of eggs and first-instar larvae include *Coleomegilla maculata* De Geer and *Hippodamia convergens* Guérin-Méneville. Other predators are the pentatomids *Podisus maculiventris* (Say) and *Stiretrus anchorago* (F.) (Waddill and Shepard, 1975). The mite *Coccipolipus epilachnae* Smiley has been observed attacking adults in El Salvador (Smiley, 1974) and United States (Schroder, 1979). Possibly the best-known natural enemy of the Mexican bean beetle is the eulophid larval parasite *Pediobius foveolatus* (Crawford) which was effectively used on soybeans in United States (Stevens et al., 1975). This parasite was introduced into Mexico and became established within three years (Carrillo-Sánchez, 1977). Carrillo also reports that the tachinid *Aplomyiopsis epilachnae* (Aldrich) can parasitize as many as 70% of larvae. The bacteria *Bacillus thuringiensis* Berliner controlled larvae under laboratory and field conditions (Cantwell and Cantelo, 1982).

Removal of plant debris and deep plowing are cultural practices that control the insect. Turner (1935) indicated that damage by beetles is decreased when plant densities are reduced. Crop associations (maize-beans) also reduce beetle populations (Martínez-Rodríguez, 1978; Sánchez-Preciado, 1977). The effect of companion plantings was studied by Latheef and Irwin (1980). Fewer beetles were found on beans bordered by french marigold,

but the beneficial effect was overshadowed by allelopathic effects of french marigold on beans.

Carbaryl, malathion, and methyl parathion effectively control this insect (Cadena-L. and Sifuentes-A., 1969). The first application is made when there are 25 adults per hectare present, a second spray may be combined with *Apion* control, and a third application is made only if necessary. In United States, farmers are advised to spray when one beetle or egg mass is found per 1.8 m of row. The beetles are counted on the ground after shaking the plant. In Wyoming, USA, Michels and Burkhardt (1981) established an economic threshold level of 1-1.5 larvae per plant. Hagen (1974) obtained an effective 10-week control with granular formulations of disulfoton, carbofuran, phorate, aldicarb, and fensulfothion which were applied at planting. The effectiveness of pyrethroids was reported by McClanahan (1981). Zungoli et al. (1983) found that the chitin inhibitor, diflubenzuron, gave adequate control with no apparent effect on the main parasite, *P. foveolatus*.

Bean leafroller (Lepidoptera: HesperIIDae)

The bean leafroller, (*Urbanus* (syn. *Eudamus*) *proteus* (L.)), is called “gusano fósforo” and “gusano cabezon” in Latin America. This insect is widely distributed from United States (Quaintance, 1898) to Brazil (Freitas, 1960) and Chile (Díaz-P., 1976).

In general, the bean leafroller is a minor pest of beans. In Florida, USA, Greene (1971a) calculated that economic damage occurs when more than 725 cm² of leaf area per plant is destroyed. Yield reduction occurs when there are more than 26 fourth-instar larvae per plant. More than 4 fifth-instar larvae per plant would also be of economic significance. However, these population levels were seldom observed, possibly because only 4% of individuals reach the fifth instar.

The adult butterfly lays one to six eggs per leaf on the lower surface. Larvae fold the leaf margin (Figure 200) and feed and pupate within the fold. Larvae are recognized by their three dorsal longitudinal lines and large red-brown head capsules (Figure 201). In Florida, eggs hatch in three days (Greene, 1971b) and larval and

pupal stages last 15 and nine days, respectively. In Colombia, van Dam and Wilde (1977) found that the egg stage lasts an average of four days, while larval and pupal stages develop in 23 and 11 days, respectively. The duration of immature stages is longer in Chile (Díaz-P., 1976).

Chemical control is seldom required. Effective natural control (21%-40% larval parasitism) was observed in Colombia (van Dam and Wilde, 1977).

Saltmarsh caterpillar (Lepidoptera: Arctiidae)

The common Latin American name for the saltmarsh caterpillar, *Estigmene acrea* (Drury) is "gusano peludo." *Estigmene acrea* is a cosmopolitan species and is basically a cotton pest. It also attacks lettuce and sugar beets and, although commonly found on beans, is not regarded as a major pest of this crop. Other host plants include maize, horticultural crops, soybean, sesame, tobacco, and several weeds (Young and Sifuentes-A., 1959).

Biological studies of this species were made by Stevenson et al. (1957) and Young and Sifuentes-A. (1959). Adult moths lay egg masses with as many as 1000 eggs. Larvae develop in 17-19 days. Young larvae remain aggregated (Figure 202) and can skeletonize isolated bean plants. Older larvae are solitary. Their bodies are covered with setae (Figure 203). Pupation takes place on the soil in plant debris.

Good levels of natural control were detected by Young and Sifuentes-A. (1959) in Mexico and by Rodas (1973) in Colombia. Economic levels are seldom reached and chemical control is rarely needed.

Hedylepta (Lepidoptera: Pyralidae)

Before the name of the genus was changed, the common name frequently used for this insect was "hedylepta." *Omiodes* (syn. *Hedylepta*; syn. *Lamprosema*) *indicata* (Fabricius) is also known as "pega-pega" in some areas of Latin America. *Omiodes indicata* is a

pest of beans, soybeans, and other legumes in Central (King and Saunders, 1984) and South America (Ruppel and Idrobo, 1962).

Adult moths oviposit on the lower surface of leaves. A female lays an average of 330 eggs. Hatching occurs in four days and green larvae (Figures 204 and 205) develop in 11 days. They pupate (Figure 206) and emerge five days later as an adult (Kappor et al., 1972). Larvae weave leaves together (hence, the alternative name, webworm) and feed on the parenchyma (Figure 207), safe from insecticides.

The level of natural control is high (García, 1975; Lenis-Lozano and Arias-Sánchez, 1976) and the insect does not usually become a serious pest. Chemical control is seldom needed and is recommended only if 33% or more defoliation occurs at flowering (de Bortoli, 1980).

Leafminers (Diptera: Agromyzidae)

Several species of leafminers (Figure 208) occur on beans in Latin America, including the cosmopolitan species *Liriomyza huidobrensis* (Blanchard) and *L. sativae* Blanchard which are polyphagous and widely distributed (Spencer, 1973). Other species include *Melanagromyza phaseolivora* Spencer in Ecuador and *Japanagromyza* species in coastal areas of Peru. Common names for leafminers in Latin America include "minadores," "tostones," and "moscas minadoras."

Liriomyza sativae has a short life cycle of 24-28 days and several generations occur per year. This species is particularly important in Venezuela as a pest of common beans, especially when young plants are attacked. The insect is usually regulated by natural enemies such as braconids, eulophids, and pteromalids (Spencer, 1973).

Lyriomyza huidobrensis is an important pest in certain areas of Ecuador such as the Catamayo and Lambayeque Valleys. The life cycle (17-25 days) was studied by Espinosa-G. and Sánchez-V. (1982). The egg stage lasts two to three days. The larval stage requires seven to nine days, and pupae five to seven days, to develop. Adults live three to six days. There are several generations per year.

Chemical control is difficult. Insecticides can provoke high populations and outbreaks, resulting in severe defoliation and significantly reduced yields (Spencer, 1973). Omethoate, permethrin, and cypermethrin are recommended (Espinosa-G. and Sánchez-V., 1982; Torres-B. and Delgado-A., 1967). The use of plastic sheets lined with adhesive and passed through the field at canopy height has been suggested by Soto-P. (1982) for reducing adult populations. An economic injury level of one to two larvae per leaf was established by Espinosa-G. and Sánchez-V. (1982).

Piercing and Sucking Insects

Leafhoppers (Homoptera: Cicadellidae)

Empoasca kraemeri Ross and Moore is the most important insect pest of beans in Latin America. It occurs in Florida, Central America, Colombia, Ecuador, Peru, and Brazil (de Oliveira et al., 1981; Ross and Moore, 1957). *Empoasca fabae* (Harris) is a closely related species and is a pest of beans in Central America (King and Saunders, 1984). However, workers question its presence south of the United States (Ross and Moore, 1957; van Schoonhoven et al., 1985). Other minor species of *Empoasca* in Latin America are listed by Bonnefil (1965), Langlitz (1964), Ruppel and DeLong (1956), and van Schoonhoven et al. (1985).

Leafhoppers are highly polyphagous (DeLong, 1971). Nymphs of *Empoasca* spp. have been collected from more than 80 cultivated and noncultivated host plants in Colombia. Common names frequently used for leafhoppers in Latin America include “empoasca,” “chicharrita,” “lorito verde,” “cigarra,” “saltahojas,” and “cigarrinha verde.”

The biology of *E. kraemeri* was studied by Wilde et al. (1976). Eggs are inserted singly into leaf blades, petioles, leaf tissues, or stems, with 50%-82% of the eggs located in petioles (Gómez-Laverde and van Schoonhoven, 1977).

Eggs hatch in eight to nine days and the five nymphal instars (Figure 209) are completed in 8-11 days. Adults are green (Figure 210) and have an adult life span, on average, of 62 days. Thirteen to

168 eggs with an average of 107 eggs per female are laid. The sex ratio is usually 1:1 and there is no parthenogenesis. In Brazil, Leite-Filho and Ramalho (1979) observed a three-day preoviposition period and a shorter adult life-span.

Damage (Figure 211) is caused by nymphs and adults feeding in phloem tissue which results in leaf curling and chlorosis, stunted growth, and severely reduced yields or complete crop loss. A toxin may be involved in plant damage but this has not been demonstrated. This species, unlike other species, does not transmit bean viruses. Damage is more severe when high populations occur at early crop-growth stages and flowering. Damage occurring after pod set does not have a significant effect on yields (van Schoonhoven et al., 1978a).

Leafhopper attack and damage is more severe during hot, dry weather and is aggravated by poor soil conditions or insufficient soil moisture. Planting date affects leafhopper populations and resulting damage. In El Salvador, Miranda (1967) obtained yields of 1182 kg/ha when common beans were planted on December 21 (end of wet season), but only 121 kg/ha when beans were planted on January 21 (middle of dry season). At the Centro Internacional de Agricultura Tropical (CIAT) in Colombia, very high populations develop during dry or semidry seasons.

Besides planting dates, various cultural practices reduce leafhopper populations and damage. Associated cropping affects leafhopper populations: smaller *E. kraemeri* populations were found on common beans planted in association with maize that was planted 15-20 days earlier. However, populations were larger when both crops were planted on the same date (CIAT, 1977; Hernández-Romero et al., 1984). Similar results were obtained by García et al. (1979) who evaluated the effect of a sugarcane-bean association. Nymphs per leaf and adults per meter row were 44% and 55% lower, respectively, in association (when beans were planted 45 days after sugarcane) than in monoculture.

Preliminary studies showed that leafhopper adult and nymphal populations decreased 43% and 70%, respectively, in bean plots which had nearly 100% weed cover (CIAT, 1976). Altieri et al. (1977) suggested that *E. kraemeri* populations were reduced, not by

increased parasite or predator activity, but by a possible chemical repellent effect of two weed species (*Leptochloa filiformis* (Lam.) Beauv. and *Eleusine indica* L.) Gaertn. The role of weed cover in reducing leafhopper infestations was further studied by van Schoonhoven et al. (1981). They found that mixtures of these grassy weeds effectively reduced nymphal and adult populations on leafhopper-susceptible and resistant cultivars. *Eleusine indica* was more competitive with the susceptible cultivars than *L. filiformis*. Both weeds competed with the resistant cultivars, preventing yield advantage. Similar results were obtained in United States by Andow (1983).

Mulching with aluminum foil and rice straw significantly reduced adult leafhopper colonization, possibly as a result of increased light reflection. Yields were greater compared to beans without mulches (Cardona et al., 1981; Wells et al., 1984). This method of control, however, has serious economical and practical limitations. Andrews et al. (1985) showed that plastic mulches can be economically viable in production of green beans, but advised against their use for common beans.

The egg parasite *Anagrus* sp. (Hymenoptera: Mymaridae) is the best known natural enemy of *E. kraemeri* in Latin America. This parasite has a functional response of two days to the presence of host eggs (CIAT, 1980). Although it parasitizes between 60% and 80% of leafhopper eggs under field conditions, it cannot keep leafhopper populations below economically damaging levels (Gómez-Laverde and van Schoonhoven, 1977). *Anagrus flaveolus* Waterhouse is present in Brazil (Pizzamiglio, 1979).

Other natural enemies include the trichogrammatid *Aphelinoidea plutella* (Girault) (Pizzamiglio, 1979), the mymarid egg parasite *Polynema* sp., and the dryinid *Agonatopus* sp. The parasitic fungi *Hirsutella guyana* and *Erynia radicans* (Brefeld) were found in Brazil (Ghaderi, 1984). *Erynia radicans* has also been observed infecting *E. kraemeri* during rainy periods in Colombia (van Schoonhoven et al., 1985) and Honduras (Caballero and Andrews, 1985).

Varietal resistance to *E. fabae* has been studied in the United States. McFarlane and Rieman (1943) classified several materials as

resistant and discussed the possibility of using them to suppress leafhopper populations. Wolfenbarger and Slesman (1961a and 1961b) later screened 1619 lines and found significant variability in plant damage and nymphal counts. A significant correlation between nymphal counts and damage scores was detected. Epidermal hairs did not correlate with nymphal populations, whereas plant height, resistance to bean common mosaic virus (BCMV), and seed color were related to various levels of resistance. Higher levels of resistance were detected more among *Phaseolus lunatus* L. and *Vigna radiata* materials than among *P. vulgaris* (Wolfenbarger and Slesman, 1961d). Chalfant (1965) found a 50% yield difference between protected and unprotected plots, regardless of their variability.

Resistance to *E. kraemeri* has been extensively studied at CIAT (Figure 212) by evaluating more than 18,000 bean accessions. Mass screenings are based solely on visual damage scores (leaf distortion and yellowing) that are recorded 25, 35, and 45 days after planting to avoid maturity and other late-season effects. Intermediate and resistant materials are rescreened in replicated nurseries in which a visual estimate of pod number per plant is also made. More indepth evaluations of bean accessions are made, calculating the yield difference between insecticide-protected and unprotected plots.

No high levels of resistance have been found in *P. vulgaris*. To date, 3%-4% of the 18,000 *P. vulgaris* accessions evaluated are classified as resistant. Most of these are small-seeded, black or cream-colored, indeterminate bush beans (Galwey, 1983). Black-seeded, late materials appear less susceptible than large-seeded red or white accessions. At high infestation levels, nymphal counts do not correlate with visual damage scores (Eskafi and van Schoonhoven, 1978; Murguido and Beltrán, 1983). Hooked trichomes are a major factor responsible for resistance of *P. vulgaris* to *E. fabae* (Pillemer and Tingey, 1976). As resistant mechanisms to *E. kraemeri* they are also important in *P. lunatus* (Lyman and Cardona, 1982), but not in *P. vulgaris* (CIAT, 1974).

Mechanisms of resistance to *E. kraemeri* have been the subject of several studies. Wilde and van Schoonhoven (1976) did not find antibiosis or definitive signs of nonpreference (antixenosis). Additional research suggested that tolerance was manifested by

reduced damage, expressed as less stunting, higher leaf area index, and more pods (CIAT, 1983). Additionally, in both free- and no-choice tests, ovipositional antixenosis was detected in the cultivars EMP 89, EMP 94, and EMP 97 (Kornegay, 1985; Kornegay et al., 1986). According to Kornegay and Temple (1986) an additive-dominance genetic model explained the inheritance of tolerance and antixenosis defense mechanisms.

Breeding for resistance to *E. kraemeri* has been complicated by the lack of adequate levels of resistance in *P. vulgaris*, lack of diversity in resistance responses, quantitative nature of inheritance (Galwey and Evans, 1982a), and strong interactions between genotype and environment (Galwey and Evans, 1982b; Kornegay et al., 1986; van Schoonhoven et al., 1985). Nevertheless, a recurrent selection program has successfully diversified mechanisms of resistance (Kornegay et al., 1986) and some lines have been consistently outstanding (van Schoonhoven et al., 1985). Some of the CIAT-developed EMP lines that yield well under high insect pressure have wide adaptation in various Latin American countries. For example, EMP 92 has been multiplied in Argentina for commercial production (Costilla, 1983) and EMP 84 is a potential new cultivar for Cuba.

In addition to *P. vulgaris*, resistance to *E. fabae* has been found among *P. lunatus*, *P. acutifolius* A. Gray, and *P. coccineus* materials (Wolfenbarger and Slesman, 1961d). When barriers to interspecific crossing are overcome, more rapid breeding progress may be possible (Galwey et al., 1985).

Chemical control of *E. kraemeri* is effective with monocrotophos, methamidophos, dimethoate, and granular carbofuran (CIAT, 1974 and 1976; Murguido, 1983). The economic injury level is two to three nymphs per leaf and is higher for resistant cultivars (CIAT, 1976 and 1983). In Central America, Andrews (1984) recommends that sprays be made when one adult per plant is found at the seedling stage. Two nymphs per leaf or two adults per plant are critical population sizes between the two-leaf stage and pod set. As many as three nymphs per leaf or three adults per plant can be tolerated during pod fill.

Whiteflies (Hemiptera-Homoptera: Aleyrodidae)

The sweetpotato or common whitefly, *Bemisia tabaci* (Genadius), is the most important aleyrodid affecting beans in Latin America. Other species are *B. tuberculata* Bandar, *Tetraleurodes acaciae* (Quaintance), *Trialeurodes abutiloneus* (Haldeman), and *Trialeurodes vaporariorum* (Westwood). These species have other leguminous and nonleguminous host plants (Russell, 1975). Common names for whiteflies in Latin America are "mosca blanca" and "mosca branca."

Bemisia tabaci is a vector of such important bean viruses as bean golden mosaic and bean chlorotic mottle (Gámez, 1971). Direct feeding does not damage bean plants and the insect becomes important only in areas where virus transmission occurs such as Central America, parts of Mexico, the Caribbean, Brazil, and Argentina (Blanco-Sánchez and Bencomo-Pérez, 1981; Cárdenas-Alonso, 1982; Costa, 1965; Gámez, 1971).

The systematics of the group has been complicated by the occurrence of host-correlated variation (Mound, 1963). Immature stages of *B. tabaci* occur in a variety of morphological forms associated with definitive types of host leaves. Races also occur (Bird and Maramorosch, 1978). This is important, especially when breeding plants for resistance to whiteflies.

Russell (1975) summarized the biology of *B. tabaci*: females lay 25-32 eggs singly or in groups on the undersurface of bean leaves where the egg pedicel is inserted into the epidermis. The immature stages (Figures 213 and 214) also occur on the undersurface of leaves. The egg to adult (Figure 215) cycle is completed in about three weeks and is similar on cotton seedlings (Butler et al., 1983).

In Brazil and other countries, soybeans act as a transitional host for whitefly infestations which then move in large numbers to beans (Costa, 1975). Wide planting periods favor population buildup and breeding of successive generations. Alonzo (1975) reported a significant effect of late planting dates on whitefly infestations in Guatemala.

Resistance to BGMV is an economic method of control, particularly as little is known about resistance mechanisms of bean

cultivars to *B. tabaci*. Hohmann and de Carvalho (1982) found that *B. tabaci* preferred Porriño Sintético but did not report resistance in four cultivars tested. Studies in Guatemala demonstrated that the resistant cultivar ICTA Jutiapan, without chemical protection against the vector, outyielded the protected susceptible check, Rabia de Gato (Aldana-De León et al., 1981). In Mexico, line D 145, without protection, outyielded the protected susceptible cultivars Jamapa and Criollo Regional (Rodríguez-Rodríguez, 1983).

Chemical control is possible with foliar applications of methamidophos 15 and 30 days after planting or applying, before planting, granular phorate or carbofuran (Mancía et al., 1973). Aldicarb also provides good protection (de Bortoli and Giacomini, 1981). Triazophos and mephosfolan were not effective in Brazil (Hohmann, 1982).

Aphids (Homoptera: Aphidae)

Several aphid species attack common beans. Their direct damage is not important but their ability to transmit bean common mosaic virus makes them important economic pests. Common names in Latin America include “áfidos,” “pulgonos,” “afidios,” and “pulgão do feijoeiro.” Species common on beans are *Aphis gossypii* Glover, *A. craccivora* Koch, *A. spiraeicola* Patch, *A. fabae* Scopoli, *Tetraneura nigriabdominalis* (Sasaki), *Myzus persicae* (Sulzer), and *Brevicoryne brassicae* (L.) (Bécquer-Hernández and Ferrándiz-Puga, 1981; Costa and Rossetto, 1972; Zaumeyer and Thomas, 1957).

High aphid mortality occurs when aphids are captured by hooked hairs on bean leaves (McKinney, 1938). Control of bean common mosaic has been achieved by incorporating resistance genes so that chemical control of aphids is not needed.

Thrips (Thysanoptera: Thripidae)

Thrips are pests of beans in several Latin American countries, but their attacks are usually of little economic importance. *Frankliniella* sp., *Sericothrips* sp., and *Caliothrips brasiliensis* (Morgan) have occurred in Brazil (Rossetto et al., 1974) and Colombia (Posada-O.

et al., 1970). In Colombia, *C. braziliensis* is the most abundant species. *Caliothrips fasciatus* (Pergande), *C. phaseola* (Hood), *Frankliniella insularis* (Franklin), and *F. williamsi* (Hood) are pests of beans in Central America (King and Saunders, 1984). Common names in Latin America include "trips" and "bicho candela."

Females insert their eggs into leaves, petioles, and stems. In laboratory studies at CIAT, eggs of *C. braziliensis* hatched in five to six days. First-instar larvae developed in one to two days and the second lasted four to five days. Pupation occurred in the soil and debris and lasted two to three days. Longevity and fecundity of adults were not studied.

Larvae and adults feed on the undersurface of cotyledonary leaves. In older plants they can also be found feeding on leaves, flowers, and petioles. When populations are high, thrips cause leaf cupping and reduction in the size and development of young plants (Figure 216). In general, they seldom become an economic pest. Most attacks occur in field borders and usually during hot, dry weather.

Chemical control of thrips is rarely needed. Adults and nymphs of *Orius tristicolor* (White) prey on *Sericothrips* sp. and *C. braziliensis*.

