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INTRODUCTION

The inefficacy and cost of many of the chemical bean-disease control measures and the susceptibility of many of the important snap and dry bean (*Phaseolus vulgaris* L.) cultivars to damaging diseases stimulated interest in the United States in the development of resistant bean cultivars through breeding. Early epidemics of bean diseases in certain sections of the country and the later discovery of physiological races or strains of many of the causal organisms proved the hopelessness of growing susceptible bean cultivars. None of the American bean cultivars used commercially before 1918 was disease resistant.

The first disease-resistant cultivar, Robust, was developed by F. A. Spragg of the Michigan Agricultural Experiment Station in 1915 (117). A white, pea-bean type, it resisted the common bean mosaic virus. The next resistant cultivar, Great Northern University of Idaho (UI) 1, developed in 1929 by Pierce and Hungerford of the Idaho Agricultural Experiment Station, was resistant to the same virus and was a selection from the mosaic-susceptible common Great Northern.

Wisconsin Refugee and Idaho Refugee introduced in 1934 were the first mosaic-resistant snap bean cultivars developed by hybridization (95). These were followed by the release of United States (US) 5 Refugee in 1935 (124). About 35 disease-resistant dry bean cultivars and 120 snap bean cultivars have been developed in the United States by seed companies, State Experiment Stations, and the US Department of Agriculture.

The greatest progress in breeding for disease resistance in beans has probably been the development of common mosaic-resistant cultivars of both dry and snap bean types. Unless a newly released cultivar is resistant to the type strain of the virus and to a widespread variant strain, generally known as the New York 15 strain, it is not well accepted by growers, the seed trade, and the bean processing industry.

IMPORTANCE OF THE CROP

In 1973, about 1.4 million acres of dry beans were harvested in the United States with a production of about 17 million 100-pound bags (122). In order of importance, Michigan, California, Colorado, Idaho, Nebraska, and New York account for almost 90% of the acreage of the United States production. Michigan alone produces about 34% of all dry beans in the country on 42% of the total acreage in beans (122). About 85,000 acres of snap beans were harvested for fresh market and about 292,000 acres for processing in 1973, this acreage producing 302 million pounds and about 1.5 billion pounds, respectively (122).

The farm value of dry beans in the United States in 1973 was approximately $514 million; of snap beans for fresh market, about $53 million; and of snap beans for processing, $77 million. Production of snap bean seed in 1973 was about 52.8 million pounds of the green-podded bush types, 3.8 million pounds of wax-podded bush types, and 2.9 million pounds of pole types. Bush type beans are mainly used for processing and the fresh market; most of the pole beans are used for fresh market and home gardens.

The dry bean is the main edible legume grown and consumed in Latin America and Mexico. In some of these countries it is the second most important crop grown; in many of them it is the principal source of protein in the diet. Brazil leads the world in acreage and total production of dry beans, with approximately 5.9 million acres producing about 53 million 100-pound bags annually. It is followed by Mexico and the United States with acreages of about 3 million and 1.4 million acres, which produce about 9.8 and 17 million 100-pound bags, respectively. The yield per acre for the United States is about 1178 pounds; for Brazil, 578 pounds, and for Mexico, 307 pounds. Argentina, Chile, and Peru have per acre yields of almost 30% less than those of the United States. To a great extent, diseases are responsible for much of these yield differences, and breeding for disease resistance to increase yields is now pursued in many of the Latin American countries.

DISEASE RESISTANCE

Virus Diseases

BEAN COMMON MOSAIC Although once a major disease, bean common mosaic (BCM) is now almost nonexistent in the United States, because most varieties of both dry and snap beans developed since 1940 are resistant to known strains of the virus (114).

Resistance to BCM in most US snap bean cultivars is derived from Corbett Refugee (136), which was selected in 1929 from Stringless Green Refugee bean, a very popular processing cultivar at the time. Later studies have confirmed that this cultivar is resistant not only to the type strain of the virus, but also to at least nine other strains described in North and South America (64). The same type of resistance is found in Blue Lake type beans. In the United States, the resistance of

Corbett Refugee has not broken down in more than 45 years. However, strains of BCMV have been reported to which neither the dominant Corbett Refugee gene nor the recessive Robust gene confer resistance (92). However, these viruses have not yet been reported to occur in the United States. If and when they are found and become widespread, they could cause severe BCM epidemics.

Ali (3) used the approach-graft inoculation technique developed by Grogan & Walker (67) to distinguish between cultivars with the Corbett Refugee type of resistance and those with the Robust type of resistance. Cultivars in which Corbett Refugee was the resistant parent showed top necrosis, whereas the Robust-type cultivars remained healthy.

Results of mechanical inoculation of the progeny of crosses, Stringless Green Refugee (susceptible) X US 5 Refugee and Idaho Refugee (resistant) indicated that a single dominant gene controlled resistance of the Corbett Refugee type. However, in crosses between Stringless Green Refugee and Robust, he found the resistance of Robust to be controlled by a single recessive gene.

Crosses between Robust and Corbet Refugee gave an F_2 ratio of 13 resistant to 3 susceptible following mechanical inoculation. By use of the approach-graft inoculation the above ratio was broken down into nine necrotic, four healthy, and three mottled. These data suggested two pairs of genes acting with dominant and recessive epistasis. This type of inheritance was confirmed by Rudorf (104). Resistance in Corbett Refugee is based on a dominant inhibitor gene I epistatic to a dominant gene A required for virus infection. Corbett Refugee confers resistance to all strains of BCMV known in the United States and elsewhere and is an excellent example of Van der Plank's (123) horizontal resistance.

