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Title: Screening Phaseolus Cultivars for Resistance to White Mold Disease Caused by Sclerotinia sclerotiorum (Lib.) de Bary

Abstract Approved : James R. Baggett

Thirty bean lines and cultivars were evaluated for resistance to white mold in field and greenhouse tests. Measurements of disease reaction in field tests included percent infected pods, number of infected plants, and disease severity ratings. Correlation coefficients for disease severity ratings versus percent infected pods and number of infected plants were r = 0.76 and r = 0.74 (P = 0.01) respectively. A positive correlation in disease reaction among entires (r = 0.53; P = 0.01) was found between the 2 years of field tests, indicating a degree of repeatability of this method for detecting differences in disease reaction. In the greenhouse test, the percent of plants which had collapsed 5 days after 40 hrs of exposure to colonized bean pods which were applied to the stems as inoculum was used to measure disease reaction. Results among entries ranged from 19.7% to

85.9% collapsed plants. Significant differences for disease reaction among the entries were found in both field and greenhouse tests, but little correlation was found between tests. However, information regarding plant architecture and days from planting to maturity was used to identify and separate disease avoidance mechanisms affecting field tests from the disease resistance indicated in the greenhouse tests. Based on results of these tests, the following 9 entries appear to possess useful levels of disase resistance for use in a breeding program: 'Cape', PI 204717, 'Red Kidney', B3749, PI 169787, 'Contender', PI 415965, 'Taylor's Dwarf', and 'Ex Rico 23'.

A field test of 351 F_3 families resulting from 8 crosses showed variability in disease reaction based on disease severity scores. Although this test was not designed as a genetic study, the variability appeared to be normally distributed within each cross, indicating resistance to white mold is a quantitatively inherited trait. There was apparent transgressive segregation among families in each cross, indicating potential for selection with these screening procedures.

Screening *Phaseolus* Cultivars for Resistance to White Mold Disease Caused by *Sclerotinia sclerotiorum* (Lib.) de Bary

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INTRODUCTION

Economic losses caused by white mold have been reported in all major snap and dry edible bean (*Phaseolus vulgaris* L.) production areas of the United States and many throughout the world. The wide host range, adaptability, and longevity in soil of viable inoculum of the causal fungus organism, *Sclerotinia sclerotiorum* (Lib.) de Bary, has hampered efforts to control this disease.

Plant breeding strategies to raise the level of resistance in commercially acceptable cultivars are dependent upon effective screening procedures for differentiating levels of resistance and identifying sources of resistance. No source of resistance controlled by a single gene has been identified. Higher levels of resistance have consistently been found in the runner bean (*P. coccineus* L.). Many conflicting reports about levels of resistance in *P. vulgaris* lines and cultivars have been published. The source of this confusion has been the lack of a single, effective screening procedure capable of identifying levels of resistance and distinguishing resistance from avoidance mechanisms.

The purpose of this study was twofold: to test the effectiveness of greenhouse and field screening procedures

for detecting levels of resistance in a group of bean lines and cultivars, and to evaluate the effectiveness of these procedures in distinguishing disease resistance from disease avoidance.

REVIEW OF LITERATURE

EPIDEMIOLOGY

The causal organism of white mold disease is Sclerotinia sclerotiorum (Lib.) de Bary. Purdy (31) reports the host range of this fungal pathogen to include 361 species of 225 genera in 64 plant families. In beans, a white mold epidemic is initiated by ascospores produced from apothecia of germinated sclerotia (2). Sclerotia require a conditioning period before producing apothecia (4). Schwartz and Steadman (34) reported apothecia production by 70% of sclerotia collected in spring versus only 10-20% by sclerotia collected in fall. Direct mycelial growth from sclerotia has not been reported to play a significant role in disease initiation or development. Only sclerotia found in the top 2-3 cm of soil are functional since apothecia with stipes longer than 3 cm are rarely produced under field conditions. Periods of cool, moist conditions favor apothecia formation. Apothecia production from sclerotia is mainly limited by the absence of prolonged periods of high soil moisture conditions. Ten days of continuous moisture at or very near the soil saturation point are generally required for apothecia development. However, Weinzierl and Koepsell, (Fifth Sclerotinia Workshop 1983, unpublished) reported that under western Oregon conditions apothecia are produced following interruptions