Stink bugs (Hemiptera: Pentatomidae)

Several species of pentatomids have occurred as pests of beans in Latin America. *Acrosternum marginatum* (Palisot de Beauvois), the green bean stink bug, is found in Central America, Mexico, the Caribbean (King and Saunders, 1984), and Colombia. The cosmopolitan and polyphagous bugs *Nezara viridula* (L.) and *Piezodorus guildinii* (Westwood) are not economically important in common beans (Costa et al., 1980 and 1981). Other pentatomids recorded on beans in Latin America are *Edessa rufomarginata* De Geer, *Euschistus bifibulus* (Palisot de Beauvois), *Padaeus trivittatus* Stål, and *Thyanta perditor* (F.). None of these are economically important (King and Saunders, 1984). Common names for these insects are "chinchés," "chinchés apestosos," and "chinchés hediondos."

The biology of *A. marginatum* was studied by Hallman et al. (1985 and 1986). The total cycle from egg to adult takes 42 days. There are five nymphal instars. The first-instar nymphs are foliar feeders, while later nymphs are pod feeders. After a 10-day preoviposition period, females lay an average of 96 eggs in masses of 3-28 eggs (average 13). The insect (Figure 217) is not commonly found in commercial fields but sometimes appear in large populations when it becomes economically important. Hallman (1985) estimated that significant yield losses occurs at infestation levels of one late-instar nymph/0.6 m² of beans.

Telenomus sp. (Hymenoptera: Scelionidae) is an important egg parasite of pentatomids in Brazil (Link et al., 1980). No other control measures are reported.

Pod-attacking Insects

Bean-pod weevil (Coleoptera: Curculionidae)

The bean-pod weevil, *Apion godmani* Wagner, is an important insect pest of common beans in Mexico and parts of Central America (Salguero, 1983a; Sifuentes-A., 1981). *Apion aurichalceum* Wagner is also important in the highlands of Mexico (McKelvey et al., 1951) and Guatemala (Salguero, 1983a). In Central America, *A. godmani* occurs in Guatemala, El Salvador, Honduras, and northern Nicaragua. It does not occur in coastal areas and is more serious at higher altitudes. Reports on the presence of this insect in Colombia have not been confirmed. Other less important species of *Apion* on beans are listed by McKelvey et al. (1947) and Mancía (1972). Host plants for *A. godmani* include *Dalea*, *Desmodium*, *Rhynchosia*, and *Tephrosia* species (McKelvey et al., 1947). Common names for these insects are "apion," "picudo de la vaina," and "picudo del ejote."

The economic importance of *A. godmani* varies. In Mexico, Sifuentes-A. (1981) estimated 50% yield losses while Guevara-Calderón (1961) reported as much as 80% damage. Salguero (1983b) found 17% average damage in the central-western plateau of Guatemala and 9%-60% damage in the southeastern plateau.

Mancía et al. (1972) observed as much as 94% bean loss in El Salvador, especially during the rainy season. In germplasm screening nurseries in Honduras, seed damage has ranged from 1% in resistant to 80%-85% in susceptible materials. *Apion aurichalceum* is less important, possibly as a result of its ovipositional behavior: the female lays about 35 eggs only in the distal portion of a pod, and the remaining seeds of the pod therefore escape attack (McKelvey et al., 1951).

The adult bean-pod weevil (Figure 218) is black and about 3 mm long. During the wet season, two generations may form, with a possible third generation occurring during the dry season. Survival sites could not be located in Mexico (McKelvey et al., 1951) or in Guatemala (Salguero, 1983b).

In the laboratory (21 °C and 75% r.h.), Mancía (1972) found that the egg stage lasted five days, the three larval instars six days, while the prepupal and pupal stages lasted two and nine days, respectively. Adults sometimes remained in the pupal chamber for three or four days but usually emerged immediately after pupation. Adults lived from 10 days to nearly a year, and mated several times. A maximum of 392 eggs per female were recorded (Mancía, 1972). The pre-oviposition period lasted 10 days.

McKelvey et al. (1951) reported a longer larval period of three weeks and four larval instars. The egg-to-adult period in Mexico lasted 6-8 weeks and adults lived an average of three months. A shorter egg-to-adult cycle of 28-30 days was calculated by Salguero (1983b) in Guatemala. The insect has not been observed during the dry season.

Adults usually appear before flowering and cause light feeding damage to leaves, pods, and flowers which is not economically important. Oviposition takes place on newly formed pods during the daytime. The female adult chews a small hole in the mesocarps of one- to four-cm-long pods, usually above the developing seed, and deposits a white, semitranslucent egg. These spots are visible as white hyperplastic deformations (Figure 219) (McKelvey et al., 1947 and 1951). Those young pods which are attacked may abort (Enkerlin-S., 1951).

Second-instar larvae bore into the mesocarp of the pod wall and feed on developing seeds (Figure 220), leaving the hilum intact. *Apion* damage is somewhat similar to that of *Asphondylia* sp., a cecidomyiid common in El Salvador and Honduras (Espinoza-R., 1985). One larvae per seed is normal, but three to five per seed have been found during heavy infestations with a maximum of seven per seed and 28 per pod (Mancía, 1972; McKelvey et al., 1947). Larvae do not feed on mature seed.

Triaspis sp., a braconid larval parasite was recorded by McKelvey et al. (1951) in Mexico and by Mancía (1972) in El Salvador. The fungus *Metarrhizium* sp. was observed attacking *Apion* adults in Guatemala (Salguero, 1983a). The efficiency of these natural enemies has not been evaluated. Bean-maize crop associations reduce *Apion* populations (Martínez-Rodríguez, 1978).

Host-plant resistance to *A. godmani* has been studied by several authors. McKelvey et al. (1951) identified bean accessions Puebla 2 and 32, and Hidalgo 6 and 24 as resistant. Guevara-Calderón (1961) identified lines derived from Hidalgo and Puebla 32 as most resistant, together with cultivars Amarillo 155 and Amarillo 156. Other Mexican resistant cultivars were selected by Ramírez-Genel et al. (1959), Guevara-Calderón et al. (1960), Guevara-Calderón (1969), and Medina-Martínez and Guerra-Sobrevilla (1973). From these studies and the intensive screening conducted in El Salvador by Mancía (1973c) and in Guatemala by Yoshii (1978), high levels of resistance (expressed as percentage of seed damage) were detected in accessions Mexico 1290, Amarillo 154, Negro 150, Puebla 152, Línea 12 Salvador, and Línea 17 Salvador.

These and other sources of resistance were used in a breeding project which identified highly resistant lines with less than 10% of pods damaged and less than 2% of seeds damaged (CIAT, 1983). Resistant lines with better adaptation to Mexican and Central American conditions have since been used in crosses to recover resistance through transgressive segregation (Beebe, 1983). Some of these parents were APN 18, APN 92, APN 64, Línea 17 (derived from Mexico 1290), and BAT 340. Simultaneously, new parents of Mexican origin were identified. Some of these are Aguas Calientes 40, Puebla 22, Puebla 36, Puebla 36-1, Puebla 49, Puebla 416, Amarillo 169, Hidalgo 46-A, and Veracruz 155. A good correlation

between percentage of pods damaged and percentage of seeds damaged has been obtained. A sequential sampling plan for resistance nurseries has been proposed by Hallman (1983).

Chemical control of *A. godmani* is still important. Monocrotophos, methamidophos, methomyl, methyl parathion, and carbaryl are effective (Mancía et al., 1972). Carbofuran is effective at a high dosage of 2.5 kg (Mancía, 1973a), but not at 1.5 kg a.i./ha (Salguero, 1983a). Sprays are more effective when made six days after flower initiation and again seven days later (Mancía et al., 1974). A tentative economic threshold of 4-6 adults/40 m of row was established by Salguero (1983b). This economic threshold appears too low and further field testing is needed.

Lepidopterous Pod Borers

Corn earworm and tobacco budworm (Lepidoptera: Noctuidae)

Damage by the *Heliothis* complex, *H. zea* (Boddie) and *H. virescens* (F.) (Figure 221), is sporadic but can be severe. Common Latin American names include "heliothis," "bellotero," "elotero," "ejotero," and "yojota."

Females oviposit on leaves. The larvae (Figure 222) undergo six larval instars during 18-30 days. Larvae attack pods, and feed on seeds after perforating the pod wall above the seeds. Pupation occurs in the soil.

At high population levels, attacks can be devastating (Turner, 1979). Several seeds per pod may be destroyed and secondary rotting may destroy any remaining seeds. Because of the sporadic nature of attacks, the *Heliothis* complex has not been well studied in beans. *Heliothis virescens* seems to be more abundant than *H. zea*.

High levels of parasitism occur. Posada-O. and García (1976) listed 26 different parasite or predator species of *Heliothis* in Colombia. As much as 89% larval parasitism has been recorded at CIAT. The egg parasites *Trichogramma* spp., the tachinid larval parasites *Eucelatoria* sp., and *Archytas piliventris* Wulp are common. Others include the braconid larval parasites *Bracon hebetor*

Say, *Chelonus antillarum* Marsh, *C. insularis* Cress., and *Apanteles marginiventris* (Cress.) (King and Saunders, 1984). *Orius* sp. and *Geocoris punctipes* (Say) are predators of eggs and first-instar larvae.

Chemical control of older larvae is difficult. Pyrethroids are widely recommended. The nuclear polyhedrosis virus (Elcar) was tested on beans in Australia (Rogers et al., 1983) and compared favorably with fenvalerate.

Epinotia pod borer (Lepidoptera: Olethreutidae)

Epinotia aporema (Walsm.) is widely distributed throughout Latin America. It is an important insect pest in Peru (Wille-T., 1943) and Chile (Brücher-E., 1941). The insect has also attacked faba beans, chickpeas, soybeans, alfalfa, and lentils (Alomia, 1974; Wille-T., 1943). Common names frequently used for this species in Latin America include "polilla del frijol," "epinotia," "polilla del brote," and "barrenador de la vaina."

Females lay an average of 100 eggs in four to eight masses during one to two weeks. The egg stage lasts four to seven days in Peru (Wille-T., 1943), Chile (Ripa-Schaul, 1981), and Colombia (Alomia, 1974). There are five larval instars which together are completed in 14-22 days. Pupation occurs in a cocoon on leaves or the ground (Wille-T., 1943) during 14-16 days. Adults live 15-22 days and are active at night.

Larvae damage beans by feeding on or in terminal buds, stems, and pods. Larvae weave their excrements together and push them out of the feeding canals. The insect may also cause flower damage and abortion. Stems and buds can be deformed (Figure 223) and pod damage can result in rotting by secondary organisms (Alomia, 1974).

The egg parasite *Trichogramma* sp. has been recorded in Chile (Ripa-Schaul, 1981). Wille-T. (1943) observed a tachinid larval parasite, *Eucelatoria* sp., in Peru. Some work on resistance to *E. aporema* has been done in Peru (Avalos-Q., 1982). In a screening of 968 bean materials, five had significantly lower levels of damaged stems and seeds than the local commercial cultivar. Adequate

chemical control is available with aminocarb, parathion, and omethoate (Torres-B., 1968). Fenvalerate or carbaryl applied 30 days after planting are also effective (Avalos-Q., 1977). Fenvalerate has a 15-day residual effect.

Lima bean pod borer (Lepidoptera: Pyralidae)

The lima bean pod borer (*Etiella zinckenella* (Treitschke)) has occurred in the United States (Stone, 1965), Puerto Rico (Scott, 1940), Mexico, parts of Central America and the Caribbean (King and Saunders, 1984), and Brazil (Ramalho et al., 1978). Little is known about the economic importance of this species in Latin America. According to King and Saunders (1984), it is more important in the Caribbean than in Central America. Attacks are sporadic and only occasionally does the insect become a serious pest. Common names for this insect in Latin America are "barrenador del ejote," "polilla de las vainas," and "medidor de las vainas."

Eggs are laid on flowers or pods. Larvae are yellow, green, or pinkish with red-brown dorsal lines. It can feed on flowers or the exterior of pods, but prefers to act as a pod borer, feeding on developing seeds. Pupation can take place inside pods or the ground. Damaged flowers and small pods can abort (Stone, 1965). *Etiella zinckenella* leaves almost no outside evidence of its presence in pods, while maruca pod borer, *M. testulalis* (Geyer), keeps exit holes open in the sides of infested pods. Larvae force feces and other waste material outside through these holes.

Chemical control of the lima bean pod borer is difficult and is best directed against small larvae before they perforate pods (King and Saunders, 1984). Some work on the resistance of bean cultivars to this insect has been carried out in Brazil by Ramalho et al. (1978) who observed variability in percentage of infested pods and seed damage.

Maruca (Lepidoptera: Pyralidae)

Maruca testulalis Geyer is an important pest of legumes in Africa and Asia (Singh and van Emden, 1979; Taylor, 1978), but is not

usually an important pest of common beans in Latin America (King and Saunders, 1984). Occasional attacks, though, can be serious. *Maruca testulalis* has occurred in Brazil (Ruppel and Idrobo, 1962), Colombia (Posada-O. et al., 1970), the Caribbean (Leonard and Mills, 1931), and Central America (King and Saunders, 1984). Common names include "maruca," "barrenador de la vaina," and "perforador de la vaina."

Like most pod borers, *M. testulalis* oviposits near or on flower buds, flowers, young leaves, and pods. There are five larval instars which together last 8-13 days (Broadley, 1977). Larvae have four black or dark gray spots on each segment (Figure 224). Larvae penetrate the pod, feed on developing seeds, and expel frass and feces. Some damage to leaves and flowers occurs before pod feeding (Scott, 1940). Pupation occurs in a cocoon woven between two pods in debris on the soil or in the soil itself.

According to King and Saunders (1984) chemical sprays may be justified when one damaged pod per two plants is found.

Storage Insects

Bruchids (Coleoptera: Bruchidae)

van Schoonhoven (1976) has listed 28 insect species occurring on stored beans. However, most are of minor importance or only accidentally found on beans. By far the most important pests of stored beans in Latin America are the Mexican bean weevil, *Zabrotes subfasciatus* (Boheman) (Figure 225) and the bean weevil, *Acanthoscelides obtectus* (Say) (Figure 226). Both are cosmopolitan (Chapter 21, this volume). Literature on the economic importance of bruchids is scarce. McGuire and Crandall (1967) estimated 35% of losses occurred during storage (Figure 227) in Mexico and Central America but did not specify if these losses resulted from insects or other factors. In Brazil, 13% losses have been estimated. van Schoonhoven (1976) calculated that in Colombia 7.4% losses were caused by bruchids. Damage was not higher because storage periods were short, averaging 44 days. Common names for these insects are "gorgojos," "gorgojo pintado," or "gorgulho de feijão"

(*Z. subfasciatus*); and “gorgojo común” or “caruncho” (*A. obtectus*).

The main difference between these bruchids is in their oviposition behavior. *Zabrotes subfasciatus* attaches the egg to the seed (Figure 225). After hatching, the young larvae bore through their egg shell and the seed coat in one process (Howe and Currie, 1964). *Zabrotes subfasciatus* does not attack in the field. In contrast, *A. obtectus* females do not glue eggs to the testa but scatter them among stored seeds or infest beans in the field by ovipositing on growing pods. The newly hatched larvae will later penetrate the seed.

Another important difference lies in their ecological adaptation. *Zabrotes subfasciatus* is a tropical species and is found predominantly in warmer areas. *Acanthoscelides obtectus* occurs at higher latitudes and altitudes, in subtropical regions, or in the cooler environment of the highlands of tropical America. In a study in Nicaragua (Peter H. Giles, personal communication), beans were infested initially with *A. obtectus* (99.7%) and *Z. subfasciatus* (0.3%) at different elevations above sea level. After 16 weeks, the percent ratios became 0:100 at 56 m; 5:95 at 450 m; and 27:73 at 680 m. Temperatures decreased as elevation increased. These data suggest that *A. obtectus* becomes a stronger competitor at lower temperatures.

In storage, the life history of *Z. subfasciatus* and *A. obtectus* is similar (Howe and Currie, 1964). Larvae of both species molt four times before pupating. During the last larval instar, the feeding and pupation cell (Figure 228) 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 or biting out the window with its mandibles. Adults normally do not eat but may consume water or nectar. Adults are short lived, and mate and oviposit soon after emergence.

Zabrotes subfasciatus adults exhibit strong sexual dimorphism. Females are large and have four characteristic cream-colored spots on the elytra. The male is entirely brown. At 28 °C and 75%-80% r.h., females lay an average of 36 eggs and live 13 days. The egg stage lasts five to six days, larval development takes 14 days, and the pupal stage takes six to seven days. Usually the sex ratio is 1:1.

At 26 °C and 75%-80% r.h., females of *A. obtectus* live 14 days and lay an average of 45 eggs. Eggs hatch in six to seven days and the larval-pupal development takes 23 days. Sex ratios tend to be 1:1. Mortality during development occurs mainly as larvae penetrate the seed or when the exit hole is not large enough for adult emergence.

Farmers have used various traditional methods to control bruchids. Among these are mixtures of grain with inert materials such as sand, crystalline silica, bentonite, and magnesium carbonate which effectively kill weevils. Ashes from fireplaces are also used as an effective physical barrier to adults (CIAT, 1975). Black pepper has been successfully used to control *A. obtectus* (Lathrop and Keirstead, 1946).

Storing beans in undamaged pods can reduce losses from *Z. subfasciatus*. Eggs deposited on pod walls hatch but larvae die inside the pods without penetrating seed. This method cannot, however, be used to control *A. obtectus* since this insect can attack beans in the pods. Labeyrie (1957) showed that storing beans unshelled or delaying the harvest considerably increases *A. obtectus* attack. This occurs because *A. obtectus* prefers to oviposit on mature pods (Labeyrie and Maison, 1954; Menten and Menten, 1984).

Vegetable oils are also effective against bruchids. van Schoonhoven (1978) found that cotton, peanut, soybean, and maize oils were equally efficient when applied at the rate of 5-10 ml per kg seed. Treated seed retained its germination ability (CIAT, 1977), while the oils caused adult mortality, reduced oviposition, and killed eggs. Unrefined oils can also be used (Hill and van Schoonhoven, 1981).

Chemical control of weevils is readily obtained with a variety of products such as malathion, pyrethrins, pirimiphos-methyl, and fenitrothion (CIAT, 1975; Salas and Ruppel, 1959). The pyrethroids, deltamethrin and permethrin, have also given excellent control. Some fungicides also protect seed (van Schoonhoven and van Dam, 1982). For large volumes of seed, the fumigants aluminum phosphide and methyl bromide are widely used in Latin America (van Schoonhoven, 1976).

Recent work on resistance to bruchids has been conducted at CIAT, Colombia, on a continuous basis (Menten and Menten,

1984; Oliveira et al., 1979; Ramalho et al., 1977). After screening more than 4000 cultivated bean accessions for resistance to *Z. subfasciatus*, van Schoonhoven and Cardona (1982) concluded that resistance levels were too low to be of economic value. Similarly, no satisfactory levels of resistance were identified when more than 10,000 genotypes were tested with *A. obtectus*.

However, very high levels of resistance to both bruchids were found in noncultivated, small-seeded wild forms of *P. vulgaris* of Mexican origin (CIAT, 1984a; van Schoonhoven et al., 1983). Resistance is expressed as reduced oviposition, longer larval development times, and reduced progeny weight. Antibiosis is the resistance mechanism. According to Osborn et al. (1986), the protein, arcelin, could be the factor responsible for resistance. Variants of this protein are present in accessions with the highest resistance levels: G 12866 (arcelin 2); G 12891, G 12895, and G 12942 (arcelin 3); and G 12949, G 12952, and G 12953 (arcelin 4).

Work is underway to genetically transfer the different arcelin types into cultivated beans and to determine the effect of arcelin on bruchid resistance and human nutrition (CIAT, 1988; Osborn et al., 1986). Evaluation of resistance sources and progenies for resistance to *A. obtectus* under field conditions is also in progress.

Other Pests

Snails and slugs

Snails are a minor pest in Africa and seldom cause damage to beans in Latin America.

Slugs (Figure 229), however, have become important pests of common beans in some parts of Central America (Andrews and Dundee, 1986). Slugs have also been reported as minor pests in Africa (Chapter 21, this volume), the Caribbean (King and Saunders, 1984) and certain areas in South America (CIAT, unpublished surveys). Common names for slugs in Latin America include "babosas," "lesmas," "ligosas," "sanguijuelas," "lipes," and "chimilias."

The veronicellid which has been identified as *Sarasinula plebeia* (Fisher) (syn. *Vaginulus plebeius* (Fisher)) is the most important species (Andrews, 1983a). It was reported for the first time from Central America in El Salvador in 1967 by Mancía (1973b). According to Andrews and Dundee (1986), this species was accidentally introduced into El Salvador. It has superimposed its range of distribution on that of native veronicellids such as *Diplosolenodes occidentalis* (Guilding) and *D. olivaceus* (Stearns). Other species reported in Central America are *Leidyula* (syn. *Veronicella*) *moreleti* (Crosse and Fisher) and *Leidyula floridana* (Binney). It is not known whether *D. occidentalis* (syn. *Vaginulus occidentalis*) and *D. olivaceus* are separate species or simply ecotypes.

By 1976 *S. plebeia* was a serious pest of beans in El Salvador, Nicaragua, and Honduras. It was first reported in Guatemala and Costa Rica in 1971 and 1981, respectively. It is not known to occur in Panama, but attacks cassava in Colombia. *Sarasinula plebeia* is a minor pest of beans in Guatemala where it borders El Salvador and Honduras (Salguero, 1981). It is not clear whether this species occurs in Mexico. Andrews and Dundee (1986) report that damage by *S. plebeia* occurred in Chiapas, Veracruz, and Yucatán. However, the Mexican Quarantine Service (Dirección General de Sanidad Vegetal de México, 1982) lists *Leidyula* (syn. *Veronicella*) *moreleti* as the responsible species.

According to Andrews (1983a), crops of 500,000 Central American farmers are affected by this pest every year. The slug problem is more serious in Honduras and Nicaragua than elsewhere. In certain years, as much as 53% of the area planted with beans can be affected (Secretaría de Recursos Naturales de Honduras, 1981).

Slugs are hermaphroditic and self-fertilization in *S. plebeia* is common. Females lay as many as 80 eggs in masses under plant debris or in soil cracks. Eggs are oval, translucent, and hatch in 20-24 days at 27 °C. Under dry conditions, eggs may take six months to hatch. Young slugs resemble adults and reach maturity in two to five months (Mancía, 1973b). Slugs live 12-18 months and reach five to seven cm in length. According to Andrews and Lema (1986), one generation takes eight weeks and there may be two generations per year in Honduras. Slugs are inactive during dry periods. Higher slug

densities occur near streams, in heavy clay soils, and in weedy fields. Most damage occurs along the borders of fields and progresses inwards, especially if vegetation and debris provide ample protection for slugs during the day.