Many dry bean varieties derive their resistance from the recessive gene present in Robust. It is effective only against the type strain of BCMV but not effective against the so-called New York 15 strain. Great Northern UI 1 also carries this recessive gene (94), and both varieties, or the resistant varieties developed from them, have been widely used in the development of other mosaic-resistant dry bean types. Both Robust and Great Northern UI 1 have given virus protection for almost 50 years. Some of the most popular mosaic-resistant dry bean varieties are Sanilac, Seafarer, and Gratiot (all navy bean types); also Great Northern UI 59, Great Northern 1140, Pinto UI 111, Red Mexican UI 36, and Big Bend. According to M. J. Silbernagel (unpublished data), resistance depends upon a different pair of recessive alleles for each strain of the virus. Some of the Michigan navy bean cultivars obtained their resistance to the New York 15 virus from the Corbett Refugee source.

BEAN YELLOW MOSAIC The distribution of bean yellow mosaic virus (BYMV) is found in most areas where beans are grown. The virus and its strains commonly infect *Melilotus alba Medic.*, *Trifolium pratense* L., *T. incarnatum* L., and *Gladiolus* sp. and are transmitted to beans by aphids from these infected hosts. The losses can be very severe if beans are grown close to infected plants of the above species or if viruliferous aphids infest them severely.

Resistance of beans to this virus appears to be conditioned by different genes. Dickson & Natti (57) reviewed the literature on the inheritance of resistance and reported single-factor dominant resistance derived from *Phaseolus coccineus* L. This species and some of its selections resist more strains of BYMV than any variety of *P. vulgaris* (13). Resistance derived from *P. coccineus* has also been reported by Baggett (12) and Baggett & Frazier (14) to be inherited recessivcly conditioned by two or three major genes with other modifiers affecting the variation in symptom expression. Buishand (28) reported that a single dominant gene from *P. coccineus* conferred resistance to top necrosis caused by a strain of BYMV. Some of the Great Northern bean cultivars such as Great Northern UI 16 and 31 resist one or more strains of the virus (16). Resistance to two severe pod-distorting strains of BYMV in Great Northern UI 31 was found to be governed by three major recessive genes with other modifiers (13). Provvidenti & Schroeder (99) found Great Northern 1140 resistant to 82 isolates of the severe strain of BYMV described by Thomas & Zaumeyer (120) and pea virus 2. They found that resistance in Great Northern 1140 is conditioned by a single recessive gene. Resistance to a pea isolate of BYMV in a Red Kidney X Black Turtle Soup cross was found to be controlled by a single dominant factor (107).

Another useful type of virus resistance in beans is insect nonpreference. Hagel et al (69) showed that aphid nonpreference in the Black Turtle Soup is related to field resistance to BYMV. This is an example of horizontal resistance (123), because it would be effective against all strains of the virus.

CURLY TOP Curly top, a virus disease of beans and many other cultivated and wild plants (142), was first reported in 1926. The disease is transmitted by the sugar beet leafhopper, *Circulifer tenellus* Baker. The virus is prevalent only in certain arid sections of the United States where this insect thrives, for example, in the Columbia River Basin of eastern Washington, where in most years only bean cultivars immune to the disease survive.

Mackie & Esau (83) were the first to show the feasibility of breeding for resistance to curly top. They recovered resistant plants from segregating populations of crosses between resistant and susceptible cultivars. The United States Department of Agriculture and the University of Idaho have had bean-breeding programs for curly top resistance for over 40 years. These studies have been conducted in the desert regions of eastern Washington and southern Idaho where the sugar beet leafhopper is usually very abundant.

The virus cannot be transmitted mechanically. Segregating populations are exposed to infection with the virus when rows of curly top–susceptible sugar beets are planted about 20 feet apart in bean plots about a month before the beans are planted. The beets attract the viruliferous leafhoppers, which become a virus reservoir. The leafhopper population on the emerging bean seedlings is increased when the leaves are cut off of the beets, thus forcing the leafhoppers to feed on the beans, which are less desirable hosts. Infection during the seedling stage is preferable, because bean plants become more tolerant of the virus with age.

California Pink, Red Mexican, and Burtner Blightless (52) were the sources of resistance for the curly top–resistant bean cultivars grown at present. California Pink and Red Mexican were used to develop the following curly top-resistant dry beans: Big Bend and UI 36, Red Mexican types; Columbia, UI 111 and 114, Pinto cultivars; Royal Red, a dark Red Kidney type; and Great Northern UI 31.

The curly top resistance found in American snap bean cultivars, such as Apollo, Custer, Idelight, Idachief, Jackpot, Rodeo, Valgold, Goldcrop, and Wondergreen, was provided by Burtner Blightless (114). Schultz & Dean (108) reported that resistance was dominant and conditioned by two factors in dominant and recessive epistasis.

Even though a number of strains of the virus have been reported (121) and differences between varieties in degrees of susceptibility have been noted (113), resistance has remained stable in the field in the United States for more than 40 years. Resistance can, however, be broken down in the greenhouse under high inoculum levels and unfavorable host development.

In Australia, Ballantyne (17) found that 56 curly top–resistant bean cultivars in the United States were resistant to Australian summer death virus. This disease, similar to curly top, is caused by a yellow-type, phloem-restricted, leafhopper-transmitted virus. The leafhopper, *Orosius argentatus* (Evans), differs from the species that transmits curly top. Also, many of the curly top, summer death-resistant cultivars have a high degree of resistance to subterranean clover stunt virus, a yellow type, phloem-restricted aphid-transmitted disease (116).

SOUTHERN BEAN MOSAIC Southern bean mosaic virus (SBMV) was isolated from mottled bean pods originating in Louisiana in 1941 (139). Little is known about the distribution of this virus in the field. Because it is highly infectious, researchers originally assumed that it would become a very serious disease of beans, but it has not. However, because symptoms of SBMV infection are similar to those produced by BCMV or BYMV, it may be more prevalent than is generally recognized. In 1948 it seriously damaged snap beans in southern Illinois. It was also reported from Tennessee and Georgia in 1952 (142). It is not seed borne and its very narrow host range may account for its relative unimportance as a bean-virus disease.

SBMV produces local lesions on some bean cultivars and systemic-mottle symptoms on others (139). No cultivar thus far tested has shown both types of symptoms. Local-lesion-susceptible cultivars can be considered commercially resistant. Most of the snap bean cultivars are susceptible to systemic infection, whereas most of the dry bean cultivars are resistant to it.