of soil saturation. Temperature can have a direct effect on apothecia production; however, in bean producing areas of the United States, temperatures during the growing season are seldom too high or too low for apothecia production. Temperature, relative humidity, and wind velocity only affect apothecia production indirectly through their effect on moisture conditions in the top 2-3 cm of the soil (11). In irrigated areas where limited rainfall occurs during the growing season, conditions conducive for apothecia production do not occur until the canopy of the plant develops sufficiently to restrict soil evaporation rates. Larger, denser plant canopies reduce the effect of temperature, relative humidity, and wind velocity on soil moisture thereby prolonging periods of soil saturation. In areas where rainfall occurs throughout the growing season, apothecia may be produced without the presence of a dense plant canopy.

Ascospores are dispersed when slight decreases in moisture tension cause forcible ejection of the spores approximately 1 cm into the air from the apothecia surface (41). This enables the ascospores to reach more turbulent air layers and be further dispersed. Distances of dispersal up to several km have been reported. Ascospores which have been deposited on bean tissue can survive up to 12 days when conditions are not favorable for germination. It is estimated that a single sclerotium has the potential

to produce apothecia yielding 2.3×10^8 ascospores (34).

Ascospores require an exogenous energy source to infect healthy tissue of bean plants (1,32). Spent bean blossoms are the common energy source. However, ascospores can directly infect a healthy plant through wounds (3). A 48-72 hr period of continuous leaf wetness is required for ascospore infection of bean flowers (2). Moist flowers infected with actively growing mycelium in contact with healthy tissue require 16-24 hrs of continuous surface wetness for infection. Dry, colonized flowers require over 72 hrs of these same conditions for infection to occur. High relative humidity does not substitute for this surface wetness requirement.

Under conducive moisture conditions, water soaked lesions develop on leaf, stem and pod tissues which are in contact with infected flower parts. These lesions give way to mycelial mats within a few days. Within 7-10 days sclerotia are produced on this mat and the area takes on a bleached, dry appearance with black sclerotia visible. These sclerotia rarely germinate during the same growing season. Secondary spread of the disease only occurs from contact between adjacent plants or plant parts. It is believed this occurs only to a very limited extent in beans (2).

CONTROL

In a survey conducted in western Nebraska from 1970 - 1973, Kerr et al (27) found that in 102 dry edible bean fields, plants averaged 30% infection and fields averaged 13% yield loss due to white mold. At the 1983 price of dry beans this amounted to a $200/ha loss. This is slightly more than twice the estimated cost of a single aerial fungicide application (39). In New York, control is even more critical. In addition to direct losses in the field, detection of more than 2% pod infection in a truckload of green beans can result in rejection of the entire load at the processing plant (39). In Oregon, the total value of the 1982 processing bean crop was $22.5 million (43). The incidence of white mold ranged from 0-28% with an estimated $1.5 million in yield losses attributed to the disease.

The two most common methods of control now recommended are crop rotation with non-host crops and fungicide sprays (22). Crop rotation with non-host crops is often recommended in an attempt to limit sclerotial inoculum buildup in soils. However, Steadman (39) reports comparable sclerotia populations in various corn, sugar beet, and bean crop rotations despite differences in occurrence of the bean host in the previous cropping history. In addition, apothecia were found in fields of non-host

crops. In a study conducted in Maryland, Adams (4) found that within 2 years, inoculum density declined to 30% of original density regardless of cropping sequence. Schwartz and Steadman (34) found no progressive accumulation of sclerotia in fields annually planted to susceptible bean cultivars nor progressive decrease in fields rotated to non-hosts during 1973-1976. Furthermore, no correlation between sclerotial populations and disease severity was found in their study. In New York, Abawi (2) found that the soil population of sclerotia did not increase in a field where 3 consecutive years of severe white mold epidemics resulted in total loss of the crop. He concludes that although most sclerotia appear to be ephemeral some are long-lived. Halkilahti (23) reports sclerotia can survive in soil for more than 4 years. The within field source plus the availability of ascospores from outside of bean fields may explain why no correlation has been found between previous cropping history of bean fields and white mold incidence and severity.