Young slug damage is apparent when whole leaves, except for veins, are consumed (Figure 230). Older slugs consume entire leaves. Entire seedlings may also be consumed, and pod damage can occur. Andrews and Huezo de Mira (1983) calculated that each active slug/m² can cause, in one night, a plant stand reduction of 20% and yield reduction of 16%. Andrews and Huezo de Mira (1983) used simple, inexpensive, pitfall traps that were baited with a mixture of bean, molasses, beer, and carbaryl (Andrews, 1983b). They determined that each captured slug represented a reduction of plant stands by 14% and yield by 11%. The authors established an economic injury level of 0.25 active slugs/m² or 0.4 slugs per trap each night. Honduran work has raised the levels to 1 slug/m² or 1 slug per trap each night (Andrews and Barletta, 1985).

At high population levels, slugs can become a health problem. They act as vectors of the nematode *Angiostrongylus costaricensis* Morera and Cespedes which is pathogenic to man, especially children (Morera, 1973).

Slugs show marked preferences for certain weeds and crops (Ramírez et al., 1985) and are repelled by several plant species. Extracts of *Canavalia* sp. and other plants may reduce slug damage (Coto-Alfaro and Saunders, 1985). Protozoans, brachylaemid flatworms, lungworms, lampyrid beetles, and sciomyzid flies have been reported as natural enemies of slugs (Stephenson and Knutson, 1966). A review and a proposal for biological control of slugs in Central America were recently prepared by Bennett and Andrews (1985).

Control of slugs is most effectively achieved by ridding fields and field borders of weeds and plant debris. Burning crop residues, land preparation, and drainage of fields are recommended (Mancía, 1973b). Chemical control is obtained with baits prepared with carbaryl, methiocarb, phorate, aldicarb, thiocarbonyl, or metaldehyde (Crowell, 1977). Metaldehyde is widely recommended (Mancía, 1973b; Navarro, 1980). Residual effects of this product are

short term, especially under wet conditions. Foliar sprays of common insecticides do not work (Wheeler and Peairs, 1980). Granular insecticides applied to the soil are also less efficient than baits (Durón-Andino et al., 1981).

Spider Mites

Tropical spider mites (Acarina: Tarsonemidae)

The tropical spider mite, *Polyphagotarsonemus latus* (Banks), causes postflowering foliar damage to beans, especially during humid and warm weather. It also attacks potato, tomato, cotton, pepper, and many weeds (Cromroy, 1958; Doreste, 1968). It is not a serious pest of beans but occasionally can become economically important (CIAT, 1975). According to van Schoonhoven et al. (1978b), the tropical mite occurs in Florida, the Caribbean, Central America, and parts of South America, and is a pest in Brazil (Costa, 1970) and in parts of Colombia. It also occurs in Africa (Chapter 21, this volume). Common names in Latin America include "ácaro blanco," "ácaro tropical," and "ácaro branco."

The tropical mite is small and green, and has a short life cycle which passes through the stages of egg, larva, pseudopupa, and adult. In Brazil, the developmental stages together last six to seven days (Flechtmann, 1972). van Schoonhoven et al. (1978b) found a shorter life cycle in Colombia where the duration of egg, larva, and pseudopupa stages was two, one, and one day, respectively. Males lived for 12 days, while females lived 15 days and laid an average of 48 eggs.

Mite-damaged leaf edges roll upwards and have a shiny appearance (Figure 231). Lower leaf surfaces may turn purple. Young leaves may turn yellow to gold and be stunted. Pods can also be attacked, becoming covered with brown wound tissue (Figure 232). Symptoms can be confused with those induced by virus, mineral deficiencies, sunscald, or pollutants (Chapter 24, this volume).

Chemical control is possible with sulfur, endosulfan, dicofol, and omethoate (van Schoonhoven et al., 1978b). Dimethoate apparently stimulates *P. latus* populations (Harris, 1969).

Spider mites (Acarina: Tetranychidae)

Several species of spider mites attack beans. *Tetranychus desertorum* Banks is common in South America where it has a wide host range (Nickel, 1960). Other species reported are *T. telarius* L. (syn. *T. cinnabarinus* Boisd.), *T. urticae* Koch, *T. ludeni* (Zacher), *Eotetranychus lewisi* (McGregor), *Oligonychus stickneyi* (McGregor), and *O. yothersi* (McGregor) (Andrews and Poe, 1980; King and Saunders, 1984). Mites are called "ácaros," "arañas rojas," and "arañitas" in Latin America.

Spider mites usually attack beans (Figure 233) near physiological maturity and are not regarded as important pests of the crop. Studies on the biology of *T. desertorum* were made by Nickel (1960) and Piedrahita-C. (1974). The resistance of bean cultivars to spider mites was studied at CIAT. Some variability was detected but the levels of resistance were not high enough to provide economic benefits (Jara et al., 1981). In Latin America, chemical control recommendations for spider mites on beans include sprays with omethoate or tetradifon (González-A., 1969).

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Chapter 23

NUTRITIONAL DISORDERS

Carlos A. Flor and Michael T. Thung*

Introduction

In Latin America beans are grown in many different types of soils. The low levels of fertility in some soils can significantly reduce bean yield. This demanding crop has specific nutritional needs that not only require soils which are rich in essential nutrients, but also has good physical properties. Elements such as aluminum and sodium, are undesirable and even in small quantities are toxic. Beans absorb nutrients in the following order: $N > K > Ca > S > Mg > P$ (Howeler, 1980; Howeler and Medina, 1978).

In Central America and western South America, beans are usually grown in mountainous areas where Andosols predominate. The low fertility of these regions is caused primarily by deficiencies in phosphorus, nitrogen, and several micronutrients (Fassbender, 1967; Howeler and Medina, 1978). Studies in Colombia show that the application of phosphorus to the majority of soils in temperate and cold Andean bean-growing areas produces favorable responses in bean crops (Flor, 1985b).

In Costa Rica, soils differ considerably to each other in their physiochemical characteristics. However, beans suffer from aluminum and manganese toxicity and from deficiencies of phosphorus and nitrogen (Corella, 1983).

In Brazil, except for the northeast, most beans are planted in soils that support the type of vegetation known as "cerrados." The soils are predominantly Oxisols, Entisols, Inceptisols, and a few Ultisols. In general, these soils have low fertility and are characterized by

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phosphorus deficiency, aluminum toxicity, low cation exchange capacity (CEC), poor moisture retention, and, occasionally, manganese toxicity (Table 1).

The bean-growing regions of Argentina are confined to the northwest provinces: Salta, Santiago del Estero, Tucumán, and Jujuy. These areas are on the same latitudes as some important bean-growing areas of Brazil (Paraná, Santa Catarina, and Rio Grande do Sul). Yet, there is a large difference in soil fertility between the two countries. Northwestern Argentina is characterized by fertile alluvial soils that have physical problems such as erosion and compaction (Table 2).

Chile's bean-producing regions are characterized by soils with medium to high fertility levels.

In general, beans in Latin America are grown in moderately acid to neutral soils, except for those areas of Peru, Dominican Republic, Cuba, and Mexico which have saline soils.

In United States, beans are grown principally in the states of Michigan, California, Idaho, Nebraska, Colorado, Wyoming, North Dakota, and New York. Many soil problems in these areas are mechanical rather than chemical because of, for example, the excessive use of heavy agricultural machinery which compacts the soil. The chemical limitations that have been reported are: manganese deficiency in some areas of Michigan State (Voth and Christenson, 1980), zinc and iron deficiencies in soils with high pH, high base saturation, and the presence of free calcium carbonate (Mahler et al., 1981 and 1983; Overcoming zinc shortage in pintos, 1969; Vose, 1982).

Very little information exists on fertility problems or responses in common bean-producing regions of Africa and West Asia. Few fields receive *Rhizobium* inoculum or chemical fertilizers, even though preliminary research suggests that beans respond to phosphorus and, certainly, to nitrogen amendments. Limited information is available for Kenya, Malawi, Rwanda, Tanzania, Uganda, Zambia (CIAT, 1981), Sudan, Jordan, and Ethiopia (CIAT, 1985). Obviously, research is needed to investigate the types and severities of soil problems which exist in these regions and to develop strategies to manage them while improving bean productivity.

Table 1. General soil characteristics of bean-producing areas in Brazil.

| Parameter | pH | P N. Carolina (ppm) | K | Ca + Mg | Cation exchange capacity | Al | Aluminum saturation | Organic matter |
|-----------|---------|---------------------------|-----------|---------|--------------------------------|-----------|------------------------|-------------------|
| | | | | | (meq/ 100 ml soil) | | (%) | |
| Range | 4.3-6.2 | 0.1-16.5 | 0.02-0.61 | | 0.35-8.10 | 0.08-2.40 | 1.1-89.4 | 0.7-6.0 |
| Average | 5.0 | 2.0 | 0.15 | 0.34 | 1.10 | 0.56 | 59.0 | 2.2 |

SOURCE: Lopes and Cox, 1977.

Table 2. General soil characteristics of bean-producing areas of Argentina.

| pH | P Bray II (ppm) | K | Ca | Mg | Al | Cation exchange capacity | Organic matter (%) |
|----------------------|-----------------------|---------|---------|---------|-----|--------------------------------|--------------------------|
| (meq/ 100 g of soil) | | | | | | | |
| 6.5-7.8 | 52.0 | 0.4-1.5 | 4.0-6.0 | 0.4-1.5 | 0.0 | 10.0 | 0.1-4.0 |

SOURCE: M. T. Thung (personal communication, 1985).

Important Diagnostic Characteristics of Nutritional Disorders of Beans

Diagnostic testing for nutritional disorders in beans can be categorized into three types: visual classification of symptoms, analysis of soils and tissues, and experimental trials.

Frequently, diagnoses emphasize deficiencies. A complete diagnosis considers simple and complex deficiencies, toxicities, and combinations of deficiencies and toxicities. It is easy to confuse the symptoms of some deficiencies with those of some viral diseases (Flor, 1985a; Menten et al., 1981). For example, Figure 234 illustrates the type of morphological abnormalities that can occur in a bean seedling because of boron deficiency. Yet, the symptoms are similar to those induced by bean dwarf mosaic virus (Figure 134, Chapter 17) or herbicide damage.

An understanding of the general morphology, anatomy, and physiology of the bean plant in its "normal state" is essential before the researcher can determine whether a plant is manifesting abnormal symptoms (Flor, 1985a).

The researcher must also be familiar with the different stages of plant growth because each stage produces physiological changes. The researcher can therefore determine whether a plant is exhibiting normal growth or showing abnormalities in organ and structural differentiation (Fernández et al., 1982; Flor, 1985a). Recent greenhouse experiments at the Centro Internacional de Agricultura Tropical (CIAT), Colombia, for example, showed that certain soil

problems can be diagnosed by observation of plant symptoms at two early stages of development: when the plant forms its primary leaves, and when it develops the first trifoliolate leaf. This method of observation has also been successfully carried out in field testing for N, P, Mg, and B levels (Figure 235). It is valuable for its potential to rapidly define a problem at the earliest stages of plant growth, thereby permitting immediate treatment (Flor, 1985a and 1985b).

Visual Classification of Symptoms

The diagnostician must gain experience in the visual observation and characterization of symptoms of nutrient toxicities and deficiencies. He has to take care not to characterize problems as having "typical symptoms," as this term is applicable to only exceptionally specific cases in soil fertility studies. Alternative expressions such as "symptom complex," "syndrome," or "range of symptoms," better describe the complex of symptoms with their different levels of intensity (Figures 236 and 237) (Flor, 1985a).

Relationship Between Nutritional Requirements and Disorders

Too much or too little of any nutrient can cause a "nutritional disorder". Nutritional disorders also include toxicities from high levels of an element, substance, or ion in the soil (Tanaka and Yoshida, 1970). Studies in nutritional disorders have focused on the capacity of beans to absorb selenium (Arvy, 1983); absorption and interactions of nickel, selenium, and arsenic (Wallace et al., 1980a and 1980b; Wallace and Mueller, 1980; Wallace and Romney, 1980); and visual symptom recognition of chromium toxicity (Schmitt and Weaver, 1980). The possibility of acute or chronic damage to beans from polluting agents, particularly ozone and SO₂, has also been recognized (Cowling and Koziol, 1982; Chapter 24, this volume). There are also other elements that beans can absorb in toxic quantities, although many of these have yet to be observed in the tropics.

Nutritional disorders of beans are directly related to the plant's nutritional requirements; its response to excesses of elements,

substances, or ions in the soil; its ability to efficiently use minimal quantities of any nutrient (Figures 238 and 239); and plant age (some symptoms of nutritional disorders disappear as the plant matures) (Flor, 1985a and 1985b; Malavolta, 1976). A lot of literature exists on all points and is briefly reviewed here.

The nutritional requirements of a given plant are demonstrated by the quantity of nutrients needed to complete normal growth. The nutritional components of the original seed itself must also be considered when discussing the plant's nutritional needs (Table 3). Nutrients can be ingested from the soil, fertilizers, and, in the case of nitrogen, the air (Flor, 1985b). For example, Colombian studies have shown that, in some soils, it is possible to find about 40 kg/ha of fixable nitrogen (CIAT, 1976 and 1977).

The nutritional requirements and nutrient absorption capacities of beans vary considerably among genotypes (Table 4). A Brazilian study demonstrated this variance by investigating the nitrogen, phosphorus, and potassium needs of 90 bean cultivars. The primary macronutrients were used in very different amounts: nitrogen

Table 3. Variation in the nutrient composition of bean seeds.

| Element | Bean cultivar | | |
|------------|---------------|--------------|---------|
| | Calima | ICA Pijao | Carioca |
| (%) | | | |
| Nitrogen | 3.00 | 3.81 | 2.3 |
| Phosphorus | 0.61 | 0.61 | 0.39 |
| Potassium | 1.51 | 1.66 | 1.3 |
| Calcium | 0.24 | 0.17 | 0.35 |
| Magnesium | 0.17 | 0.19 | 0.2 |
| Sulfur | 0.15 | 0.19 | — |
| (ppm) | | | |
| Iron | 91.5 | 70.0 | 68.0 |
| Manganese | 17.0 | 17.0 | 22.0 |
| Copper | 10.0 | 11.2 | 7.0 |
| Zinc | 27.0 | 30.0 | 36.0 |
| Boron | 12.2 | 8.8 | 15.5 |

SOURCES: CIAT, 1978, and adapted from Feitosa et al., 1980.

Table 4. Differences in nutrient absorption in different cultivars of common bean.

| Cultivar and growth habit | Vegetative period (days) | Absorption (kg/ha) | | | | | |
|---------------------------|--------------------------|--------------------|-------|---------|----|----|----|
| | | N | P | K | S | Ca | Mg |
| ICA Gualí (I) | 74 | 111 | 16 | 89 | — | — | — |
| Porrillo Sintético (II) | 88-99 | 134-147 | 18-21 | 123-133 | — | — | — |
| Puebla 152 (III) | 91 | 149 | 23 | 110 | — | — | — |
| Magdalena 3 (IV) | 100 | 175 | 23 | 140 | — | — | — |
| Roxinho group | | 102 | 9 | 93 | 25 | 54 | 18 |

SOURCES: Cobra-Netto et al., 1971; Flor, 1985b; Laing, 1977.

varied between 50 and 425 kg/ha, phosphorus between 20 and 65 kg/ha, and potassium between 100 and 262 kg/ha (Amaral et al., 1980).

Analyses conducted in Kenya determined the rate beans extract nutrients from the soil. The order of absorption was N, K, Ca, Mg, P, S, Fe, Mn, Zn, and Cu. Seeds accumulate the highest quantities of N, P, Mg, and S (Qureshi, 1979).

Another study, carried out in Colombia, investigated the response of 13 bean cultivars to different levels of boron (Swann and Mora, 1975). Two cultivars, ICA Gualí and ICA Calima, did not react to boron deficiency, whereas the other 11 cultivars were severely affected (Figure A).

A better nutrient uptake does not necessarily mean that a plant will yield more. It merely shows that cultivars differ in their ability to efficiently use nutrients for seed production (Amaral et al., 1980). For example, CIAT has established four levels of phosphorus uptake efficiency and/or response (Figure B) (Thung et al., 1984).

The relationship between nutritional disorders and absorption curves for different nutrients varies from cultivar to cultivar (Figure C). The transport mechanisms, nutrient distribution, and nutritional requirements of each plant part, each growth stage, and/or growth cycle of the plant affect the quantity and timing of demand for specific nutrients (Figure D).

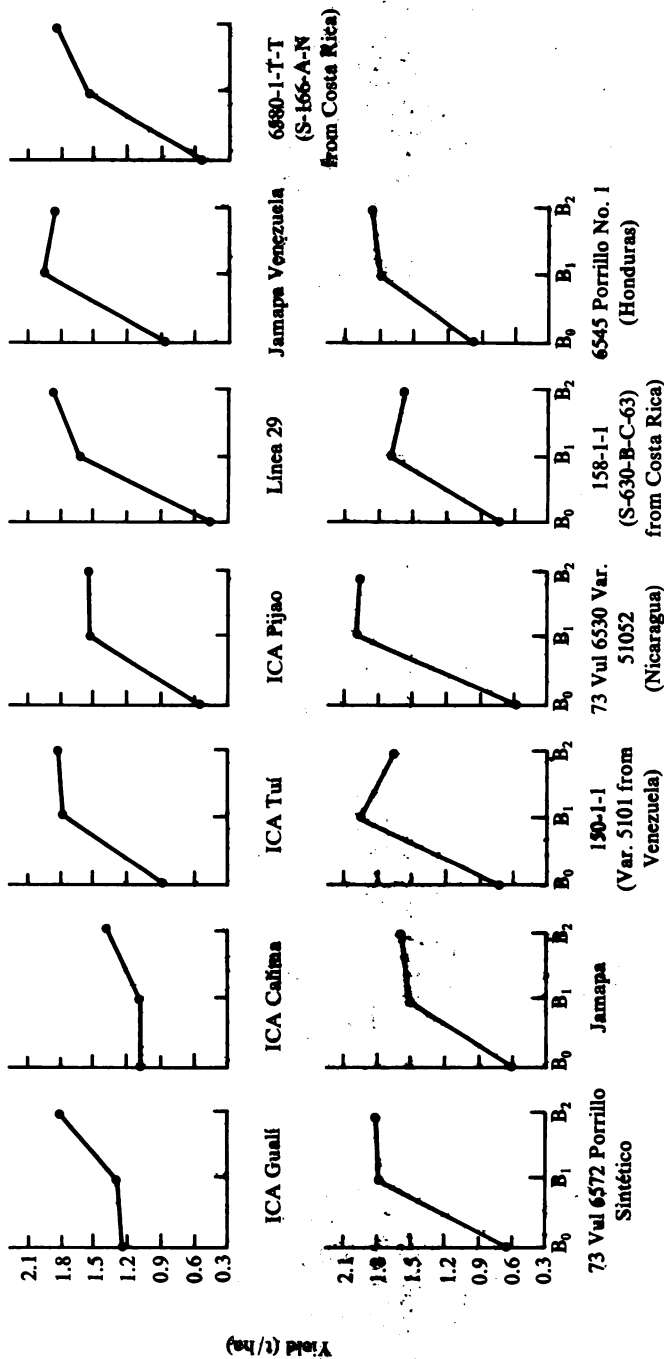


Figure A. Response of 13 bean varieties to applications of three levels of boron (B_0 , B_1 , B_2). (Taken from Swann and Mora, 1975.)

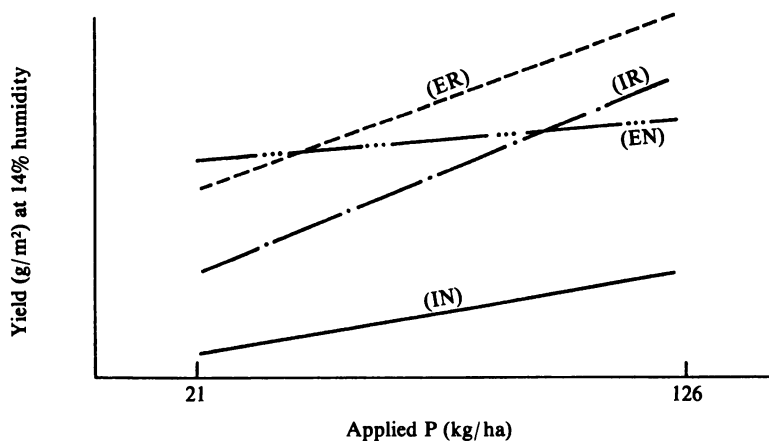


Figure B. Response and efficiency of common beans to phosphorus application. E = efficient; I = inefficient; R = some response; N = no response. (Taken from Thung, 1979.)

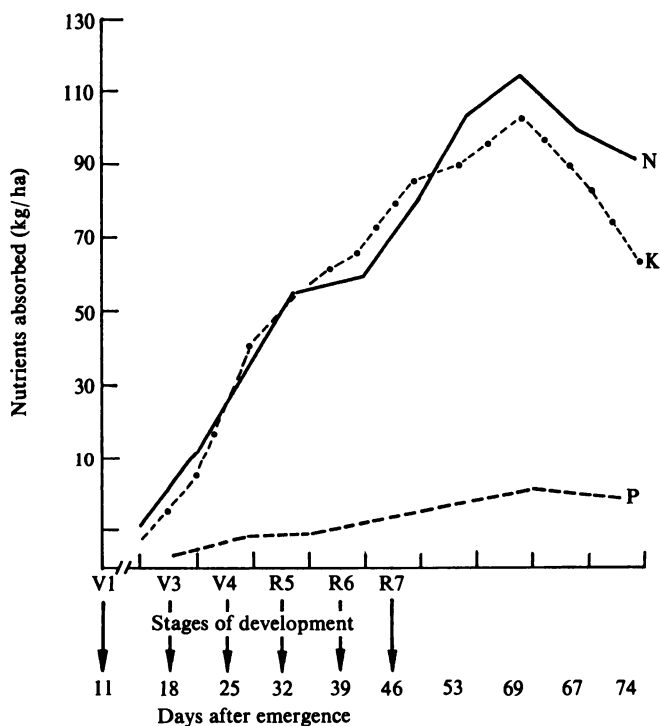


Figure C. Absorption curves for N, P, and K in the variety Porrillo Sintético. (Taken from Cardona et al., 1982.)

