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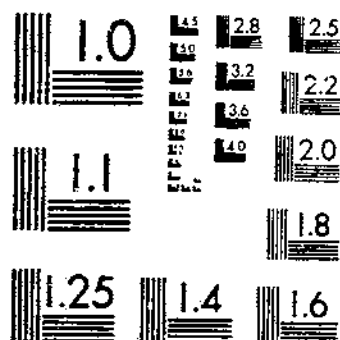
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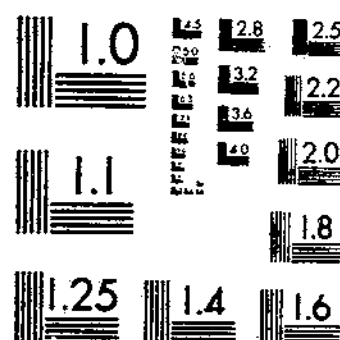
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PL 86-544 1957 USDA TECHNICAL BULLETINS 1400 DATA  
A MONOGRAPHIC STUDY OF BEAN DISEASES AND METHODS FOR THEIR CONTROL  
ZADOKS, H. J. THOMAS, JR. 1 OF 2

# START



MICROCOPY RESOLUTION TEST CHART  
NATIONAL BUREAU OF STANDARDS-1963-A



MICROCOPY RESOLUTION TEST CHART  
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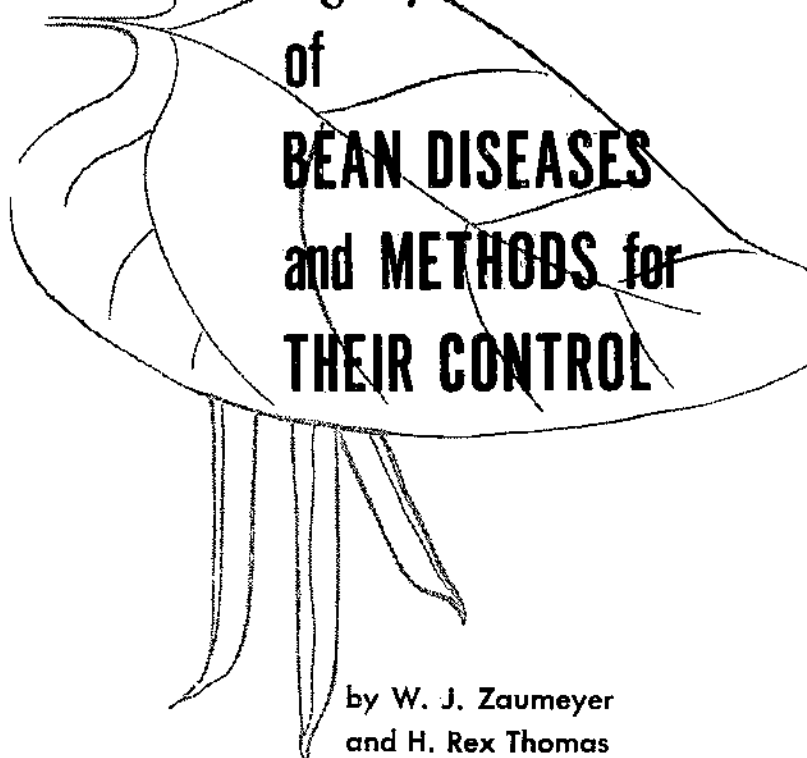
# A Monographic Study

of

**BEAN DISEASES**

and **METHODS for**

**THEIR CONTROL**



by W. J. Zaumeyer  
and H. Rex Thomas

**Technical Bulletin No. 868**

**UNITED STATES DEPARTMENT OF AGRICULTURE**

Revised February, 1957



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# A Monographic Study of Bean Diseases and Methods for Their Control<sup>1</sup>

By W. J. ZAUMEYER, principal pathologist, and H. REX THOMAS, assistant chief, Horticultural Crops Research Branch, Agricultural Research Service

## INTRODUCTION

Dry (field) and snap (garden) beans (*Phaseolus vulgaris* L.) and lima beans (*P. lunatus* (Benth.) Van Eselt.) are practically universal in their distribution and constitute one of the most important food crops of the world. They are grown commercially to some extent in almost every State in the United States (figs. 1 to 3). Even in regions where they are not grown commercially, they are cultivated in nearly every home garden.

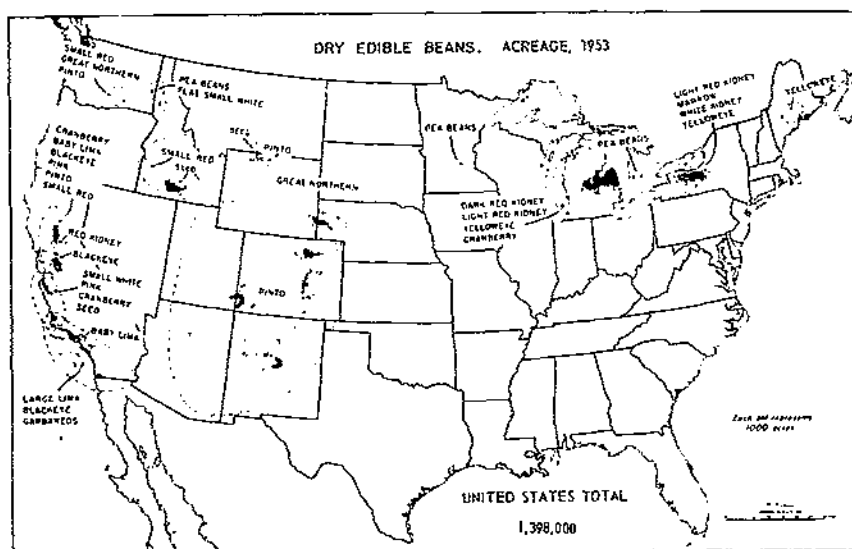


FIGURE 1.—Varietal distribution and acreage of dry beans grown in the United States in 1953. Acreage in 1953, 1,398,000 acres. Adapted from a map prepared by U. S. Department of Agriculture, Office of Foreign Agricultural Relations.

<sup>1</sup> Original edition was prepared by L. L. Harter, formerly senior pathologist, and W. J. Zaumeyer, pathologist.

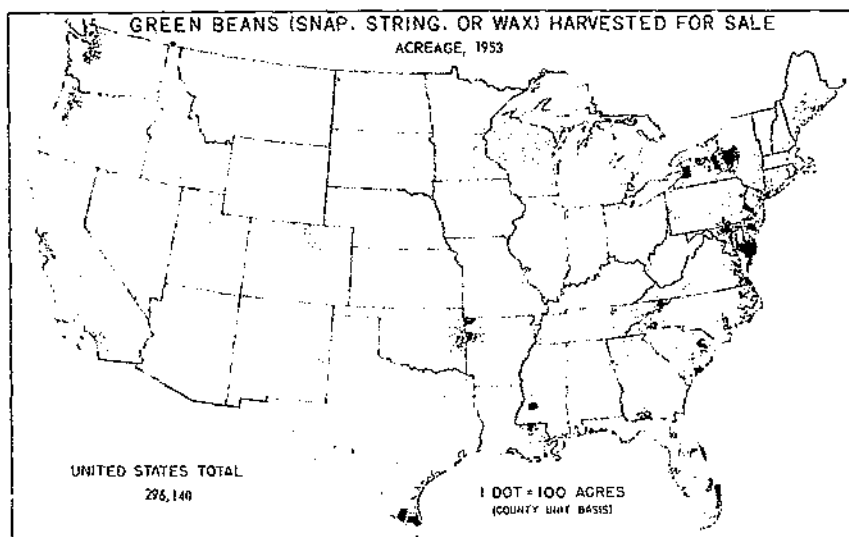


FIGURE 2.—Distribution and acreage of snap beans grown in the United States in 1953. Acreage in 1953, 296,140 acres. Adapted from a map prepared by U. S. Department of Commerce, Bureau of the Census.

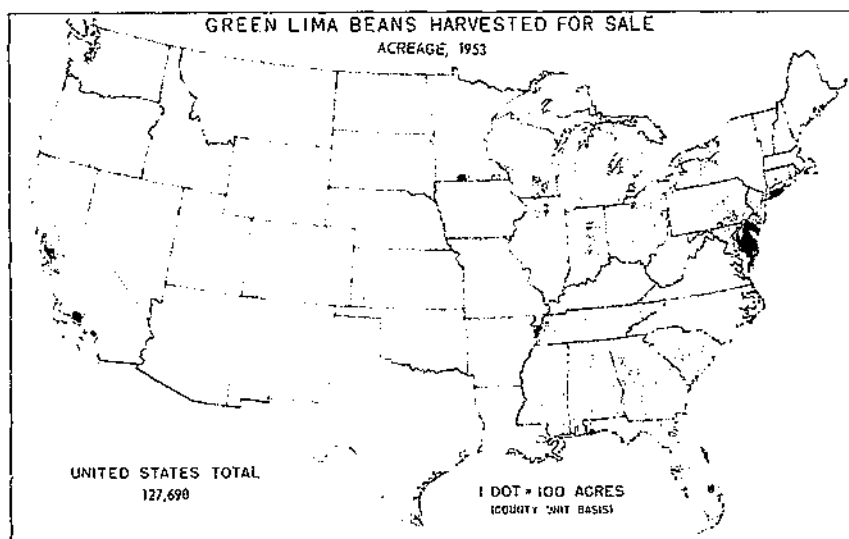


FIGURE 3.—Distribution and acreage of green lima beans grown in the United States in 1953. Acreage in 1953, 127,690 acres. Adapted from a map prepared by the U. S. Department of Commerce, Bureau of the Census.

The origin of the snap and dry bean and of the lima bean has been a moot question among plant historians. De Candolle (185)<sup>3</sup> was of the opinion that the bean originated in the Old World, while Sturtevant (982) and others believed that it originated somewhere in America, and from there was distributed to other parts of the world. The American origin of the common bean now appears to be fixed by descriptions and references showing that it was found in many scattered points over the Americas about 1500. Guatemala is now believed to be the country of origin of the lima bean. Its distribution from here has been traced by the various early varieties left along Indian trade routes. One route extended into Peru where the larger seeded types were developed in the warm coastal areas. The name "lima bean" probably came from Lima, Peru, where the species was found by early American explorers. The evidence indicates that beans were important articles of food in both the Old and the New World as early as the fifteenth century.

The bean growers in the United States may be roughly divided into four classes: (1) Growers of dry shelled beans; (2) market gardeners who grow beans for consumption as a green vegetable; (3) processors; and (4) seed growers.

The farm value of dry beans (1042), including snap and lima beans grown for seed in the United States in 1953, was approximately \$138,750,000; that of snap beans for market, \$46,912,000; and that of snap beans for processing, \$36,664,000. The farm value of lima beans for market amounted to \$4,045,000, and for processing, \$16,206,000. The total value to the farmer was roughly \$242,577,000.

In 1953, 1,398,000 acres of dry beans produced about 16,761,000 100-pound bags. There were 158,620 acres of snap beans for market and 187,520 acres for processing, which produced 17,486,000 bushels and 298,580 tons of beans, respectively. Lima beans were planted on 18,500 acres for market and on 109,190 acres for processing, which produced 1,433,000 bushels of pod beans and 105,900 tons of shelled beans.

Dry beans rank high as a cheap source of nourishing food. While not so high in calories as potatoes and sweetpotatoes, they outrank most vegetables in the amount they contain. They are high in protein, calcium, and iron, and contain a large amount of vitamin B<sub>1</sub> (976).

Snap beans are prized as a green vegetable and are valuable as a source of calcium, riboflavin, and iron. Although not so high in nutritive value as the dry bean, they possess about the same nutritive qualities as some of the green leafy vegetables, such as kale, beet tops, swiss chard, and spinach.

The production of beans, like that of many other crops, is greatly reduced every year by fungus, bacterial, and virus diseases (1197). Just how prevalent such diseases are and what the losses amount to depend on environmental factors, such as temperature and humidity, as well as on the quality of the seed. During seasons

<sup>3</sup> Italic numbers in parentheses refer to Literature Cited, p. 192.

of abundant rainfall accompanied by high humidity and high temperatures, certain diseases are worse than others. It is not to be expected that an entire season would pass without there being present some of the environmental conditions favorable for infection by and spread of certain parasites. All the diseases do not occur every year in a single locality. Some years a certain disease may be prevalent and destructive and the next year entirely absent or present to such a limited extent as to be of no consequence.

The average losses (\$20) from the principal diseases to the bean crop in 1938 (the last complete report) throughout the United States was estimated at approximately 12 percent. In a rainy, cool season they may cause losses amounting to \$15,000,000 to \$20,000,000. Good cultivation and the use of the proper kind and amount of fertilizer may hold the diseases in check and thereby enable the grower to produce a fair crop in spite of the diseases. It must not be assumed, however, that such practices are a substitute for the application of control measures or make it no longer necessary to develop and use resistant varieties. In some cases investigations have shown that plants supplied with certain plant nutrients that cause a vigorous succulent growth are more easily parasitized than those in a weakened condition.

The investigations on bean diseases are extensive; not only are many of the results conflicting and confusing, but they also are often published in little-known or obscure journals and in many foreign languages. The purpose of this bulletin is to assemble as much of the pertinent information as possible from these numerous publications, together with the results of extensive investigations conducted by the writers over a period of many years. Where not otherwise specified, facts reported in this bulletin are supported by the results of the writers' own researches and observations.

The chemicals mentioned in this bulletin are injurious and some of them extremely poisonous to man and other animals when taken internally. Care should be taken in handling them to prevent their contact with the mouth, eyes, or nostrils. When using these chemicals in dust form, be careful not to inhale them. When large quantities of seed are being treated with dust, a respirator or dust mask should be worn. (This warning applies to the use of dusts on plants in the field.) When small quantities of seed are treated in the open air or in a well-ventilated room the use of a respirator or mask is not necessary. When a large quantity of solution is used, oiled leather gloves and a rubber or oilcloth apron should be worn. Care should be taken to dispose of the unused solutions so that they cannot be drunk by chickens, cattle, or other livestock. After completing the treatment all vessels should be thoroughly cleaned and the hands and clothing washed.

## FIELD DISEASES OF SNAP AND DRY BEANS

### Fungus Diseases of Major Importance

#### Anthracnose

##### Geographical Distribution and Economic Importance

When and where bean anthracnose, caused by *Colletotrichum lindemuthianum* (Sacc. & Magn.) Scrib., originated will probably never be known. The earliest satisfactory evidence of its collection dates back to 1843 (277, p. 344). In 1875 it was collected at Padua and Bonn (852, 856), and Lindemuth reported it to Frank (351) in the same year. In 1880 the same disease was found on kidney beans at Bedford, England (94).

In 1884 (857) anthracnose was common in Italy, France, Germany, and North America. Since then it has been reported from practically all countries of Europe, Japan, Formosa (Taiwan), India, many islands of the East and West Indies, Belgian Congo, Transvaal, Uganda, Brazil, Guatemala, Venezuela, Canada, Cuba, Australia, New Zealand, and the Union of Soviet Socialist Republics.

In 1891 the bean crops in parts of Italy were completely destroyed (1055). Serious epidemics in 1915 and 1916 in Germany were reported by Fischer (344).

Since the earlier observations of Ellis and Everhart (325) and Scribner (888, 889), the disease has been found in many States of the United States, Alaska, and Hawaii. It probably has occurred in all except those where it is excluded because of prohibitive climatic conditions.

From about 1912 to 1920 anthracnose was considered the most serious bean disease in the United States east of the Mississippi River. Many infected seed rotted in the ground without germinating or, if they did germinate, the seedlings were destroyed before they emerged. Plants were frequently destroyed in the seedling stage or soon thereafter. Those that survived the seedling stage were so devitalized that they produced a small crop or none at all. Sometimes, good vegetative growth was produced, but the pods were badly flecked by anthracnose, making it necessary to discard many of them at the time of picking as diseased pods materially reduced the market value. Since losses occur from the time the seed is sown to the time of marketing, no accurate estimate of total loss is possible. Losses are much less at the present time than they were in 1926 and earlier. The reason for the decided reduction in losses since 1927 is the fact that more disease-free seed is being planted.

The anthracnose organism requires humid weather with comparatively low temperatures for infection to take place. If dry

weather predominates, no anthracnose results even though infected seed is planted. Much of the seed that was distributed previous to 1927 was grown in Michigan and New York in regions especially favorable by temperature and humidity conditions to infection and growth of the anthracnose organism, which is seed-borne. However, since about 1930, more of the snap bean seed has been produced in the arid States, where the causal organism does not thrive because of the unfavorable weather conditions. Barrus (76) stated that the disease occurred in cycles in New York from 1906 to 1919. In 1915 the losses varied from 30 to 100 percent of the crop. Heavy losses were reported in 1892 to have occurred in New York (82) the previous year and in New Jersey (420) in 1896.

The losses in Michigan (704) in 1914 were estimated at approximately \$1,500,000, and double that amount in 1915. States reporting for 1918 listed losses ranging from 0 to 18 percent, the greatest losses being recorded for the Northern States (614).

In 1927 anthracnose was observed in a number of States (631). The infection varied from a trace in some States to 1.5 percent in Maryland and 10 percent in Massachusetts and Tennessee. The losses indicated were in sharp contrast to those of 1936 (319), when the greatest loss reported was 4 percent and the average for all the reporting States that grew beans for canning was only 0.2 percent. Most of the States reported no loss or only a trace. Table 12 of the same report, which included States that grew snap beans for the market, showed an average of only 0.1 percent. Before the planting of disease-free seed became the usual practice, many crops were ruined in Florida by anthracnose and thousands of baskets were discarded as worthless after they had been shipped. Most of the dry bean acreage grown in Michigan is planted with locally grown seed. In 1950 conditions for spread and development of anthracnose were favorable, and the loss from the disease was estimated at \$1,400,000.

### Symptoms

The symptoms of anthracnose on the seed are not always easy to distinguish from those caused by certain other organisms. The anthracnose organism produces yellowish to brown sunken cankers (fig. 4, C), which may be rather small or extend over a large part of the seed coat. The common bacterial blight organism and the halo blight organism cause cankers similar to those produced by the anthracnose fungus on the seed. These diseases can usually be distinguished by the types of lesion produced. In many cases the lesion caused by the common blight organism may be distinguished by the yellow deposit of bacteria under the seed coat.

The infection of the hypocotyl of seedlings usually results from the spores washing down from an infected cotyledon and causing few or many lesions. The lesions, which often attain considerable size, begin as minute flesh- to rust-colored specks, that gradually enlarge lengthwise of the stem and to a lesser extent around it. The lesions (fig. 4, A) finally become sunken. Myriad of spores

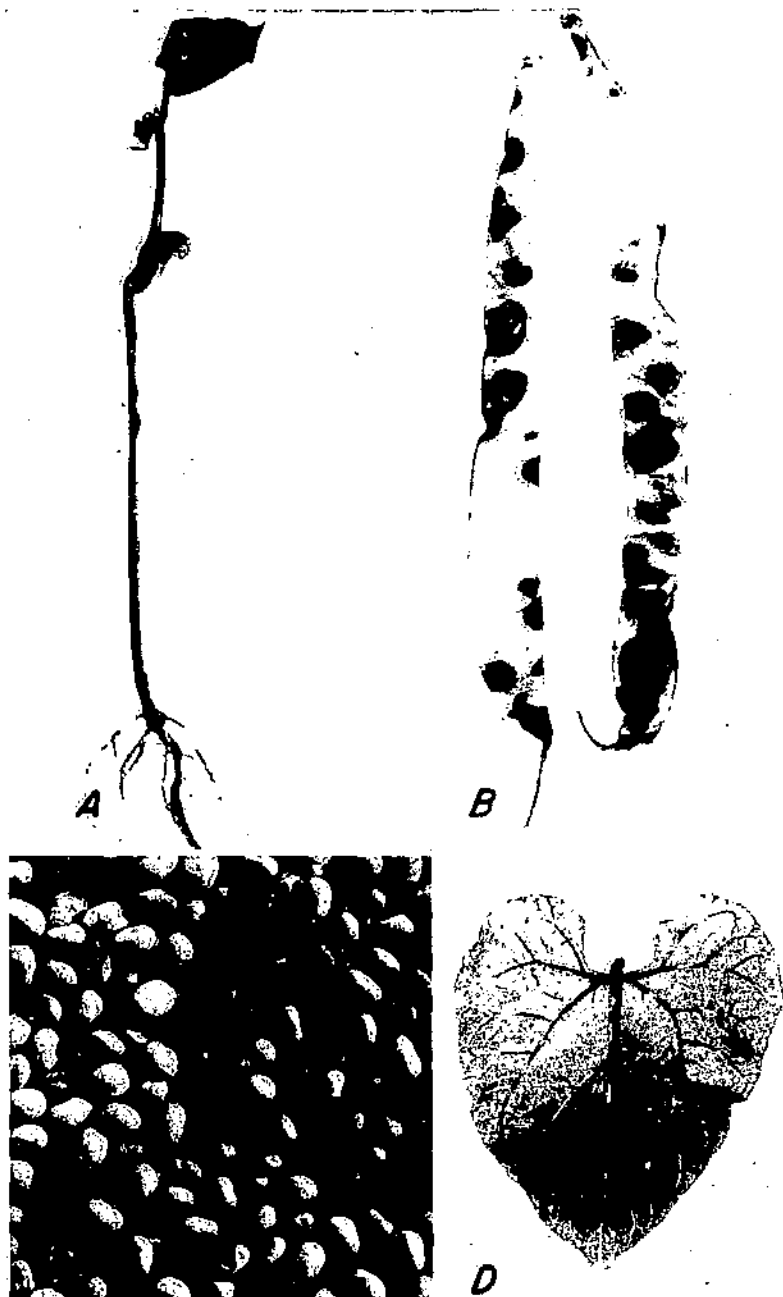


FIGURE 4.—Anthracnose, caused by *Colletotrichum lindemuthianum*: A, Young infection on the stem of a seedling, showing slightly sunken lesions; B, pods showing sunken lesions in which numerous conidia are massed; C, dark-colored and somewhat shrunken, infected seed, illustrating the seed-borne nature of anthracnose; D, infection of the underside of a leaf, showing invasion of the veins and veinlets by the causal fungus.



massed in them give them a rust color. If the lesions are numerous and favorable conditions prevail for the development of the fungus, the stem may be so weakened that it is unable to support the top of the plant.

Infection may occur on both the petiole and the veins of the leaf. If the petiole is badly infected, the leaf droops and recovery to its normal position is not possible. Infections occur on the underside along the veins (fig. 4, D), causing a dark, brick-red to purplish color that later turns dark brown or almost black. Small lesions, in which spores are generally present, may be produced on the petioles and larger veins. Fewer spores are to be found in the infected portions of the small veins. Somewhat similar symptoms on the veins and veinlets are sometimes caused by certain physiological disturbances with which anthracnose might be confused.

Anthracnose is the most easily recognized and the symptoms are most clearly defined on the pods. The first evidence is noted as small flesh- to rust-colored spots. The very young lesions may be longer in one direction than in the other, but a fully developed lesion (fig. 4, B) is usually nearly circular. The lesions vary in size from 1 to 10 mm. in diameter, averaging about 5 to 7.5 mm. Barrus (76) described the disease in part as follows:

A canker may extend through the endocarp and even to the seed, particularly if infection takes place early, in which case the pod sometimes fails to develop and becomes shriveled and dried. Cankers resulting from infections that occur during the later growth of the pod seldom extend below the endocarp. As the pod matures, the lesion is marked at the edge of a canker by a slightly raised, black ring with a cinnamon-rufous to chestnut-colored border. The center of the spot is then somewhat light buff in color. Flesh-colored spore masses on the surface of a young canker dry down to gray, brown, or even black granulations or to small pimples.

## Causal Organism

### Nomenclature

Since the anthracnose fungus produces setae on some specimens and not on others, there has been some uncertainty as to its valid name. Saccardo and Magnus (852) described the fungus in 1878 as *Gloeosporium lindemuthianum* from specimens collected by Lindemuth at Bonn, Germany. Some years later, Scribner (888), noting the presence of setae on his material, stated that it seemed "probable" that the name must be changed from *Gloeosporium* to *Colletotrichum*. In October 1889 Scribner (889) published another article on bean anthracnose and definitely cited the fungus as *C. lindemuthianum*. In the same year Briosi and Cavara<sup>4</sup> used the same binomial credited to themselves. In view of the fact that Scribner first suggested the transfer in 1888 and actually made it in 1890, quite probably before Briosi and Cavara's publication appeared, it appears desirable to give Scribner credit for the name.

<sup>4</sup> BRIOSI G. and CAVARA, F. I. FUNGHI PARASSITE DELLE PIANTE COLTIVATE OD UTILI. Fasc. II, No. 50. 1889.

In 1880 Berkeley (94) ascribed the cause of anthracnose to *Ascochyta*. In 1893 Halsted (419) conducted cross inoculations with the bean and watermelon anthracnose organisms and concluded that the two were identical, and referred both organisms to *Colletotrichum lagenarium* (Pass.) Ell. & Halst. Halsted's data were not regarded as conclusive and were not generally accepted.

No one yet has definite proof of a perfect stage of the organism; only once have perithecia with mature asci been reported, and those were in culture. In 1913 Shear and Wood (905) found asci and perithecia in cultures made from anthracnose spots on beans. These did not occur with great frequency except in one line of cultures that produced perithecia but not conidia. There is no evidence that Shear and Wood demonstrated the parasitism of this particular strain. They were apparently convinced that it was a perfect stage of *Colletotrichum lindemuthianum* and accordingly called it *Glomerella lindemuthianum* Shear.

Krüger (578) accepted the conclusions of Shear and Wood, but Edgerton (316) suggested that they may have been working with one of the saprophytic species of *Colletotrichum* frequently found on beans. The ascigerous stage has not been reported since.

Desmazières (277) described a fungus that he collected on *Phaseolus* as *Septoria leguminum* Desm., which Saccardo was unable to distinguish from *Gloeosporium lindemuthianum*. Barrus (76) found two distinct fungi on the specimens distributed by Desmazières as *S. leguminum*, one of which contained spores typical of *Colletotrichum lindemuthianum*. Richon (842) described an organism found on the stem of beans as *G. phaseoli* Richon, but Allescher (29) intimated that it was probably identical with *G. lindemuthianum* or that it may have been a *Colletotrichum*. These descriptions and specimens distributed by Desmazières and others have led to considerable confusion as to the exact identity of some of these organisms. Some of these descriptions and distributions probably led to the error of ascribing to *S. leguminum* the organism on the pods of *P. vulgaris* distributed by Von Thümen<sup>5</sup> as *S. leguminum* and by Roumeguère<sup>6</sup> as *S. leguminum* var. *phaseolorum* Roum. Other fungi collected from beans have been described, but the descriptions are in some cases too incomplete for anyone to determine whether they are identical with the bean anthracnose organism. In some cases the specimens themselves are too meager or too poor to be of much value.

Takimoto (989) reported a new species of anthracnose from azuki bean in Japan which was infectious to bean and cowpea and described the organism as *Colletotrichum phaseolorum*.

### Morphology and Physiology

The conidia of *Colletotrichum lindemuthianum* are hyaline and one-celled and vary considerably in size and shape. They are borne in acervuli, and in mass are salmon, ochraceous, or pinkish. Setae

<sup>5</sup> THUMEN F. VON. MYCOTHECA UNIVERSALIS [Exsiccati]. No. 2096. 1882.

<sup>6</sup> ROUMEGUÈRE, C. FUNGI GALlici [Exsiccati]. No. 2791. 1884.

are sometimes present on the host and frequently in culture, especially if the organism is grown on agar. They are less frequent on bean pods than the other parts of the host. The setae are often pointed, stiff, simple, septate, brown hairs, varying in length from  $30\mu$  to  $100\mu$ . They arise from among the conidiophores or at the margin of the acervulus. The spores, or conidia, are borne on hyaline, unbranched, erect, continuous conidiophores,  $40\mu$  to  $60\mu$  in length, and packed close together in the acervulus. They are generally oblong with rounded ends, although there are many modifications of form. Some spores are rounded at one end and somewhat pointed at the other. A few are kidney-shaped and some are S-shaped, while others are slightly curved at one end.

Spore germination, which begins in 6 to 9 hours under favorable conditions, can be observed to the best advantage in a nutrient solution. The percentage of germination is very low in distilled water, and the germ tubes are usually abnormal. Leach (592) showed that the highest percentage of spores germinate on green-bean and potato-dextrose agars. He also found that in Czapek's solution without sugar, germination was practically prohibited when the pH was lower than 2.6 and higher than 9.6. Mathur and coworkers (657) reported that the gamma strain which does not sporulate abundantly on synthetic media gave excellent sporulation on a medium containing glucose, mineral salts, and neopeptone. There was no conidial formation below pH 3.0, but the formation was greatest between pH 5.2 and 6.5. The anthracnose fungus is one of those organisms that require relatively cool temperatures for growth, infection, and development. The writers have found that a temperature of about  $14^{\circ}$  to  $18^{\circ}$  C. is the most favorable for spore production on snap bean pods. At temperatures of  $30^{\circ}$  or above no spores, or only a few abnormal ones, are produced. Leach (592) found that maximum growth in culture occurred at  $22.5^{\circ}$ ; maximum normal spore germination occurred at  $27.5^{\circ}$ .

Appressoria are generally formed if the spores are germinated in a solution low in nutritive value, provided the germ tube comes in contact with a hard surface. It is with the aid of these organs that the germ tube penetrates the host tissue. On the surface of the leaf, a germinating spore soon forms an appressorium, which is fastened to the leaf by means of a sticky or mucilaginous substance. A small thread is developed from the appressorium, and penetration of the host cell is apparently accomplished by mechanical means. Once inside the cell the hyphal thread enlarges and continues its growth, penetrating other subcuticular layers through small openings probably made by enzymic action. This process continues for several days without killing the cells. Strands of mycelium increase beneath the epidermis and produce cavities that become acervuli containing the spore masses. A single acervulus may contain 3 to 50 or even more conidiophores, depending on the size of the lesion. Each acervulus is composed of a stromatic layer from the surface of which the conidiophores arise. For a more detailed study of the structure of the appressoria and the process of penetration, the reader is referred to the work of Dey (278).

## Pathogenicity

In 1892 Halsted (415, 416) carried out inoculation experiments to prove the parasitism of *Colletotrichum lindemuthianum* and obtained positive results in 36 hours. Similar results were obtained by Scribner (889).

Some years later Barrus (73) observed that some varieties of beans were susceptible to anthracnose while others were not. Following up this lead, he inoculated a number of bean varieties with several different isolates. His results clearly demonstrated that he was working with at least 2 physiologic races. Barrus' results were corroborated by Edgerton and Moreland (318). In 1918 Barrus (75) published results of inoculating with 10 different isolates a large number of varieties of dry and snap beans, a number of lima bean varieties, and several other legumes more or less closely related to beans. He designated one of the races as alpha and the other as beta. In New York Burkholder (162) isolated from the White Imperial variety a third race that he designated as gamma.

Leach (592), as a result of inoculating 14 varieties of beans with isolates from various sources, concluded there were 8 distinct races. There was no evidence that he had in his possession the alpha and beta races of Barrus (75) and the gamma race of Burkholder (162). From these results it is conceivable that 11 distinct races were recognized at this time. Leach (593) in grafting experiments between susceptible and resistant beans, found no indication that resistance or susceptibility was influenced by either stock or scion.

Muller (702) reported five physiologic forms from Holland that differed from those of Barrus, Burkholder, and Leach. He (701) also isolated a strain of a species of *Gloeosporium* from *Phaseolus multiflorus* that he found to be infectious to bean and apple, and identified it as *G. fructigenum* f. *hollandica*. When this strain was passed through *P. vulgaris*, its pathogenicity to apples was decreased but its virulence toward bean was increased (701). Yerkes and Orteiz (1163) reported at least four new groups of races in Mexico that differed from the alpha, beta, and gamma races.

In Germany Shaffnit (877), Shaffnit and Böning (878), Böning (106), Budde (148), Peuser (770), Schreiber (882, 883), and Reichelt (822) carried on investigations dealing principally with 2 different phases of the problem: (1) Varietal susceptibility or resistance and (2) races of the causal organism. The most extensive investigations were conducted on 57 varieties of beans, mostly of European origin by Schreiber, who concluded that there were 34 distinct races that could be divided into 3 major groups corresponding to alpha, beta, and gamma. In Mexico De la Garza (381) found that of the 180 Mexican bean varieties tested, about 60 percent were resistant to the alpha race while 90 percent possessed a high degree of resistance to beta and gamma races. Andrus and Wade (48), in their studies on anthracnose resistance, reported a new race, delta, isolated from bean material from

North Carolina. They were unable to identify this race with any of the 34 races described by Schreiber. Frandsen (350) in Germany reported 7 additional races that differed from any previously recorded in Holland and Germany. Reynolds (836) reported that liquid cultures made from undiluted dried extracts from young plants of certain bean varieties were found to prevent growth of *Colletotrichum lindemuthianum*. Preliminary studies indicated that varietal differences were noted in quantity of toxic action. An extensive study of the susceptibility and resistance to anthracnose was made by Rands and Brotherton (802) in the United States with 663 varieties and strains, of which 170 were of American origin and 493 foreign, and by Isariishvili (514) in the Union of Soviet Socialist Republics. Rands and Brotherton showed that 27 varieties were resistant, of which 6 were practically immune to all the then-known forms.

Johnson (538) reported that water congestion in leaf tissues may be a predisposing factor to leaf penetration of the anthracnose pathogen. Basic factors involved in heavy infection appeared to depend upon the presence of water congestion, together with a conditioning of the epidermis or cuticle favoring penetration.

#### Dissemination

The causal organism is seed-borne and will survive from one season to the next as dormant mycelium within the seed coat or even in the cells of the cotyledons or as spores between the cotyledons or elsewhere in the seed. These facts make it clear that the seed is the principal means of dissemination over long distances from one State to another or from one section of the United States to another.

There are several ways the disease may be disseminated over short distances. The spores are embedded in a sticky gelatinous substance from which they are not easily freed. This mucilaginous substance is soluble in water and may be dissolved during rains and the spores scattered about by the splashing raindrops to nearby plants and to other parts of the same plant. Laborers picking beans may act as distributing agents, especially if the picking is done when the vines are wet with dew or rain.

The many insects that frequent bean plants may crawl over spore masses, and the mucilaginous substance containing spores may adhere to their bodies; in their visits to other plants they deposit the spores on the leaves or pods where a new infection may be started.

#### Hosts

Anthrachnose is largely restricted to *Phaseolus vulgaris*. The Scarlet Runner (*P. coccineus* L., formerly called *P. multiflorus* Willd.) is slightly susceptible, and lima bean (*P. lunatus*) has been infected. The tepary bean (*P. acutifolius* var. *latifolius* Freeman) is very susceptible, and the mung bean (*P. aureus* Roxb.) slightly so.

### Control

Seed treatment to be effective against anthracnose must destroy the mycelium and spores in the seed without greatly impairing its germination. From the time anthracnose was recognized as a serious disease of beans, control experiments have been carried out. These experiments include seed treatment with a variety of chemicals and by hot water and dry heat. Halsted and Kelsey (422) and Whetzel (1113) concluded that different strengths of bordeaux mixture had little effect on the mycelium within the seed. Bedford (86) and Sevey (898) soaked bean seed in different concentrations of formalin and claimed a reduction in the amount of the disease without any appreciable effect on germination. On the other hand, a number of other investigators (82, 250, 317, 344, 702) worked with a variety of different chemicals, and, although in some cases they noted some promise of beneficial results, the general conclusion was that any chemical that would destroy the organism would reduce the percentage of germination below the point of practicability.

The wet method of seed treatment has been opposed by some investigators on the ground that the seed is badly injured by soaking in water. On the other hand, Tilford, Abel, and Hibbard (1020) concluded that soaking bean seed *per se* was not injurious. They found that the embryo was damaged by bacteria or bacterial products that accumulate during the period of soaking. When clean seed was soaked in sterile distilled water and aerated, a germination of 97 percent was obtained after it had been 3 days under water. It was noted that, if the seed was supplied with sufficient oxygen and the carbon dioxide and the decomposing by-products were removed, beans would germinate under water as readily as in the soil.

Beneficial results have been reported (967) from treating the seed for half an hour in a 0.125-percent solution of Ceresan. It not only increased the percentage of germination but it also stimulated the seed to earlier germination. Seed treated with Ceresan developed great vigor in the field and remained healthy, while much of the untreated seed was destroyed by anthracnose. In view of the reduction in the amount of disease and the stimulative effect on growth, treatment of the seed with Ceresan was recommended.

Several investigators have attempted control by heating the seed, either wet or dry, to a specified temperature, with the hope of killing the fungus without injuring the seed. Kirk (564) obtained good results by soaking the seed 5 minutes in water at a temperature of 60° C. or 15 minutes at 54.5°, and Edgerton (315) was inclined to believe beneficial results were possible by soaking the seed for 10 to 15 minutes at 50°. Kidd and West (559) obtained poor germination from soaking the seed at different temperatures for periods ranging from 6 to 72 hours. Muncie (703) tried heating the seed, both wet and dry, with the result that in those cases where the pathogen was killed the germination was considerably reduced.

Although Steinberg (967) obtained beneficial results from seed treatment, the evidence indicated that anthracnose could not be adequately controlled by any of the seed treatments so far devised. According to Andersen (34), treating infected seed with Vancide 51 at concentrations of 7.5 or 15 percent in a 2 to 3 percent methocel slurry for 10 to 40 minutes was very effective in controlling the disease. Seed treated for 20 minutes in methocel containing 5 to 10 percent of the fungicide gave nearly complete control. In Switzerland (406) Ceretan (Ceresan) dust, 0.5 gr. per 100 gm. of seed, eliminated the organism if applied when only the seed coat was involved, but it was of questionable value after the hypha had penetrated the cotyledon.

Disease control in the field was demonstrated with mycotox-4 (substituted phenyl ester) and onyx DL-1 (didodecyl dimethyl ammonium bromide) in Shell Horticultural base oil No. 7 used with a mist blower at 3 gallons per acre (350). One aerosol treatment using an organic copper formula produced 96 to 100 percent protection for 15 to 30 days against anthracnose. In New Zealand, Reid and Brien (829) reported that spray applications of bordeaux mixture (6-8-100) and cupron (copper oxychloride (5-100)) appreciably reduced anthracnose infection and increased yields. McNew (634) found that beans sprayed with ferbam at the rate of 2 pounds per 100 gallons of water effectively reduced infection. Wilson (1123) reported ziram as giving good control in Ohio. More recently McNew, McCallan, and Miller (635) reported the following fungicides effective in reducing anthracnose infection: dichlone, zineb (Dithane Z-78), nabam (Dithane D-14), ziram (Zerlate), ferbam (Fermate), lime sulfur, wettable sulfur, bordeaux mixture, copper lime dust, and fixed or insoluble copper.

Natti and Szkolnik (714) reported that beans treated with 2,4-D were less severely infected by anthracnose than similar beans treated with other plant hormones. Control was probably the result of suppression of development of susceptible tissues rather than modification of metabolism of plants induced by growth regulators.

Crop rotation should be practiced. It should comprise at least 3 years' duration, since it has been shown that the anthracnose fungus will live at least 2 years in the soil. Tochinal and Sawada (1027) in Japan reported that the spores of the fungus were unable to live through the winter but were found to live in the dry tissue for 2 years. They also showed that the organism can survive for 2 years in the seed.

As anthracnose does not occur in the Rocky Mountain States and in States farther west, any bean seed grown in such regions is practically free of the disease and can be safely used. The writers have found that anthracnose-free seed will give an almost clean crop the first year. Since the writers have demonstrated the value of clean seed, anthracnose has ceased to be of any consequence in those localities where from 1912 to 1928 it was a limiting factor in the growth of the crop.

While the use of western-grown seed is controlling anthracnose at the present time, breeding studies are in progress in Michigan

to produce a pea bean resistant to the 3 races of the organism common in the United States. Pea beans are not generally grown in the West, and practically all of the seed is grown in Michigan and New York. In these studies, Emerson No. 847, a strain produced at Cornell University but never released to the trade and resistant to alpha, beta, and gamma strains, was used as the resistant parent. Some reference has been made already to this phase of the problem (p. 11). A selection from Red Kidney that showed considerable resistance to anthracnose was brought to the attention of Barrus (74) in 1913. He tested this strain under controlled conditions and found it to be highly resistant to the alpha and beta races of the organism. The variety was named Wells Red Kidney. Burkholder (158) produced a resistant White Marrow, and McRostie (636, 637) a resistant white pea bean. These results have been supplemented by the breeding and selecting of varieties resistant to physiological races of anthracnose in Germany.

Reid (826, 827) in New Zealand tested many varieties for resistance to anthracnose and found several that were free from the disease; namely, Small White, Burbank, and Ideal Market. He also noted quite a number of varieties that were highly resistant. In Australia (2, 4) resistant Tweed Wonder variety is recommended where the disease is common. In Mexico (381) inoculation experiments with alpha, beta, and gamma races to local varieties showed most varieties susceptible to the alpha race but a high degree of resistance to beta and gamma races. Hubbeling (501) reported Dubbele Witte Stringless and Groniger Weekschil virtually immune from anthracnose in Holland.

The results indicate that three methods — the use of clean seed, spraying with fungicides, and the development of resistant varieties — may be employed to control anthracnose. Clean seed can be produced in the United States, but in some foreign countries weather conditions may be such that production of clean seed is not possible; in that event, breeding or selection of resistant varieties would be necessary.

### Ashy Stem Blight

#### Geographical Distribution and Economic Importance

Ashy stem blight, caused by *Macrophomina phaseoli* (Maubl.) Ashby, and sometimes called blight, stem blight, root rot, charcoal rot, and macrophoma rot, was first described on beans by Maublanc (662) in 1905.

The disease has been reported from many parts of the world: Africa (921, 1074), Canada (682), Ceylon (412), Cyprus (715), Egypt (140), Formosa (823), Greece (873), India (327, 1043), Italy (389, 390), Palestine (823), Philippine Islands (845), and Venezuela (1148).

According to Kendrick (552) ashy stem blight was first reported on beans in the United States in 1919 from California. If, however, the causal organism is the same as the one causing



charcoal rot of sweetpotatoes, then it has been known in the United States for many years. In 1925 Ludwig described it as a new stem rot of beans in South Carolina (610). The following years Wedgworth (1095) reported it from Mississippi. In the next few years ashy stem blight was reported or collected on lima, snap, and dry beans from Colorado, Florida, Georgia, Kansas, Maryland, Nebraska, North Carolina, and Virginia. More recently it was reported as serious in Texas.

Ashy stem blight occurs on the spring crop of beans in the Southern States throughout the entire growing season. Ludwig (611) reported its occurrence in considerable abundance in South Carolina, and losses of 60 to 65 percent have occurred in some fields in Mississippi. The writers observed the disease in 5 to 50 percent of the plants in different fields in the southern part of Georgia in 1927, when it caused the death of many plants. From what is known of the disease at the present time, it is likely that the losses were more than they appeared to be. It is seed-borne and may destroy many germinating seeds before they emerge from the soil (44, 552, 613). Kendrick (552) proved that high temperatures favored the disease, and observed that serious field outbreaks followed periods of warm weather.

### Symptoms

The ashy stem blight fungus frequently causes conspicuous black, sunken cankers on the seedlings before or soon after they emerge from the soil. A small, somewhat irregular, dark, sunken lesion on the stem at the base of the cotyledon (fig. 5, *D*) is usually the first evidence of the disease. From these cankers the infection may extend in either direction (fig. 5, *B*) and frequently into the petioles of the primary leaves. The progress of the lesion may be so rapid that within a short time the entire stem is involved and the growing tip is killed. The cankers at the base of the cotyledonary node are sunken (fig. 5, *D*) and dark-colored and have a rather sharp margin. Sometimes concentric rings within the canker are characteristic of the cotyledonary node infections. This type of infection frequently weakens the stem, and the plant breaks off later as the result of injury resulting from cultivation or strong winds. Black sclerotial bodies form in the diseased tissue a few days after infection. Numerous sclerotial bodies that macroscopically resemble pycnidia may develop in the stems of diseased plants.

Sometimes, however, the fungus may produce a multitude of tiny fruiting bodies (pycnidia) on the surface of the infected stem (fig. 5, *C*). These appear as minute black pustules about the size of pin points on the gray background, which has a characteristic ashen appearance quite different from the symptoms of any other bean disease. Kendrick (552) never observed pycnidia in California, but numerous sclerotial bodies occurred on the stems of dead or dying plants.

If infection is delayed until after the primary leaves are fully developed, the progress of the fungus is materially slower than



FIGURE 5.—A, *Fusarium* root rot, caused by *Fusarium solani* f. *phaseoli*, on underground part of stem and on roots. B-D, Ashy stem blight, caused by *Macrophomina phaseoli*: B, Primary infection at the cotyledonary node and a secondary infection of a primary leaf; C, pycnidia on stems of large plants; D, primary infection at the cotyledonary node.

if infection occurs before or soon after the plant emerges from the soil. The development of the disease is frequently more pronounced on one side of the plant, resulting in the drooping and death of the primary leaf on that side and the yellowing of the trifoliate leaves if present.

In Georgia (613, 1100) ashy stem blight and *macrophomina* leaf spot infection, which occurs mostly on mature plants, are primarily caused by infection from air-borne conidia. Charcoal rot is a disease of young seedlings and results from mycelial infection in the soil.

## Causal Organism

### Nomenclature

The fungus causing ashy stem blight was originally called *Macrophoma phaseoli* Maubl. (662). In the United States this organism has been variously designated. Kendrick (552), Mackie (624), and Tompkins and Gardner (1028) called it *Rhizoctonia bataticola* (Taub.) Butl.; Taubenhause (992), *Sclerotium bataticola* Taub.; Wedgworth (1095), *M. phaseoli*; and Andrus (44), *Macrophomina phaseoli*. Ashby (54) partly cleared up the confusion regarding the synonymy of the different organisms.

### Morphology and Physiology

*Macrophomina phaseoli*, one of the Fungi Imperfecti, does not fruit readily in culture, and frequently its pycnidia are not found on diseased plants in the field. In the Western States pycnidia occur very rarely, but in some of the Southern States they have been noted several times. In Ceylon pycnidia have developed on beans, jute, and other hosts (413). They have been obtained a few times in culture (44, 413). Luttrell (612) isolated a strain that produced pycnidia on agar medium. Sclerotial bodies are readily produced, both on the plants in the field and in artificial cultures.

Haigh (413) worked with 27 different strains of the fungus, which he separated into 3 groups largely on the basis of the size of the sclerotial bodies. Pycnidia were found only in the small-sized sclerotial group. One form that arose by saltation produced pycnidia in culture either with or without sclerotia. When inoculated into sweetpotatoes it produced a rot similar to the charcoal rot (992); either pycnidia or sclerotia developed in the tissue of the host.

The pycnidia are at first buried beneath the lead-gray epidermis in which many sclerotial bodies are generally mixed. The conidiophores are more or less straight, sometimes crooked with a truncate tip, and range from  $12\mu$  to  $20\mu$  wide by  $6\mu$  to  $25\mu$  long. The conidia are one-celled, more or less fusiform, straight, or slightly curved. One end of the conidium is often pointed and the other is blunt. The conidia range from  $15\mu$  to  $30\mu$  long and from  $5\mu$  to  $8\mu$  wide.

### Pathogenicity

Kendrick (552) demonstrated the parasitism of *Macrophomina phaseoli* on several varieties of beans and produced the typical charcoal rot of sweetpotatoes by inoculation with the bean ashy stem blight fungus. Tompkins and Gardner (1028), using isolates from various hosts, found considerable variation in cultural characters and also that most of the infections of beans and cowpeas took place through the cotyledons. With the exception of a culture from begonia, the isolates pathogenic to beans were likewise pathogenic to cowpeas. The parasitism of the organism isolated from beans has been demonstrated also by inoculation (823) of the soil. Additional proof of the parasitism of *M. phaseoli* on various hosts has been contributed by several investigators (140, 901, 922, 923, 924) in Egypt, India, and Uganda. In Georgia cross-inoculation experiments with cultures from bean, cowpea, lupine, partridge peas, lespedeza, and clover were infectious to bean but not to cotton, whereas an isolate from cotton was not infectious to these plants (883).

Luttrell (613) was able to infect beans through the soil only during the seedling stage. In contrast, when shoots were inoculated with conidia of the same fungus, 100-percent infection resulted at all stages of growth, the infections becoming increasingly severe with increasing age of the plants.

### Hosts

*Macrophomina phaseoli* was found by Mackie (624) to attack many varieties of snap and dry beans grown commercially in California. *Phaseolus lunatus* f. *macrocarpus* was not affected, but all varieties of *P. lunatus* except Hopi were susceptible. There are more than 100 hosts, including corn and sweetpotatoes (873). Johnson (536) reported the same organism as a cause of a disease of *Strophostyles helvola* (L.) Britt. in Georgia, and Henson and Valleau (479) reported it as a common pathogen of red clover in Kentucky. Additional hosts are catalpa, cedar, alfalfa, Sudan grass, mung bean, and broomcorn (493). In Peru the organism has been reported on sour orange (81).

### Control

Since the causal organism of ashy stem blight is seed-borne, it is recommended that no seed grown in the Southern States be planted. Most of the seed planted in the South is of western origin, and no cases are known in which an epidemic of ashy stem blight has been traced to seed from this source.

Since the causal organism is parasitic on many different hosts, crop rotation would be of little value, although it has been recommended. Neal and Wedgworth (719) stated that the bean seedlings may be protected against *Rhizoctonia* sp. and *Macrophomina phaseoli* by dusting the seed thoroughly with some of the organic mercury compounds, provided the seed is not planted too deeply. In Georgia (384) dusting the seed with 2 percent Ceresan effec-

tively controlled infection. Reichert and Hellinger (823) recommended burying deeply in the soil or burning all infected plant residue and not planting seed on infested soil for 5 years. Applying manure with plant ashes, potash, or lime is also recommended as a result of experiments made in Formosa (Taiwan) and India.

## Fusarium Root Rot

### Geographical Distribution and Economic Importance

Fusarium root rot, caused by *Fusarium solani* (Mart.) Appel & Wr. f. *phaseoli* (Burk.) Syd. & Hans. was first recognized in 1916 by Burkholder (156) in western New York, where it was particularly prevalent. As many as 90 percent of the plants in several counties in western New York were infected (159) and the losses (157) in some cases were very large.

Fusarium root rot, sometimes called dry root rot, has been reported from several foreign countries. Benloch and Del Cañizo (89) reported its occurrence on several varieties in Spain, where it apparently caused considerable loss. It is known to occur in England (737), Bulgaria (572), Canada (281), and Peru (12). In 1929 the disease was prevalent in England and all the garden varieties commonly grown in the Evesham district were reported susceptible (734). In 1939 fusarium root rot caused losses of 95 percent in some varieties in New South Wales (3). In 1949 severe damage was reported from Idaho (663).

### Symptoms

The first symptoms of the fusarium root rot are characterized by slightly reddish discoloration of the taproot, which is evident about 1 week after the plant appears aboveground. The general structure of the root at this time is normal. The reddish discoloration gradually increases in intensity and extent, more or less covering the taproot (fig. 5, A), with no definite margin, or it may occur in streaks that may extend nearly to the surface of the soil but rarely above it. The red color on the taproot is later replaced by a brown discoloration, which is frequently accompanied by longitudinal fissures. The infections are sometimes local rather than involving all the stem below the soil line and the taproot. Sometimes the main root and the lower part of the stem become pithy. The small lateral roots (fig. 5, A) that normally develop from the taproot are usually killed, whereupon a cluster of fibrous roots develops above the lesions just below the soil line.

Secondarily developed roots keep the plant alive, and if subsequent weather conditions are favorable, an almost normal crop may be produced. Occasionally one of these roots enlarges to take the place of the taproot, but more often the plant is supplied with nutriment by a large number of rootlets an inch or two beneath the surface of the soil.

Although the lesions do not extend above the surface of the soil, the loss of lateral feeding roots may be very apparent. This

is true in the latter part of the season, especially if the soil moisture is rather low. During the early part of the season the development of diseased plants is somewhat retarded and remains so throughout the summer. If dry weather occurs later, the leaves turn yellow and may even drop off, and the pods are few and imperfectly filled with the undersized seed. There is generally no wilting of the plant.

## Causal Organism

### Morphology and Physiology

The spore sizes and number of septations of *Fusarium solani* f. *phaseoli* vary considerably with the culture media on which the fungus is grown. The predominating number of septations is three, though in some cases four-septate spores are more numerous. Burkholder's (159) description of the organism is in part as follows:

Macroconidia mostly 3-septate (44.5 by 5.1  $\mu$ ), 4-septate (50.09 by 5.3  $\mu$ ), rarely 5-septate, of nearly even diameter throughout, more or less curved near apex, with somewhat rounded but slightly pointed apex, usually apiculate. Microconidia rare. Aerial mycelium in culture scanty and usually white. Spores borne mostly in pseudopionnotes . . . Spores in mass frequently yellowish. Chlamydospores terminal or intercalary, single or in short chains (11.6  $\mu$  in diameter).

Too much dependence should not be placed on spore sizes, as they may be greatly influenced by the culture medium used and the environmental conditions (445, 446) to which the culture is exposed.

The life history of *Fusarium solani* f. *phaseoli* does not differ from that of many other species of *Fusarium*. The only spore forms commonly found are the conidia and chlamydospores. The causal organism lives from one season to the next in one of these stages or possibly by means of the mycelium in decayed vegetable matter. This species, like many others of the *Fusarium* group has the ability to live almost indefinitely as a saprophyte, and as such might continue to exist on almost any kind of substance occurring in nature as a substrate. The dead roots of bean plants would be presumably utilized as long as they were available. The mycelium may penetrate the healthy epidermis and remain intercellular for a short time after entrance. The main hyphal strands extend longitudinally with the taproot. A physiological difference, however, does exist. *F. martii* collected from various sources has been shown by Burkholder (159) not to be parasitic on beans, while *F. solani* f. *phaseoli* readily infects beans. This significant physiological difference was not sufficient to justify a new species, but according to Burkholder (159) justified varietal rank. Reinking (835) found that certain strains isolated from beans could cause flecking and slight streaking of peas and vice versa. However, in no case was a bean isolate very virulent on pea or a pea isolate very virulent on bean.

Burke (153) studied the pathogenicity of the fungus in different soils. Pure-culture or infested-soil inocula of the fungus in

concentrations sufficient to cause severe bean rot in a virgin soil in the greenhouse produced much less damage in a soil from a root-rot-free beanfield. This pathogen-suppressing property of the soil from the beanfield could be removed by heat or chemical sterilization, indicating it was microbiological in nature.

Weimer and Harter (1099) described *Fusarium aduncisporum* as a new species, principally because of morphological differences between its conidia and those of *F. solani* f. *phaseoli*. This species and *F. martii* are now considered synonymous (947).

#### Dissemination

The causal organism is not borne within the seed, although it is possible that the spores might adhere to the surface of the seed-coat and be transported in that manner. The spores may be present in all cultivated soils and probably in virgin soils. The ability of this fungus to live as a saprophyte enables it to survive almost indefinitely under any situation where moisture and decayed organic material are available. Common methods of distributing an organism of this type are to feed the infected straw and refuse to livestock and to use it for bedding. The fungus then finds its way into the compost heap and is carried from there to the field. Heavy infestation of soils has been traced to such practices. Drainage water or the runoff during heavy rains is one of the most important agencies for the dissemination of parasitic organisms, and in several cases this agency has been found to explain the occurrence of a disease in fields where the host crop has never been grown before. If the crop is planted in high ground, the spores from the refuse of diseased plants may, and probably do, wash to the low-lying fields during heavy rains. Menzies (665) reported that the disease in the Columbia Basin section of Washington behaves as though the pathogen were native in the area. The pattern of development is the same whether on old land or on new land that may even be isolated and irrigated from deep wells.

#### Pathogenicity

The parasitism of *Fusarium solani* f. *phaseoli* has been amply demonstrated. A high percentage of infection was obtained in all cases by inoculation of the seed and of the soil before planting or by inoculating the plants. Plants in all stages of development appear to be susceptible, but the disease is worse when infection occurs early in the life of the plants. No difficulty was experienced in carrying the fungus in a viable condition through the winter (157, 159) in a compost of bean roots and sheep manure. Reddick (816) found that the yields of air-dry seed from diseased plants grown at soil temperatures of 22° and 34° C. were reduced 26 and 34 percent, respectively, when compared with seed from healthy plants. Burkholder (160) concluded that high and low soil temperatures have no, or but little, effect on the severity of fusarium root rot. Burkholder (163) found that infected plants show a greater reduction in yield in dry soils than in medium wet or wet soils. The hydrogen-ion concentration of the soil did not affect susceptibility (167).

### Hosts

Other plants besides beans known to be susceptible to *Fusarium solani* f. *phaseoli* are *Phaseolus acutifolius* var. *latifolius*, *P. coccineus*, *P. angularis* (Willd.) W. F. Wight, *P. aconitifolius* Jacq., *Vigna sinensis*, *Pueraria thumbergia* (Sieb. & Zucc.) Benth., *Phaseolus lunatus*, and *Pisum sativum* L.

### Control

There are no adequate control measures for fusarium root rot. Crop rotation is helpful if continued long enough. Experiments have shown that beans growing repeatedly on the same land increases the incidence of the disease, and if infested soil is not planted to beans for a number of years the loss to the crop is reduced. A 2- or 3-year rotation would be of little value, but one of 6 to 8 years might be beneficial.

Land that has not been used for beans for several years should be selected when possible. Manure containing infected bean straw should not be applied to fields that are more or less free of the pathogen. The disease is seldom serious where long rotations have been followed.