Fungicidal protection of bean flowers has been more successful than crop rotation in control of white mold. To date, benomyl has been the predominant fungicide for this use but others such as thiophanate methyl are used. In Oregon, a conditional use permit has been issued for vinclozolin. Other fungicides continue to be tested for effectiveness of control. Studies by Hunter et al (24)

demonstrated the importance of thorough flower coverage and spray timing for effective protection in snap beans. Spraying entire plants or only flowers provided protection against infection. However, spraying the entire plant except for the flowers provided no protection. Therefore, a single application at the early bloom stage or applications at early and full bloom stage can provide good protection. This type of protection has been less successful in dry edible beans. Steadman (38,39) attributes this to the fact that most snap bean cultivars are determinate and flower for only 2 wks after initial flowers open. Most dry bean cultivars are indeterminate and flower for 4 wks after first flowers open. During the last 2 wks of this period it is impossible to achieve good plant coverage because of the dense canopy formed by these cultivars.

BREEDING FOR RESISTANCE

No single source possessing a high level of host resistance to white mold has been found in *P. vulgaris*. A higher level of resistance has consistently been found in *P. coccineus* and in crosses between *P. coccineus* and *P. vulgaris* (1,8). However, no commercially acceptable cultivars of dry edible or green bean types have been released as a result of these crosses. There are many conflicting reports in the literature regarding the levels

of resistance in given cultivars. For example, Coyne et al (14) reported that the dry edible bean cultivar 'Black Turtle Soup' possessed a high level of genetic resistance whereas 'Aurora' tended to avoid infection due to its erect growth habit. Yet in 1982 Hunter et al (25) reported 'Black Turtle Soup' possesses little to no genetic resistance. Furthermore, in another study Coyne et al (13) found 'Aurora' to be susceptible at a plant spacing of 30.5 cm within the row but tolerant at a spacing of 4.5 cm. Abawi et al (1) reported resistance to white mold in the P. coccineus selection B3749 to be controlled by a single dominant gene. A study by Dickson et al (19) indicated that resistance in B3749 and other lines is quantitatively inherited by accumulation of minor genes. Studies by Roberts et al (33) and Agbo and Wood (6) support the hypothesis of quantitative inheritance. In both studies, narrow sense heritability estimates were low, ranging from 0.05-0.30 in different crosses used in the Roberts study and averaging 0.12 in Agbo and Wood's work.

Two major factors have contributed to this confusion. One is the lack of a single, effective, consistent screening test which can identify levels of resistance. The other is the fact that numerous studies (7,12,20,35, 40) indicate that plant architecture can act as a disease avoidance mechanism. Thus, under some conditions a plant

may escape the disease due to its plant architecture and yet possess no genetic resistance.

Field evaluation of beans for white mold resistance generally involves planting in an area where the disease has occurred in previous growing seasons to insure the presence of sclerotia as inoculum. In the absence of naturally occurring inoculum, preconditioned sclerotia can be placed in the trial area to serve as inoculum (17). Conducive conditions for infection are provided by keeping the soil saturated just prior to and during the bloom period. This stimulates production of apothecia from sclerotia. In the absence of naturally occurring conducive conditions, prolonged periods of leaf wetness are insured by periodic sprinkler irrigation. Often windbreaks of corn or sunflower are planted around the test area to restrict air movement thereby prolonging periods of leaf wetness (17,28).

Evaluation of disease reaction generally involves disease severity scores. These take the form of severity rating scales (18), percentage estimates of population infection (20), and individual plant evaluations of infection (29). Field evaluation has distinguished differences in disease reaction among various bean lines, cultivars, plant introductions, and species. However, this procedure does not determine whether these differences are due to disease avoidance or physiological resistance.