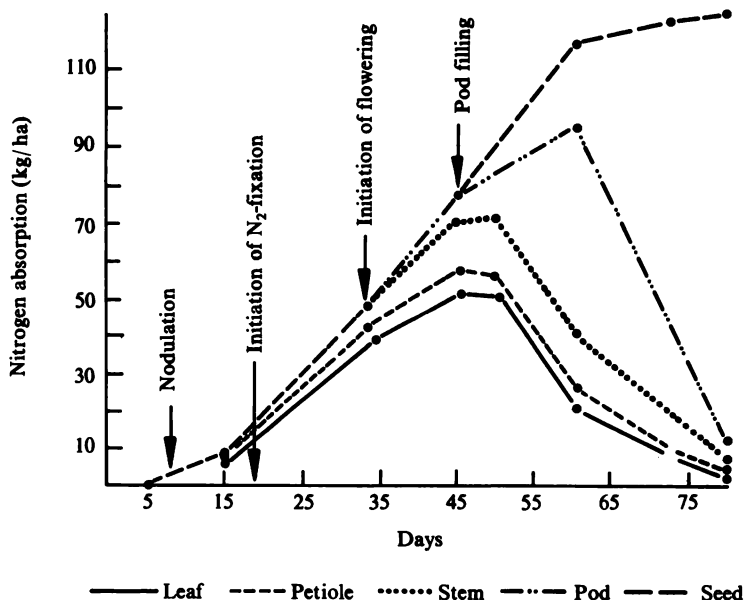


Figure D. Nitrogen absorption by the variety Porrillo Sintético throughout the growth cycle. Note the high percentage of N absorbed after flowering and the loss of N suffered by leaves and pod walls during pod filling. (Taken from Graham, 1979.)

Relationship of Nutritional Disorders to Critical Levels in Soil and Tissues

One way of determining the nutritional requirements of a cultivar is by discovering the concentration of nutrients in the soil, media, or nutritive solution (external requirements). The nutritional requirements can also be determined through the plant (internal requirements). This definition of "nutritional requirements" is equivalent to the critical levels in soil or plant. Critical levels are determined by the lowest level of nutrient application which causes a response in the plant and by the highest level of nutrient application to which the plant will still show a response, especially in yields (Howeler, 1983; Howeler and Medina, 1978).

Measuring the critical levels in tissues permits distinction between species, but not between genotypes within a species. Critical levels vary between different organs of the same plant and

with tissue age. They are also affected by the presence or absence of other nutrients and, especially, by environmental conditions.

Critical levels used for soil analysis, especially if such analysis includes recommendations to add lime or sulfur, and fertilizers, are much more valuable when they result from careful correlations between analytical methods and a well-designed field trial. The critical levels of an element in the soil varies with the method of extraction. In reality, each critical level is a range of values where the deficiency is manifested by a wide variety of symptoms, reflecting deficiencies that are light, medium, or severe (Howeler, 1983; Howeler and Medina, 1978; Thung et al., 1984; Thung et al., 1985).

Tables 5 and 6 show the values of critical levels in soils used in CIAT (Colombia) and in the Centro Nacional de Pesquisa de Arroz e Feijão (CNPAP), Brazil (Cardona et al., 1982; de Oliveira, 1983). Such data, however, cannot be generalized to other areas because critical levels vary according to local conditions. Nevertheless, soil analyses are more useful than tissue analyses for conclusive diagnoses. Tables 7 and 8 show approximations of critical levels of nutrients in bean leaves.

Table 5. Estimations of critical levels of soil nutrients needed by beans.

| Factor | Method | Critical level |
|---------------|-----------------------|---------------------------|
| pH | Soil to water = 1:1 | 5 and 8.1 |
| Al | KCl, 1N | 1 meq/100 g |
| Al saturation | Al/(Al + Ca + Mg + K) | 10% |
| P | Bray I | 11 ppm |
| | Bray II | 15 ppm |
| | Olsen-EDTA | 14 ppm |
| | North Carolina | 13 ppm |
| K | Ammonium acetate, 1N | 0.15 meq/100 g |
| Mg | Ammonium acetate | 2.0 meq/100 g |
| Ca | Ammonium acetate | 4.5 meq/100 g |
| Conductivity | Saturation extract | 0.8 mmhos/cm ^a |
| Na saturation | Ammonium acetate, 1N | 4% |
| B | Hot water | 0.4-0.6 ppm |
| Zn | North Carolina | 0.8 ppm |
| Mn | North Carolina | 5 ppm |

a. 0.8 mmhos/cm = 0.08 S/m (SI units of measurement).

SOURCE: Cardona et al., 1982.

Table 6. Interpretation of soil analyses (following CNPAF)^a from Brazil.

| Analysis | Interpretation (soil content) | | |
|-------------------------------------|----------------------------------|---------|---------------------|
| | Low (less than) | Medium | High (more than) |
| Organic matter (%) | 1.5 | 1.6-3.0 | 3 |
| Phosphorus (ppm) | 10 | 11-20 | 20 |
| Potassium (ppm) | 30 | 31-60 | 60 |
| Calcium + magnesium (meq/100 g) | 2 | 2.1-5 | 5 |
| Aluminum (meq/100 cm ³) | 0.3 | 0.4-1.0 | 1 |

a. CNPAF: Centro Nacional de Pesquisa de Arroz e Feijão, Brazil.

SOURCE: de Oliveira, 1983.

Table 7. Estimations of deficient, adequate, and toxic levels of nutritional elements in foliar tissue of beans (*Phaseolus vulgaris* L.).

| Element | Level | | |
|------------|--------------------------|-----------|----------------------|
| | Deficient (less than) | Adequate | Toxic (more than) |
| (%) | | | |
| Nitrogen | 2.50 | 2.80-6.00 | — |
| Phosphorus | 0.20 | 0.25-0.50 | — |
| Potassium | 1.50 | 1.80-2.50 | — |
| Calcium | 0.50 | 0.80-3.00 | — |
| Magnesium | 0.20 | 0.25-0.70 | — |
| (ppm) | | | |
| Iron | 50 | 100-450 | 500 |
| Zinc | 15 | 20-100 | 200 |
| Manganese | 20 | 30-300 | 500 |
| Boron | 20 | 30-60 | 200 |
| Copper | 5 | 10-20 | 30 |

SOURCE: Wilcox and Fageria, 1976.

In tissue analyses, the plant part most commonly used is the completely unfurled, trifoliate leaves at flower initiation, without the petiole (CIAT, 1976, 1977, and 1978; Howeler, 1983; Howeler

Table 8. Estimations of critical levels of nutrients in bean tissue (*Phaseolus vulgaris* L.).

| Source | N (%) | P (%) | K (%) | Ca (%) | Mg (%) | Mn (ppm) | S (%) |
|-------------------------------|-----------------------|----------|----------|-----------|-----------|-------------|----------|
| Cobra-Netto et al., 1971 | 1.54 (D) ^a | | | | | | |
| Howeler, 1983 | 3.00 (D) | | | | | | |
| Ramírez, 1969 | 3.00 (N) ^b | | | | | | |
| MacKay and Leefe, 1962 | 5.10 (N) | | | | | | |
| Howeler, 1983 | 5.20 (N) | | | | | | |
| Cobra-Netto et al., 1971 | | 0.13 (D) | | | | | |
| Howeler, 1983 | | 0.25 (D) | | | | | |
| Howeler, 1983 | | 0.40 (N) | | | | | |
| MacKay and Leefe, 1962 | | 0.40 (N) | | | | | |
| Cobra-Netto et al., 1971 | | | 0.93 (N) | | | | |
| Howeler, 1983 | | | 1.00 (D) | | | | |
| MacKay and Leefe, 1962 | | | 2.00 (N) | | | | |
| Howeler, 1983 | | | 3.00 (N) | | | | |
| Cobra-Netto et al., 1971 | | | | 0.42 (D) | | | |
| Howeler, 1983 | | | | 1.25 (D) | | | |
| Howeler, 1983 | | | | 1.60 (N) | | | |
| Abrufia et al., 1974 | | | | 2.00 (N) | | | |
| Blasco-L. and Pinchinat, 1972 | | | | 5.00 (N) | | | |
| Ramírez, 1969 | | | | | 0.25 (D) | | |
| Howeler, 1983 | | | | | 0.30 (D) | | |
| Berrios and Bergman, 1968 | | | | | 0.35 (N) | | |
| Cobra-Netto et al., 1971 | | | | | 0.48 (D) | | |
| Howeler, 1983 | | | | | 0.85 (N) | | |
| Howeler, 1983 | | | | | | 20 (D) | |
| Howeler, 1983 | | | | | | 140 (N) | |
| Blasco-L. and Pinchinat, 1972 | | | | | | 386 (N) | |
| Ramírez, 1969 | | | | | | 439 (N) | |
| Howeler, 1983 | | | | | | | 0.14 (D) |
| Ramírez, 1969 | | | | | | | 0.14 (D) |
| Howeler, 1983 | | | | | | | 0.25 (N) |
| Cobra-Netto et al., 1971 | | | | | | | 0.70 (D) |

a. (D) = Deficient level.

b. (N) = Normal level.

and Medina, 1978). Variations inherent in tissue analysis depend on the plant part, plant age, and genotype sampled. For example, the potassium content of leaves varies dramatically during the day (de Morães and Arens, 1973). Even the plant's health affect nutrient concentrations; for example, the more severe the infection by a virus such as bean yellow mosaic virus, the lower the content of calcium and magnesium in primary leaves and the higher the content of manganese and zinc in trifoliolates (Rosen et al., 1980). When analyzing for micronutrients, care must be taken to avoid contamination from fungicide applications. Comparison and contrast methodology compares the analysis of normal leaves with the analysis of "problem leaves" and is very helpful in the diagnosis of specific problems (Flor, 1985a).

Important Nutritional Disorders in Bean-Producing Regions

Introduction

The majority of the world's bean-producing regions lies in acid soil zones. These regions suffer frequent problems of low phosphorus content, high capacity to fix phosphorus, high levels of exchangeable aluminum and, therefore, frequent low levels of calcium and magnesium, and manganese toxicity (Howeler, 1980; Howeler and Medina, 1978; Thung, 1979; Thung et al., 1985).

Phosphorus deficiency and aluminum toxicity are the principal nutritional problems of beans in Latin America (Abruña et al., 1974; Fassbender, 1967; Mascarenhas et al., 1967a and 1967b; Müller et al., 1968). The availability of phosphorus is associated with moisture content so the incidence of phosphorus deficiency increases when water is scarce.

Most beans are cultivated in Oxisols, Ultisols, and acid Inceptisols which have a pH less than 5.5, high aluminum content, and low levels of calcium and magnesium. The aluminum-saturation method facilitates the assessment of soil acidity (Jones, 1984; Kamprath, 1970; Pearson, 1975). It can be calculated in the following form:

$$\text{Aluminum saturation (\%)} = \frac{\text{Al}}{\text{Al} + \text{Ca} + \text{Mg} + \text{K}} \times 100$$

where all elements are expressed in meq/100 g of soil.

There is a relationship between pH and bean yield (Figure E) (R. H. Howeler, 1985, personal communication), even though different bean genotypes respond to soil acidity differently. The majority of genotypes are noticeably affected by pH less than 4.9.

Figure F shows the relationship between percentage of aluminum saturation and bean yield. Although it demonstrates again that genotypes respond differently to aluminum excesses, the critical level of aluminum saturation is about 10% (Howeler, 1980; Howeler and Medina, 1978). However, in some Oxisols of Brazil, the critical level increases as much as 25%-30% (de Eira et al., 1974; Mohr, 1960). In some Ultisols, it is as high as 60%. Such variation is influenced by the percentage of organic matter content in the soil.

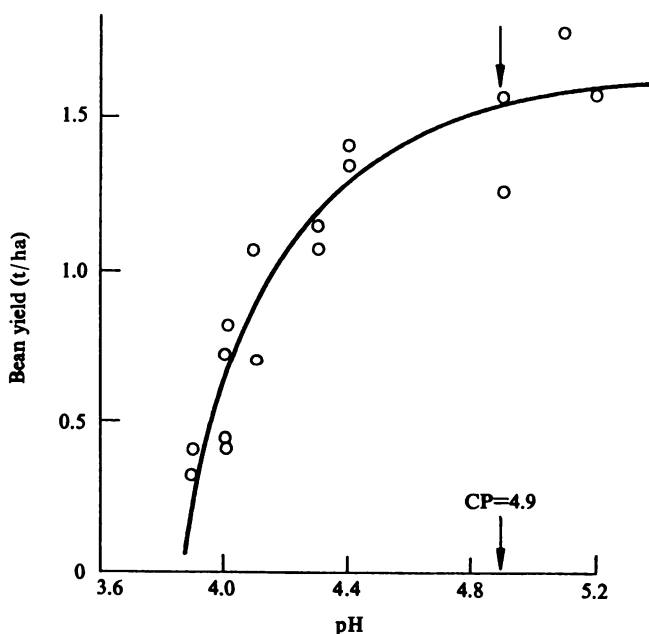


Figure E. Relationship between pH and bean yield (CP = critical point) (R. H. Howeler, personal communication, 1985).

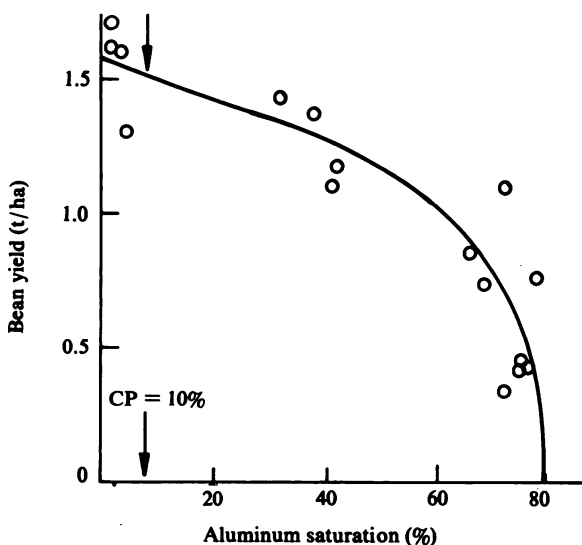


Figure F. Relationship between bean yield and the percentage of aluminum saturation CP = critical point). (Taken from Howeler and Medina, 1978.)

Phosphorus deficiency

Phosphorus deficiency is common in acid soils. It causes short, sometimes dwarfed, plants with thin stems and shortened internodes. Upper leaves are small and dark green and, when the deficiency is severe, early defoliation occurs. The vegetative period is prolonged for some days and the reproductive phase is shortened. Flowering is late, few flowers are produced, and the level of aborted blossoms is high. Few pods form and contain only a small number of seeds (Figure 240) (Howeler, 1980; Howeler and Medina, 1978; Malavolta, 1972 and 1981; Thung et al., 1984).

Phosphorus deficiency can be controlled chemically by band application of various rock phosphates or superphosphate fertilizers. Band application optimizes the use of phosphate fertilizers because only 20%-25% of this fertilizer can be used by plants. The remainder stays fixed in the soil (Kick and Minhas, 1972; Mandal and Khan, 1977; Thung et al., 1982). This residual fixed phosphorus is difficult to release and its effectiveness is therefore minimal (de Eira et al., 1974). Beans respond to phosphorus application primarily by increasing the number of pods per plant (de Oliveira et al., 1977;

Mahatanya, 1977; Thung et al., 1982). Also, better root development and penetration occurs, thereby improving the plant's ability to withstand dry periods and to compete more successfully with soil-borne pests.

Genotypes vary considerably in their ability to efficiently use low quantities of phosphorus from the soil (Amaral et al., 1980; H. P. Haag et al., 1967; W. L. Haag et al., 1978; Lindgren et al., 1977; Salinas, 1978). Examples of those cultivars which give reasonable yields even when soil phosphorus levels are very low are Carioca, Rico Pardo 896, Iguacu, G 4000, G 5059, G 5201, and G 5054—all from CIAT's germplasm bank. There are also genotypes which produce poorly under low phosphorus conditions, but respond remarkably to the application of phosphate fertilizers (Ortega, 1985; Thung, 1979).

A methodology to identify genotypes that efficiently use minimal amounts of phosphorus (CIAT, 1976; Thung et al., 1984), or respond well to applied phosphates, can be established by using these characteristics as parameters (Figure B). A certain "essential" quantity of phosphorus for survival must be provided before further delineations can be done. However, this "essential" level is not universal and must be determined for each location. CIAT genotypes that efficiently use phosphorus and respond well to additional phosphate applications are A 440, A 254, NAG 24, A 230, A 275, A 251, and 82 PVBZ 1771. The efficiency of phosphorus use is a highly heritable characteristic (Fawole et al., 1980).

Aluminum toxicity

Aluminum toxicity is easy to recognize: plants are very small and feeble, have yellow lower leaves with necrotic borders (Figure 241), and a poorly developed root system characterized by numerous white adventitious roots near the base of the stem. Lime applications to neutralize the aluminum will affect only the first 20 cm of soil. This often causes roots to grow horizontally rather than deeper into the nonaffected soil. The plant is therefore stunted and grows poorly. Aluminum usually accumulates within and on roots (Naidoo et al., 1978) and is not readily translocated to aboveground plant parts.

Aluminum toxicity is strongly related to phosphorus and calcium deficiencies. It is usually corrected by lime applications which not only neutralize the aluminum but also adjust the proportions of calcium and, if made with dolomitic lime, magnesium. Application levels for lime vary enormously and are specific to each soil type. In Santander de Quilichao in Colombia, for example, aluminum toxicity is controlled with 1 t/ha of CaCO_3 , whereas in the Cerrados of Brazil applications of 5 t/ha are normal.

However, high lime applications can induce deficiencies of zinc, boron, and magnesium. A deficiency of phosphorus can also be induced by the precipitation of phosphorus and calcium because calcium phosphate cannot be assimilated near roots (Jacobsen, 1979; Kamprath, 1970).

Bean cultivars show considerable diversity in their susceptibility to aluminum (de Oliveira and Malavolta, 1982; Foy et al., 1967; Salinas, 1978). Brazilian cultivars such as Carioca, Rio Tibagi, G 5059, Rico Pardo 896, and IPA 1 are tolerant to moderate levels of aluminum (CIAT, 1977; Ortega, 1985; Pearson, 1975). Researchers at CIAT have identified as tolerant to aluminum the genotypes A 283, A 254, A 257, A 440, and 82 PVBZ 1736.

Calcium deficiency

Acid soils with pH between 4 and 5.5 normally have low levels of calcium and magnesium, that is, $\text{Ca} + \text{Mg} = 0.5$ meq/100 g soil (Table 9). However, the plant's need for calcium is relatively high

Table 9. Characteristics of acid soils with phosphorus deficiency and aluminum toxicity.

| Country | pH | P North Carolina (ppm) | K | Ca + Mg | Al | Mn | Fe |
|----------|-----|---------------------------------|--------------------|---------|------|-------|-----|
| | | | (meq/ 100 ml soil) | | | (ppm) | |
| Brazil | 4.7 | 1.9 | — | 0.5 | 1.04 | — | — |
| Colombia | 5.6 | 4.3 | 0.22 | — | 2.00 | 50 | 4.3 |

SOURCE: M. T. Thung (personal communication, 1985).

(Table 4) even though calcium is basically immobile within the plant (de Oliveira, 1983). Calcium deficiencies are almost always found in plants suffering from aluminum toxicity or from aluminum and manganese toxicities together.

Symptoms of calcium deficiency are death of growing tips, dark green older leaves, yellow new leaves, poor root development, and sometimes collapse of the hypocotyl (Helms, 1971). Calcium deficiency particularly affects plant height and production of dry matter (Malavolta, 1981). This deficiency is commonly corrected by liming and applying simple superphosphates.

Manganese toxicity

Manganese toxicity occurs normally in soils of volcanic origin having a pH lower than 5.5. Soils with poor drainage enhance toxicity, for example, those soils of the Várzea zone of Brazil. The plant rapidly absorbs Mn^{2+} which accumulates in new leaves. Old leaves show necrosis between the ribs and new leaves turn yellow. When the toxicity is severe, leaves become wrinkled and deformed (Figure 237), appear burnt, and the plant may die. When the level of manganese toxicity is light, plants show symptoms that are easily confused with those produced by various viruses (Figure 242).

Variability exists in genotypic susceptibility or tolerance to manganese toxicity. Improving drainage conditions, tilling or preparing the soil to sufficient depth, and applying organic matter and lime alleviates manganese toxicity.

Magnesium deficiency

Magnesium deficiency usually occurs in acid soils with low base levels and on volcanic ash soils with low levels of potassium and calcium. Several acid-soil bean-producing regions have high levels of organic matter. In these soils liming not only serves to neutralize possible aluminum toxicity but also adjusts calcium and magnesium levels. This can be achieved by applying dolomitic lime (Howeler, 1980; Howeler and Medina, 1978).

Magnesium is a component of the chlorophyll molecule and is a mobile nutrient. A characteristic symptom of magnesium deficiency is interveinal chlorosis of lower leaves (Figure 243). The range of symptoms of magnesium deficiency are shown in Figure 244.

Nitrogen deficiency

Although beans are legumes which can fix nitrogen when inoculated with appropriate strains of *Rhizobium* (Figure 245) (Graham and Halliday, 1977; Graham and Rosas, 1977), cultural, varietal, or inoculation difficulties can limit this fixation ability (CIAT, 1976, 1977, and 1978; Graham, 1981). The plant is therefore left dependent on residual soil nitrogen or on applied nitrogen fertilizers.

Nitrogen deficiency occurs on all acid soils. It is especially severe in sandy soils that have low organic matter content.

Beans need more nitrogen than any other nutrient. A large quantity of nitrogen is needed for making the high percentage of protein in seeds. A study of nutritional requirements of 90 bean cultivars in Piracicaba, Brazil, found that the protein content of seeds varied between 21% and 34%, with a mean of 27%. Nitrogen extraction ranged from 50 to 425 kg of N per hectare (Amaral et al., 1980). This study revealed important differences among genotypes in their nutritional requirements. However, genotypes showing the highest nitrogen uptake were not necessarily the highest yielding beans as genotypic variability in efficiency of nitrogen use also existed.

Nitrogen deficiency first appears on lower leaves as a uniformly pale green color; these leaves later turn yellow. The deficiency is always most serious in the lower leaves because nitrogen is a mobile element. Trifoliolate leaves are small and branching is slight. Figures 246 and 247 show that nitrogen deficiency can be correctly diagnosed at the primary leaf stage (Graham, 1979; Howeler, 1980). Normal, unfurled, trifoliolate leaves contain about 5% nitrogen, but if these leaves are deficient they may contain as little as 3% nitrogen. Petioles are more useful than leaf surfaces in the diagnosis of the deficiency.