If the fusarium root rot is serious, shallow cultivation should be practiced. The root system of diseased plants is near the surface and should not be disturbed any more than necessary.

Chemical treatment of the soil is not entirely effective. According to Burkholder and Crosby (172, 173), any treatment sufficient to sterilize the soil injures the plants. Leach and Snyder (599) found that adding 25 percent nabam at the rate of 1 gallon per acre or 2 pounds of the dry powder in the row at time of seeding markedly reduced root rot infection. Watson (1088), using a similar method, obtained a reduction in root rot severity by using formaldehyde at 1½ gallons per acre and chlorobromopropene at rates from 0.2 to 2 gallons per acre. In Colorado (1039) some control of fusarium root rot on pinto bean was obtained by using zineb, crude penicillin, and Ceresan M as seed treatments.

Walters (1078) did not control the root-rotting organism when he used various fungicides applied as dusts to the seed or in conjunction with methocel as a sticker. He (1079) showed that root rot infection was less and yields of dry beans were higher with light frequent irrigations every 5 to 7 days throughout the season than with heavy, infrequent irrigation.

Since no very effective control measures are known, breeding and selection for disease resistance offer the best solution. Burkholder and Crosby (172, 173) found one variety known as the Flat Marrow to be very resistant.

Ogilvie and Mulligan (736) tested a number of bean varieties for susceptibility to fusarium root rot; most of these were very susceptible. The following were reported as being somewhat tolerant: Flageolet Victoria, Saxony, Incomparable, Flageolet, St. Andrews, and Dwarf Sharpes Goliath.



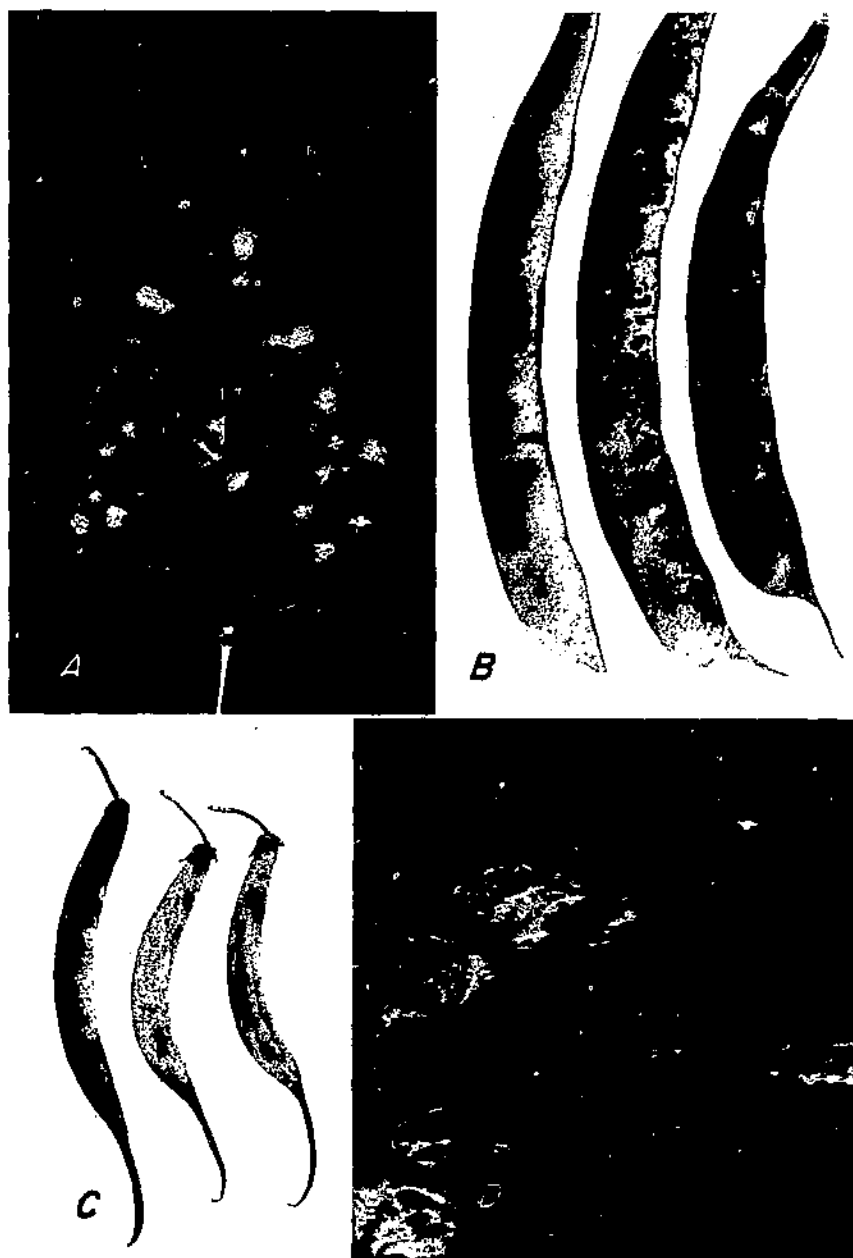


FIGURE 6.—Powdery mildew caused by *Erysiphe polygoni*: A, Infection of leaf showing conidia and mycelium; B, russetting of full-grown pods; C, young pods killed by severe infection; D, advanced infection on leaves.

Benlloch and Del Cañizo (89) found Riojana, the best variety grown in Spain, to be very susceptible, while Pineses and Asturiana, two varieties of poor quality, were less susceptible. The visible symptoms did not appear until late in the season. They recommended the following as control measures: (1) Early planting; (2) crop rotation; (3) application of lime; (4) application of barnyard manure to which basic slag, superphosphate, or kainite was added; (5) growing of resistant varieties; and (6) burning of diseased plants.

In New South Wales (4) it was reported that Tweed Wonder, Canadian Wonder, Hawkesbury Wonder, and Brown Beauty were moderately resistant.

Workers at Cornell University found several varieties of the runner bean (*P. coccineus*) and a few foreign varieties of *P. vulgaris* having considerable resistance to root rot. These varieties are being used as parental material in a breeding program for root rot resistance.

## Powdery Mildew

### Geographical Distribution and Economic Importance

*Erysiphe polygoni* DC. ex Merat., the cause of powdery mildew of beans, was described as a new species by De Candolle (587, p. 273) in 1805, but the description is not adequate to distinguish it from other species of *Erysiphe*. Merat gave the first valid description of the species. Races of *E. polygoni*, which are not morphologically distinguishable on the various hosts, are now probably worldwide in distribution.

In 1889 (374), 1922 (1114), and 1925 (733) powdery mildew was reported to cause considerable damage in Bermuda. Cook and Horne (234) reported its occurrence in Cuba in 1908.

In the United States the losses vary from year to year, but in certain localities they may exceed half the crop (227). Powdery mildew occurs in the Southern States on the fall crop, but very little is present on the spring crop. In 1917 Sherbakoff (907) included it among the important diseases of truck crops in Florida. Along the Pacific coast it occurs every year on snap and lima beans. It reduces yields by arresting the development of the plants. Although this disease is common on some other legumes in Colorado, Utah, Wyoming, Idaho, and Montana, it does not occur extensively there on beans.

### Symptoms

The powdery mildew fungus attacks all parts of the plant except the roots. It occurs first on the leaves (fig. 6, A, D) and spreads from them to the stem and pods (fig. 6 B, C). The earliest symptoms are characterized by very faint, slightly dark areas on the leaf that may be mistaken for mottling. These obscure areas later develop into small, white, powdery spots (fig. 6, A) that enlarge and combine with others to form eventually an almost complete coating of a whitish powder (fig. 6, D) over the leaf. The powdery mass is composed of the mycelium and an innumerable

number of one-celled hyaline conidia. If infection is extensive, the leaves are somewhat malformed and pale yellow. In severe cases of the disease defoliation occurs, sometimes leaving only the naked branches.

The pods may be stunted and malformed, the retardation in growth the result of the reduced vitality of the plant and the direct damage to the pods. When the powdery coating is rubbed off the pod a brownish to purplish color, sometimes known on the market as russetting, is revealed.

## Causal Organism

### Morphology and Physiology

*Erysiphe polygoni* is representative of a large group of parasitic fungi. It draws its nourishment from its host by means of haustoria attached to the leaf, stem, or pods. It produces two types of reproductive bodies: (1) Conidia, or summer spores, and (2) ascospores borne in perithecia. The latter are rarely found on beans.

The conidial stage was originally described as *Oidium balsanii* Mont. in 1854. The conidia are cylindrical, oval or nearly orbicular, simple, hyaline, one-celled spores with rounded ends and successively cut off from the ends of short, simple, erect, stout, septate, colorless hyphae. They range in length from  $25\mu$  to  $30\mu$  and from  $1\mu$  to  $18\mu$  in width. Childs (205) reported a diurnal cycle of conidiophore development and observed that the period of abstriction of conidia occurred between 10 a. m. and 2 p. m.

The perithetical stage serves to carry the fungus through the winter. The perithecia are nearly spherical, sometimes flattened, dark-colored bodies with hyphal appendages and no ostiole. The asci are released only after the thick perithecial wall disintegrates or is ruptured. The asci vary in number from two to eight. They are oblong or ovate and range from  $45\mu$  to  $70\mu$  in length and from  $30\mu$  to  $45\mu$  in width. Each ascus bears three to eight spores,  $20\mu$  to  $25\mu$  long and  $9\mu$  to  $14\mu$  wide.

*Erysiphe* spp. are exceedingly variable and are composed of a number of physiologic races (723, 821, 868). Salmon (867), Reed (821), and others have shown that spores borne on a certain host are capable of infecting only that host or in most cases only species of the same genus or related genera. Races that can pass from one genus to another are not common, and forms morphologically distinct are thought to be separate species.

Cook (228) found that conditions unfavorable to the growth of the bean, such as low temperature and lack of soil moisture, are favorable to the development and spread of powdery mildew. The writers, on the other hand, obtained the best results from inoculation of vigorously growing plants. The experimental results of Yarwood (1148) indicate that infection may be correlated with relatively low humidities and low temperatures. His results show that, while the spores will germinate at a very high relative

humidity, they will germinate also when the humidity is low. The physiologic races studied by him seem to be especially well adapted to very dry atmospheric conditions. His results apparently agree with the general field observations that *Erysiphe polygoni* and certain other powdery mildews are adversely affected by rain and can develop luxuriantly and possibly better under rather dry conditions. Yarwood (1155) also reported that mildew infection was more severe on plants grown in soils of low rather than high moisture. On beans grown in water culture, mildew development increased with increasing nutrient concentration. Miller (681) reported the treating of greenhouse-grown plants at the primary leaf stage with a spray application of maleic hydrazide or pentachlorobenzoic acid. This inhibited the growth of the trifoliate leaves, and the primary leaves were retained for a longer period than normal ones with an increase in vigor. Primary leaves infected with the powdery mildew organism could thus be sprayed once with a fungicide and kept in the greenhouse until the fungus built up sufficiently for a satisfactory disease reading.

#### Dissemination

While *Erysiphe polygoni* might be carried on the seed, the seed is probably not important as a means of distribution. The organism is probably present everywhere, so dissemination by means of the seed would not be a factor of any importance. The conidia are not believed to have long rest periods or to survive extremely adverse conditions. They are easily detached from the conidiophores and transported by wind, rain, and insects. They may be washed from the leaves onto the stems and pods in any light rain and disseminated over greater distances by rainstorms and strong winds.

#### Hosts

*Erysiphe polygoni*, a common fungus species, is destructive not only to beans and peas (*Pisum sativum* L.) but also to other genera (968) of economic importance. Among them are *Brassica*, *Calendula*, *Catalpa*, *Dahlia*, *Delphinium*, *Lupinus*, *Lycopersicon*, *Medicago*, *Scabiosa*, *Trifolium*, *Valeriana*, and *Vicia*. Salmon (868) found that conidia grown on *T. pratense* L. would not infect other species of the same genus.

It was found that the disease could be controlled in Cuba (234) by spraying with bordeaux mixture or other fungicides. Dusting with sulfur has been a commercial control practice in various parts of the United States. In 1917 Sherbakoff (907), and later Moore (689) and Cook (229), recommended its use. Cook recommended four applications at weekly or 10-day intervals. The writers have found that a single dusting, if applied when the disease first appears, may represent the difference between a marketable crop and no crop at all. The best control was obtained when the first application was made when the disease first appeared (229). Moore concluded that sulfur-lime dust (75 to 25) was the most economical and efficient. Applications should begin

at the first sign of the disease and should be repeated every 10 days or 2 weeks thereafter if necessary. Two to three treatments are usually sufficient. Yarwood (1149) found that infected bean leaves sprayed with water reduced the amount of infection. The spores are readily dislodged and are unable to regenerate themselves from haustoria.

Yarwood (1152) also reported a 95-percent eradication of powdery mildew with a spray containing about 0.04 percent bluestone plus a spreader, while a similar control without a spreader required about 2.4 times as much copper in the form of bluestone. He found that bordeaux mixture was not so effective as bluestone. In greenhouse tests Felber and Hamner (341) showed that an aqueous solution of actidione sprayed on bean leaves infected with powdery mildew controlled the disease in 48 hours on the leaf surfaces that received the treatments. Keil and others (551) found that Karathane (dinitrocaprylphenyl crotonate) gave excellent control in 1.5, 3.0, and 5.0 percent dusts and was also effective as an eradicant.

Variations in the resistance of different bean varieties have been observed. Thirty-four varieties subjected to natural field infection were arranged by Moore (689) into three classes as follows: (1) Light infection, (2) moderate infection, and (3) severe infection. The lightly infected group included Asgrow Valentine, Dwarf and French Horticultural, Great Northern, Grenell Rustproof, Hodson Wax, Keeney Rustless Wax, Round Pod Kidney Wax (Brittle Wax), Stringless Green Refugee, Stringless Kidney Wax, and Sure Crop Wax. Investigations by Dundas (304) showed that Pinto, Hungarian, and selections from Lady Washington, Pink, and Yellow were resistant. In addition, Alabama No. 1, Contender, Florida Belle, Logan, Pinto, Tenderlong 15, Ranger, Idaho Refugee, Sensation Refugee Nos. 1066 and 1071, U. S. 5 Refugee, Rival, Topcrop, and Wade have been reported to be resistant to one or more races of the fungus. The fact that many of these varieties may not be resistant in all areas may be attributed to the presence of different races in the various localities.

### Pythium Root Rots

Pythium root rot, sometimes called damping-off, hollow stem, root rot, stem rot, or wilt, may be caused by several species of *Pythium*. Those most commonly found on beans in the United States are: *Pythium ultimum* Trow (299, 671), *P. debaryanum* Hesse (40, 537, 672, 690), *P. aphanidermatum* (Edson) Fitz. (*P. butleri* Gubr.) (449, 627, 672, 1172). Other species causing root rot of beans in the United States are *P. myriotylum* Drechs. (299), *P. helicoides* Drechs. (298), *P. oligandrum* Drechs. (40), *P. rostratum* Butl. (671), *P. pulchrum* Mind. (671), *P. vexans* DBy. (40), *P. anandrum* Drechs. (672), and *P. acanthicum* Drechs. (672).

In Hawaii *Pythium hydnaporium* (Mont.) Schroet. has been reported on beans (756). Meurs reported beans in the Philippines

to be attacked by *P. irregulare* Buis. (669). *P. paroecandrum* Drechs. was isolated from rotting bean pods in India (66). Other reports of *Pythium* on beans include *P. ultimum* from South Africa (1064, 1076); *P. debaryanum* from South Africa (1064), Sweden (423) Puerto Rico (232), Philippine Islands (805), and *P. aphanidermatum* from Japan (56, 991) and China (1168). There have been other references to unidentified species of *Pythium* occurring on beans (671).

### Symptoms

*Pythium* root rot is sometimes confused with rhizoctonia root rot and possibly with root rots caused by other fungi. If the plants are attacked when very young, *Pythium* causes a wet rot and soon kills the plants (fig. 7, D). The disease is then known as damping-off. In such cases the pith of the stem is destroyed, giving rise to the descriptive term "hollow stem." The stem of the plant at or below the surface of the soil may be infected; and from the point of infection the fungus spreads up and down and produces a semisoft, colorless to dark-brown rot (fig. 7, C). If conditions are dry, such as may occur in the greenhouse, the stems may be neither softened nor discolored at the base, but instead are somewhat flattened or collapsed from the soil line upward for a distance of 5 to 10 cm. (299). When half-grown plants become infected, they may survive for a time but they later wilt and finally die. On large, well-developed plants an unimportant injury in the form of brown scars of various sizes is produced near the surface of the soil.

When the weather is hot and moist, a rot of the stem and lateral branches as far as the leaves may occur. This symptom has been referred to as pythium wilt (449). The cortex of the stem becomes soft and slimy and separates readily from the vascular tissue. If the weather is very moist a cottony growth is visible. Large plants 8 to 12 or more inches high are as susceptible as small ones. Soon after the cortex has been softened the leaves curl and become flaccid during the warmest part of the day, but they regain their normal turgidity at night. After a few days the leaves remain permanently wilted, followed by the death of the plant. The mycelium invades the cortex and pith of the stem and in later stages some of the lower branches.

### Causal Organism

#### Physiology

The pythia develop best under moist conditions. The different species vary in their temperature requirements. *Pythium ultimum* and *P. debaryanum* prefer cool temperatures. At high temperatures *P. aphanidermatum* is commonly isolated (299). When unusually warm weather occurs *P. myriotylum* may occur in the field (299). It is found more often in the greenhouse during the hot summer months (299).



FIGURE 7.—A, B, Stem canker caused by *Rhizoctonia solani*; infection may occur at any point on the stem. C, Pythium root rot, caused by *Pythium debaryanum*; note the discolored primary root and the dead fibrous roots. D, Death of bean seedling caused by *Pythium butleri*.

In Colorado pythium wilt caused by *Pythium butleri* occurred only in irrigated fields (449) and at high temperatures. It appeared in Virginia after a long period without rain. Records taken there during the summer showed that, although the soil was dry and no rain had fallen for several weeks, the humidity in the vicinity of the plants was near saturation for several hours during the night. Observations and records seem to indicate that this disease is favored by high temperature and high humidity. In this connection it occurred in the San Joaquin Valley of Cali-

fornia, one of the driest sections of the State, on both snap and lima beans; it has occurred on dry beans along the coast where the temperature is much lower. In both of these regions the beans were being grown under irrigation.

*Pythium aphanidermatum* is the species usually associated with the wilt symptom (449, 450), but *P. ultimum* and *P. myriophyllum* have also been isolated (299).

### Control

*Pythium* root rot is rarely prevalent or destructive enough to call for the application of control measures. Sherbakoff (907) recommended that the soil where diseased plants had grown be drenched with a 1-percent solution of copper sulfate. Seed treatments have been erratic in controlling damping-off. Porter (732) found that beans treated with Ceresan germinated better in soil infested with *Pythium debaryanum* and *P. graminicolum* Subr. than untreated seeds. (See seed treatment, p. 190).

Thin seeding provides better aeration and there is less danger of the organism reaching other plants in the row if the plants are not too close.

### Rhizoctonia Root Rot

#### Geographical Distribution and Economic Importance

*Rhizoctonia* root rot, caused by *Rhizoctonia solani* Kühn, is a cosmopolitan fungus causing diseases of many plants widely separated taxonomically. It is sometimes referred to as stem canker, hollow stem, damping-off, and stem rot. It has been reported on beans from many States in the United States and from many foreign countries (9, 140, 150, 184, 336, 372, 425, 494, 946, 1049, 1071). Its occurrence on lima beans has also been reported (124).

Losses from *rhizoctonia* root rot vary greatly in different localities and from year to year. Five to ten percent loss is not uncommon, and more has been noted in situations especially favorable to the fungus. Nacion (710) reported *Rhizoctonia solani* causing as much as 40 percent loss to lima beans in the Philippines.

### Symptoms

*Rhizoctonia* root rot is worst on young bean plants; on seedlings the disease is of the type known as damping-off. *Rhizoctonia solani* may attack the roots and the stem above and below the surface of the soil. Later the infection may spread to nearby plants. The cankers, often reddish brown (fig. 7, A, B), extend longitudinally and cause somewhat sunken lesions on the stem. If the plant is young and succulent, it usually dies soon after infection. If, on the other hand, the stem is somewhat woody, the plant shows little indication of being diseased, although the yield is generally considerably reduced. The fungus may enter the pith where it causes a brick-red coloration. It has been isolated from the pith in the



region of the lower branches. Sclerotia are often found within the stem of moribund plants.

*Rhizoctonia* is a common soil-inhabiting fungus, and branches and pods resting on the soil frequently become infected. It causes slimy, water-soaked spots on the pods, which serve as sources of infection to beans during transportation.

## Causal Organism

### Nomenclature

The sporiferous stage of *Rhizoctonia solani* was first observed in this country by Rolfs (846) in 1903 on potato stems; it was described by E. A. Burt as *Corticium vagum* var. *solani*. In Europe this same fungus is generally known as *Hypochnus solani* Prill. & Del. or *C. solani*. Rogers proposed the name *Pellicularia filamentosa* (Pat.) Rogers to include the organisms previously classed as *Corticium vagum* var. *solani*.

### Morphology and Physiology

The basidial stage of *Rhizoctonia solani* has been seen a few times on bean plants. The shape of the basidiospores, the character of the hyphal septations, and other morphological characteristics of the fungus are no different from those of this fungus on other hosts. Notwithstanding the fact that the different isolations seem to be very similar morphologically, Matsumoto (658) has obtained evidence to show that isolations from different hosts may differ parasitically. His investigations were carried on with isolations made from potatoes, beans, lettuce (*Lactuca sativa* L.) eggplants (*Solanum melongena* L.), and *Habenana* sp. His results showed that the different isolates differed among other things in their nitrogen metabolism and in their tolerance to acids and alkalis. Inoculation experiments on beans, lettuce, potatoes, and eggplants demonstrated that certain strains would attack all those hosts, others only certain ones, and two strains none of them. Infection took place most readily through the roots. Penetration was mechanical, the fungus entering directly through the epidermis.

Person (761) found that a sector variant produced in culture was less pathogenic than the original culture on beans, soybeans, cowpeas, and English peas. Using beans as a test host, Person (762) distinguished four rather clear-cut groups of isolates on the basis of pathogenicity: (1) isolates from sugarcane and from sclerotia found on white potato tubers, which were not pathogenic on beans; (2) isolates from peas, which caused only a very slight amount of damping-off and only a moderate stem infection; (3) the isolate from rice, which almost completely prevented emergence but caused less severe stem lesions than the pea isolates; (4) isolates from bean, tomato, eggplant, and sugar beet, which were capable of reducing stands somewhat and caused very severe lesions on stems.

Kotila (568) described a strain that causes a blight of sugar beet foliage. It also caused a blighting of bean foliage in addition

to stem cankers. The perfect stage was frequently found on leaf blades and petioles of sugar beet. Field observations indicated that basidiospores play an important role in dissemination of the fungus. The single-basidiospore isolates were found to have cultural characters distinctly different from those of the mycelial isolate from which they were obtained. They also differed among themselves in growth habit. They varied greatly in pathogenicity to sugar beet seedlings, infection ranging from 100 percent to almost none; on sugar beet foliage infection ranged from mild to severe.

Sanford in Canada (870) found that *Rhizoctonia solani* disappeared from the soil in 120 days or less when associated with wheat, oat, barley, or corn plants replanted every 21 days, but was still fairly abundant in soils bearing potato, bean, or pea plants. He believed that the fungus depends essentially on parasitic nutrition for its persistence in the soil.

*Rhizoctonia* root rot is favored by warm weather. Richards (839) found that the severity of the disease depended on the temperature of the soil. The lesions on beans were produced at temperatures ranging from 9° to 29.5° C. (optimum 15° to 18°). Kendrick (554) found that lima bean plantings made so that mean soil temperature was 65° to 70° F. at 6-inch depth for the initial 12 weeks had less *rhizoctonia* root rot infection than those planted later when soil temperature for a similar period was above 70°.

### Control

*Rhizoctonia solani* is chiefly a soil-inhabiting fungus, and as such is difficult to control. Since it is parasitic on a large number of hosts, crop rotation is of little value. Anderson (37) found that beans grown in sand culture with a continuous flow of solutions of high-nutrient salt concentration were markedly more severely attacked than were those grown in solutions of low-nutrient concentration. Solutions favorable to host growth and severe attack were also favorable, upon addition of dextrose, to the growth of the fungus in liquid culture. While impractical on a large scale, destroying diseased plants as soon as they are observed has been recommended. Where *R. solani* is very prevalent, beans should not be planted too close together. The causal organism has been isolated from the seed, and some measure of control may be expected if only disease-free seed is used. Observations in Florida (1021, 1023) indicated that turning under vegetation a short time before planting lima beans and snap beans increases the damage caused by *Rhizoctonia*. Preparing soil 5 days or more in advance of planting reduced the disease incidence (1024). Luttrell and Garren (613) found in Georgia that *rhizoctonia* root rot usually does not appear in plantings made in early spring, but in later plantings it may seriously reduce stands.

Moore and Conover (693) showed that PCNB (pentachloronitrobenzene) as a soil treatment significantly reduced *rhizoctonia* root rot of snap beans. Cardona (188) found in Colombia that the

variety Uribe Redondo was the most resistant variety to rhizoctonia root rot. Machacek and Brown (623) reported that Navy and Great Northern beans were more resistant to rhizoctonia root rot than other varieties tested.

## Rust

### Geographical Distribution and Economic Importance

Bean rust, caused by *Uromyces phaseoli* var. *typica* Arth., was first reported from Germany in 1795 (766). Since then it has been reported from almost every part of the world: Africa (118, 288, 292, 388, 494, 562, 1072), Australia (28, 217, 615), Bulgaria (645), Canada (224), Chile (5), China (997), Cyprus (716), Formosa (Taiwan) (876), France (779), Germany (522), Guatemala (540), India (844, 1043), Italy (769), Japan (578), Mexico (580, 1164, 1206), New Zealand (254), Philippine Islands (831), Puerto Rico (204), Rumania (24, 874), Serbia (809), South America (60, 100, 382, 456, 476, 744), Turkey (129), Union of Soviet Socialist Republics (41, 111), and the West Indies (210). Chardon and Toro (200) reported this rust on both kidney and lima beans in South America. In 1916 Lakon (586) found rust prevalent on various species of *Phaseolus* in Germany.

Rust infection is possible in all localities where a high relative humidity is maintained for 8 or 10 hours, but it is rare at humidities below 95 percent. In the dry parts of California, for example, rust rarely if ever occurs, but in humid parts it occurs annually and frequently as an epidemic.

In only a few early accounts was bean rust said to be serious. Beach (82) reported that it assumed economic importance locally in New York in 1892. Halsted (421) stated that it was serious in New Jersey in 1901, that it was not restricted to cultivated crops, and that varieties differed in susceptibility (fig. 8, A to D). Several years later Whetzel (1112) reported the occasional occurrence of the disease on snap and lima beans in New Jersey. Fromme and Wingard (360) reported that rust sometimes caused complete loss of the snap bean crop in Virginia but that lima beans were almost immune.

In recent years bean rust (fig. 9) has assumed economic importance in a number of States. In Colorado hundreds of acres were completely destroyed in 1927. Large losses occurred at about the same time along the coast in southern California and to a less extent in Virginia, Massachusetts, New Jersey, Louisiana, Washington, and other States. Townsend (1030) reported that in 1938 losses in certain counties in Florida ranged from 40 to 80 percent and that hundreds of acres were never harvested. The following year the losses would have been very high also had it not been for the application of control measures. The writers estimated the losses in one county in Colorado to be approximately \$1,000,000 in 1942. In 1945 (1181) rust was widespread and caused heavy crop losses in some of the major dry-bean producing sections of Wyoming and Montana. In one Montana district rust

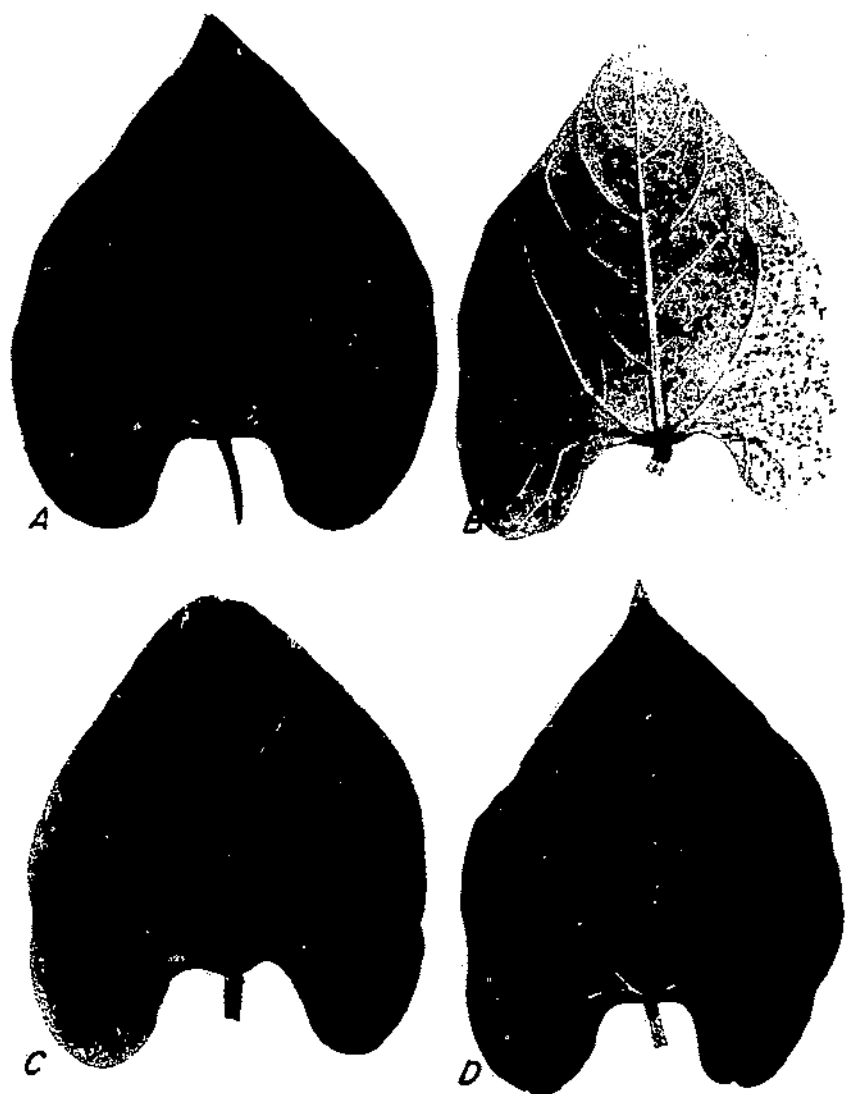


FIGURE 8.—Leaves infected by *Uromyces phaseoli* var. *typica*, showing grades of resistance from most resistant (A) to most susceptible (D).

appeared in practically all fields in 1944, and in 1945 only those fields where control measures were applied were free of disease.

The severity of these losses could be explained on the assumption that the volume of spores has been built up tremendously. Simultaneously, there may have been an increase in the number of physiologic races, thus bringing more bean varieties into the susceptible class.



FIGURE 9.—Typical rust infection on Pinto, a very susceptible bean variety.

### Symptoms

Bean rust attacks the leaves, pods, and very rarely the tender parts of the stem and branches. It is most abundant and conspicuous on the leaves (figs. 8 and 9). Whether the initial infection occurs on the upper or lower surface of the leaf, the symptoms usually appear first on the lower surface. Infection is first evident as minute, almost white, slightly raised spots known as sori. Under favorable conditions they are evident about 5 days after the spores have been applied to the susceptible host.

The germ tubes enter the stomata and grow in the stomatal cavities and intercellular spaces, gradually enlarging the sorus, which in 8 to 10 days ruptures the epidermis and exposes the urediospores. The sorus continues to enlarge for several days longer. On highly susceptible varieties the sorus may reach a diameter

of 1 to 2 mm. (fig. 8, D). In it the rust-colored urediospores appear first. On very susceptible varieties a ring of secondary sori often develops outside the primary sorus, and outside the secondary ring often a third one develops. The individual sori composing the ring may eventually merge and form a single sorus. The occurrence of secondary and tertiary rings of sori depends to some extent on the susceptibility and vigor of the host. Toward the end of the growing season as the plants become older and the leaves somewhat moribund, teliospores replace the urediospores except in tropical regions where they are rarely noted. The sorus gradually becomes black as the dark-brown teliospores replace the urediospores.

## Causal Organism

### Nomenclature

The bean rust fungus was for many years commonly known as *Uromyces appendiculatus* (Pers.) Fr. (357), but this name cannot be used since it is antedated by *U. appendiculatus* Ung. of 1836, which applies to a rust on a very different host.

Persoon, as the starting point author, gave the name *Uredo appendiculata phaseoli* (767). Rebutish (815), working in Germany, first gave the fungus specific rank as *Puccinia phaseoli* Reb. Transfer to *Uromyces* was made by Winter in 1880. Arthur (52) established the variety *typica*, which under the International Code of Botanical Nomenclature becomes *phaseoli*, the complete name of the fungus then being *Uromyces phaseoli phaseoli*. Other names have been assigned to the fungus at later dates but they have been relegated to synonymy.

### Morphology and Physiology

The causal fungus, now called *Uromyces phaseoli* var. *typica*, is one of a large number of obligate parasites of cultivated plants. It is autoecious, i. e., it has its life cycle confined to a single host.

The complete life cycle of the bean rust was produced by Andrus (43) in a greenhouse. The aecia are rare in nature, but have been observed by the writers in beanfields in Oregon, by Eastham (312) in British Columbia, and by McWhorter<sup>7</sup> in Washington.

The urediospores, frequently called the summer spores, are produced in great numbers in sori on the upper and lower surfaces of the leaf. They spread the disease over great distances and cause severe epidemics. The urediospores will germinate as soon as they are mature, and under favorable conditions they produce another generation of the same kind of spores in about 10 to 15 days. The rapidity with which they increase enables them to produce several generations in a single season. Naito (711) studied the influence of temperature on germination of the urediospores and found that in water moderate germination occurred from 10° to 25° C. Between 10° and 20° germination started 1 hour after sowing of the spores and was completed after 4 hours. Maximum growth of the germ tubes occurred at 20°.

<sup>7</sup> From personal correspondence.

The catenulate urediospores are dilute brown, spiny, one-celled, and thin-walled. They are globoid to ellipsoid and about  $8\mu$  to  $24\mu$  (average  $22.5\mu$ ) by  $20\mu$  to  $37\mu$  (average  $28\mu$ ). Generally they have two equatorial or superequatorial pores, which sometimes are almost indistinguishable.

Sometimes urediospores and teliospores may be observed in the same sorus. The teliospores are more abundant and appear earlier in northern latitudes than in milder climates. In the Southern States and Hawaii, teliospores are said not to be produced; this might indicate that temperature, light intensity, or both influence their production. The age of the leaves and the physiological condition of the host appear to exercise some influence on teliospore production. There is some evidence that some physiologic races are more inclined than others to produce teliospores under identical conditions. Some races that produce only urediospores in a semitropical climate develop teliospores when transported to a northern region, but all races do not behave alike. One race from southern California cultured for several years in the greenhouse at Washington, D. C., did not produce teliospores, although collections from many other regions normally produced teliospores there. The writers have repeatedly observed that in the more northerly regions teliospores are not so readily produced in the late spring and early summer, when the days are the longest and the light more intense, as in the late summer and early fall. Whether this is caused by the length of day, light intensity, or a combination of such factors has not been determined. Waters (1936) found that factors such as light, temperature, and moisture may so influence the host that the fungus reacts by changing from the uredinal to the telial generation, or under proper manipulation in the reverse direction.

The teliospores are dark brown, one-celled, and thick-walled. They are frequently called winter spores, largely because they develop late in the season and serve to carry the organism through the winter months. They will not germinate without a resting period. This period can be shortened by storing them at a freezing temperature. The teliospores are amphigenous and globoid to broadly ellipsoid and range in width from  $20\mu$  to  $28\mu$  (average  $24\mu$ ) and in length from  $25\mu$  to  $35\mu$  (average  $30\mu$ ). The wall is uniformly  $3\mu$  to  $4\mu$  thick with a hyaline papilla over the pore. The spores are mostly smooth. The pedicel is hyaline and short.

Sempio (1892) found that temperatures of  $34^{\circ}$  to  $36^{\circ}$  C. for  $2\frac{1}{2}$  days completely killed bean rust mycelium in the leaf when the treatment was applied 4 days after inoculation. He (1893) also studied the effect of carbon dioxide, light, radiation, and humidity on infection. Later, he showed that darkness during the first 3 days after inoculation stimulated infection and that during the period of mycelial growth darkness had a retarding effect. Exposure to darkness during the fruiting period showed less infection than the control. Sempio (1894), investigating metabolic resistance in plants, noted that bean leaves inoculated with urediospores and kept in the dark in distilled water for 10 days were yellow but showed no infection. Leaves kept in a 1-percent levulose solution

under the same conditions remained green and many became infected. Yarwood (1158) showed that infection on leaves was directly correlated with the amount of guttation. He also found that when pinto bean primary leaves infected in localized areas with the uredinial stage of the fungus were reinoculated, infection from the second inoculation did not occur close to the first infection (1159). Wei (1097) found that an excess of nitrogen increased the amount of infection of the leaf, while potassium had the opposite effect. The effect of phosphorus was not definite. He also indicated that light was essential during the infection period for successful entrance of the fungus and that reduction in the light intensity prolonged the incubation period.

Yarwood (1157) showed that rust-infected leaves were more susceptible to virus infection of alfalfa mosaic, tobacco necrosis, and tobacco ring spot.

Cohen (218) reported that water-infiltrated bean leaves were more resistant to rust than normal leaves. The infiltrated condition of the leaf did not interfere with appressoria formation, stomatal penetration, or the formation of substomatal vesicles, but subsequent infection hyphae were rare.

#### Dissemination

Bean rust, which is not seed-borne, may be disseminated locally by farm implements, insects, and animals, but the wind is the principal agent in the dissemination of the spores over long distances.

As urediospores remain viable for only a short time, it is probable that too few overwinter to give rise to an epidemic. Townsend (1080) believed that in Florida rust epidemics depended on the dissemination of urediospores from the States farther north, because teliospores do not develop there to carry over the fungus from year to year.

Teliospores, which require a rest period and retain their viability for many months, are largely instrumental in carrying over the organism from year to year.

#### Hosts

Arthur (52) listed several different species of *Phaseolus* as being susceptible to infection by bean rust, as, for example *P. multiflorus* (*P. coccineus*), *P. polystachyus* (L.) B.S.P. (*P. polystachios*), and *P. sinuatus* Nutt. (*P. sinuatus* Torr. & Gray). The writers have obtained good infection on *P. acutifolius* var. *latifolius* and on some varieties of *P. lunatus*, and mild infection on a number of other hosts. In general lima beans are more resistant than the dry and snap beans, but they show a similar type of reaction to the several physiologic races of bean rust. Fromme and Wingard (361) inoculated a number of varieties of lima bean and cowpea, one variety of asparagus-bean, and one variety of horsebean without obtaining infection, but they did obtain infection on one variety of lima bean.

Fromme (359) concluded from his investigations of the cowpea rust that the rusts on *Phaseolus adenanthus* G. Mey., *P. aniso-*



*trichus* Schlecht., *P. atropurpureus* Moc. & Sess., *P. coccineus*, *P. dysophyllus* Benth., *P. lunatus*, *P. obvallatus* Schlecht., *P. polystachyus*, *P. retusus* Benth. (*P. metacalfi* Woot. & Standl.), and *P. vulgaris* were correctly named *Uromyces appendiculatus* (*U. phaseoli* var. *typica*). He expressed some doubt as to the status of rusts on certain other legumes. Sydow and Sydow (1986) reported *U. appendiculatus* on *P. mungo* L., *Vigna sinensis* (Torner) Haask, and *P. vulgaris*.

A rust on *Strophostyles helvola*, occurring commonly in Virginia and other States along the Atlantic coast, was thought at one time to be closely related to bean rust, if not identical with it. By cross inoculation, Harter, Andrus, and Zaumeyer (447) demonstrated conclusively that the two were very different pathologically, although they were somewhat alike morphologically.

### Control

In the United States, dusting with sulfur at frequent intervals, if begun when the plants are very small, has given good control. Townsend (1030) found that sulfur dust or sprays gave good control, but that bordeaux mixture was of no value. Zaumeyer and Goldsworthy (1188) found that liquid lime-sulfur, chlorinated naphthoquinone, and nabam were effective in eradicating 24-hour infections of bean rust. Zaumeyer (1182, 1183) reported that sulfur applied at the rate of 20 to 25 pounds per acre was a very effective method of control. If finely ground sulfur (325-mesh or finer) is dusted on beans before any rust is noticeable, the treatment protects the crop from infection. The sulfur destroys the rust pustules present at the time, preventing the spread of spores and the formation of their infection centers. If dusting is done after infection has become widespread, it must be repeated and the control is not so effective.

In the State of Washington (180) 1 to 6 applications of 1 in 100 lime-sulfur spray plus 1 in 100 stop fire (Saonichton KS-resin spray) gave good control of rust in fields with overhead irrigation. Milbrath (675) found that dusting with Kolo dust greatly reduced rust infection when applied at 7-day intervals, if the dusting program was started before aeciospore formation. He also reported that large numbers of teliospores adhere to old stakes from fields of pole beans infested with rust and dipping the stakes in lime-sulfur (1 to 10) or in copper sulfate (8 to 100) prevented infection from these stakes when used in newly planted beanfields. Yarwood (1150) reported killing of 5-day-old uredial pustules of bean rust without host injury by exposing the plants to the vapors from lime-sulfur solutions or from dilute hydrogen sulfide gas. He (1151) also showed that by increasing the amount of lime to bluestone solutions, the protective value of these solutions for bean rust was increased but the eradicant value was decreased. Later, he (1154) reported that penetrating applications of dilute lime-sulfur were effective in eradicating bean rust 8 days after inoculation with little host injury. The effectiveness was increased with increasing lime-sulfur concentrations and with the addition of a spreader or zinc sulfate to the lime-sulfur-spray (1153). The

vapors from lime-sulfur solutions ranging from 0.001- to 100-percent lime-sulfur eradicated bean rust from 4- and 7-day-old infections with a maximum therapy from 0.1-percent lime-sulfur. Yarwood (1156) also found that when bean leaves which had been inoculated for 3 to 5 days were placed in sealed jars with the vapors from sodium sulfide solutions that rust mycelium was killed without leaf injury, provided the pH and the concentration of the sodium sulfide and the time and temperature of exposure to sodium sulfide were properly adjusted. Yarwood (1161) determined by radioautographs of bean leaves inoculated with rust 4 to 21 days before treatment that infected areas showed greater uptake of tracer sulfur than noninfected areas.

Wager (1062) found that in southern Africa bean rust was controlled by spraying the plants at weekly intervals, especially just after rains, with a 4-4-50 bordeaux mixture. Straib (979) reported that in Germany old bean poles are disinfected in a 0.1 percent formalin solution before being used. Repeated sprayings with bordeaux mixture are practiced, and sulfur is used only when the summer rainfall is low. In Australia (11) dusting with sulfur at the rate of 15 to 25 pounds per acre at intervals of 1 to 2 weeks until a few days before flowering is recommended for rust control. Spraying with wettable sulfur is also recommended.

Brien and Jacks (137) reported that lime-sulfur (1 to 150) plus colloidal sulfur 2 pounds to 100 gallons of water was effective in controlling bean rust in New Zealand. In Holland, Van der Vliet (1054) showed satisfactory control of bean rust with zineb used in a mist blower at a concentration of 5 percent or as a spray at 0.35 percent.

The breeding of rust-resistant beans is complicated because of the relatively large number of physiologic races involved. Harter, Andrus, and Zaumeyer (447) demonstrated the existence of 2 physiologic races on the basis of their reaction to certain Kentucky Wonder bean types. Dundas and Scott (309) reported 2 additional races. Later, Harter and Zaumeyer (452) identified 20 physiologic races from collections of bean rust obtained from various parts of the United States and Hawaii by the use of 7 differential hosts. In Hawaii (457, 755, 757) several new strains of rust were reported which attacked varieties that were previously resistant. It is not known whether these strains differ from those reported in the United States. Dundas (308) reported mutation in urediospores when stored at 0° C. for a year. Isolations from such stored material inoculated to differential varieties differed in every case from the race as stored, and in most instances from previously described races. Fisher (346) reported 10 new races, and Marcus (652) isolated a race in Maryland that was the same as one of those reported by Fisher. Sappenfield (871) described another new race from New Mexico. Menezes (664) reported 4 races from Brazil, all of which had been reported previously from the United States. Waterhouse (1085) found a new race in Australia which infected bean varieties that were resistant to the previously described races in that country.

The breeding of rust-resistant beans naturally becomes more

complicated as the number of physiologic races increases. A hybrid resistant to several races may prove to be susceptible to those not known at the time the crosses were made. A few bean varieties, however, show a high degree of resistance or at least tolerance to many of the races so far identified. Wingard (1133) listed a number of varieties that could be used in localities where rust was prevalent. Many of the varieties were later found to be susceptible. A more complete list of the degree of resistance or susceptibility of a large number of commercial varieties to 14 physiological races was published by Harter and Zaumeyer (452). Their lists contain a few varieties that were resistant or tolerant to each of the physiologic races.

The size of the mature rust pustule is the criterion for fixing the degree of susceptibility and resistance. Wingard (1134) grouped varieties in 3 classes on the basis of their reaction to rust: (1) Immune, (2) those showing severe flecking as a result of infection but without the production of many spores, and (3) those on which numerous sori are produced. He showed histologically that resistant varieties are hypersensitive (1135). Wei (1097) distinguished 5 major types of infection (0 to 4). He considered 0, 1, and 2 as resistant, and 3 and 4 as susceptible. Harter, Andrus, and Zaumeyer (447) used a scale of 0 to 10, 0 denoting immunity and 10 the highest degree of susceptibility, with intermediate grades between these extremes. Readings lower than 5 denoted grades of resistance; those from 5 to 10, grades of susceptibility.

Harter (443) released 2 white-seeded, rust-resistant Kentucky Wonder beans, and Zaumeyer and Harter (1193) developed 2 rust-resistant pinto varieties, No. 5 and No. 14. Wingard (1137) developed 10 rust-resistant pole varieties that were named Virginia Victory No. 1 to No. 10. Frazier and Hendrix (353, 354) introduced the rust-resistant Hawaiian Wonder pole variety that is adapted to high elevations in Hawaii where rust is serious. Wellhausen and coworkers (1106) reported the bean varieties Rocamex 1, 2, and 3 were resistant to the rust prevalent in the central Plateau of Mexico. Cass Smith (196) and others reported a new rust-resistant high-yielding pole bean in Australia known as Westralia that is a natural cross between Golden Harvest and a rust-resistant strain of Kentucky Wonder.

Beans should not be planted on land that produced a heavily rust-infected crop the preceding year or close to stacks of old bean straw infested with rust, as the new crop may become infected earlier in the season and more severely than it would have under other conditions. Infested bean straw should not be used for feeding or bedding livestock.

## Sclerotinia Wilt

### Geographical Distribution and Economic Importance

Sclerotinia wilt, caused by *Sclerotinia sclerotiorum* (Lib.) DBy., and also known as white mold (307) and wilt, causes heavy

losses in the field and during transportation (p. 176). It attacks practically all vegetable crops.

*Sclerotinia* wilt has been reported from Argentina (455), Australia (13, 102), Bermuda (1114), Canada (68), Denmark (147), England (98), France (728), Germany (523), Italy (67, 112), Mexico (1162), New Zealand (136), and Tanganyika Territory (16, 1077). In 1903 Marchal (649) reported in Belgium a disease of beans caused by *Sclerotinia trifoliorum* Eriks. Weston (1111) in England and Cormack (244) in Canada also reported this species causing injury to beans.

*Sclerotinia sclerotiorum* is widely distributed and may be found in beanfields in practically every part of the United States where weather conditions are favorable (691). In 1939 the disease became a serious problem in parts of southern Florida. In some years it is a serious disease of beans in the irrigated regions of Wyoming, Idaho, Montana, and Oregon. It was quite widespread in Michigan in 1953 and was found in more fields in Colorado in that year than ever before; in some cases it caused losses up to 50 percent.

The disease frequently occurs after a period of warm, humid weather. A few days of such weather may result in large crop losses. In the Middle Atlantic States outbreaks of *sclerotinia* wilt are more prevalent on the fall than on the spring crop, especially if warm rainy weather occurs in September. Biffen (98) found that the sclerotia were sometimes formed on the roots of plants grown on heavy clay. There is, however, no general correlation of soil type or texture with the amount of crop loss. In general, the disease is more prevalent in the Southeastern States, some of the irrigated sections of the Mountain States, and in the moister areas of the Pacific Northwest than in other areas of the United States. Crops grown on low ground surrounded by woods, which reduce air circulation, suffer the greatest loss. The incidence of the disease is also increased by thick planting and the prevalence of a luxuriant growth of weeds.

The losses vary greatly from year to year. In the Northern States *sclerotinia* wilt occurs usually in July and August and has been so severe that entire fields have been plowed under. McClintock (616, 617) reported serious losses in Virginia in 1915 and a loss of more than 30 percent in parts of the State in 1916.

### Symptoms

The first symptoms of *sclerotinia* wilt under field conditions appear as irregular-shaped, water-soaked spots on the stems, followed later by similar lesions on the branches and even the leaves. The causal organism grows rapidly, and the lesions gradually enlarge, causing a somewhat soft watery rot of the affected parts. Often a sticky, brown liquid exudes from the pods and stands out on them in small drops. After the watery stage, the affected tissues dry out and become light and punky. If several days of warm, wet weather follow the initial infection, a cottony growth of the fungus spreads over the branches and leaves. A



FIGURE 10.—*Sclerotinia* wilt, caused by *Sclerotinia sclerotiorum*, showing pod and stem infection. Spherical to elongated white bodies on pods are the formation of sclerotia; black bodies on pods in lower center are mature sclerotia.

few days thereafter the sclerotia begin forming and frequently occur in large numbers (fig. 10).

The leaves of the plant turn first pale, then bright yellow, and later brown. If the lesions are numerous on the stem, death may follow within a few days.

Although the fungus may spread from the branches and leaves to the nearby pods (fig. 10), those in contact with the soil are the ones most frequently attacked (fig. 10). The water-soaked lesions may occur on any part of the pod. They gradually enlarge until they extend from the dorsal to the ventral suture and may finally attain an inch or more in length. The middle lamella of the parasitized tissue is dissolved, giving it a slimy water-soaked consistency.

### Causal Organism

#### Nomenclature

The American workers had almost exclusively referred to the causal fungus as *Sclerotinia libertiana* Fekl. until the appearance in 1924 of a brief article on the nomenclature involved by Wake-

field (1066), who pointed out that Europeans had generally used the specific name *sclerotiorum* and not *libertiana* so that the valid name is *S. sclerotiorum* (Lib.) DBy. A careful scrutiny of the facts relating to its synonymy would seem to justify her conclusions. The fungus was described and distributed in 1837 by Libert<sup>8</sup> under the name of *Peziza sclerotiorum* Lib. Fuckel later transferred the species to his genus *Sclerotinia* and changed the specific name to *libertiana*, instead of using the specific name *sclerotiorum* already given to it. Other names applied to the fungus were *Peziza sclerotii* Fckl., *Phialea sclerotiorum* (Lib.) Gill., and *Hymenoscypha sclerotiorum* (Lib.) Phill.

### Morphology and Physiology

*Sclerotinia* is one of the numerous genera in the order Pezizales. It produces an abundant mycelial or vegetative growth and eventually forms resting bodies called sclerotia. The mature sclerotia are irregular-shaped, black, hard bodies varying in size from 1 mm. to several in diameter. They possess a considerable degree of resistance to drought, cold, and other destructive agencies. After a period of cold weather and in the presence of a sufficient amount of moisture, the sclerotium gives rise to the apothecium. This organ is supported by a long stalk on which is borne a saucerlike structure, the ascocarp, that bears the asci. If the spores, upon escaping from the asci, lodge on a susceptible host, a new infection may originate. The apothecia are generally scattered, smooth, pale, and funnel-shaped, and vary in diameter from 4 to 8 mm. The stalk is slender and often curved. According to Saccardo (861) the asci are cylindrical,  $125\mu$  to  $130\mu$  long by  $8\mu$  to  $10\mu$  wide, and dilute blue. The spores are arranged in a single row in the ascus. They are ellipsoidal, guttulate, and  $9\mu$  to  $13\mu$  long by  $4\mu$  to  $6.6\mu$  wide. The paraphyses are somewhat club-shaped. Jones (542) gave somewhat different spore measurements from those of Saccardo for the same organism on *Helianthus annuus* L. She recorded also a greater variation in the length and width of the asci. Such differences in size of these structures may be expected when material is taken from hosts widely separated in relationship.

Tanrikut and Vaughan (990) have shown that the nutrient requirements of the fungus are not specific. Although the fungus shows preferences for certain elements, it also has the ability to grow on almost any substrate, regardless of the presence or absence of any specific mineral or organic nutrient.

### Pathogenicity

McClintock (618) isolated *Sclerotinia sclerotiorum* from beans, tomatoes, parsley, cauliflower, and eggplant in Virginia. Cross inoculations made with the *S. sclerotiorum* from beans and lettuce proved the two isolates to be identical. The writers have on several occasions isolated *S. sclerotiorum* from beans and successfully inoculated other plants, thereby verifying the results of

<sup>8</sup> LIBERT, M. A. PLANTAE CRYPTOGAMICAE ARDUENNAE. [Exsiccati]. No. 326. 1837.

McClintock and others. Ramsey (806) showed by cultural studies and cross inoculations to lettuce and certain other crops that *S. minor* Jagger was identical with *S. libertiana*.

#### Dissemination

Just how *Sclerotinia sclerotiorum* is disseminated from one field to another is not definitely known, although there are several possibilities. The sclerotia are often embedded in the host, and any method whereby the affected plants are carried from one field to another would accomplish the introduction of the parasite in a new environment. The storage of vegetables or the feeding of them to livestock, with the subsequent spreading of the manure and the refuse to the field, would result in the introduction of the fungus into new locations. Cultivators and other farm implements, as well as the roaming of livestock from one place to another, would tend to scatter the sclerotia. The spores on being liberated from the asci, like those of many other similar fungi, are disseminated by the wind and rain and probably by insects.

Blodgett (103) isolated the fungus from infected seeds. Hungerford and Pitts (504) found that less than 1-percent seed-borne infection resulted when beans from seriously infected fields were planted the year following production. It is likely that a small percentage of infected seeds and sclerotia may pass when the beans are milled or even handpicked, and thus provide a means of introducing the fungus into new areas.

#### Control

No adequate control measures are known for sclerotinia wilt. The numerous sclerotia produced in the soil and on diseased plants are difficult to remove or destroy. Sanitary measures should be employed when practical. Burning the old bean vines in the field after the crop has been removed has been recommended and doubtless will reduce the amount of infection.

The rotation of 2 or 3 years or longer with such crops as corn, small grains, and hay should be practiced when possible. How long a rotation would be necessary to kill out the fungus is not known. However, Pollock (780) showed that the *Sclerotinia* causing the brown rot of stone fruits will remain viable in the soil for at least 10 years. Hungerford and Pitts (504) found that sclerotia held in storage for 7 years germinated readily and produced ascospores which caused infection. Crops such as lettuce, cucumbers, eggplant, peas, tomatoes, turnips, and celery, which are susceptible to *S. sclerotiorum*, should be avoided (965).

There are no varieties of beans immune to the disease. McClintock (616) stated that Bountiful, Stringless Green Pod, Celestial, Golden Wax, and Extra Early Black Valentine are more tolerant than Extra Early Red Valentine and Extra Early Refuge. More recent studies have not corroborated these results.

According to Moore (692), a high percentage of the sclerotia can be destroyed by flooding infected fields for periods of 3 weeks

or more. Because high temperatures hasten decay, the best season for flooding is summer. Stoner and Moore (978) showed that moving water rotted sclerotia in 20 days when covered to a depth of 3 inches, whereas 35 days were required to rot them with static water.

Broadcast applications of calcium cyanamid at the rate of 1,000 pounds per acre have given very good results on marl soils but not on sandy soils (694). Cyanamid injures germinating seed; therefore, it must be broken down through natural chemical action in the soil before the treated area becomes safe for planting. Bridgmon and Starr (133) in Wyoming found that the application of 900 pounds per acre for controlling sclerotia was not encouraging.

Since high humidity favors infection, any method of bean culture that will reduce the amount of moisture in the surface soil and around the plants will aid in controlling the disease. The field where the disease is found should not be irrigated more often than necessary. Wider spacing of rows and reduced rates of seeding, which prevent the vines from meeting early in the season and allow better air circulation, are two other methods for reducing the moisture around the plants and thus checking development of the rot fungus.

Blodgett (103) recommends that bean straw and cull beans contaminated with sclerotia should not be fed to animals if the manure is to be used on land where beans may soon be planted. Bridgmon and Starr (133), however, report that in Wyoming the sclerotia were destroyed by passage through the alimentary tract of cows and by trampling of livestock. Screenings from bean cleaning mills that may contain large quantities of sclerotia should be burned.

Workers in Oregon (270, 1046) reported significant reduction of aerial infection by using bismuth subsalicylate, bioquin, ziram, and Flotox as a dust and ziram plus Sulfuron, ferbam plus Sulfuron, and Cop-O-Zinc as sprays in the Northwest. Later they (1047) reported that ziram and Cop-O-Zinc, either as dust or spray, gave good control. Sprays were more effective than dusts.