Zaumeyer & Harter (138) studied the inheritance of symptom expression to infection by SBMV in bean crosses with nine cultivars. Inheritance was governed by a single gene with local-lesion development dominant to systemic mottling.

POD MOTTLE Little is known of the distribution of pod mottle virus (PMV). It was first isolated by Zaumeyer & Thomas (141) in 1945 from severely mottled bean pods of the Tendergreen cultivar grown at Charleston, South Carolina. In 1948 and

1949 it was isolated in combination with SBMV from beans grown in southern Illinois, and in 1950 it was found in Florida-grown beans.

Although unrelated to SBMV, PMV also produces local lesions on some bean varieties and systemic mottle symptoms on others. Varieties susceptible to local infection are immune from systemic infection, and those susceptible to systemic infection are immune from local infection.

As with SBMV infection, all cultivars susceptible to local infection can be considered commercially resistant.

Most green-podded snap bean and dry bean cultivars are susceptible to local infection; most of the wax-podded cultivars are susceptible to systemic infection. Practically all of the cultivars resistant to BCMV are also resistant to systemic infection by PMV. Reaction of most of the green-podded, bush, snap bean cultivars to PMV differs from that of SBMV (141).

Thomas & Zaumeyer (119) found that the inheritance of the expression of symptoms of PMV was governed by a single pair of factors. Plants with the dominant factor are susceptible to the local-lesion type of infection whereas those with the recessive factor are susceptible to the systemic mottle type infection.

Fungus Diseases

ANTHRACNOSE Anthracnose caused by *Colletotrichum lindemuthianum* (Sacc. & Magn.) Briosi & Cavara does not occur in the Rocky Mountain states and in states farther west. Thus, bean seed grown in such regions is free of the disease. Before the use of western-grown seed, bean anthracnose was very serious in the humid areas of the United States (142). The widespread use of western-grown seed has practically eliminated the disease in this country, making it unnecessary to incorporate anthracnose resistance in domestic snap bean breeding programs. In Michigan, however, where the disease is found, and where much acreage of dry bean seed is grown for seed stock, anthracnose resistance has been incorporated into most of the recent navy bean releases.

In Europe, the environment in areas where bean seed is produced is ideal for the development and spread of the anthracnose organism. Hence, the incorporation of resistance to this disease is an important phase of every European bean-breeding program (65). General use of resistant cultivars resulting from these programs has reduced anthracnose to a minor problem. However, in Central and South America, anthracnose resistance is considered one of the primary breeding objectives (114).

Four races of the causal organism are distinguished by their differential pathogenicity to several host cultivars and are designated by alpha, beta, gamma, and delta. Barrus (23) was the first to demonstrate physiologic races of anthracnose—in fact, of any plant pathogen.

Hubbeling (76), Goth & Zaumeyer (66), Walker (126), and Yarnell (133) have reviewed the sources of resistance, differential cultivars, and the inheritance of resistance to bean anthracnose. Emerson No. 847, a bean strain developed at Cornell University but never released to the trade, resists the alpha, beta, and gamma races of the organism. Wells Red Kidney was reported to be resistant to the alpha and beta races (23). Goth & Zaumeyer (66) summarized the status of varietal resistance

to the various races. Michelite 62 and Seaway are resistant to beta and gamma; Sanilac, Saginaw, Gratiot, Seafarer, and Manitou, a recent Red Kidney release from Michigan, resist all three races of the organism. They also noted that Plant Introduction (*PI*) 304110 resisted beta, gamma, and delta races but was susceptible to the alpha race. Cornell 49-242 resists all four races but has undesirable genetic linkages.

In 1918 Burkholder (33) studied anthracnose resistance. His work provided the first contribution on the inheritance of resistance to any bean disease. When segregating progenies of crosses between Wells Red Kidney, resistant to two physiological races of anthracnose (alpha and beta), and Perry Marrow, resistant to only one race (alpha), were inoculated in the F_2 generation with a single race of the organism, resistance was found to be governed by a single dominant gene. Later work by Bredemann (26) confirmed the dominance of resistance and established the presence of several genes for resistance.

Schreiber (105) supplemented these studies using 37 isolates of the organism which he divided into three main groups corresponding to alpha, beta, and gamma races. Reciprocal crosses between Dry Shell No. 22 and Konserva and between Dry Shell No. 22 and Wachs Best von Allen showed a 3:1 ratio with resistance dominant when inoculated in the F_2 generation with only one race of the organism. When progenies of the same crosses were inoculated with two races together, a 9:7 ratio of resistance to susceptibility was noted, indicating two complementary dominant factors. When inoculations were made with all 37 isolates together, a three-factor difference was indicated. If the races used for inoculation were selected from two of these groups, the F_2 hybrids always showed a 9:7 ratio, but if two strains were chosen from the same group, the ratio was always 3:1. Schreiber concluded that each of the three factors for resistance was on a different chromosome.

In 1933 Schreiber (106) reported the inoculation of the same crosses with a mixture of other physiologic races of the pathogen. From these studies he concluded that at least eight dominant genes were responsible for resistance.

Andrus & Wade (5) studied the inheritance of resistance to beta, gamma, and delta races. In crosses of resistant X tolerant and resistant X susceptible parents, resistance was always dominant. In crosses of tolerant X susceptible parents, susceptibility was dominant. Monohybrid and dihybrid ratios were obtained with all three races and trihybrid ratios with two races. A system of ten genes in three allelomorphic series with both duplicate and complementary genes for resistance, one dominant gene for susceptibility, and gene interactions at three points was proposed as the simplest Mendelian hypothesis that would coordinate all the data for the inheritance of reaction to beta and gamma races of anthracnose. Three independent genes were proposed for resistance to the delta race.