Greenhouse screening procedures have been developed in an attempt to identify sources of physiological resistance. Adams et al (5) inoculated plants by placing an oat seed colonized by the fungus on the soil next to the stem. Abawi et al (1) sprayed flowering plants with a suspension of ascospores. Schwartz et al (35) used mature, detached, colonized bean flowers placed in the axils of leaves to initiate infection. These workers concluded that a more sensitive and critically controlled environment was needed to detect differing levels of resistance. In response to this need, Hunter et al (26) modified Abawi's ascospore flower technique, refined Schwartz's detached flower leaf axil inoculation procedure, and developed a juvenile stem inoculation procedure. Hunter's procedures make use of the concept of limited term inoculation which is based on the hypothesis that resistance results in slower growth of the organism. Use of his procedures has tended to support this hypothesis (10,15,19,25).

The concept of limited term inoculation involves direct application of the inoculum to plants, placement of inoculated plants in conditions conducive to disease initiation and development for a limited amount of time, removal of the original inoculum from plants, removal of plants from the conducive conditions, and evaluation of disease reaction (26). In Hunters ascospore flower inocu-

lation technique, flowering plants are sprayed with a suspension of ascospores and placed in a greenhouse mist chamber for 7 days. They are then removed and scored for disease reaction using a 0-5 subjective rating scale (0 = no disease; 5 = severe disease characterized by a large, water soaked lesion covered with cottony mycelium and/or collapse of the stem). In this procedure, contact between flowers and green tissues is left to chance. In the detached flower inoculation method, detached fully open bean flowers are sprayed with a suspension of ascospores and placed in the axils of leaves. Plants are then placed in a greenhouse mist chamber for 7 days. After this time, the original inoculum is removed from the plants and plants are removed from the mist chamber to be subjectively scored for disease severity using the same 0-5 scale. The juvenile stem inoculation test is similar but small pieces of colonized celery petiole or canned green bean pods are used as inoculum. Colonization is achieved by placement of the celery or bean pod on a rapidly growing in vitro culture of the fungus for 24 hrs at 22°C. The colonized pieces are then placed on the second or third internode of 4-5 wk old bean plants. These plants are in turn placed in a mist chamber for 48 hrs after which time the inoculum is removed and the plants are returned to the greenhouse for disease severity evaluation using the same 0-5 scale. Hunter et al (26) found a good

correlation between results of the juvenile stem test and the detached mature flower techniques. However, they found a significant difference between these procedures and the ascospore flower inoculation procedure. These differences were attributed to the fact that in the latter procedure contact between infected blossoms and green tissue was left to chance. Similar results were found by Cline and Jacobson (10) using the juvenile stem test and the ascospore flower technique to differentiate white mold disease reaction in soybeans (Glycine max).

Since publication of the Hunter et al study in 1981, some modification has been introduced to increase the sensitivity of their procedures in distinguishing differences in disease reaction. In their most recent method, Dickson and Hunter (17) grow plants in the greenhouse during the winter for 21 days at 21°C ±5 under metal halide lights to avoid spindly growth. For the juvenile stem test, the inoculum is prepared by placing canned green bean pods in a pan which is then autoclaved, cooled, and sprayed liberally with an ascospore suspension (4000 spores/ml sterile water). The pans are covered with sterilized aluminum foil and incubated for 4 days at 22°C. Water is squeezed out of the mycelium covered pods until the weight is only about 20% of the original canned weight. A piece of colonized pod is wrapped around the first node above the cotyledons and plants are placed in a

mist chamber for 15 hrs. After this time, the inoculum is removed and plants are returned to the greenhouse. After 7 days plants which have not collapsed will survive and produce seed. A disease severity rating scale is no longer used. Only the proportion of plants which have collapsed is recorded for each line being tested. Differences in this proportion are used to distinguish levels of resistance. The detached flower test is unchanged except that the plants are held for 5 rather than 7 days in the mist chamber and the disease rating scale is now based on the proportion of collapsed plants rather than a severity index.

Using the field screening procedure and both the juvenile stem test and detached blossom leaf axil inoculation procedure, Dickson and Hunter (15,18,19) believe that progress is being made both in identifying superior parents with resistance to white mold and development of white mold resistant lines. They believe that these methods allow them to make selections for physiological resistance rather than escape mechanisms due to plant architecture.

MATERIALS AND METHODS

FIELD PLOT DESCRIPTION

Twenty-nine lines and cultivars of *P. vulgaris* and 1 line of *P. coccineus* were evaluated for resistance to white mold in 1982 and 1983. Most *P. vulgaris* cultivars were green pod or dry edible types. The exceptions were 1 green shell and 1 wax pod type. Three lines were obtained from the U.S. Plant Introduction Station in Pullman, Washington (Table 1). In 1982, 351 F_3 families were also evaluated for resistance.