## Southern Blight

### Geographical Distribution and Economic Importance

Southern blight, southern wilt, or crown rot, caused by *Sclerotium rolfsii* (Curzi) West was first recognized as a distinct disease by Rolfs (847) for whom Saccardo (861) named the species in 1911. It causes diseases of a large variety of crops besides beans in the United States (759, 993) and in the tropical and semitropical regions (190, 920, 1110) of the world. Many of the susceptible crops are grown in rotation with beans, and most of the soils in the southern part of the United States and along the Pacific coast are infested. No estimate is possible of the losses to beans caused by *S. rolfsii* alone. It also causes decay of beans in transit (p. 175).



## Symptoms

The symptoms produced by *Sclerotium rolfsii* are sufficiently clear-cut to distinguish southern blight from root and stem diseases caused by other fungi. The earliest symptoms are a slight yellowing of the lower leaves and the water soaking and slight darkening of the cortex of the stem just below the soil line. As the disease progresses, leaves in the upper branches of the plant become yellowed and eventually drop off. The infection of the underground part of the stem and taproot extends downward and destroys the cortex, which may readily be separated from the stele. Occasionally the causal organism may invade the vascular bundles of the stem as far as the lower branches, causing a dark discoloration of this tissue. At the base of the stem and on the ground about the plant a moldy growth of white mycelium intermixed with a large number of sclerotial bodies is produced in all stages of maturity. The mycelium extends into the soil about the stem, and if the plant is pulled up a collar of soil and mycelium adheres to it. If there has been an abundance of rain, the softening of the cortex will extend 2 inches or more above the soil line. Coarse, fan-shaped mycelial strands radiate over decayed pods or other decayed vegetable matter in contact with the soil.

## Causal Organism

### Nomenclature

*Sclerotium rolfsii* was supposedly a sterile fungus. No fruiting stage in its life history was known before 1926. Reproduction was thought to be accomplished wholly by means of the hyphae and sclerotia. Nakata (712) collected cultures from various parts of the world and obtained spore development in one culture from Japan and another from America. From cultural and morphological studies he believed the fungus was identical with *Hypochnus centrifugus* (Lév.) Tul. Bresadola (131) had transferred this species earlier to *Corticium*, and this disposition of the species was followed by Von Höhnelt (491) and other workers. Burt, in his monograph (177, pp. 206-208), reported its occurrence on decaying wood, leaves, and fallen branches without any suggestion of a sclerotium stage. Curzi (261) also reported that he had found a perfect stage, which he named *C. rolfsii*. In 1947 West transferred it to *Pellicularia rolfsii*. Barrett (69) observed the basidial stage of the fungus in the United States. West (1107) found a perfect stage on climbing fig in Florida, which he placed in the genus *Pellicularia*.

### Morphology and Physiology

The hyphae are somewhat coarse, the cells ranging from 150 $\mu$  to 250 $\mu$  long by 2 $\mu$  to 9 $\mu$  wide. According to Higgins (486) they are characterized by the presence of clamp connections and a peculiar method of branching, which are valuable aids in fixing its taxonomic position. The sclerotia ( $\frac{1}{2}$  to 1 $\frac{1}{2}$  mm. in diameter), at first white but becoming brown with age, are more or less

smooth and glossy and somewhat resemble mustard seed. Higgins (486) pointed out that the affinity of *Sclerotium rolfsii* to the Basidiomycetes is indicated by the septate binucleate mycelium and by the clamp connections at the septa.

*Sclerotium rolfsii* is one of those organisms whose development is closely associated with weather conditions. It prefers a warm climate and considerable soil and air moisture. When a combination of these conditions occurs, the disease is active and a large percentage of the plants in a field may be killed. The summer of 1937, with reasonably high temperatures accompanied by an abundance of rain, provided ideal conditions for southern blight to flourish in most of the regions along the Atlantic seaboard.

The causal organism is said to be susceptible to injury by low temperatures, and for that reason it does not usually damage crops to any extent in the North. However, it is found as far north as Illinois, Maryland, and New Jersey, where it may cause considerable damage. Fajardo and Mendoza (335) studied 8 isolations of *Sclerotium rolfsii* from various hosts in different parts of the Philippine Islands and found the isolates fell into 3 groups based on variation in growth rate, type of colony, time required for sclerotial formation, and number, size, and color of the sclerotia. They found that sclerotia buried at 5 to 6 inches were killed after 45 to 60 days. Milthorpe (683) studied 8 isolates and showed that the abundance and type of vegetative growth are determined largely by the nature of the medium and temperature. Abundance and size of the sclerotia, but not shape or color, are also affected by these factors. The optimum temperature for growth is 30° C. This growth decreases markedly as the temperature drops to 15° or as it increases to 37°.

#### Dissemination

The dissemination of *Sclerotium rolfsii* differs from that of many other fungi. Since spore production is not known to occur in the United States, its dissemination is assumed to be by means of the sclerotia and the mycelia.

The fungus may spread several feet through the soil, and the mycelium can often be traced from one plant to another. Cultivators and other implements used on the farm naturally drag diseased plants to new locations in the field and conceivably to additional fields. By means of such plants as sweetpotatoes, tomatoes, and other crops that are grown in seedbeds for commercial purposes and shipped perhaps long distances, the fungus is distributed more widely. To discard all diseased plants is impossible as the early stages of infection cannot be detected. Sclerotia can pass through the digestive tract of cattle and sheep without complete loss of vitality (598).

#### Pathological Histology

Milthorpe (683) studied the mechanism of parasitism in bean hypocotyls. The fungus penetrates plant tissue mechanically and

gives rise to coenocytic intra- and intercellular hyphae. Death of the cytoplasm precedes the invading hyphae by one cell layer. Dissolution of the middle lamellae occurs shortly after entry. Protopectinase and pectinase, but not pectase, are produced and are responsible for its toxic action. Higgins (487) believed that the death of cells is caused by the toxic action of oxalic acid and that oxalic acid is secreted by the fungus hyphae.

### Hosts

There are innumerable hosts for *Sclerotium rolfsii*. Infection experiments carried out by Harter (437) and others (366, 994, 1142) clearly demonstrated that *S. rolfsii* is parasitic on a number of hosts widely separated in taxonomic relationship. Practically all vegetables and many weeds are susceptible. The fungus frequently develops on trees and shrubs. Although its complete host range has never been determined, little or no infection has been observed on such hosts as grasses, small grains, cotton, and corn.

### Control

No very effective measures can be recommended for the control of southern blight. The destruction of diseased plants and the suppression of susceptible weeds may help to reduce the loss. Rotation with cereals and other crops resistant to the disease is a precaution that may assist to some extent. Luttrell (613) found seed treatment ineffective. Borzinia and Picco (115) reported variation in susceptibility and resistance in five varieties they tested.

## Fungus Diseases of Minor Importance

### Alternaria Leaf Spot

Alternaria leaf spot, caused by *Alternaria* sp., was reported from central Florida in 1951, causing considerable damage to about 100 acres of beans. The symptoms first appear on the leaflets as small, irregular, reddish-brown flecks with a dark-brown border. As the spots increase in size, they become circular with concentric zones within the affected area. The older portion of the lesion may fall out, leaving a shot-hole appearance. Lesions often coalesce, forming large areas of dead tissue. Other symptoms on leaves may appear as individual leaflets dying back from the tip or from the margin, and the lesions may kill the entire leaf. Sometimes the central growing point of the plant is killed.

Although *Alternaria fasciculata* (CKe. & Ell.) L.R. Jones & Grout has been reported on beans in the United States (984), it is not known whether it has caused any damage to the crop as did the species from Florida. Neergaard (720) reported *A. brassicae* f. *phaseoli* Brun. occurring in Denmark in 1894. The spore measurements of this species vary considerably from those of the Florida isolate and appear to be distinct.

No control measures are known at the present time. It can be assumed, however, that control measures for other *Alternaria* species would also control this species.

### Angular Leaf Spot

#### Geographical Distribution and Economic Importance

Angular, or gray, leaf spot, caused by *Isariopsis griseola* Sacc., is widely distributed in the United States and abroad. Saccardo (354) made the first report of it in 1878 from Italy. It has also been reported from Argentina (950), Austria (146), Australia (4), Brazil (696, 915), Bulgaria (573), French West Africa (646), Germany (411, 563), Hawaii (191), Japan (511), Kenya (622), the Netherlands (881), Palestine (818), Peru (375), Portugal (263), Rumania (875), U.S.S.R. (Russia) (524, 727, 912, 913, 1056), Spain (88, 1037), Southern Africa (287), Tanganyika (1071), Trinidad (1019), Turkey (130), and Yugoslavia (919). Brock (141) regarded it as the most important disease affecting seed crops on the south coast of New South Wales in 1948 and 1949. It has been reported to the writers that it is one of the most common diseases of bean in Columbia.

Angular leaf spot (239) has been reported at various times from most States and Puerto Rico. Although it reduces the vitality of the plants and lowers yields, it usually is not considered of much economic importance. Occasionally it has caused considerable damage to beans in West Virginia and Connecticut. Locally in Florida many plants affected by angular leaf spot have been defoliated, and the crop has been practically ruined in some years. In 1918 pod spotting and defoliation caused by *Isariopsis* resulted in widespread damage in Puerto Rico. Some native varieties showed some resistance.

#### Symptoms

One of the outstanding symptoms of angular leaf spot is described by this common name. Spots, which originate on the underside of the leaf, are delimited by the veins and veinlets. The lesions, which are gray at first and brown later, do not have colored borders. This absence of color in the leaf and the striking angularity of the spots (fig. 11) distinguish this disease from the blotch caused by *Cercospora cruenta* and from other leaf diseases. In a few instances angular leaf spot has caused almost complete defoliation of pole beans, beginning with the lowest leaves. Leaves are infected more frequently than pods.

On the pods the spots are superficial at first and have nearly black borders and reddish-brown centers. The centers and borders are sharply defined. The spots vary in size, and ultimately they may become so crowded that they coalesce and occupy the width of the pod. The centers of the spots are studded with columnarlike growths, or coremia (fig. 11, C, D).

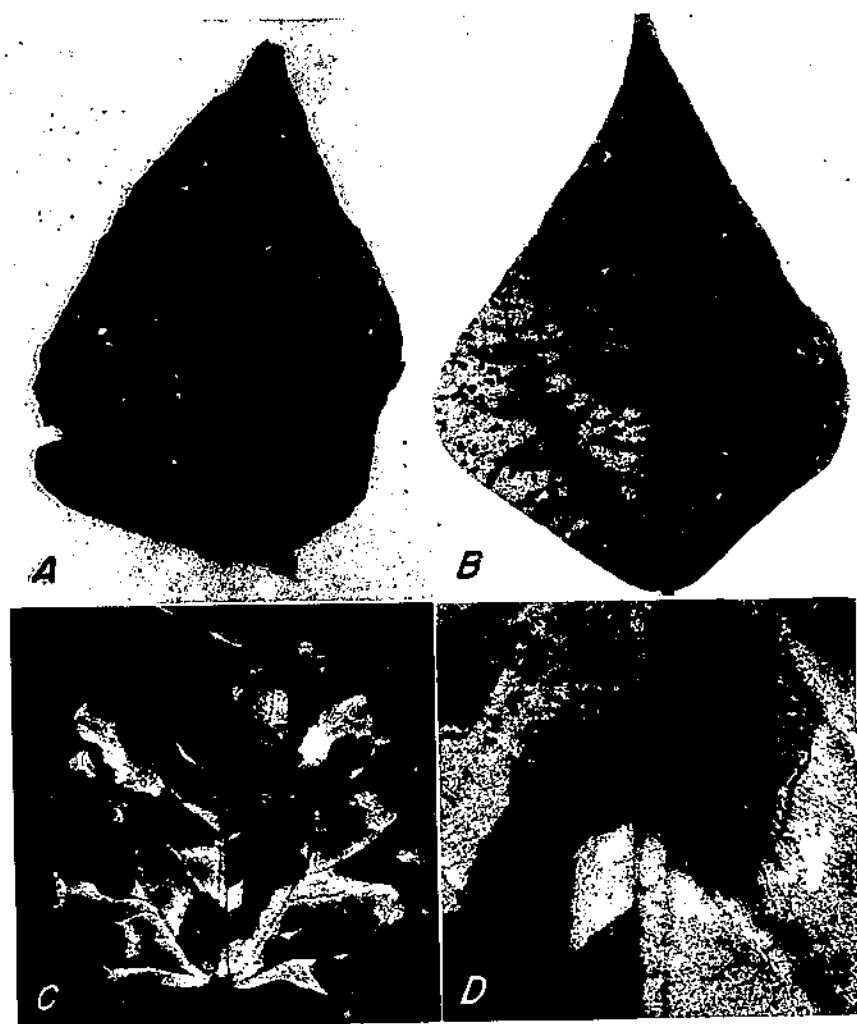


FIGURE 11.—Leaf spots caused by *Phyllosticta phaseolina* (A) and *Isariopsis griseola* (B-D). The centers of the irregular-shaped lesions caused by these fungi eventually fall out, leaving ragged holes. Lesions caused by *I. griseola* are more angular than those caused by *P. phaseolina*. C, Large spreading lesions, showing dark-colored coremia. D, Enlargement of several lesions.

### Causal Organism

#### Nomenclature

*Isariopsis griseola* was described by Saccardo (854) in 1878 on *Phaseolus vulgaris* in Italy. In 1909 Ferraris (843) concluded that the genus *Isariopsis* was identical with the genus *Phaeoisariopsis* and proposed the combination *P. griseola* (Sacc.) Ferr. This generic change has not been generally followed. In 1881 Ellis

(324) described the causal organism as *Graphium laxum* Ell., and in 1886 Saccardo (359) described what appeared to be the same fungus as *I. laxa* (Ell.) Sacc. Material apparently of the type collection of *G. laxum*<sup>9</sup> was distributed by Ellis. On the label of Fungi Columbiani 2434,<sup>10</sup> *G. laxum* is given as a synonym of *I. griseola*. Ellis and Everhart (326) described *Cercospora columnare* from dead leaves of beans collected in New Jersey. The following year they collected material from living leaves, which was issued under the same name as North American Fungi 3394,<sup>11</sup> although they had previously used the name *I. griseola* on the label of North American Fungi 2487,<sup>12</sup> collected from the same locality and presenting essentially the same characteristics. A comparison of authentic Italian material of *I. griseola* with the other exsiccati just listed and with other material of American origin in the National Fungus Collections of the United States Department of Agriculture shows them to be identical. In all specimens examined the synnema is the same. There is a definite similarity of the spores and a very general agreement in their size.

#### Morphology and Physiology

*Isariopsis griseola* is a member of a relatively small genus of the Stilbaceae. The coremium is composed of a smaller number of hyphae that grow erect and more or less parallel into a sheaflike structure. It is dark-colored at the base and gradually becomes lighter in color toward the tip. The coremia (fig. 11, C, D) range in thickness from 20 $\mu$  to 40 $\mu$ . The conidia are borne on the tips of the columnar hyphae. They are one- to three-septate, rarely four-septate, light gray, cylindrical to spindle-shaped, sometimes slightly curved, and not constricted. They range in length from 50 $\mu$  to 60 $\mu$  and in width from 7 $\mu$  to 8 $\mu$ .

#### Control

Control measures have not been perfected for angular leaf spot, possibly because they usually have not been needed. Spraying the vines with a 4-4-50 bordeaux mixture has been recommended and practiced to some extent, but the results have been uncertain.

Brock (141) tested 164 lines of beans for resistance to *Isariopsis* leaf spot. No defoliation or typical sporebearing lesions on the trifoliolate and very few typical lesions on the first leaves occurred on Alabama No. 1, Cafe, California Small White, Case Knife (variety of *Phaseolus coccineus*), Epicure, Mexican Black, McCaslan, Navy, Negro Costa Rica, Scotia, and Rojo Chico. These are classed as highly resistant. All 8 varieties of lima bean that Brock tested were susceptible or very susceptible.

<sup>9</sup> ELLIS, J. B. NORTH AMERICAN FUNGI. [Exsiccati] No. 546. 1881.

<sup>10</sup> BARTHOLOMEW, E. FUNGI COLUMBIANI. [Exsiccati]. No. 2434. 1907.

<sup>11</sup> ELLIS, J. B., and EVERHART, B. M. NORTH AMERICAN FUNGI [Exsiccati]. No. 3394. 1896.

<sup>12</sup> ELLIS, J. B., and EVERHART, B. M. NORTH AMERICAN FUNGI [Exsiccati]. No. 2487. 1890.

### Ascochyta Leaf Spot

Ascochyta leaf spot, caused by *Ascochyta boltshauseri* Sacc., was originally described in 1891 (110) on snap beans in Europe. It was later reported on lima beans also in Europe (486). Kirk (565) reported it on beans in New Zealand in 1906, and Sprague (953) reported it for the first time in the United States in 1935 on beans in Oregon and in 1948 from Washington (954). Jorstad (546) discovered it on beans from Norway and Neergaard from Denmark (721). Bazan de Segura (81) reported it on beans from Peru. Snee (944), in comparative studies of *A. phaseolorum* and *A. boltshauseri*, found that the diseases caused by these two species are indistinguishable macroscopically. He reported that all varieties in Holland are susceptible to both.

Crossan (252), in cross-inoculation studies, showed that *A. abelmoschi* from okra, *A. phaseolorum* from snap bean and lima bean, and *A. gossypii* from cotton are all pathogenic to snap bean, lima bean, cotton, and okra. Comparative studies showed that the pathogens from the several hosts are morphologically very similar and are indistinguishable on the basis of spore size and shape. The studies suggested that the three species of *Ascochyta* are identical. Inoculation experiments by Sprague proved the susceptibility of snap beans, mung bean, adzuki bean, and Scarlet Runner. Although he obtained some slight infection on pea, the amount was too small to justify the conclusion that it is a susceptible host. *A. boltshauseri* has been reported also on lima beans (972).

Sprague (953) gave no description of the symptoms caused by *Ascochyta boltshauseri* further than to state that it produced dark to drab zonate lesions. The following account was given of the morphology of the fungus:

... pycnidia numerous, obscure, light brown to dark brown (on pods) 120 to 150 $\mu$  in diameter; pycnidia on inoculated leaves golden brown, 60 to 130 $\mu$  in diameter. Spores, on pods, predominately one-septate (many immature), 15 percent to 20 percent of the spores were 2- to 5-septate; mostly 2-septate; one septate spores 10-27  $\times$  2.5-6.6 $\mu$  mean spore size 18  $\times$  4.5 $\mu$ ; multiseptate spores 16.6-34  $\times$  4.5-7.1 $\mu$  mean spore size 21.5  $\times$  5.6 $\mu$ ; spores in pure culture on potato-dextrose agar mostly nonseptate, constricted in 2 or more places 6-11  $\times$  2-3 $\mu$ , a few much longer; spores on lesions from artificially inoculated leaves the same size as on pods of naturally inoculated material.

*Stagonopsis phaseoli* Eriks., reported to cause a disease of beans in Europe, was found by Sprague to be identical with *Ascochyta boltshauseri*.

### Black Root Rot

Black root rot, caused by *Thielaviopsis basicola* (Berk.) Ferr., although principally a disease of tobacco, has been reported on beans in New Hampshire, New Jersey, and New York. Burkholder (156) reported it on the roots in association with other organisms, principally with a species of *Fusarium*, while Von Kirchner (563) in Germany and Curzi and Barbaini (262) in

Italy reported it on the roots of various hosts, including beans. The losses caused by this disease are of minor importance.

*Thielaviopsis basicola* causes brown spots on the hypocotyl and to some degree the destruction of the pith. It also attacks the main root and frequently extends to many of the laterals. Williams (1120) showed that in one case the blackened pith contained masses of the winter spores. He germinated bean seed on blotting paper and inoculated the plants with pure cultures of the organism. In 3 days brown streaks were produced half an inch above the point of inoculation similar to those on the original diseased plants.

Black root rot has been observed so seldom and has been responsible for so little injury that no control measures have been worked out, and probably none are needed. The causal organism is usually associated with other fungi, and in view of this fact it is uncertain how much of the injury is caused by *Thielaviopsis basicola* and how much by companion organisms.

### Cercospora Leaf Blotch

#### Geographical Distribution and Economic Importance

*Cercospora* leaf blotch, caused by *Cercospora cruenta* Sacc., has been reported from Alabama, Florida, Georgia, Mississippi, South Carolina, Texas, and Wisconsin. The writers have collected specimens from other States as follows: Maryland, Virginia, North Carolina, New Jersey, and Delaware. In 1932 it was reported from China (998).

*Cercospora* leaf blotch is generally more or less prevalent every year in the United States, but the losses are only slight, having probably never exceeded more than 1 or 2 percent during any 1 year. Welles (1104), on the other hand, found it very destructive to *Phaseolus aureus* in the Philippines. Seventy-five percent of the pods containing partially grown seed were attacked, but young pods remained uninjured. The spots on the stem were not so numerous as those on the leaves and pods, and the direct damage to the stem was of little importance.

#### Symptoms

*Cercospora cruenta* generally attacks mature bean leaves when the crop is fairly well advanced and produces brown, rust-colored patches (fig. 12, A, B) of different sizes and shapes. The spots are mostly angular, somewhat resembling those caused by *Isariopsis* in that the veins act as barriers to its spread, with the consequent production of a checkerboard appearance. Usually a dark-colored border surrounds the spots, which may attain a diameter of a centimeter or more by the coalescence of several primary infections into one. After the death of the tissue, the center of the lesion frequently dries, cracks, and drops out, leaving ragged holes of various shapes and sizes. In the early stages the spots are more or less circular, but they lose some of their original shape as they enlarge. The conidia are usually borne in the centers



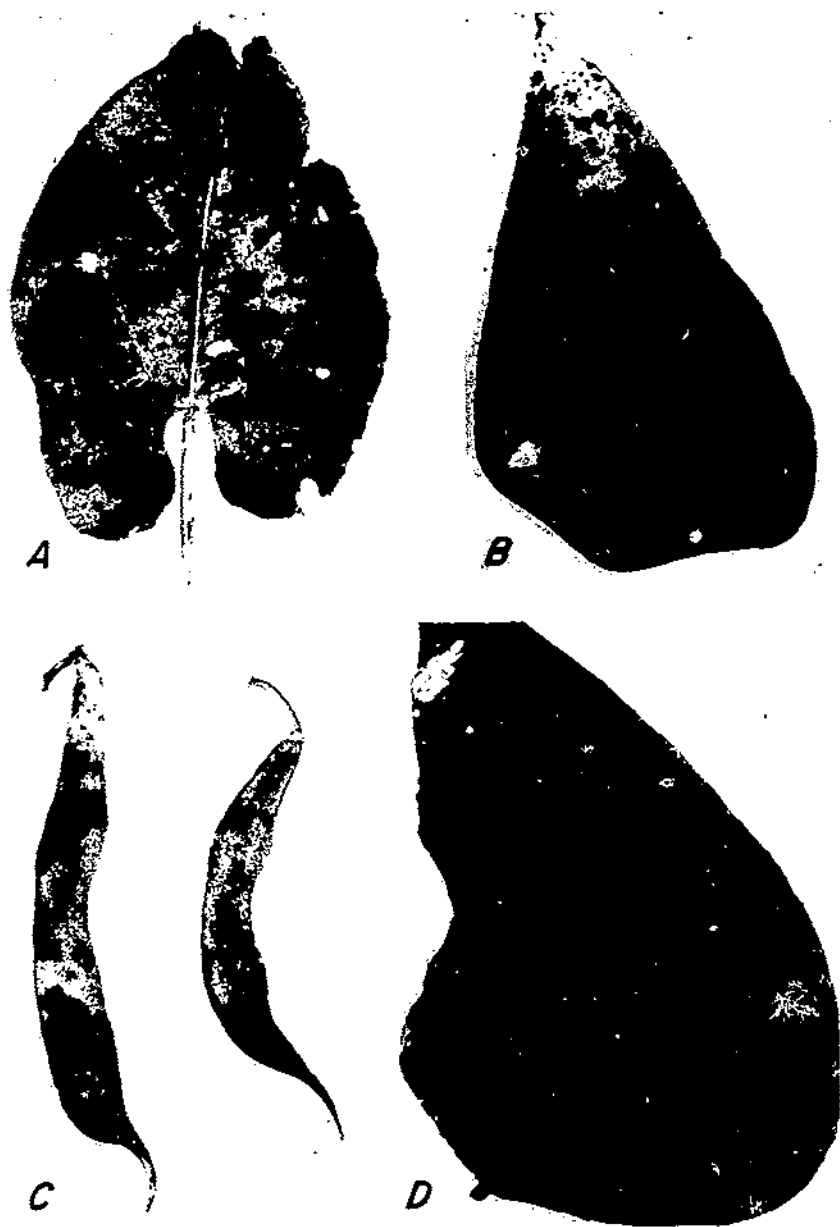


FIGURE 12.—A and B, Leaf blotch, caused by *Cercospora cruenta*, which appears as dark, somewhat circular, dead areas on leaves of snap (A) and lima (B) beans. C and D, Sooty spot, caused by *Heterosporium* sp. The black areas are fasciculate bundles of mycelium and conidia.

of the spots. The combined effect of the attack at various places results in the premature falling of the leaves. The causal organism has been isolated from the seed.

Under field conditions *Cercospora cruenta* often behaves in a surprising manner, especially on the fall crop. The primary leaves are frequently covered with numerous spots, while at the same time none of the trifoliate leaves are attacked. The infections on the primary leaves may serve as a source of infection for the trifoliate leaves after they have matured and have begun to decline as a result of senility. Vigorously growing leaves rarely become infected.

## Causal Organism

### Nomenclature

*Cercospora cruenta* is one of several species of the genus *Cercospora* reported to be parasitic on beans and other legumes in the United States and other parts of the world. Latham described the perfect stage as *Mycosphaerella cruenta*. Whether the various species described are actually different has not been determined. It is becoming well recognized that morphological differences alone are insufficient to establish satisfactory taxonomic relationships. Welles (1104), after working with several species of *Cercospora* from the Philippines, concluded that there may be constant morphological differences of some taxonomic value, but if such exist he was unable to observe them. His conclusions were based on the fact that morphological variations in the size of fruiting structures were influenced by certain environmental factors, such as moisture. If Welles' deductions are correct, it is possible that *C. canescens* (328) is the same as *C. cruenta*. That these two organisms and possibly others are identical was suspected by Butler (178), who reported *C. cruenta* on *Vigna sesquipedalis* (L.) Frutwirth in Italy and on *Phaseolus aureus* and *P. aconitifolius* in India. He further suggested that two other species of *Cercospora*, *C. canescens* on a cultivated *Phaseolus* and *P. lunatus* in the United States and on *P. aureus* in China and *C. vignae* Rac. on cowpeas in Java, are probably the same as *C. cruenta*. The last-named fungus and *C. vignae* have been reported on *V. cylindrica* (L.) Skeels from the United States. *C. lussoniensis* Sacc., described by Saccardo (863) and reported by Reinking (831) to occur on beans in the Philippines, may be included also. *C. cruenta* is likewise reported by Thorold (1019) on *V. catjana* (*V. cylindrica*) and *P. aconitifolius* in Trinidad. Chupp (209) considers *C. phaseolorum*, *C. vignae*, *C. lussoniensis*, and *C. phaseoli* synonymous with *C. cruenta*.

### Morphology

*Cercospora cruenta* was originally described by Saccardo (855, 858). Welles (1104) gave the following measurements of the organism from *Phaseolus aureus*:

... Conidiophores 55 to 91 microns in length, with 3 to 5 septa, conidia 51 to 153 microns in length by 6 to 9 microns in width, with 3 to 7 septa.

He found that the average spore measurements of the same organism varied greatly when obtained from different hosts. Individual spores likewise varied greatly, and it is probable that the range of size is actually very much greater than that given by Saccardo.

### Control

The application of control measures is rarely necessary for cercospora leaf blotch, and only a few recommendations have been made. It is caused by a fungus that yields readily to treatment. Spraying early with a copper fungicide to prevent the organism from becoming established has been recommended.

### Cercospora Leaf Spot

*Cercospora canescens* Ell. & Mart., the cause of a leaf spot of beans and occasionally of lima beans, was originally described in 1882 (328).

Miles (677) reported *Cercospora canescens* from Puerto Rico in 1917, where it caused some, but not serious, loss to the bean crop. It has also been collected from Alabama, Florida, Kansas, Missouri, New Jersey, and Texas. In 1936 it was reported from China (988). The same fungus has been reported on lima beans in Missouri (1139).

The spots caused by this fungus occur on the upper surface of the leaf. They are nearly circular or slightly angular, 2 to 10 mm. in diameter, gray in the center, sometimes surrounded by a slightly reddish border. The spots on lima beans are usually smaller than those on snap and dry beans and contain more reddish color in the border.

The several-septate, caespitose, hyaline-tipped conidiophores of *Cercospora canescens* range from  $50\mu$  to  $100\mu$  long and from  $3\mu$  to  $4.5\mu$  wide. The conidia are hyaline, slender to club-shaped, straight or curved, few- to many-septate,  $80\mu$  to  $220\mu$  long by  $3\mu$  to  $4.5\mu$  wide. An occasional conidium exceeds these dimensions.

### Downy Mildew

Downy mildew, caused by *Phytophthora parasitica* Dast. (*P. terrestris* Sherb.), was reported by Stevenson (970) to be very destructive in parts of Puerto Rico, where all the bean plants in some of the gardens were often ruined. It has also been reported from India (1048). The causal organism was originally described by Sherbakoff (906) as the cause of the buckeye rot of tomatoes. This was the first report of its occurrence on beans in the United States.

No control experiments have been conducted. As the fungus attacks the pods resting on the ground, pole varieties should be planted or bush types that do not fruit too near the soil should be used.

### Fusarium Yellows

Fusarium yellows, caused by *Fusarium oxysporum* Schlecht. f. *phaseoli* Kendrick & Snyder, was originally collected by Harter (440) on dry beans in California in 1928. Several years later the disease was observed in Colorado, Idaho, and Montana. It has not become an important disease.

The causal organism invades and discolors the vascular bundles of the stem and often the petioles and peduncles. Dwarfing results if the plant is attacked when young. The infected plants do not wilt in the characteristic manner of wilts of many other plants attacked by species of *Fusaria*. The initial symptoms are slight yellowing and dropping of the lower leaves, followed by those of the next leaves in order up the stem. As the disease becomes more severe, the leaves become increasingly yellow and finally bright yellow. Diseased plants can be recognized at some distance by the distinctly yellow foliage.

Harter (440) was able to produce typical symptoms of the disease in a small percentage of plants by inoculating them with a *Fusarium* isolated from the vascular bundles. Kendrick and Snyder (553) extended the work of Harter to include inoculations of cowpeas, soybeans, and lima beans. None of these crops was susceptible to infection by the bean organism.

Single-spore cultures recovered from tissue plantings from the vascular bundles of diseased beans were found to agree morphologically with *Fusarium oxysporum* as amended by Snyder and Hansen. The causal organism was described by Kendrick and Snyder (553) as *F. oxysporum* f. *phaseoli*.

Kendrick and Snyder (553) stated that fusarium yellows is seedborne, the fungus probably being transported by the spores adhering to the seed. They showed that the causal organism did not enter the seed but that the spores were deposited on the surface of the seed during harvesting. Dusting the seed with 8 ounces of Semesan or with 4 ounces of Ceresan per 100 pounds of seed completely eliminated the disease in greenhouse tests.

### Gray Mold

Gray mold, caused by *Botrytis cinerea* Fr., is better known as a transit and storage rot of vegetables than as a field disease. It does occur, however, under field conditions on different parts of many plants and especially on beans.

While gray mold has been reported as occurring on beans in various parts of the world, there are very few references of any considerable loss caused by it. Campbell (181) reported losses up to 50 percent in many pole bean fields in Oregon in 1948. Beaumont (85) reported the occurrence of *Botrytis cinerea* in Devon and Cornwall, England, in 1935, and Ishiyama (516) reported it among other parasites of beans in southern Saghalien, Japan. Pape (747) recorded its occurrence in Germany in 1921, where it was found starting as a wilt on bean seedlings and finally killing the plants. Conners (221) reported isolated specimens of

a wilt of beans caused by the same fungus in Canada. Its development and spread are favored by warm, humid conditions. It causes a softening of the plant tissue that is generally easy to recognize by the greenish color which the fungus produces. It is usually restricted to soft, succulent tissue; however, it may sometimes occur on somewhat woody tissue that is moist or wet.

*Botrytis cinerea* is present in most soils. It may attack the pods and the stems of young plants, producing a water-soaked condition, followed by wilt and later death. Pods resting on the ground often become infected and develop a slimy rot, which may result in considerable loss in transit. Infection is favored by wounds. On aboveground plant parts infection usually occurs where the old blossom has fallen on the plant or has been retained at the tip of the pod. Campbell (181) found that gray mold was most destructive where rows were close together, where the stand and growth of foliage were greatest, where the rows ran at right angles to the direction of the prevailing winds and where air movement was restricted. In pole beans, control suggestions included spacing the rows at least 5 feet apart, planting in hills or thinning the plants in drilled rows to approximately 4 inches apart. Limited tests with fungicides failed to give control.

### Phyllosticta Leaf Spot

#### Geographical Distribution and Economic Importance

The leaf spot caused by *Phyllosticta phaseolina* Sacc. (853) is often found on beans grown in the eastern and southern parts of the United States, but usually it is not serious. It has a wide distribution in foreign countries, having been reported from Belgium (650), Brazil (699), Bulgaria (520), Canada (297), Denmark (606), Japan (510), Serbia (809), and Union of South Africa (287).

#### Symptoms

*Phyllosticta phaseolina* is restricted largely to mature leaves, but it has been observed also on other parts of the plant (436). The spots (fig. 11, A), irregularly scattered over the leaf, are 2 to 10 mm. in diameter and have light centers and deep rusty-brown margins. Lesions on the petioles and stems are narrower at first, but later they may extend around the petioles and halfway around the stems. Flower buds usually turn brown and drop off soon after infection. Small lesions, usually less than 1 mm. in diameter with dark centers and reddish margins, often occur on the pods.

As the season advances, the disease becomes more severe and dark-colored lenticular pycnidia scattered over the centers of the spots appear.

#### Causal Organism

The pycnidia are  $70\mu$  to  $90\mu$  in diameter. The pycnospores are hyaline, mostly straight, ovoid to oblong, and  $4\mu$  to  $6\mu$  by  $2\mu$  to  $2.5\mu$ .

Other species of *Phyllosticta* reported on *Phaseolus* may or may not be identical with *P. phaseolina*. *Phoma subcircinata* Ell. & Ev., reported by Halsted (418) on lima beans, seems to be different.

### Hosts

*Phyllosticta phaseolina* occurs on *Phaseolus vulgaris*, *P. lunatis* f. *macrocarpus*, *P. polystachios* (*P. polystachyus*), *Strophostyles helvola*, and *Vigna cylindrica*. On kidney and lima beans and cowpeas, Smith (926) produced characteristic leaf spots, sometimes sterile, with a pure culture of *Phyllosticta phaseolina*.

### Control

*Phyllosticta* leaf spot is not generally regarded of enough economic importance to require the application of control measures. Fungicides such as bordeaux mixture, fixed coppers, and carbamates might be effective.

### Sooty Spot

Sooty spot, caused by a species of *Heterosporium*, develops occasionally on beans in the latter part of the growing season in commercial field plantings in the greenhouse.

The organism is a weak parasite on beans and is able to establish itself only under the most favorable conditions. Infection begins on the upper surfaces of the lower and mature leaves. Sooty spot (fig. 12, D) is characterized by the formation of numerous almost black fascicles of mycelium on the upper surface of the leaf. The fascicles may appear on any part of the leaf, but frequently they are clustered at the base about the petiole or near the margin on only one side of the midrib. The leaf gradually turns yellow as the number of spots increases and finally drops off. The surfaces of yellow, almost dead leaves, are mostly covered with thick mycelial growth and conidia. The fungus appears to grow superficially over the epidermis, and necrotic lesions are not produced. Symptoms on the petioles and lamina are almost identical.

The spot may develop on very young pods. The symptoms on the pods differ somewhat from those on the leaves in that the spots have a more nearly even border (fig. 12, C). With the increase in the age of the pods, the mycelium spreads over additional surface, forming an almost continuous covering of black hyphae.

Peduncles are also attacked. Infection has not been observed on the stem.

### Texas Root Rot

#### Geographical Distribution

Texas root rot, caused by *Phymatotrichum omnivorum* (Shear) Dug. is perhaps best known as a disease of cotton and alfalfa.

It occurs in Arkansas, Arizona, California, Louisiana, Nevada, New Mexico, Oklahoma, and Texas. It has also been reported from Mexico by Ramirez (804). Although it may be restricted to spots in the field, such is not always the case. These infested spots, which enlarge from year to year, can usually be identified from the behavior of the plant growth on them. The disease is worst on black, poorly drained soils.

### Symptoms

Texas root rot appears about the time the plants begin to bloom as a sudden wilting of the plant. The infected surface is darkened, sunken, and softened so that the epidermis may be easily peeled from the roots and crown. Occasionally the diseased parts are covered with minute, wartlike masses consisting of whitish to yellowish fungus threads. Young, slightly infected rootlets, or even those partly destroyed, are often colored a pinkish buff.

### Causal Organism

#### Nomenclature

Texas root rot was attributed by Pammel (745) to *Ozonium auricomum* Lk., although he recognized that the Texas root rot fungus differed essentially from that species. In 1907 Shear (902) described it as a new species, *O. omnivorum* Shear. In 1916 Duggar (301) reported the presence of conidia-bearing hyphae on the characteristic mycelium of the *Ozonium* found on the roots of affected plants. The conidial stage of the fungus studied by Duggar (301) was referred to the genus *Phymatotrichum*, and the new combination *P. omnivorum* was proposed. Duggar was unable to obtain infection by inoculation with conidia. In 1925 Shear (903) found a hymenomycete on the stem of an Osage-orange (*Machura aurantiaca* Nutt.; now called *M. pomifera* (Raf.) Schneid.) in close association with mycelium of the Texas root rot fungus, and on the strength of this association described it as *Hydnum omnivorum* Shear. He was unable, however, to demonstrate the connection between *Ozonium* and the *Hydnum*.

#### Morphology

Duggar described three fairly distinct types of mycelium, i.e., the large-celled type, the strand hyphae, and the acicular type. The large-celled type, measuring as much as  $20\mu$  in diameter with cross walls  $60\mu$  to  $120\mu$  apart, is strikingly similar to *Rhizoctonia* and is found abundantly on the margins of the conidial areas. The acicular type, derived from the arachnoid mycelium, has fairly rigid hyphae, branching in pairs and at right angles. In the early stages of growth, the hyphae are nearly hyaline but become brownish with age. Duggar found that the spores were produced at first on the characteristic, large hyphae and on small branches of those hyphae that make up the strands. The attached conidia were formed in heads about short, swollen, but not necessarily spherical, branches of the strand, or short-celled, hyphae. A sclerotial stage was described by King and Loomis (561).

### Pathogenicity

Pammel (745), Duggar (301), and Shear and Miles (904) studied the disease extensively, but they did not record successful inoculation experiments. Satisfactory proof of parasitism, however, was later obtained by King and Loomis (561) and Taubenhause and Ezekiel (996).

### Control

The Texas root rot fungus attacks a large number of wild and cultivated plants. Beans are listed by Taubenhause, Dana, and Wolff (995) as one of the highly susceptible crops. It was believed at one time that the grasses and cereals were immune to the disease, but Taubenhause and Ezekiel (996) later showed that the monocotyledons as a group were highly resistant and that most of the dicotyledons were susceptible. On the other hand, King and Loomis (561) reported root rot on certain grasses in Arizona. This fact should be taken into account when a crop rotation is being planned for Texas root rot control.

Crop rotation of 3 or 4 years with highly resistant crops, such as those belonging to the grass family, has been recommended by several investigators. The grasses shown by King and Loomis (561) to be susceptible should not be used. During the rotation period all weeds should be destroyed, as many of them are highly susceptible.

Rea (814) claimed that uninfested parts of a field may be protected from infestation by a barrier of closely planted sorghum, a nonsusceptible crop, and also that the use of organic manures, clean fallow, and deep tillage would reduce the loss.

Other investigators who studied methods of control are Duggar (300), Shear and Miles (904), Peltier, King, and Samson (760), King (560), and Eaton and King (313).

### Web-Blight

#### Geographical Distribution and Economic Importance

Web-blight is caused by *Rhizoctonia microsclerotia* Matz, which was described by Matz (659) as the cause of a disease of figs in Florida and later of a destructive wilt of beans and cowpeas in Puerto Rico (667). Weber and others reported it on beans in Florida (1093) and on a number of other hosts in Alabama, Louisiana (58), and Texas (1094). It also occurs in Brazil (276), Burma, Ceylon, Japan, and the Philippines (1094). That web-blight possesses great destructive potentialities was shown by the investigation of Weber (1094), who reported serious losses to snap and lima beans in Florida over a period of several years.

### Symptoms

The web-blight fungus produces small, circular, water-soaked spots on the leaf and spider-weblike mycelial growth on the stems, pods, and foliage, in which many small, brown sclerotia are embedded. Invaded cells die by the time the spots attain 1 mm. in



diameter. The spots are much lighter in color than the adjacent healthy tissue and appear as if they had been scalded. The diseased areas, varying from 1 to 3 cm. in diameter, later become tan brown and frequently zonate and are surrounded by dark borders. As the infected area enlarges, light-tan hyphae develop on both surfaces of the leaf and spread rapidly over the noninfected areas. With favorable weather conditions the mycelium spreads to all parts of the plant, binding the leaves, petioles, flowers, and pods together with a web or mat of hyphal strands.

The pods are attacked in all stages of their development. On young pods the early infections are light tan and irregular in shape. On mature pods the spots are dark brown, more or less circular, slightly zonate, and definitely sunken. When the spots are numerous they involve the entire pod; when few and scattered they enlarge up to 1 cm. in diameter, or as much as the width of the pod. The ovules may also be attacked.

### Causal Organism

The causal organism was first described as *Rhizoctonia microsclerotia* Matz. Later the basidial stage was found on beans and figs by Weber (1094). Basidiospores from both hosts were germinated on potato-dextrose agar, and the mycelia and sclerotia from the two sources were indistinguishable. Cultures from both sources differed from those obtained from basidiospores of *Corticium solani* and *C. stevensii* Burt (1094). The results of different investigators with the organism lead to the conclusion that it should now be known by the name *C. microsclerotia* (Matz) Weber. More recently Rogers considered it as a synonym of *Pellicularia filamentosa* (Pat.) Rogers, and in 1953 the name *P. filamentosa* f. sp. *microsclerotia* (Matz) Exner was proposed.

Matz (659) described *Rhizoctonia microsclerotia* as follows:

Sclerotia superficial, small 0.2 to 0.5 mm. in diameter, white when young, brown to dark brown at maturity, nearly homogenous in structure and color, sub-globose, free from tufted mycelium, not smooth usually single, sometimes conglomerated.

Vegetative hyphae 6 to 8 $\mu$  wide, first hyaline and granular, brown and more or less empty with maturity, septate.

The basidiospores are oval, hyaline, thin-walled, 9 $\mu$  to 11 $\mu$  long and 5 $\mu$  to 6 $\mu$  wide.

### Hosts

The causal fungus parasitizes the foliage and fruit of a number of wild and cultivated annuals and perennials. Inoculation experiments have demonstrated that the fungus is parasitic on such vegetables as tomatoes, beets, eggplants, cucumbers, cantaloupes, watermelons, and carrots. The wide range of hosts would suggest that extensive losses may be possible in those sections of the country where the crops are grown during the season of the year when high temperature and high humidity occur.

### Control

In the Tropics beans should be planted so that they may complete their growth before the beginning of the rainy season. The

planting should be well-aerated situations in rows and not broadcast. Rotation should be practiced with nonsusceptible crops, such as tobacco, corn, and grasses.

## Bacterial Diseases of Major Importance

### Common Blight

#### Geographical Distribution and Economic Importance

Common blight, caused by *Xanthomonas phaseoli* (E. F. Sm.) Dows., is often prevalent in all the States east of the Rocky Mountains. While it may occur in California, Idaho, Nevada, Washington, and Oregon, common blight rarely causes enough loss to be of any economic importance.

Common blight has been reported from Australia (644), Bulgaria (57, 572), Canada (983), China (832), France (275), Hungary (558), Japan (511), Madagascar (119), Norway (545), the Philippines (834), Russia (373), Spain (25), Turkey (128), Union of South Africa (286), Uruguay (95), and Yugoslavia (999).

It is not always possible to separate the losses caused by the common and halo blights. They frequently occur together in the same field and probably on the same plant.

In 1928 Hedges (467) reported heavy losses from common and halo blights in several of the Western and Southern States. In some fields the crop was almost a complete loss. No accurate estimate of the total losses caused by these two diseases is possible, but such losses amount to an enormous sum (865).

The severity of the blights varies more or less from year to year, depending somewhat on weather conditions. In 1918, 75 percent of the fields in New York were affected and serious losses occurred. In 1919 losses ranged from 40 to 60 percent of the crop in Colorado. The losses to snap beans in 1936 in the United States were estimated to range from a trace in several States to 20 percent in Colorado and 50 percent in Oklahoma. Calculated on the basis of weight, the losses were more than 3,400,000 pounds of snap beans and about 34,700,000 pounds of dry beans. These estimates are not so high as they were in some previous years. Losses of about 22 to 28 percent of the seed yields of snap beans and 25 to 30 percent of the field beans occurred in Weld County, Colo., in 1937 (1204). Andersen (38) estimated that bacterial blights caused a \$3,500,000 loss to growers in three Michigan counties in 1951. In 1953 the disease was widespread in western Nebraska, and the loss caused was estimated at a little more than \$1,000,000.

### Symptoms

See Halo Blight (p. 74) and figures 13, 14, 15, and 16.

## Causal Organism

### Nomenclature

Common blight was recognized for the first time in the United States by Beach (82) in 1892. About the same time Halsted (417) described a bacterial disease of the pods and seed of beans and obtained positive results from inoculations. In 1897 Smith (928, 929) described and named the organism *Bacillus phaseoli* E. F. Sm. In 1901 Smith (930) described the cultural characteristics of the organism and transferred it to the genus *Pseudomonas*. Up to that time Migula's (674) classification had been followed by most bacteriologists. In 1905 Smith (931) transferred the name of the blight organism to the genus *Bacterium*, so that it became *Bact. phaseoli*. Later another system of classification was proposed (90); if this were followed, the blight organism would be known as *Phytomonas phaseoli* (E. F. Sm.) Bergey et al. Later the name was changed to *Xanthomonas phaseoli* (294).

### Morphology and Physiology

For a complete description of *Xanthomonas phaseoli* consult Elliott (321, pp 128-131).

Hedges (463) compared culturally *Xanthomonas phaseoli* var. *sojense* (Hedges) Starr and Burk. with *X. phaseoli*. Many disease-producing bacteria are very similar culturally to *X. phaseoli*, and their separation is often possible only by their associations with definite diseases of certain hosts, the difference being biological. Such is the case with *X. campestris* (Pam.) Dows., the cause of black rot of cabbage, and *X. phaseoli*. Because of the similarity of some of the well-known pathogens, Link and Sharp (607) attempted to differentiate serologically *X. campestris*, *Corynebacterium flaccumfaciens*, *X. phaseoli*, and *X. phaseoli* var. *sojense*, and found that these four pathogens could be distinguished by agglutination tests. Serologically, *X. campestris* is closely related to *X. phaseoli* and *X. phaseoli* var. *sojense*, but not so closely related to *C. flaccumfaciens*. Sharp (900) reported morphological, physiological, serological, virulence, and acid-agglutination studies on the organisms just mentioned, with the exception of *X. campestris*, and concluded that the three species differed and could be differentiated by the use of the agglutination test.

Elrod and Brown (330) found that the *Xanthomonas phaseoli* group had common group components. Absorption of any of the individual antisera by a heterologous organism of the group left specific factors and removed all group components. Starr (966) studied the minimal nutritive requirements of many species and varieties of the genus *Xanthomonas*, excluding *X. phaseoli* and *X. phaseoli* var. *fuscans*.

Hedges (472) studied the changes in *Xanthomonas phaseoli* which occurred during 2 years' association with common bean mosaic virus in vivo. A decrease in toxicity during this period is considered to be the result of the increased development of the weekly pathogenic R to S white and nonpathogenic S pink vari-

ants, which resulted in the partial subordination of the virulent S yellow form of the bacterium.

Sherf (908) has shown that slant cultures of *Xanthomonas phaseoli* covered with a layer of sterile mineral oil have retained their viability and pathogenicity for at least 13 to 18 months.

Feder and Ark (339) have obtained a toxic complex and polysaccharide component from *Xanthomonas phaseoli* that induces a wilt in Russian Mammoth sunflower and Bonny Best tomato cuttings. Leach and others (595) reported that the exudate was a polysaccharide-like substance with a molecular weight above 19,000,000, as determined by light-scattering measurements. Maximum live-cell content was reached on the fourth or fifth day on glucose agar, the stationary phase lasting until the seventh day, after which the count decreased rapidly. Approximately 50 percent of the cells survived in drops of exudate collected before the seventh day and dried on coverglasses over Drierite. Twelve percent were still viable after storage for 440 days at 5° C. and 20 percent relative humidity.

Hedges and Fisher (474) studied the effect of supplying various levels of nitrogen to bean plants infected with a mixture of common bean mosaic and *Xanthomonas phaseoli* and found that the most virulent development occurred when most optimum growth of the host occurred.

#### Pathogenicity

The pathogenicity of *Xanthomonas phaseoli* was demonstrated in 1897 (929). Since then the organism has been extensively studied by many investigators in the United States and in many foreign countries. Infection takes place through the stomata, and successful inoculations can be obtained by rubbing the leaves with a suspension of the causal organism or can be obtained less easily by spraying the leaves with a fresh culture and confining the plants for about 24 hours in an infection chamber. When the plants are inoculated in this way, the bacteria frequently enter and follow the vascular bundles for some distance from the point of entrance.

Some investigators prefer to confine the plants some hours in a moist chamber at a high humidity before inoculation. The relation of humidity to infection has been investigated by Zau-meyer (1170) in a series of experiments in which he confined plants in a moist chamber for 24 hours before and 24 hours after inoculation. His results indicated that infection was obtained without premoist treatment when plants were held in an infection chamber for 24 hours after inoculation.

Several methods of inoculation have been used to evaluate the resistance of varieties and hybrid seedlings. Carborundum dusted on the leaves before rubbing with a pad soaked with a suspension of the organism increased infection (109). This method has been used by the authors very successfully. A multiple-needle inoculator consisting of 90 No. 0 insect mounting pins with their head ends embedded in a cylindrical block of sealing wax has been used to puncture primary leaves that have been placed on a rubber or

cellulose sponge saturated with bacterial suspension (45). The reaction area is measured quantitatively after 12 to 14 days. Bridgmon (132) obtained the highest percentage of infection when the plants were atomized with a bacterial suspension, both in the seedling and mature stages, under high relative humidity. When time is not a limiting factor and good germination is not required, he recommended needling or soaking shoots in a bacterial suspension.

#### Dissemination

The methods of dissemination of the causal organism are important because of their relation to possible control. The manner of distribution can be divided into two categories: (1) Over long distances, as from one part of the country to another; and (2) from plant to plant or from field to field.

The disease is seedborne and is capable of being transported over long distances. Since it is the usual practice for canners and seed dealers to purchase seed from seed firms for distribution to farmers, the shipments may be from the west to the east coast. In fact, seed grown in Colorado, Idaho, New York, and in several other States is shipped throughout the Middle West, East, and South for planting. If this seed is produced where the bacterial blights occur, it is likely to be infected, and as a result dissemination by that means to all parts of the country actually occurs.

Infected seed becomes the source of considerable local dissemination. Plants grown from infected seed frequently bear lesions on the cotyledons, cotyledonary nodes, or the primary leaves. These lesions gradually enlarge, and, if the weather conditions are sufficiently humid, slimy masses of bacterial exudate accumulate on the surface. From the slimy mass the bacteria may spread to other plants by several different agencies. Rain accompanied by wind washes the bacteria either to other parts of the plant or to other plants where, if conditions are ideal, new infections start. These plants become centers of infection for nearby ones.

Menzies (665) used artificially established infection centers in beanfields irrigated by sprinkler for various time intervals, and found that practically no spread of blight infection was obtained during July and August with up to 30 hours of continuous irrigation. In September, as the crop approached maturity, disease sometimes developed rapidly but because of late occurrence the damage was negligible. He concluded that beans grown in the Columbia Basin of Washington can be irrigated by overhead sprinkling without serious danger from bacterial blight, provided they are planted early enough to mature before October.

Insects have been reported to spread bacterial blight from plant to plant. Sackett (864) stated that "insects play an important part in disseminating the trouble." It is possible that many insects that visit bean plants carry the bacteria on their legs or other parts of their bodies and distribute them to other plants. Grasshoppers (*Melanoplus* spp.) and the Mexican bean beetle (*Epilachna varivestis* Muls.) have been shown to be agencies in dissemination.

Fields of beans with only a small percentage of infected plants have become severely damaged by blight within a few days after a severe windstorm and hail. The wounds made by the hail serve as infection courts for the bacteria.

How the blight organisms overwinter, especially in the Northern States, where the winters are long and the temperatures low, has been variously argued. There are several plants other than beans that are susceptible to *Xanthomonas phaseoli*, and in the South, where a succession of crops is grown throughout the year, the perpetuation of the organism by a series of different hosts is not improbable. In fact, beans may be grown most of the year in some of the Southern States. Harrison and Barlow (435) stated that the bacteria can live over at least one winter in the stems and leaves, if these are allowed to remain on the ground. McCready (620) stated:

The disease is carried over from year to year in the seed from a diseased crop, in the soil on which a diseased crop has grown, in straw from infected fields, and in bedding or manure.

Muncie (703) isolated the blight organism from diseased bean stubble that had remained in the field over winter.

Zaumeyer (1170) related observations that lend support to the possible overwintering in the field of the blight organism. He observed that beans planted in a field where the disease was prevalent one year became severely infected the following year, while seed from the same source gave a clean crop when grown on noninfested nearby fields. Circumstantial evidence indicates that the organism can overwinter in the soil.

Hedges (473) placed lima bean leaves infected with *Xanthomonas phaseoli* into pots in the fall and kept them buried during the winter. The next spring she was unable to obtain any infection on snap beans planted in these pots.

Soaking bean seed in a solution containing the nodule organism has been practiced to improve the growth of the plant. This practice is subject to some risk, especially if the seed should be infected with blight. The blight bacteria escape from the seed into the water and contaminate all the seed in the lot and great losses have resulted (1170). This practice is condemned by Barss (77), who pointed out that the soaking of the seed in a liquid culture of *Bacillus radicola* Beij. (*Rhizobium phaseoli* Dang.) for nodule inoculation results in a general contamination of the entire seed lot, even if only a few seed are infected by blight bacteria.

### Pathological Histology

Under natural conditions *Xanthomonas phaseoli* enters the leaves through stomata or wounds. It then invades the intercellular spaces (561), causing a gradual dissolution of the middle lamella. Later the cells begin to disintegrate with the formation of bacterial pockets. The bacteria may enter the stem in three ways: (1) Through the stomata of the hypocotyl and epicotyl; (2) through the vascular elements leading from the leaf, and (3) from infected cotyledons. Bacteria together with the matrix in the xylem vessels may cause a wilting of the plant, either by plugging

the vessels or by the disintegration of the cell walls. Because of the composition of the cell wall, little infection is found in the secondary xylem.

The pathogen is harbored below the seed coat. It enters the sutures of the pods from the vascular system of the pedicel and then passes into the funiculus and through the raphe leading into the seedcoat. The micropyle also serves as a point of entry into the seed. Direct penetration through the seed coat has not been observed.

The bacteria either remain in the seed coat or pass into the region of the cotyledons and enter these structures when the seed germinates. With the increase in the size of the cells after germination, rifts in the epidermis of the cotyledon are formed and the bacteria pass through these openings into the intercellular spaces of the cells below and may invade the entire cotyledon. The vascular elements may be invaded; whence, infection of the young plant takes place (932). Microchemical tests have shown that after germination a great share of the cotyledonary tissue becomes soluble, and bacterial action is probably influenced to a great extent by the solubility of this material.

Allington and Chamberlain (31) studied the multiplication of *Xanthomonas phaseoli* in a susceptible plant, bean, and in a resistant plant, soybean. Multiplication in the intercellular spaces was about equal for the first day in both plants. After 5 or more days the inhibitory effect of soybean to *X. phaseoli* was manifested by the less rapid increase in the bacterial population.

Sanford (869) recovered a mixed bacterial flora from thin sections cut aseptically from the steles of apparently healthy beans. Thomas and Graham (1018) recovered six different species of bacteria from apparently healthy bean plants, among them were: *Xanthomonas phaseoli*, *X. phaseoli* var. *fuscans*, and *Corynebacterium flaccumfaciens*. Schnathorst (879) isolated bacteria from certified bean seeds produced in Idaho and from plants grown from such seed. The genus *Bacillus* was most prevalent. None of the bacteria were pathogenic.

The pathological histology caused by *Pseudomonas medicaginis* var. *phaseolicola* is almost identical with that caused by *Xanthomonas phaseoli*. The histology of both is very different from that caused by *Corynebacterium flaccumfaciens*.

### Hosts

*Xanthomonas phaseoli* has been reported to be parasitic on *Phaseolus mungo*, *P. aureus*, *P. coccineus*, *Dolichos lablab* L., *Strophostyles helvola* (377), *Glycine max* (L.) Merrill, *P. lunatus*, *P. acutifolius* var. *latifolius*, *P. aconitifolius*, *P. angularis*, *Vigna sinensis*, *Stizolobium deeringianum* Bort., and *Lupinus polyphyllus* Lindl., in addition to the garden and field beans. Sherwin and Lefebvre (909) found none of the cowpea varieties they tested were susceptible to *X. phaseoli*.