Resistance to alpha, gamma, and delta races found in *Phaseolus aborigineus* Burkart gave a 9:7 ratio and was controlled by two complementary dominant factors (103). Resistance to beta was inherited as a simple dominant factor. Hubbeling (77) proposed about eight dominant, allelic genes for resistance to all four races of the organism. Masterbroek (84) recently found a new dominant *ARE* gene in Cornell 49-242, which gives resistance to all four races of anthracnose.

Resistance to alpha, beta, and gamma races was studied by Cardenas-Ramos (35). In eight crosses, resistance to alpha was dominant. Crosses between resistant parents indicated that at least two genes were involved. In three of eight crosses, susceptibility to beta was dominant. Two hypotheses were proposed: (*a*) the existence of duplicate genes, each able to confer resistance and (*b*) complementary action of two genes conferring resistance. One of the duplicate genes is linked with one of the gamma-resistant genes. Thus, cultivars susceptible or resistant to both beta and gamma occur more often than cultivars resistant to beta and susceptible to gamma, or vice versa. In the crosses of gamma-resistant with gamma-susceptible varieties, resistance was always dominant, but F_2 ratios indicated two duplicate genes or two complementary genes, as with alpha.

As many as ten different sites in the chromosomes can govern resistance or susceptibility of the plant to alpha, beta, and gamma races (37). The accumulation of favorable genes at all ten sites (loci) would be a difficult task. No known variety has this combination. Certain PI strains have the single gene *ARE,* which confers resistance to all the races simultaneously.

Resistance to anthracnose in most European cultivars is controlled by the single dominant *ARE* gene derived from Cornell 49-242, which confers resistance to all known European races (82, 84). Mexico 222 and Mexico 227 also are resistant to the European races. These likewise are controlled by a single dominant gene, which differs from the *ARE* gene (20).

The races of anthracnose found in Australia differ from those reported in Europe and the United States. Cornell 49-242 resists all of the Australian isolates collected during the past ten years (B. Ballantyne, unpublished data).

Oliari et al (91) reported the identification of seven physiologic races of *C. lindemuthianum* on *P. vulgaris* in Viscosa, Brazil, and neighboring locations in the state of Minas Gerais. Races BA-1 and BA-2 belong to the alpha group, race BA-3 to the Brazilian group II, races BA-4 and BA-5 to the Brazilian group V, and races BA-6 and BA-7 to the Mexican group II. *Phaseolus aborigineus* 583 and Costa Rica 1031 were used to subdivide the races within groups. Cornell 49-242 was resistant to all seven races.

In Uganda, Leakey (81) reported that the *ARE* gene from a number of French bean accessions appeared to confer virtual immunity to all isolates of *C. lindemuthianum,* except to two races that produced slight anthracnose lesions, but with negligible sporulation. French accessions were used in preference to Cornell 49-242, in which the linkages are very undesirable. The most promising of the French cultivars are Confinel, Peonel, and Verdon.

RUST *Uromyces phaseoli* (Reben) Wint. has been reported from almost every part of the world (142). Before 1945 it was one of the principal diseases of dry beans in the irrigated areas of Colorado, Western Nebraska, Wyoming, and Montana, but in recent years it has been of minor concern in these areas. It is often important in fall-grown snap bean crops along the Atlantic seaboard and in late fall and winter bean plantings in Florida. Recently, it has been serious in Arkansas and Tennessee. In Central and South America it is considered to be the most important bean disease.

It is of concern also when some of the European cultivars are grown in Africa and other tropical seed-producing areas. In such areas, beans are grown for many months of the year, allowing for a buildup of inoculum.

No varieties thus far have been shown to resist all of the reported races of rust. About 35 races have been described (142) in the United States, and unquestionably many more exist. Fifteen other races of the organism were reported from Mexico by Crispin & Dongo (51). Ballantyne (19) lists 95 additional races from Mexico, Latin America, East Africa, Australia, New Zealand, and the Netherlands, but how many of these duplicate previously described races is unknown.

US Pinto 5 and 14 cultivars (140) resisted more US rust races than any other cultivars at the time of their release in 1946, but are not grown commercially. Quinones (101) developed Luna, a new rust-resistant Pinto bean, for New Mexico. Wingard (131) described ten varieties of pole beans that were resistant to the races prevalent in Virginia. Seminole (132), Florigreen (129), and Dade (38), beans developed in Florida, have shown resistance to races prevalent there. Recently a new race that attacks Florigreen and Dade has appeared (86). Kantzes & Hollis (79) reported that Extender, Wade, Tenderwhite, and Harvester cultivars have a commercial level of tolerance to a new race found in Maryland. In recent field tests in Maryland, the following snap beans are highly resistant: Bush Blue Lake 290, Custer, Mountaineer White Half Runner, and Oregon 1604 (88). Augustin, Coyne & Schuster (10) inoculated 25 varieties with Brazilian rust race B11 and found that only Great Northern 1140 (143) and Kentucky Wonder 765 showed a high degree of resistance.

An earlier report indicated that Westralia was resistant to all races of rust in Australia (7). More recently, reactions of 158 bean lines to natural field infection of rust were assessed by Ballantyne (18, 19) in Australia. She found that many of the green-podded bush and the red kidney types were only slightly infected; this suggested that some of these types showed nonspecific resistance to races of rust. The pole beans and most dry bean types showed either a high level of specific resistance or severe infection; there was no evidence of nonspecific resistance in these groups.

In Mexico, immunity from all but one of the 15 rust races reported there by Crispin & Dongo (51) has been found in Guerrero 6, Guanajuato 10A5, Vera Cruz 10, and Negro 150. N. Vakili in Puerto Rico (unpublished data) has selected 22 lines of dry beans possessing resistance to many races in the western hemisphere.

Meiners (unpublished data) found the following varieties highly resistant to three isolates from Maryland: PI 165426 and 152326, Venezuela 54, Villa Gro, Puerto Rico (PR) 15-R-52, PR 15-R-55, and PR 15-R-57, Aurora, and Cornell 49-242.