Seeds contain 6-20 mg of nitrogen. At first, beans fulfill their nitrogen requirements from the reserve present in cotyledons. However, beans begin to exhibit symptoms of nitrogen deficiency 14-20 days after emergence if they do not receive nitrogen fertilizer. It is at this stage of development that beans form nitrogen-fixing nodules. However, because nodules do not function well until they are about 30 days old, beans during this time are especially prone to nitrogen deficiencies. From about days 30 to 60, the nitrogen requirement increases almost linearly, with maximum absorption occurring about day 56. With the formation of pods, a great part of the plant's nitrogen passes to the developing seeds. By harvest time almost 90% of the nitrogen in a bean plant is found in the seeds (Graham, 1979).

Pod filling is another stage when bean plants are susceptible to nitrogen deficiency. After flowering, photosynthesis, and consequently nitrogen fixation, decreases. Some researchers obtained positive results by applying foliar nitrogen fertilizer at this stage, although the majority of recent studies have not confirmed them (Fernández et al., 1982; Graham, 1979).

Rhizobium studies have determined that the following factors are important for nodulation and fixation of nitrogen:

- presence and supply of an appropriate *Rhizobium* strain;
- specificity between *Rhizobium* strain and the host;
- soil acidity;
- soil temperature;
- nutritional factors;
- use of chemical products;
- cultural factors (farming system, etc.); and
- competition between native *Rhizobia* and introduced high-efficiency strains (Graham, 1978 and 1981).

There is clear evidence that differences in nitrogen-fixing capacity exist among genotypes. In general, genotypes with long vegetative cycles (growth habit IV) have the highest capacity for nitrogen fixation. Slow-growing cultivars also fix more nitrogen. Nitrogen deficiencies can be controlled by applications of nitrogen fertilizers and organic matter. There is little difference in quality between the principal sources of nitrogen which are urea, sodium nitrate, and

ammonium sulfate. Neither are there important differences in times of application, except that repeated applications of nitrogen in rainy areas are helpful (Graham, 1978, 1979, and 1981; Graham and Rosas, 1977; Kick and Minhas, 1972).

Potassium deficiency

The major bean-producing areas of Latin America have soils containing medium to high levels of potassium. The response to additional potassium applications is therefore negligible. A Brazilian study showed that, of 232 trials, only 15 responded positively to potassium supplements (Howeler, 1980; Howeler and Medina, 1978; Malavolta, 1972). Deficiencies occur in Oxisols and Ultisols with very low fertility, in soils with high calcium and magnesium contents, or in highly permeable sandy soils.

Potassium is a mobile element and therefore a deficiency first appears in the lower leaves. Primary leaves manifest serious symptoms when potassium deficiency is severe (Figure 235). The affected plant has very weak stems with short internode length, reduced root growth, and a proneness to collapse (Figure 248). Genotypes differ in their ability to efficiently use small quantities of soil potassium. Potassium use is controlled genetically by one simple gene (Shea, 1966; Shea et al., 1968).

Potassium deficiency can be corrected by applying commercial products such as potassium chloride (KCl, 50% K) and potassium sulfate (K_2SO_4 , 42% K).

Micronutrient deficiencies

Zinc deficiency. Zinc deficiencies occur principally in soils that have a high pH. They also occur in acid soils that have been treated with too much lime and/or phosphorus. Elevated absorption of other nutrients such as iron, can also induce zinc deficiency.

Zinc deficiency has also been reported in slightly alkaline soils with high moisture content. Affected plants show a yellowing of younger leaves. These chlorotic leaves have a high phosphorus content, causing an imbalance in the P:Zn ratio. High soil moisture

apparently increases the availability and absorption of phosphorus which, in turn, induces zinc deficiency (Khan and Soltanpour, 1978). A physiological antagonism between zinc and phosphorus thus occurs within the plant.

The predominantly Oxisol and Ultisol soils of Brazil's Cerrados and Colombia's "Llanos Orientales" experience zinc deficiencies. Here, the deficiency is associated with a low nutrient content in the parent material.

Zinc deficiency first appears as an interveinal chlorosis of young leaves. Later, clear brown spots appear on leaves and folioles lengthen and become deformed (Figure 249).

Zinc sulfate (ZnSO_4) is commonly used to control zinc deficiency. Foliar applications of this chemical easily control light to moderate deficiencies.

Genotypes vary in their reactions to deficiencies, and to excesses, of zinc. For example, the cultivar Saginaw is more tolerant of zinc deficiency than of an excess of zinc, whereas the reverse is true for the cultivar Sanilac. In Saginaw, an excess of zinc induces ferric chlorosis (Brown, 1978) which it does not in Sanilac, because Sanilac absorbs more iron and phosphorus than does Saginaw (Ambler and Brown, 1969).

Boron deficiency. Boron deficiencies occur in various soil types: sandy textured soils that have low organic matter content and high levels of aluminum and iron hydroxide; alluvial soils that have high pH and low total boron content (CIAT, 1976, 1977, and 1978); and neutral or alkaline soils that are subject to dryness and intense sunlight. The deficiency is more critical in sandy soils because of the instability of soil particles. Liming lessens the availability of boron (Malavolta, 1976).

The first symptom of boron deficiency is the death of the main growing tip. Lateral buds produce many small branches, but the terminal buds die. Primary leaves thicken, deform, and become leathery. The folioles curl and petioles become brittle. The trifoliate leaves may form only one or two deformed folioles. Flowers, and consequently pods, are not formed, and the root system is poorly developed (Howeler et al., 1978; Swann and Mora, 1975)

(Figures 234 and 236). Nutritional requirements double under intense sunlight, raising the boron level in leaves (Howeler et al., 1978; Swann and Mora, 1975). Dryness and low soil moisture also intensify deficiency symptoms (Malavolta, 1976).

Varieties differ in their susceptibility to boron deficiency (Figure 234). In general, black beans are more susceptible than red beans.

Iron deficiency. Iron deficiency is rare, but can occur in calcareous soils containing free calcium carbonate (Coyne et al., 1973). It can also occur in acid soils that have been excessively limed.

Although the literature reports that iron is absorbed in its Fe^{3+} form, beans grown at CIAT in soils with a pH above 7.5 have shown chlorosis in the youngest leaves after extremely heavy rainfalls. Foliar analysis showed that these affected leaves contained elevated levels of iron in its Fe^{2+} form which predominates in soils under iron-reduction conditions. Excess absorption of Fe^{2+} affects the Fe:Zn ratio and produces zinc deficiency.

Symptoms of iron deficiency appear in young leaves which become pale yellow to almost white, while the veins remain green (Figure 250). Iron is extremely mobile within the plant (de Oliveira, 1983; Howeler and Medina, 1978).

Iron deficiency can be corrected by applying chelates. Inorganic iron salts cannot be recommended because they are easily leached out of soils with high pH (Coyne et al., 1982; Heinonen and Waris, 1956).

The Great Northern cultivars Valley, Emerson, and U.I. 59 are tolerant to iron deficiency.

Manganese deficiency. Manganese deficiency occurs in soils having a pH higher than 6.7, in organic soils, poorly drained soils, or in acid soils that have been heavily limed. The principal symptoms are dwarfism and the presence of gold-yellow coloring between veins on leaves. Affected leaves contain less than 30 ppm manganese, whereas the manganese content in normal leaves is between 75 and 250 ppm (Howeler, 1980).

Sulfur deficiency. Sulfur deficiency is not common in Latin America, although it occurs in some Oxisols of the Cerrados of Brazil. The symptoms of sulfur deficiency are very similar to those of nitrogen deficiency, but differ in there being a uniform chlorosis of lower leaves which later spreads to younger leaves (de Oliveira, 1983; Howeler, 1980; Howeler and Medina, 1978).

A correct proportion of nitrogen to sulfur is important for the formation of proteins. A N:S proportion of about 15 is adequate (Ligero and Lluch, 1982).

Sulfur deficiency is usually corrected by the application of powdered sulfur at a rate of 10-20 kg/ha. Some fertilizers such as ammonium sulfate (24% S) or simple superphosphate (12% S), can also be used.

Copper deficiency. Compared with other crops, beans are not particularly sensitive to copper deficiency (Lucas and Knezek, 1972). The plant's need for copper is so small that practically any soil can supply the demand.

Very little research has been done in Latin America on copper deficiency. However, it occurs in organic soils, sandy soils, and in over-limed acid soils (Howeler, 1980). Beans with copper deficiency are stunted and have short internodes. Young leaves are gray or blue-green.

Copper deficiency can be corrected by applying 5-10 kg of copper per hectare, using copper sulfate. Minor deficiencies can be corrected by foliar applications of copper sulfate or copper chelate (Howeler, 1980).

Molybdenum deficiency. Molybdenum is an immobile nutrient. Symptoms of molybdenum deficiency resemble those of nitrogen deficiency (de Oliveira, 1983). In general, the deficiency occurs in soils with a pH of less than 5.5 and in which the presence of iron and aluminum reduces molybdenum solubility.

Sodium and saline toxicities

Beans are very sensitive to soil salinity and/or sodium content of a soil. In general, sodium content becomes a problem for beans

when the percentage of saturation is more than 4%. Salinity affects beans when conductivity is more than 0.8 mmhos/cm (0.08 S/m in SI units) (Cardona et al., 1982).

Genotypes vary considerably in growth and survival in saline soils and/or soils with high sodium content. Susceptible genotypes suffer severe growth reduction, leaf burn, and eventual death. Damage at germination and seedling stage is high and may significantly reduce plant population (Ayoub, 1974 and 1975; Colmenares-M. and Blasco-L., 1974; León and Medina, 1978). Especially in soils with excessive sodium content, the occurrence of unfavorable physical properties such as compaction, complicates the problem.

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Chapter 24

ADDITIONAL PROBLEMS

H. F. Schwartz*

Introduction

Many other factors besides plant pathogens, nematodes, insects, 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 are sometimes confused with those caused by other problems described elsewhere in this book. Proper identification of the causal agent often requires the construction of a complete history of all past and current factors in the problems of bean production of a given region. This chapter describes briefly some miscellaneous problems which may occur in bean production, with emphasis given to Latin and North America.

Biotic Problems

Parasitic plants such as dodder, are known to cause damage to cultivated crops, including common beans (USDA, 1953; Walker, 1969; Wellman, 1972; Westcott, 1971). *Cassythia filiformis* L. parasitizes bean plants under controlled conditions (Wellman, 1972). *Cuscuta epithymum* (L.) Murr. (clover dodder) is a parasite

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of many legumes (Westcott, 1971). Dodder produces slender, nearly leafless, vines (Figure 251) which may be white, yellow, orange, or reddish purple. When vines invade a host such as a bean plant, they wrap themselves around plant parts and develop haustoria or suckers through which the dodder obtains nutrients from the bean plant. The vines then spread from plant to plant and can seriously reduce yields (Walker, 1969).

Pieces of dodder vine and seeds can be disseminated by animals, man, farm implements, and surface irrigation water. Control measures are sanitation before the dodder produces seeds, burning residue to destroy seeds, and rotation with resistant crops such as cereals, soybeans, or cowpeas (USDA, 1953; Westcott, 1971).

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, giving rise to certain cultivars that are peculiarly adapted to growing conditions unique to specific production areas. However, even these cultivars can be affected by extremes or variations resulting from one or another environmental factor during a season.

Moisture. Plants may suffer high or low moisture stresses that influence physiological processes, plant development, and susceptibility to plant pathogens. For example, low soil-moisture content damages plants because there is insufficient water for roots; toxic ions such as magnesium and boron, accumulate; stomata close; uptake of CO_2 is restricted; and the plant wilts, either temporarily or permanently (NAS, 1968).

High soil moisture and flooding leach out important nutrients necessary 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 also increase the rate of respiration (NAS, 1968; Walker, 1969; Zaumeyer and Thomas, 1957).

High soil moisture or relative humidity induces intumescence in cultivars which have abundant foliage and pods that are not directly exposed to the sun. Cells elongate and multiply, resulting in raised dark green spots that appear on leaves or pods. The spots burst (edema) if high moisture conditions persist (Zaumeyer and Thomas, 1957).

Leaves can be damaged by the impact of large droplets of water during rainstorms, causing leaf wilt or defoliation (Natti and Judge, 1971). Hail and lightning damage can also occur during rainstorms, stunting plant development, creating wounds through which secondary disease agents enter, and even causing plant death (Natti and Judge, 1971; Walker, 1969).

Temperature. Beans also are affected by soil and air temperatures; sudden changes affect the plant's ability to absorb soil moisture. Low temperatures produce chilling or frost damage (Figure 252) which appears as dark water-soaked areas on wilted leaves or plants. If these low temperatures persist they stunt general plant development.

High temperatures induce flower abortion (Westcott, 1971), increase the rate of evapotranspiration, and cause plant wilt if there is insufficient soil moisture or limited root growth. High temperatures and winds compound plant stresses from low soil moisture by physically inducing soil aggregation, cracks, and subsequent root damage (NAS, 1968). Seedlings develop basal lesions at the soil line if the soil surface becomes too hot (NAS, 1968; Walker, 1969; Westcott, 1971; Zaumeyer and Thomas, 1957).

Sunscauld. Sunscauld of bean leaves, stems, branches, and pods occurs during periods of intense sunlight (that is, high radiation of ultraviolet wave length), especially after periods of high humidity and cloud cover (Walker, 1969; Zaumeyer and Thomas, 1957). High temperatures also induce sunscauld damage (Walker, 1969). 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 (Figure 253).

These symptoms resemble those caused by tropical spider mite and air pollutants.

Bean development is also influenced by light intensity, quality, and duration (photoperiod). Reduced light causes etiolation, characterized by succulent growth and long stem internodes, and often reduces chlorophyll content and flower production (NAS, 1968; Walker, 1969). Cultivars sensitive to photoperiod and planted at high latitudes do not flower normally, producing only a few pods late in the growing season. However, plants often appear healthy and green unless low temperatures cause abnormalities. High light intensity scorches or burns leaves and pods (russet) and causes flower-and-pod abortion. It also intensifies damage caused by chemical spray droplets or air pollution, especially that caused by photochemical pollutants (NAS, 1968; Zaumeyer and Thomas, 1957).

Wind. Wind speed and direction affect plant development. Evapotranspiration rates are increased by persistent winds and so aggravate plant moisture stress. Violent plant movement damages roots and predisposes them to subsequent root-rot problems. It also breaks stems and branches and causes plant lodging, especially if soil moisture is high (NAS, 1968).

Beans are also damaged by the abrasive action of wind and airborne soil particles (Bubenzer and Weis, 1974; Zaumeyer and Thomas, 1957). For example, after a 20-minute exposure to winds of 15.5 m/s in the field, there was a yield loss of 8% from plants that suffered leaf damage as seedlings (Figure 254). There was a 14% yield loss when flowering plants lost buds and blossoms (Bubenzer and Weis, 1974).

Mechanical. Bean plants can be damaged physically during cultivation, application of pesticides, or preparation of irrigation furrows if care is not taken and bean plants have produced abundant vegetation. Wounds on leaves and other plant parts provide entry for various bean pathogens, especially bacteria.

Bean seeds can be mechanically or physically damaged during harvesting, threshing, processing, and planting operations, especially when seed-moisture content is low (Copeland, 1978; Westcott, 1971; Zaumeyer and Thomas, 1957). External seed damage consists of cracked seed coats and cotyledons. Internal damage consists 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 which plants survive only by producing buds in the cotyledon axils (Figure 255). A similar symptom, snakehead, occurs from damage by insects or common bacterial blight. Seedlings which survive the effects of mechanical damage are often stunted and yield poorly (Copeland and Saettler, 1978; Zaumeyer and Thomas, 1957).

Genetic Problems

Beans occasionally exhibit physiological and genetic abnormalities which may be confused with symptoms induced by plant pathogens or abiotic factors. Albino seedlings occur but usually die within a few days because they lack chlorophyll. Leaf variegations appear as mosaic patterns of green, yellow, and white tissue (Figure 256) and can cause abnormal development of plant and pods. Individual leaves or branches may be affected or the entire plant is variegated (Westcott, 1971; Zaumeyer and Thomas, 1957). General plant chlorosis and pseudo-mosaic symptoms can be heritable traits. Certain cultivars exhibit small chlorotic spots (yellow spot) on primary and trifoliolate leaves, but still develop normally. The trait is heritable (Zaumeyer and Thomas, 1957).

A heritable seedling wilt, that is, not caused by root rot, causes primary leaves to become pale, bronzed, curl slightly, and senesce, resulting in plant death. Internal necrosis is also a heritable trait and produces brown necrotic spots on the flat surface of cotyledons (Zaumeyer and Thomas, 1957). Cripples or abnormal plant development occur and are probably caused by genetic abnormalities.

Seed-coat splitting occurs in certain cultivars and is probably inherited. The cotyledons and seed coat grow unevenly, exposing the cotyledons which then extend beyond the seed coat. They are cone shaped, rough, and serrated (Zaumeyer and Thomas, 1957). Other factors such as moisture and temperature, are often involved.

Chemical Problems

Chemical toxicities. If chemicals are not applied according to manufacturers' recommendations, beans will be injured, especially if the

chemicals are applied during germination and seedling development. Toxic concentrations of various chemicals and fertilizers placed too close to seeds create problems if they do not dissolve and leach rapidly throughout the root zone (NAS, 1968; Zaumeyer and Thomas, 1957). Insecticides (Figure 257), paraquat spray drift (Figure 258), and 2,4-D spray drift (Figure 259) produce distinctive necrotic or morphological symptoms on affected leaves or plant parts. Other physiological disorders are caused by chemicals which contain impurities or products that are metabolized by soil microorganisms into toxic byproducts or aggravated by specific soil and environmental conditions.

Root injury by herbicides and pesticides are increased by soil-moisture stress, low temperature, deep planting, soil compaction, and mechanically damaged seed (Wyse et al., 1976b). Chemically damaged roots are often predisposed to subsequent infection by root-rot pathogens (Mussa and Russell, 1977; Wyse et al., 1976a, 1976b, and 1976c).

Air pollution. Air pollution is important in many parts of the world where beans are planted close to pollution sources such as near industries that release gaseous byproducts, downwind of urban areas, close to gaseous byproducts generated by transport, or where natural environmental processes pollute the air. Air pollutants which affect beans are ozone, peroxyacetyl nitrate (PAN), sulfur dioxide, fluorides, solid particles (that is, sand or soil), and chlorine. Air pollutants also influence the interactions between beans and plant pathogens.

Ozone (O_3) is a common air pollutant formed by electrical discharge during thunderstorms. However, by far the most important source of phytotoxic O_3 is the photochemical production from gases liberated by combustion engines (EPA, 1978). Yield losses greater than 50% have been reported on common beans (Saettler, 1978). Kohut and Laurence (1983) report that 0.06-0.09 ppm ozone concentrations during pod filling causes foliar injury, extensive defoliation, and yield reductions of 24%-27% under field conditions. Ozone injury or bronzing first appears on the upper leaf surface as small water-soaked or necrotic lesions which coalesce and become bronze or reddish-brown (Figure 260). They resemble sunscald damage (EPA, 1978; Hofstra and Ormrod, 1977; Saettler,

1978; Weaver and Jackson, 1968). Premature senescence and defoliation then occurs, especially if ozone concentrations reach 100 ppm (Saettler, 1978). The severity of plant damage is affected by ozone concentrations, cultivar sensitivity, leaf age, light (Figure 261), temperature, humidity, soil moisture and texture, and plant nutrition (Brennan and Rhoads, 1976; EPA, 1978; Saettler, 1978; Tonneijck, 1983). A series of successive short exposures to ozone was more damaging than continuous exposure to the same concentration for the same total time (Stan and Schicker, 1982).

Guri (1983) reports that two major interacting genes and an undetermined number of genes with minor effects control the expression of ozone insensitivity in *P. vulgaris*. Hucl and Beversdorf (1982) report that field selection for insensitivity is affected by maturity and injury levels. They recommend that early generation selections be made under controlled conditions, to be followed by field evaluations as lines approach homozygosity.

Peroxyacetyl nitrate (PAN) is formed by photochemical interaction between hydrocarbons, resulting from incomplete combustion of petroleum products, and oxides of nitrogen. PAN damage first appears on the lower leaf surface as a water-soaked, shiny or silvery area (Figure 262) that eventually becomes bronzed (Metzler and Pell, 1980). Symptoms resemble those induced by frost, sunscald, or various insects (EPA, 1978) such as the tropical spider mite.

Sulfur dioxide (SO₂) is formed during the combustion of fossil fuels and either acts directly as an air pollutant or combines with water to form sulfuric acid mist (EPA, 1978). SO₂ injury appears on the upper or lower leaf surface as a dull, dark-green, water-soaked area which eventually turns necrotic or bleached (Figure 263) (EPA, 1978; Hofstra and Ormrod, 1977). SO₂ injury is usually more serious on younger leaves than on older ones (EPA, 1978), especially when temperature, soil moisture, and relative humidity are high (Davids et al., 1981).

Other air pollutants exist which damage beans, but they are usually not as common as ozone, PAN, or SO₂. Hydrogen fluoride damages young leaf tips and margins which then become necrotic, causing leaf edges to curl downwards. Plant problems are severe

near sources of hydrogen fluoride such as aluminum smelters, phosphate fertilizer operations, or chemical plants.

Chlorine gas induces dark green leaf spots or flecks on the upper leaf surface. These spots later become light tan or brown and resemble ozone damage. Chlorine also causes interveinal bleaching similar to SO_2 damage.

Hydrochloric acid (HCl) causes yellow-brown to brown, red, or nearly black necrosis (flecks or spots), surrounded by a cream or white border on leaf margins or interveinal tissue on the upper leaf surface. HCl also causes a glazing on the lower leaf surface which resembles PAN damage. Swiecki et al. (1982) report that cuticular resistance, influenced by the amount of epicuticular wax, determines the degree of leaf glazing by gaseous HCl .

Nitrogen oxide and nitrogen dioxide (NO_2) can cause chlorotic or bleached symptoms on the upper leaf surface. These symptoms extend to the lower leaf surface and resemble SO_2 damage. Necrotic lesions induced by NO_2 fall out of the leaf, leaving a shot-hole appearance (EPA, 1978).

Air pollutants interact with each other or with plant pathogens to alter the type and intensity of damage to beans. For example, additive, synergistic, or antagonistic interactions occur between ozone-PAN and ozone- SO_2 . The type of interaction depends on the concentration of each pollutant and sensitivity of plants (Hofstra and Ormrod, 1977; Jacobson and Colavito, 1976; Kohut and Davis, 1978). Various pollutants also influence plant pathogens and the resulting symptoms on infected or exposed plants (EPA, 1978).