### Control

Spraying and dusting have not been generally effective in controlling common blight of beans, although Christow (208) obtained good control by spraying with bordeaux mixture. Edgerton and Moreland (317) did not obtain satisfactory results with this spray. Burke and Starr (154) found that bordeaux mixture (12.75 percent) dust and spray, puratized spray (5 percent, 1 in 800), and cuprox dust (copper oxychloride) were the best treatments on hail-damaged plants, while bordeaux spray, bordeaux dust, and puratized spray were the most effective on undamaged plants. None of the treatments gave complete control.

The use of clean, or disease-free, seed is the most important remedy and will usually give a commercially profitable crop so far as bacterial blight is concerned. Clean seed cannot always be obtained, but a considerable quantity of it is being grown in those parts of the intermountain region of the West and of the Pacific Coast States where bacterial blights are not a factor (626). Some of the seed grown in these regions is practically free of seedborne blight and should be demanded by canners and seed dealers for distribution to the farmer. Crop rotation should be practiced along with the use of disease-free seed. The damage from the disease is often correlated with the date of planting. Damage is not usually very severe until during the hottest part of the season, and if the crop can be planted early enough it will mature the hottest weather and possibly escape severe injury.

In New South Wales (1128) seed raised for certification is grown in high rainfall areas, where it is easier to detect trace infections of blight than in arid regions. There are no areas with a sufficiently low rainfall to permit the consistent production of blight-free seed of susceptible varieties unless clean seed is used for a seed crop. No tolerance for blight is allowed in fields under certification.

Katznelson (549) and Katznelson and coworkers (550) have developed a technique to detect rapidly internal seed infection by *Pseudomonas phaseolicola* and *Xanthomonas phaseoli*. The method is based upon the increase in number of phage particles when the bacteria are present in a sample.

Several investigators have tested numerous varieties with the hope of finding some that could be recommended for planting and that could be used as parents for the breeding of disease resistance (14, 132, 152, 164, 208, 332, 366, 451; 718, 808, 983, 1080, 1170).

Variety trials for disease resistance show that no varieties are immune. Some may be more tolerant than others, but under conditions very favorable for blight development all of the current snap bean and field beans grown in the United States are severely damaged. Fullgreen (46), released in 1950, is reported to be resistant to common blight. It possesses a greater degree of resistance to halo blight than common blight.

The bacterial blight organism lives for several years in the seed, and some investigators have advised not to plant infected seed



until the bacteria die. This practice would not be practicable, however, unless the seed would outlive the bacteria. Under certain conditions the seed loses its vitality in a few years. Christow (208) found 7-year-old seed practically free of live bacteria and germinating satisfactorily. He advised, however, not to use seed more than 8 years old. Rapp (811) found seed 2 to 3 years old free of blight. More recently, however, the blight organism has been isolated from seed that was 10 years old.

Seed treatments for common blight control have been recommended by several investigators, but in general they have not been used. Edgerton and Moreland (317) treated seed for 18 to 20 minutes in a solution of either 1 to 50 benetol or 1 to 1,000 corrosive sublimate.

Person and Edgerton (763) reported promising results in the treatment of blighted seed by immersing it 12 to 14 minutes in a solution of 1 to 500 mercuric chloride in 70 percent ethyl alcohol plus 2 percent acetic acid. Effective control of initial infection of the primary leaves was obtained in all tests.

Kreitlow (576) employed several different solutions as disinfectants and reduced the amount of blight from 23.6 percent in the untreated to 0.2 percent in the treated lots. The yields were 2 to 7 times those of the controls. Effective control was obtained by use of the following solutions: (1) 1 to 500 mercuric chloride in diethyl ether; (2) 1 to 20,000 brilliant green in 50 percent ethyl alcohol plus 3 percent acetic acid; (3) 1 to 500 mercuric chloride in 70 percent ethyl alcohol plus 3 percent acetic acid; (4) 1 to 20,000 gentian violet in 50 percent ethyl alcohol plus 3 percent acetic acid.

Burke (154) reported that seed treatment with dry heat at 80° C. for 35 minutes followed by New Improved Ceresan for 24 hours proved effective in reducing the number of diseased plants from infected seed.

The antibiotics Aureomycin (182, 1101), streptomycin (220), Circulin (220), and Polymyxin (549) have been effective against *Xanthomonas phaseoli* in vitro. The number of diseased plants grown from infected seed was reduced when the seed was soaked in a 1 to 100 aqueous solution of streptomycin prior to planting (153). Dihydrostreptomycin sulfate and streptomycin sulfate were absorbed by the stems of bean seedlings and translocated upward to the primary leaves. Within 3 to 4 days they accumulated in sufficient amounts to inhibit or prevent the growth and development of halo and common blight organisms (684, 685, 686).

Marlatt (653) reported that streptomycin used as a spray or dust and in seed treatments failed to control common blight under field conditions in New Mexico.

Gray (397, 398) showed that the addition of glycerin to streptomycin sprays caused a marked increase in the effectiveness of the antibiotic against the common bacterial blight of beans in greenhouse tests.

Chemotherapeutants have been applied to beans, but none has yet proved suitable for practical control (234).

## Fuscou Blight

Fuscou blight, caused by *Xanthomonas phaseoli* var. *fuscans* (Burk.) Starr and Burk., was isolated by Burkholder (166) from beans grown in Switzerland.

### Geographical Distribution and Economic Importance

*Xanthomonas phaseoli* var. *fuscans* has been reported from Colorado (1002), Michigan (33), Montana (1002), New York (168), Wisconsin (168), and Wyoming (1002). The disease has also been found in Australia (8), Russia (51, 332, 373), South America (168), Switzerland (166), and Yugoslavia (1000).

Because of the similarity of symptoms between fuscous and common blight, the damage caused by fuscous blight is not known. The writers have isolated the causal organism from infected plants growing in Colorado, Montana, Nebraska, and Wyoming. Andersen (32) made isolations from infected seed delivered to the elevators and found that 75 percent of the seed was infected with *Xanthomonas phaseoli* var. *fuscans*, 9 percent with *X. phaseoli*, 3 percent with *C. flaccumfaciens*, and 13 percent with weakly pathogenic unidentified organisms.

### Symptoms

The symptoms of *Xanthomonas phaseoli* var. *fuscans* produced on seedlings and older plants are not significantly different from those caused by *X. phaseoli*, except that in some cases a slight hypertrophy develops in the tissue about wounds on the stem. It is the writers' experience that in greenhouse inoculations the symptoms are more severe on plants infected with fuscous blight than on plants infected with common blight. On young seedlings, considerable darkening of the stem occurs about the point of artificial inoculation.

### Causal Organism

#### Morphology and Physiology

The reader is referred to Elliott (321, pp. 131-132) for a complete description of *Xanthomonas phaseoli* var. *fuscans*.

#### Dissemination

Same as common and halo blights (pp. 68 and 82).

#### Hosts

Burkholder (166) obtained infection on *Phaseolus vulgaris*, *P. lunatus*, and *P. coccineus*.

### Control

Control of fuscous blight is similar to that recommended for common blight (p. 71).

Burkholder and Bullard (171) tested 40 bean varieties for their reaction to fuscous blight infection and found Great North-

ern U. I. No. 1 and Norida to be the most tolerant. The Scarlet Runner bean (*P. coccineus*) was more resistant. The reaction of additional varieties has been listed by Cornell workers.

### Halo Blight

Halo blight, caused by *Pseudomonas phaseolicola* (Burk.) Dows., was first described by Burkholder (165) in 1926.

### Geographical Distribution and Economic Importance

In the United States halo blight occurs in the same general areas where common blight is found.

Halo blight has been reported from Australia (15, 18, 345), Belgium (651), Brazil (697), Canada (223, 311), Denmark (395, 1091), England (1116, 1117), France (581, 582), French Morocco (1), Germany (107, 126, 569, 962, 963), Kenya (717), Mauritius (739), the Netherlands (1119), New Zealand (824), Poland (1169), Russia (373), South Africa (1063), Spain (372), Switzerland (166), Tasmania (293), and Uruguay (95).

Halo blight is less important than common blight in the United States, mainly because most of the field bean varieties in the United States are resistant. Halo blight is favored by cool temperatures and common blight by warm temperatures. This accounts for the more general occurrence of common blight in the South on snap beans.

### Symptoms

The symptoms of the halo and common blight diseases are very much alike. It is seldom possible from a superficial examination to be certain which disease is present. In many cases, isolation of the organism provides the only means. In foreign countries halo blight has often been referred to as the grease spot, a name that is quite applicable to the spots on the pods (fig. 16). The halo blight spots are water-soaked, possibly more so than those caused by the common blight (fig. 15, D), but water soaking is not a dependable characteristic, since the degree is somewhat dependent on weather conditions. Both organisms cause stem girdle (fig. 15, C). Both are said to cause wilt, but in the writers' experience, wilting is not common in the United States. Both diseases affect the seeds, pods, leaves, and stem in a similar manner.

The first symptoms produced by *Pseudomonas medicaginis* var. *phaseolicola* on seedlings are identical with those produced by *Xanthomonas phaseoli*, being small, water-soaked spots on the underside of the leaf or leaflets that enlarge and coalesce with similar infected areas (fig. 14, B). Later, *P. medicaginis* var. *phaseolicola* produces a halolike zone of greenish-yellow tissue (fig. 14, A) outside the water-soaked area that aids in distinguishing halo blight symptoms from the symptoms produced by *X. phaseoli*. The latter organism causes the infected region to appear flaccid, giving it a sunscalded appearance. This area, encircled by a comparatively narrow zone of lemon-yellow tissue

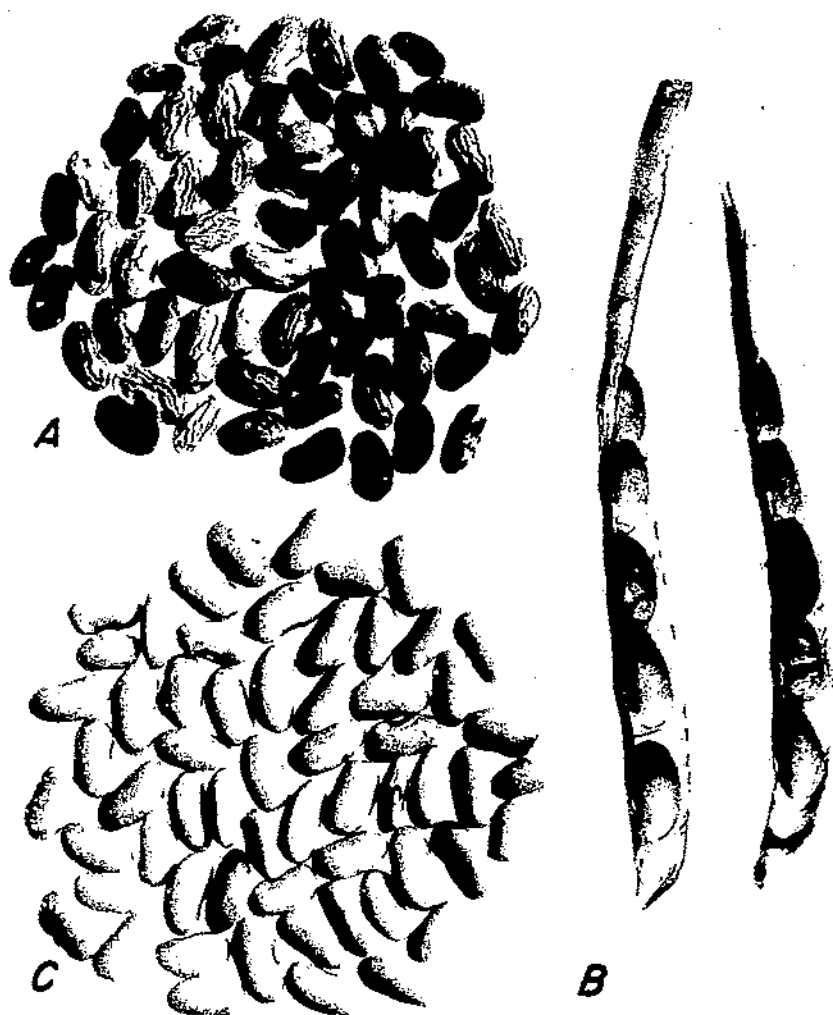


FIGURE 13.—Common blight, caused by *Xanthomonas phaseoli*: A, Infected seed; B, pod containing both healthy and diseased seed; C, disease-free seed.

(fig. 14, C), turns brown and rapidly becomes necrotic and may be so extensive as to cause defoliation.

A single water-soaked spot caused by *Pseudomonas medicaginis* var. *phascolicola* may produce a halolike zone 2.5 cm. in diameter. A chlorotic area apparently caused by toxins, which are produced by and work in advance of the organism, surrounds the infection court. Systemic infection of the plant may frequently produce considerable chlorosis and leaf malformation without the appearance of much external infection (fig. 14, D).

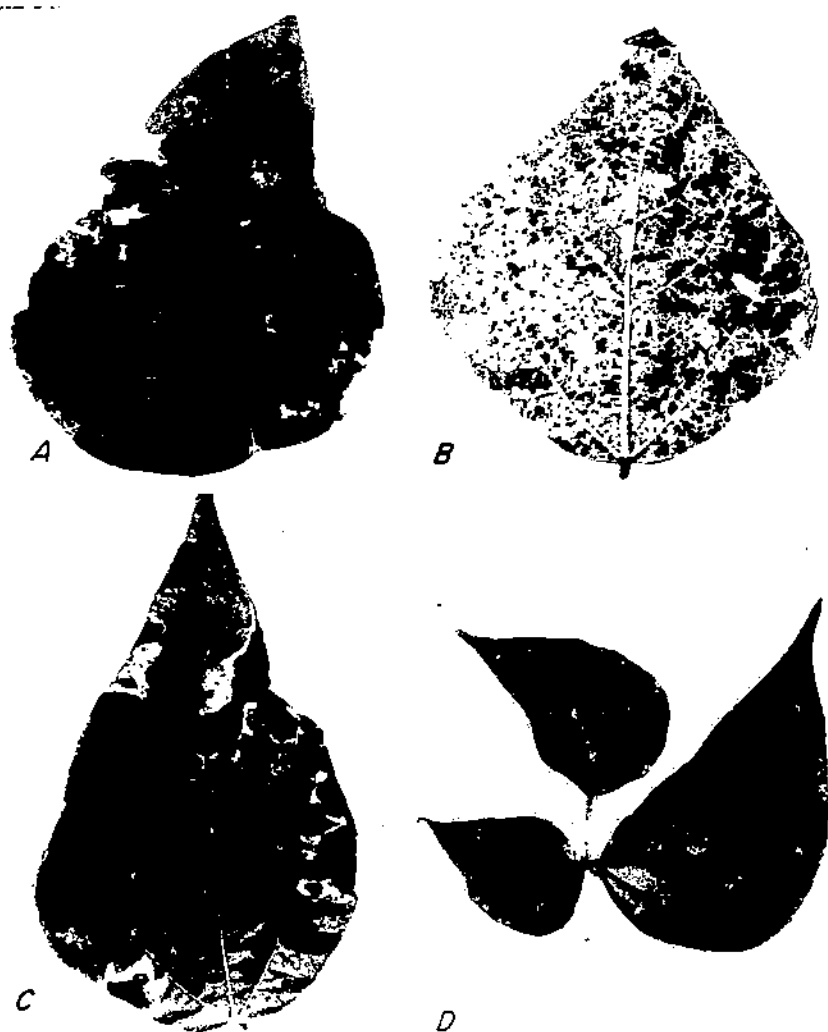


FIGURE 14.—A, Halo blight infection, caused by *Pseudomonas phaseolicola*, showing yellow-green areas surrounding necrotic lesions; B, early stage of infection of common blight caused by *Xanthomonas phaseoli*, showing primary water-soaked lesions; C, a late stage of the disease showing the lesions bordered by bands of lemon-yellow tissue; D, halo blight on trifoliate leaflets with systemic infection, causing malformation of leaflets.

In the early stages of development, plants in a beanfield infected with the halo blight organism often can be distinguished from those infected by *Xanthomonas phaseoli* by the characteristic yellow color of the leaves. *X. phaseoli*, on the other hand, causes the leaves to turn brown quite readily, giving the appearance of a burning effect. The symptoms produced by *Pseudomonas medicaginis* var. *phaseolicola* on the stems, pods (fig. 15, A),



FIGURE 15.—A, B, Halo blight, caused by *Pseudomonas phaseolicola*: A, Infection along suture; B, infected seeds from pod. C, D, Common blight caused by *Xanthomonas phaseoli*: C, Stem girdle; D, seriously infected pods showing exudate.

and seed (fig. 15, B) are indistinguishable from those produced by the common blight organism (fig. 13, A), except when bacterial exudate is noted in the lesions. The color of the exudate of *X. phaseoli* (fig. 15, D) is yellow, whereas that of the halo blight organism is light cream or silver-colored. Infection of the sutures of the pod is common (fig. 15, A).



FIGURE 16.—Pod infection caused by *Pseudomonas phaseolicola* in the field; the exudate is light-cream or silver-colored.

If the bacteria enter the vascular tissue of the leaf, different symptoms develop. The initial infection usually begins in the small veinlets, from which the bacteria pass to the large veins and finally to the midrib. Severe infections produce reddish discolorations with water soaking of the tissues adjacent to the

veins. If the leaf infection starts from the petiole, the main vein and its branches appear water-soaked at first and later take on a brick-red color.

The lesions on the stem of young seedlings are sometimes sunken and begin as water-soaked spots that gradually enlarge. Later they appear as reddish streaks extending longitudinally along the stem. The surface of the stem often splits, and a bacterial exudate may accumulate in the lesion.

In the earliest stages of pod formation a characteristic lesion known as stem girdle, or joint rot (fig. 15, C), frequently develops (705). It is very common in plants that originate from infected seed and is produced by the organism developing internally in the stem. The infection starts at the node above the cotyledonary attachment as a small water-soaked area and encircles the stem as it enlarges. Later, the diseased region becomes amber-colored. The girdling is usually completed when the pods are half mature, and the affected stem is further weakened by the increasing weight of the top so that the plant often breaks at the node.

The infections on the pod may occur at any place as small, water-soaked spots that gradually enlarge. There may be distinct zoning and a narrow, reddish-brown or brick-red band of tissue surrounding the spot. Infections may occur in the vascular elements of the dorsal and ventral sutures, causing a water soaking of the adjoining tissue. The infection often follows the vascular system of the sutures, finally entering the seed by way of the funiculus.

Plants from infected seed often exhibit a characteristic wilting, which consists of a slight drooping of the leaves at the pulvinus. During the daytime when there is a large amount of water loss, the leaves droop and become more or less flaccid, but they regain their normal position and turgidity again at night when the water loss is reduced.

Both organisms may cause several types of lesions on the seed (fig. 13, A). If the infection occurs when the pods are young, the seed may rot or shrivel (fig. 13, B). If the bacteria enter by way of the funiculus, only the hilum may be discolored. On dark-seeded varieties the discoloration is difficult to detect, but it is very noticeable on varieties with white or light-colored seedcoats.

Frequently seedlings grown from infected seed appear above ground with their growing tips injured or even entirely destroyed. Injury of this type has been referred to as snakehead (see fig. 28, B, C). If such plants do not die, buds may arise in the axils of the cotyledons and produce a dwarfed plant with few pods. Such injury resembles that attributed to seed-corn maggot by Hawley (458) and to the baldhead attributed by Harter (441) to mechanical injury. Large, angular, water-soaked areas frequently occur on the opposite sides of the primary leaves at similar positions, indicating that the initial infection occurred while they were still folded together.

Goss (391) has recently shown that high temperatures modified the symptoms of halo blight and that under the influence of such



temperatures the typical halo was not noted, although the number of infected plants was greater than at lower temperatures. In such cases it would be impossible to differentiate the two diseases.

There sometimes occurs in the field a type of insect injury to the leaves that so closely resembles the symptoms of halo blight that the two can hardly be distinguished. The absence of bacteria in the symptomatic area at once precludes the possibility that it is the halo blight. The spots are more or less irregular in shape and outline and variable in size. They are a pale green to yellow and occur mostly between the veins and rarely extend beyond them, although the small veins or veinlets do not act as barriers.

Often on the underside of the leaf at the center of each spot, a nymph of the white fly (*Trialeurodes abutilonea*)<sup>13</sup> may be seen. This is a very small insect scarcely visible to the unaided eye.

### Causal Organism

#### Nomenclature

At about the time Burkholder (165) recognized halo blight, Hedges (465) was studying a bacterial disease of the kudzu. In 1927 she named and described the causal organism as *Bacterium puerariae* Hedges. A more complete account of the disease was published by her (468) in 1928. Later she (469) compared the kudzu and the halo blight organism, and as a result of cross-inoculation studies discovered that the two organisms produced identical symptoms on both beans and kudzu. Burkholder's description having been published first, *Bact. puerariae* becomes a synonym. The organism is now classified as *Pseudomonas phaseolicola* (Burk.) Dows.

#### Morphology and Physiology

The reader is referred to Elliott (321, pp. 78-80) for a complete description of *Pseudomonas phaseolicola*.

Adam and Pugsley (17) have shown that *Pseudomonas phaseolicola* may occur in at least two forms, smooth and rough, and listed the differences in physiological properties between the two forms. It was demonstrated by means of the agglutination test that the change from smooth to rough involves a corresponding modification of a heat-stabile or somatic antigen. The smooth strain was sensitive and the rough nonsensitive to a bacteriophage obtained from diseased bean seeds. According to Pugsley (796) the halo blight organism possesses both a heat-labile and heat-stabile antigen.

Artemieva (51) made a comparison of the biochemical and serological properties of several isolates of *Pseudomonas phaseolicola*, *Xanthomonas phaseoli* var. *fuscans*, and *P. mori* (Boyer and Lambert).

Israelski and Struminskaya found that extracts of beans infected with different bean bacterial organisms gave specific pre-

<sup>13</sup> Identified by C. F. W. Muesebeck, formerly principal entomologist, U. S. Department of Agriculture.

cipitin reactions with sera prepared against the corresponding organism. Similar results were obtained with infected seeds after removal of nonspecific precipitates (517).

A selective medium was developed by Wilson (1126) to detect seed infection by *Pseudomonas phaseolicola*, as well as isolation of the organism from soil and other sources.

#### Pathogenicity

Burkholder's (165) first account of halo blight carried considerable evidence that infection would take place only through wounds. Spraying the pathogen on the plants gave negative results, whereas inoculations made through wounds produced good infection. He subsequently obtained different results, for he said in a later publication that the organism evidently gains entrance to the plant through the stomata, since mechanical injury is not necessary for infection to take place. These results are in agreement with those of Zaunmeyer (1174) and of Wager (1063), who had no difficulty in obtaining infection without wounding. When inoculated into the stem, petioles, or larger veins of the leaf, the organism enters the xylem and in some cases traverses almost the entire vascular system of the plant. The writers have experienced no difficulty in obtaining good infection by spraying a suspension of the bacteria on the leaves if the plants are confined in an infection chamber with a relative humidity of 95 to 98 percent for about 24 hours after the inoculation. The infected leaves of very small plants often droop at the pulvini, and most of them die in about 14 days. Large plants are not so readily infected by the organism as are young ones. Good infection has been obtained in the field by inoculating plants with a bacterial suspension at 200 pounds' pressure with a power sprayer. No effort was made to maintain a high humidity after inoculation.

At temperatures of 24° to 28° C. the symptoms of halo blight appear in 6 to 10 days and at slightly lower temperatures from 2 to 3 days later (190). The typical halo symptom is noted chiefly at 20° and below, and occasionally a slight halo is present at 24°. At 28° to 32° no halo symptoms are noted, but infection points are greater in number than at lower temperatures; the spots are small and somewhat inconspicuous. These can serve as a source of inoculum for the further spread of the organism.

Jensen and Livingston (532) studied 13 isolates of halo blight and found that on the basis of pathogenicity and symptoms they fell into 3 main groups. Some of the isolates that produced only halo-less lesions at 28° C. gave rise to the typical halo form at 16° and 22°, while others produced exclusively halo-less lesions at all 3 temperatures.

Skoog (918) reported that little or no toxin was produced by *Pseudomonas phaseolicola* when grown in pure culture at temperatures of 20° C. or higher. The optimum temperatures for toxin production are below 20°, which corresponds closely with the optimum temperatures for the production of halo symptoms on bean leaves. Skoog found that the bacteria multiplied about equally in the resistant Red Mexican and susceptible Red Kidney

varieties for the first 3 days after inoculation. After this time a lower population was found in the Red Mexican.

Johnson (539) obtained no infection of beans in the absence of water congestion in the leaf. The preexposure of bean plants outdoors for 5 to 10 days prior to inoculation increased the rate and amount of congestion and the amount of infection.

### Dissemination

Dissemination of the halo blight organism is similar to that of the common blight organism.

Reid (825) found in New Zealand that the dissemination of the disease is limited, rarely spreading for distances of greater than 132 feet.

Hedges (478) was unable to obtain infection on snap beans grown in the spring in a plot where snap beans heavily infected with halo blight had been plowed under the previous autumn. In Australia, Wilson (1127) added cut-up infected plants to the soil and obtained infection in the next crop from seed sown 22, 41, and 52 days later, but not after 64 days, 5 months, or 18 months. He concludes that the soil is an unlikely source of the carryover of halo blight bacteria from one growing season to the next.

It was shown by Diachun and Valteau (280) that *Pseudomonas phaseolicola* can multiply on the roots of wheat, tomato, bean, and soybean in the laboratory. In field tests they were not able to recover the organism from the roots of wheat plants grown in soil to which *P. phaseolicola* had been added.

Bortels (113) reported that the swarming of *Pseudomonas phaseolicola* was depressed during low pressure (cyclonic), and stimulated by high pressure (anticyclonic) atmospheric conditions. The intensity of swarming was reduced by the presence of clouds and increased by their absence.

### Hosts

The halo blight organism has been transmitted to *Phaseolus lunatus*, *P. coccineus*, and *Pueraria thunbergiana*, in addition to *Phaseolus vulgaris*.

### Control

The control of halo blight of beans is in part identical with that recommended for common blight (p. 71). These recommendations consist in the use of disease-free seed and crop rotation. The value of clean seed cannot be emphasized too strongly, because its use largely assures a clean crop the first year. In the United States snap beans or field beans grown in the arid sections of the Intermountain, Southwestern, and Pacific Coast States is relatively free of blight. In New Zealand Reid (825) found that the removal of diseased plants was successful in eliminating the disease in beanfields grown for seed production. Hähne (407) found in Germany that the disease was so widespread that healthy seed was difficult to obtain. Eradication in the field was not satisfactory, because of the masked symptoms of many infected plants.

There have been many experiments conducted with the hope of

finding a fungicide that may be applied to the foliage or in which the seed may be soaked to control the halo blight. In this country the results have not been very conclusive.

In New Zealand satisfactory control has been obtained with various copper sprays (256, 828, 830). At least three applications were recommended, with the intervals between them adjusted to afford protection from the seedling stage to crop harvest.

In Europe (407, 567) the use of several sprays of 0.5 or 1 percent bordeaux mixture reduced infection. In Montana applications of 4-4-50 bordeaux effectively controlled halo blight (21, 695). In Wyoming copper oxychloride, copper sulfate, and yellow cuprocide dusts were effective in greatly reducing the spread of halo blight (1080). Streptomycin sulfate and dihydrostreptomycin sulfate are translocated within the bean for a short distance (684) and will protect the plant from halo blight infection. Translocation of streptomycin from leaves to stems and fruits was not demonstrated. Absorption of streptomycin from soil treated with relatively large amounts could not be demonstrated. The halo blight organism was eradicated by dipping artificially inoculated plants in a 0.025 percent solution of streptomycin shortly after the appearance of initial symptoms. Control was less effective if treatment was delayed several days (686). In the field 0.1 percent aqueous solution of streptomycin sulfate used as a spray gave good control of halo blight when 3 applications were made (1199, 1200). Polymyxin and Aureomycin inhibit the growth of *Pseudomonas phaseolicola* in culture (549).

Seed treatment is of questionable value, because the organism is carried beneath the seed coat and out of the reach of a disinfectant, unless the treatment is rather long, in which case the germination may be considerably impaired. Böning (108) found that seed treated with a fungicidal dust or spray gave much higher yield than untreated seed. Favorable results were obtained by the use of Ceresan and also by soaking the seed in hot water for 30 minutes at 45° C. and for 10 minutes at 50°. Burkholder and Crosby (172) suggested a 20-minute soaking in 1 to 1,000 corrosive sublimate, and Bremer and Hähne (127) reported beneficial results by immersing the seed for 15 to 20 minutes in water at a temperature of 52° to 55° after they had been soaked for 12 hours. Wark (1084) found that treatment of artificially infested seed with a 1 in 500 solution of mercuric chloride in 70 percent ethyl alcohol acidified with 3 percent acetic acid plus 1 in 20,000 gentian violet was effective in reducing infection but impracticable, as it reduced germination. The antibiotics streptomycin, neomycin, bacitracin, chloromycetin, subtilin, and penicillin have reduced the prevalence of halo blight when infected bean seeds were soaked in aqueous solutions of these antibiotics before planting (403, 489, 943, 964). When halo blight infected bean seed was soaked for 2 hours in the culture filtrates of *Penicillium chrysogenum* and *Streptomyces griseus*, the percentage of healthy stands in the field was raised from 46 percent in the untreated seed to 87 and 92 percent, respectively. The incidence of infection was reduced from 35 to 0.2 and 0.1 percent, respectively (566).

Several methods have been developed to detect infected seed lots. Wilson (1124) soaked the seed before sowing, which resulted in an increase in the number of affected seedlings and accelerated the appearance of symptoms. The detection of severely affected samples was also determined in a few days by soaking some of the seeds in water and inoculating bean pods with the infusion. Katznelson (548) made use of a rapid phage-plaque count technique.

Experiments on resistance and susceptibility of varieties to halo blight have been conducted in Australia (778, 797, 810), England (735), Germany (957, 958, 959, 960), Holland (656), New Zealand (255, 826, 827), Poland (1169), Union of South Africa (1063), and United States (132, 175, 488, 1080).

Many field bean varieties are highly resistant to halo blight infection. Among them are such field beans as Michelite, Great Northern, Pinto, Pinks, and Red Mexican, which do not become infected in the field. When leaves of these varieties are rubbed with a suspension of halo bacteria small necrotic spots develop with little or no systemic spread of the organism. The Great Northern variety was used in developing the resistant snap bean Fullgreen (46). In Australia, Richmond Wonder, Clarendon Wonder, Hawksbury Wonder (910), and Windsor Longpod (10) are resistant varieties developed by hybridization. Jensen and Goss (531), in a search for resistant varieties and a method for identifying them, found that small, inconspicuous necrotic lesions on inoculated leaves instead of large necrotic spots that develop on susceptible varieties indicated physiological resistance. Their results showed that the Red Mexican and Schwert 27, a German variety, in all stages of growth were resistant at temperatures of 16°, 22°, and 28° C. Pod inoculation on these two varieties produced small, rusty-colored necrotic lesions instead of large water-soaked ones.

## Wilt

### Geographical Distribution and Economic Importance

Bean wilt, caused by *Corynebacterium flaccumfaciens* (Hedges) Dows. (*Bacterium flaccumfaciens* Hedges), was first recognized and described as a new disease in 1922 (461). Since wilt occurs in certain localities with the common and halo blights and the three diseases have certain symptoms in common, wilt may be confused with the other two diseases.

The exact distribution of wilt is not known. The causal organism has been reported from the District of Columbia, Maryland, Michigan, Montana, New York, North Dakota, Ohio, South Dakota, West Virginia, and Wyoming. Since 1946 it has been observed by the writers in Colorado, Nebraska, and Idaho. The disease has also been reported from southern Africa (1063), Australia (16), Belgium (956), Bulgaria (57, 571), France (464), Germany (956), and New Zealand (824).

Wilt has been serious in certain bean-producing areas since 1946. In 1946, 1947, and 1948 serious reductions in yield because of wilt infection were observed near Riverton, Wyo., by the

writers. In 1951 many fields near Wiggins, Colo., were almost a total loss because of the severity of the disease. Burke and Starr (155) found that of 53 Wyoming beanfields surveyed, wilt occurred alone in 19, and in combination with *Xanthomonas phaseoli* in 15. *X. phaseoli* was isolated alone from beans in 10 fields and *Pseudomonas phaseolicola* from beans in 9 fields. In one field all 3 bacterial diseases occurred.

### Symptoms

The wilt organism is seedborne; because of that fact the seedling is subjected to possible infection as soon as the seed germinates. Young plants 2 or 3 inches tall may be attacked, and at that size are usually killed. If they survive an early attack or if infection first becomes manifest after they reach considerable size, they may live throughout the season and mature seed.

The leaves become flaccid and hang limp along the stem and branches of the plant and finally wilt and die (fig. 17, B). The earliest wilt symptoms are observed easiest during the warmest part of the day when the leaves become flaccid. If the disease is not too far advanced the leaves gradually regain their turgidity during the cooler part of the day. When water is cut off from the leaves as a result of bacterial plugging of the vascular bundles, they become brown and drop off. The typical wilting symptoms sometimes are not observed. Instead, golden-yellow, necrotic leaf lesions, closely resembling those of common blight, occur. The margin of the lesions tends to be more irregular than that produced by common blight.

On the surface of stems, leaves, or pods affected by wilt, water-soaked spots so characteristic of the common and halo blights are not conspicuous. The causal organism follows the sutures of the pods just as common and halo blight bacteria do (fig. 17, A) and may infrequently appear on the surface. The vascular bundles of this tissue are sometimes darkened. The wilt organism may penetrate the hilum of the seed and form yellow masses of bacteria under the seedcoat, or, without entering the seed, form incrustations on the outer surface. Seed infection is often evident only after the pod is opened and the seed examined (fig. 17, A). If the disease appears on the pods, other than along the sutures, the spots are occasionally water-soaked and resemble those caused by common and halo blights. Infected seed of white-seeded varieties are conspicuously yellow even when infected systematically, as the bright-yellow masses of bacteria are plainly visible through the seedcoat; but they are not easily recognized in beans with colored seedcoats. The wilt is more conspicuous on ripe pods, where it produces an olive-green color, than on green ones. The causal organism is readily isolated from infected stems, leaves, pods, and seed.

### Causal Organism

#### Morphology and Physiology

*Corynebacterium flaccumfaciens* is described in detail by Elliott (321, pp. 20-21) as *Bacterium flaccumfaciens*. Distinguishing features of the wilt and common blight organism were studied

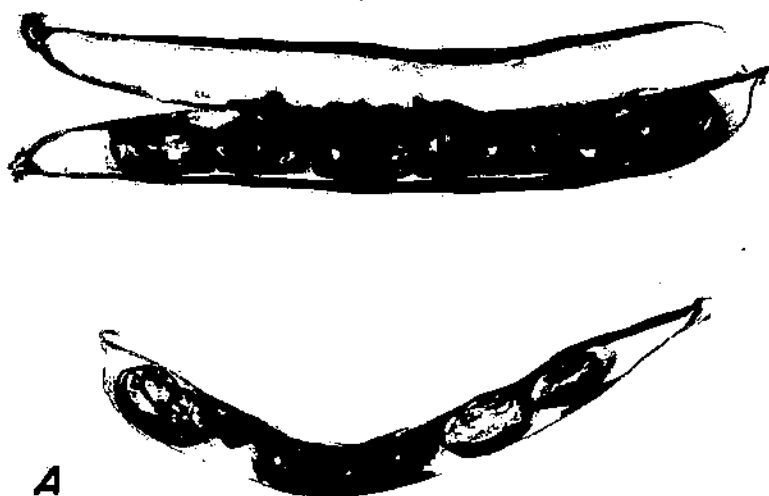


FIGURE 17.—Bacterial wilt caused by *Corynebacterium flaccumfaciens*: A, Infected seeds; B, late stage of the disease showing complete wilting of a plant.

by Hedges (462, 464), who compared their cultural characteristics. She demonstrated that the wilt organism is Gram-positive, a characteristic that definitely distinguishes it from the common blight organism, which is Gram-negative. Schuster and Christiansen (886) found a strain of the wilt organism in Nebraska that produces an orange coloration of infected bean seed.

#### Dissemination

Similar to common and halo blights (pp. 68 and 82).

Observations made by the writers in the irrigated bean-growing areas in Colorado indicate that the wilt bacteria are spread within a beanfield by the irrigation water and can cause infection in the absence of rain. Because of limited external development of the disease on plants, wilt is less rapidly spread by rains than are halo, common, and fuscous blights. However, after hailstorms wilt has been observed to spread over almost the entire field where only a few infected plants were previously noticed. The organism infects the seed (466) and is known to live at least 5 years in the seed.

#### Pathological Histology

Zaumeyer (1173, 1174) investigated the comparative pathological histology of the common, halo, and wilt pathogens. *Corynebacterium flaccumfaciens* is primarily a vascular parasite. Both the common and the halo blight organisms may invade the vascular bundles, but they show a preference for parenchymatous tissue. The wilt organism does not infect through the stomata, while the common and halo blight organisms do. This difference in habit means that very little water soaking of any part of the surface of the plant is induced by the wilt pathogen, while it is a common characteristic of both common and halo blights. The bacteria causing common and halo blights fill the stomatal cavities and produce water-soaked spots of various sizes from which they emerge, forming incrustations of bacterial slime on the surface. This is not true of wilt. All three organisms behave much alike in their ability to dissolve the cell walls.

#### Hosts

Hedges (461) demonstrated the wilt organism to be parasitic on King of the Garden lima bean (*P. lunatus* f. *macrocarpus*) and *Glycine max* var. Iota San. Burkholder (166) later added Scarlet Runner bean (*P. coccineus*), adzuki bean (*P. angularis*), cowpea (*Vigna sinensis*), yard-long bean (*V. sesquipedalis*), and hyacinth-bean (*Dolichos lablab*) to the list. Rands and Brotherton (808) added *P. aureus* and *P. mungo*. Wellhausen (1105) was able to kill maize seedlings in the greenhouse at 80° to 90° F. by inoculation with *Corynebacterium flaccumfaciens*. No one has attempted to determine the entire range of susceptible hosts.

#### Control

The isolation of the wilt organism from seed 24 years old suggests that the bacteria are not easily killed (170). Kovachevsky (571) has shown that the bacteria were not killed in seed heated



for 20 hours at 52° C., then kept at 75° for 20 hours, and finally at 85° for 3 to 5 hours. This treatment, while not completely destroying the bacteria, materially reduced the percentage of seed that germinated. Burkholder (166) was able to isolate the organism from seed heated for 1 hour at 100°. The wilt pathogen probably does not spread as readily as the common and halo blight organisms do. It is not known if it will live over winter in the soil or on the debris from previous crop. Inasmuch as the organism infects only through seed and wounds, infection by means of splashing from the soil during rains and dissemination from the leaves, pods, and other sources to other plants would not occur to the extent it would with common and halo blight. The danger of infection during seed inoculation by nitrogen-fixing bacteria (*Rhizobium phaseoli*) by the wet method is indicated by the results of Leonard (602, 603), who demonstrated wilt to be much worse in fields sown to treated seed than in those where the seed was untreated. Hedges (470) pointed out that the practice of examining the fields both early and late during the growing season and using as seed only that which has a minimum of disease, accompanied by hand picking, contributed greatly to the cleanup of the seed stock. No immune or resistant varieties are known.

Very little research has been attempted in the way of developing resistant varieties. Kovachevsky (574) stated that none of the beans native to Bulgaria are resistant to wilt and that the growing of the crop is becoming unprofitable because of the heavy losses. Rands and Brotherton (808) conducted a test in the United States with a large number of American and foreign varieties, but the results were not conclusive enough to enable them to class the varieties according to the degree of susceptibility. None were found to be resistant. There is no effective method for controlling wilt. Probably the only permanent and effective method must await the production of resistant varieties by hybridization and selection.

## Bacterial Diseases of Minor Importance

### Bacterial Brown Spot

Brown spot, caused by *Pseudomonas syringae* Van Hall (*Phytophthora vignae* var. *leguminophila* Burk.), was described by Burkholder (166) from specimens collected in New Jersey as a new disease of beans. It is a disease of rare occurrence in the United States. Its symptoms are very similar to those caused by other bean bacterial pathogens, except that a ring spot is produced on the pod. If the disease becomes systemic, lesions occur along the stem. Older plants are somewhat resistant. The organism develops rapidly when first inoculated on the leaves and stems, but a few days thereafter the lesions enlarge very little. In this respect it differs materially from other bacterial pathogens of the bean.

A strain of the organism isolated from *Vigna sesquipedalis* was found to be a weaker parasite than *Pseudomonas syringae*, and, unlike the latter, the strain never produces a ring spot on the pod. Otherwise the symptoms caused by the two organisms are

very similar. The strain from *V. sesquipedalis* infected *Phaseolus vulgaris*, *Pueraria hirsuta* (*P. thunbergiana*), and *V. sinensis* (166). The New Jersey strain infected *Phaseolus lunatus*, *Dolichos lablab*, *Glycine max* var. Wilson (*Soja max*), *Vicia faba*, *Vigna sinensis*, *V. sesquipedalis*, and *Pueraria hirsuta*. It causes symptoms on the different legumes similar to those produced on the bean. The New Jersey strain is the only one of the bacterial group pathogenic on beans that is also pathogenic on *Vicia faba* L. This information is valuable as a means of identifying the organism.

### Brown Rot

*Xanthomonas solanacearum* (E. F. Sm.) Dows. (*Bacillus solanacearum* E. F. Sm.), the cause of a disease principally of tomatoes, eggplants, and potatoes, was isolated in 1919 by Smith and McCulloch (933) from snap beans grown in Florida. The symptoms are characterized by a browning and wilting of the leaves and petioles, a browning of roots, and a local decay of the epidermis. The vascular bundles of the stem and roots are stained dark. Inoculation experiments showed that the organism was parasitic on snap and lima beans as well as on certain other legumes, such as peas, cowpeas, and peanuts. Other investigators have shown that the organism was parasitic on a number of other species. The expression of disease symptoms are rather prompt and decisive following inoculation by the needle-prick method.

The disease has never been of much economic importance on beans in the United States, although the losses in Florida for a single year were said to amount to 20 percent of the crop. It is possible that it may have been confused with or mistaken for other bacterial diseases of beans.

The organism originally described by Smith (927) as *Bacillus solanacearum* was later transferred by him to the form genus *Bacterium* (92). A condensed account of this organism, including a description, its synonymy, and citations to additional literature are given by Elliott (921, pp. 139-142).

### Gall Blight

Gall blight, caused by *Pseudomonas viridiflava* (Burk.) Clara (*Phytomonas viridiflava* Burk.), was described by Burkholder (166), who recorded the results of inoculation experiments and the behavior of the causal organism on culture media. This pathogen was isolated from bean pods collected in Switzerland in 1927. The writers are not aware that this bacterium has been observed or reported in the United States. Needle-prick inoculations of young seedlings resulted in visible infection in 2 days and killed the plants in 4 days, thus showing that the organism was extremely virulent. When the stems were inoculated, the lesions extended less than an inch above and below the points of inoculation, the distance covered depending on the age or the succulence of the host tissue. The stem tissue is eventually killed, and the upper part of the plant dies. Below the lesion the plant remains alive and

beneath the epidermis a gall that often becomes twice the diameter of the stem begins to form. No hypotrophy was observed on the pod, and there was not the water soaking so characteristic of early infections of the common and halo blight organisms. No infection was obtained except through wounds.

Inoculation experiments by Burkholder (166) proved the susceptibility of the following hosts: *Phaseolus coccineus*, *P. lunatus*, *Dolichos lablab*, *Vigna sinensis*, and *Pueraria hirsuta* (*P. thumbergiana*). Elliott (321, p. 100) also lists *Delphinium cultorum*.

### Streak

Streak, caused by (*Erwinia lathyri* Manns and Taub.) Holland (*Bacillus lathyri* (Manns and Taub.)), is principally a disease of the sweetpea (*Lathyrus odoratus* L.), but it has been shown by Manns (647) to cause a similar disease of peas and snap and lima beans. *E. lathyri* was isolated from the stems of snap beans, and infection was obtained by the use of cultures of the organism derived from the sweetpea.

The disease on the sweetpea has been reported from Delaware, Maine, Massachusetts, and New York and from the British Isles. So far it has attained no economic importance as a bean disease in the United States. The parasite is a yellow, rod-shaped organism having rounded ends and peritrichiate flagella. The streak disease at one time was attributed to *Thielaviopsis basicola* and a species of *Alternaria*, or confused with diseases caused by them. According to a number of investigators there appears to be no convincing proof of the pathogenicity of this organism.

## Virus Diseases of Major Importance

### Common Bean Mosaic

#### Geographical Distribution and Economic Importance

Common bean mosaic (bean virus 1) was first observed by Iwanowski (521) in Russia about 1894. In 1908 Clinton (215) reported an infectious chlorosis of beans, which was probably common bean mosaic, in Connecticut. Stewart and Reddick (974) reported that the disease caused large losses to the crop in New York during the summers of 1916 and 1917.

Subsequently, Stone and Howitt (977), Archibald (49), and Güssow (404) reported it from Canada. About 25 percent of the plants were infected. Barss (77) stated that the same disease had been observed in Oregon in 1917 and that it had occurred there at an earlier date. According to Spragg and Down (952), mosaic may have been present in Michigan as early as 1908. Ogilvie (732) reported its occurrence in Bermuda in 1923, and Porter (781) in eastern China in 1926. Grainger (394) reported that bean mosaic occurred in England in 1929 but that he believed the disease had been present for a much longer time. Chamberlain (197) reported it from New Zealand in 1933.

Bean mosaic is probably worldwide in its distribution. In 1925 Rands and Brotherton (808) tested several hundred varieties of beans collected from many parts of the world for studying resistance to several bean diseases. They noted mosaic symptoms on seedlings from the following: Argentina, Belgian Congo, Brazil, Chile, Colombia, Czechoslovakia, Ecuador, England, France, Germany, Guatemala, Honduras, Italy, Japan, Java, Mexico, the Netherlands, Peru, Union of Soviet Socialist Republics, Uruguay, and Venezuela. Feiginson (340) reported that beans suffer considerable loss from mosaic in the U.S.S.R. In the United States it has been reported from 42 States, and it is not unlikely that it is present in every State. Cass Smith (195) reported the widespread nature of the disease in Australia and its possible menace to the bean industry there.

In Idaho the loss caused by mosaic amounted to about 5 percent in 1920 and to about 10 percent in 1921. In 1927 Harter (439) reported mosaic in southern Idaho to range from none to 85 percent, depending upon the variety. In 1929 Zaumeyer (1171) noted from 20 to 100 percent infection in the Stringless Green Refugee variety in the western seed-producing States. He reported mosaic widespread in southern Idaho, slightly less in Colorado and Utah, and still less in Montana, Wyoming, and California. The same year almost 100 percent infection with a reduction in yield occurred in the Stringless Green Refugee variety in Michigan. In 1930 Horsfall (496) visited 42 fields of snap beans for canning in New York, and in 32 of them he found from a trace to 100 percent infection. He estimated an average loss of about 10 percent. In 1932 Harrison (434) and others found mosaic to be of considerable importance in all localities in New York. More than 75 percent of the fields of Stringless Green Refugee examined showed 80 to 100 percent infection. Harrison and Burkholder (433) reported bean mosaic as the most destructive disease observed in New York on the Stringless Green Refugee in 1936, the infection ranging from 10 to 100 percent.

In 1945 (1181) the disease was widespread in Colorado and Idaho in fields of susceptible varieties. In many fields in Colorado, 75 percent of the plants were infected, reducing the yields in such fields by one-third. In 1947 (1195) the disease was again serious in southern Idaho in fields of susceptible varieties. The strain of common bean mosaic reported by Richards and Burkholder in New York in 1943 was quite widespread in fields of susceptible varieties such as Great Northern U.I. 15 and Red Mexican 3 and 34 in Idaho in 1945 and 1947. In one field of Red Mexican 34 practically 100 percent of the plants were infected. In 1953 common bean mosaic was widespread in fields of Red Mexican 3 in eastern Washington, where it caused considerable yield reductions.

### Symptoms

Bean mosaic rarely kills the plant, and slight infections do not produce conspicuous symptoms. Like mosaic diseases of many other crops, it stunts (282) the plant and causes mottling and various types of leaf malformations (430) (fig. 18). The symptoms differ slightly with the variety and age of plant and

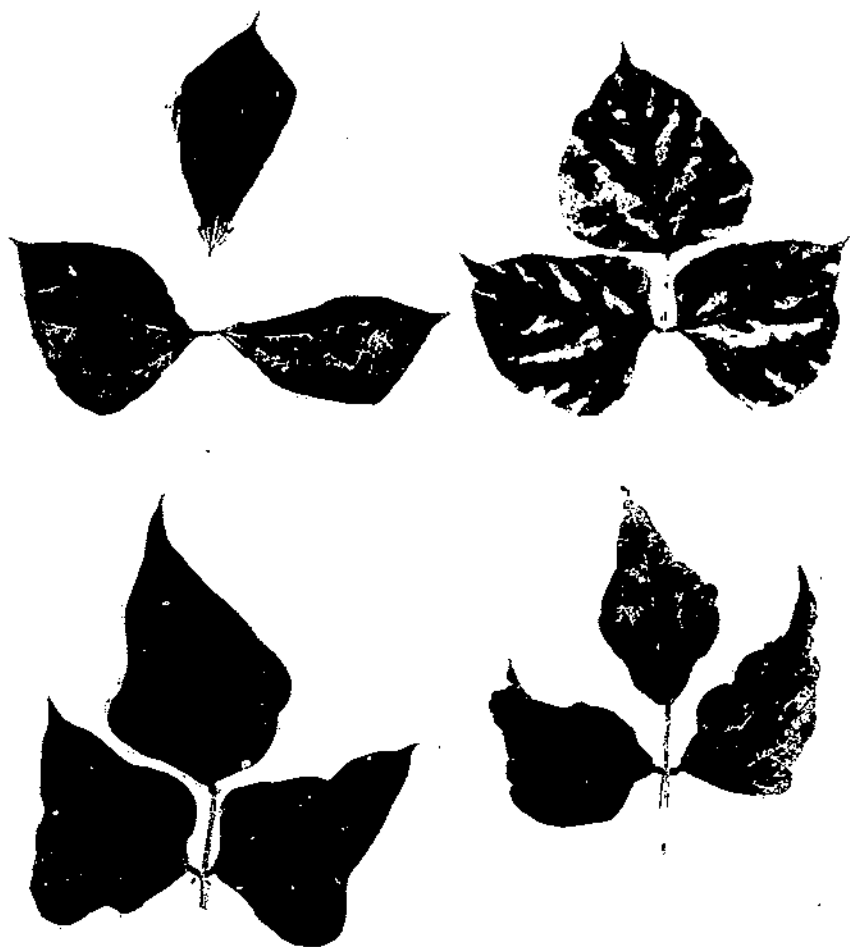


FIGURE 18.—Common bean mosaic, showing variations in type of leaf symptoms of several bean varieties.

somewhat with the conditions under which the plant is grown. Sometimes the symptoms may be noted on one or both of the simple leaves, which are slightly mottled, curled, and undersized. Trifoliate leaves affected with mosaic usually have irregular-shaped, light-yellow and green areas (fig. 18) of various sizes, a type of mottling characteristic of mosaics in general, which may be the only evidence of the disease. Besides the characteristic mottling, there may be considerable puckering, malformation, and other alterations in the shape of the leaves. Infected leaves may be narrower and longer than normal ones, with downward cupping caused by the unequal growth of the tissue. Bean plants attacked early in the season usually are yellowish in color, often are

dwarfed and spindling, and fail to produce a normal crop. The symptoms of mosaic cannot be recognized on the stems or seed.

Failure to set pods on mosaic-infested plants has been attributed by Nelson (725) to defective pollen, although other factors may be partially responsible. The pods on severely infected plants are usually undersized and contain fewer ovules than those produced on normal plants. They are occasionally covered with small, dark-green spots (431) and often mature very late, and when harvested the seed is frequently shriveled and undersized. High temperature favors the expression of symptoms, and a low temperature has a tendency to mask the symptoms.

In 1940 Jenkins (528) described what appeared to be a new virus disease of beans, which he named black root. In 1941 he (529) published the results of a histological study of the different tissues affected by it. During the 1941 season black root caused 40 percent mortality in the experimental plots in Georgia. Under field conditions, the first symptoms are a slight wilting of the leaflets near the top of the plant during the blooming or early podding period. The plants lose their bright-green color and appear grayish green. A permanent general wilting of all the leaves and finally death of the plant follows, although under certain conditions some viable seed is produced. These symptoms are accompanied by a vascular necrosis of the root, stem, leaves, and pods (fig. 19, A, B, D.)

The roots are dark, and in severe cases the taproot is almost black. External dark discoloration of the lower hypocotyl and petioles is also evident. The dark discoloration of the stem is sometimes confined to one side (fig. 19, A.)

A streaking frequently appears along the dorsal and ventral sutures of the pods (fig. 19, D). Young pods shrivel, but older ones, when severely infected, show an inky appearance as a result of vascular infection. Cross sections of such pods show vascular discoloration of varying degrees throughout the entire pod (fig. 19, C.)

Under greenhouse conditions, infected seedlings manifest symptoms not observed in the field. The first noticeable symptoms on the inoculated leaves are relatively large spreading local necrotic lesions or merely the necrosis of one or more veins (fig. 19, B). Occasionally the infection makes no further progress, but more frequently the young trifoliate leaves first show a slight wilting followed by a necrosis of the growing tip and, later, death. The necrosis may extend down the stem to the soil line and the plant finally dies.

The systemic necrosis referred to as black root was found to be a symptom expression of common bean mosaic virus (401, 1195). The symptoms of black root are produced on varieties whose resistance to common bean mosaic is derived from Corbett Refugee, Kentucky Wonder, and Creaseback types when inoculated with these viruses. These symptoms do not develop on varieties deriving their resistance from Great Northern U.I. No. 1 and Robust. Thomas (1005) found that high air temperatures were necessary for the systemic necrotic symptoms to develop. When young bean plants were exposed at 32° C. or above, veinal necrosis was ob-

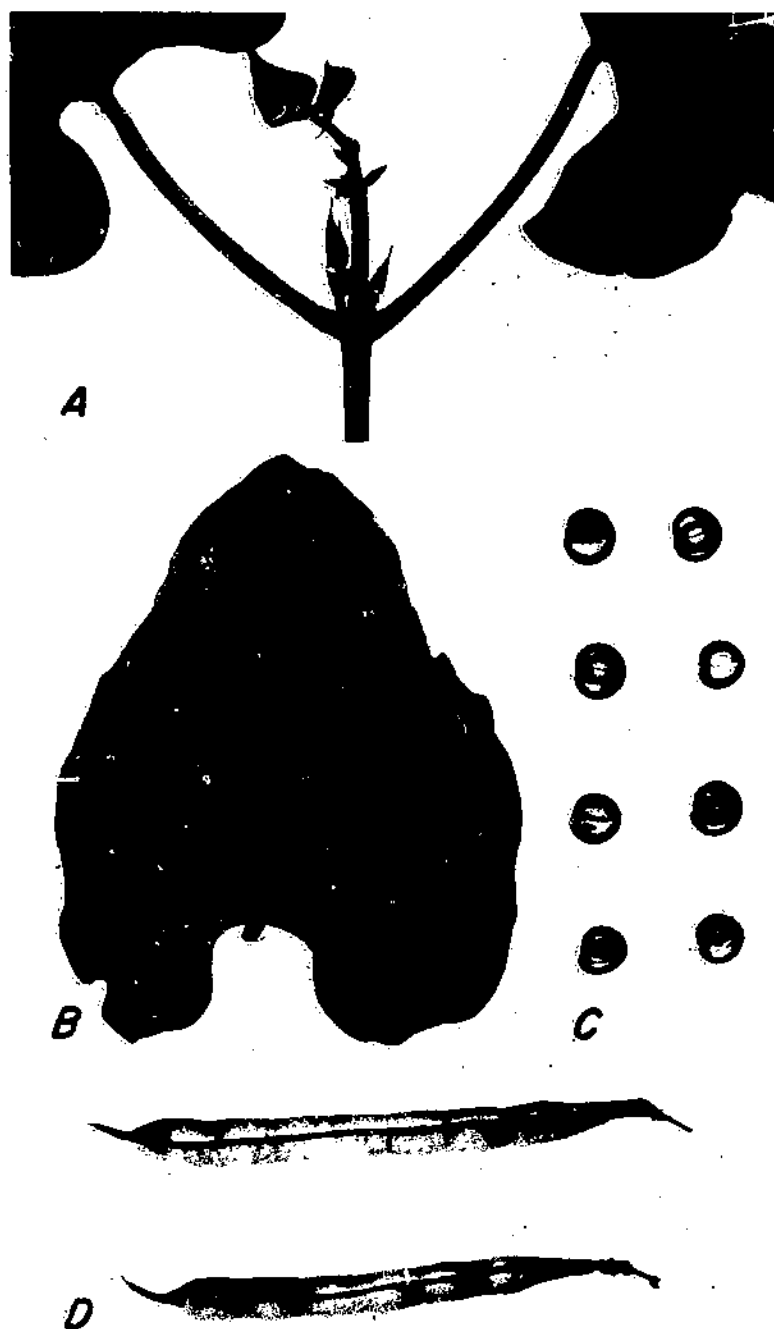


FIGURE 19.—Black root virus showing variations in symptoms: *A*, Vascular necrosis of stem (dark streak along stem); *B*, vernal necrosis or inoculated primary leaf; *C*, cross sections of pods showing vascular discoloration; *D*, dark discoloration of dorsal suture of pods.

served in inoculated plants within 40 hours. Growing plants in soil at 20°, 25°, and 30° had little or no effect on the development of leaf veinal necrosis.