The field trials were conducted at the Oregon State University Vegetable Research Farm near Corvallis, Oregon in an area where bean plots infected with white mold were located in 1980 and 1981. Each of 3 adjacent trial areas contained 12 rows of bean plots surrounded by a windbreak consisting of 4 rows of sweet corn (*Zea mays*) planted parallel to the bean rows. Two transverse rows of corn were planted at each end of the trial. The peripheral windbreaks were seeded 23 June 1982 and 23 May 1983. One trial area was used to evaluate the cultivars while the other 2 were used for the F_3 Families.

On 9 July 1982 and 6 June 1983 all bean cultivars were seeded in single plots 1.8 m in length in rows 92 cm apart. Single rows of green bean ('Oregon 1604') were planted to serve as a border between the plots and the

Table 1. Cultivars evaluated for white mold disease resistance in 1982-83 field trial and greenhouse tests.[z]

Cultivar	Seed Source[y]	Culinary Type
Astro	2	green pod
Aurora	6	dry edible
B3749	4	scarlet runner
Black Turtle Soup	6	dry edible
Black Valentine	9	green pod
Bountiful	7	green pod
BBL 53	2	green pod
Cape	2	green pod
Checkmate	2	green pod
Coloma	7	green pod
Contender	7	green pod
Ex Rico 23	8	dry edible
Gabriella	2	wax pod
Gallatin 50	10	green pod
Geneva 19-2	5	green pod
Green Crop	7	green pod
Harvester	2	green pod
Midnight	6	dry edible
Orbit	2	green pod
Oregon 1604	6	green pod
Pinto UI III	6	dry edible
Provider	7	green pod
Red Kidney	9	dry edible
Roma	7	green pod
Taylor's Dwarf	7	green shell
Tendercrop	7	green pod
Tidal Wave	1	green pod
PI 169787	3	dry edible
PI 204717	3	dry edible
PI 415965	3	dry edible

[z] All cultivars are *Phaseolus vulgaris* except B3749 which is *Phaseolus coccineus*.

[y] Sources: 1 = Ferry Morse Seed Company; 2 = Asgrow Seed Company; 3 = USDA Plant Introduction Station, Pullman, WA; 4 = J. Meiners, USDA; 5 = G.S. Abawi, Geneva, NY; 6 = Idaho Seed Bean Company; 7 = Rogers Brothers Seed Company; 8 = J.C. Tu, Harrow, Ontario, Canada; 9 = Oregon State University; 10 = Gallatin Valley Seed Company.

parallel corn rows. Single transverse corn rows were also seeded at 9 m intervals through the entire trial area at this time. In addition, 8 cultivars were chosen from this group for more intensive study. Each of these cultivars was planted in the same trial area with the single row plots but in adjacent rows 3.7 m long. This was done to more closely simulate commercial field conditions. Each of the 8 cultivars planted in this manner was considered 1 entry and only the center row was used for observation. In 1982, these 8 cultivars were also planted in similar 3-row plots in another area of the farm which did not have a recent history of white mold. This area was considered a low disease potential or control area. No windbreak was planted in this area. In all cases a randomized block design with 4 replications was used. 'Oregon 1604' was included as the known susceptible check.

In 1982, each of the 351 F_3 families was planted in plots 1.8 m in length using a randomized block design with 2 replications. These 2 blocks were located on each side of the trial area used for cultivar screening and separated from it by the parallel corn rows. These families were derived from 8 crosses made in 1980 between cultivars considered to have white mold resistance and susceptible cultivars. F_3 seed was collected from random F_2 plants in 1981. In each block, 1 plot of 'Oregon 1604' was planted as a check in each of the 9 sections divided by transverse

corn rows.