Wingard's (130) studies on the inheritance of rust resistance in 1933 before the discovery of a large number of physiologic races of the organism showed resistance to be dependent on a single dominant factor. It is assumed that he worked with only one race.

Zaumeyer & Harter (137) extended the study of inheritance of resistance to six races of rust involving four different crosses of six bean cultivars. Their results showed that resistance to races 1 and 2 in the hybrids was governed by a single 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. 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.

In New Zealand, resistance to US races 10, 17, and 28 was dominant in the F_1 seedlings, but the plants were susceptible when older (134).

Augustin et al (10), in studies with Brazilian race B11, found that in crosses between Great Northern 1140 and four susceptible lines a major gene controlled disease reaction, with resistance being dominant.

Resistance to rust is based on the size of the leaf lesions. At least three systems have been used (51, 53, 70), with ratings from 0 to 5, 1 to 5, and 0 to 10. The lower number denotes immunity, and the higher number extreme susceptibility; intermediate grades denote degrees of resistance. In the 0 to 10 scale, pustule size from 0 to 5 denotes field resistance, whereas larger sizes denote susceptibility (70).

ROOT ROTS Root rots of bean have long been a problem wherever beans are grown. In the principal bean-growing regions of the United States, fusarium root rot is the most serious, followed by rhizoctonia, pythium, and thielaviopsis root rots. More emphasis has been given to breeding for resistance to fusarium root rot; however, the others can be very destructive when conditions favor the development of the respective organisms.

Fusarium root rot Breeding for resistance to the root rot caused by *Fusarium solani* (Mart.) Appel & Wr. f. sp. *phaseoli* (Burk.) Snyd. & Hans. has been one of the most difficult problems to solve in the history of the crop. The importance of the disease was first recognized by Burkholder (34) in 1916 in western New York. There as many as 90% of the plants in several counties were infected, with corresponding losses.

Burke (29–32) reported that many cultural and environmental factors affect the severity of the disease. Many attempts have been made to control the disease by the use of organic soil amendments and chemical treatments, but none has succeeded.

Breeding for disease resistance has been hampered by a lack of a high resistance in parental material. Disease tolerance, however, has been found in PI 203958 collected in Mexico by Norvell and in *P. coccineus* (14, 128). Smith & Houston (115) also reported it in PI 165426 and 165435.

Although several bean breeders have measurable root-rot tolerance in advanced breeding lines, the first commercial cultivars resistant to fusarium root rot have recently been released by the US Department of Agriculture and the Washington Agricultural Experiment Station. Deriving their root rot resistance from PI 203958, these include a Red Mexican type, Rufus, and three Pink cultivars, Roza, Viva, and Gloria. The last was released also by the California Agricultural Experiment Station. Besides their root-rot resistance, they are resistant to curly top and to the type and New York 15 strains of BCMV. The cultivars are less resistant to root rot than PI 203958, but more resistant than any known commercial variety. Tests have shown that Rufus yields 15–30% more seed than any other Red Mexican variety

in fusarium-infested soil, and the resistant Pink cultivars also are superior in yield under exposure to fusarium root rot.

McRostie (87) was the first to report that resistance to fusarium root rot was recessive to susceptibility and that two factors were involved in the inheritance.

In crosses between *P. vulgaris* and *P. coccineus,* Azzam (11) showed that tolerance was recessive and probably controlled by three major genes or two genes with modifying factors. His results indicated no relation between vigor of the root and resistance.

Bravo, Wallace & Wilkinson (25) concluded that resistance to fusarium root rot, whether derived from PI 203958 or scarlet runner, is completely dominant. Additive gene effects are larger than dominant gene effects. Estimates of the number of genes controlling resistance ranged from three to seven, and the effects of individual genes could not be distinguished.

Testing in a greenhouse, Hassan et al (72) found that PI 203958 had four genes for resistance and that breeding line 2114-12, which derived its resistance from *P. coccineus,* had five to six genes for resistance. They concluded that four of the genes from *P. coccineus* are the same as those in PI 203958, and that gene action is mostly additive but that partial dominance of resistance appears in 9- to 13-week-old field-tested plants. Broad-sense heritability was estimated as 62–64% in the greenhouse and as 22% and 79%, respectively, in 5- and 9- to 13-week-old field-tested plants.

Some investigators believe that factors, such as the ability of the seed to germinate in the cold, the ability to develop a large, vigorous root system, and the presence of inhibitory substances in the seed coat and hypocotyls (118) may increase the level of genetic tolerance.

Rhizoctonia root rot Several PI lines have been reported to resist rhizoctonia root rot caused by *Rhizoctonia solani* Kühn; namely, PI 165426, 165435, 109859, 163583, 174908, 226895, and Venezuela 54 (97, 98). Venezuela 54 and PI 165426 were reported to have the highest degree of resistance (97). Yerkes & Freytag (135) believed that resistance to *Rhizoctonia* in the scarlet runner bean was superior to that found in *P. vulgaris.*

McLean et al (85) reported 12 bean breeding and PI lines among 600 tested as showing some resistance to *R. solani* in artificially infested greenhouse soil. The resistant lines were as follows: PI 165426, 165435, 181954, 318696, 318697, 318699, 318700 and breeding lines B3866, Venezuela 54, and 165426 X Alabama 1. No commercial rhizoctonia root rot–resistant varieties have yet been released whose resistance has been derived from the above sources. However, J. R. Deakin of the U.S. Vegetable Breeding Laboratory, Charleston, South Carolina, released a highly tolerant wax bean cultivar named Goldcoast in 1970 and two breeding lines, B3088 and B3787 (unpublished data).

Deakin & Dukes (54) reported that resistance to *R. solani* is highly heritable, although the precise mode of inheritance is unknown. They found also that resistance is associated with colored seed, and they were unable to obtain resistant white-seeded lines because of epistatic effects.