Bridgmon (184) has shown experimentally that southern bean mosaic virus may induce symptoms of black root in varieties of beans expressing the local-lesion type of symptom expression. He suggests that the original description of black root may have referred in part to systemic necrosis induced by the southern bean mosaic virus.

Although 100 percent black root infection, accompanied by killing of the plants, was noted in a number of experimental plantings in the West, only a trace of such infected plants has been observed in commercial plantings of varieties resistant to common bean mosaic. This has been true even in years when common bean mosaic has been very prevalent on susceptible varieties.

In 1943 a strain of common bean mosaic was reported by Richards and Burkholder (840) and later by Dean and Hungerford (272). The symptoms produced by this virus cannot be distinguished from those of common mosaic. Grogan and Walker (401) showed that this virus also produced black root symptoms on the same varieties as were produced by the common bean mosaic virus. This strain of the virus is most readily differentiated from common bean mosaic by its infectiousness of certain bean varieties that are resistant to the common bean mosaic virus. Some varieties that are resistant to the common bean mosaic virus but susceptible to the strain are Michelite, Pinto U.I. Nos. 72, 78, and 111, and Red Mexican U.I. 3 and 34. Snow (945) reported another strain of the common bean mosaic virus which was infectious to Red Mexican U.I. 3, Topcrop, and Blue Lake beans, alsike clover, and gladiolus.

### Cause

Common bean mosaic is caused by *Marmor phaseoli* Holmes (125). The virus is inactivated when held for 10 minutes between 56° and 58° C. Dilution end point in fresh plant extract from infected bean is between 1 to 800 and 1 to 1,000. It resists aging for 28 hours in vitro at 18°C. but not for 32 hours.

Preliminary studies by the writers showed that the physical properties of the strain are comparable to those of the common bean mosaic virus.

Hedges (471) found that the virus of common bean mosaic persisted in cultures of *Xanthomonas phaseoli* for 6 weeks and in those of a variant of this organism for 11 days.

### Transmission

#### Mechanical Transmission

The infectious nature of bean mosaic was first demonstrated in 1918 by Reddick and Stewart (818), who obtained infection by rubbing the upper surface of young leaves with crushed leaf tissue from mosaic-infected plants. Clinton (215), on the other hand, failed to reproduce the disease in healthy plants by this method. Later investigations by Elmer (329), Fernow (342),



1 TB 868 (1957) USDA TECHNICAL BULLETINS 868 - 12 OF 3  
A MONOGRAPHIC STUDY OF BEAN DISEASES AND METHODS FOR THEIR CONTROL  
ZAUNEYER, W. J. THOMAS, H. R.

Merkel (666), Pierce and Hungerford (776), Fajardo (334), and Nelson (725) showed that the disease is not easily transmitted in this way.

Zaumeyer and Wade (1201) obtained a relatively high percentage of infection without the use of an abrasive. The writers obtained almost 100 percent infection by the use of an abrasive such as carborundum powder dusted on the leaves previous to inoculation. The new strain of the virus is transmitted as readily as common bean mosaic. Atomizing plants with diluted infected juice containing carborundum powder at about 30 pounds' pressure resulted in good infection (841). Thomas and Fisher (1006) described a rapid method of testing snap beans for resistance to common bean mosaic virus. Young inoculated plants were kept at 90° F. for 3 to 4 days until necrosis developed on beans with the dominant genetic factor for resistance. No necrosis developed on the susceptible plants.

#### Insect Transmission

In 1922 Nelson (724) reported the transmission of bean mosaic with *Macrosiphum solanifolii* (Ashm.). Elmer (329) believed that *Pseudococcus maritimus* (Ehr.) was a vector. Fajardo (333) was able to transmit bean mosaic with *Aphis rumicis* L., *Myzus persicae* (Sulz.), *Macrosiphum solanifolii*, and an undetermined species of mealybug. Pierce and Hungerford (776) transmitted the virus with an undetermined species of black aphid, but not with a green aphid from alfalfa, one of the green leafhoppers, and the tarnished plant bug. Merkel (666) reported that *A. rumicis* and *Macrosiphum pisi* (Kalt.) transmitted the bean virus. Smith and Barker (942) in Haiti reported the transmission of bean yellows, which resembles mosaic, by *Empoasca* sp. It is possible Smith and Barker may not have been working with the common bean mosaic.

Zaumeyer (1176) and Zaumeyer and Kearns (1194) reported transmission with *Aphis gossypii* Glov., *A. medicaginis* Koch, *A. rumicis*, *A. spiraeicola* Patch, *Brevicoryne brassicae* (L.), *Hyalopteris atriplicis* L., *Rhopalosiphum pseudobrassicae* (Davis), *Macrosiphum ambrosiae* Thos., *M. solanifolii*, *M. pisi*, and *Myzus persicae*, but not with *Neothomasia populicola* Thos. They were unable to transmit the disease with other insects found on beans, such as *Epitrix cucumeris* (Harr.), *E. fuscata* Crotch, *Aceratagalla sanguinolenta* Prov., *Empoasca fabae* (Harr.), *E. filamenta* DeL., *Lygus oblineatus* (Say), and several others. Their results indicated that, although aphids were not so numerous on beans as many other species of insects, they were responsible for the dissemination of mosaic in the field. Zaumeyer and Kearns believed that even though the bean is not a favored host for aphids, which do not frequent it in large numbers, they may feed on infected bean plants and transmit the virus to healthy ones, in their search for a more favored host.

#### Seed Transmission

Reddick and Stewart (820) demonstrated the seedborne nature of the bean virus and noted considerable variability in the per-

centage of virus transmission in the seed from different mosaic plants. Burkholder and Muller (174) found that plants affected with the mosaic seldom give rise to more than 50 percent diseased plants. Merkel (666) reported that only 34.5 percent of the seedlings from 1,000 seeds collected from field-grown infected plants showed the mosaic symptoms. These results agree closely with those of Pierce and Hungerford (776), who found that seed from infected plants transmitted the virus to about 33 percent of the seedlings. About 48 percent of primary-infected seed transmitted the disease. Nelson (725) showed that about 50 percent of the seed of primary-infected plants was infected. He maintained that the variability of mosaic transmission in the seed might be explained by certain characteristics in the vascular anatomy of the bean pod, assuming that the virus moves through the vascular tissue. He argued that the virus might be present in only certain elements of the vascular bundle and that only the seed having a direct connection with the infected tissues would become infected.

Fajardo (535) proved, and later was confirmed by Harrison (432), that the percentage of seed infection is correlated with certain stages in the growth of the plant. If infection occurs before the plants bloom, the seed might carry the virus. If, on the other hand, they became infected after the blossoms are set, no seed infection results. Nelson (725), however, had observational evidence that indicated that in rare instances the Refugee variety, which is very susceptible, appeared to transmit the virus through the seed after the blossoming period. Smith and Hewitt (935) noted that, in general, varieties most severely infected produced a higher percentage of infected seed than those less affected.

In testing the viability of the seed of several bean varieties collected from 1897 to 1899, Pierce and Hungerford (776) obtained germination of two seeds of one variety in a greenhouse test. One of these plants showed mosaic; from this, they concluded that the virus had survived in the seed for at least 30 years.

#### Pollen Transmission

In 1918 Reddick and Stewart (818) suggested that pollen may be a means of transmitting the virus from diseased to healthy plants. However, Merkel's (666) investigations on virus transmission by pollen gave only negative results. Nelson and Down (726), on the other hand, showed the virus to be present in about one-fourth of the ovules and pollen grains in crosses between mosaic-infected Refugee and healthy Early Prolific varieties. They obtained additional proof in crosses between Refugee and Robust, a resistant variety, in which about one-fourth of the hybrid seedlings were severely infected with mosaic.

#### Hosts

Reddick and Stewart (818) reported *Phaseolus lunatus* f. *macrocarpus*, *P. acutifolius* var. *latifolius*, and *Vicia faba* as being susceptible. Fajardo (535) was unable to transmit the virus to any other hosts except bean. Nelson (725), relying on natural field infection, reported, among others, the following legumes to be susceptible: *P. acutifolius* var. *latifolius*, *P. angularis*, *P. aconiti-*

*folius*, *P. calcaratus* Roxb., *P. mungo*, *P. limensis* Macf. (*P. lunatus* f. *macrocarpus*), *P. coccineus*, *V. faba*, and *Vigna sesquipedalis*. Pierce (773) confirmed Reddick and Stewart's results (819) and found also that *P. calcaratus* and *P. lunatus*, as reported by Nelson, were susceptible. He was unable to confirm Nelson's results on the susceptibility of *P. angularis*, *P. coccineus*, and *V. sesquipedalis*.

Zaumeyer and Wade (1201) were unable to confirm the results of these workers, and found that *Phaseolus vulgaris* was the only susceptible host. Species tested represented 16 genera in the Leguminosae. Preliminary studies by the writers showed that the new strain infected the same hosts that were infected by the virus of common bean mosaic.

### Control

The only satisfactory method of control for common bean mosaic is the use of resistant varieties, of which a number, both of field and snap bean varieties, are available. Roguing the infected plants is helpful in reducing the amount of secondary spread but costly if a high percentage of mosaic-infected plants are present. Some seed companies practice roguing where the acreage is relatively small. In large commercial acreages it is practically impossible to profit greatly by this method, because of secondary spread unless plants grown nearby are likewise free of mosaic.

According to Pierce and Hungerford (776), beans planted early usually suffer less from mosaic than those planted late in the season, probably because insect activity is less in the early spring. Unfortunately, it is not always desirable or practicable to plant early, since if beans are planted too early the yields may be poor because of poor germination, frost, or other unfavorable conditions.

Harrison (431) subjected mosaic-infected seed to temperatures as high as 100° C. for several hours, but was unable to destroy the virus. Exposing the seed to formaldehyde fumes and to X-rays had no influence on the virus.

Reddick and Stewart (818, 819) tested many dry and snap bean varieties for susceptibility to mosaic, but they reported only a few as resistant; all of these except Robust were later found to be susceptible.

Rands and Brotherton (808) tested a large number of varieties of both foreign and domestic origin and found all except Robust to be susceptible. In 1929 a mosaic-resistant Great Northern was distributed by the University of Idaho as U.I. No. 1. Since then several other Great Northern types, namely, U.I. No. 59, U.I. No. 81, and U.I. No. 123, have been developed and have replaced the common Great Northern variety in commercial production (772). In 1938 Michelite (291), a mosaic-resistant type similar to Robust, was introduced. In 1940 there were introduced two mosaic- and curly-top-resistant Red Mexican beans known as Red Mexican U.I. Nos. 3 and 34 (709). A little later a Great Northern selection known as Great Northern U.I. 15 was released (708). The variety is no longer being produced because of its susceptibility to the strain of common bean mosaic. In 1949 U.I. 15 was

replaced by two other similar strains; namely, U.I. Nos. 16 and 31, which resist both the common and the variant strain of common bean mosaic as well as curly top viruses.

In 1945 there were introduced several mosaic- and curly-top-resistant Pinto varieties known as U.I. Nos. 72, 78, and 111, and in 1946 Small Flat White U.I. No. 1 was introduced (502). It is resistant to common bean mosaic.

Monroe, a mosaic-resistant pea bean type, was released by the New York Agricultural Experiment Station at Cornell.

In 1932 a mosaic-resistant type known as Corbett Refugee was selected from Stringless Green Refugee and later hybridized with the latter. Two mosaic-resistant progenies derived from this cross known as Wisconsin Refugee and Idaho Refugee were introduced commercially (777, 1067). A cross between U.S. No. 1 Refugee and Corbett Refugee resulted in the development of mosaic-resistant U.S. No. 5 Refugee (1061).

A number of mosaic-resistant snap beans are also available. In 1941 Sensation Refugees No. 1066 and No. 1071 (39), two types resembling Idaho Refugee, and Medal Refugee, a type similar to U.S. No. 5 Refugee, were introduced commercially. Other recent bush snap bean introductions are Contender (179), Florida Belle (1031), Idagreen, Improved New Stringless, Logan (1059), Puregold, Rival, Tenderlong 15, Topcrop (1134), Wade (492), Hyscore, Kingreen, Processor, and Seminole (1141). Some of the older resistant varieties are the Kentucky Wonder types, the Blue Lake strains, except Blue Lake 65, and Ideal Market.

All of the above-mentioned varieties are also resistant to the variant strain of common bean mosaic except Red Mexican U.I. Nos. 3 and 34, Pinto U.I. Nos. 72, 78, and 111, Robust, and Michelite.

## Yellow Bean Mosaic

### Geographical Distribution and Economic Importance

The distribution of yellow bean mosaic (bean virus 2) and its strains in the United States is not definitely known, although it has been observed and reported in widely separated localities. The only other reports of its occurrence outside of the United States are from East Africa (1073), Germany (349, 800), Canada (92), Holland (500), Italy (648), and Switzerland (120). Since it is not seedborne, its occurrence is largely correlated with the growth of *Melilotus alba* Desr., one of its principal hosts (20), as well as *Trifolium pratense*, *T. incarnatum*, and *Gladiolus* sp.

The losses caused by yellow bean mosaic are frequently very large. As the symptoms of yellow bean mosaic and its strains and common bean mosaic often overlap, it is almost impossible to assign any particular amount of loss to an individual virus. Furthermore, it is likewise difficult to determine whether any of the strains of yellow bean mosaic are of more economic importance than the typical, or original-described, virus.

Blood (104) in 1947 reported an unusual heavy infection of yellow bean mosaic in northern Utah. All but 75 of 240 acres of Blue Lake pole beans were abandoned because of the disease.

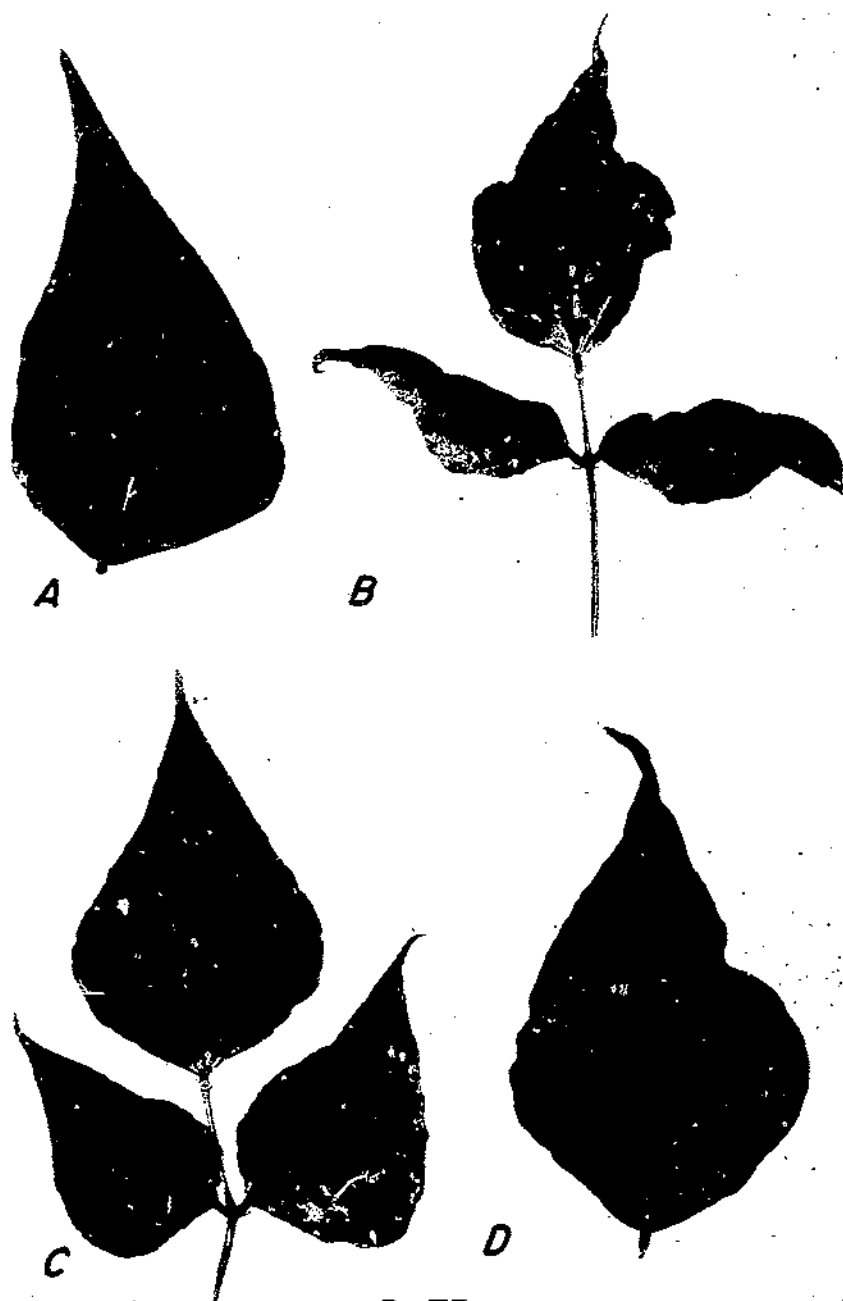


FIGURE 20.—A to D, Yellow bean mosaic, showing variations in type of symptoms on leaves of several bean varieties.

Zaumeyer and Thomas (1195) reported that most fields of Blue Lake in the same area showed 100 percent infection. Boyle (121) and later McWhorter and coworkers (641, 642) reported a severe necrotic strain of yellow bean mosaic on Blue Lake beans in Oregon that usually occurred in fields in close proximity to plantings of gladiolus, which was the source of the inoculum. Considerable damage frequently resulted in such beanfields. The name given to this strain of the virus was X-disease. The economic importance of the pod-distorting strain reported on beans by Grogan and Walker (400) from Wisconsin is not known. McWorther and Hardeson (643) reported severe injury of subclover (*Trifolium subterraneum*) grown for seed in Oregon by yellow bean mosaic virus.

### Symptoms

The first symptoms of typical yellow bean mosaic in the greenhouse appear about 1 week after inoculation and are characterized by drooping of the leaflets at the pulvini. Later small, halolike, chlorotic spots, ranging in diameter from 1 to 3 mm., develop on these leaves and on those formed later. The spots gradually enlarge and, upon coalescing, produce a more or less typical chlorosis (fig. 20, A, C). Young leaves have a tendency to become brittle, concave on their upper surface, and glossy. Infected leaves may be malformed and distorted (fig. 20, B), especially if the infection occurs in one-half of the leaflet. The mottling of contrasting yellow and green areas becomes more intense as the plants grow older (fig. 20, C, D). This intensity serves as a means of distinguishing yellow bean mosaic from common bean mosaic. Yellow bean mosaic virus causes decided dwarfing of the plant and bunchiness, the result of shortening of the internodes and multiplication of the branches. Maturity of infected plants is greatly retarded, and the production of seed is reduced.

The symptoms of the X-strain are variable. The most common symptom on the Blue Lake or similar pole varieties is purpling of the leaf bases of the lower leaves. Purpling is accompanied by an internal killing of tissues with frequent death of the plants. The lower leaves are usually yellow, and the veins of such leaves may be blackened. Frequently leaves of infected plants are malformed and have dark, irregular spots that may be accompanied by a blackening of the veins. A plant may first develop these symptoms, but later the leaves may show typical mottle. Under greenhouse conditions, local necrotic spots are formed on inoculated leaves on some varieties. A necrosis of the terminal growing point is frequently noted.

The symptoms of the pod-distorting strain of the virus are likewise variable, depending upon the variety of bean infected. In some cases the mottle is not unlike that produced by the common strain of the virus, but it is usually more severe, causing more leaf malformation and plant stunting. Some varieties show a top necrosis followed by death of the plant, while in other cases a modified type of necrosis is observed in which the tip leaves turn yellow and absciss, but the growing point is not killed. Pods from infected plants are severely malformed.

The necrotic-lesion-producing strain of yellow bean mosaic (641) develops two types of local lesions on beans, depending on the variety: (1) A distinct and somewhat circular spot, and (2) spreading veinal necrotic lesions that frequently cover a large portion of the leaf (fig. 21, B). The systemic mottle symptoms are rather similar to those produced by the type yellow bean mosaic virus, but on some varieties they are more intense (fig. 22, A). Necrosis of the trifoliate leaves is produced on a few varieties. Stem, petiole, and top necrosis, as are produced by the pod-distorting virus and X-virus on certain varieties, are seldom produced by the new virus strain. Infected pods are not malformed.

The severe yellow mosaic virus (1011) that produces local lesions on tobacco causes reddish-brown blotches on infected Blue Lake bean pods under field conditions. Veinal leaf necrosis and stem necrosis are also observed. Plants are stunted, and leaves are reduced in size, curled, and mottled. Infected pods are severely misshapen.

### Cause

Yellow bean mosaic is caused by a number of virus strains that commonly infect *Melilotus alba*, *Trifolium pratense*, *T. incarnatum*, and *Gladiolus* sp. The type strain of the virus was described by Pierce (773) and later by Zaumeyer and Wade (1202). In 1946 McWorther and Boyle (641, 642) reported a necrotic or the X-strain of the virus on Blue Lake beans in Oregon. In 1949 McWhorter (640) reported nine additional strains isolated from beans, gladiolus, alfalfa, and peas in Oregon and Washington. He stated that these strains showed considerable variation with regard to symptomology, minor host preference, and ability to develop cytological evidence of the virus in the leaf cells of *Vicia faba*. Grogan and Walker (400) reported a pod-distorting strain of the virus from Idaho Refugee and other varieties of beans. From variable symptoms noted on beans in the field, there are probably many other strains of the virus.

Bridgmon and Walker (135) isolated the type strain and the pod-distorting strain from naturally infected gladiolus. Quantz (799) isolated what appeared to be the pod-distorting strain from beans in Germany. He reported the natural hosts of yellow bean mosaics as yellow and white lupine and alsike clover. Berkeley (92) also reported the isolation of yellow bean mosaic from gladiolus. Frey (363) records the identification of the pea mosaic virus in New Zealand on gladioli that was transmissible to bean. Houston and Oswald (497) isolated two strains of the virus from Ladino clover.

Conover (225) isolated a virus from soybean that he regarded as a strain of the type virus. Hagedorn (409) and Hagedorn and Walker (410) described four isolates of yellow bean mosaic virus, of which one appeared fairly distinct from the type strain. Zaumeyer and Fisher (1187) described a new necrotic-lesion-producing strain of the virus. Besides producing local lesions on beans, the virus produced mottle symptoms more intense on many varieties than those produced by the type strain. Thomas and Zaumeyer (1011) reported on a strain of yellow bean mosaic



virus known as severe yellow mosaic, which produced local lesions on tobacco. Frandsen (349) in Germany described a strain differing from the typical yellow and pod-distorting strain, which he designated as *Marmor manifestum* n. nom. It produces necrosis on Kentucky Wonder beans and yellow mosaic symptoms on many other varieties.

McWhorter (638) demonstrated cytologically in *Vicia faba* infected with the typical bean virus 2 the presence of isometric crystals in both the cytoplasm and nuclei of infected host cells. They usually occur within the nucleoli of diseased cells, which assume cubical shapes as they become filled with the isometric crystals. They were not found in the leaf cells of healthy plants. McWhorter believes that these crystals are related to the causal agent and are a function of the virus, rather than of the host. Rich (837) showed a probable connection between nucleolar material and the development of crystalline inclusions characteristic of the virus. He later showed that in broadbeans infected with this virus the inclusions were produced just prior to the first appearance of external symptoms and that they were found only in plant parts that contained infective quantities of virus (838).

The typical bean virus 2 and the pod-distorting strain are inactivated between 58° and 60° C. in 10 minutes in plant juices. Dilution end point in fresh plant extract from infected bean is between 1 to 1000 and 1 to 2000. They resist aging in vitro at 18° for 24 hours but not for 32 hours. The properties for the X-strain have not been reported. The properties of the pod-distorting virus and the necrotic lesion producing virus are comparable with those of the type strain. Severe yellow mosaic is inactivated between 50° and 55° in 10 minutes and at 18° between 48 and 72 hours.

Grogan and Walker (399) showed that in Stringless Green Refugee and Sensation Refugee 1066 common bean mosaic effectively immunized beans against infection with the typical yellow bean mosaic and, likewise, this virus partially immunized them against common bean mosaic. They presented limited evidence, indicating that yellow bean mosaic caused a reduction in the amount of seed transmission of common bean mosaic. From this, together with their similarities of physical properties, they inferred that these viruses are closely related. Frausden's (350) findings in his cross-protection investigations did not support the hypothesis of a close relationship between his strain and the regular strain of yellow bean mosaic. He reported that the damage to the plants inflicted by a combination of the two viruses was greater than by either alone.

Zaumeyer and Fisher (1187) showed that the viruses of common bean mosaic, the New York 15 mosaic, and the type strain of yellow mosaic did not completely protect bean plants from infection by the new necrotic strain. Thomas and Zaumeyer (1011) found that no protection was obtained against severe yellow mosaic virus from the viruses of common bean mosaic and the necrotic lesion strain of yellow bean mosaic. Beemster and Van der Vant (87) found that bean virus 1 and bean virus 2 were serologically related.

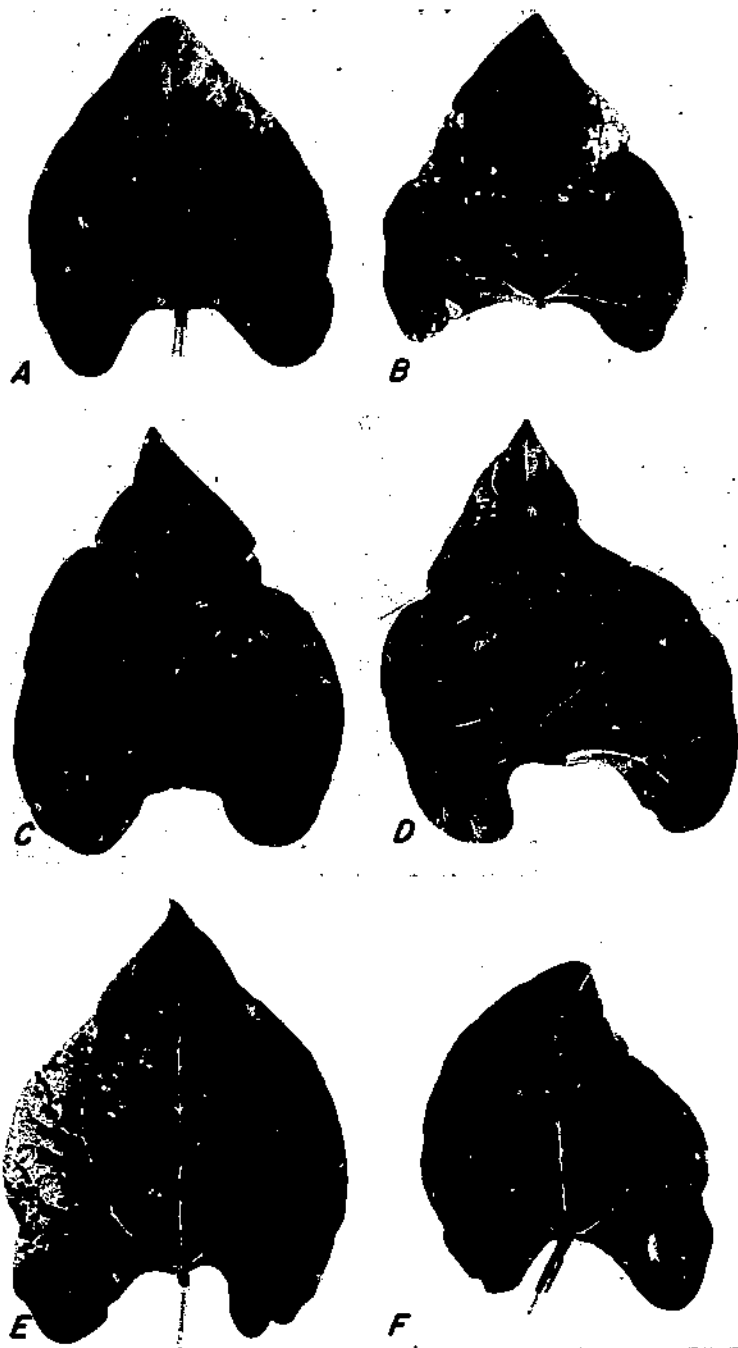


FIGURE 21.—Local lesions produced by several bean mosaic viruses on bean: *A*, Alfalfa yellow mosaic; *B*, necrotic strain of yellow bean mosaic; *C*, common alfalfa mosaic; *D*, red node; *E*, left half of leaflet, southern bean mosaic, right half of leaflet, pod mottle; *F*, bean strain of ring spot.

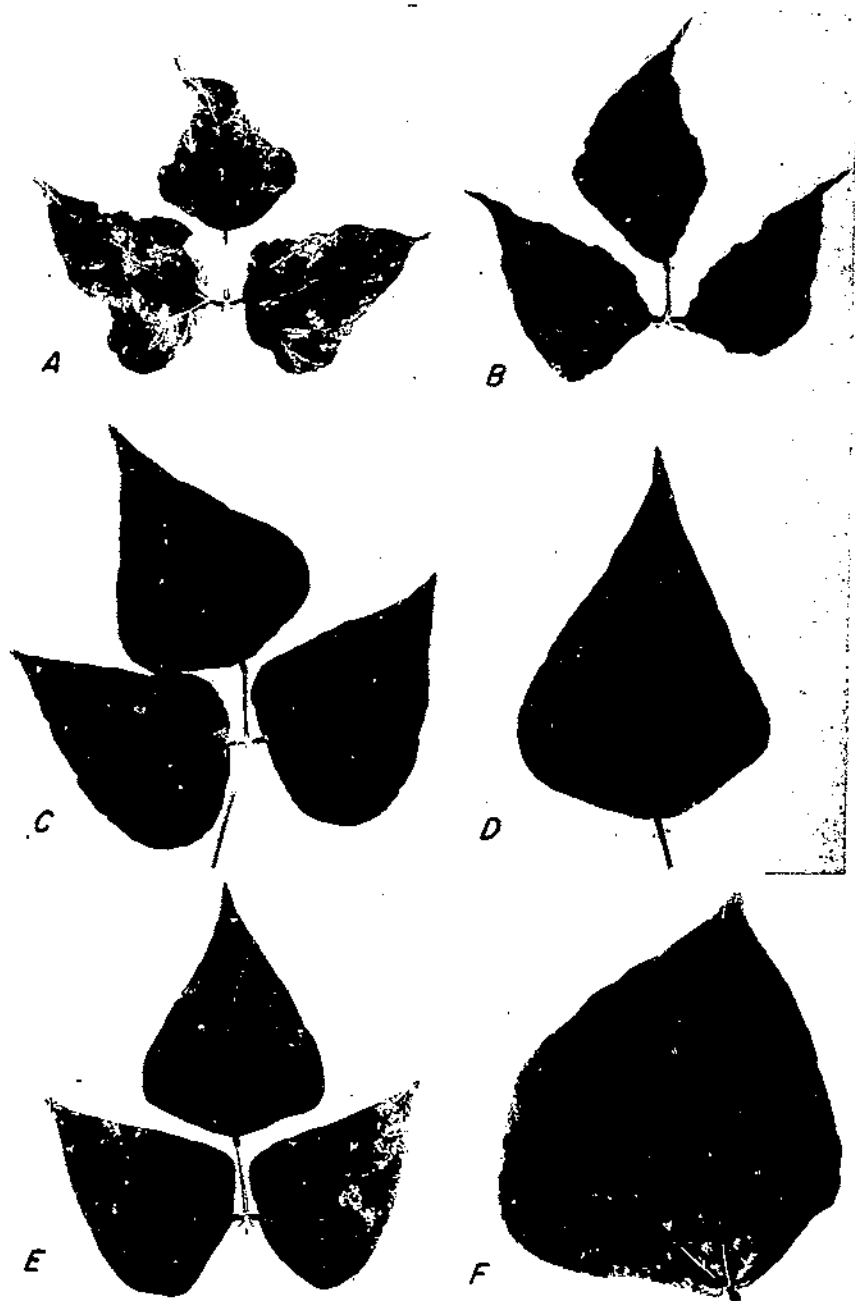


FIGURE 22.—Leaf mottling of bean caused by several viruses: *A*, Necrotic lesion strain of yellow mosaic; *B*, sweetclover ring spot; *C*, pod mottle; *D*, alfalfa yellow mosaic; *E*, yellow stipple; *F*, southern bean mosaic.

### Transmission

The yellow bean mosaic viruses like many similar viruses can be transmitted by rubbing the juice from a diseased plant on to a healthy one. Under natural conditions insects are the chief transmitting agents. Pierce (773) obtained experimentally transmission of the type virus with *Illinoia* (*Macrosiphum*) *pisii* and *Macrosiphum solanifolii*. Other aphids are probably involved.

Grogan and Walker (400) reported the green aphid *Myzus persicae* as a vector of the pod-distorting strain of the virus.

### Hosts

Pierce (773) found the host range of yellow bean mosaic virus paralleled that of the common bean mosaic virus very closely. With the exception of the Burpee Bush lima (*Phaseolus lunatus*), Henderson Bush lima (*P. lunatus*), and rice bean (*P. calcaratus*), all the hosts of common bean mosaic were found susceptible to yellow bean mosaic. According to Zaumeyer and Wade (1201), yellow bean mosaic is infectious to *P. aureus*, *Cajanus indicus* Spreng. (*C. cajan* (L.) Millsp.), *Cicer arietinum* L., *Lathyrus odoratus*, *Lens esculenta* Moench, *Melilotus alba*, *Pisum sativum*, *Trifolium pratense*, *Vicia faba*, and *V. americana* Muhl.

McWhorter (641, 642) showed that *Trifolium incarnatum*, *T. pratense*, and *Gladiolus* sp. were susceptible to the X-strain of the virus. He later showed that *Medicago sativa* was susceptible to one of the other strains he experimented with. Grogan and Walker (400) reported additional susceptibles of the typical bean virus 2. These are *T. hybridum*, *T. incarnatum*, *Vicia monantha* (L.) Desf., *V. villosa* Roth, *V. sativa* L., *V. atropurpurea* Desf., *Glycine max*, and *Trigonella foenumgraecum* L. Brierley (138) reported that the mild mosaic of gladiolus is caused by yellow bean mosaic. He showed that this virus also infected various other ornamentals such as *Freesia*, *Babiana*, *Iris*, *Sparaxis*, and *Tritonia*. Freesias are sometimes killed by the virus.

Zaumeyer and Fisher (1187) reported *Phaseolus lunatus*, *Vigna sesquipedalis*, and *V. sinensis* as susceptible to the necrotic strain but resistant to the other strains of yellow bean mosaic virus. *Pisum sativum*, *Trifolium incarnatum*, *T. pratense*, and *T. repens* were not infected by the necrotic virus strain, while certain other strains infect these species. This virus also infects *Nicotiana tabacum* and *N. rustica*. No other strain of the yellow mosaic virus except severe yellow mosaic virus has infected these hosts.

Thomas and Zaumeyer (1010) found that the host range of severe yellow mosaic was quite similar to the type strain, except that the former did not infect *Trifolium pratense*, but was infectious to *Nicotiana tabacum*, *N. rustica*, and *N. sylvestris*.

Frausden (349) reported that the strain of yellow mosaic he studied in Germany was infectious to *Phaseolus lunatus* and *Lupinus luteus* L.

Hungerford and Hillyer (503) reported *Crotalaria spectabilis* Roth, *Lupinus deusiflorus* Benth., *Medicago lupulina* L., *Trifolium hybridum* L., and *Vicia villosa* as additional hosts for the type strain of the virus.

### Control

There is no adequate control of yellow bean mosaic. It is not seedborne, and susceptible varieties might escape infection if planted in regions where white and yellow sweetclover, red clover, crimson clover, and gladiolus, the principal hosts of the virus, do not occur. However, in view of the wide distribution of certain of the above-mentioned clovers, control by completely evading them would appear to be extremely remote.

A number of snap bean varieties showed mild symptoms, indicating they might be tolerant enough to produce a crop in spite of the disease. Since the virus is infectious to most of the varieties that are resistant to common bean mosaic, it can readily be distinguished in this manner. Grogan and Walker (400) inoculated 37 varieties or strains of beans with the typical yellow bean mosaic virus and the pod-distorting virus and found that all were susceptible to the yellow bean mosaic virus, but that Great Northern U.I. Nos. 59, 81, and 123 were the only varieties that showed mild infection. These 3 varieties were the only ones that were resistant to the pod-distorting virus.

Zaumeyer and Fisher (1187) inoculated 44 varieties of beans with the necrotic lesion strain and they found all were susceptible, but that Great Northern U.I. Nos. 16, 31, and 123 were susceptible only to local lesion infection. The others were susceptible to either systemic infection alone or to both local and systemic infection. Thomas and Zaumeyer (1010) found that severe yellow mosaic virus was infectious to all varieties of beans tested except Great Northern U.I. 123.

Crumb and McWhorter (253) demonstrated that dusting beans with insecticides for the control of the vectors was unsatisfactory for economic control of yellow bean mosaic.

Rudorf (851) reported obtaining resistance to both common bean mosaic and yellow bean mosaic viruses in progeny from a cross between *Phaseolus vulgaris* and *P. coccineus*.

### Curly Top

#### Geographical Distribution and Economic Importance

Curly top, a virus disease of beans and many other cultivated and wild plants (355, 395), was first reported on beans by Carsner (194) in 1926. The disease is known to occur in California, Washington, Oregon, Idaho, Montana, Utah, and New Mexico, and in parts of Colorado, Wyoming, and Texas. It has also been reported from British Columbia (222).

The amount of loss to beans occasioned by curly top cannot be accurately measured. There is some reduction in yield every year; and, during years when the population of the insect that transmits curly top is large, the bean crop may be almost completely ruined. Although snap bean varieties are as susceptible as the dry beans, the greatest loss is caused to dry beans, which are grown more extensively.

### Symptoms

The initial symptoms of curly top of young plants remotely resemble those of common bean mosaic, but in later stages they are quite distinct. Young plants show the most pronounced symptoms on the trifoliate leaves, which become slightly puckered, curl downward, turn yellow, and die (fig. 23, A, B). These as well as the primary leaves are thicker than normal, very brittle, and readily break off the main stem. Plants affected in this way usually die in the seedling stage. Older plants seriously diseased may drop their blossoms, become chlorotic, and die. The young leaflets often curl downward and cease to grow. Affected plants are decidedly dwarfed and have short internodes, which give them a bunched appearance (fig. 23, C).



FIGURE 23.—Curly top. A, B, Plants infected when young; such plants usually die. In A, the growing point has died. C, Symptoms of curly top on plant (center) in field.

Plants infected late in the season do not always develop typical symptoms and generally grow to maturity, but any pods produced on such plants may be stunted.

The symptoms vary with the variety, the difference being somewhat correlated with susceptibility or resistance. The partially resistant varieties may produce a few pods. Such plants usually retain their normal green color for a considerable length of time. In rare cases a deeper green color, which shows a strong contrast to the partial clearing of the veins and veinlets, is apparent for a while.

### Cause

Curly top is caused by the virus *Ruga verrucosus* Cars. & Bennett and is carried in the body of the beet leafhopper (*Circulifer tenellus* (Baker)). Giddings (385, 386) reported 10 distinct strains of the virus that differed in virulence. Two strains induced severe injury in the Red Mexican bean, a variety heretofore considered resistant to curly top. Lackey (585) found in Great Northern U.I. 81, a variety tolerant to curly top, that strain 1 was inactivated or multiplication inhibited, while in Bountiful it was not. Also, that strain 4 reached a slightly higher concentration than strain 1 in the root tips of Bountiful, a susceptible variety.

### Transmission

Curly top is transmitted only by the beet leafhopper. The leafhoppers become transmitting agents of the virus by feeding on wild and cultivated plants that are affected with curly top, such as Russian-thistle (*Salsola kali* var. *tenuifolias* Zausch.), mustard (*Brassica nigra* (L.) Koch), and beet (*Beta vulgaris* L.). then transmit the disease to other plants for an indefinite period. The insects overwinter and produce their spring broods on various perennials and winter annuals as well as on other host plants. Many of these plants that are hosts for both the insect and the virus, as well as many of the insects, survive the winter. The insects and plants serve as sources of infection the following season.

The spring brood of leafhoppers may acquire the curly top virus in their breeding grounds and carry it with them into cultivated fields of beets, beans, tomatoes, and other crops to which they migrate. This migration usually takes place when the plants on which the leafhoppers have fed in the abandoned and desert areas begin to dry and mature.

The symptoms on beans appear about 10 days to 2 weeks after the viruliferous insects feed on a plant. Since the virus is transmitted only by *Circulifer tenellus*, curly top is prevalent only where that insect thrives. The severity of the disease is dependent on the size of the leafhopper population, which varies from year to year, and the percentage of viruliferous leafhoppers.

Proof of the susceptibility of beans to the curly top virus was demonstrated by Carsner (194), who found that of the seven varieties tested, Black Valentine was the most susceptible, while Great Northern was but slightly injured. The other varieties were intermediate. More recent investigations by Murphy (707) have shown the Great Northern to be highly susceptible. Although

considerable damage is caused to beans, they are not the favorite host of *Circulifer tenellus*, as indicated by the fact that the insect, when restricted to the bean as the only source of food, dies within a short time. No great amount of damage to beans is likely except in years when the insects are very abundant.

### Hosts

Carsner (192) reported 11 species of wild plants and 3 species of economic plants belonging to 11 families susceptible to curly top. He also reported (193) that the virus of curly top became attenuated when passed through certain weeds, such as *Chenopodium murale* L., *Rumex crispus* L., and *Suaeda moquini* Greene. Severin and coworkers (897) reported that 174 species of plants in 115 genera belonging to 37 families were either experimentally infected or demonstrated to be naturally infected with curly top. Fifty-three species of plants in 38 genera of 16 families were proved to be naturally infected with curly top. He later reported (896) that weeds growing on the uncultivated plains and foothills in California and in the cultivated areas experimentally infected with curly top included 57 species in 28 genera belonging to 16 families. The wild plants growing in the uncultivated areas and naturally infected with the virus included 14 species in 13 genera belonging to 8 families, while in the cultivated areas 26 species of weeds in 15 genera belonging to 9 families were found to be naturally infected with the disease in nature.

### Control

Curly top can be best controlled by growing resistant varieties. Resistance (265) was first found among field bean varieties such as Burtner Blightless, California Pink, California Red, Jenkins, and Red Mexican (267). Commonly grown dry bean varieties resistant to curly top and common bean mosaic are Red Mexican U.I. Nos. 3 and 34 (708, 709), Great Northern U.I. Nos. 16 and 31, Pinto U.I. Nos. 72, 78, and 111. Pioneer, a curly-top-resistant small white bean that can be used either as a snap or as a dry variety was released in 1944 (268). Columbia is a resistant Blue Lake variety developed for the Pacific Northwest. Idaho Bountiful and Golden Gem are bush snap beans developed by the Idaho station (273), which are resistant to curly top and common bean mosaic viruses. Among the susceptible varieties of snap beans, some that can be grown without danger of severe loss from curly top in years of medium leafhopper population, are Burpee Stringless Green Pod, Idaho Refugee, and Landreth Stringless Green Pod. Some of the varieties that are most seriously damaged by curly top are Bountiful, Plentiful, Black Valentine, and Kentucky Wonder (414).

Susceptible varieties can sometimes escape infection if planted at the proper time. It was observed in southern Idaho that if planting is done just before or at about the start of the spring migration of the beet leafhopper in the Twin Falls district the crop will nearly always escape severe injury from curly top disease (589).

The virus is not seedborne.



## Virus Diseases of Minor Importance

### Alfalfa Mosaic

The alfalfa mosaic virus, *Marmor medicaginis* Holmes var. *typicum* Black & Price, has been reported from the United States (783, 1098, 1178), Canada (91), Bulgaria (574), China (1166), and New Zealand (362). A number of strains of the virus have been reported: Alfalfa mosaic 1A and 1B (1177), alfalfa N strain (639), pepper strain (91), potato calico strain (743), tobacco strain (533), Ladino clover strain (577), tuber necrosis strain (743), yellow dot (1003), and alfalfa yellow mosaic (1185). It is not known whether the virus reported by Snyder and Rich (949) on celery or the one by Fry (362) on white clover, red clover, and lucerne in New Zealand differs from any of these strains. Calico dwarf, a strain of potato calico, was recently reported by Milbrath (676). Only two described strains have been reported as occurring naturally on beans, yellow dot and the alfalfa yellow mosaic viruses, although Thomas (1004) reported several isolates of alfalfa mosaic virus collected from mottled bean leaves in Washington and Idaho that may differ from these viruses. These were not compared in detail with one another or with the other previously described strains of the virus.

Houston and Oswald (497) reported two strains of the virus that they isolated from Ladino clover. It is not known whether they differ from any of the previously reported strains.

The type virus and some of its strains produce only local lesions on beans that range from 0.5 to 3.00 mm. in diameter, depending upon the variety of beans inoculated (fig. 21, A, C). Alfalfa N, potato calico, calico dwarf, tuber necrosis, yellow dot, and alfalfa yellow mosaic viruses produce both local and systematic infection on bean, but potato calico and tuber necrosis viruses do so only occasionally. They cause necrosis of the stem and dieback of the growing point. Yellow dot produces a mild systemic mottle, calico dwarf a bright gold mottle, and alfalfa yellow mosaic an intense mottle (fig. 22, D) and necrosis of the leaves and stems. Yellow dot and alfalfa yellow mosaic cause no terminal killing. The strains vary slightly in host response and physical properties. None of the strains are of economic importance on beans.

The synonyms (1102) of the type virus are: Alfalfa mosaic virus (1098), alfalfa ringspot virus (1102), alfalfa virus 1 (773), alfalfa virus 2 (773), Medicago virus 2 (574), alfalfa mosaic virus 1 (1178), chilli mosaic virus (574), Medicago virus 2 var. *typicum* (101), and Lucerne mosaic virus (512).

The host range includes 83 species of 28 families. Transmission is readily accomplished by inoculation of expressed juice. It is also transmitted by the pea aphid, *Macrosiphum pisi* (Kalt.) and *Myzus persicae* (Sulz.). It is not transmitted by seed.

The type strain of virus is inactivated between 60° and 65° C. Some strains are inactivated at 53° to 55°, whereas one strain is inactivated at 65° to 70°. It has a dilution end point at approximately 1 to 3000, and its longevity in vitro at 20° is 4 to 5 days. The virus was purified, and a high molecular weight nucleoprotein

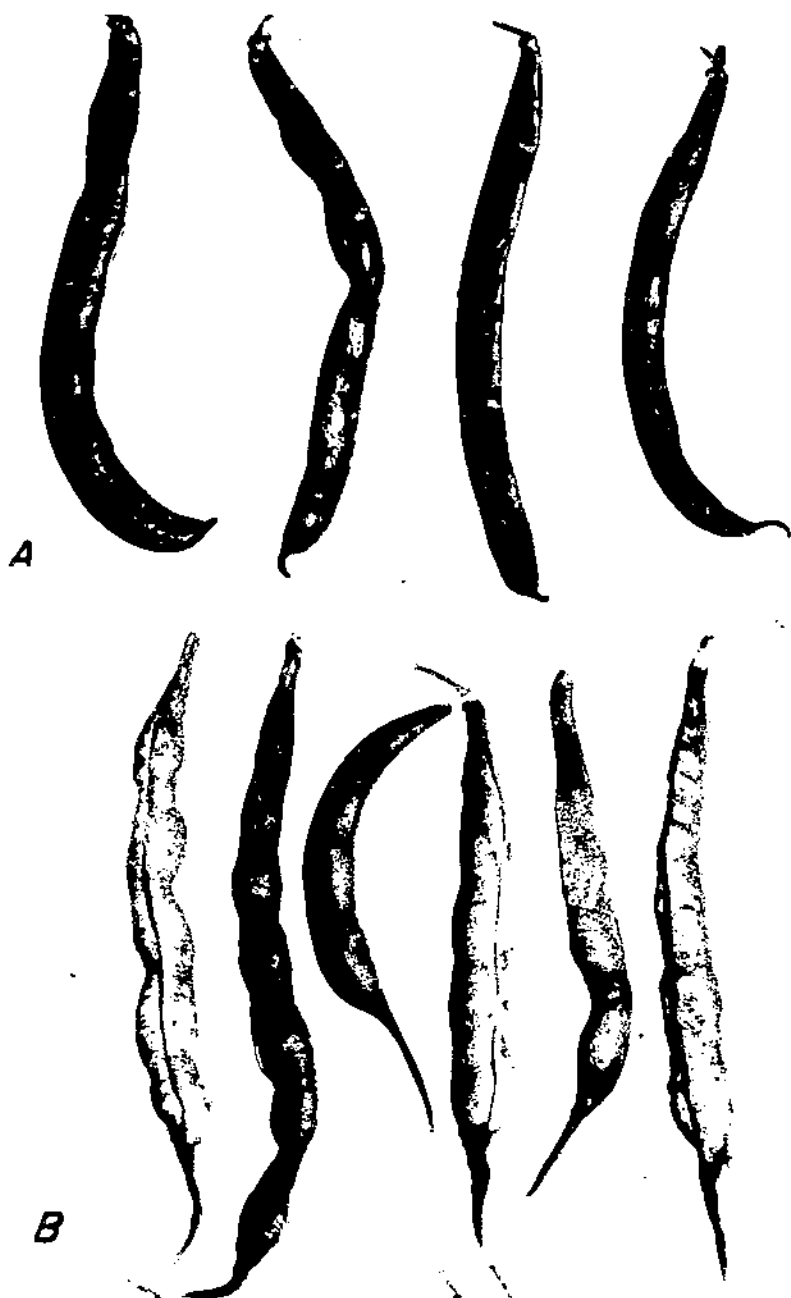


FIGURE 24.—*A*, Pod mottle virus, showing characteristic mottling and malformation of infected bean pods; *B*, pod malformation caused by a strain of yellow bean mosaic virus.

having the properties of this virus was isolated (849). The virus crystals are essentially spherical in shape and have a specific gravity of 1.48. It is the smallest plant virus that has been isolated.

### Pod Mottle

#### Geographical Distribution and Economic Importance

Not a great deal is known about the distribution or economic importance of pod mottle. The virus was isolated by Zaumeyer and Thomas (1196) in 1945 from severely mottled pods observed on plants of the Tendergreen variety grown at Charleston, S. C. In 1948 and 1949 it was isolated in combination with southern bean mosaic and yellow stipple virus from beans grown in southern Illinois. In 1950 it was isolated from pods of Black Valentine beans grown in Florida. In 1952 and 1953 it was again isolated from beans grown near Charleston, S. C.

### Symptoms

Similar to southern bean mosaic virus, pod mottle virus produces local lesions on some varieties of bean (fig. 21, *E*) and systemic mottle on others. Varieties susceptible to local infection are immune from systemic infection and those susceptible to systemic infection are immune from local infection. The local lesions appear 3 or 4 days after inoculation, are lighter brown and more diffuse than those produced by southern bean mosaic virus, and give the impression of being subepidermal (fig. 21, *E*). They average about 2 mm. in diameter, and the size of the lesions depends on the variety, the age of the inoculated leaf, and the number of lesions per unit area.

The systemic mottle symptoms of pod mottle are in general more intense than those of southern bean mosaic, and on a few varieties they are more severe than those of common bean mosaic. Infected leaves of very susceptible varieties are malformed and mottled, but they show no puckering or blistering (fig. 22, *C*). The trifoliate leaves of some varieties are both mottled and necrotic and may die. It is very difficult to differentiate the three viruses by systemic symptoms alone.

On the pods, pod mottle virus produces more marked symptoms than does southern bean mosaic virus (fig. 24, *A*). Infected pods are severely mottled, malformed, are darker green than normal, and often resemble beans that have been frozen. They are not so malformed, and so rough and warty as those produced by the pod distorting strain of yellow bean mosaic virus (fig. 24, *B*).

### Cause

Pod mottle is caused by the virus *Marmor valvularum* Zaum. & Thomas. It is inactivated between 70° and 75° C. in 10 minutes in plant juices. Its dilution end point in fresh plant extract from infected bean is between 1 to 10,000 and 1 to 100,000. The virus resists aging for 62 days at 18°, but it is inactive after 93 days.

### Transmission

Infection is more readily obtained with pod mottle virus than with common or yellow bean mosaic viruses, but local lesion production is not quite so readily obtained as with southern bean mosaic virus. The local lesions produced are usually not so numerous on the inoculated leaves as those produced by southern bean mosaic virus. Handling plants systemically infected by pod mottle virus and then healthy plants of varieties susceptible to local infection, as bean pickers handle plants in the field, produced local lesions on such susceptible plants. Since the virus is readily transmitted mechanically, it is likely that it could be spread by field laborers, by farm machinery such as cultivators, by wind-blown sand, and by other means. From limited tests, it appears that the virus is not seedborne. The insect vectors of the virus are not known.

### Hosts

The host range of pod mottle virus is very similar to that of southern bean mosaic virus. Zaumeyer and Thomas (1196) found that besides *Phaseolus vulgaris*, the only two other susceptible species in Leguminosae from 12 that were tested were *P. lunatus* and *Glycine max.* Recently the tepary bean, *P. acutifolius* var. *latifolius*, has been found to be susceptible to systemic infection. No species outside of Leguminosae have been infected.

### Control

The control of pod mottle is similar to that for southern bean mosaic. All varieties susceptible to local infection can be considered commercially resistant, since the damage caused by local infection, if found under field conditions, would be negligible.

Many bean varieties and strains have been inoculated with the virus (1196). The majority of the green-podded varieties of snap beans and most of the dry beans are susceptible to local infection. Most of the wax varieties are susceptible to systemic infection. Practically all of the varieties resistant to common bean mosaic are also resistant to systemic infection of the pod mottle virus. In general, the reaction of most of the green-podded bush varieties of snap beans to pod mottle virus is different from that of southern bean mosaic. There are notable exceptions—Full Measure, Landreth Stringless Green Pod, Longgreen, Plentiful, and Tendergreen—which are susceptible to systemic infection of both viruses.

If this virus should become a serious factor in crop production, the matter of breeding for resistance to systemic infection in susceptible varieties of snap beans would not be a difficult problem. There are a number of varieties of good commercial type that are resistant and could be used as parental material in a hybridizing program.

### Red Node

#### Geographical Distribution and Economic Importance

Red node, a virus disease, has been reported a number of times in the bean-growing areas of the Mountain States, but only in a

few instances has it been of economic importance. It was observed for the first time in 1938 in northeastern Colorado, but at that time its cause was unknown and was referred to as a physiological breakdown. A year or two later similar diseased plants were observed in western Colorado where the disease caused considerable damage in some fields of Pinto bean. In 1942 the disease was quite common in Wyoming and southern Idaho. In 1943, Virgin (1953) described the same disease in Idaho and named it "red node." His preliminary evidence indicated that it was related to yellow bean mosaic. It occurred erratically in some of the Western States in 1945 and 1946 (1195, 1201). In 1947 in Colorado, Skiles and Thomas (916) reported that the disease in some fields caused losses often amounting to 75 percent.

### Symptoms

The first noticeable symptoms of red node under field conditions are a reddish discoloration of the nodes of the stem and pulvini of the leaves and leaflets, together with reddish concentric ringed patterns on the pods (fig. 25, B). These may vary from small indistinct markings to large reddish sunken blotches that may involve most of the pod. In extreme cases there is a decided shriveling of the pod without seed formation. Discoloration of the stem, growing point, veins, and veinlets of the leaves may occur.

When the affected area on a pod is large and sunken, the pods become malformed. Often the more advanced pods develop seeds even when they become infected, but younger pods on a cluster may dry and shrivel. The infected seeds are often discolored with concentric, ringlike markings.

Under greenhouse conditions, necrotic lesions develop on the inoculated leaves (fig. 21, D). The veins become reddened and the discoloration progresses into the stem and growing point and in severe cases the young plant may die within 5 days. Plants that are not killed have reddened nodes and pulvini, reddish necrotic streaks on the stem (fig. 25, A), and are severely stunted.

### Cause

Red node is caused by the virus *Annulus orae* var. *phaseoli* Thomas & Zaum. Based on similarity of properties, symptoms on beans, jimsonweed, Turkish tobacco, and other hosts, and limited cross-protection tests, it was concluded by Thomas and Zaumeyer (1009) that the red node virus is related to the tobacco streak virus (369). The virus is inactivated between 56° and 58° C., withstands a dilution of 1 to 500 but not 1 to 1,000, and resists aging in vitro at 18° for more than 24 hours but less than 48 hours. In dried leaf tissue stored at room temperature, infection occurred after 20 days of storage. Thomas and Graham (1016) reported that the virus may remain active at least 7 months in dried or frozen bean tissue.