All plots in both years were thinned to 10-12 plants/30 cm. Cultural practices and pest control for all plots were similar to those used in commercial green bean production in western Oregon. Approximately 600 kg/ha of 8-24-8 granular fertilizer was applied in a band prior to planting. A granular soil insecticide (fonofos) was incorporated prior to planting at 2 kg a.i./ha. Dinitro amine was applied as a post planting pre-emergence herbicide at 4 kg a.i./ha. No fungicides were applied on any of the trial areas.

Irrigation was provided by a sprinkler system which delivered water at a rate of 0.85 cm/hr. Irrigation was applied as needed for satisfactory growth, approximately every 7-10 days, until plants began flowering. In the case of the 8 cultivars planted in the control area of the farm without a windbreak, this irrigation regime was used throughout the season. Beginning at the time of first flower, all plots in the disease area were irrigated for 5 min periods 3-4 times a day to provide a favorable environment for disease initiation and development. The last irrigation each day was applied at dusk to insure the presence of moisture in the canopy through the night. This practice continued for the remainder of the growing season except when rainfall provided adequate moisture. Adequate amounts of rainfall to maintain soil saturation

and especially to prolong periods of leaf wetness in the canopy were rare in both years. The presence of dew sometimes eliminated the need for morning irrigation. However, even on cloudy, humid days supplemental irrigation was required to maintain leaf wetness.

DISEASE EVALUATION

In 1982 all bean cultivars were evaluated for disease severity 5 times between 14 September and 7 October. The F_3 families were evaluated 3 times. Disease severity was evaluated using a 0-5 rating scale where 0 = no symptoms, 1 = leaf lesions, 2 = stem and leaf lesions, 3 = majority of plants with stem and leaf lesions, few plants wilting, 4 = almost all plants with stem and leaf lesions, many plants wilting and dehiscing leaves, and 5 = completely diseased plot. Growth habit for all cultivars and families was classified using the Centro Internacional de Agricultura Tropical (CIAT) classification scheme (36) when most plants were at the full bloom stage. A record was made of the date at which each cultivar reached the physiological stage equivalent to processing maturity of green beans.

In addition to these observations, individual plants were collected from the center row of each of the 8 cultivars planted in adjacent 3-row plots. Ten plants were chosen at random on each of the first 4 disease

scoring dates in 1982. Samples were taken from both the disease area with the windbreak and the control area which received only commercial cultural practices. The number of infected plants (NIPL), total plant weight (PW), percent of infected pods (PIP), and total number of pods (TNP) were recorded from each of these samples.

In 1983, all plots were evaluated for disease severity only once on 25 August when most green pod cultivars were at processing maturity. The 0-5 rating scale was used as in 1982. A canopy index scale was devised to indicate the erectness of the plant habit of each cultivar where 1 = strongly erect with no pods contacting the soil surface, 2 = semi-erect with some pods contacting the soil surface, and 3 = sprawling plant habit with much of the plant in contact with the soil surface. In addition to these observations, 20 plants were randomly collected from the center row of each triple row plot. These were individually rated for disease severity using a 0-5 scale corresponding to the plot rating scale.

GREENHOUSE STUDY

The same 30 cultivars planted in the field in 1982 and 1983 were evaluated in the greenhouse during the winter of 1982-83 for resistance to white mold. Seed of each cultivar was planted in 3 pots (18 cm diameter x 23 cm depth). Pots contained a soil mix of 1/3 soil, 1/3

sand, and 1/3 peat. A complete fertilizer blend including micronutrients was incorporated at the time the mix was made. After emergence, pots were thinned to 5 plants/pot. Pots were watered as necessary for vigorous growth. Temperatures were maintained at 24°C day and 16°C night. Fluorescent lights were utilized on a 12 hr day/12 hr night schedule. Prior to inoculation, plants were grown in the greenhouse for approximately 3 wks until the first trifoliate leaf expanded.

The disease screening procedure utilized was based on the limited term inoculation method developed by Hunter et al (26). Small pieces (2 x 2 mm) of whole or cut sclerotia were surface sterilized in a 5% solution of sodium hypochlorite for 30 sec and placed on petri plates containing potato dextrose agar. When actively growing cultures were obtained and before sclerotia production began, small pieces (4 x 8 mm) of commercially canned green bean pods were placed on the plates. After incubation for 24 hrs at 22°C, the bean pieces were colonized with cottony mycelium visible on the surface of the pods.