Black root rot Resistance to black root rot caused by *Thielaviopsis basicola* (Berk. & Br.) Ferr. was reported by Hassan et al (73) in PI 203958 and a New York State breeding line 2114-12. Both had the same genes for resistance to the organism. Resistance was found to be partially recessive and controlled by about three genes (65). Also, the genes controlling resistance to black root rot differed from those responsible for fusarium root rot resistance and were not linked (74).

PYTHIUM BLIGHT Resistance to pythium blight caused by five species of *Pythium* was recently found in PI 203958 by Adegbola & Hagedorn (2). They noted it also in Bush Green Pod, a Blue Lake type snap bean.

Kim & Kantzes (80) tested 138 cultivars and lines of *Phaseolus vulgaris* for resistance to *Pythium aphanidermatum* (Edson) Fitzpatrick and found PI 201389 somewhat resistant. They noted PI 203958, previously reported as resistant, susceptible. PI 164893 and 234258 of *Phaseolus lunatus* L., and PI 180466 and 288600 of *Vigna mungo* L. were more resistant than were lines and cultivars of *P. vulgaris.*

Dickson & Abawi (55) found a white-seeded snap bean breeding line 1273 resistant to seed decay and damping-off caused by *Pythium ultimum* Trow in artificially infested soil under growth chamber conditions. Resistance was derived from PI 203958. The results indicated that the association of dark seed-coat color and *Pythium* resistance can be broken.

ANGULAR LEAF SPOT Angular leaf spot caused by *Isariopsis griseola* Sacc. is of minor importance in the United States, but is an important disease in tropical and subtropical regions. In tests conducted in Colombia, Olave (90) found that the most resistant cultivars were Mexico 11, Mexico 12, and Cauca 27a. In Australia, Brock (27) tested 164 cultivars and found 19 resistant and 11 highly resistant. The latter included Alabama No. 1, Cafe, California Small White, Epicure, Mexico Black, McCaslan, Negro Costa Rica, Scotia, Rojo, Chico, and Case Knife. In Spain, Puerta & Alonso (100) reported the cultivars Boriole and San Fiacre to be resistant.

Cardona-Alvarez (36), in Colombia, reported resistance to be controlled by a single dominant gene. Barros, Cardenosa & Skiles (22) reported resistance to be recessive in some crosses and dominant in others. Resistance of cultivars Decal, Maravilla, and Huila 14 is attributed to three recessive genes (89).

POWDERY MILDEW Studies by Dundas (58) showed that Pinto, Hungarian, and Pink were resistant to powdery mildew caused by *Erysiphe polygoni* DC. ex St.-Amans. Also, Alabama No. 1, Contender, Logan, Tenderlong 15, Idaho Refugee, Sensation Refugee No. 1066 and 1067, US 5 Refugee, Topcrop, and Wade have been reported to be resistant to one or more races of the fungus (142). Dundas (59–61) found that the inheritance of resistance in several cultivars of field and snap beans to 12 of 14 physiologic races of mildew was controlled by a single dominant factor for resistance. He noted also one dominant factor for tolerance and one for susceptibility during 5 to 7 days after emergence (61). These studies were in part based on the dish-culture method, in which diseased leaflets were floated on a 10% sucrose solution in Petri dishes and inoculated with spores of the powdery mildew fungus.

WHITE MOLD In a greenhouse screening procedure for resistance to *Sclerotinia sclerotiorum* (Lib.) de By., Adams et al (1) found nine *Phaseolus coccineus* cultivars (Scarlet and White runner types) and *P. vulgaris* lines PI 203958 and 300659, and cultivars Soldier and Steuben Yelloweye tolerant to the organism. In a study of 20 bean cultivars and breeding lines, Anderson et al (4) reported that Black Turtle Soup, Sanilac, Capitol, Aurora, and New York (NY) 6207-2 were tolerant to white mold in western Nebraska in 1973. Whether the tolerance observed in these cultivars resulted from certain physiological or morphological characteristics was not shown.

Nematode Diseases: Root Knot

Barrons (21) studied the inheritance of resistance to root knot caused by *Meloidogyne incognita* (Kofoid & White, 1919) Chitwood, 1949 in a cross between resistant Alabama No. 1 (78) variety and susceptible Kentucky Wonder. He found the inheritance of resistance governed by two recessive genes. Barrons believed that the inheritance is quantitative: all individuals with two or more dominant genes appeared susceptible to root knot and those with one dominant gene appeared intermediate. Later work by Blazey et al (24), using Contender and Cherokee Wax as susceptible parents and Wingard Wonder and Springwater Half Runner as resistant parents, produced a resistant F_1. The F_2 segregated 1 resistant to 15 susceptible plants. They found that bean cultivars as well as *P. coccineus* resistant to *M. incognita* were susceptible to four other species of *Meloidogyne* (24). Certain of these results are considered to be consistent with those of Barrons. Fassuliotis et al (62) reported that PI 165426 and 165435 resisted the root knot nematode.

In crosses between resistant Alabama No. 1 and susceptible Hawaiian Wonder, Hartmann (71) reported that the two-gene hypothesis for resistance reported earlier did not account for the segregation patterns of the F_3 families. Instead, he explained it with a three-gene hypothesis. He found that three pairs of genes equal in action are needed for resistance, but a minimum number of genes for susceptibility is necessary before all resistance is lost.

Bacterial Diseases

HALO BLIGHT The epidemics of halo blight caused by *Pseudomonas phaseolicola* (Burkh.) Dows. from 1963 to 1970 in the snap bean seed–growing areas of southern Idaho stimulated much interest in research on the control of bacterial diseases of bean (63).

Many dry bean cultivars are highly resistant to halo blight infection in the field (47, 142). In the greenhouse, small necrotic spots develop on inoculated leaves of such cultivars, with no systemic spread of the organism (142). Most United States snap bean cultivars are very susceptible. However, some Bush Blue Lake types show some tolerance.

European bean breeders have devoted more attention to the devleopment of halo blight–resistant snap beans than have breeders in the United States. Many French, German, and Dutch cultivars have much resistance to halo blight. for example,

Chicobel and Colana were found resistant with a rating of 1 on a 1 to 9 scale with 1 being resistant and 9 susceptible. Nine varieties were rated 8, 2 as 7, 14 as 6, and 4 as 4 (9).