### Transmission

Beans are readily infected when inoculated mechanically with freshly expressed juice of the virus from beans, but not from in-

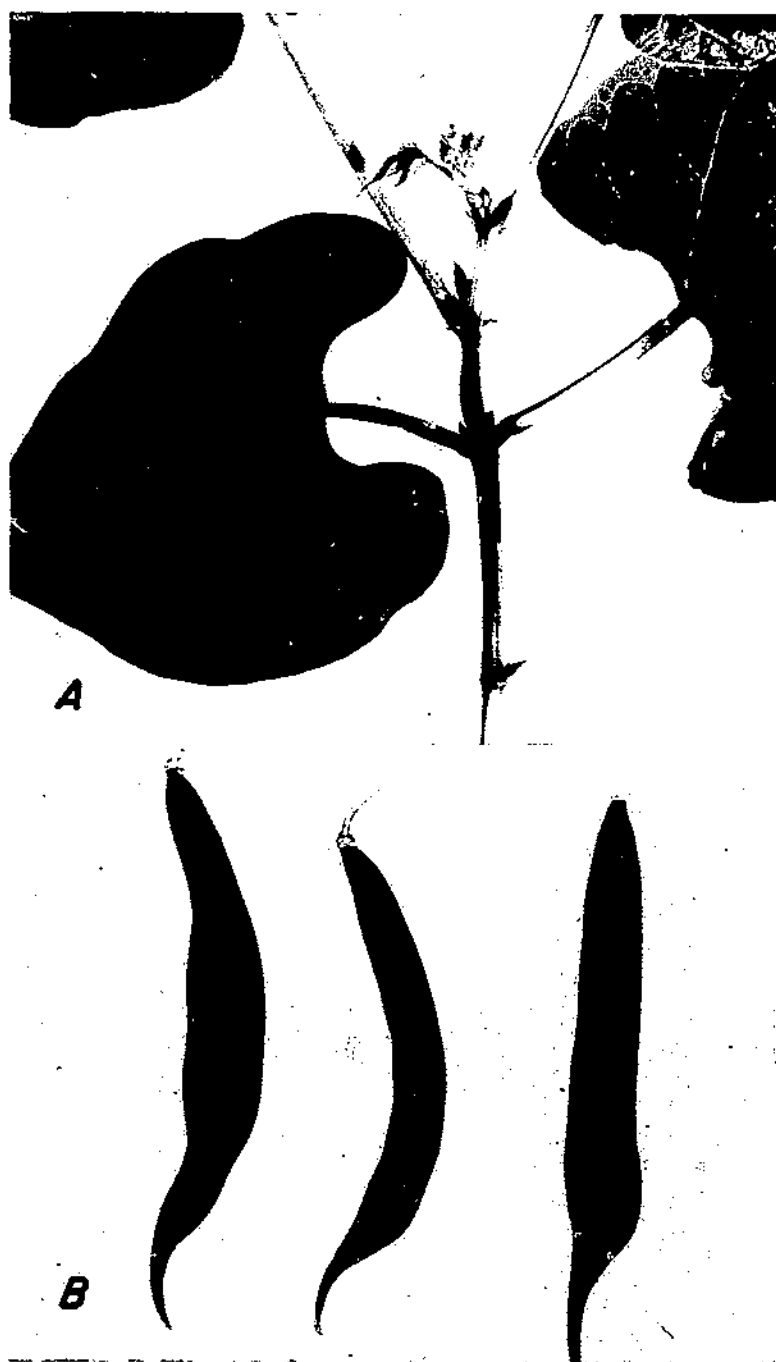


FIGURE 25.—Red node: *A*, Necrotic red streaks on stem, petiole, and necrosis of leaves and reddening of nodes; *B*, necrosis on pods.

fecting tobacco. Similarly, tobacco is readily infected with inoculum from infected tobacco but with difficulty when expressed from red-node-infected beans. Lesions on beans were produced in 2 days at 26° C., in 5 days at 14° and 18°, but no lesions appeared within 8 days at 10°. No insect vectors are known, but the frequent concentration of infection along the edges of beanfields suggests insect transmission from susceptible cultivated or wild host plants. Thomas and Zaumeyer (1009) reported no seed transmission, although Thomas and Graham (1017) showed that certain seed lots carried as much as 26 percent infection.

### Hosts

Besides beans, Thomas and Zaumeyer (1009) reported the following legumes susceptible to the virus: *Crotalaria spectabilis*, *Cyamopsis tetragonoloba* (L.) Taub., *Lupinus albus* L., *L. angustifolius* L., *Melilotus alba*, *Phaseolus aconitifolius*, *P. aureus*, *P. coccineus*, *P. mungo*, *Pisum sativum*, *Glycine max*, *Trifolium incarnatum* L., *T. pratense*, *Vicia faba*, *V. sativa*, *Vigna sesquipedalis*, and *V. sinensis*. The susceptible nonleguminous hosts were *Nicotiana tabacum* L. and *N. rustica* L. Thomas (1013) reported squash and pumpkin as also being hosts, and later he (1015) reported a large number of additional hosts.

### Control

No control measures for red node can be recommended at present, because the natural source of the virus and the means of transmission are not yet known. Since a relatively high percentage of red node has been noted in those portions of beanfields in close proximity to white or yellow sweetclover, it is recommended that these hosts be controlled if growing wild along fencerows or ditchbanks. Graham (393) reported a 75 percent reduction of infection by spraying beans at 10-day intervals with basic copper arsenate and nicotine sulfate plus Triton. No resistant bean varieties were noted among those tested. Skiles and Thomas (916) and Thomas (1014) reported Kentucky Wonder No. 780 and Kentucky Wonder Brown No. 814 resistant to the virus. This is not in agreement with the writers' results.

## Southern Bean Mosaic

### Geographical Distribution and Economic Importance

The name "southern bean mosaic virus" was employed for a mosaic disease (1190, 1191) obtained on mottled bean pods originating in Louisiana in 1941. The virus was designated as bean mosaic virus 4.

Little is known regarding the distribution of this virus under field conditions. Because the mottled leaf symptoms produced by it do not differ greatly from those produced by bean virus 1 or yellow bean mosaic, it is believed that it may have been overlooked or confused with them. By itself it does not produce symptoms as intense as those produced by the other two viruses. It was first observed as a severe mottling of the pods by market inspec-

tors in Louisiana in 1941. In 1948 serious damage resulted from this virus to snap beans in southern Illinois. It has also been reported from Tennessee and Georgia in 1952. The virus was also obtained from bean specimens collected in New York. A closely related strain of this virus was isolated from material grown in California, Colorado, Idaho, and Maryland, but as yet the disease is of no importance in these States. Bain (61) described another possible strain of this virus from Mississippi that is found very commonly in poorly drained areas of fall-planted Stringless Black Valentine beans.

### Symptoms

The virus produces local lesions (791, 1191) (fig. 21, *E*) on some varieties and systemic-mottle symptoms (fig. 22, *F*) on others, depending upon the variety inoculated. No plant thus far tested has exhibited both types of symptoms.

The necrotic lesions appear 2 or 3 days after inoculation. They are nearly circular, brownish red, and 1 to 3 mm. in diameter. Their size is determined in part at least by the variety infected, age of leaf, and the number of lesions per unit area. When the virus extract used for inoculation is highly concentrated, the lesions may be so numerous that the leaves die prematurely and drop off.

The first systemic symptoms are characterized by a mild mottle of the trifoliate leaves similar to that caused by bean virus 1, the mottling attaining its greatest intensity about the time the plants reach maturity. Vein banding is common, the interveinal tissue being lighter green than the tissues adjacent to the veins. The leaves may be puckered and blistered, typical of the symptoms of common bean virus 1. Reduction in leaf size and malformation of the leaf are noted on very susceptible varieties. On a strain of Blue Lake beans a severe necrosis of the leaves and stems and a rosetting of the young trifoliate leaves were reported (1192).

The symptoms on the pods are more pronounced than those produced by bean virus 1. They appear as irregular-shaped, dark-green, water-soaked blotches on green-podded varieties and as greenish-yellow areas on wax-podded varieties. The symptoms produced by a mixture of common bean mosaic and southern bean mosaic are more severe than are produced by the separate viruses (1186).

### Cause

Southern bean mosaic is caused by *Marmor laesiofaciens* Zaum. & Harter. Price (788, 789) crystallized the virus, and the purified virus gave positive tests for protein and negative tests for carbohydrate and was found to contain spherical particles having a mean diameter of about 33.6 mμ. They remained active for months when held at about 3° C., but soon became noninfectious when frozen. The crystals were either rhombic bipyramids joined by two pinacoids or rhombic prisms. Solutions of the prisms were highly active and produced typical symptoms of southern bean mosaic. By means of electron micrographs, Price and others (793)



showed that the elementary particles of the southern bean mosaic virus are essentially spherical bodies of about the same size as the particles of the tomato bushy stunt virus.

Price and Black (790) found that the virus is antigenic and that it is serologically distinct from tobacco necrosis virus and tomato bushy stunt virus, which it resembles in some of its physical properties. Their data showed that southern bean mosaic virus is an independent and distinct virus species. Miller and coworkers (678, 679, 680) investigated the physical and chemical properties of the virus and determined the particle size, shape, hydration, elementary composition, crystallization by dialysis, together with electrophoretic and nucleic acid investigations. Lauffer and Price (590) purified southern bean mosaic virus, obtaining a pigment not removable by centrifugal fractionation or by crystallization by means of electrolysis. Leonard and others (601) reported a close resemblance in respect to size, shape, and hydration between southern bean mosaic and tobacco necrosis virus.

The virus is inactivated between 90° and 95° C., is infectious at a dilution greater than 1 to 500,000, and resists aging in vitro at 18° for 32 weeks. It is still infectious after a 30-minute treatment with 95 percent alcohol. It is not destroyed by a 1 to 100 dilution of 37 percent formaldehyde for 30 minutes.

### Transmission

The transmission of southern bean mosaic virus is in general very similar to that of other bean viruses, although the virus is much more highly infectious than any of the other viruses that infect beans. Ordinary handling of diseased plants or touching infected plants with a hose when watering in the greenhouse can transmit the disease from diseased to healthy plants. It is believed that bean pickers and cultivating or similar equipment could spread the virus throughout a field of beans or to other fields. The virus is seedborne to a very limited extent. The insect vectors of the virus are not known, but it is assumed that it is transmitted by aphids as are common and yellow bean mosaic viruses.

Gupta and Price (405) reported that the filtrates from various fungi such as *Actinomyces* sp., *Aspergillus niger*, *Botryotinia convoluta*, *Memnoniella echinata*, *Trichothecium roseum*, and others were found effective in inhibiting infection of the virus.

Cheo (202) reported that the virus in the embryo was inactivated very rapidly when dehydration of the seed occurred, which may have been caused by certain chemical changes produced during final stages of ripening. Crude extracts from mature bean seeds caused inactivation of the virus.

### Hosts

The host range of southern bean mosaic is much more limited than it is for common or yellow bean mosaic. In addition to *Phaseolus vulgaris*, a number of varieties of *P. lunatus* and *Glycine max* were the only susceptibles found in the Leguminosae. No species outside this family has been infected.

### Control

Although all varieties tested were found to be susceptible to either the local or systemic infection by southern bean mosaic virus, those varieties that are resistant to systemic infection can be considered commercially resistant. It is unlikely that local lesions severe enough to be of any economic importance would occur under field conditions.

Most of the common bean varieties were tested with the virus. A number of the varieties resistant to common bean mosaic are susceptible to systemic infection of southern bean mosaic. Certain of the exceptions are the following: Blue Lake, Decatur, Great Northern U.I. Nos. 15, 59, and 123, Kentucky Wonder, Kentucky Wonder White, Pinto U.I. No. 78, Ranger, and Red Mexican U.I. No. 34. Great Northern U.I. Nos. 16, 31, and 81, Pinto U.I. Nos. 72 and 111, and Red Mexican U.I. No. 3 were not tested, but it is believed that their reaction would be similar to their sister strains.

Gray (396) found that leaf spray applications to beans of 125 p. p. m. of partially purified noformicin, an antibiotic produced by a species of *Nocardia*, inhibited local lesions and systemic infection caused by southern bean mosaic virus. Application of the antibiotic to the roots of Pinto beans growing in sand reduced the number of local lesions produced on the leaves by the virus. Thiouracil likewise showed antibiotic activity against the virus.

### Stipple Streak

Stipple streak of bean (80, 500, 1081), which is a strain of the tobacco necrosis virus, has not been reported in the United States but only from Holland, where it is serious on pole beans in some parts of the country. Another strain of this virus was reported for the first time occurring naturally in the United States on *Primula obconica* Hance by Price and coworkers (794). In 1950, Fulton (370) isolated several strains of the virus from the roots of guar growing in the greenhouse in Wisconsin and also from field-grown tulips. Fulton (368) reported the virus associated with strawberry plants in Arkansas, and Yarwood (1160) from lettuce in California.

Under greenhouse conditions the virus produces dark-colored local lesions 3 to 4 days after inoculation. Within a week, black streaks appear on the petioles and stems and necrotic lesions develop on or near the veins of inoculated young leaves. The infected plants are stunted, have twisted and chlorotic leaves, and usually shrivel and die.

Under field conditions scattered brown or black spots and streaks appear on stems, petioles, and veins. Often, only a part of a plant may be affected. Necrotic spots, rings, or streaks are noted on the pods.

The virus persists in the soil, and natural infection may develop in young plants within a week after planting, but the symptoms do not appear until considerably later; sometimes as late as 6 weeks after planting (1081). All varieties of beans that were

tested were susceptible. The Scarlet Runner bean (*Phaseolus multiflorus*) was resistant (1081).

Stipple streak appears to be a variant of the Rothamstead culture of the tobacco necrosis virus (80). The two viruses are serologically related, crystallize in the same manner, and have particles of similar sizes, but differ in the manner in which they infect beans. Stipple streak becomes systemic very rapidly in beans when artificially inoculated, while the Rothamstead strain only produces local lesions on the inoculated leaves.

Tobacco necrosis virus (786, 936, 937, 939) has been found to infect more than 88 species in 37 families (370, 787). About 7 species in Leguminosae were found susceptible. Whether the stipple streak strain is infectious to all of these hosts is unknown.

The thermal inactivation point of stipple streak virus lies between 85° and 90° C., and the virus has a dilution end point of about 1 to 1,000,000 (80). The virus withstands desiccation in dried tissue.

## Yellow Stipple

### Geographical Distribution and Economic Importance

Yellow stipple has only been reported once, and then in combination with southern bean mosaic and pod mottle viruses (1198). Although no bean variety has been found immune, no appreciable crop loss would result, if under field conditions this virus produces symptoms no more intense than in the greenhouse.

### Symptoms

The mottle symptoms of yellow stipple virus are considerably milder than those caused by common bean mosaic, yellow bean mosaic, southern bean mosaic, and pod mottle viruses. Infected leaves are slightly malformed and mildly mottled (fig. 22, E), not showing the severe puckering and blistering, yellow blotching, and malformation that frequently results from infection with the other viruses. The plants are not stunted, and the pods are not mottled or malformed. On dry bean varieties, such as Great Northern and Pinto, the yellow stippling is very distinctive. At first, the young trifoliate leaves show a mild mottle and, later, small yellow chlorotic spots (fig. 22, E) that may coalesce and form irregular-shaped patches as large as 10 mm. in diameter appear. These areas may involve as much as three-quarters of a leaflet. These symptoms appear identical with a condition known as yellow spot, reported by Parker (752) as a heritable-deficient Mendelian character. Infected leaves are almost normal in size and only slightly roughened or distorted. Local lesions have not been produced on any variety tested thus far.

Seedlings of some varieties infected mechanically by rubbing often show a necrosis of the veins and veinlets of the inoculated leaves. This necrosis extends into the petioles of the leaves and into the stem to the soil line, where it is frequently severe and causes a slight shrinking of the tissues.

### Cause

Yellow stipple is caused by *Marmor flavopunctum* Zaum. & Thomas. It is inactivated between 72° and 75° C. in 10 minutes in plant juices. Dilution end point in fresh plant extract from infected bean is between 1 to 50,000 and 1 to 75,000. It resists aging for 5 days in vitro at 18° and more than 80 days in desiccated leaf tissue stored at room temperature.

### Transmission

The virus is readily transmitted to beans by inoculation with expressed juice wiped on leaves dusted with carborundum. It does not appear to be seed-transmitted, and the insect vectors are not known.

### Hosts

Zaumeyer and Thomas (1197) found the following hosts susceptible: *Cyamopsis tetragonoloba*, *Phaseolus acutifolius* var. *latifolius*, *P. calcaratus*, *P. mungo*, *Pisum sativum*, *Glycine max*, and *Vigna sinensis*. No species outside Leguminosae were infected.

### Control

Thirty-six representative snap and dry bean varieties were shown to be susceptible. In most cases the mottle symptoms were mild.

### Other Viruses Reported Infectious to Beans

There are a number of other viruses that affect beans, but they have not been reported as causing any damage to the crop under field conditions or as occurring naturally on beans. The tobacco ringspot virus, as shown by Wingard (1132), Pierce (773), and Allington (30), produces local necrotic lesions on beans (fig. 21, F). LeBeau's top necrosis virus of beans is believed to be the same as soybean bud blight (600), a strain of tobacco ringspot virus. The watermelon ringspot described by Pound (784) is infectious to beans. Kote (570) described a tobacco ringspot virus from bean pods collected in Germany. Cooper (241) described a strain of ringspot virus, as shown by Wingard (1132), Pierce (773), and which produced similar symptoms on bean. Cheo and Zaumeyer (203) described a strain of tobacco ringspot isolated from mottled bean pods collected in Virginia and Delaware. Quantz (802) reported a leaf necrosis of beans caused by a ringspot virus which is closely related serologically to *Solanum deformans* virus of potatoes.

Price (785) and Silberschmidt and Kramer (914) showed that tobacco mosaic virus produces local necrotic lesions on beans. Chamberlain (198, 199) reported the production of local lesions on beans with the pea streak virus (*Pisum* virus 3). In addition, this virus produces necrosis of the stem and of the veins of the upper leaves. This is accompanied by distortion and wilting, and death of the plant may result,

White clover mosaic (1175) (white clover virus 1) causes inconspicuous blotches of light green or yellow on the trifoliate leaves of beans (774). These blotches often coalesce, forming a single spot covering as much as three-fourths of the leaf. The surface of infected leaves is sometimes uneven, but the leaves are not malformed or reduced in size. Infected plants are not stunted, and under greenhouse conditions the symptoms are often masked.

White clover mosaic infects *Trifolium repens* L., *Medicago sativa*, and *Phaseolus lunatus* (1202) in addition to the hosts susceptible to yellow bean mosaic virus.

Pea mosaic viruses 4 and 5 and alsike clover mosaic viruses 1 and 2 were shown by Zaumeyer (1179) and Zaumeyer and Wade (1203) to cause mottling on beans. In general the symptoms were similar to those produced by yellow bean mosaic and the systemic symptoms of white clover mosaic viruses.

Johnson and Jones (535) showed that the virus of severe mosaic of peas was infectious to bean. It produced yellow stipplelike symptoms on several varieties. They also showed that the pea enation mosaic virus produced a mild mottle on Corbett Refugee bean. Other investigators were unable to infect beans with the pea enation virus.

Pea virus 2 of Osborn (741) is infectious to bean and produced symptoms similar to those of yellow bean mosaic. Osborn reported a close relationship between these two viruses.

Smith and Markham (940) reported two new viruses that were infectious to beans. The first was isolated from *Arabis hirsuta* and produced systemic infection on beans. Small yellow flecks first appeared, followed by raised blisters, a bright mottle, and finally a severe necrosis, resulting in death of the growing point and young leaves. The second virus, referred to as the tobacco broken ringspot virus, may be a strain of the tobacco ringspot virus. It causes local and systemic infection in beans.

These same writers (941) reported a virus from *Ligusticum scoticum* that produced yellow chlorotic spots on the inoculated leaves of Canadian Wonder beans and systemic necrosis and death of the growing point of the plant.

Whipple and Walker (1115) reported a strain of cucumber mosaic infectious to bean. On certain varieties the symptoms resembled those produced by the yellow bean mosaic virus. Fulton (367) described six strains of cucumber virus 1, four of which infected beans systemically. In addition, one strain caused death of the inoculated leaves. Bhargava (97), working with four strains of this virus, found that beans were immune during the summer but developed local lesions in the winter. Hagedorn (408) isolated a new strain of cucumber virus 1 from peas that infected beans. Doolittle and Zaumeyer (290) described two additional strains of the virus from pepper and alfalfa that caused systemic mottling on bean.

Murakishi (706) reported a virus from Kaimi (Spanish) clover that infected *Phaseolus vulgaris* and *P. lunatus*.

Fulton (371) mechanically transmitted rose mosaic virus to bean. Warid and Plakidas (1083) isolated a virus from cowpea,

which they called Vigna virus 1 and which was found to be transmissible through bean seed.

Capoor and Varma (186) in India reported a *Dolichos* enation mosaic that causes systemic necrosis in beans, finally killing the plant. The thermal inactivation point of the virus is similar to that of southern bean mosaic. In addition, they (187) reported a yellow mosaic of *Phaseolus lunatus* that they named double bean mosaic and which was only transmitted to bean by bud grafting. Infected leaves have scattered discolored patches that gradually turn bright yellow. Affected plants are not dwarfed, but yield is reduced.

Aitken (23) described a virus from subterranean clover in Victoria, Australia, that was transmitted to 23 bean varieties. The symptoms produced are much more severe than for common bean mosaic. They are not unlike those produced by some strains of yellow bean mosaic.

Lupine virus 1 reported by Mastenbrock (655) from Holland is infectious to beans and is distinct from any previously reported legume virus.

Smith's (938) black ring disease of tomato was reported infecting beans.

Beans and lima beans with witches-broom symptoms were reported by Hoyman (499) in Arizona.

A white clover mosaic virus complex was reported by Zaumeyer and Wade (1202) to produce both local and systemic mottle symptoms on beans. The local lesions were somewhat similar to those produced by alfalfa mosaic virus on beans. The systemic mottle symptoms appear as a mild leaf blotching of light green or yellow. The mottling phase of the virus was classified as *Trifolium* virus 1 (1102).

Johnson (534) regarded *Trifolium* virus 1 as a mixture of two distinct viruses, pea mottle (*Marmor efficiens* Johns.) and pea wilt virus (*Marmor repens* Johns.). These viruses may be related to the virus of Zaumeyer and Wade (1202) that produces mottling symptoms on beans.

The sweetpea streak virus reported by Ainsworth (22) in England produces on the inoculated leaves of beans a few pale-green spots, each surrounded by a fine necrotic line. On the trifoliate leaves small yellow spots appear and the leaflets turn downward. Later, veinal chlorosis develops and death of the plant usually results within 4 to 6 weeks.

Bawden and others (79) reported a broadbean mottle virus that produced a bright interveinal mottle on Canadian Wonder bean. Local lesions may develop on beans a month after inoculation. The virus is not transmitted by seed.

Fukushi (365) transmitted a virus from red clover to bean by means of *Aphis laburnii*. The relationship of this virus to the typical red clover mosaic virus, which is not transmissible to bean, is not known.

Quantz (801) reported a new mosaic virus from broadbean that was infectious to kidney bean.

## Diseases Caused by Nematodes and Insects

### Root Knot

#### Geographical Distribution and Economic Importance

The first published account of the disease is credited to Berkeley (93), who in 1855 described and illustrated roots of affected plants and recognized the animal nature of the causal organism.

Root knot is caused by nematodes, or eelworms, of several species of the genus *Meloidogyne*,<sup>14</sup> which also attack a great number of other cultivated and wild plants. Root-knot nematodes occur in all parts of the United States, being almost universally present in the soils of the warmer parts of the country and not uncommon in the most Northern States. Root knot is widely distributed throughout the rest of the world.

Root-knot nematodes, because they attack such a large variety of crops, are the cause of very severe losses. Beans of all kinds, with the exception of a few varieties, are highly susceptible to attack, and losses that amount to 50 percent of the crop have been reported in heavily infested soil. Because of root-knot nematodes, bean growing has been discontinued on some farms.

### Symptoms

Root knot of beans may be suspected when the plants are pale and yellowish, somewhat dwarfed, and have a tendency to wilt during the warmer part of the day. The disease can be easily identified by examination of the roots. These show typical galls or enlargements, ranging from pinhead size on the smaller roots to one-half inch or more in diameter on the larger roots. These galls are enlargements of the root tissue and cannot be detached without breaking the root. This characteristic enables them to be readily distinguished from the nodules formed by the beneficial nitrogen-fixing bacteria, which are more or less loosely attached to the side of the root. In severe infection with most species of root-knot nematodes, the root system is much reduced. The roots are abnormally shortened and thickened, with a marked absence of lateral roots. However, one of the root-knot nematode species (*Meloidogyne hapla* Chitwood) produces only small galls, usually with several short roots arising above the gall.

Root-knot galls interfere seriously with the normal functioning of the roots, making it difficult for the plant to obtain water and fertilizer from the soil. This results in the yellowish color, tendency to wilt, and reduction in yield. In severe infections the roots may be a mass of galls (fig. 26, A, B), causing the plant to die prematurely.

<sup>14</sup> Until 1949, root-knot nematodes were grouped under the name *Heterodera marioni* (Cornu) Goodey or *H. radiculicola* (Gree) Muller, but it is now known that a number of species, differing in morphology and in host preferences, exist.



FIGURE 26.—Root knot, caused by several species of *Meloidogyne*: A, On lima bean; B, on snap bean.

### Control

Beans become infected by root-knot nematodes only if they are present. Control is therefore a matter of eliminating the nematodes from the soil before the beans are planted. Several methods have been recommended, but in general only two are ordinarily feasible. These are control by chemicals (soil fumigation) and crop rotation.

The two soil fumigants available at present for use on bean-fields are (1) a mixture of dichloropropene and dichloropropane and (2) ethylene dibromide, usually mixed with naphtha as a diluent. These are easily applied by injection into the soil. Their use is economically practical only where the infestation of the soil with nematodes and other soil organisms, such as wireworms, is fairly heavy and the crop increases following fumigation are of sufficient magnitude to make the investment profitable.

Root-knot nematodes can also be controlled by crop rotations with plants that are either highly resistant or immune to attack. A number of rotation crops have been recommended for this purpose in the past (1936). The discovery that there are a number of different species of root-knot nematodes differing in host habits has made these recommendations of doubtful value. At present, no general recommendations can be made for control by rotation. A study of the host habits of the local populations of the root-knot nematodes must first be made.

Isbell (515) and Barrons (71) found that beans differ in their susceptibility to root-knot nematode infection. Isbell obtained by selection two strains of beans, Alabama No. 1 and Alabama No. 2, that are highly resistant to the root knot. The nemas were found



to penetrate the root just behind the growing tip. They made little progress after infection and caused very little reduction in yield. Spartan and State, half-runner types, are also resistant to root knot.

Resistance to root knot infection was found in lima beans obtained from the Hopi Indians (625), Peru (625), and Puerto Rico (27). The resistance in the latter source was used in developing the resistant Westan, a dry, edible, baby lima. Bixby is a baby lima released by the Oklahoma station in 1952 that is tolerant to root knot (176). The resistance in some of the Hopi lima beans is being used to develop other small-seeded limas (243).

Barrons (70) developed a technique for determining the root knot resistance of beans and cowpeas in the greenhouse. Wester (1109) showed that controlled greenhouse tests were more effective than field tests for selecting resistant lima bean plants.

### Other Nematodes

In addition to the root knot organism, the following nematodes have been reported on beans or lima beans: stubby-root (*Trichodorus* spp.) (206, 207); meadow (*Pratylenchus* spp.) (530, 713, 1103); sting (*Belonolaimus gracilis*) (207); sugar beet (*Heterodera schachtii*) (812); soybean (*H. glycines*) (509); clover (*H. trifolii*) (509); *Ditylenchus* spp. (738, 798); and an unidentified *Heterodera* spp. (454). Of this group the meadow or root lesion, the stubby-root, and sting nematodes are the most important in the United States.

### Hopperburn

Hopperburn of beans (687), caused by the potato leafhopper (*Empoasca fabae*), occurs widely in the United States; but this leafhopper is a pest of importance only in the eastern half of the country, and it also occurs in some foreign countries. In the southern United States the damage caused by it is often so enormous in some years that it is very difficult to produce a crop unless the pest is controlled. Ogilvie (732) reported considerable damage to the fall crop of beans in Bermuda.

The earliest symptoms are characterized by an upward curling of the leaf, beginning at the apex and spreading along the margin (fig. 27, A) and later inward toward the base. The affected part appears parched and slowly turns deep brown. In mild cases a narrow strip along the midvein escapes damage. If the insect infestation is severe, this area as well as the midvein is injured. The destructive processes proceed more rapidly during dry weather.

Monteith and Hollowell (687) studied the effects of *Empoasca fabae* on a number of different legumes, including beans, and produced the typical tip and marginal browning as well as conspicuous reddening, bronzing, and yellowing of the leaves, petioles, and stems. There is usually a pronounced dwarfing of the plant, accompanied by a retardation in the development of floral structures and a consequent reduction in yield.

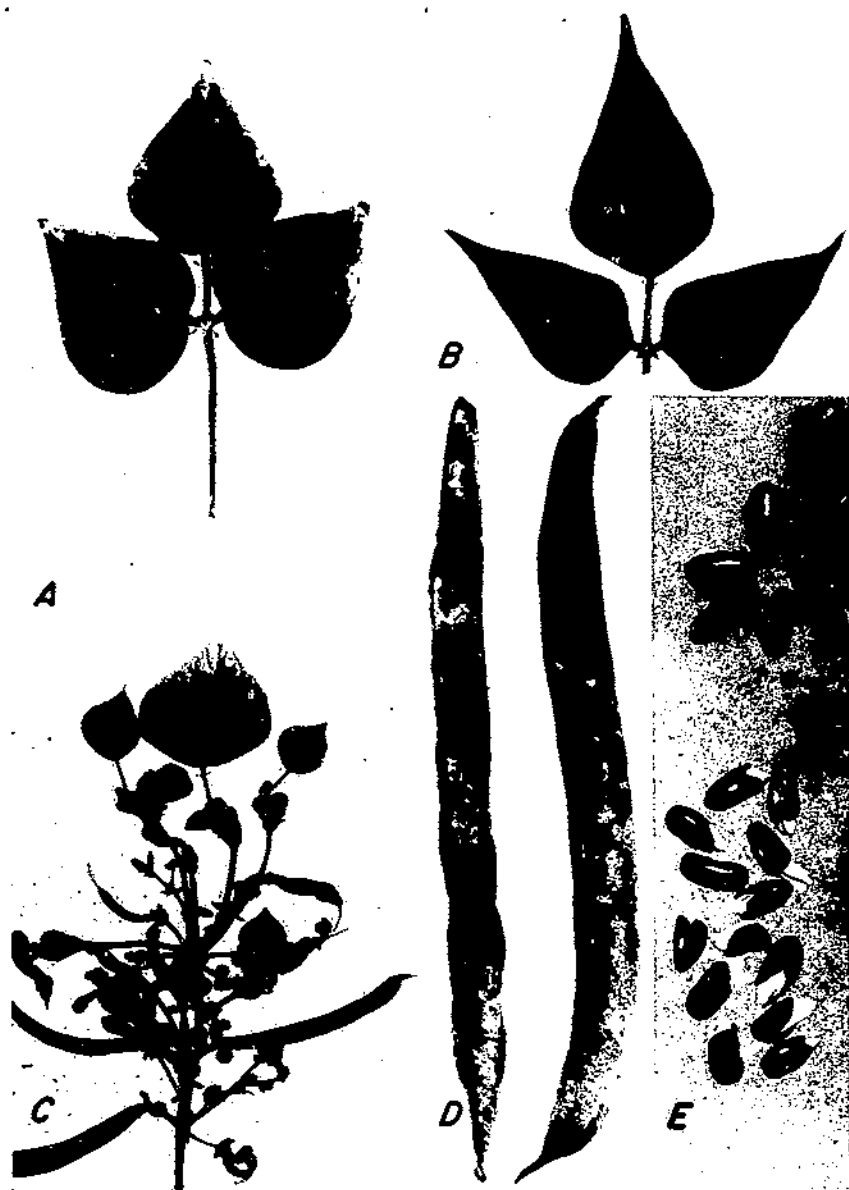


FIGURE 27.—*A*, Leafhopper injury; *B*, healthy leaf; *C*, phylloidy, caused by aster yellows virus; *D*, dark spots on the pods caused by exposure to the sun; *E*, seedcoat splitting because of uneven growth of cotyledon and seedcoat.

The writers have found considerable variation in the susceptibility of different varieties. Beyer (96) conducted a field test of 60 varieties and obtained similar results. None, however, were entirely immune; but some, especially Wells Red Kidney, showed a marked degree of resistance. The bush lima beans showed some resistance, while the pole varieties proved to be quite susceptible. Garden beans came next to pole beans in susceptibility.

The physiological principles underlying hopperburn are not positively known, but they are thought to result from some disturbance to the metabolic processes brought about by the injection of a toxin into the leaf by the insect. The toxic principle apparently remains more or less localized, and under favorable conditions plants sometimes recover.

### Control

Leafhoppers have been reported partially repelled by spraying the crop with a 4-4-50 bordeaux mixture to which a one-half pint of nicotine sulfate has been added (96). Treating the crop with a dust containing parathion, malathion, methoxychlor, or DDT also gives good control. Sprays of these compounds are effective.

### Snakehead

Snakehead is a name applied to an injury (fig. 28, A) of germinating bean seed, which according to Hawley (458) is caused by the seed-corn maggot (*Hylemya ciliatella* (Rond.)). This insect has caused serious losses to beans and occurs throughout the United States, but it is particularly a pest of beans in the East from Virginia northward and in Michigan. It also occurs in Canada. The symptoms of the injury are similar to those caused by the bacterium *Xanthomonas phaseoli* (fig. 28, B, C) and by the fungus *Thielaviopsis basicola* (161) and to baldhead (441).

The symptoms of snakehead differ from those of baldhead in that the plumule and other affected parts are made ragged by the chewing insects, whereas with baldhead the fracture or injury is clean-cut.

The larvae of the seed-corn maggot may feed on the plumule, cotyledons, and radicle underground without injuring the cotyledons. The plants usually shrivel and later die, but occasionally one may live and produce axillary buds and grow into a dwarfed plant, which may produce a few pods. If the maggot does not destroy the growing tip, a normal plant may result.

The larva may eat into the cotyledon and may consume the interior, leaving only a shell. Damage to the cotyledon alone does not usually cause much injury to the plant. If the larva enters the radicle, it burrows upward through the fleshy part of the stem. This type of injury is not always fatal, since the maggot seldom feeds on the vascular tissue.

The seed-corn maggot can be controlled by treating the seed with a wettable powder containing 75 percent of aldrin, dieldrin, or lindane. In addition, preventive measures should be taken to guard against loss. The land should be prepared in a manner to promote rapid germination and growth of the seedlings. Planting

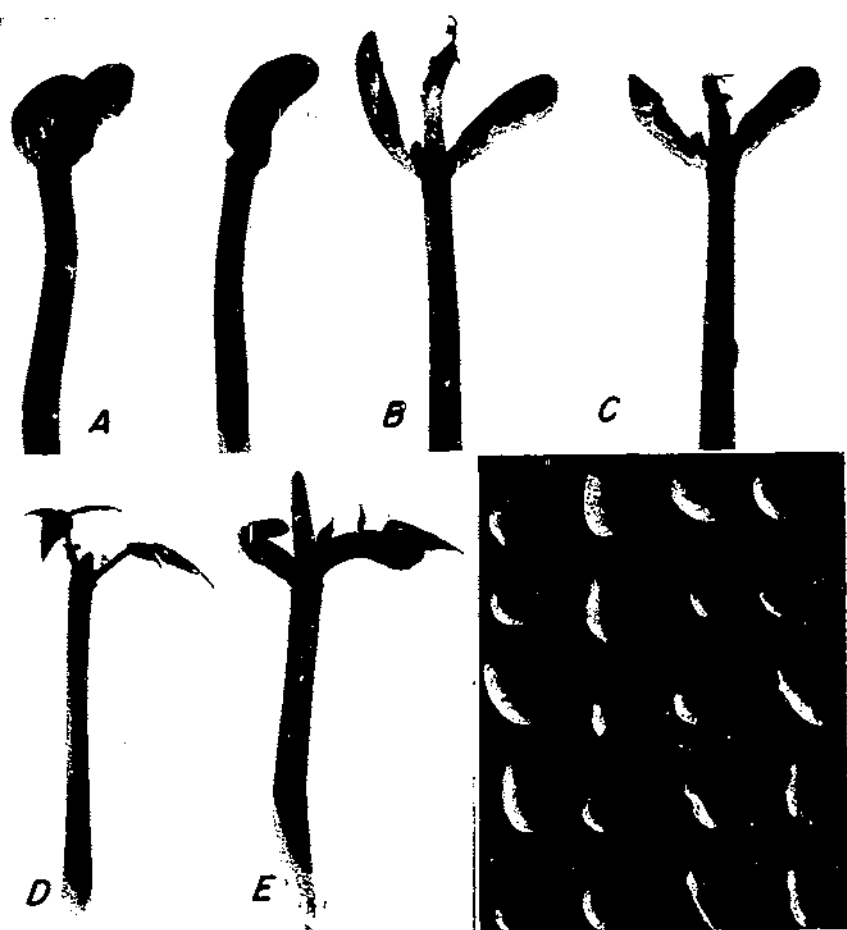


FIGURE 28.—A-C, Snakehead: A, Abnormal bean seedlings resulting from injury caused by the seed-corn maggot; B, C, another type of snakehead in which the terminal bud is injured by parasitic bacteria such as *Xanthomonas phaseoli*. D, E, Baldhead (mechanical) plants showing destruction of the terminal bud and the development of buds in the axils of the cotyledon. F, Cracking of lima bean seed caused by the thresher; such seed often develop into baldhead seedlings.

during cool, wet weather should be avoided. Shallow planting, thereby enabling the seedlings to come up quickly, has been shown to reduce injury. The application of manure immediately before planting beans should be avoided. Similarly, beans should only be planted in soil in which a green manure or cover crop has been turned under long enough to become well rotted. The use of commercial fertilizers high in organic matter should be avoided. Beans should not be planted at a time the maggots are known to be abundant in the soil.

## Nonparasitic Diseases

### Alkali Injury

The diagnosis of alkali injury to beans is not possible without some knowledge of the environmental conditions under which they were grown. The stunted growth and yellowing of the leaves are characteristic symptoms that may be caused by the lack of certain soil nutrients or by an excess of some of the same elements. The leaves of beans grown in an excess of alkali often resemble those of plants that have grown in soil flooded by water for several days. The surfaces of the stems and roots are often injured by the corrosive action of the alkali, thereby retarding or completely destroying the absorptive power of the roots.

Alkali injury occurs only in certain localities in the West where the rainfall is slight and leaching and drainage have not been sufficient to remove the alkali from the soil. It occurs in spots of various sizes in the field and more abundantly in low places. The spots are easy to locate by the effect of the alkali on the vegetation. The alkali salts are usually most concentrated at the centers of the spots and gradually become less concentrated toward the outer edges. At the center all or most of the plants may be killed, and toward the outer edge of the spot the number of living plants may increase. If the salts are abundant in the soil, a white or brown incrustation, depending on the kind of alkali present, collects on the surface; this is an aid in determining its extent and location.

There is no control for alkali injury to beans. They are quite sensitive to the action of both the white and the black alkalies and should not be planted where either is present. Some crops, as, for example, sugar beets, are more tolerant than beans. A number of native grasses, weeds, and bushes are very tolerant, and some thrive best where it is present.

### Arsenical Injury

In regions where the control of grasshoppers is attempted by spreading bran containing sodium arsenite between the bean rows, some burning occurs on pods and leaves that have been accidentally sprinkled with the poison. It causes brown, scorched, angular lesions somewhat resembling those caused by *Xanthomonas phaseoli*, except there is no water soaking of the tissue on the underside of the leaf. On the pod the scorched areas turn a reddish brown, a characteristic that is useful in differentiating arsenical injury from common bacterial blight.

Brannon (122) found that calcium arsenate-hydrated lime (1-2-50), applied to beans grown in soil of pH 7.6 and 7.0, caused severe leaf burning, leaf shedding, stunting, and reduction in yield; however, on soils of a higher acidity (pH 4.8), no injury was noted.

## Baldhead

### Economic Importance

Baldhead, a condition in beans caused principally by thresher injury of the seed in which the growing tip of the young plant is injured, was described by Harter (441). His results showed a considerable variation in varieties and that all were liable to injury. Varieties such as Full Measure, Early Stringless Refugee, Red Kidney, Improved Kidney Wax, Black Wax, and many others are often injured to a very considerable extent. Baldhead in lima beans ranged from 2 percent in the sieva types to 16 percent for the Fordhook types.

Borthwick (114) studied various types of mechanical injury in baby lima beans in California. He found that baldhead ranged from 2 to 10 percent, and other injuries from 0 to 28 percent. The total damage to a lot of baby lima beans containing 9.1 percent moisture ranged from 7.6 to 52.5 percent at threshing speeds of 770 to 1,560 feet per minute. A similar lot containing 16.4 percent moisture showed 1 and 21 percent damage, respectively, at cylinder speeds of 770 and 1,785 feet per minute. Beans rolling down a 45° incline 6.4, 10.5, and 15.0 feet were damaged 4.5, 8.6, and 13.3 percent, respectively.

Crosier (251), Drake (296), Wester (1108), Ingalls (513), and Nuttle (731) reported that baldhead beans were retarded in growth, produced smaller plants, and were inferior in yield to normal plants. Hardenburg and Eto (429) found that of the various types of snakehead (baldhead) seedlings only those involving the loss of both primary leaves were sufficient to cause a significant loss of yield.

Whitney (1118) calculated that 50 to 80 percent of the seed-coats and cotyledons of lima beans were sometimes damaged in threshing, and Goss (392) found that threshing and subsequent handling and cleaning were sources of serious loss of Fordhook lima beans in California.

### Symptoms

The external symptoms of baldhead are characterized by fissures or cracks of various sizes in the seedcoat and cotyledon (fig. 28, F). The extent of cracking varies from almost invisible fissures to bad mutilation of seed.

The internal symptoms cannot be detected until the seed germinates. These are of several types. When the epicotyl is cracked just below the plumule the latter drops off, leaving the seedling without a growing tip (fig. 28, D, E). This is what Harter (441) described as baldhead, but it is sometimes called snakehead by the growers. This is an entirely different injury from that described by Hawley (458) as being caused by the seed-corn maggot (fig. 28, A) and from the trouble attributed to *Xanthomonas phaseoli* and *Thielaviopsis basicola*.

Other types of injury may occur on the embryo. A common one is the detachment of one or both cotyledons from the hypocotyl. In some cases, however, the cotyledons are not entirely

broken off but are held in place by a few cells. In the region of the broken tissue a callus develops from which adventitious buds may arise.

Mutilation of the hypocotyl and radicle and complete loss of the radicle are not uncommon. A callus forms over the wound from which adventitious roots arise. These roots may serve the purpose of keeping the plant alive for a long time or even result in the production of a crop, although the plant's development is considerably delayed in the beginning. The severance of the radicle is usually accompanied by a decided curvature of the hypocotyl near the fracture.

A fracture of the hypocotyl at some point between the cotyledons and radicle frequently occurs. If the break extends entirely through the hypocotyl, the plant frequently dies. If, on the other hand, a strand of unbroken tissue holds the two parts of the hypocotyl together, a firm joint may be formed. Such plants can nearly always be detected by the angle and enlargement that often forms where the union takes place.

### Cause

Mechanical injury to the seed is of a twofold nature: (1) External injury or damage to the seedcoat and cotyledons; (2) internal injury, in which the epicotyl, plumule, hypocotyl, and radicle are affected. Harter (441) was the first to attribute the poor stands in snap, dry, and lima beans to mutilation (fig. 28, *F*) of the seed during threshing operations. His investigations showed that the injury consisted in a cracking of the epicotyledonary tissue, just below the plumule, so that the latter was unable to develop. These results were later supplemented by investigations of Borthwick (114), who found that the thresher may cause damage to the cotyledons, hypocotyl, and radicle of the sieva lima bean also. Bainer and Borthwick (62) showed that the amount of mechanical injury is more or less correlated with the speed of the cylinders in a threshing machine; the greater the peripheral velocity of the cylinders the greater the damage. A direct correlation was also found between the moisture content of the seed and thresher injury: the drier the seed the greater the damage. They also found that lima beans sliding down a long incline dropping some distance into a bin were very similarly injured.

The Associated Seed Growers, Inc. (53) and Toole and co-workers (1029) showed that in addition to thresher injury of bean seed that damage also occurred during processing of the seed, such as milling, hand picking, polishing, and seed treating. It was noted that injury caused by these operations is the result of additional impacts caused by each handling process.

### Control

The most important factor in preventing baldhead is proper threshing of the seed. In general, there is less injury caused by the large, special bean combines than by the small, grain combines. When a two- or three-cylinder threshing machine is used, a slow speed of the first cylinder should be employed to prevent

seed injury. The speed of the second and third cylinder might be increased if necessary without too much risk.

The most important point is the reduction of the peripheral speed of the cylinder. This actually determines the force of impact against the seed. High moisture content in the beans permits higher cylinder speeds without an increase in the total damage. Commercially, it may not always be easy to control the moisture content, but if the threshing can be done before the seed is too completely desiccated, much of the damage can be prevented. More damage to the seed occurs during light feeding of the threshing machine than during heavy feeding.

Equipment used for processing the seed after it is threshed should be so designed and operated to prevent as much as possible seed impacts that cause injury.

### Fertilizer Injury

Fertilizer injury is largely restricted to germinating seed and small plants. The greatest damage occurs when the fertilizer is drilled in the row and the seed planted at the same time or soon thereafter. If it rains between the time the fertilizer is applied and the seed is sown, thereby bringing the fertilizer into solution, the injury is considerably reduced. Experiments conducted by Townsend (1030) on Florida soils showed that kainite, which contains some sodium chloride, was more damaging to germinating bean seed than the muriate and sulfate of potash when applied in equal amounts. The results revealed that kainite at the rate of 180 pounds to the acre reduced germination to 27 percent. When equal amounts of muriate and sulfate of potash were applied, the germination was 67 to 100 percent, respectively. Injury to germinating seed and young plants is worse in light, sandy soils than in heavy soils containing a considerable amount of organic material.

### Heat Injury

In 1920 MacMillan and Byars (632) observed considerable injury caused by extreme heat on young plants in Colorado. The injury, characterized by a noticeable constriction of the stem at or just a little above the surface of the soil, was found by them to occur mostly in sandy soils. In the region where the constrictions occurred, temperatures were recorded as high as 120° to 130° F. in the warmest part of the day. Badly injured plants seldom recovered enough to produce a crop and often died.

### Internal Necrosis

Internal necrosis (740) of beans, nonparasitic in nature, is characterized by brown necrotic spots in the center of the flat inner surfaces of the cotyledons. There are no external symptoms.

Plants grown from seed of such plants produce the same condition in their progenies, indicating the possibility of a heritable condition. The seed in some pods are affected.



### Intumescence

Intumescence, which results from elongation and multiplication of the cells, is not uncommon on the leaves and pods of certain varieties of beans grown under certain conditions. It often suggests an early infection of the common and halo blights, but differs from them in that the spot is raised and not sunken. It may occur on any part of the pod, but there is a tendency for several individual spots to appear more or less localized at the end nearest the spur. The domelike elevations in intumescences are darker green than the surrounding tissue.

There appears to be a rather definite relation between intumescence on beans and soil moisture, since it often occurs after a number of days of rainy or humid weather. It is found mostly in varieties that produce abundant foliage and on pods that are more or less hidden from the direct rays of the sun and poorly aerated because of their position on the plant. If the humid weather continues long enough the distended cells often burst, causing a condition sometimes referred to as edema.

### Nutritional Disorders

In general, nutritional disorders frequently produced by a lack of some of the essential salts in the soil cause chlorosis (fig. 29) of the leaves and sometimes death of the plant. They have mostly been attributed to a deficiency of iron in the soil, but recent investigations have shown that the lack of other salts, as, for example, zinc, copper, and manganese, may also produce a chlorosis in beans. Notwithstanding the chemical differences of these salts, each may cause symptoms in certain stages of development so much like those caused by other salts that they are inseparable. Nutritional disorders are prevalent in certain localities in the Southern States, especially in sandy soils containing a low amount of organic matter.

### Copper Deficiency

Copper deficiency, according to Townsend (1930), is prevalent in the sawgrass soils of the Everglades in Florida. The symptoms caused by copper deficiency have never been adequately described. However, Townsend pointed out that when beans were planted on recently turned sawgrass soil, the plants were stunted and chlorotic; and they finally withered and died.

Production was restored in the Florida soils by applying broadcast, and then disking in, about 100 pounds of copper sulfate per acre several months before planting. If it is not possible to apply the copper sulfate in advance of planting, it may be applied with the fertilizer. Townsend (1930) found that an increased yield sometimes resulted when the plants were sprayed with a copper fungicide, even though no fungus diseases were present.

Seal (1890) tested the susceptibility of 48 bean varieties in Florida, and found that some were resistant to chlorosis caused by a copper deficiency. As a control measure he recommended the



FIGURE 29.—Nutritional disorders on leaves: A, Manganese deficiency, which causes loss of color between veins; B, zinc deficiency; C, normal leaf.

addition of copper sulfate, magnesium sulfate, and sulfur to the fertilizer.

### **Magnesium Deficiency**

An insufficient amount of magnesium in the soil causes chlorosis of the leaves and a stunting of the growth of the plants of beans. It has been a prevalent disorder in certain soils in the South, especially in South Carolina. Cooper and Moore (240) recommended the use of sulfate of potash-magnesium as a corrective. Basic slag at the rate of 400 to 1,500 pounds to the acre was also effective. Similar control measures have been recommended by Carolus and Brown (189) and by Wedgworth (1096).

### **Manganese Deficiency**

Manganese deficiency (fig. 29, A) was described by Townsend and Wedgworth (1032) as occurring in Florida, where the reaction of the soil was such as to render unavailable the manganese required by beans and other vegetables. Symptoms of manganese deficiency usually develop when the reaction of the soil is higher than pH 6.8 and are absent in soils where the reaction is pH 6.8 or lower (387).

Manganese deficiency causes retardation of growth (1030), and if it is not corrected, the leaves become golden yellow dotted with dead spots as contrasted with the normal leaf (fig. 29, C). A chlorosis develops between the small veins, giving the leaf a mottled appearance. In severely affected plants the leaves may drop, bud growth may be arrested, and root development may be greatly restricted.

Townsend (1030) found that 50 to 100 pounds of manganese sulfate per acre applied with the fertilizer will correct the disease and also that spraying the plants with a solution containing 4 pounds of manganese sulfate in 50 gallons of water is a good corrective. Two or three applications at the rate of 50 gallons per acre are often necessary if the disease is severe. In Florida, 50 to 100 pounds of sulfur to the acre applied with the fertilizer has given good results. The sulfur increases the acidity of the soil to the point where the manganese compounds are dissolved, rendering them available to the plant. In South Africa (285, 667) and England (485) spraying beans with 0.25 percent potassium permanganate or 0.25 percent manganese sulfate solution corrected deficiency symptoms (285, 667). Adding stable manure to soils deficient in manganese gives results as good as those obtained where only fertilizers are applied and the plants sprayed with manganese. Seed beans grown in manganese-deficient soil will give symptoms of this disorder even if planted in normal soil, and the seed should be soaked in a 0.25 percent manganese solution for 4 to 6 hours immediately before planting (235).

### **Molybdenum Deficiency**

In the Gosford-Wyong area of New South Wales (4, 6, 748) beans grown on dark gray, very acid, fine, sandy loam soils make

poor growth and develop symptoms locally referred to as scald. The first symptom is a collapse of the tissue between the main veins and along the edges of the leaflets. The collapsed tissue is light greenish brown, later yellowish brown. This is followed by stunting, defoliation, and failure to set pods.

An abnormally high uptake of manganese, iron, and possibly aluminum by "scalded" plants, as well as chlorosis associated with magnesium deficiency, was characteristic of the disorder. The scald and chlorosis symptoms were believed expressions of mineral depletion of soils, giving rise to excessive acidity (748). Scald was associated with seed raised and again planted in acid soils of pH 4.2 to 5. Similar seed planted in fertile soil of low acidity produced healthy plants.

Wilson (1129) found that adding sodium molybdate in amounts as low as one-quarter ounce per acre caused plant recovery. Combined with the evidence regarding the association between seed origin and scald, the responses of affected plants to molybdenum suggest that deficiency of this element is responsible for the condition, which develops only when the supply from both seed and soil is inadequate.

### Zinc Deficiency

In many of the peat soils of Florida zinc has been found to occur in insufficient amounts to produce a good crop of beans (1030). The plants may grow normally for 4 or 5 weeks in such soils, after which time growth is retarded and the leaves (fig. 29, B) become distorted, thick, and tough. At about this time yellow, brown, or even white chlorotic areas develop between the principal veins. The leaves fall prematurely.

Under these conditions, zinc deficiency can be corrected by the addition of 100 pounds of zinc sulfate to a ton of fertilizer. If the zinc sulfate is used alone, about 10 to 20 pounds should be applied to an acre. Townsend (1030) found that the best results were obtained by spraying the plants with 2 pounds of zinc sulfate in 50 gallons of water. About 50 gallons of this solution should be applied to an acre.

In California (78) zinc-deficiency symptoms developed on beans grown on an old corral site. A foliage spray of 2 pounds zinc sulfate and 1 pound of hydrated lime in 50 gallons of water per acre restored normal growth.

Beans grown on some of the newly irrigated lands in Yakima Valley and in the Columbia Basin Project of Washington make poor growth because of zinc deficiency (1051, 1052). Severely affected plants are stunted, yellow or light green, and all or parts of the lower leaves may turn brown and fall off. Flowers are scarce, and the few pods that do set do not fill normally and are conspicuously slow to mature. In milder cases of deficiency, early growth is stunted and the plants are very light green or chlorotic. But early in July the vines start to grow and appear to catch up with normal beans. Applications of 0.3 percent  $\text{ZnSO}_4 \cdot 7\text{H}_2\text{O}$  as a foliage spray at low pressures to Red Mexican bean produced a resumption of growth and restoration of the normal color of

foliage. Fifty gallons of spray per acre when the plants have about four true leaves is recommended.

Boawn and coworkers (105) found that phosphate fertilizers had no effect on the uptake of either applied zinc or native soil zinc in bean plants.

### **Boron Toxicity**

In California beans are listed among the crops sensitive to excessive concentrations of boron (314). In Holland an excess of boron causes leaf stunting, chlorosis, marginal necrotic spotting, and upward curving of the lamina (505). This condition may arise when beans follow beets, as borax is usually added to this crop in California to control heart rot.

### **Manganese Toxicity**

Löhnis (608, 609) reported that beans are highly susceptible to manganese toxicity in acid soils in the Netherlands. The leaves of affected plants have interveinal chlorosis and later spotting and curling of the leaves, a brownish-purple spotting of the petioles and stems, general stunting of the plants, and poor development of the root system. The addition of calcium carbonate corrected the condition.

### **Chlorosis Induced by Excess Water**

Zimmerley (1207) reported a chlorosis of beans after periods of heavy rainfall on soils whose pH reaction ranged from 7.0 to 7.2. During periods of drought, the apical leaves developed normally and the older, yellowed foliage returned to its normal color. Deep planting and excessive irrigation may also induce chlorosis (460). Beans are especially sensitive to excessive moisture in the soil, which may result from frequent rains and from too heavy and too frequent irrigation.

### **Phyllody**

Phyllody (fig. 27, C) on snap and lima beans is apparently induced by one or more strains of aster yellows virus. It has appeared on aster, scabiosa, delphinium, chrysanthemum, marigold, cosmos, and carrot, which are known to be hosts of the aster yellows virus. On beans, it occurs late in the growing season and is associated with late branching, renewed growth, and witches'-brooms (5, 269). A similar malformation occurs on other crops, such as alfalfa, sweetclover, carrots, and squashes. In general it causes changes of the floral parts into leaf structures and an extension of the axis between whorls of the phylloid flower where there was no close union of vascular traces that supply these adjacent whorls. In the snap bean secondary or accessory phylloid flowers, inflorescences with phylloid flowers, and deformed shoots are found (266, 269).

Dana found that a large amount of starch accumulation, which characterized the big bud of tomatoes (264) and which is a similar disease, also occurred in the pith of the bean plant and to a less extent in that of soybean. He reported metamorphosis of the carpels into leafy structures with marginal veins and that ovular leaflets arranged alternately and attached at the margin of such structures sometimes developed.

Dana (269) cleft-grafted scions from phylloid inflorescences of naturally infected bean plants on greenhouse-grown bean plants. Phyllody occurred in 9 of 10 inoculated plants, and scions from their phylloid inflorescences transmitted phyllody to 11 of 20 plants. No transmission was obtained by inserting wedges of tissue from phylloid plants into stems of 32 inoculates.

### Sand Injury

Injury to beans by blowing sand is not common, although it does occur occasionally in certain parts of the United States where strong winds prevail. The drifting sand not only causes mutilation of leaves but abrasion of pods as well (fig. 38, *D*). In 1924 Burger (151) reported serious sand injury to young plants and tender foliage in certain parts of Florida. The unsightly appearance of the pods reduces the market value of the product. Following injury to the pods a layer of cork cells, which serves to protect against invasion by fungi and bacteria, eventually forms beneath the wounded tissue.

### Seedcoat Splitting

Seedcoat splitting (fig. 27, *E*) appears to be the result of an uneven growth of the cotyledon and seedcoat. Under favorable conditions such seed when planted germinate as well as normal seed. If, however, the environmental conditions for germination are not favorable, the seed may rot. Seedcoat splitting can usually be detected early in the development of the ovule by opening the young pod. The cotyledons protrude beyond the seedcoat and somewhat resemble a cone in shape. The exposed part is pointed, roughened, and serrate. The protrusion points toward the pedicel in practically all, if not all, cases. Not every seed in a pod is involved.

Seedcoat splitting is more prevalent in some varieties than others. It is very common in the Commodore and also noted in Pencil Pod Black Wax, Brittle Wax, and Full Measure.