Rust and halo blight infection are altered by interaction with fluorides. For example, smaller, but more numerous, rust pustules develop more slowly in the presence of fluorides than in their absence (Laurence, 1981). Ozone-sensitive beans, inoculated with bean common mosaic virus, were less damaged than normal after exposure to the pollutant (Davis and Smith, 1974). Population growth of the common bacterial blight pathogen on leaves was not affected by SO_2 exposure (Laurence and Reynolds, 1984b). However, the bacterium produced smaller lesions and had a longer latent period after exposure to SO_2 (Laurence and Reynolds, 1982) or hydrogen fluoride (Laurence and Reynolds, 1984a).

Ozone damage has been reduced on various crops, including tobacco and onions, by applying antioxidants such as dichlone and the dithiocarbamates (Kohut and Davis, 1978). Bean damage by oxidants can be reduced by applying benomyl (Manning et al., 1974; Pell, 1976) and N-[2-(2-oxo-1-imidazolidinyl)ethyl]-N¹-phenylurea or EDU (Carnahan et al., 1978). Other control measures are identifying and developing cultivars that are less sensitive to damage by various pollutants and their interactions.

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APPENDICES

Appendix I. Official Common Names and Formulae of Chemicals Cited in Text

The chemical compounds listed below were cited by authors in various chapters of this book. The list is intended to aid the proper identification of these chemicals and does not constitute an endorsement of them by CIAT.

| Official common name | Chemical formula ¹ |
|----------------------|--|
| ANTIBIOTICS | |
| Streptomycin | 2,4-Diguanidino-3,5,6,-trihydroxycyclohexyl-5-deoxy-2-O-(2-deoxy-23 methylamino- α -glucopyranosyl)-3-formyl pentantofuranoside |
| Terramycin | Oxytetracycline hydrochloride |
| Tetracycline | Prepared from chlortetracycline or oxytetracycline |
| ANTIOXIDANTS | |
| Dithiocarbamates | <i>See fungicides</i> Ferbam, mancozeb, maneb, metiram, zineb, ziram |
| EDU | N-[2-(2-oxo-1-imidazolidinyl)ethyl]-N ¹ -phenylurea |
| FUMIGANTS | |
| Aluminum phosphide | Al P |
| Chloropicrin | Trichloronitromethane |
| D-D | Mixture of E+Z isomers of 1,3-dichloropropene and 1,2-dichloropropane |

1. SOURCES: Farm chemicals handbook '88. 1988. Meister Publishing, Willoughby, OH, USA.
Thomson, W. T. 1986. Agricultural chemicals, books I-IV. Thomson Publications, Fresno, CA, USA.

Official common name**Chemical formula**

Dichloropropene
Methyl bromide
Vorlex

1,3-Dichloropropene
Bromomethane
Mixture of methyl isothiocyanate,
1,3-dichloropropene, and other chlorinated C₃
hydrocarbons

FUNGICIDES

Benomyl

Methyl 1-(butylcarbamoyl)-2-benzimidazole-carbamate

Bitertanol

β -([+1,1'-biphenyl]-4yloxy)- α (1,1 dimethyl-ethyl) 1H-1,2,4-triazole-1-ethanol

Bordeaux mixture

Mixture of copper sulfate and calcium hydroxide (lime)

Busan 30A

2-(Thiocyanomethylthio) benzothiazole

Captafol

cis-N-((1,1,2,2-Tetrachloroethyl) thio)4-cyclohexene-1,2-dicarboximide

Captan

cis-N-Trichloromethylthio-4-cyclohexene-1,2-dicarboximide

Carbendazim

2-(Methoxycarbonylamino)-benzimidazole

Carboxin

5,6-Dihydro-2-methyl-N-phenyl-1,4-oxathiin-3-carboxamide

Ceresan

Ethylmercury chloride. Discontinued.

Chloroneb

1,4-Dichloro-2,5-dimethoxybenzene

Chlorothalonil

Tetrachloroisophthalonitrile

Copper hydroxide

Cupric hydroxide (Cu(OH)₂)

Copper oxides

Cuprous oxide (Cu₂O); and cupric oxide (CuO)

Copper oxychloride

Basic cupric chloride (approximately 3Cu(OH)₂.CuCl₂)

Copper sulfate

Cupric sulfate pentahydrate

Dazomet

Tetrahydro-3,5-dimethyl-2H-1,3,5-thiadiazine-2-thione

Dichlone

2,3-Dichloro-1,4-naphthoquinone

Dicloran

2,6-Dichloro-4-nitroaniline

Dinocap

Mixture of 2,4-Dinitro-6-octyl-phenyl-crotonate, 2,6-dinitro-4-octyl-phenyl crotonate, and nitrooctyl-phenols

Fenaminosulf

Sodium [4-(dimethylamino)phenyl] diazene sulfonate

Fentin acetate

Acetoxy-triphenylstannane

| Official common name | Chemical formula |
|----------------------|--|
| Fentin hydroxide | Triphenyltin hydroxide |
| Ferbam | Ferric dimethyldithiocarbamate |
| Iprodione | 3-(3,5-dichlorophenyl)-N-(1-methylethyl)2,4-dioxo-1-imidazolidinecarboxamide |
| Kasugamycin | [5-amino-2 methyl-6- (2,3,4,5,6,-pentahydroxy cyclohexyloxy) tetrahydropyran-3-yl] amino- α -iminoacetic acid |
| Lime sulfur | Aqueous solution of calcium polysulfides |
| Mancozeb | Manganese ethylene bisdithiocarbamate with zinc ion |
| Maneb | Manganese ethylenebisdithiocarbamate |
| Mercuric chloride | HgCl ₂ . Discontinued. |
| Metalaxyl | N-(2,6-Dimethylphenyl)-N-(methoxyacetyl)-alanine methyl ester |
| Metiram | Tris {ammine {ethylenebis(dithiocarbamato)} zinc(2+)} {tetrahydro-1,2,4,7-dithiadiazocine-3, 8-dithione}; polymer |
| Nabam | Disodium ethylene-1,2-bisdithiocarbamate |
| Oxycarboxin | 5,6-Dihydro-2-methyl-N-phenyl-1, 4-oxathiin-3-carboxamide 4,4-dioxide |
| PCNB | Pentachloronitrobenzene |
| PMA | Phenylmercury acetate |
| Procymidone | N-(3,5-dichlorophenyl)-1, 2-dimethylcyclopropane-1,2-dicarboximide |
| Propiconazole | 1-{2-(2,4-dichlorophenyl)4-propyl-1,3-dioxolan-2-ylmethyl}-1H-1,2,4-triazole |
| Prothiocarb | Ethyl-N-(3-dimethylamino-propyl)-thiolcarbamate hydrochloride. Discontinued. |
| Pyroxychlor | 2-chloro-6 methoxy-4-(trichloromethyl) pyridine |
| Sulfur | Elemental sulfur |
| Terrazole | 5-Ethoxy-3-trichloromethyl-1,2,4-thiadiazole |
| Thiabendazole | 2-(4'-Thiazolyl)-benzimidazole |
| Thiophanate | Diethyl {1,2-phenylenebis(iminocarbonothioyl)}-bis{carbamate} |
| Thiophanate-methyl | 4,4'-o-phenylenebis {3-thioallophanate} |
| Thiram | Tetramethylthiuram disulfide |
| Triadimefon | 1-(4-Chlorophenoxy)-3,3-dimethyl-1-(1H-1,2,4-triazol-1-yl)-2-butanone |
| Tridemorph | N-Tridecyl-2, 6-dimethylmorpholine |

| Official common name | Chemical formula |
|----------------------|--|
| Triphenyl phosphite | $(C_6H_5O)_3 P$. Not commercially available as a fungicide. |
| Vinclozolin | 3-(3,5-Dichlorophenyl)-5-ethenyl-5-methyl-2,4-oxazolidinedione |
| Zineb | Zinc ethylenebisdithiocarbamate |
| Ziram | Zinc dimethyldithiocarbamate |

HERBICIDES

| | |
|---------------|---|
| Alachlor | 2-Chloro-2'-6'-diethyl-N-(methoxymethyl)-acetanilide |
| Atrazine | 2-chloro-4-ethylamino-6-isopropylamino-1,3,5 triazine |
| Avadex | S-(2,3-Dichloroallyl)diisopropylthiocarbamate. Discontinued. |
| Bentazon | 3-(1-Methylethyl)-1H-2,1,3-benzothiadiazin-4(3H)-one 2,2-dioxide |
| Cycloate | S-ethyl-N-cyclohexyl-N-ethylthiocarbamate |
| Dinitramine | N_4, N_4 -Diethyl-a,a,a-trifluoro-3,5-dinitrotoluene-2,4-diamine |
| Dinoseb | 2-(sec-butyl)-4,6-dinitrophenol |
| Eptam | S-Ethyl dipropylthiocarbamate |
| Fluorodifen | 2,4'-Dinitro-4-trifluoromethyl-diphenyl ether. Discontinued. |
| Fluometuron | 1,1-Dimethyl-3-(a,a,a-trifluoro-m-tolyl) urea |
| Fusilade | RS butyl 2-{4-(5-trifluoromethyl-2-pyridol oxy) phenoxy} propionate |
| Glyphosate | isopropylamine salt of N-(phosphono-methyl) glycine |
| Linuron | 3-(3,4-Dichlorophenyl)-1-methoxy-1-methyl-urea |
| Metribuzin | 4-Amino-6-(1,1-dimethylethyl)-3-(methylthio)-1,2,4-triazin- 5(4H)-one |
| Paraquat | 1,1'-Dimethyl-4,4'-bipyridinium ion |
| Pendimethalin | N-(1-ethylpropyl)-3,4-dimethyl-2,6-dinitro-benzenamine |
| Simazine | 2-chloro-4,6-bis(ethylamino)-s-triazine |
| Trifluralin | a,a,a-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 | (1R,4S,4aS,5S,8R,8aR)-1,2,3,4,10,10-hexa- chloro-1,4,4a,5,8,8a-hexahydro-1,4:5,8- dimethanonaphthalene (not less than 95%). |
| Aminocarb | 4-(Dimethylamino)-3-methylphenolmethyl- carbamate |
| Carbaryl | 1-Naphthyl N-methylcarbamate |
| Carbofuran | 2,3-Dihydro-2,2-dimethyl-7-benzofuranyl meth- ylcarbamate |
| Chlorpyrifos | O,O-Diethyl O-(3,5,6-trichloro-2-pyridyl)- phos- phorothioate |
| Cypermethrin | (RS)- α -cyano-3-phenoxybenzyl (1RS, 3RS; 1RS, 3SR)-3-(2,2-dichlorovinyl)-2,2-dimethyl- cyclopropanecarboxylate |
| Deltamethrin | (S)- α -cyano-m-phenoxybenzyl (1R, 3R)-3(2,2- dibromovinyl)-2,dimethylcyclopropane-car- boxylate |
| Diazinon | O,O-Diethyl O-(2-isopropyl-4-methyl-6-py- rimidinyl) phosphorothioate |
| Dicofol | 1,1-Bis (chlorophenyl)-2,2,2-trichloroethanol |
| Disflubenzuron | 1-(4-chlorophenyl)3-(2,6 difluorobenzoyl) urea |
| Dimethoate | O,O Dimethyl S-(N methylcarbamoylmethyl) phosphorodithioate |
| Disulfoton | O,O-Diethyl S-[2-(ethylthio) ethyl] phos- phorodithioate |
| Endosulfan | 6,7,8,9,10,10-Hexachloro-1,5,5a,6,9,9a-hexa- hydro-6,9-methano-2,4,3-benzodioxathiepin -3-oxide |
| Fenamiphos | Ethyl 3-methyl-4-(methylthio) phenyl (1-meth- ylethyl) phosphoramidate |
| Fensulfothion | O,O-Diethyl O-[4-(methylsulfinyl)phenyl] phos- phorothioate |
| Fenitrothion | O,O-Dimethyl O-4-nitro-m-tolyl phosphoro- thioate (IUPAC) |
| Fenvalerate | (RS)- α -Cyano-3-phenoxybenzyl (RS)-2-(4- Chlorophenyl)-3-methylbutyrate |

| Official common name | Chemical formula |
|----------------------|---|
| Fonofos | O-Ethyl-S-phenylethylphosphonodithioate |
| Malathion | O,O-dimethyl phosphorodithioate of diethyl mercaptosuccinate |
| Mephosfolan | 2-(diethoxyphosphinylimino)-4-methyl-1,3-dithiolane |
| Metaldehyde | Metacetaldehyde |
| Metasystox (i) | S-[2(ethylthio)ethyl] O,O-dimethylphosphorothioate |
| Methamidophos | O,S-Dimethyl phosphoramidodithioate |
| Methiocarb | 3,5-Dimethyl-4-(methylthio)phenyl methylcarbamate |
| Methomyl | S-Methyl-N-((methylcarbamoyl)oxy)-thioacetimidate |
| Methyl parathion | O,O-dimethyl-O-4-nitrophenyl phosphorothioate |
| Monocrotophos | dimethyl-(E)-1-methyl-2-methylcarbamoyl-vinyl phosphate |
| Nicotine | 3-(1-Methyl-2-pyrrolidyl)pyridine |
| Omethoate | O,O-dimethyl S-[2-(methylamino)-2-oxoethyl] phosphorothioate |
| Oxamyl | Methyl N'N'-dimethyl-N-[(methylcarbamoyl)oxy]-1-thiooxamimidate |
| Parathion | O,O-diethyl O-4-nitrophenyl phosphorothioate |
| Permethrin | 3-(phenoxybenzyl)(1RS, 3RS: 1RS,3SR)-3-(2,2-dichlorovinyl)-2,2-dimethylcyclopropane carboxylate |
| Phorate | O,O-Diethyl S-{(ethylthio) methyl}phosphorodithioate |
| Pirimicarb | 2-Dimethylamino-5,6-dimethylpyrimidin-4-yl dimethylcarbamate |
| Pirimiphos-methyl | O-(2-Diethylamino-6-methylpyrimidin-4-yl) O,O-dimethyl phosphorothioate |
| Pyrethrum | Six related esters: pyrethrins I and II, cinerins I and II, and jasmolins I and II |
| Tetradifon | 4-chlorophenyl 2,4,5-trichlorophenyl sulfone |
| Thiocarboxime | 1-(2-Cyanoethylthio)ethyldeneamino N-methylcarbamate |
| Triazophos | 1-phenyl-1.2.4-triazolyl-3-(O.O-diethylthionophosphate) (IUPAC) |
| Trichlorfon | Dimethyl (2,2,2-trichloro-1-hydroxyethyl) phosphonate |

Appendix II. Taxonomic Classification and Common Names of Various Host Plants Cited for *Phaseolus Vigna* Genera

| Scientific name with common names | Synonym |
|---|--|
| <i>Arachis hypogaea</i> L. Groundnut Peanut | |
| <i>Cajanus cajan</i> (L.) Millsp. Pigeonpea | <i>Cajanus indicus</i> Spreng. |
| <i>Glycine max</i> (L.) Merrill Soybean | |
| <i>Lablab purpureus</i> (L.) Sweet Hyacinth bean | <i>Dolichos lablab</i> L. <i>Lablab niger</i> Med. |
| <i>Lens culinaris</i> Med. Lentil | <i>Lens esculenta</i> Moench. |
| <i>Macroptilium atropurpureum</i> (DC.) Urb. Purple bean Siratro | <i>Phaseolus atropurpureus</i> DC. <i>P. dysophyllus</i> Benth. |
| <i>M. bracteatum</i> (Nees ex Mart.) Maréchal et Baudet | <i>Phaseolus bracteatus</i> Nees ex Mart. |
| <i>M. erythroloma</i> (Benth.) Urb. | <i>Phaseolus erythroloma</i> Mart. ex Benth. |
| <i>M. lathyroides</i> (L.) Urb. Phasemy bean | <i>Phaseolus lathyroides</i> L. |
| <i>M. longepedunculatum</i> (Benth.) Urban | <i>Phaseolus longepedunculatus</i> Mart. ex Benth. |
| <i>P. acutifolius</i> A. Gray var. <i>acutifolius</i> Tepary bean | <i>Phaseolus acutifolius</i> A. Gray var. <i>latifolius</i> Freeman |
| <i>P. coccineus</i> L. Scarlet runner bean | |

| Scientific name with common names | Synonym |
|--|---|
| <i>P. coccineus</i> ssp. <i>obvallatus</i> (Schlecht.) M.M.S. Wild <i>P. coccineus</i> | |
| <i>P. filiformis</i> Bentham | |
| <i>P. leptostachyus</i> Bentham | <i>Phaseolus anisotrichus</i> Schlecht. |
| <i>P. lunatus</i> L. Lima bean | <i>Phaseolus limensis</i> Macfady |
| <i>P. maculatus</i> Scheele | <i>Phaseolus retusus</i> Benth. |
| <i>P. polyanthus</i> Greenman | |
| <i>P. polystachyus</i> (L.) B.S.P. | |
| <i>P. polystachyus</i> var. <i>sinuatus</i> (Nutt) M.M.S. | <i>Phaseolus sinuatus</i> Nutt ex Torr. & Gray |
| <i>P. vulgaris</i> L. Common bean Dry bean | |
| <i>Phaseolus vulgaris</i> var. <i>aborigineus</i> (Burk.) Baudet | <i>Phaseolus aborigineus</i> Burk. <i>P. aborigineus</i> Burk. var. <i>hondurensis</i> Burk. |
| <i>Pisum sativum</i> L. Pea | |
| <i>Pueraria lobata</i> (Willd.) Ohwi Kudzi vine | <i>Pueraria hirsuta</i> (Thunb.) C. K. Schneid. <i>P. thunbergiana</i> (Siebold et Zucc.) Benth. |
| <i>Vigna aconitifolia</i> (Jacq.) Maréchal Moth bean | <i>Phaseolus aconitifolius</i> Jacq. |
| <i>V. adenantha</i> (G. F. Meyer) M.M.S. | <i>Phaseolus adenanthus</i> G. F. Meyer |
| <i>V. angularis</i> (Willd.) Ohwi et Ohashi Adzuki bean | <i>Phaseolus angularis</i> (Willd.) W. F. Wright |

| Scientific name with common names | Synonym |
|--|--|
| <i>V. longifolia</i> (Benth.) Verdcourt | <i>Phaseolus trichocarpus</i> C. Wright |
| <i>V. luteola</i> (Jacq.) Bentham | <i>Vigna repens</i> (L.) Kuntze |
| <i>V. mungo</i> (L.) Hepper Black gram Urd bean | <i>Phaseolus mungo</i> L. |
| <i>V. radiata</i> (L.) Wilczek var. <i>radiata</i> Golden gram Green gram Mung bean | <i>Phaseolus aureus</i> Roxb. <i>P. radiatus</i> L. |
| <i>V. radiata</i> var. <i>sublobata</i> (Roxb.) Verdc. | <i>Phaseolus trinervius</i> Wight et Arn. |
| <i>V. umbellata</i> (Thunb.) Ohwi et Ohashi Rice bean | <i>Phaseolus calcaratus</i> Roxb. <i>P. riccardianus</i> Tenore |
| <i>V. unguiculata</i> (L.) Walpers ssp. <i>unguiculata</i> Common cowpea Cowpea | <i>Vigna sinensis</i> Savi ex Hassk. |
| <i>V. unguiculata</i> ssp. <i>unguiculata</i> var. <i>sesquipedalis</i> (L.) Verdc. | <i>Vigna sesquipedalis</i> (L.) Fruhw. |
| <i>V. vexillata</i> (L.) A. Rich. | |

Appendix III. Acronyms and Abbreviations Used in Text

| | |
|------|-----------------------|
| AbMV | Abutilon mosaic virus |
| AG | Anastomosis groups |
| ALS | Angular leaf spot |
| AMV | Alfalfa mosaic virus |
| ANT | Bean anthracnose |

| | |
|-----------------|--|
| ARS/USDA | Agriculture Research Service of the United States Department of Agriculture |
| ASC | Ascochyta blight |
| ASU | Agroecological Studies Unit of CIAT |
| AVRDC | Asian Vegetable Research and Development Center, Taiwan |
| BALSIT | Bean Angular Leaf Spot International Test |
| BCDMV | Bean curly dwarf mosaic virus |
| BCIMV | Bean chlorotic mottle virus |
| BCMV | Bean common mosaic virus |
| BCTV | Beet curly top virus |
| BGMV | Bean golden mosaic virus |
| BMMV | Bean mild mosaic virus |
| BPMV | Bean pod mottle virus |
| BRMV | Bean rugose mosaic virus |
| BSMV | Bean southern mosaic virus |
| BYMV | Bean yellow mosaic virus |
| BYSV | Bean yellow stipple virus |
| CBB | Common bacterial blight |
| CCMV | Cowpea chlorotic mottle virus |
| CEC | Cation exchange capacity |
| cfu | Colony-forming units |
| CIAT | Centro Internacional de Agricultura Tropical, Colombia |
| CLRV | Cherry leafroll virus |
| CMi | Commonwealth Mycological Institute, United Kingdom |
| CMMV | Cowpea mild mottle virus |
| CMV | Cucumber mosaic virus |

| | |
|--------------|---|
| DNA | Deoxyribonucleic acid |
| ELISA | Enzyme-linked immunosorbent assay |
| EMV | Euphorbia mosaic virus |
| FAO | Food and Agriculture Organization of the United Nations, Italy |
| HB | Halo blight |
| ICA | Instituto Colombiano Agropecuario, Colombia |
| ICTA | Instituto de Ciencia y Tecnología Agrícolas, Guatemala |
| IPM | Integrated pest management |
| ISNAR | International Service for National Agricultural Research, Netherlands |
| meq | milliequivalent |
| mho | unit of electrical conductance |
| MLOs | Mycoplasma-like microorganisms |
| NVRS | National Vegetable Research Station, United Kingdom |
| PAN | Peroxyacetyl nitrate |
| PDA | Potato dextrose agar |
| RMV | Rhynchosia mosaic virus |
| RNA | Ribonucleic acid |
| RR | Root rots |
| SBMV | Southern bean mosaic virus |
| SMV | Soybean mosaic virus |
| UV | Ultraviolet |
| WB | Web blight |
| XCP | <i>Xanthomonas campestris</i> pv. <i>phaseoli</i> (Smith) Dye. Also known as common blight bacterium |
| YDC | Yeast-extract-dextrose calcium carbonate agar |

Appendix IV. Metric Conversion Tables for Measurement Units Cited in Text

Metric to Non-metric Units

Temperature

Degrees Centigrade = $(F^{\circ} - 32)/1.8$

Length and Area

1 centimeter = 0.39 inches
1 meter = 3.28 feet
1 kilometer = 0.62 mile
1 square meter = 10.76 square feet
1 hectare = 2.47 acres

Weight

1 gram = 0.04 ounces
1 kilogram = 2.21 pounds
1 metric ton = 1.10 tons

Volume

1 cubic centimeter = 0.03 fluid ounces
(ml)
1 liter = 0.26 gallons
1 gram/liter = 0.13 ounces/gallon
1 milliliter/liter = 0.13 fl. ounces/gallon
1 kilogram/hectare = 0.89 pounds/acre
1 liter/hectare = 0.11 gallons/acre

Non-metric to Metric Units

Degrees Farenheit = $(C^{\circ} \times 1.8) + 32^{\circ}F$

1 inch = 2.54 centimeters
1 foot = 0.31 meters
1 mile = 1.6 kilometers
1 square foot = 0.09 square meters
1 acre = 0.41 hectares

1 ounce = 28.35 grams
1 pound = 0.45 kilograms
1 ton = 0.91 metric ton
1 pound/square inch = 70.3 g/square
centimeter

1 fluid ounce = 29.57 cubic
centimeters (ml)
1 gallon = 3.79 liters
1 ounce/gallon = 7.49 grams/liter
1 ounce (fl.)/gallon = 7.81 milliliters/liter
1 pound/acre = 1.12 kilograms/hectare
1 gallon/acre = 9.35 liters/hectare

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 (μm) = 1×10^{-4} centimeter = 3.94×10^{-5} inch

1 dalton = 1/16 of an oxygen atom = about 1.65×10^{-24} g

1 lux = 1 lumen/square meter

SI Prefixes Used in Text

m = milli = 10^{-3}

μ = micro = 10^{-6}

n = nano = 10^{-9}

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Bean Production Problems in the Tropics

Color Plates

COLOR PLATES

Chapter 2



Figure 1. Climbing beans growing in association with banana in Rwanda.