One colonized bean piece was placed on the stem just below the node of the primary leaf on each 3 wk old plant. At this time plants were placed in a mist chamber which was set to mist 30 sec every 5 min. Randomization occurred by subjectively placing each of the 3 pots of each cultivar in a different section of the mist chamber.

After 40 hrs the inoculum was removed from the plants and they were returned to the greenhouse. After 5 days the total number of plants with collapsed stems was recorded for each cultivar. This same procedure was repeated each week for 9 wks with the weeks being considered blocks in a randomized block design.

STATISTICAL ANALYSIS

Analysis of variance was performed on field and greenhouse disease scores. The arcsin transformation was used on the greenhouse data which is reported as percent collapsed plants. Correlation coefficients were computed where information about relationships between variables was desired. Spearman's rank correlation method (37) was used to give an indication of the relationship between the disease scoring system used both years and the greenhouse screening results. Pooled scores were obtained for each cultivar in the field trial using the method of Ostle (30). All statistical analysis was done using the SAS data analysis system.

RESULTS AND DISCUSSION

CULTIVAR FIELD TRIALS

Results of the 1982 field trials involving cultivars planted in 3-row plots in the disease and control areas are given in Table 2. Table 3 presents corresponding data from the disease area planted in 1983. To minimize the effect of disease avoidance due to maturity differences, values are from the observation date when each individual cultivar was judged to be at the green bean processing maturity stage. In all cases except plant weight, the mean value for each variable was significantly different ($P = 0.05$) between the two locations. Significant interactions between cultivar and location were not found.

As indicated by the triple row disease score (TRDS), disease severity in the control area was about 1/2 that of the disease area. At the lower severity level, neither the NIPL, PIP, nor the TRDS were significantly different among the 8 cultivars. PW and TNP did reflect significant cultivar differences in the control location. PW is a measure of biological yield and TNP is a component of this yield. It appears, therefore, that at the low disease level found in the control area these variables reflect normal cultivar differences for yield rather than disease reaction.

In the disease area, each of the 3 variables

Table 2. White mold disease reaction and yield measurements[z] for cultivars grown in high disease infection (disease) and low disease infection (control) areas in 1982.

Cultivar	Location[y]	TRDS	SRDS[x]	PW	TNP	PIP	NIPL
Aurora	Disease	3.0	3.0	1.4	124	6.5	6.5
	Control	2.0	---	1.2	179	0.8	0.8
Black Turtle Soup	Disease	1.9	1.8	1.1	130	0.6	1.3
	Control	1.3	---	1.1	128	0.0	0.0
Black Valentine	Disease	2.8	2.4	1.5	105	4.3	5.3
	Control	1.5	---	1.3	80	0.0	0.3
Ex Rico 23	Disease	2.4	2.3	1.1	112	1.8	1.0
	Control	1.4	---	1.8	212	0.0	0.0
Midnight	Disease	2.1	1.9	1.3	93	1.9	2.5
	Control	1.0	---	1.4	159	0.2	0.3
Oregon 1604	Disease	3.9	3.9	1.5	119	7.0	9.3
	Control	2.0	---	1.7	133	1.0	0.5
Pinto UI III	Disease	3.1	2.5	0.9	79	1.3	3.3
	Control	1.5	---	1.3	110	0.3	0.5
Red Kidney	Disease	2.1	1.9	1.4	80	0.4	1.5
	Control	1.0	---	1.3	73	0.0	0.3
	Disease $\bar{x}$ =	2.7*	2.5	1.3	106*	3.0*	3.8*
	Control $\bar{x}$ =	1.5	---	1.4	134	0.3	0.3
LSD (P=0.05)	Disease	0.6	0.8	NSD	NSD	4.4	2.7
	Control	NSD	---	0.4	39	NSD	NSD

[z] TRDS = triple row disease score; SRDS = single row disease score; PW = plant weight in kg/10 plant sample; TNP = total number pods/10 plant sample; PIP = percent infected pods/10 plant sample; NIPL = number infected plants/10 plant sample. For disease scores 0 = no symptoms, 5 = completely diseased plot.

[y] Location by cultivar interactions not significant.

[x] Single row plots not planted in control location.