Results obtained by the writers in crosses between certain varieties indicate that this tendency is heritable, and many hybrid lines have been discarded because of the high percentage of split seedcoats.

Farooqui and McCollum (337) reported the highest amount of seed rupture occurred in pods from the earliest flowers that developed while the plants were in the most vigorous condition. The data indicated a relation between high yields and increased seed rupture. They also showed that the rate of development

in the testa in relation to that of cotyledons may also be a factor in rupture.

### Sunscald

Sunscald, or light injury (fig. 27, *D*), occurs everywhere in the United States and may affect leaves, stems, branches, and pods. Leaves that have about completed their growth and nearly mature pods are more apt to be affected. This condition is apparently directly connected with intense sunlight following conditions of high humidity and cloudy weather. There also appears to be some association with heavy applications of fertilizers. In fields where light injury occurs, the uppermost and outermost leaves, which are exposed to the direct rays of the sun, are mostly the ones that show the characteristic symptoms. In general the leaves that are protected from the direct rays of the sun during the warmer part of the day are least damaged.

Light injury begins on the leaf as a slight browning or bronzing of the epidermis in small patches between the veins, often at the central part of the leaf or sometimes near or at the margin. In later stages the discoloration increases in extent and results in the production of large islands of dead tissue. The tissue becomes thin and brittle and crumbles readily when dry. If the causative conditions continue long enough, a considerable amount of defoliation follows.

The first indication of sunscald on the pods is the development of tiny brown or reddish spots on that part exposed to the direct rays of the sun (629). The spots gradually enlarge and finally develop into short streaks extending from the ventral toward the dorsal sutures. In 2 days they may appear water-soaked, sometimes becoming slightly sunken and often tinged with red. Several small spots often coalesce (fig. 27, *D*), forming a larger one, that may involve the entire side of the pod exposed to the sun, whereas the other side may be practically without this discoloration. Light injury on the pods, especially in the early stages, is frequently mistaken for primary infection of bacterial blight. No varieties are known to be immune.

By using the arc lamp MacMillan (630) and the writers showed that light injury is caused by the short waves. The epidermal cells are killed, and indeterminate symptoms similar in some respects to those of mild mosaic result.

## FIELD DISEASES OF LIMA BEANS

### Fungus Diseases

#### Anthracnose

Although anthracnose, caused by *Colletotrichum lindemuthianum*, is a common and potentially a serious disease of the snap and dry beans (p. 5), it is not of the lima bean. It was reported from West Virginia, Maryland, Ohio, and Mississippi, but in no case were the losses of any consequence.

Edgerton (315) attempted inoculation experiments and obtained a slight infection on the small, white pole lima and later good infection on the Fordhook Bush lima. Edgerton and Moreland (318) noted varietal differences in susceptibility. They also observed that some isolates of the organism would attack varieties of different species but would not infect all varieties of the same species. Barrus (75) inoculated lima beans with two physiologic races of the anthracnose organism and obtained slight or no infection in most cases. On the other hand, some varieties gave fair infection and others good infection, showing variations existed in susceptibility to different races as with varieties of *Phaseolus vulgaris*. Leaves, stems, and pods of a number of the sieva and Fordhook lima beans, both dwarf and pole, have been successfully infected.

#### Downy Mildew

##### Geographical Distribution and Economic Importance

Downy mildew, caused by *Phytophthora phaseoli* Thaxt., was found by Thaxter (1001) to be widespread in Connecticut about 1889 and by Sturgis (980) in 1898, where it caused serious damage to the lima bean crop. Since its early discovery by Thaxter, it has been studied by a number of investigators and reported in Delaware (925), Maryland (214), Virginia (358), Minnesota (955), West Virginia, and Ohio (40). In addition, downy mildew is known to occur in New Jersey, Kentucky, Louisiana, Pennsylvania, Massachusetts, and Vermont. In 1941 it was reported from Colorado (453). In most of the States listed, downy mildew has been reported to be very destructive during one or more years. It has not been reported from California (948). Outside the United States downy mildew has been reported in Ceylon (768), Canada (310), Puerto Rico (478), and Russia (now U.S.S.R.) (951).

#### Symptoms

The downy mildew fungus attacks the pods (fig. 30) more readily than any other part of the host, but it may occur also on young leaves, shoots, and floral parts. The infected leaves often



show irregular-shaped spots with a somewhat purplish discoloration, especially in the region of the veins, with little evidence of mycelial growth. From an infected spot the fungus growth spreads and eventually may cover a considerable part of the pod with a dense, pure-white fungus felt (fig. 30) in irregular-shaped patches. In the arid regions of the West, the fungus felt is more appressed than it is in the more humid regions of the Atlantic



FIGURE 30.—Downy mildew, caused by *Phytophthora phaseoli*. Infected pods are covered with white mycelial growth.

seaboard, and in that respect it differs from the cottony, feltlike mycelial covering of the pod as described by Thaxter (1001). As the pods become more completely covered, they shrivel, wilt, and slowly die. The mycelium penetrates the pod and may even enter the seedcoat. During the active growth of the fungus, a reddish band, 1 to 2 cm. in width, which changes to almost black when the pod becomes dry and shriveled, separates the healthy from the diseased tissue. Other fungi, such as *Fusaria*, *Alternaria*, or *Cladosporia*, may follow the mildew and discolor the pure white of the mildew mycelium.

## Causal Organism

### Nomenclature

*Phytophthora phaseoli* was described by Thaxter in 1889. In 1925 Leonian (604) proposed to make *P. phaseoli* a variety of *P. infestans* (Mont.) DBy., so that, according to his studies, *P. phaseoli* would become *P. infestans* var. *phaseoli* Leon. In general this arrangement has not been followed.

### Morphology

Thaxter (1001) described *Phytophthora phaseoli* as follows:

Mycelial hyphae branched, rarely penetrating the cells of the host by irregular haustoria. Conidiophores slightly swollen at their point of exit through the stomata, arise singly or one to several in a cluster; simple or once dichotomously branched, and once to several times successively inflated below their apices. Conidia oval or elliptical, with truncate base and papillate apex;  $35-50\ \mu \times 20-24\ \mu$ . Germination by zoospores, usually 15 in number, or rarely by a simple hypha of germination. Oospores unknown.

De Bruyn (145) later found oospores, which were said by Du Porte (310) to carry the fungus through the winter. The conidia of Colorado material mentioned above ranged in length from  $30\ \mu$  to  $55\ \mu$  and in width from  $20\ \mu$  to  $35\ \mu$ .

Wilson (1122) stated that *Phytophthora phaseoli* differs rather markedly from the other American species of the genus in the method of branching of the conidiophore, which is very long, simple, or commonly branched at the base and bears a single apical conidium; below this are several swellings on which the majority of conidia fail to develop.

### Physiology

Hyre and Cox (507, 508) studied the effect of relative humidity and temperature on longevity of sporangia and the effect of temperature on germination of sporangia and zoospores and germ tube growth of zoospores. At  $20^{\circ}\text{C}$ ., viability was 37 percent after 27 hours at 100 and 95 percent relative humidity; less than 10 percent after 4 hours at 90; after 1 hour at 86; or after 15 minutes at 81 percent relative humidity. At  $30^{\circ}$ , the viability of sporangia was greatly reduced even at 93 to 100 percent relative humidity. After 24 hours at  $10^{\circ}$ ,  $15^{\circ}$ ,  $20^{\circ}$ ,  $25^{\circ}$ , and  $30^{\circ}$ , percentage of indirect sporangial germination was 90, 88, 20, 1, and 0, respectively; percentage of zoosporial germination was 16, 77, 87, 83, and 64; and average zoospore germ-tube growth was 62, 79, 99, 114, and 19 mm.

Maximum radial growth on oat agar occurred at  $20^{\circ}\text{C}$ .; no growth occurred at  $5^{\circ}$  and  $30^{\circ}$ . Two weeks' exposure to  $30^{\circ}$  apparently was lethal to the fungus (508). Cox (247) found that when sporangia were used to inoculate host tissue at constant temperatures no disease developed at  $10^{\circ}$ ; symptoms developed after 10 days at  $15^{\circ}$ ; symptoms appeared after 4 days at  $20^{\circ}$ ; symptoms occurred only once at  $25^{\circ}$ ; and no disease developed at  $30^{\circ}$ . Disease development occurred after  $2\frac{1}{2}$  days on sporangia-inoculated leaves when successfully incubated at  $15^{\circ}$  for 6 hours and at  $25^{\circ}$  for 18 hours.

Sakseno and Bhargava (866) found that the fungus is unable to grow on mineral salts, dextrose, and inorganic nitrogen but requires for its growth a special amino acid, d-alanine, supplemented with thiamine. The ammonium ion was toxic to the growth of the fungus.

#### Dissemination

The fungus may be spread by infected seed plant debris, wind, rain, and insects. It is unlikely that infected seed is important in disseminating the fungus, as most of the lima bean seed is produced in California where the disease is not known to occur. In Delaware the fungus overwintered on lima bean refuse (248, 249). All observations suggest that airborne sporangia are a factor in local spread but they are not important in introducing inoculum from outside areas. The percentage of infected flowers is usually small and the symptoms inconspicuous, but insects visiting diseased blossoms spread the organism to healthy pods, leaves, and petioles. The spread of the fungus and the destructiveness of the disease are increased during cloudy, rainy weather.

#### Hosts

Cox and Hyre (248) inoculated 21 crop and weed plants of the Leguminosae and Solanaceae and obtained infection only on lima bean. Leonian (604) reported isolating *Phytophthora phaseoli* from navy bean. The writers have repeatedly inoculated *Phaseolus vulgaris* with negative results.

#### Control

De Bruyn (145) recommended crop rotation to control downy mildew of lima beans, and Du Porte (310) advised burning the dead leaves and other parts of plants, since the resting spores survive the winter in them. Heuberger (481) suggested: (1) Use only western-grown seed; (2) do not spread lima bean refuse on fields to be used for lima bean production; (3) rotate crops; (4) plant only in well-drained soil; and (5) plant rows in the direction of the prevailing wind.

Spraying or dusting with copper sprays has been recommended by several workers (83, 213, 230, 259, 483, 484). Some injury may occur from the use of copper fungicides (259, 484). In Delaware, zineb and Parzate sprays and dusts were not so effective as copper compounds, but they caused no visible injury to plants (483).

Zaumeyer and Wester (1205) reported the control of the disease with the commercial streptomycin formulations, Agri-mycin 100, Agristrep, and Phytomycin under greenhouse conditions.

Spore traps have been used by Hyre (506) as an aid in forecasting the appearance of downy mildew.

Several resistant lima bean lines were found among a collection of foreign and domestic lima beans (1007). None of the resistant lines were of acceptable type, and a hybridizing program has been initiated to develop desirable resistant varieties.

## Gray Mold

Gray mold of lima beans, caused by *Botrytis cinerea* Fr., is seldom important in the field. It has been reported causing a pod rot in California and Washington (40). A severe outbreak was reported in Ventura County, Calif., in 1948 (556).

The symptoms on lima beans are similar to those on snap and dry beans. The name comes from the grayish cast on affected parts produced by abundant sporulation of the fungus under humid conditions. Lesions originate on any portion of the plant in contact with *Botrytis*-contaminated plant debris. All aboveground parts of the plant may be attacked. According to Kendrick and Middleton (556) blossoms may be directly invaded by germinating spores and may be infected at any stage of maturity. Pods may also become infected at various stages of maturity.

Proper spacing of rows to allow adequate air circulation and avoidance of excessive irrigation, especially after the foliage closes the rows, are suggested for control rather than the use of fungicides (556).

## Podblight

### Geographical Distribution and Economic Importance

Podblight, caused by *Diaporthe phaseolorum* (Cke. & Ell.) Sacc., appears to be restricted largely to the Eastern and Southern States. Outside the United States podblight has been reported in Cuba (143), Bermuda (732), and Tanganyika Territory (1075). In 1924 Doidge (287) reported a podblight of beans caused by *Phoma subcircinata* in southern Africa.

The loss caused by *Diaporthe phaseolorum* is not generally very great, although considerable damage has been reported locally (231) from several States. It is not a serious trouble on the pods until the season is well advanced, and in many cases only after the marketing of the green beans has been discontinued.

## Symptoms

Although the ascigerous and conidial stages of this fungus have been connected, the conidial stage alone is responsible for the damage of the crop. In view of this fact the symptoms are largely those caused by the imperfect stage of the fungus. During the earlier part of the season the leaves function as a host for the fungus; from them, it spreads to the pods. Large, subcircular, brown, often bordered, patches (fig. 31, A), which often attain a diameter of 1 to 3 cm., are produced on the leaves. Infection may occur on any part of the leaf, but it is largely restricted to the region bordering the midvein. The fungus spreads in all directions from the infection point but may often be delimited by the veins. In this moribund or dead tissue the pycnidia are produced. They are arranged concentrically and appear first as gray or grayish raised pimples, which later darken and become nearly black. The dead tissue finally becomes dry and falls out, leaving ragged holes.

Podblight should not be confused with a leaf spot of lima and other kinds of beans, as well as cowpea, caused by *Phyllosticta*

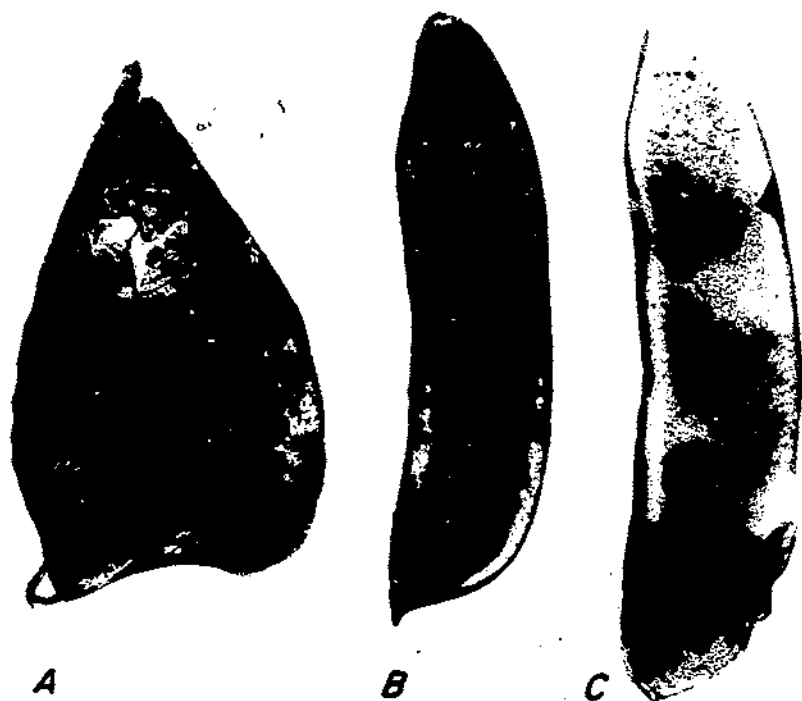


FIGURE 31.—Podblight of lima bean, caused by *Diaporthe phaseolorum*: A, Leaflet showing a large, ragged spot studded with pycnidia; B, small initial lesions on green pod; C, late pycnidial stage on green pod.

*phaseolina*; such spots are smaller and more nearly round and have smaller and fewer pycnidia.

In New Jersey, Delaware, and Maryland the disease appears on the pods in the latter part of July or August, at a time when the vines have reached almost full growth. It progresses slowly, requiring 7 to 10 days after inoculation to produce a spot 4 or 5 mm. in diameter. Infection occurs at any point on the pod (fig. 31 B, C), but more frequently at or near the ventral suture. Infection then spreads in all directions in a circular or semicircular manner. Later, numerous minute elevations, which represent the early stages in the formation of pycnidia, appear (fig. 31, C). After the death of the pod the fungus grows rapidly through the tissue, forming pycnidia over the entire surface. Fruiting bodies are rarely found on the stem until after the death of the plant.

Invasion of dead branches and pods by *Diaporthe phaseolorum* var. *sojae* and production of pycnidia has been reported by Luttrell (612).

Infected seed is dark and shriveled, and if it is attacked when immature its growth is much retarded if not entirely arrested.

The disease is much more prevalent on the pole than on the bush lima bean.

## Causal Organism

### Nomenclature

Podblight was reported by Halsted (418) in 1892 as the cause of damage to the pods and leaves of pole lima beans in New Jersey. He attributed it to a species of *Phyllosticta*.

In 1893 Ellis and Everhart (326) apparently observed the same organism on the pods of lima beans at Newfield, N. J., and described it as a new species *Phoma subcircinata*, pointing out that it differed from *P. leguminum* West. in the subcircinate arrangement of the pycnidia and the rather large binucleate spores. The name given by Ellis and Everhart was apparently accepted by Halsted (421), who in 1901 referred to the cause of podblight by that name and somewhat enlarged his earlier description. Two illustrations showed the characteristic appearance of the disease on the pods and on the leaves, both typical of the trouble as known at the present time. When Ellis and Everhart described *P. subcircinata*, they were unaware that the fungus was the pycnidial stage of *Diaporthe* (*Sphaeria*) *phaseolorum* described by Cooke and Ellis (236) on the stalks of beans a number of years previously.

The relationship between *Phoma subcircinata* and *Diaporthe phaseolorum* was established by Harter (438), who obtained a typical culture of *P. subcircinata* from a single ascospore of *D. phaseolorum*. The cultures of ascospore origin likewise produced typical symptoms of the disease.

Before the perfect, or ascigerous, stage was known, the fungus was referred to either *Phyllosticta phaseolina* or *Phoma subcircinata*. A study of the morphological structures showed a chambering of the pycnidia, formation of a sclerotial stroma, the presence of stylospores, and other characters typical of the form genus *Phomopsis* (438). According to Diedicke's (233) revision of certain closely related genera, the imperfect stage of the podblight fungus belongs to the form genus *Phomopsis*.

### Morphology

The perithecia are small ( $158\mu$  to  $355\mu$  in diameter), buried, and gregarious. The asci are clavate,  $28\mu$  to  $46\mu$  long by  $5.0\mu$  to  $8.0\mu$  wide (average  $37.4\mu$  by  $6.7\mu$ ). Ascospores are biserial, oblong, lanceolate, four-nucleate, only slightly constricted,  $10\mu$  to  $12\mu$  long by  $3\mu$  wide.

The pycnosporangia range from  $6.0\mu$  to  $8.6\mu$  long by  $2.4\mu$  to  $4.1\mu$  wide (average  $7.5\mu$  by  $3.23\mu$ ). The pycnidia range in diameter from  $158\mu$  to  $475\mu$  (average about  $245\mu$ ). Stylospores may or may not be present.

### Pathogenicity

Harter (438) obtained infection with both pycnosporangia and ascospores. Wounding of the pod was not necessary for infection. *Diaporthe phaseolorum* has been reported on snap bean (899), pepper (1034), and tomato (123, 495). Luttrell (612) inoculated snap bean, cowpea, lima bean, soybean, peanut, tomato, and pepper with pycnosporangia from cultures of *D. phaseolorum* and *D.*

*phaseolorum* var. *sojae*. *D. phaseolorum* infected only lima bean and did not produce lesions on any of the other plants and never fruited on them after they had died from other causes. On lima bean it was definitely pathogenic, entering through the non-wounded epidermis of immature pods and producing characteristic lesions. *D. phaseolorum* var. *sojae* never infected seedling plants and never produced lesions on mature plants of any host with the exception of pepper and tomato.

### Control

In 1901 Halsted (421) published some experimental results on the control of the podblight of lima beans and showed that spraying the dwarf limas with bordeaux mixture or soda-bordeaux mixture produced very favorable results. Since the fungus attacks the leaves and later the pods, spraying should be started when the plants are 1 to 2 feet high and repeated often enough to keep the foliage covered.

As the fungus is seedborne, only disease-free seed should be used for planting. However, the use of infected seed is not likely, since so far as known the disease does not occur where lima bean seed is grown commercially. Infected seed would be a source of danger only in those cases where a grower might choose to save seed for the next crop in some locality where podblight occurred.

### Root Rot

In California, root rot of lima bean may be caused by a complex group involving the fungi *Fusarium solani* f. *phaseoli*, *Pythium ultimum*, *Rhizoctonia solani*, and *Thielaviopsis basicola* (673).

Root rot is found more prevalent in light soils than in heavy soils. Kendrick (554) found less rhizoctonia root rot on lima beans planted when the mean soil temperature was 65° to 70° F. at 6-inch depth for the initial 12-week period than those planted when soil temperature was above 70°. He found that the optimum temperature for disease development for different isolates varied from 59° to 95°. This prevents any generalization as to what conditions favor rhizoctonia root rot, unless the growth behavior of the strains in a locality is known.

Increased stands of lima beans have been obtained by treating the seed with various fungicides (13, 258, 482, 596, 628, 635, 668, 1022, 1070). Arasan and Spergon are the two most commonly used products (635).

The severity of the root rot complex on lima beans was reduced by the application of fumigants and insecticides over that for lima beans grown on untreated soil (673). This reduction was attributed in part to a decrease in the wireworm population.

High tolerance to root rot, caused principally by *Rhizoctonia solani* and *Fusarium solani* f. *phaseoli*, was found in a breeding selection in California (555). This line is being used as a root-rot-tolerant parent in crosses with susceptible green processing-type lima beans.

## Scab

### Geographical Distribution and Economic Importance

The scab of lima beans, caused by *Elsinoe phaseoli* Jenkins, has never been found in the United States. Its known distribution is in Puerto Rico, Cuba, Dominican Republic, Jamaica, Guatemala, Mexico (211, 525, 527), Costa Rica, and Nicaragua (527).

It occurs on all wild or naturalized lima beans and on all cultivated (527) varieties. It is the most serious disease of lima beans in Cuba and Puerto Rico. It was known by the growers in Cuba as verruga and was at first confused with bean anthracnose, caused by *Colletotrichum lindemuthianum*, to which it bears some resemblance.

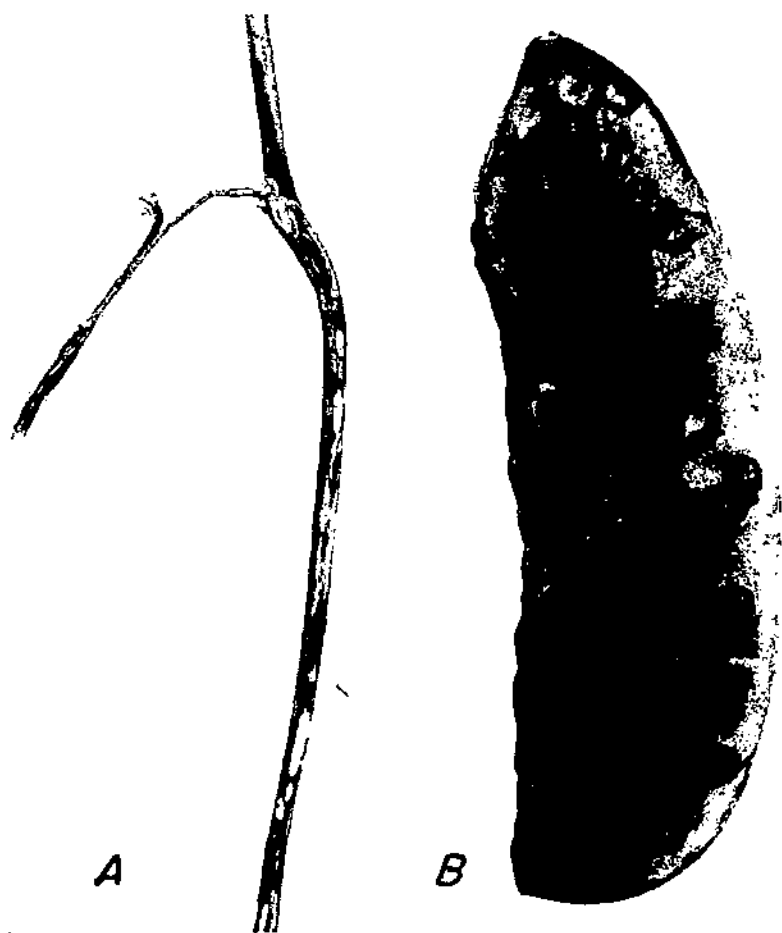


FIGURE 32.—Scab, caused by *Elsinoe phaseoli*: A, Light-colored linear lesions on stems; B, pod infection.



### Symptoms

Lima bean scab (526) occurs on the stems (fig. 32, A), leaves, and pods (fig. 32, B). The lesions, varying from a few to several hundred, are generally more conspicuous on the upper than on the lower surface of the leaf. They are usually circular in outline and buff-colored and range from minute spots to those 4 mm. or more in diameter. Those near the veins are somewhat larger and elongated in the direction of the vein. The lesions may be convex on the upper surface of the leaf and concave on the lower.

The stem cankers are raised, elliptical, or elongated spots as much as 1 cm. in length. They extend parallel to the long axis of the stem. Some malformation frequently occurs when young succulent shoots are attacked.

The symptoms on the pods, which may be badly disfigured, are more noticeable than on any other part of the plant. Individual lesions are irregular, elliptical, or subcircular in outline and range in size from mere flecks to more than 1 cm. in diameter. They may be grouped or scattered and more abundant near one suture of the pod than the other.

According to Jenkins (526) immature lesions often range in color from brick red to brown with age, becoming light drab or deep red bordered by maroon.

### Causal Organism

#### Nomenclature

In 1931 the lima bean scab fungus was tentatively identified as *Elsinoe canavaliae* Rac. by Jenkins (526), who later described it as a new species, *E. phaseoli*. Bruner and Jenkins (144) made a series of inoculations in which the parasitism of the causal organism was studied and its morphology compared with closely related species on other hosts. Their results showed that *E. phaseoli* differed from *E. canavaliae* and *E. calopogonium* Syd., two closely related species. These three fungi are similar morphologically but differ parasitically and in their geographical distribution.

The form genus *Elsinoe* is an ascomycete belonging to the Myriangiales. Its conidial stage is a *Sphaceloma*.

#### Pathogenicity

Bruner and Jenkins (144) obtained infection of young leaves of wild and several horticultural lima bean varieties, both by the cotton-plaster method and by spraying the plants and then confining them for 24 hours under bell jars. The best results were obtained on young leaves and pods. Infection on the leaves became evident in 5 to 9 days.

#### Control

No control has been worked out for scab of lima beans. Spraying at intervals with 4-4-50 bordeaux mixture has been suggested (143).

In Cuba (621) the fungus is believed to overseason on living lima bean plants, either wild or escaped plants. Any infected

plants adjacent to new plantings should be removed, as field evidence suggests that the spores are windborne (621).

### Stem Anthracnose

#### Geographical Distribution and Economic Importance

Stem anthracnose, caused by *Colletotrichum truncatum* (Schw.) Andrus & Moore, has a wide distribution, especially in the Southeastern States where it occurs on both lima and snap beans. Its known distribution includes Alabama, Georgia, Louisiana, Mississippi, North Carolina, Pennsylvania, South Carolina, Texas, Virginia, Maryland, and Iowa.

In some fields in the South a large percentage of the crop has been reported destroyed in certain years. A loss of 5 to 10 percent is not uncommon. Additional losses have been reported after the beans have reached the wholesale markets (123).

#### Symptoms

Stem anthracnose occurs on the stems, leaves, pods (fig. 33), and seed. In severe cases of infection the plant is dwarfed and the leaves show marked chlorosis. Drooping of the foliage and death of the plant are not uncommon. The initial infections usually occur along the veins on the undersides of the leaves and on the young succulent part of the stem and petioles (fig. 33, B), where a brick-red color is produced. Cox (246) reported lesions on various parts of the inflorescence, causing abscission of flowers and immature pods.



FIGURE 33.—Lima bean stem anthracnose, caused by *Colletotrichum truncatum*: A, Pods showing different stages of the disease; B, necrotic lesions on leaf and petiole.

Elongated cankers, more or less irregular, rough, and somewhat fissured, may occur on the stem, which may be partially or completely girdled. Complete stem girdling causes the death of the plant.

Initial infection of the pod (fig. 33, A) is characterized by small, reddish blotches, which may later spread over the entire surface. At this stage there are no definite lesions. Cox (246) has occasionally found sunken lesions on green pods. Later the infected areas, in which are embedded the acervuli containing the conidia and setae, become light brown or tan. Macroscopically, the acervuli resemble pycnidia. Dead pods covered with these structures closely resemble the pod symptoms produced by the imperfect stage of *Diaporthe phaseolorum*. However, a careful study of such specimens reveals the fact that there is no similarity in the fruiting structures.

The causal organism penetrates the pod and frequently develops the fruiting structures on the inside of the pod wall. The seed is often infected and may serve as one of the means of disseminating the fungus.

## Causal Organism

### Nomenclature

The stem anthracnose fungus was first collected on *Phaseolus* sp. by Schweinitz (887) in Pennsylvania in 1832 and described as *Vermicularia truncata* Schw. In 1883 Cooke (235) collected what was probably the same fungus in South Carolina and described it as *V. polytricha* Cke. What appears to be the same fungus was later collected in Texas on the Kentucky Wonder variety by Heald and Wolf (459, 460), who described it as a new species, *Colletotrichum caulicolum* Heald & Wolf.

Andrus and Moore (47) compared Schweinitz's type material with several collections of field material from various sections of the country and came to the conclusion that the different descriptions by Von Schweinitz, Cooke, and Heald and Wolf were all made of the same organism. They concluded that it was a *Colletotrichum*, and accordingly formed the combination of *C. truncatum* without examining type specimens of the organism described by Heald and Wolf. The morphology of the fungus studied by Andrus and Moore does not agree in several important details with that described by Heald and Wolf (459), so it is possible that there may be two distinct species. The symptoms differed also; this might be expected, as Heald and Wolf described symptoms on snap beans and Andrus and Moore on lima beans.

### Morphology

The acervuli are very different in appearance from those of the common bean anthracnose caused by *Colletotrichum lindemuthianum*. Macroscopically, they bear a close resemblance to pycnidia. They are black on the outer surface, somewhat rugose, and sometimes sclerotiumlike in appearance. Numerous long, thread-like, brown setae that range in length from  $60\mu$  to  $300\mu$  and from  $3.5\mu$  to  $8\mu$  in width are present. The conidia are hyaline and

curved to sickle-shaped, and range in length from  $18\mu$  to  $30\mu$  and in width from  $3\mu$  to  $4\mu$ . The fruiting structures may be found on the leaves, stems, petioles, and pods.

### Pathological Histology

Cox (246) found that on inoculated lima bean leaves spores germinate, produce appressoria from which minute infection hyphae penetrate the cuticle directly, and form primary intra- and inter-cellular hyphae in epidermal cells within 22 hours. Within 46 hours, subepidermal cells are penetrated and their contents collapsed. Symptoms become visible within 48 to 72 hours. A vascular discoloration as far as 150 mm. from the point of inoculation was evident within 5 days after injecting spore suspensions into stems and petioles with a hypodermic needle. The occurrence of discoloration at disjunctured intervals along the stem and the presence of mycelium in the vessels, according to Cox, suggest that spores were transported in the xylem when introduced in this manner.

### Hosts

Andrus and Moore (47) successfully infected 18 varieties of *Phaseolus vulgaris*, the symptoms ranging from slight scarification of the stem and leaves to severe injury of the stem accompanied by defoliation.

The writers have artificially inoculated a number of varieties of lima beans and have found considerable difference in their susceptibility. The leaves and petioles of some varieties were so badly damaged that the plant died in a few days, while others showed only a few inconsequential lesions.

Cox (246) reported that in addition to *Phaseolus vulgaris* and *P. lunatus* the following legumes were susceptible: *Medicago sativa*, *Lotus corniculatus* L., *Trifolium pratense*, *Vigna sinensis*, *Glycine max*, *Vicia sativa*, *Lathyrus hirsutus* L., *Lespedeza striata* Thunb., and *Pisum sativum*. No infection was obtained on one snap bean selection and *Trifolium incarnatum*, *T. subterranean*, *Arachis hypogea* L., and *Melilotus alba*. Persons (765) isolated *Colletotrichum truncatum* from eggplant (*Solanum integrifolium* Poir.).

### Control

Cox (246) noted the fungus lived over for at least 1 year in or on the seed. Seed grown in western United States where the disease does not occur should be planted. The fungus may overwinter on exposed lima bean refuse, and fall plowing may eliminate this source of inoculum. In North Carolina (246, 323) several fungicides were effective in controlling stem anthracnose. Weekly applications of zineb (Dithane Z-78) or dichlone (Phygon) sprays were recommended.

The writers have artificially inoculated a number of varieties of lima beans and have found that there was considerable difference in their susceptibility. Cox (247) found one lima bean breeding line to be resistant in greenhouse tests and Jackson Wonder lima bean tolerant.

## Seed Pitting

### Geographical Distribution and Economic Importance

Seed pitting, often referred to as yeast spot, seed puncture, or dimpling, was observed on common bean in 1895 in Michigan (271), in 1919 in New York (458), and Idaho (911), where it also occurred on lima bean. This injury was attributed to lygus bug feeding. A similar injury was reported in Illinois (38) on dried lima beans from California. Yeast spot, a similar type of injury associated with a yeast carried by the southern green stinkbug, was observed in lima bean and cowpea in Virginia in 1921 (1130). Yeast spot of lima bean has been reported in the United States from Alabama (1131), Illinois, Maryland, Mississippi, North Carolina, Tennessee, Virginia, and West Virginia. Elsewhere it has been reported from Bermuda (1087), Ceylon (749), and the West Indies (729, 730).

Losses from seed pitting of 90 to 100 percent have been reported (1131), but in general they do not exceed 10 to 20 percent. In California (65) losses from lygus feeding also resulted from: (1) Shedding of blossoms and young pods; (2) discarding beans having conspicuous pits or lowering the grade in fresh and frozen lima beans, particularly the latter, if such seeds were not removed; (3) cost of hand removal of pitted seeds from dry beans and from fresh limas to be used for quick-freezing; (4) occasional withering of the affected bean in an otherwise normal pod.

### Symptoms

Seed pitting, as described by Wingard (1130), occurs on the seed in the pod. It causes numerous dark sunken areas on the cotyledons. The organism is apparently able to attack the seed at any time during development, but the most severe injury seems to result when infection takes place before the seed is half grown. In case of early infection the seed may either die prematurely or fail to grow to normal size. In the majority of cases the testa remains unbroken, the infected spot being dark brown and somewhat sunken and wrinkled (fig. 34). This, however, is not always true, since in some cases the testa is ruptured, the presence of the disease being evident from the craterlike lesions on the cotyledons. The lesions are grayish brown and granular, and masses of asci and a small number of vegetative cells occur in them. Pods that appear healthy may contain badly diseased seed.

Similar symptoms on lima bean seed produced by lygus bug feeding have been described (65, 911). However, in California the yeast *Nematospora coryli* Pegl. was not found associated with the damaged seeds. If lygus bug feeding took place on blossoms or young pods the blossoms and pods were quickly shed. At the time of puncture of the pod a small hole would be visible from which sap oozes, and internally the tissue surrounding the puncture would soon turn brown (911). Older pods have smooth, firm intumescences up to 10 mm. across and 2 mm. high on the inside surfaces. These intumescences frequently cause distortion by pressure against the developing seeds and usually are in contact with

pitted areas of a seed. It is not possible to detect accurately either internal pod intumescences or seed pitting by external appearance of the pod, except by the infrequent incidence of slight swellings at the point of feeding.



FIGURE 34.—Seed pitting of lima bean seed, caused by lygus bug feeding. Yeast spot, a similar or identical type of injury, is associated with a yeast carried by the southern green stinkbug.

### Causal Organism

#### Nomenclature

Seed pitting of lima beans is reported to be caused by the yeast *Nematospora coryli* Pegl. (*N. phaseoli* Wingard), which enters through punctures made by the southern green stinkbug (*Nezara viridula* (L.)) and probably other insects.

In California and Idaho seed pitting results from activity of two lygus bugs (*Lygus hesperus* Kngt. and *L. elisus* Van Duzee) and possibly other insects. The toxin secreted by these insects apparently causes the death of a considerable number of cells around the puncture.

It is possible (65) that the pitting attributed to yeast in certain areas may involve also an effect of insect toxins.

### Morphology and Physiology

The cells of the yeast organism show a wide variation in form. The elliptical and spherical ones predominate in young cultures. Myceliumlike strands and cells shaped like tennis rackets and walking sticks are not uncommon in 24-hour-old cultures.

In young cultures one to five buds arise from the mother cells. In some cases the mother cells send out a group of bud cells from one end and myceliumlike strands from the other. The strands are septate and in most cases form buds at the cross walls.

Elliptical cells in young cultures range from  $8\mu$  to  $14\mu$  long and from  $6\mu$  to  $10\mu$  wide. The mature spherical cells are about  $20\mu$  in diameter, and the myceliumlike strands range in length from  $90\mu$  to  $140\mu$  and in width from  $2.5\mu$  to  $3.5\mu$ .

The methods of spore germination have been observed. In one case, which is the most common, the basal cell swells at the transverse septum, forming a sphere about  $6\mu$  in diameter; from this a germ tube protrudes and grows into a myceliumlike strand of considerable length, with cross walls and branches that give it the appearance of a true mycelium. In the other case, the sphere is formed as just described but instead of its sending out a septate strand a spherical cell buds from it.

The minimum and maximum temperatures for the growth of *Nematospora coryli* on media are about  $15^{\circ}$  and  $40^{\circ}$  C., respectively, and for successful infection about  $25^{\circ}$  to  $30^{\circ}$ . The minimum temperature for ascospore formation is about  $18^{\circ}$ , the optimum  $25^{\circ}$  to  $30^{\circ}$ , and maximum  $34^{\circ}$ .

### Pathogenicity

Infection is apparently restricted entirely to the fruiting structures of the host; this restriction suggests that infection is dependent upon the presence of an abundant supply of available carbohydrates, such as is found in the cotyledons of certain seeds.

Spraying the young lima bean pods (1130) with a water suspension of a pure culture of the organism as well as smearing them with a pure culture failed to produce infection. The seed was readily infected when the pods were punctured with a needle dipped into a pure culture of the yeast. The symptoms were evident within 2 or 3 days after inoculation. They were very conspicuous within 7 to 10 days. Infection rarely occurred on snap beans, but it may occur on tomatoes.

The only tests that have been reported (1131) on transmission of the yeast to lima bean by the southern green stinkbug were made by transferring the insects from previously infected bean plants, and no results with uncontaminated bugs have been reported. Baker and others (65) suggested that the possibility exists that a toxin introduced by the bugs might have caused damage, sometimes accompanied by yeast infection.

### Dissemination

According to Wingard (1131) natural infection is dependent on the puncture made by the green bug (*Nezara hiliaris* (L.)), more properly called southern green stinkbug (*N. viridula* (L.)),

and probably by other insects. This insect is always present wherever infection occurs, and lesions always develop on the cotyledons around its punctures. The severity of infection and distribution depend directly upon the number of insects present. Leach and Chulo (594) failed to find yeast spot in association with stinkbugs on lima beans in West Virginia. When insects were taken from yeast-spot-infected plants they failed to find *Nematospora* within the body of the insects, indicating that transmission may be entirely external and mechanical. Underhill (1038) found that lima beans were not the preferred host of the stinkbug and that they usually migrated to beans after feeding for some time on certain preferred noncultivated plants.

### Hosts

The results of different investigators indicate that there are numerous hosts for *Nematospora coryli*. Wingard (1131) obtained positive infection of *Phaseolus lunatus*, *P. vulgaris*, *Ipomoea batatas*, and *Vigna sinensis*. Other hosts are cotton (729, 975), soybean (588), mung bean, *Bauhinia golpini* (688), birdseye bean (1130), *Vigna catjang*, *V. unguiculata*, *Dolichos lablab*, *Cannavallia gladiata*, *Crotalaria juncea*, *C. retusa*, and *Tephrosia*. *Indigofera*, and *Cassia* spp. (730), citrus, pomegranate (338), tomatoes (880), and pecan (1092).

### Control

No control measures for the yeast-spot type of seed pitting have been developed. The large Fordhook lima bean was reported to be less susceptible than the small sieva type (1131).

In California and Idaho, seed pitting that results from lygus bug feeding and possibly other insects has been controlled by the use of 5 percent DDT (65, 670). Field experience in California suggests that the growers of lima beans should carefully avoid planting limas next to perennial plants favored by these insects, such as seed beets and alfalfa.

## Other Field Diseases

### Bacterial Spot

#### Geographical Distribution and Economic Importance

Bacterial spot of lima beans, caused by *Pseudomonas syringae*, may occur wherever lima beans are grown in the United States, with the possible exception of the Pacific coast and certain intermountain regions where it has never been reported so far as the writers know. Little is known about its occurrence in foreign countries. Gardner and Kendrick (379) stated that it may possibly occur in Japan, Cook (233) reported it from Puerto Rico, and Wilson (1125) reported it from New South Wales.

The heaviest losses, which may range from 5 to 20 percent of the crop and are the result of defoliation and of damage to the pods, occur along the Atlantic coast and in the Southern States.



### Symptoms

Bacterial spot (fig. 35) occurs on the leaves, stems, and pods. The leaves are usually infected first, and from them the organism spreads to the stem and pods. The lesions appear first on the upper surface of the leaf as small spots, which soon develop reddish-brown borders. As the spots enlarge the centers turn light gray, become dry and papery, and later fall out (fig. 35, A). Two or more spots often coalesce, forming one large lesion of irregular shape and outline. Single lesions range from 1 to 3 mm. in diameter. The leaves sometimes become distorted or malformed, but this malformation is not a constant characteristic of the disease.

On the leaves the bacterial spot resembles in many respects the symptoms of common blight, caused by *Xanthomonas phaseoli*, and of halo blight, caused by *Pseudomonas medicaginis* var. *phaseolicola*. There are, however a few macroscopic differences that assist in distinguishing them. In the early stages of infection, the common and halo blights cause water-soaked lesions on the leaves. Such lesions never result from infection by the bacterial spot organism. The lesions are more regular in outline and generally somewhat smaller than those of common and halo blights. The reddish-brown color surrounding the lesion is very characteristic of bacterial spot. On the lower surface the spots are depressed and their margins are lighter than those on the upper surface; these symptoms are not characteristic of the common and halo blights.

Reddish-brown lesions and streaks may occur on the stems and petioles. On the pods (fig. 35, B) they are more nearly circular in outline except when they are on or adjacent to the sutures, in which case they may be largely confined to and extend along them for some distance. A cream-color exudate frequently occurs on the pods, stems, and petioles. Early infection of the peduncle is generally followed by the dropping of the blossoms. Pod lesions are brown at first and surrounded by a water-soaked halo. They are never as large as those produced by the common blight organism (fig. 35, C).

### Causal Organism

*Pseudomonas syringae* was first observed in 1892 by Beach (82), who noted that it caused symptoms on snap beans that differed from those caused by *Xanthomonas phaseoli*. He successfully infected lima beans but not snap beans. His description of the symptoms indicated that the causal organism was the same as *P. syringae* described some years later as the cause of a similar disease of cowpeas. The same disease was reported (981) to occur in Connecticut in 1897. In 1921 Tisdale and Williamson (1025) announced that a bacterial disease of lima beans had been under their observation since 1917. In 1923 they (1026) published an account of the disease as it occurred on the lima bean and described the causal organism as a new species, *Bacterium viridifaciens* Tisdale and Williamson. Gardner and Kendrick (378), who were investigating a bacterial disease of cowpea at the same time, described the causal organism as a new species, *Bact. vignae*

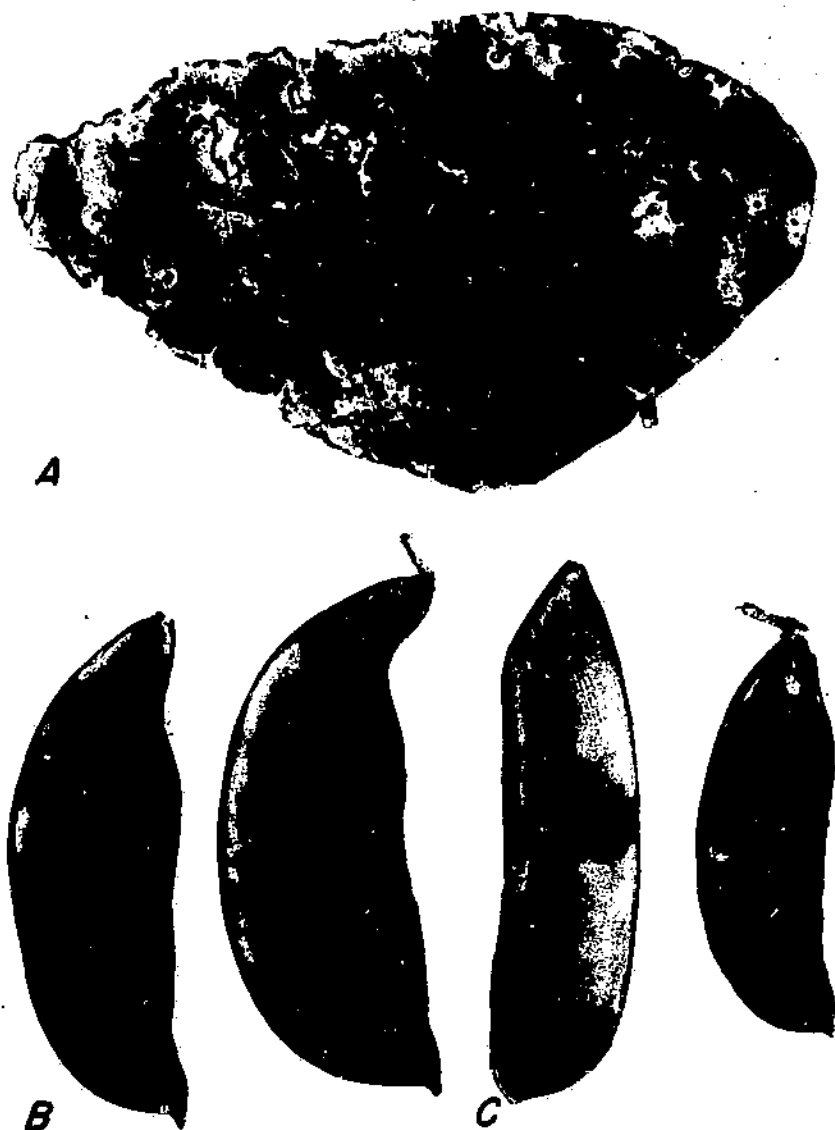


FIGURE 35.—Bacterial spot and bacterial blight caused by *Pseudomonas syringae* and *Xanthomonas phaseoli*, respectively: A, Leaf infected with bacterial spot showing small lesions and the characteristic ragged, necrotic tissue at the margin; B, pods infected with bacterial spot; C, pods infected with bacterial blight.

Gardner and Kendrick. The organism originally described by Tisdale and Williamson on lima bean and the one described by Gardner and Kendrick on cowpea were later shown (379) to be identical. Gardner and Kendrick's article appeared in March 1923

and the one by Tisdale and Williamson in July 1923; consequently, *Bact. vignae* takes precedence over *Bact. viridifaciens*. *Bact. vignae* is now regarded as a synonym of *P. syringae* (212).

Elliott (321) gives a complete description of *P. syringae*.

Beach (84) found that the migration of *Bacterium vignae* through the tissue of lima bean is in the form of zoogloea. Progress through a tissue is mostly intercellular.

### Hosts

Elliott (321) reported the following hosts to be susceptible: *Desmodium cunescens* (L.) DC., *Dolichos lablab*, *Phaseolus angularis*, *P. limensis*, *P. limensis* var. *limenanus* (*P. lunatus* f. *macrocarpus*), *Stizolobium deeringianum*, *Vigna catjang* (*V. cylindrica*), *V. sesquipedalis*, and *V. sinensis*. Wilson (1125) obtained infection on *P. vulgaris*, *P. multiflorus*, *Glycine max*, *Vicia faba*, *Medicago sativa*, *Citrus limonia*, *Syringa vulgaris*. A blossom blighting of *Pyrus* (751, 848, 1121), *Prunus* (751, 1121), and *Malus* (751, 848) has been reported as caused by *Pseudomonas syringae*. This organism has been reported on Long Island, N. Y., as infecting *Syringa* plantings and also *Prunus* spp. in the fence rows. From these plants the organism spreads to lima beans where at times it becomes very troublesome (751).

### Control

Since the organism causing bacterial spot of lima beans is seedborne, the use of clean seed reduces the chances for its establishment. No information is available as to how the organism lives from one season to the next, except in the seed. Most of the lima bean seed used in the United States is produced where bacterial spot is not known to occur; as a consequence, the danger from that source is probably not important. The organism is known to be parasitic on a number of hosts, some of them grown as cover crops in the winter. It is likely that infection may originate in the spring from such sources. The use of resistant varieties and crop rotation help to hold the disease in check.

Clayton (213) has shown that spraying the plants both before and after blooming prevented a large part of the loss in New York, the yield being increased thereby as much as 43 percent. A 4-6-50 bordeaux mixture gave excellent control and was safe to apply. Copper-lime dust was somewhat less effective than liquid bordeaux. Since first infections are usually on the lower leaves, the first spraying should be applied while the plants are still young.

### Mosaic, a Virus Disease

#### Geographical Distribution and Economic Importance

Lima bean mosaic is caused by a strain of the cucumber mosaic virus (442, 444) (cucumber virus 1 of Johnson). In 1917 McClintock (619) reported a mosaic on several varieties of lima beans in Virginia. Since then, it has been reported in Delaware (19), Indiana (742), Puerto Rico (232), and Cuba on the Challenger va-

riety.<sup>15</sup> The writers have observed lima bean mosaic in Delaware, Maryland, New York, Michigan, and New Jersey, in addition to the locations already reported.

Ho and Li (490) reported a mosaic of lima beans from China. Whether this is the same virus reported here is unknown. Yu (1167) reported a mosaic disease of cowpea that was transmitted mechanically to lima bean.

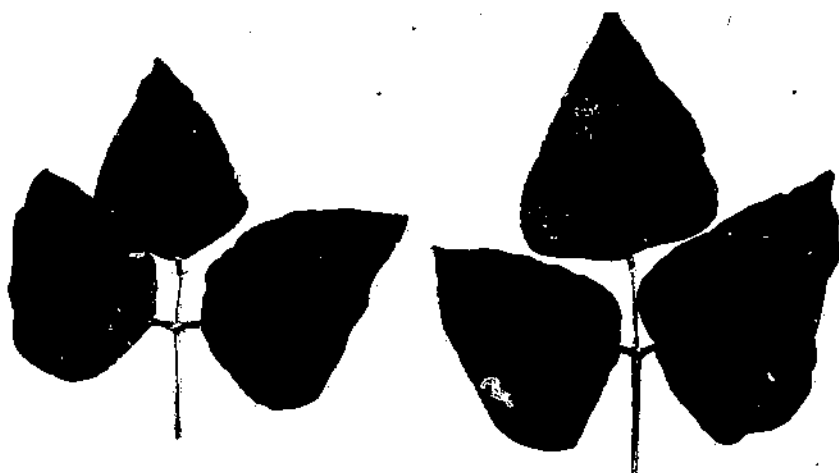


FIGURE 36.—Lima bean mosaic: Lima bean leaves, showing different stages in the development of symptoms.

### Symptoms

The earliest symptoms of the disease are characterized by a slight clearing of the veins of the juvenile leaves. Soon thereafter, the lamina of the leaflet may bend downward to an angle of  $45^{\circ}$  or more, accompanied at the same time by a downward recurving of the midrib. The apical end of the vine often curves downward instead of assuming the erect position of a normal plant. Under the most favorable conditions, symptoms appear in 5 to 6 days when the inoculations are made on nearly full-grown primary leaves. If the plant survives the initial attack, the drooping leaves slowly return to their normal position and at about the same time develop a slight yellow flecking. Sometimes instead of flecks, large light-colored areas of various sizes occur (fig. 36); often these are near the margin. By the time the leaf reaches maturity, the flecks combine to form alternate light and dark spots characteristic of mosaic of many other plants; irregularities in the growth of different parts of the leaf or other causes result in the production of islands of raised or depressed spots of various sizes that are composed of dark-green tissue. The entire series of symptoms is completed by the time the plant has developed the third or fourth set of trifoliate leaves.

<sup>15</sup> Unpublished data, from W. A. McCubbin.

### Relationship to Other Viruses

The close resemblance of the lima bean mosaic to the cucumber mosaic has already been suggested. While the differences between the lima bean and cucumber mosaic viruses are not wide, they are considered sufficient to justify classifying the lima bean mosaic virus as a strain of the cucumber mosaic virus (cucumber virus 1 of Johnson). Some of the differences between the two are as follows: (1) On tobacco, lima bean mosaic virus produces a type of local lesion not caused by the cucumber mosaic virus; (2) on *Vicia faba* it produces both local and systemic infections, which are not produced by the cucumber mosaic virus; (3) the lima bean mosaic virus is destroyed by heating for 10 minutes at 70° C., while 75° is necessary to destroy cucumber mosaic virus; (4) the lima bean mosaic virus will withstand aging in vitro for 8 days at a temperature of 22°, while the cucumber mosaic virus is inactivated in 3 to 4 days.

### Transmission

The lima bean mosaic virus is apparently not seedborne. *Aphis gossypii* and *Myzus persicae*, the only aphids tested, transmitted the virus. It is also readily transmitted mechanically by rubbing the juice of mosaic-infected plants into the leaves of healthy ones.

### Hosts

In addition to the small-seeded sieva types of lima beans, the following hosts are susceptible (444): *Cucumis sativus* L., *Nicotiana tabacum* var. Turkish, *Zinnia elegans* Jacq. var. Double Fantasy, *Lycopersicon esculentum* var. Globe, *Phytolacca americana*, *Solanum melongena* var. Superior High Bush, *Capsicum annum* L. (*C. frutescens* L.) var. World Beater, *Petunia hybrida* Vilm., *Vicia faba*, *Vigna sinensis* var. Black and Blackeye, and *Zea mays* var. *erecta* Bailey.

### Control

Plants of 23 varieties of lima beans were inoculated (444) with the virus from infected plants. Eight varieties, all of the small-seeded sieva type, expressed typical symptoms of the disease, whereas the large-seeded Fordhook sorts were resistant. Among the resistant varieties are a number that are extensively grown commercially, as, for example, Fordhook, Challenger, Burpee Best, Carpinteria, Large White, and Leviathan. Pryor and Wester (795) reported U.S. 243 lima as resistant and U.S. 343 susceptible, even though both were selections from the same Fordhook × sieva cross.

### Blossom Drop, a Nonparasitic Disease

Dropping of the blossoms of lima beans is a common occurrence during the summer months when the soil and atmosphere are dry and the air temperatures high. In some cases no pods set until the growing season is rather well advanced. Under normal conditions many of the blossoms fail to produce fruit, but shedding of

practically all the blooms must be attributed to some definite cause.

Cordner (242) found that plant size and pod set are related to environment during the prebloom and flowering stages, respectively. He also found a correlation between blossom abscission and a high air temperature and a dry atmosphere. A similar relation between weather and blossom drop has been shown by Binkley (99) for a number of varieties of snap beans. A correlation between shedding of the blossom and a fluctuation in the supply of soil moisture has also been shown. Clore (216) obtained poor yields when, in the Yakima Valley of Washington, lima bean seed was planted early in the season and came into bloom when the temperature was high and the relative humidity low. On the other hand, if the seed was planted after June 1, when mild temperatures and relatively high humidities prevailed during the blossoming period, the yield was higher and the quality better. Andrews (42) corroborated in general the conclusions of other investigators, by experiments in two localities in South Carolina conducted under different sets of conditions with Henderson and Fordhook varieties. His experiments, designed primarily to study the influence of environment on yield, showed that the best results were obtained where the mean temperature was low and the relative humidity high. A high yield of pods was found to be associated with a high chlorophyll content of the leaves.

## MISCELLANEOUS FUNGI AND BACTERIA REPORTED ON SNAP, DRY, AND LIMA BEANS

*Agrobacterium tumefaciens* (E. F. Sm. & Towns.) Conn.—Crown gall, occasional in experimental cultures (1103).

*Alternaria fasciculata* (Cke. & Ell.) Jones & Grout.—Described by Jones and Grout (544) as the cause of a leaf disease of potatoes and reported by them on the dead leaves of beans. Anderson and others (40) reported its occurrence on beans in Illinois, Louisiana, Michigan, North Dakota, and Utah.

*Aristastoma oeconomicum* (Ell. & Tracy).—Tehon (1103) reported it as a leaf spot of beans from Virginia and Georgia.

*Ascochyta phaseolorum* Sacc.—Reported to cause ochraceous leaf spots on *Phaseolus vulgaris* in Japan, Uganda, and Europe (972), and also from Peru (81). Reported by Ellis in 1952 (322) from beans in North Carolina.

*Ascochyta pisi* Lib.—Reported on beans in southern Africa (289) and in Russia (now U.S.S.R.) (912).

*Aspergillus flavus* Lk.—Reported on lima beans in Indiana (1045).

*Asteroma phaseoli* Brun.—Reported on *Phaseolus lunatus* and *P. vulgaris* in the Philippines (64, 833). The ripening pods and unhealthy leaves were frequently found covered with large, felt-like black spots.

*Brachysporium pisi* Oud. (perhaps a *Curvularia*).—Reported from Alaska causing a leaf spot of beans (1103).

*Cephalosporium gregatum* Allington & Chamberlain.—Causes a brown stem rot of beans; reported from Illinois (1103).

*Ceratophorum setosum* Kirchn.—Reported as infecting beans (843).

*Cercospora caracallae* (Speg.) Greene.—Reported from Wisconsin as causing a leaf spot (1103).

*Cercospora columnaris* Ell. & Ev.—Saccardo (860) reported the fungus as causing pale to grayish-yellow spots on the leaves of cultivated beans in New Jersey. Chupp (209) considers this synonymous with *Isariopsis griseola* (p. 51).