Figure 2. Seeds grown from farmers' bean varietal mixture in Rwanda.



Figure 3. Bean stems and pod damaged by scab.

Chapter 4



Figure 4. Typical lesion development and accompanying chlorosis caused by *Phaeoisariopsis griseola* infection of bean leaves.



Figure 5. Infection by angular leaf spot fungus in pod (left) and stems (right).

**Figure 6. Synnemata
production on lower
surface of bean leaf.**



Figure 7. Bean plant infection from previously infested bean debris.

Chapter 5



Figure 8. Initial anthracnose lesions on veinlets of lower leaf surface.



Figure 9. Anthracnose symptoms on upper leaf surface and petiole.



Figure 10. Sporulating pod lesions caused by anthracnose infection.

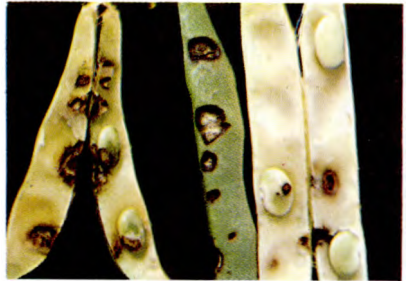


Figure 11. Mycelial development of the anthracnose fungus within the developing bean pod.



Figure 12. Seed infection by anthracnose.



Figure 13. Resistant (left) and susceptible (right) bean germplasm.

Chapter 6



Figure 14. A field suffering severe root-rot damage.



Figure 15. Young lesions caused by *Rhizoctonia solani*.



Figure 16. Hypocotyl cankers produced by the rhizoctonia root-rot fungus.



Figure 17. Old cankers and pith infection caused by *Rhizoctonia solani*.



Figure 18. Root and hypocotyl lesions caused by *Fusarium solani* infection.



Figure 19. *Fusarium* root-rot symptoms start behind lateral roots in bean.

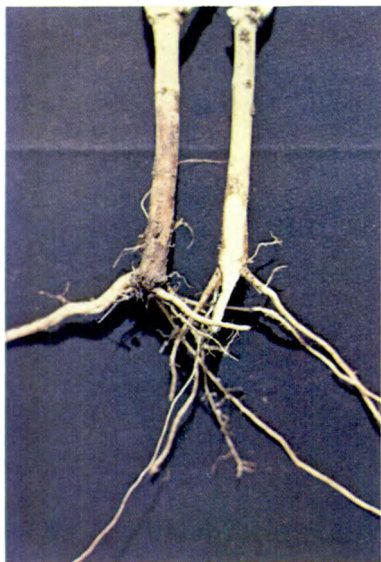


Figure 20. Root and hypocotyl infection by *Fusarium oxysporum*.



Figure 21. A severely infected plant showing diagnostic pinkish orange spore masses of *Fusarium oxysporum*.



Figure 22. Leaf yellowing caused by *Fusarium oxysporum*.



Figure 23. Seed decay after infection from *Pythium ultimum*.



Figure 24. *Pythium* root-rot symptoms on infected plants (left) and healthy plants (right).



Figure 25. Sunken lesions caused by *pythium* root rot.



Figure 26. Postemergence damping-off caused by *Pythium* species.



Figure 27. *Pythium* damage to feeder roots. Healthy root on left and damaged root on right.

Figure 28. Hypocotyl and root lesions and sclerotia produced by *Sclerotium rolfsii*.





Figure 29. Symptoms of root infection by *Thielaviopsis basicola*.



Figure 30. Symptomatic streaks of *Aphanomyces* infection spread up the stems.

Chapter 7



Figure 31. Pycnia of bean rust, *Uromyces appendiculatus*, on upper leaf surface.



Figure 32. Aecia of bean rust, *Uromyces appendiculatus*, on lower leaf surface.



Figure 33. Rust uredia on susceptible bean leaf.



Figure 34. Rust uredia on susceptible bean pod.

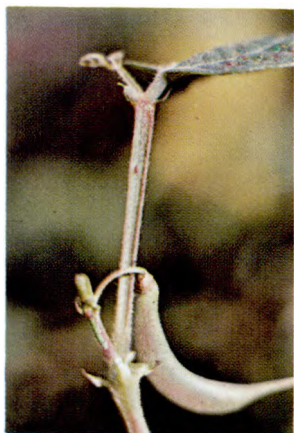


Figure 35. Rust uredium on susceptible bean petiole.



Figure 36. Immature rust uredia 5 to 6 days after infection of susceptible bean leaf.



Figure 37. Rust telia and teliospores on maturing bean leaf.



Figure 38. Interaction between bean rust and anthracnose fungi observed in the field.



Figure 39. Necrotic ring development around bean rust uredia caused by interaction with unidentified virus.

Figure 40. Rust-resistant bean cultivar on left; susceptible cultivar on right.



Chapter 8



Figure 41. Old leaf lesions caused by web blight fungus.



Figure 42. Pod infection by web blight fungus.



Figure 43. Plant severely infected by web blight fungus during a natural epidemic.



Figure 44. Microsclerotia produced on infected leaf tissue.



Figure 45. Initial leaf infections caused by basidiospores and mycelia of the web blight fungus.

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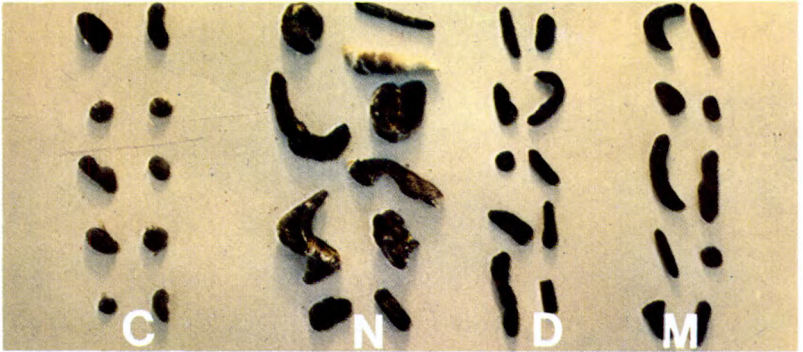


Figure 46. Sclerotial forms produced by the white mold fungus. C = culture produced; N = unconditioned and naturally produced; D = dry conditions; M = moist conditions.



Figure 47. Apothecia produced in field from germinated sclerotium.



Figure 48. Bean blossoms colonized by ascospores of *Sclerotinia sclerotiorum*.



Figure 49. Watery soft rot and sclerotia production in bean pod infected by white mold fungus.



Figure 50. Mycelia and sclerotia production on infected bean pod.



Figure 51. White or bleached symptom of bean plant severely infected by white mold fungus.



Figure 52. Canopy wilt caused by white mold infection of bean vegetation.

Chapter 10

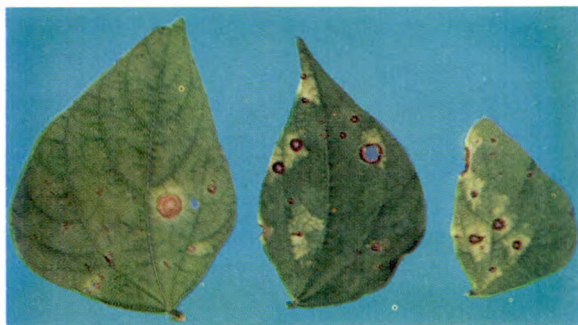


Figure 53. Leaf lesions caused by *Alternaria* infection.



Figure 54. Blemish on bean leaf caused by *Alternaria* species.



Figure 55. Blemish on bean pods caused by *Alternaria tenuis*.



Figure 56. Upper and lower leaf surface lesions caused by *Ascochyta* spp.



Figure 57. Petiole and pod lesions caused by *Ascochyta* spp.



Figure 58. Old pod lesions caused by *Ascochyta* spp.



Figure 59. Seedling infection produced by *Macrophomina phaseolina*.



Figure 60. Initial infection by ashy stem blight fungus on one side of plant.



Figure 62. Pycnidia of *Macrophomina phaseolina* on infected bean stem.

Figure 61. Sclerotia of *Macrophomina phaseolina* on infected bean stem.

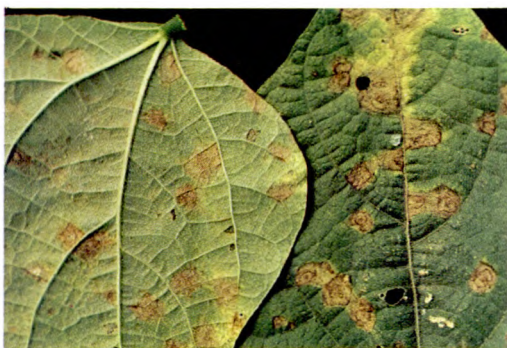


Figure 63. Lesions on infected bean leaves caused by *Cercospora* spp.

Figure 64. Leaf lesions caused by chaetoseptoria leaf spot.





Figure 65. Pod infection caused by *Phytophthora* species.

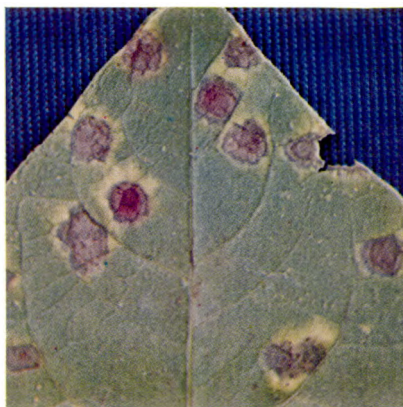


Figure 66. Leaf lesions caused by *Entyloma* species.



Figure 67. Lower leaf lesions caused by *Ramularia phaseoli*.



Figure 68. Upper leaf lesions caused by *Cercospora vanderysti*.



Figure 69. Mycelium and spores produced on lower leaf surface by gray leaf spot.



Figure 70. Severe plant infection by the gray leaf spot fungus.



Figure 71. Apothecium and conidia produced by *Botryotinia fuckeliana*.



Figure 72. Pod infection by gray mold.



Figure 73. Powdery mildew lesions on bean leaf.



Figure 74. Severe plant infection by *Erysiphe polygoni*.



Figure 75. Pod infection by *Erysiphe polygoni*.



Figure 76. Leaf lesions caused by white spot fungus.



Figure 77. Mixed leaf infection by gray and white spot fungi.

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Figure 78. Water-soaked spots caused by leaf infection of common and fuscous blights.



Figure 79. Common blight lesions, showing lemon-yellow and necrotic symptoms. .



Figure 80. Severe foliage infection by common bacterial blight.



Figure 81. Stem girdle and breakage caused by common bacterial blight.

Figure 82. Pod and seed infection by common bacterial blight.

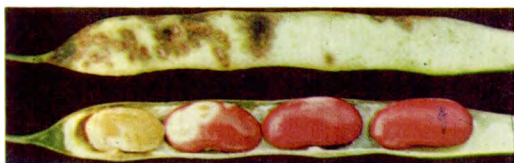


Figure 83. Variation shown by *Phaseolus vulgaris* germplasm for its resistance to infection by common blight bacteria (susceptible left, resistant right).

Chapter 12



Figure 84. Symptoms of halo blight on leaves.



Figure 85. Severe plant infection during a halo blight epidemic.



Figure 86. Greasy spot symptom produced by halo blight on pods.



Figure 87. Bacterial exudate
produced by
Pseudomonas syringae
pv. *phaseolicola*.



Figure 88. Systemic plant
chlorosis caused by
halo blight.

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Figure 89. Seed discoloration caused by different pathogenic strains of the bacterial wilt organism.



Figure 90. Bacterial wilt in bean plants.



Figure 91. Bacterial wilt in bean vascular system.

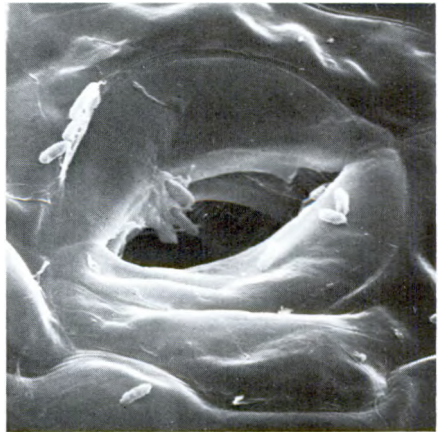


Figure 92. Scanning electron microscope photograph of *Pseudomonas syringae* cells on the edge of a plant stomata (X 5000).



Figure 93. Bacterial brown spot lesions on bean foliage.



Figure 94. Bacterial brown spot lesions on bean pods.



Figure 95. Symptoms of bacterial wildfire on bean foliage.

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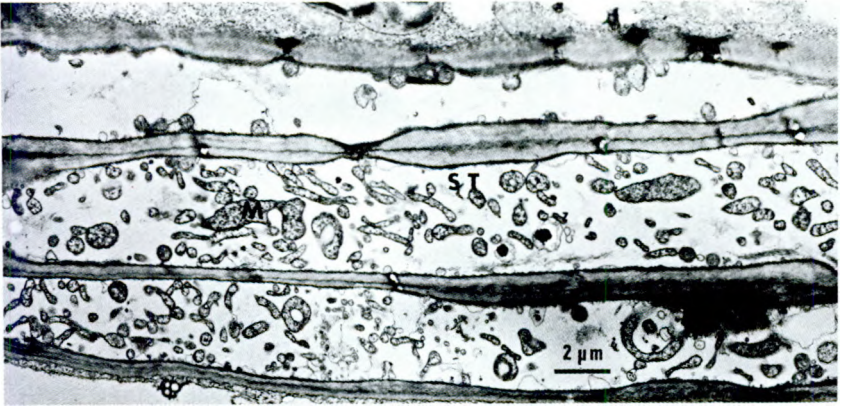


Figure 96. Electron microphotograph of longitudinal section of bean sieve tubes (ST) containing numerous pleomorphic corpuscles (M).



Figure 97. Electron microphotograph of mycoplasma-like corpuscles (M) showing absence of cell wall and presence of plastids (P).



Figure 98. Leafhopper vector (*Scaphytopius fuliginosus*) of bean mycoplasma-like organism in Colombia.



Figure 99. Phyllody caused by mycoplasma infection of bean.



Figure 100. Pod deformation caused by bean mycoplasma infection.



Figure 101. Leaf and petiole deformation caused by bean mycoplasma infection.



Figure 102. Witches' broom symptom in infected plant.



Figure 103. Premature germination of bean seeds in immature pod.



Figure 104. Mycoplasma symptoms in soybean.



Figure 105. *Crotalaria juncea* with symptoms of "machismo."

Chapter 15



Figure 106. Curling, stunting, and malformation of leaves infected by bean common mosaic virus (BCMV).



Figure 107. Leaf mosaic symptoms induced by BCMV infection.



Figure 108. Leaf curling and malformation induced by BCMV infection.

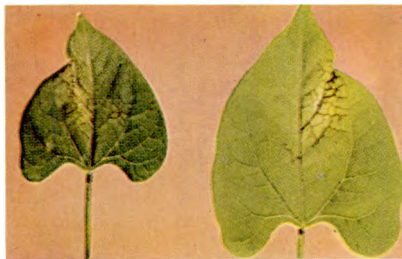


Figure 109. Initial leaf symptoms of black-root reaction induced by BCMV.



Figure 110. Plant wilting and systemic necrosis symptoms of black root.

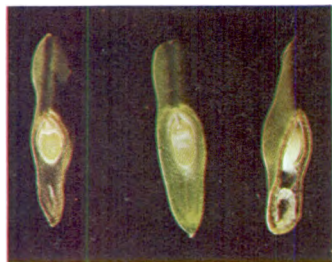


Figure 111. Black-root-induced necrosis in vascular system of bean pods.

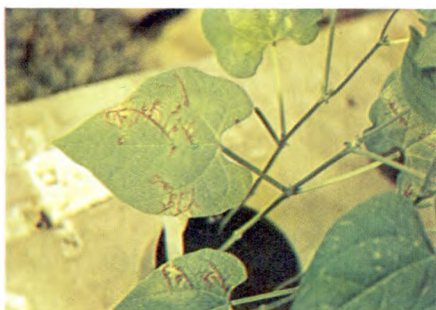


Figure 112. Local lesions produced by BCMV in inoculated bean leaves.



Figure 113. Cytoplasmic inclusions or pinwheels (X 25,000) produced by BCMV.



Figure 114. Winged aphid adults such as these, may act as virus vectors.



Figure 115. Chlorotic leaf symptoms caused by bean yellow mosaic virus (BYMV) infection.



Figure 116. Leaf malformation induced by BYMV infection.

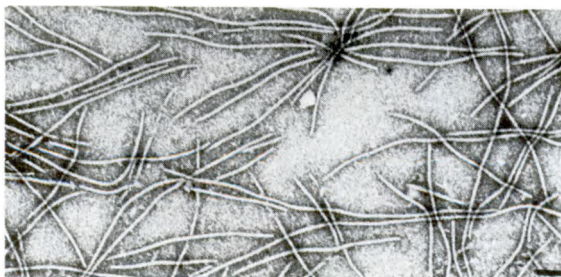


Figure 117. Filamentous particles of BYMV.



Figure 118. Leaf symptoms of cucumber mosaic virus in infected cucumber plants.

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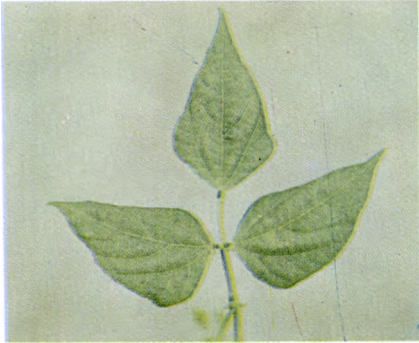


Figure 119. Light chlorosis and leaf curling induced in leaves of the bean cultivar Diacol Calima inoculated with bean southern mosaic virus.

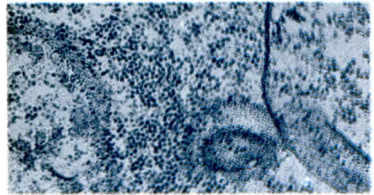


Figure 120. Electron microscope photograph of the isometric particles of bean southern mosaic virus (X 15,000).



Figure 121. Leaf symptoms induced by bean mild mosaic virus infection of the bean cultivar Porrillo 1.



Figure 122. Leaf blisters and malformation induced by bean rugose mosaic virus infection.



Figure 123. Adult beetle of *Diabrotica balteata*.

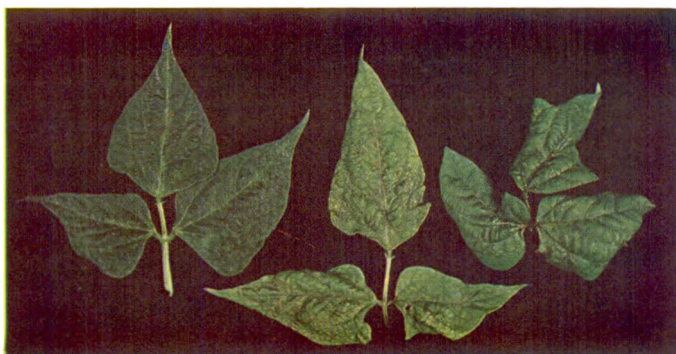


Figure 124. Variation in leaf symptoms induced by bean curly dwarf mosaic virus infection of bean cultivars 27 R, Porrillo 1, and El Salvador 184 (left to right).



Figure 125. Plant and leaf symptoms induced in the bean cultivar Porrillo 1 by a mixed inoculation with bean curly dwarf mosaic and bean mild mosaic viruses.

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Figure 126. Beans infected by bean golden mosaic virus.

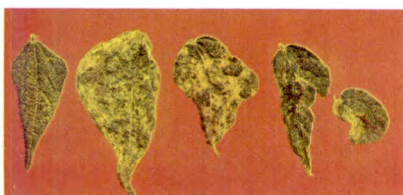


Figure 127. Mosaic symptoms and malformation induced by bean golden mosaic virus infection in bean leaves.



Figure 128. Malformation induced by bean golden mosaic virus infection in bean pods.

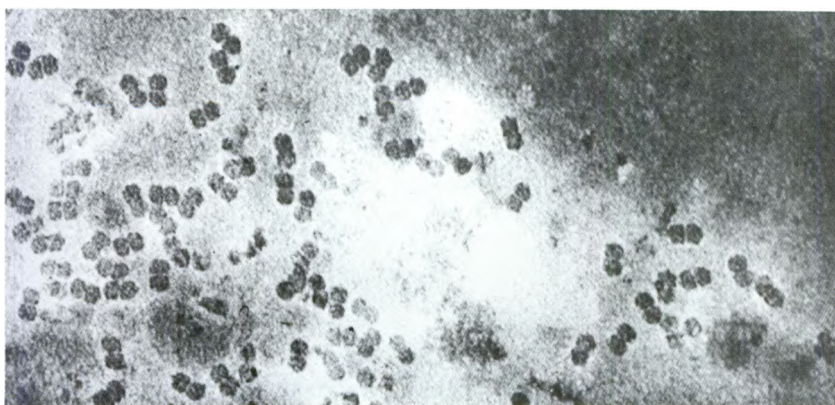


Figure 129. Geminate particles of bean golden mosaic virus (X 160,000).