In Australia, Richmond Wonder, Clarendon Wonder, Hawksbury Wonder (112), and Windsor Longpod (6) are resistant cultivars developed by hybridization. Sources of resistance include Cornell 49–242, Great Northern UI 59, Great Northern 1140, Great Northern Nebraska No. 1, Scout Pinto, Pinto UI 111, Red Mexican UI 34, Red Kote, California Small White varieties Ferry Morse (FM) No. 51, FM No. 59, Sanilac, Seafarer, Bush Blue Lake, Oregon State University (OSU) 190, OSU 10183, and Oregon 1604, PI 181954, 203958, and 150414 (8, 56, 63, 102, 142). Coyne et al (47) and Baggett & Frazier (15) summarized the sources of resistance in beans and their differential reaction to races 1 and 2 of the halo blight organism.

The general pattern of inheritance of resistance to halo blight is not well understood. In South Australia (125) two genes were found to govern resistance in beans to halo blight. Disease-resistant selections were isolated from a Canadian Wonder X Burnley Selection cross. Schuster (109) crossed the resistant varieties Red Mexican and Ankara Yellow to the susceptible US 5 Refugee and reported resistance due to a single major recessive gene. In the cross Red Mexican with susceptible Asgrow Stringless Green Pod two recessive genes were involved. Using a mixed inoculum of races 1 and 2, Dickson & Natti (56) found resistance in PI 181954 to be due to one or two recessive genes with modifiers that can increase resistant levels. Walker & Patel (127) reported hypersensitive resistance against race 1 in Red Mexican UI 3 to be controlled by one dominant gene whereas tolerance against race 1 and 2 in a selection from PI 150414 was conditioned by one recessive gene when crossed with Tenderwhite (93). Coyne et al (50) reported that tolerance in Great Northern Nebraska No. 1 selection 27 to halo blight race 1 was conditioned mainly by a major dominant gene when crossed with white-seeded Tendergreen. Coyne et al (48) reported that systemic chlorosis of the trifoliolate leaves and watersoaking of the primary leaves caused by race 2 of *Pseudomonas phaseolicola* was heritable. In the cross Gallatin 50 (susceptible to both reactions) X Great Northern Nebraska No. 1 selection 27 (tolerant to both reactions), both reactions were controlled mainly by a single dominant gene. In the cross Gallatin 50 X PI 150414 (tolerant to both reactions), both reactions were controlled mainly by a single recessive gene. In the cross Pinto UI 111 (susceptible only to watersoaked lesions) X Dark Red Kidney (susceptible to both reactions), the systemic chlorosis reaction was governed by duplicate recessive genes. Coupling linkage was detected between the genes controlling each of the reactions.

Hill et al (75) reported that the reaction to pod infection by race 1 of the halo blight organism was controlled by another gene, independent of the genes controlling the two leaf reactions. A fourth major gene independent of the genes described above was found to control the wilting reaction of halo blight–inoculated leaves. This genetic analysis of halo blight reaction shows the importance of selecting bean plants with pods that are tolerant to infection and nonsystemic trifoliolate leaf infection.

COMMON BLIGHT Unlike halo blight, very few commercial cultivars of *Phaseolus vulgaris* are tolerant to common blight caused by *Xanthomonas phaseoli* (E. F. Sm.) Dows. and fuscous blight caused by *X. phaseoli* var. *fuscans* (Burkh.) Starr and Burkh. Coyne et al (45) tested 1080 PI accessions, cultivars, and breeding lines of *P. vulgaris* for reaction to *X. phaseoli.* None was found free of symptoms after heavy inoculation, and 28 showed slight symptoms. Two accessions of tepary bean, *P. acutifolius* var. *latifolius* Freeman, showed no infection when inoculated in the field. Using Great Northern Nebraska No. 1 selection 27 as a source of resistance to common fuscous blight, Coyne & Schuster (40, 41) developed the Great Northern varieties Tara and Jules. Tara has moderate tolerance and Jules high tolerance to both organisms. Schuster et al (110, 111) reported the strains of *X. phaseoli* to which Great Northern Nebraska No. 1 selection 27 and some other recently discovered sources of resistance (44) were susceptible. PI 207262 gave a tolerant reaction with all isolates they used.

In crosses between Great Northern 1140 and Great Northern Nebraska No. 1 selection 27, Coyne et al (43, 49) found that the latter contributed several genes for tolerance to the hybrid. In advanced self-pollinated or backcross generations, the pattern of segregation suggested polygenic inheritance for resistance.

Pompeu & Crowder (96) reported in crosses between two bean lines resistant to common blight and two susceptible cultivars (Red Kidney and Black Turtle Soup) that resistance was conditioned by several partially dominant genes. This character was quantitative and highly heritable. Transgressive segregation was observed in all of the crosses studied.

BACTERIAL WILT Although not widespread in the dry bean-growing areas of the United States, bacterial wilt caused by *Corynebacterium flaccumfaciens* (Hedges) Dows. occasionally causes serious losses in western Nebraska. No commercial variety of *P. vulgaris* is resistant to the wilt bacterium. A number of PI lines have shown much tolerance, including PI 165078, 165422, 167399, and 169727. Coyne & Schuster (42) developed the variety Emerson, a great Northern type and the first cultivar tolerant to the bacterial wilt disease.

In the cross Great Northern 1140 X PI 165078 from Turkey, Coyne et al (46) determined that resistance to the organism was inherited quantitatively. In crosses with PI 165078 X Great Northern Nebraska No. 1 selection 27, susceptibility appeared to be conditioned by two complementary dominant genes, with absence of either gene or both genes resulting in tolerance.