*Cercospora fabae* Faut.—Woodward (1147) found that, although it does not readily infect beans, it causes round, black spots with grayish centers on the stems. The conidia are seven- to nine-septate,  $60\mu$  to  $100\mu$  long, and  $5\mu$  to  $7\mu$  wide. This organism has not been reported in the United States and is probably not a serious pest of beans anywhere.

*Cercospora lussoniensis* Sacc.—Reported from the Philippines to cause a leaf disease of snap and lima beans (63, 833). The conidiophores are  $28\mu$  to  $35\mu$  long and  $3.5\mu$  to  $5\mu$  wide; conidia are  $35\mu$  to  $45\mu$  long and  $3.5\mu$  wide, commonly three septate. Welles (1104) found that the conidia and conidiophores varied in size, depending on whether they were collected during a rainy or dry

season. Chupp (209) considers this fungus synonymous with *C. cruenta*.

*Cercospora phaseolina* Speg.—Reported to occur on beans in Colombia and Argentina (201).

*Cercospora phaseolorum* Cke.—Reported to cause a leaf spot of beans in South Carolina (235). Chupp (209) considers this synonymous with *C. cruenta*.

*Cercospora stuhlmanni* P. Henn.—Produces dark, irregular-shaped spots on the upper side of the leaves of beans in Tanganyika (971). The organism was originally described by Hennings (475). The conidia are dark, three- to five-septate, cylindrical to somewhat club-shaped or fusoid, not constricted at the septum. They measure from  $40\mu$  to  $65\mu$  in length and  $6\mu$  to  $8\mu$  in width. Chupp (209) considers this synonymous with *C. columnaris*.

*Cercospora zonata* Wint.—Reported (972) to occur on beans in Brazil, Portugal, Union of Soviet Socialist Republics, Italy, and Germany.

*Chaetomium indicum* Cda.—Reported on beans in Canada by Skolko and Groves (917).

*Chaetoseptoria wellmanii* Stevenson.—Causes spots on bean and cowpea leaves in El Salvador (803, 973).

*Cladosporium album* Dows.—Reported on beans in Uganda (426, 427).

*Cladosporium herbarum* Lk. & Fr.—Reported on lima beans (63, 833) from the Philippines. Baker (63) reported *Diplodia phaseolina* Sacc. and *Cercospora lussoniensis* to be associated with *Cladosporium herbarum*.

*Cladosporium herbarum* Pers. ex Fr.—Causes a spotting of pods and seeds from California, Florida, Georgia, Maryland, New Jersey, and Virginia; also on lima beans from the Philippines (63, 834). Baker (63) reported *Diplodia phaseolina* Sacc. and *Cercospora lussoniensis* to be associated with *Cladosporium herbarum* (1103).

*Corynebacterium fascians* (Tilford) Dows.—Reported to produce galls on *Phaseolus vulgaris* in England (583, 584).

*Diaporthe* sp.—Reported from Alabama as a secondary root rot (1103).

*Diaporthe aretii* (Lasch) Nits.—Reported on bean stems from Georgia (1103).

*Dimerium grammodes* (Kze.) Garman.—This fungus was found by Stevens (969) and Garman (380) to occur commonly on the snap and lima beans in Puerto Rico. See also *Parodiella*.

*Diplodia natalensis* P. Evans.—Reported on bean seed from Brazil (1050).

*Diplodia phaseolina* Sacc.—Cause of small black spots on ripening pods of snap and lima beans (831) in the Philippines.

*Epicoccum duriaeanum* Mont.—Reported on *Phaseolus lunatus*.<sup>16</sup>

*Epicoccum neglectum* Desm.—Isolated from bean in Japan but unable to infect healthy beans (519). Also on beans as a secondary leaf spot from Maryland and New Jersey.

<sup>16</sup> ELLIS, J. B. NORTH AMERICAN FUNGI [Exsiccati]. No. 638. 1881.



*Fomes lignosus* Kl.—Hanson (423) produced infection in navy beans artificially.

*Fusarium macroceras* Wr. & Reinking.—On mature pods of beans (1146) in Honduras.

*Fusarium roseum* Lk.—Reported on lima bean in Missouri (1139).

*Fusarium vasinfectum* Atk.—Reported on beans in England and Wales.

*Gloeosporium corallinum* (Peyl.) Sacc. & Trav.—Reported on beans (862).

*Glomerella cingulata* (Ston.) Spauld. & Schrenk.—Reported by Halsted (204) on beans in New Jersey.

*Helminthosporium victorise* Meehan & Murphy.—Reported on beans in North Carolina (1138).

*Hypochnus centrifugus* (Lév.) Tul.—Reported by Endo (331) on lima bean and more than 160 other hosts.

*Hypochnus cucumeris* Frank.—Parasite of *Phaseolus vulgaris* (972) in Japan, Great Britain, Denmark, and Germany. The stem is attacked at the ground level, where a thin grayish layer of hyphae forms. The plants turn yellow, collapse, and finally die.

*Isariopsis laxa* (Ell.) Sacc.—Reported from New Jersey (327) and Indiana by Van Hook (1044). It appears first on the leaves and then on the pods, which become spotted and later rot. Müller (698, 700) reported it from Brazil and Guatemala.

*Leptosphaeria phaseolorum* Ell. & Ev.—Reported (327) on old vines in association with *Diaporthe phaseolorum*.

*Macrosporium commune* Rab.—Reported on beans from Sweden (1140).

*Macrosporium consortiale* Thuem. (*Stemphylium consortiale* Thuem.).—Reported from bean and Scarlet Runner bean seed by Groves and Skolko (402).

*Macrosporium leguminis phaseoli* P. Henn.—Reported on lima beans in South America (477).

*Macrosporium phaseoli* Faut.—Reported on beans in southern Africa (289).

*Microsphaera diffusa* Cke. & Pk.—Reported on beans in Georgia (40), Maryland, and Illinois (1103).

*Microsphaera euphorbiae* (Pk.) Berk. & Curt.—Reported by Gardner (376) to cause reddish-brown patches on leaves of lima beans in Indiana.

*Mycorrhizal fungus*.—Jones (543) listed bean and other legumes among the plants in which the roots are parasitized by a nonseptate mycorrhizal fungus found only in the cortex. Jones did not determine the taxonomic position of the parasite, but he suggested its possible connection with the Phycomycetes. Some investigators have suspected a symbiotic relation between the host and parasite, but Jones suggested that there was some evidence indicating that the fungus was more or less injurious.

*Mycosphaerella cruenta* (Sacc.) Latham.—Causes a leaf blotch on beans and reported from New Jersey to Florida; also in Texas, Arkansas, and Wisconsin (1103).

*Mycosphaerella phaseolicola* (Desm.) Ideta.—Reported to cause (972) pale, reddish leaf spots on beans in Japan and France.

*Nectrea* sp.—Reported on lima beans in Costa Rica (771).

*Nematospora coryli* Pegl.—Reported on lima beans and other legumes in the West Indies (54), the Tanganyika Territory (1071), and Ceylon (750). Ashby and Nowell (55) believed that *N. phaseoli*, earlier described by Wingard (1130), is a strain of *N. coryli*. See Yeast Spot, page 155.

*Oedocephalum roseum* Cke.—Reported on beans in Michigan (238).

*Parodiella perisporioides* (Berk. & Curt.) Speg.—Reported on *Phaseolus vulgaris* and *P. lunatus* in the West Indies (891). *Dimerium grammodes* is given as a synonym.

*Phakospora vignae* (Bres.) Arth.—Reported on *Phaseolus lunatus* in Puerto Rico (891) and in Venezuela (557).

*Phoma terrestris* Hansen.—Isolated from the roots of lima beans (424) and other legumes in Texas, also from bean roots in California.

*Phyllachora phaseoli* (P. Henn.) Theiss. & Syd.—Reported on lima beans in Costa Rica (985).

*Phyllosticta phaseolina* Sacc.—Causes a leaf and pod spot of beans. Reported occasionally from New Jersey to Florida, Texas, Michigan, and Indiana (1103); also from Japan, Formosa, and Italy (972).

*Phyllosticta phaseolorum* Sacc. & Speg.—Reported to cause ochraceous spots (972) of beans in Japan, Formosa (Taiwan), and Italy.

*Physarum cinereum* (Batsch) Pers.—Reported by Johnston and Stevenson (541) and by Seaver and Chardon (891) on green leaves of beans in Puerto Rico.

*Physopella concors* Arth.—Reported on lima beans in Puerto Rico (968). Synonym of *Phakospora vignae*.

*Phytophthora* sp.—A disease of tomato caused by a species of *Phytophthora* was investigated by Reddick (817), and when the fungus was inoculated to young navy bean pods in wounds it caused watery lesions in 24 hours, and in 4 days the pods were nearly destroyed.

*Phytophthora* sp.—Reported by Coombes and Julian (237) as affecting beans in Mascarene Islands in the Indian Ocean.

*Phytophthora cactorum* (Leb. & Cohn) Schroet.—Reported by Young (1165) to infect beans. No data are available as to the extent of the disease and its occurrence under natural conditions.

*Phytophthora capsici* Leon.—Reported on *Phaseolus lunatus* from Argentina (356).

*Phytophthora parasitica* Dast.—Causes a stem and pod rot of beans. Reported from Puerto Rico (1103); also from India (1048).

*Pleospora herbarum* (Pers. & Fr.) Rab.—*Stemphylium botryosum* Wallr. Reported causing leaf spot of beans (295, 1103).

*Pseudomonas aptata* (Brown and Jameson) F. W. Stevens.—The bacterial blight of sugar beet reported to infect bean (50).

*Pseudomonas coadunata* (Wright) Chester.—Causes a stickiness of green beans (1103); reported from California.

*Pseudomonas ovalis* (Ravenel) Chester.—Reported as causing a stickiness of seed from Virginia (1103).

*Pseudomonas tabaci* (Wolf and Foster) F. L. Stevens.—The "wildfire" disease of tobacco. First described by Wolf and Foster (1144, 1145). Reported as causing a bacterial leaf spot of beans from Massachusetts and North Carolina (1103). Also *Phaseolus lunatus* in Formosa (876).

*Pullularia pullulans* (DBy.) Berkhout.—Reported as a seed spot from New York (1103).

*Ramularia* sp.—Reported on beans in Brazil (699).

*Rhizoctonia ferruginea* Matz.—Reported parasitic on snap beans (1033) and sugarcane (660) in Puerto Rico.

*Sclerophoma phaseoli* Karak.—This organism was found to occur on mature beans in Union of Soviet Socialist Republics (547), and to cause dark, small, irregular-shaped, slightly convex spots on the pods. In some cases the whole surface of the pod becomes thickly covered with pycnidia. They ranged from 100 $\mu$  to 300 $\mu$  in diameter. The spores were hyaline, oval, sometimes almost spherical or irregular in shape, 5.5 $\mu$  to 11.5 $\mu$  long, and 3 $\mu$  to 5.5 $\mu$  wide. Condiophores were absent.

*Sclerotinia fuckeliana* Dby.—Reported on beans in Argentina (455).

*Sclerotinia trifoliorum* Erikss.—Reported on stem of beans in Belgium (649).

*Sphaerotheca humuli* var. *fuliginea* (Schlecht.) Salmon.—Reported on *Phaseolus vulgaris* from China (987).

*Stagonospora phaseoli* Dearn. & Barth.—Said to be (274) a parasite associated with *Cercospora canescens* on the leaves of beans.

*Stemphylium botryosum* Wallr.—Causes a leaf spot of beans in Washington (1103).

*Streptomyces scabies* (Thaxt.) Waks. and Henrici.—Experimentally, it infected beans (1103).

*Uromyces fabae* (Pers.) DBy.—Reported on broadbean in England (245) and on *Phaseolus vulgaris* in England and New Zealand (565).

*Vermicularia polytricha* Cke.—Reported in South Carolina (764).

*Xanthomonas vignicola* Burk.—Pathogenic on beans inoculated experimentally in United States (169).

## TRANSIT AND MARKET DISEASES

The term "market diseases," as used in this bulletin, is intended to cover deterioration of snap and lima beans that takes place in transit, at the markets, and in the kitchen. Snap beans are not subjected to long storage for two reasons: (1) Probably because they are available most of the year, thereby eliminating the necessity for storage; and (2) they deteriorate rapidly at temperatures above 20° C. independently of any micro-organisms that may be involved. In experiments conducted by Lauritzen, Harter, and Whitney (591) it was shown that at a temperature of 6° to 7°, they were edible for 4 to 15 days; and at a temperature below 6°, for 6 to 15 days. It was also shown that shipment below a temperature of 10° was desirable when both the quality of the bean and the damage from diseases were taken into consideration. The destructive activity of the micro-organisms and the physiological deterioration are considerably reduced at temperatures of 10° or below.

Most of the market diseases originate in the field. However, those caused by *Rhizopus nigricans* Ehr. ex Fr. and *R. tritici* Saito are two possible exceptions; where the infections caused by them originate is uncertain. As they are cosmopolitan in their distribution, it is probable that infection might take place in the field, in transit, or on the market. Most of the organisms that cause leaf diseases do not cause market diseases. The common and halo blight and anthracnose organisms are exceptions, as they all cause much loss both in the field and on the market.

### Fungus Diseases

#### Anthracnose

Anthracnose, caused by *Colletotrichum lindemuthianum*, is best known as a field disease of the pods (fig. 4, B) and the leaves (fig. 4, D), (p. 5). Infected spots on pods in the field enlarge in transit and the spores spread to healthy pods, thereby exposing all the beans to infection. Infection is possible at temperatures from 7° to 29° C., the optimum being from 22° to 25°. The time for visible infection spots to develop after inoculation increases as the temperature decreases. Lauritzen, Harter, and Whitney (591) showed that the shortest incubation period was 5 days at 22° to 27°, and, like the lesions of the bacterial blights, they developed on apparently healthy beans after all the pods with anthracnose cankers had been sorted out.

#### Cottony Leak

Cottony leak may be caused by several different species of *Pythium*, any one of which may produce a nesting (fig. 37, A)



FIGURE 37.—A, Cottony leak, caused by various species of *Pythium*; the illustration shows the condition of the product on arrival at market. B, Gray mold rot, caused by *Botrytis cinerea*, on beans in transit.

in the shipping container resembling those caused by the several species of *Sclerotinia* and in general appearance indistinguishable from them. Species of *Pythium* often cause a more rapid destruction of beans than *Sclerotinia* does and produce abundant growths of almost pure-white, cottony mycelium.

*Pythium butleri*, the cause of a field disease of beans (p. 28), is also the most prevalent species occurring in transit, especially during the warmer months of the shipping season, and is able to infect either wounded or unwounded tissue. Like the species of *Sclerotinia*, it is prevalent in most soils. The initial infection takes place on pods resting on the ground. From these primary infections the fungus spreads to other pods in the shipping hampers.

Harter and Whitney (448) showed as a result of inoculation experiments that *Pythium ultimum*, *P. splendens* Braun, and *P. myriotylum* Drechs., although not isolated from shipped beans, caused a nesting similar to that produced by *P. butleri*. *P. debaryanum* (p. 28) caused some decay when pods were inoculated by wounding, but it was unable to attack sound tissue.

None of the species of *Pythium* with spiny oogonia that were tried would parasitize beans.

The range of temperature at which infection would occur, according to Lauritzen, Harter, and Whitney (591), ranged from 12° to 35.6° C. Infection at 12° and 15.5° was limited to inoculated beans in storage for 16 days. The optimum temperature for infection was found to be about 31°.

### Downy Mildew

Downy mildew of lima beans, caused by *Phytophthora phaseoli*, is a field disease (p. 142) and not a transit and storage disease, except that pods affected in the field might not be sorted out and as a result would be shipped to the market. There is no evidence, so far as the writers know, that the fungus develops or spreads in transit. In the early stages of infection the tissue of the pod is watery and soft. Later a dense fluffy, white mycelial growth covers the affected area (fig. 30). The mycelium may even penetrate the pod wall and later the seed. Affected seed are unfit for food.

### Gray Mold Rot

Gray mold rot is caused by *Botrytis cinerea*, another cosmopolitan fungus that seems to be present in most soils. It produces a grayish, powdery fungus growth on the surface of bean pods (fig. 37, B) that distinguishes it from watery soft rot and cottony leak, both of which are characterized by an abundance of almost white, fluffy mycelial growth. *B. cinerea* attacks a large number of different food plants in storage and transit over a range of temperature from 0° to 35.5° C. Infection (591) occurs at 6° and 8° in 6 days, at 2° in 11 days, and at 0° in 15 days, and it is greatly facilitated by high humidities. Its development can be arrested by a reduction of the moisture in the air.

### Podblight

Podblight of lima bean, caused by the fungus *Diaporthe phaseolorum*, is primarily a field disease (p. 146) of the leaves (fig. 31, A) and pods (fig. 31, B, C). It is not a transit or market disease and is important only when diseased pods are overlooked at the time the crop is gathered and sorted. The fungus penetrates the pod enough to affect the seed only in cases of very severe infection. The losses are caused entirely by a reduction in market value because of the unsightly appearance. Spots on infected pods enlarge in transit and storage.

### Rhizopus Soft Rot

*Rhizopus nigricans* and *R. tritici* are present everywhere. Both species cause rapid decay of beans. In the early stages at least, a characteristic, sour, acid odor that distinguishes rhizopus soft rot from rots produced by *Sclerotinia*, *Pythium*, *Botrytis*, and *Corticium* is noted. Other distinguishing characteristics are the fluffy, stringy mycelium and the dark to nearly black sporangial heads that stand erect above the mycelium, causing what is often referred to as whiskers. Rhizopus soft rot, being favored by high humidity, is likely to cause considerable loss (807) in cars that have been kept too warm and poorly ventilated. *R. stolonifer* (Ehr. ex Fr.) Lind has also been reported as causing a soft rot of beans.

Infection takes place mostly through wounds. The infected areas enlarge rapidly and often lead to a watery condition of the beans. Careful attention to handling so as not to bruise the pods, together with proper ventilation and refrigeration, will serve as an effective control measure.

### Scab

Scab, a disease of the stem, pods (fig. 32, A, B), and leaves of lima beans, is caused by the fungus *Elsinoe phaseoli* (p. 150). It produces unsightly scab marks on the pods but does not affect the seed. It has been reported to occur in the United States, but it has been collected frequently on the markets from beans shipped from some of the islands of the West Indies. The food value is not generally impaired, but the scabby appearance reduces the market value.

### Slimy Soft Rot

Slimy soft rot is caused by a group of closely related, actively motile, rod-shaped bacteria that are collectively referred to as *Erwinia carotovora* (L. R. Jones) Holland (*Bacillus carotovorus* L. R. Jones). These organisms are not always able to attack sound, healthy plant tissues, but they are capable of entering cells that have been weakened or predisposed to infection and decay by chilling, freezing, wounding, sunscald, and aging. They are present everywhere and have been found to invade plant tissue



FIGURE 38.—A, Soil rot, caused by *Rhizoctonia solani*; this common transportation disease usually originates in the field. B, Southern wilt, caused by *Sclerotium rolfsii*; this disease originates in the field and spreads rapidly in the hampers during shipment. C, Russet, which may be due to various causes. D, Sand injury.



parasitized by fungi and eventually to overrun and crowd out the original invader. This organism has been reported to attack beans under field conditions in Australia (7). Under favorable environmental conditions, such as an abundance of moisture and a fairly high temperature, the bacteria multiply rapidly and disintegrate the plant tissue by dissolution of the middle lamella, which permits the cells to fall apart.

The organism causes a slimy, somewhat watery rot accompanied by a putrid odor. Unless secondary infection has occurred mycelia are absent, a characteristic that serves to distinguish it from other transit and storage diseases. When beans are picked and sorted for shipment during damp, rainy weather there is grave danger that large losses may be caused by slimy soft rot before the beans reach the market. Transportation at low temperatures would tend to retard the progress of the decay.

### Soil Rot

Soil rot is caused by *Rhizoctonia solani*, another common inhabitant of all soils; like species of *Pythium* and *Sclerotinia*, this fungus infects the pods (fig. 38, A) resting on the ground (p. 31). Soil rot causes neither as much loss in transit as the rots caused by *Pythium* and *Sclerotinia* nor as much nesting in the container.

*Rhizoctonia solani* grows at a wide range of temperatures. Lauritzen, Harter, and Whitney (591) showed that infection of snap beans occurred at temperatures from 9° to 35.5° C. It grows somewhat more slowly than *Sclerotinia*, requiring 20 days for infection to start on inoculated beans stored at 0.9° and 2°, and 16 days at 5.5° and 8°. The optimum temperature for infection ranges from 24° to 32°.

### Southern Wilt

Southern wilt is caused by *Sclerotium rolfsii*, a very common soil-inhabiting fungus. It is better known as the cause of a field disease (p. 47) than as a transit or storage trouble. *S. rolfsii* persists indefinitely in the soil and attacks pods (fig. 38, B) resting on the ground. If infected pods get into the shipping container, the fungus grows rapidly under the influence of high humidity and spreads to other pods, forming a mass of rotted beans more or less bound together by the coarse mycelium. *S. rolfsii* develops somewhat parallel, fan-shaped, coarse strands of mycelium that cling close to the invaded bean pods. The destruction of the host tissue is finally followed by the production of numerous, small, at first white, but later brown, sclerotial bodies about the size of mustard seed, which, together with the characteristic mycelium, serve as an easy means of distinguishing the southern wilt from all other transit and storage diseases.

*Sclerotium rolfsii* will grow and infect beans at temperatures from 8° to 36° C. Infection is favored by a high temperature and high relative humidity.

### Watery Soft Rot

Watery soft rot is caused by four different species of *Sclerotinia*, of which *S. sclerotiorum* is the most important. They are primarily soil fungi and attack mostly the lower part of the stem and those pods that rest on the soil. Pods that become infected in the field (p. 42) serve as the means of introducing the disease into the containers in which the beans are shipped to the market. Any badly diseased pods if detected would probably be sorted out, but the slightly infected ones might be overlooked. Under the influence of high humidity and high temperature, the organisms spread rapidly in the shipping container to other pods in contact with them. In the course of a few days many may be involved, and it is not unusual for a peck or more to be decayed in a bushel hamper, forming a watery, slimy mass known as nesting (fig. 39). Infection has been obtained with *S. sclerotiorum* under controlled conditions from 0° to 28° C., the optimum temperature being about 19° to 24°.

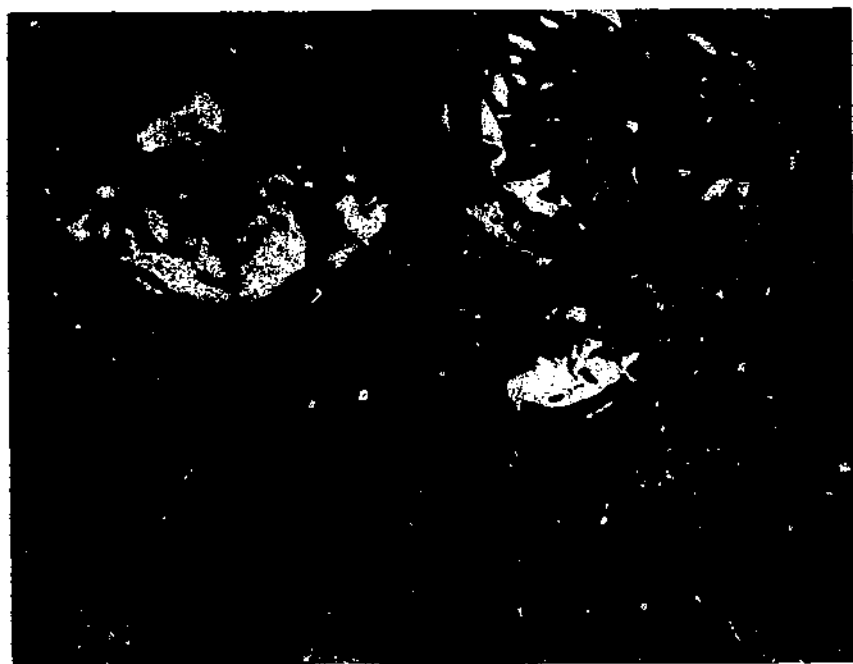


FIGURE 39.—Nesting in hampers of beans on arrival at market, caused by *Sclerotinia sclerotiorum*.

Ramsey (806) investigated the relation of four species of *Sclerotinia* to decay in vegetables, including beans. *S. sclerotiorum* was obtained in about 90 percent of the isolations, indicating that it was by far the most prevalent and destructive species. The

three other species studied were *S. minor*, *S. intermedia* Ramsey, and *S. ricini* Godfrey, and all were capable of decaying beans. *S. minor* caused a more rapid decay than any of the others.

The *Sclerotinia* species as a group parasitize a large number of different hosts and are able to adapt themselves to a wide range of temperatures. They are probably present in all soils in either the mycelial or the sclerotial stage. Any of the species parasitic on bean will infect unwounded as well as wounded tissues.

In case of doubt as to the identity of any of the fungi causing transit and storage rots, it is suggested that some of the material be collected and incubated in a warm, damp chamber. If a white cottony mycelium develops in 25 to 48 hours, the fungus is probably either *Sclerotinia* or *Pythium*. If the surface of the incubated beans is covered by a stringy mycelium with nearly black sporangia, it is probably *Rhizopus*. If sclerotia are present, the organism is very likely to be *Sclerotium*, *Sclerotinia*, or *Botrytis*.

## Bacterial Diseases

### Bacterial Blights

The bacterial blights, being almost identical in their mode of infection and behavior, in general will be discussed together. Extensive investigations showed that wound-free beans could not be infected (591) by spraying them with a water suspension of *Xanthomonas phaseoli*. Infections through wounds made by a needle readily occurred at temperatures from 2° to 31° C. When all visibly infected pods (fig. 15, D) were sorted out from commercial lots, blight lesions developed at temperatures from 1.2° to 35.0° on many pods. The development of the lesions was very slight above 35° and slow at 7° and below. As long as the blights continue to be diseases of economic importance in the bean-growing sections of the country, they will continue to be a hazard in transit and on the market.

### Stickiness and Spotting

Stickiness and spotting are two diseases of shelled lima beans that develop during shipment. Both have been studied in detail by Brooks and McCulloch (142), whose results indicate that stickiness may be caused by several bacterial organisms, the more active ones being identified as probably *Pseudomonas ovalis* (Ravenel) Chester, *Achromobacter coadunatum* (Wr.) Bergey et al., and *A. lipolyticum* (Huss) Bergey et al. *Cladosporium herbarium* caused spotting, although other fungi were occasionally found associated with it.

The spots caused by *Cladosporium* range from 1 to 3 mm. in diameter and have indefinite margins. In the early stage only the testa is affected, but later brown spots occur on the cotyledons. The color is usually brown but may become olivaceous if the beans are held under very humid conditions.

## Nonparasitic Diseases

### Russet

Various types of surface injuries to the pods (fig. 38, C) in the field and in transit are often referred to as russet. The symptoms may be roughly characterized as brownish discolorations of various shapes and sizes arranged more or less indiscriminately over the surface of the pod. The discolored tissue is usually sound and may or may not be sunken.

Some of the so-called russet originates in the field as the result of light injury to the pods. The writers have definitely established the fact that the injury caused by powdery mildew has been referred to often as russet (fig. 38, C) by the shippers and by the consignees at the terminal markets. Natural deterioration also causes a condition known as russet. Beans that have passed their prime before they were picked frequently become russeted, especially in the fall crops grown in some of the Southern States. Apparently healthy when picked and shipped, they develop russet symptoms by the time they reach the market, especially if they have been enroute many days. The outer layers of cells are often damaged, thus predisposing the cells to the attack of slimy soft rot bacteria or even to fungi, if the temperature and humidity are favorable.

### Sunscald

Sunscald caused by too long exposure of the pods to the direct rays of the sun is sometimes referred to as russet. The first symptoms are very small reddish-brown spots, which later may develop into diagonal streaks (fig. 27, D) on the side of the pod exposed to the sun. The side of the pod not exposed to the sun is generally normal in color. In early stages of sunscald the spots are water-soaked and might be confused with the common and halo blights.

## Control of Transit and Market Diseases

The losses from transit and market diseases of beans and lima beans (807) can be reduced (1) by controlling, in the field, certain diseases such as bacterial blights and anthracnose, (2) by picking the beans only when the vines are dry, (3) by sorting out all damaged beans before they are packed for shipment, (4) by keeping the beans out of the sun and rain when awaiting transportation, and (5) by keeping the beans cool during transportation.

Fungi and bacteria require plenty of moisture for their best growth. In view of this fact, it is important that the beans should be picked when the vines are dry. Picking beans when the vines are wet not only introduces conditions favorable for decay in the hampers but also actually spreads the disease organisms on the hands of the pickers to other plants in the field. If, however, the beans must be picked when the vines are wet, care should be taken to dry them if possible before they are packed. If the pods are packed in the shipping container while wet, molds represented

by many kinds of parasites and saprophytes immediately begin to develop. Some of them grow very rapidly, and in the course of a few days mold may damage a considerable percentage of the shipment. When beans are brought in from the field, all those showing any kind of blemishes and wounds should be discarded. After they are harvested and dried they should be kept out of the rain and sun while awaiting shipment.

The growth of most organisms is retarded by cool temperatures. Precooling of beans before shipment would contribute greatly to their arrival at the market in sound condition. Beans resting on the ground are likely to be infected by such organisms as *Rhizoctonia solani*, *Sclerotinia sclerotiorum*, *Pythium butleri*, and other soil-inhabiting fungi. They are the source of initial infections that may result in extensive nesting and consequently considerable loss by the time the shipment reaches the market.

Stickiness and spotting (142) of lima beans appear to be favored by high humidity and a high temperature. Lowering the humidity reduced the amount of stickiness but did not give satisfactory control. Temperatures of 50° or even of 41° F. held the troubles in check for only a few days. Complete control was obtained by washing the beans in a 30-percent solution of ethyl alcohol. Washing the pods in a 4-percent solution of chlorinated lime gave complete control of spotting and commercial control of stickiness.

## INHERITANCE OF DISEASE RESISTANCE AND OF CERTAIN ABNORMALITIES

The extreme susceptibility of many of the most popular snap and dry bean varieties to such diseases as anthracnose, mosaic, bacterial blights, rust, and powdery mildew and the inefficacy of many of the control measures developed up to the present time have probably been the means of initiating and stimulating interest in the development of resistant varieties. The epidemics of bean anthracnose that have occurred in the past in certain sections of the United States and the subsequent discovery that there were several physiological races of the causal organism were convincing proof of the hopelessness of growing susceptible beans in spite of the disease. The idea soon became general that possibly most varieties would be susceptible to one or the other of the physiological races.

Studies on the varietal susceptibility and resistance to several of the bean diseases have shown that a few varieties are tolerant or resistant to a number of the important diseases. Advantage has been taken of this fact to develop disease-resistant strains by hybridization. Coincidental with the production of disease resistance by hybridization, studies on the mode of inheritance were conducted, in many instances, in connection with not only the diseases but also certain leaf abnormalities, the symptoms of which in some cases resembled those of virus diseases. Although more intensive research in the field of disease resistance has been conducted on crops other than beans, it is believed that as large a number of bean diseases and other abnormalities as of other crops have been investigated with reference to their mode of inheritance.

For a more comprehensive review of the literature dealing with the inheritance of disease resistance and physiological abnormalities in general, the reader is referred to articles by Wingard (1136), Walker (1068, 1069), Wade (1057), Boswell (116, 117), Frazier (352), and Thomas and Zaumeyer (1009).

### Disease Resistance

#### Anthracnose

The first contribution on the inheritance of resistance to a bean disease was made by Burkholder (158), who investigated disease resistance to anthracnose, caused by *Colletotrichum lindemuthianum*. Crosses between Wells Red Kidney, a strain resistant to the two physiological races (alpha and beta) known at that time, and White Marrow, resistant to only one of them, when

inoculated in the  $F_2$  generation with a single race of the organism, indicated a single-factor difference in resistance to the race used and showed that resistance was dominant to susceptibility.

Later McRostie (636) crossed Wells Red Kidney and Robust and reported a two-factor difference when two races of the organism were used in the inoculations.

Schreiber (882) supplemented these studies by the use of 37 races, which he divided into 3 main groups corresponding to alpha, beta, and gamma. Reciprocal crosses between anthracnose-resistant Dry Shell No. 22 and Konservä and between Dry Shell No. 22 and Wachs Best von Allen showed a 3:1 ratio, with resistance as dominant when inoculated in the  $F_2$  generation with only 1 race of the organism. When progenies of the same crosses were inoculated with 2 races together, a 9:7 ratio of resistance to susceptibility was noted, indicating a 2-factor difference. When inoculations were made with all 37 races together, a 3-factor difference was indicated. If the races used for inoculation were selected from 2 of these groups, the  $F_2$  hybrids always showed a 9:7 ratio, but if 2 strains were chosen from the same group, the resulting ratio was always 3:1. Schreiber concluded that each of the 3 factors for resistance depended on a different chromosome.

In 1933 Schreiber (883) reported on the inoculation of the same crosses with a mixture of a number of other physiologic races of the pathogen. The first generation was entirely resistant, but the succeeding generations segregated into ratios that led to the conclusion that at least eight different dominant genes were responsible for resistance.

Andrus and Wade (48) studied the inheritance of resistance to beta, gamma, and delta races. Fifteen parent varieties and selections were used in 30 combinations. In crosses of resistant  $\times$  tolerant and resistant  $\times$  susceptible parents, resistance was always dominant. In 2 crosses of tolerant  $\times$  susceptible parents, susceptibility was dominant in the  $F_1$  generation and subsequent generations. Monohybrid and dihybrid ratios were obtained with all 3 races, and trihybrid ratios also were obtained with 2 races. A system of 10 genes in 3 allelomorphic series, involving both duplicate and complementary genes for resistance, 1 dominant gene for susceptibility, and gene interactions at 3 points, is proposed as the simplest Mendelian hypothesis that will coordinate all the data for beta and gamma anthracnose. A simple explanation of 3 independent pairs of genes would account for delta race.

### Common Mosaic

Several investigators have conducted studies on the inheritance of resistance to bean virus 1, but their results did not agree; this disagreement may have been caused by difficulty in transmitting the virus as well as by the influence of environment.

In 1921 McRostie (637) reported on crosses with several dry bean varieties, including Robust, and found that resistance to the virus was partially recessive to susceptibility. The type of inheritance was explained on the basis of more than 1 factor.

The grouping of his data indicated a ratio of approximately 9 susceptible to 7 resistant plants.

Pierce (775) found that when Robust was crossed with Stringless Green Refugee, the  $F_1$  progenies were susceptible; but when either Great Northern U.I. No. 1 or Corbett Refugee were crossed with Robust, the  $F_1$  plants were resistant. Corbett Refugee crossed with Stringless Green Refugee and the reciprocal were also resistant in the  $F_1$  generation, and in the  $F_2$  gave 88.8 percent and 82.2 percent resistant plants, respectively. The  $F_1$  generation of a cross of Great Northern U.I. No. 1 with Stringless Green Refugee was susceptible. The  $F_2$  progenies segregated 15 to 18 percent resistant plants. No factorial explanation of the results was attempted, although it was suggested that the inheritance of resistance may have been in part non-Mendelian.

Parker (754) showed that plants from a cross between Stringless Green Refugee (mosaic-susceptible) and Robust (mosaic-resistant) were susceptible in the  $F_1$  generation, and 99 percent of the  $F_2$  individuals were susceptible. The reciprocal cross gave 82 and 56 percent resistant plants in the  $F_1$  and  $F_2$ , respectively. In  $F_3$  some plants classed as resistant in  $F_2$  gave rise to both susceptible and resistant plants, and susceptible  $F_2$  plants gave rise to some resistant plants in  $F_3$ . When Stringless Green Refugee was used as the female parent and Corbett Refugee as the male parent, some of the  $F_2$  results approached a 3:1 ratio of resistance to susceptibility. Parker interpreted his results on the basis of cytoplasmic inheritance.

Crosses between two resistant varieties produced some susceptible plants in  $F_2$ ; this indicated that the resistant varieties differed in their type of resistance. This behavior was also noted by Pierce (775).

Results of a cross of Black Valentine, which is tolerant to bean virus 1, with mosaic-resistant U.S. No. 5 Refugee and its reciprocal made by Wade and Andrus (1060) indicated that resistance to bean virus 1 was dominant to tolerance and that a single Mendelian factor was responsible for resistance. Their data were collected under field conditions where insects were responsible for the transmission of the virus.

Ali (26) utilized the approach-graft inoculation technique developed by Grogan and Walker (401) to distinguish between varieties with the Corbett Refugee type of resistance and those with the Robust type of resistance. The Corbett Refugee varieties showed top necrosis, while the Robust-type varieties remained healthy.

The crosses Stringless Green Refugee  $\times$  U.S. No. 5 Refugee and Stringless Green Refugee  $\times$  Idaho Refugee showed an  $F_2$  ratio of 3 resistant to 1 susceptible plant following rub-inoculation. This indicated a single dominant gene difference controlling the resistance of these varieties.

In crosses between Stringless Green Refugee and Robust it was found that the resistance of Robust is controlled by a single recessive gene difference.

Crosses between Robust and Corbett Refugee gave an  $F_2$  ratio of 13 resistant to 3 susceptible, following rub-inoculation. By



using the approach-graft inoculation, the above ratio was broken down into 9 necrotic: 4 healthy: 3 mottled. These data suggested 2 pairs of genes acting with dominant and recessive epistasis.

Ali presented the following factorial scheme to explain the results. A dominant gene A is required for virus infection. Another dominant gene, I, when present with the basic gene A, permits the development of top necrosis. The genotypes of the four varieties used are Stringless Green Refugee, AAii (susceptible); U.S. No. 5 Refugee and Idaho Refugee, AAIi (field resistant, top necrotic); Robust, aaii (resistant, no top necrosis).

The necrotic reaction following graft-inoculation is conditioned by gene I only in the presence of A. Plants with the genotypes aaI-, aaii, or A-ii do not show this reaction. The first two genotypes remain healthy while the third develops mosaic symptoms.

Andersen and Down (36) studied the resistance of the variant strain of the common bean mosaic virus and reported that resistance in U.S. No. 5 Refugee is governed by a single dominant gene. Crosses between Great Northern U.I. No. 31, a resistant variety, and six susceptible varieties gave two different types of reaction, depending upon the resistance of the susceptible parent to common bean mosaic virus. The common bean mosaic virus resistant bean plants crossed with Great Northern U.I. No. 31 produced  $F_1$  plants resistant to mosaic and  $F_2$  plants that segregated three resistant to one susceptible, indicating that resistance is governed by a dominant gene. Common bean mosaic virus susceptible plants crossed with Great Northern U.I. No. 31 produced  $F_1$  and  $BC_1$  plants that were susceptible and the  $F_2$  generation segregated one resistant to three susceptible, which indicates that resistance is governed by a recessive gene.

### Curly Top

Schultz and Dean (884) reported that resistance to curly top virus was dominant in its mode of inheritance. Common Red Mexican, Burtner, and Great Northern U.I. No. 15 were used as resistant parents and Red Kidney, Dark Red Kidney, and Bountiful as susceptible parents. The accumulated data indicated that two factors in dominant and recessive epistasis may explain the mode of inheritance to the curly top virus.

### Downy Mildew of Lima Bean

Resistance to downy mildew of lima beans was found in four lima bean collections (1006). Crosses made between these and the susceptible Thorogreen variety showed that resistance to the fungus was controlled by a single dominant gene.

### Fusarium Root Rot

McRostie (637) reported that resistance to fusarium root rot, caused by *Fusarium solani* f. *phaseoli*, was recessive to susceptibility and that two factors were involved in the inheritance. The  $F_2$  generation corroborated the results of the  $F_2$ .

### Halo Blight

Schuster (885) studied the inheritance of resistance to *Pseudomonas phaseolicola* in crosses between Red Mexican and U.S. No. 5 Refugee, Arikara Yellow  $\times$  U.S. No. 5 Refugee, and Red Mexican  $\times$  Asgrow Stringless Green Pod. The resistant varieties Red Mexican and Arikara Yellow when crossed with U.S. No. 5 Refugee indicated a monofactorial type of inheritance, with susceptibility dominant. The  $F_2$  progeny from the cross between Red Mexican and the susceptible variety segregated into a ratio of nine susceptible to seven resistant plants. This suggests a bifactorial mode of inheritance.

In South Australia (1065) two genes were found to govern resistance of bean varieties to halo blight. Disease-resistant selections have been isolated from a Canadian Wonder  $\times$  Burnley Selection cross.

### Lima Bean Mosaic

In crosses between certain lima bean varieties resistant and susceptible to lima bean mosaic virus, the  $F_1$  plants from a cross between Fordhook (resistant) and sieva (susceptible) varieties were resistant (1012). A segregation of nine resistant to seven susceptible plants was obtained in the  $F_2$  generation when the resistant variety was Fordhook or Peerless varieties and the susceptible was Triumph or sieva.

### Pod Mottle

Thomas and Zaumeyer (1008) found that the inheritance of the expression of symptoms of pod mottle virus was governed by a single allelomorphic pair of Mendelian factors. Plants carrying the dominant factor are susceptible to a local-lesion type of infection. The homozygous recessive plants are susceptible to a systemic mottle type of infection.

### Powdery Mildew

The inheritance of resistance to powdery mildew in certain varieties of field and garden beans has been studied by Dundas (305, 306, 307). Most of the varieties carried a single dominant factor for resistance to 12 of the 14 forms of mildew studied. There is also a dominant factor for semiresistance and one for susceptibility during 5 to 7 days after emergence (307). These studies were in part based upon the dish-culture method in which detached leaflets were floated on a 10-percent sucrose solution in petri dishes and inoculated with spores of the powdery mildew fungus to determine susceptibility to the fungus.

### Root Knot

Barrons (72) studied the inheritance of resistance to root-knot nematodes in a cross between the resistant bean Alabama

No. 1 and susceptible Kentucky Wonder variety. The  $F_1$  plants were susceptible to root knot. An  $F_2$  segregation of approximately 11 susceptible: 4 intermediate: 1 resistant was obtained. It was concluded that resistance to root knot in Alabama No. 1 bean is inherited as a double recessive. Barrons believed that the inheritance is on a quantitative basis, with all individuals possessing 2 or more dominant genes appearing susceptible to root knot and those with 1 dominant gene appearing intermediate.

### Rust

The inheritance of rust resistance was shown by Wingard (1133) in 1933 to be dependent on a single dominant factor. His work was conducted before the discovery of various physiologic races of the organism, and it is assumed that he worked with a single race.

The study of the inheritance of resistance was extended by Zaumeyer and Harter (1189) to six physiologic races of rust, involving four different crosses of six bean varieties. The results showed that resistance to races 1 and 2 in the hybrids investigated was the result of a single Mendelian factor, but that more than one factor was involved in the resistance to races 6, 11, 12, and 17. Resistance was dominant in the hybrids inoculated with races 1, 2, 6, and 12, and incompletely dominant in those inoculated with races 11 and 17. A transgressive segregation was indicated in the progenies inoculated with race 11, since one-fourth of the  $F_2$  plants exhibited more resistance than the tolerant parent.

### Southern Bean Mosaic

Zaumeyer and Harter (1190) studied the inheritance of symptom expression to infection with southern bean mosaic virus in five bean crosses involving nine parents. Inheritance was governed by a single gene with local lesion development dominant to systemic mottling.

## Heritable Abnormalities

### Leaf Variegations

Parker (753) studied and described the inheritance of a leaf variegation in beans characterized by large, irregular patches deficient in chlorophyll (fig. 40, A. B). In a single leaf pure white and all gradations from normal green to light and yellow green were observed. Retarded growth of chlorophyll-deficient areas resulted in malformation of the leaflets.

The inheritance proved to be maternal. When a variegated plant was used as the female parent, all of the offsprings in the  $F_1$  generation were variegated. When a reciprocal cross was made with the variegated plant as the male parent and the normal green as the female parent, the  $F_1$  progeny were normal green. In the  $F_2$  generation the highest percentage of offspring from crosses

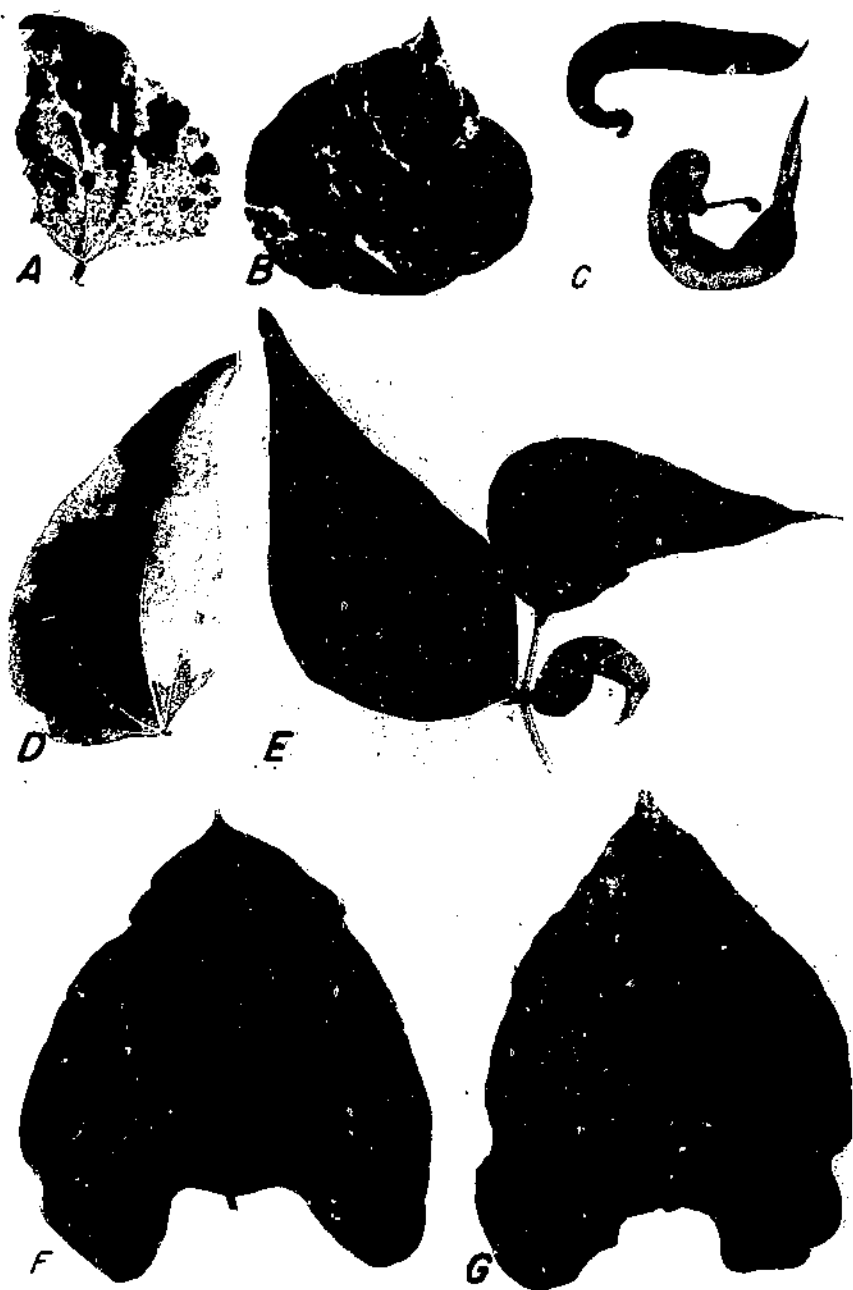


FIGURE 40.—Heritable abnormalities: *A, B*, Leaf variegations; *C*, distorted and malformed pods affected by mottle-leaf; *D, E*, leaves affected by mottle-leaf, showing (*D*) color sectoring and (*E*) dwarfing and curling; *F*, leaf affected by yellow spot; *G*, leaf affected by mottle-leaf.

between variegated female and normal green male were variegated. In 5  $F_2$  families consisting of 313 plants, 262 were variegated and 51 were normal green. In the reciprocal cross where a normal green plant was used as the female parent, only 28 plants of 381 were variegated.

The variegated character appeared to be inherited independently of nuclear factors. Parker explained his results by assuming that the cytoplasm governed in some manner the expression of the variegated character. Whether the plastids or other constituents of the cytoplasm were responsible or the cytoplasm alone was capable of causing this condition is not known.

Another heritable leaf variegation resembling mosaic was described by Zaumeyer (1178, 1180). The symptoms somewhat suggested those produced by a virus, but inoculation studies proved that it was not of a virus nature. The symptoms varied considerably. The primary leaves (fig. 40, *G*) in some cases were practically devoid of chlorophyll, and in such instances they frequently died. In other cases sectors of yellow (fig. 40, *D*) characterized the abnormality.

When severely variegated plants grew beyond the seedling stage they were always stunted, their leaves (fig. 40, *E*) and pods (fig. 40, *C*) were distorted, and their internodes were shortened. Frequently they manifested this condition on only half of each leaflet; this manifestation was accompanied by a curling (fig. 40, *E*) of the normal portion toward the variegated, owing to the unequal growth of the normal and affected parts of the leaf.

In the above-cited studies two types of leaf variegation were encountered. One appeared on the primary and later on the trifoliate leaves, and the other with similar symptoms was confined to the trifoliate leaves.

Breeding studies on the primary-leaf type showed the  $F_1$  plants from reciprocal crosses of variegated by normal green plants were green. A ratio of 15 normal green to 1 variegated plant was obtained in the  $F_2$  generation. The  $F_2$  progenies of the green  $F_2$  plants segregated into 3 classes: All green, 15 green to 1 variegated, and 3 green to 1 variegated. These progenies segregated approximately in the ratio of 7:4:4, respectively. The variegated recessive progenies, except in a few instances where the populations were small, did not breed true in  $F_2$ . It is assumed that this lack of true breeding was caused by 1 or several inhibiting factors which suppressed the variegation character.

Wade (1058) also studied the inheritance of a variegation that occurred on the trifoliate leaves of certain hybrids. In crosses between variegated plants of U.S. No. 5 Refugee and normal green plants of Black Valentine, he interpreted his results on a Mendelian 3-factor difference. A ratio of 27 normal green plants to 37 variegated plants resulted in the  $F_2$  generation. The  $F_3$  generation appeared to corroborate the  $F_2$  data, although there was a deficiency of variegated plants noted in the segregating families. In the 3:1 segregating families of the  $F_3$  generation 3 types of variegated plants were observed. It is not improbable, however, that Wade may have actually been dealing with 3 distinct types of

trifoliate leaf variegation, each being inherited on a monofactorial basis instead of 1 type inherited on a triplicate-factor basis.

In Australia, Conroy and Wilson (226) described a seed-transmitted noninfectious leaf mottling of Tweed Wonder bean. Primary leaves showed a mottling, varying from a few scattered yellow patches to a general yellowing. On the trifoliate leaves, one or more leaflets displayed a few small islands of yellow or white tissue, giving a speckled appearance to the leaflet. A leaflet distortion resembling the leaf variegation described by Zaumeyer (1180) developed on severely affected plants.

### Pale

A heritable chlorophyll deficiency in beans known as pale, characterized by a slightly paler green color than normal leaves, was reported by Smith (934) in a cross between Robust and Pink. The plants were apparently normal in growth habit and in seed characteristics. The pale color persisted throughout the life of the plant.

Reciprocal crosses between Robust and a pale selection showed that the inheritance of this character was controlled by a single recessive gene. The  $F_1$  generation produced normal plants, and the  $F_2$  generation, three normal green to one pale green. The data of the  $F_3$  generation substantiated the  $F_2$  results.

### Pseudo-Mosaic

Pseudo-mosaic was described by Burkholder and Muller (174) in 1926. The leaves manifested pseudo-mosaic symptoms and were longer and narrower than those affected with bean virus 1. The dark areas on the leaves were fairly normal in appearance and were not raised or cupped as in the true mosaic. The light areas were yellowish and at times occurred as streaks on the leaves. At the center of the light areas occurred an anastomosing of the veins, which may have been the main cause of the yellow appearance. Pseudo-mosaic did not stunt the plant or affect the yield.

Investigations showed that pseudo-mosaic was not of an infectious nature but was a heritable abnormality. In the  $F_1$  generation, the pseudo-mosaic character did not appear, indicating that it was recessive. In the  $F_2$  generation 15 normal green to 1 abnormal plant resulted, indicating that the inheritance was governed by 2 factors.

### Seedling Wilt

A seedling wilt that appeared to be of an infectious nature was described by Burkholder and Muller (174). No organism was found to be associated with such plants, and it was later shown to be of a genetic nature.

No symptoms were noted until the young trifoliate leaves began to unfold. The color of the seedlings was somewhat abnormal but hardly noticeable except in contrast with healthy plants. The

primary leaves became pale and somewhat bronzelike in appearance. They showed no rapid flagging but curled slightly and dropped off. Growth of the plant ceased and death followed.

Seedling wilt was first found in crosses between Wells Red Kidney and Robust varieties (72) and appeared only in those hybrids of which one of the parents was Robust, California Wonder, or London Horticultural. This same condition has been observed by the writers in crosses between Red Kidney by Corbett Refugee and Corbett Refugee by U.S. No. 1 Refugee.

Genetic studies (72) gave a ratio of 15 normal green to 1 wilted seedling in the  $F_2$  generation, indicating a 2-factor difference. The  $F_3$  data did not fully confirm these results. No wilted seedlings appeared in more than one-half of the families, while those that did produce them segregated into a ratio of 15 normal green to 1 wilted plant. The expected 3:1 ratio was not obtained.

### Yellow Spot

The inheritance of yellow spot (fig. 40, *F*), a chlorophyll deficiency characterized by definite circular, yellow spots on the primary and compound leaves, was investigated by Parker (752). The spots were numerous on some leaves and formed yellow blotches upon coalescing. Affected plants did not lack vigor and produced as well as normal plants.

Parker (752) made reciprocal crosses between normal-green and yellow-spotted plants and found that in the  $F_1$  generation all plants were moderately yellow spotted, indicating that the yellow was dominant to normal green. The results in the  $F_2$  generation showed that the inheritance was governed by a single factor, with a ratio of 3 yellow-spotted to 1 normal-green plant. Nine families consisting of 755 plants were grown; 564 of these showed the spotted character, and 191 the normal green. When the plants were classified into intensely yellow and moderate-yellow spot, a 1:2:1 ratio resulted with the moderately spotted plants representing the heterozygous class. The  $F_3$  results corroborated those of the  $F_2$ , thus proving that this character was governed by a single Mendelian factor.

## SEED TREATMENT

Seed treatment of beans and lima beans is used for two purposes: (1) To destroy disease-producing fungi or bacteria in or on the seed and thus prevent seedling infections; and (2) to coat the seed with a fungicide that will protect the seed and young seedlings against decay and damping-off caused by organisms in the soil.

There have been many treatments suggested to free the seed of such seedborne diseases as the bacterial blights and anthracnose. The organisms causing these diseases may be borne within the seed, and an effective treatment must destroy the mycelium and spores without greatly impairing its germination. This is difficult, and none of the treatments so far reported are in general use.

For the control of the bacterial blights hot-water (127, 574), various chemicals (108, 152, 172, 406, 576, 763), and antibiotics (152, 489, 942, 964) have been tried. These treatments, although in many cases they reduce the incidence of blight, have not been commonly used.

To control anthracnose, a number of investigators (34, 82, 86, 250, 317, 344, 406, 422, 704, 898, 967) have applied chemicals to the seed with varying degrees of success. Dry and wet heat treatments have been tried, but in general these treatments were not satisfactory.

Bean and lima bean seed are more commonly treated to protect the seed from rotting and the young seedlings from infection by such soil-inhabiting organisms as *Rhizoctonia*, *Fusarium*, and *Pythium*. The degree of effectiveness of seed treatment in increasing stands of snap and field beans is uncertain. Bean varieties vary in their tolerance to damping-off (35). This probably accounts for some of the variation in the effectiveness of seed treatment reported by different workers. Lima beans, particularly the large-seeded Fordhook types, are generally treated because of the excessive rotting and damping-off that may occur under moist soil conditions (633). Heuberger (480) found Spergon and Arasan equally effective for controlling lima bean seedling diseases. In California an insecticidal compound such as lindane is often added to lima bean seed for the control of wireworms (*Limonius* spp., *Melanotus* spp., and *Aleous* spp.) and seed-corn maggots (*Hylemya cilicrura* (Rond.)). In the absence of serious insect damage this treatment usually causes a reduction in the emergence of Fordhook and Ventura lima bean seed. When the insecticide is combined with a fungicide, however, this adverse effect is eliminated or greatly reduced (584, 597).

Various chemicals have been used as seed protectants (13, 35, 219, 257, 258, 260, 279, 482, 575, 599, 628, 668, 783, 1022, 1070). The results of State experiment stations have been summarized



(184, 1040, 1041). Cohn and DeZeeuw (210) reviewed the literature and reported that Spergon proved to be the best protectant for beans. According to McNew and coworkers (635) choranyl (Spergon) and thiram (Arasan) are the most commonly used materials. Cuprocid, Semesan, New Improved Ceresan, and corrosive sublimate are also used. The use of insecticides to control seed-corn maggot in combination with a fungicide is used. Howe and Schroeder (498), working with lima beans, found chlordane, lindane, dieldrin, and aldrin gave the best control of seed-corn maggot, and Arasan the most effective in preventing seed decay and in counteracting the toxic effects on the seeds exhibited by the insecticides.

One of the disadvantages to hot-water treatment of beans has been the wrinkling and slipping of the seed coat. The use of carbon tetrachloride instead of water eliminated this trouble (1089). Beans and lima beans were able to survive extended exposures in hot carbon tetrachloride, while the seeds were killed in a relatively short time when exposed to water of the same temperature.

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