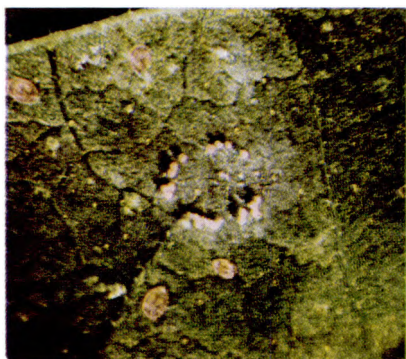


Figure 130. Eggs and immature forms of the whitefly *Bemisia tabaci* on the lower surface of a leaf.

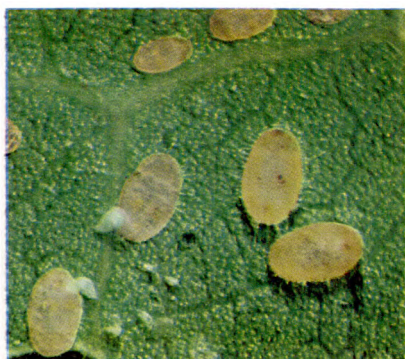


Figure 131. Immature forms of the whitefly *Bemisia tabaci*.



Figure 132. The adult whitefly (*Bemisia tabaci*), vector of bean golden mosaic virus.



Figure 133. Bean golden mosaic virus screening nursery in the Dominican Republic.



Figure 134. Plant stunting and witches' broom induced by bean dwarf mosaic virus.



Figure 135. Chlorotic mottle symptoms on leaves infected by bean dwarf mosaic virus.



Figure 136. Leaf rugosity, suspected to be induced by bean dwarf mosaic virus.



Figure 137. Chlorotic mottling induced by abutilon mosaic virus infection of *Pavonia sidaefolia*.



Figure 138. Symptoms of infectious chlorosis of Malvaceae in a *Malva* sp. plant.



Figure 139. Leaf wrinkling and chlorosis of a *Euphorbia* sp. plant infected with euphorbia mosaic virus.



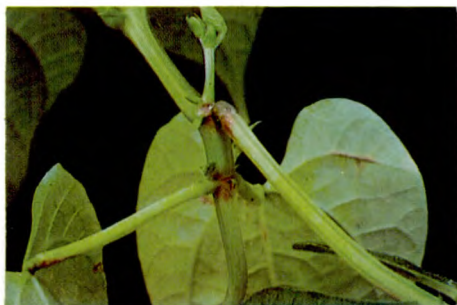
Figure 140. Bean leaves infected with rhynchosia mosaic virus.

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Figure 141. Bean plant infected with beet curly top virus.

Figure 142. Bean plant, showing red-node symptom after infection by tobacco streak virus.



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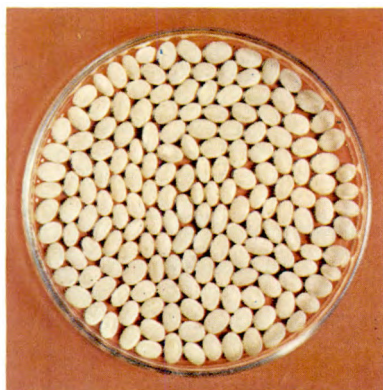


Figure 143. Sample of seed relatively free of seed-borne organisms.

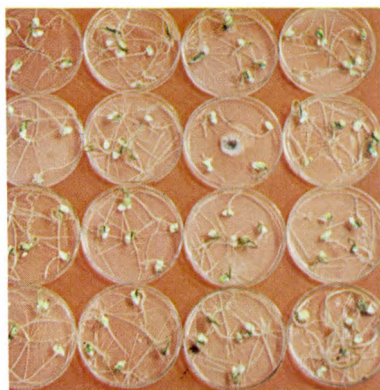


Figure 144. Seed from clean-seed sample that was surface-disinfected and incubated on potato-dextrose agar.



Figure 145. Sample of seed severely contaminated by seed-borne organisms.



Figure 146. Seed from contaminated-seed sample that was surface-disinfected and incubated on potato-dextrose agar.



Figure 147. Pod and seeds infected by anthracnose fungus.



Figure 148. Seed sample that was harvested at maturity, surface-disinfected, and incubated on potato-dextrose agar.



Figure 149. Seed sample that was harvested 2 weeks after maturity, surface-disinfected, and incubated on potato-dextrose agar.



Figure 150. Seed infected by *Sclerotium rolfsii*.

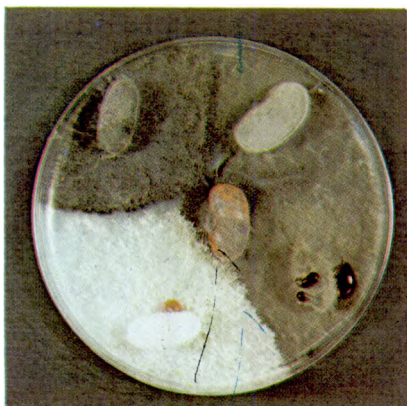


Figure 151. Seed infected by *Macrophomina phaseolina* (black mycelia) and *Phomopsis* species (white mycelia).

Chapter 20



Figure 152. Adult female and egg mass of *Meloidogyne* species.

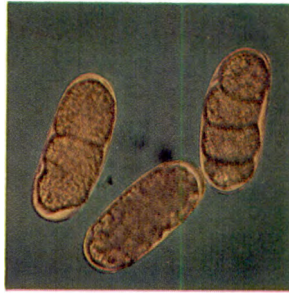


Figure 153. Egg containing developing larvae of *Meloidogyne* species.



Figure 154. Young larva of *Meloidogyne* species.



Figure 155. Adult female of *Meloidogyne incognita*.



Figure 156. Mature female root-lesion nematode (*Pratylenchus* sp.).



Figure 157. Plant chlorosis and stunting caused by infection from *Meloidogyne* species.



Figure 158. Root galls produced after infection by *Meloidogyne* species.

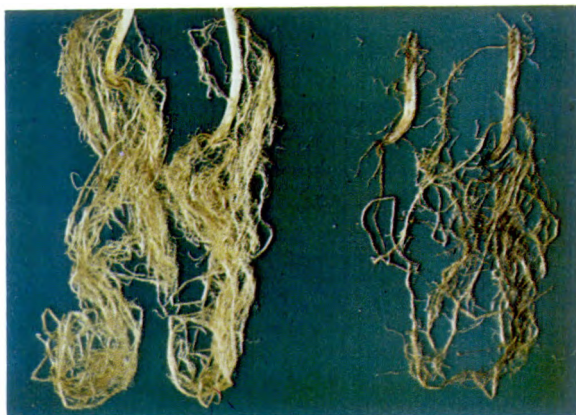


Figure 159. Root damage caused by *Pratylenchus scribneri* feeding.

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Figure 160. Adult bean fly (*Ophiomyia phaseoli*) on bean leaf.



Figure 161. Bean-fly pupae shown by cutting away the stem epidermis of bean plant.



Figure 162. Bean-fly damage on bean stem and hypocotyl.



Figure 163. Development of adventitious roots after damage by bean-fly larvae.



Figure 164. Bean seedling, withering and drying after being attacked by bean fly.



Figure 165. Bean leaf, showing ovipunctures made by larvae of the leafminer, *Liriomyza trifolii*.



Figure 166. Bean plants, showing serpentine tunnels made by larvae of the leafminer, *Liriomyza trifolii*.



Figure 167. Black bean aphids (*Aphis fabae*) on bean stems.



Figure 168. Cowpea aphid (*Aphis craccivora*) on bean pod, surrounded by nymphs.



Figure 169. Maggot of hover fly (*Xanthogramma aegyptius*) feeding on bean aphids.



Figure 170. Foliage beetle (*Ootheca bennigseni*) on bean leaf.



Figure 171. Typical foliar damage by *Ootheca* spp. on bean plant.



Figure 172. Striped foliage beetle (*Luperodes quaternus*) feeding on bean pods.



Figure 173. Striped bean weevil (*Aldidodes leucogrammus*) on the swollen stem of common bean.



Figure 174. Grub of striped bean weevil in swollen stem of common bean.



Figure 175. Flower beetle (*Mylabris tristigma*) devouring a flower of common bean.



Figure 176. Blister beetle (*Coryna kersteni*) feeding on bean flower.



Figure 177. Flower thrips (*Megalurothrips sjöstedti*) on flower.



Figure 178. Moth of legume pod borer (*Maruca testulalis*).

Figure 179. Pod and seed damage caused by the larvae of legume pod borer (*Maruca testulalis*).



Figure 180. Typical damage symptoms of *Heliothis armigera* larvae. Note the frass near the larva.

Figure 181. Larvae of American bollworm (*Heliothis armigera*) feeding on bean pods. Note the characteristic circular holes in the pods.

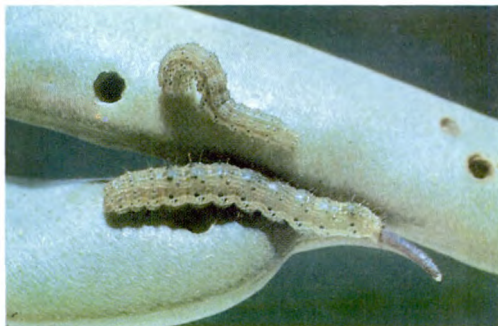




Figure 182. Bean pods damaged by the larvae of *Heliothis armigera*.



Figure 183. Adult of spiny bug (*Clavigralla tomentosicollis*).



Figure 184. A nymph of green stink bug (*Nezara viridula*).



Figure 185. Mating pair of green stink bug on common bean plant.



Figure 186. Snail (*Limicolaria kambeul*) feeding on bean leaves.

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Figure 187. White-grub larvae extracted from the base of infested plants.



Figure 188. Bean plant severed by a cutworm larva.



Figure 189. Typical cricket damage on a bean plant.



Figure 190. Larvae of seedcorn maggot (*Hylemya cilicura*) on bean seedling.



Figure 191. Damage caused by seedcorn maggot (*Hylemya cilicura*) on bean seedlings.



Figure 193. Damage caused by lesser cornstalk borer (*Elasmopalpus lignosellus*).



Figure 192. Mature larva of the lesser cornstalk borer (*Elasmopalpus lignosellus*).



Figure 194. Adult *Diabrotica balteata*.

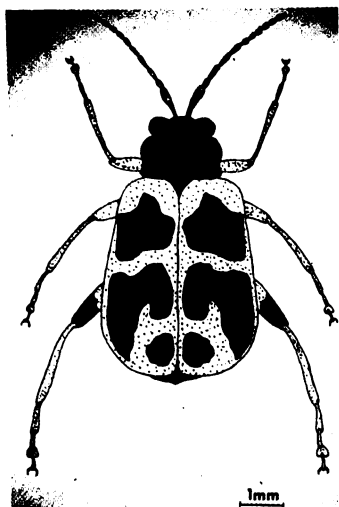


Figure 195. Adult *Cerotoma facialis*.



Figure 196. Severe damage by adult chrysomelids.



Figure 197. Damage by larva of *Cerotoma facialis* on bean hypocotyl.



Figure 198. Mature larva of Mexican bean beetle (*Epilachna varivestis*).



Figure 199. Adult Mexican bean beetle (*Epilachna varivestis*) on lower surface of a bean leaf.



Figure 200. Bean leaf folded by young larva of the bean leafroller (*Urbanus proteus*).



Figure 201. Mature larva of bean leafroller (*Urbanus proteus*).



Figure 202. Young larvae of the saltmarsh caterpillar (*Estigmene acrea*) clustered on a bean leaf.



Figure 203. Mature larva of saltmarsh caterpillar (*Estigmene acrea*).

Figure 204. Mature larva of *Omiodes indicata*.





Figure 205. Typical damage by *Omiodes indicata*.



Figure 206. Pupa of *Omiodes indicata* among leaves woven together by the larva.



Figure 207. Leaves woven together by larvae of *Omiodes indicata*.



Figure 208. Leafminer (*Liriomyza sativae*) damage on bean leaf.

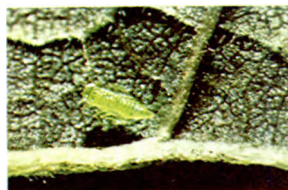


Figure 209. Nymph of leafhopper (*Empoasca kraemeri*).



Figure 210. Adults of leafhopper (*Empoasca kraemeri*).



Figure 211. Typical leafhopper damage characterized by curling and yellowing of leaves.

Figure 212. Bean plants susceptible (left) and resistant (right) to *Empoasca kraemeri*.





Figure 213. Eggs of whitefly (*Bemisia tabaci*).

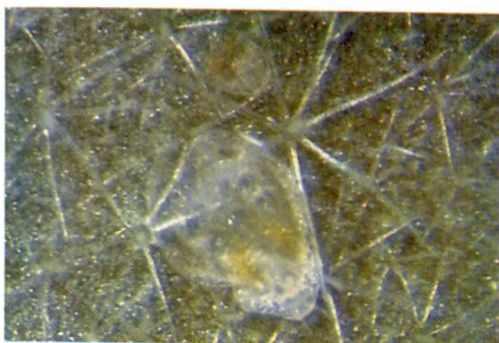


Figure 214. Pupa of *Bemisia tabaci*.



Figure 215. Adults of *Bemisia tabaci*.



Figure 216. Thrips damage on young bean plant.



Figure 217. Adult of *Acrosternum marginatum* (center) and immature (above).



Figure 218. Adult bean-pod weevil (*Apion godmani*).



Figure 219. Hyperplastic deformations caused by ovipositing females of *Apion godmani*.



Figure 220. Damage caused by larva of *Apion godmani* in bean pod.



Figure 221. Severe damage by *Heliothis* sp.

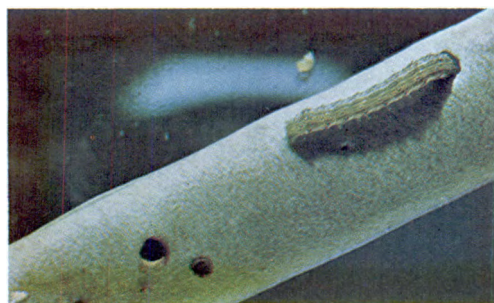


Figure 222. Larva of *Heliothis* sp. entering a bean pod.



Figure 223. Bud deformation caused by larval feeding of *Epinotia* sp.



Figure 224. Larva of *Maruca testulalis*.



Figure 225. Adults of *Zabrotes subfasciatus*. Note fresh eggs glued to the testa.



Figure 226. Adult *Acanthoscelides obiectus*.



Figure 227. Pupal cells of *Zabrotes subfasciatus*. Note hatched eggs attached to the testa.



Figure 228. Bean seeds destroyed by bruchids.



Figure 229. Adult slug.



Figure 230. Leaf damage from slug feeding.

Figure 231. Leafrolling caused by tropical spider mite.



Figure 232. Discoloration of bean pods caused by tropical spider mite.

Figure 233. Damage caused by tetranychid mites on bean leaf.



Chapter 23



Figure 234. Extreme boron deficiency as manifested in the primary leaves. The seedling at right, below, is normal, with primary leaves that are unifoliate, cordiform, and opposite.



Figure 235. Characteristic symptoms of N, P, K, and Mg deficiencies in the primary leaves. The leaf on the far left is normal.

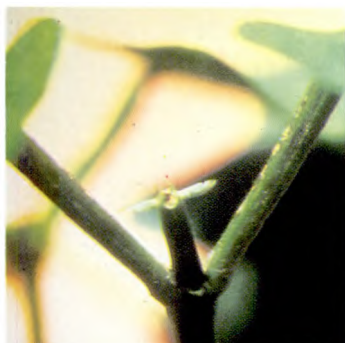


Figure 236. Symptom complex caused by boron deficiency.



Figure 237. Symptom complex caused by manganese toxicity.



Figure 238. Genotypic differences in reactions to aluminum toxicity.



Figure 239. Genotypic differences in reactions to phosphorus deficiency.



Figure 240. Symptom complex of phosphorus deficiency. In photo at right, roots on left are normal.

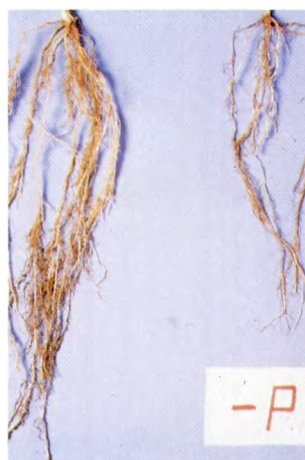




Figure 241. Symptoms caused by aluminum toxicity. Note the horizontal growth of roots.



Figure 242. The symptoms of manganese toxicity are most visible in the young leaves.

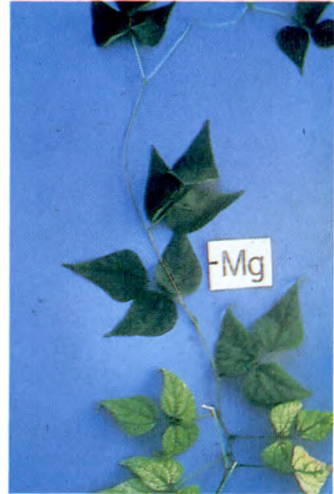


Figure 243. Magnesium deficiency. Note that symptoms appear initially in the lower leaves.



Figure 244. Symptom complex resulting from magnesium deficiency.



Figure 245. *Rhizobium* nodules on bean root.



Figure 246. Nitrogen deficiency. Note color and size differences between primary leaf stage and first and second trifoliate leaf stages. Normal leaves are on the right.

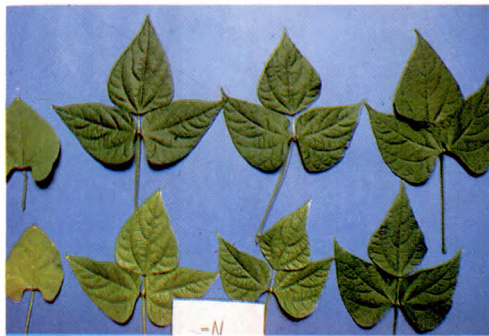


Figure 247. The symptoms of nitrogen deficiency are most intense in the primary leaves. In the foreground, affected primary, first, second, and third trifoliate leaves are shown. In the background are their normal counterparts.



Figure 248. Potassium deficiency. Note that the lower leaves are most affected.



Figure 249. Symptom complex caused by zinc deficiency.



Figure I. Symptoms caused by excess of salts.



Figure II. Other symptoms of phosphorus deficiency (see also Figure 240).



Figure 250. Symptom complex in young leaves, caused by iron deficiency.

Chapter 24

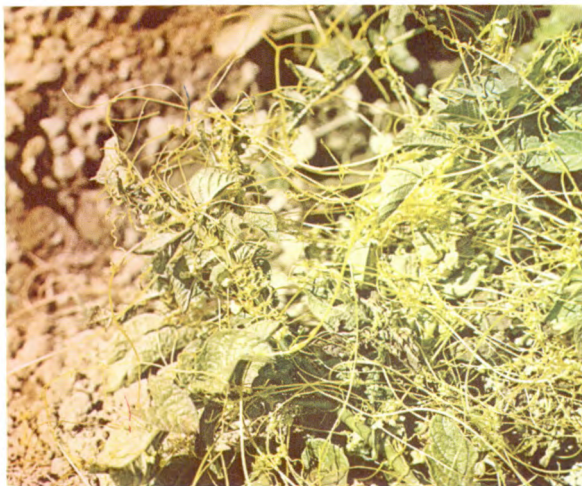


Figure 251. Dodder parasitizing potatoes.



Figure 252. Frost damage to climbing bean cultivar grown in association with maize.



Figure 253. Sunscald damage on bean pods.



Figure 254. Primary leaf damage caused by wind and airborne soil particles.



Figure 255. Baldhead symptoms induced by physically damaged seed.

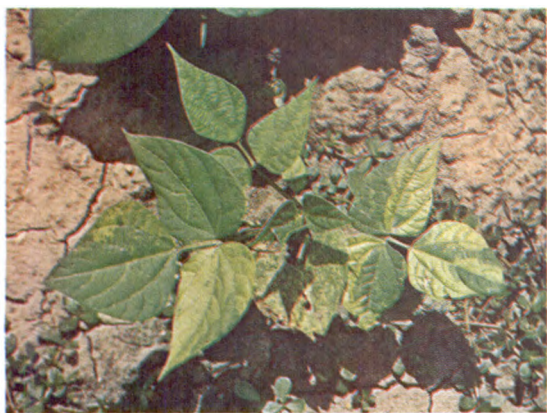


Figure 256. Leaf variegation caused by a genetic abnormality.



Figure 257. Insecticide damage to bean leaves.



Figure 258. Paraquat spray drift damage to beans.

Figure 259. Damage to beans caused by 2,4-D spray drift.

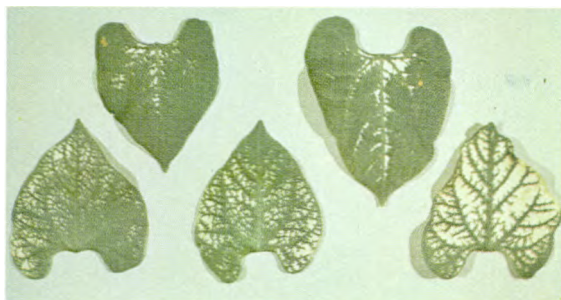
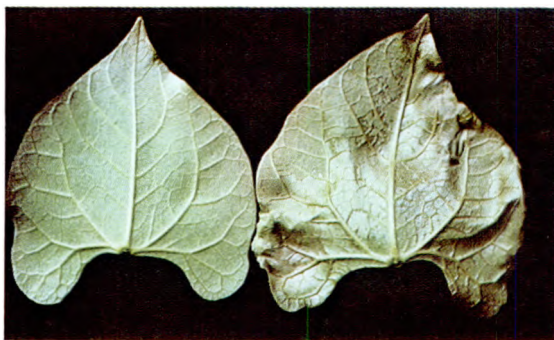


Figure 260. Flecking produced by ozone (50 pphm for 3 hr) on bean leaves.



Figure 261. Ozone damage (42 pphm for 1 hr) to bean leaves in shade (left) and in sun (right) at 22 °C.

**Figure 262. Peroxyacetyl
nitrate (PAN)
damage to
Pinto bean
leaf on right.**



**Figure 263. Sulfur
dioxide
damage (1
pphm for 1
hr) to Pinto
bean leaves.**

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