BROWN SPOT Similar to bacterial wilt, brown spot caused by *Pseudomonas syringae* van Hall is generally of minor importance to bean growers, although serious outbreaks of the disease occurred in the mid-1960s in snap beans grown in Wisconsin. Resistance to the disease was reported by Coyne & Schuster (39) in Great Northern 1140 and Tempo. Truegreen was found to be tolerant to one strain of the organism, but susceptible to another. In Wisconsin, Hagedorn et al (68) in field tests reported Earliwax, Michelite, Processor, Puregold, Sanilac, Saginaw, Tempo, Truegreen, and ten PI lines as tolerant to the disease. Greenhouse studies did not

substantiate their field results. Inheritance of resistance to this organism has not been studied.

SOURCES OF GERM PLASM

The National Seed Storage Laboratory at Fort Collins, Colorado, keeps stocks of many commercial bean varieties and many older genetic stocks. The US Department of Agriculture Plant Introduction Station at Pullman, Washington, keeps world bean collections of over 8000 items as well as several thousand accessions of related *Phaseolus* species, some of which can be hybridized with *P. vulgaris.* The Norvell Bean Collection of several thousand accessions recently has been purchased by the US Department of Agriculture and will be added to the bean collections at Pullman. This collection consists mostly of bean introductions from Latin America, including wild species and cultivars from all parts of the world. A large segment of the collection is from centers of origin of *Phaseolus* spp. in Mexico. Centro Internacional de Agricultura Tropical (CIAT) at Cali, Colombia, also maintains a world collection of *P. vulgaris* and related species.

GENETIC VULNERABILITY

Genetic vulnerability in snap beans was reported by the Committee on Genetic Vulnerability of Major Crops of the National Research Council (37). In 1970, of 35.5 million pounds of seed of green-podded bush types produced, about 46% had Tendercrop germ plasm in their ancestry, 15% had Blue Lake germ plasm, and about 15% had Harvester germ plasm. Thus, about 76% of the total seed produced of all green-podded bush varieties had germ plasm from only three major sources. The wax-podded bush varieties had more genetic diversity, although the cultivar Earliwax in this class comprised about 20% of the total seed produced.

The production of 80% of the United States' supply of bush snap bean seed in a relatively small area of southern Idaho is dangerous. Potentially the situation is very vulnerable for the users of this seed throughout the United States (37).

In dry beans, according to the Committee (37), genetic vulnerability could likewise be a problem. The most outstanding example is in Michigan where about 500,000 acres are planted annually to four closely related navy bean cultivars derived from the variety Michelite.

The pinto bean rivals the navy bean in total production. The two major pinto cultivars produced in the United States are Pinto UI No. 111 and 114, which are closely related. That about half of the total acreage is in Colorado makes this class of bean potentially vulnerable. However, the pinto bean is grown also in several western states reducing the disease hazard somewhat.

CHALLENGES FOR THE FUTURE

Most of the United States' supply of bush snap bean seed are produced in a relatively small area of southern Idaho. Also the Columbia Basin of central Washington is ideal for bacterial blight- and anthracnose-free seed to grow but the area is subject

to serious curly top infection. Therefore, greater effort should be made to develop more curly top–resistant cultivars for seed production in this area. Thus, more of the southern Idaho acreage could be diverted to the Columbia Basin, where thousands of acres are available, to ensure a dependable supply of disease-free bean seed.

Fusarium root rot causes about 6% loss to beans in the United States, if crop rotation is not rigidly adhered to. Although several tolerant dry bean cultivars have recently been released, efforts should be made to incorporate similar resistance into snap bean cultivars.

In some of the Central and South American countries and in parts of Africa, anthracnose, rust, and angular leaf spot cause heavy losses to bean crops. Parental material that resists the four important races of the anthracnose organism is available, such as Cornell 49–242, Mexico 222, and Mexico 227. Breeding should be begun to incorporate the resistant genes into the important cultivars grown in these countries.

Rust is possibly the most important bean disease in many of the Latin countries, and a number of resistant lines have been developed in several countries. This work should be expanded. When lines or cultivars are found that have a wide base of resistance, efforts should be made to develop, through breeding, resistant cultivars that will be acceptable in many of the countries. To facilitate such breeding for resistance, an International Bean Rust Nursery, similar to the International Cereal Rust Nurseries, has been organized recently. It will be coordinated by CIAT, Palmira, Colombia.

In many tropical areas of Latin America, angular leaf spot often seriously reduces yield during the rainy seasons. Several highly resistant local varieties (27R and Antioquia) were observed several years ago in El Salvador (unpublished data). Others such as Mexico 11, Mexico 12, and Cauca 27a have been reported by Olave (90) from Colombia. Efforts should be made to develop resistant varieties for the areas where the disease is a problem.

Other sources of resistance to common blight and especially fuscous blight are urgently needed. The fuscous blight organism constitutes about 50% or more of the isolates in Michigan (37). The tepary bean is a potential source of resistance to fuscous blight, but the specific barrier to the transfer of genes to *P. vulgaris* remains nearly intact, since a hybrid was obtained from only one of thousands of crosses attempted.

Because many dry bean cultivars resist the halo blight organism, breeders should make every effort to develop resistant snap bean cultivars.

Vertical or specific genes for resistance have been stable for most bean diseases for many years. Nevertheless, breeders should consider using newer methods of breeding to incorporate or retain so-called horizontal or nonspecific resistance to bean diseases. Combination of horizontal resistance with major genes could provide more lasting types of genetic control.

The genetics of resistance to diseases in beans has not been elucidated for many disease-host interactions. These should be studied vigorously, to provide a scientific basis for the practical breeding research.

In recent years, the nature of resistance in crop plants, including beans, has been emphasized. Much information is available on the development of phytoalexins,

such as phaseolin and other chemical constituents, that influence disease resistance or susceptibility in bean plants. Such research is aimed, at least in part, toward the development of chemical tests for resistance. The tests would be of value in screening germ plasm and breeding materials for disease resistance. To date, the authors are not aware that such methods are being applied to practical breeding problems, but a certain amount of effort should continue on basic studies that could develop such tests.

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