* Significant location differences at 5% level.

Table 3. White mold disease reaction[z] of cultivars grown in high disease infection area in 1983.

Cultivar	TRDS	SRDS	ADS
Aurora	3.3	3.4	3.1
Black Turtle Soup	2.0	1.5	1.8
Black Valentine	3.5	2.6	3.2
Ex Rico 23	2.0	2.0	1.7
Midnight	2.9	1.4	2.6
Oregon 1604	3.6	3.8	3.2
Pinto UI III	4.4	4.1	4.2
Red Kidney	1.8	1.6	1.2
$\bar{x}$ =	2.9	2.6	2.6
LSD (P=0.05) =	1.1	1.0	0.9

[z] TRDS = triple row disease score; SRDS = single row disease score; ADS = average disease score of 20 plants rated individually. For disease scores: 0 = no symptoms, 5 = completely diseased plot or plant.

measuring disease reaction produced significant cultivar differences. PW and TNP were included in hopes of getting a measurement of yield loss at the 2 disease severity levels. However, in the diseased location where the cultivar differences were found for disease reaction, significant yield differences were not found. Although the average TNP in the control area was significantly greater than in the disease area ($P = 0.05$), a cultivar by cultivar comparison using the TRDS does not indicate a causal relationship of disease severity. In fact, 'Black Turtle Soup', 'Black Valentine', and 'Red Kidney' had higher TNP and TRDS values in the disease area than in the control area. PW differences were probably obscured by the irrigation practices in the diseased area which kept plants wet at all times including the sampling periods. This also may account for the lack of a significant difference between the average PW in the control area versus the disease area. These conclusions agree with those of Agbo and Wood (6) that variability in biological yield of beans reflect cultivar differences for this trait and are minimally affected by white mold disease severity

The average TRDS in the disease area versus the control area indicates the effectiveness of the windbreak and the irrigation practices in promoting disease initiation and development. However, the disease area may have contained more inoculum. In the disease area, symptoms

first became apparent 3 September 1982. The number of infection sites and the severity of symptoms increased rapidly until the first week in October when unfavorable conditions appeared to inhibit further disease and plant development. Although the disease did occur in the control area, the first symptoms appeared almost a week later. On 14 September 1982 the average TRDS in the disease area was 1.9 while in the control area it was 0.5. On 7 October 1982 it was 3.4 in the disease area and 2.1 in the control area. The single row disease scores (SRDS) for the 8 cultivars which were planted in 3-row and single row plots are included in Tables 2 and 3. This information along with the other data in these tables was used to develop the correlations given in Table 4. PIP and NIPL were positively correlated to TRDS. This gives some objective support to the reliability of the subjective rating system for evaluating differences in white mold disease reaction. In addition, a highly significant positive correlation was found between the TRDS and the SRDS in this trial. Apparently, interaction between various cultivars in adjacent single row plots was not sufficient to detectably affect disease reaction of cultivars.

In 1982 the cultivar field trial generally showed that: 1) disease reaction differences could not be detected at low levels of disease; 2) PW and TNP did not provide an indication of yield loss relative to disease

Table 4. Correlations between triple row disease score (TRDS)[z] of each year and other variables to measure disease reaction in field trials.

Variables Correlated with TRDS	Correlation Coefficient
1982	
Number of Infected Plants (NIPL)	0.74 **
Percent Infected Pods (PIP)	0.76 **
Single Row Disease Score (SRDS)	0.75 **
1983	
Single Row Disease Score (SRDS)	0.70 **
Average Disease Score (ADS)	0.96 **

[z] Scored only center row of triple row plot.

** Significant at 1% level.

severity level; 3) the use of frequent irrigation and a windbreak was an effective promoter of white mold disease initiation and development; 4) the 0-5 disease scoring scale was effective in detecting disease reaction differences among cultivars; and 5) significant cultivar by location interactions were not found. In addition, single row plots appeared to be adequate for disease reaction evaluation. However, since Fuller et al (21) did not find this to be true, it was decided to repeat the triple and single row planting scheme in 1983.

In 1983, the first disease symptoms appeared during the first week in August. Disease severity again increased rapidly until the 25 August scoring date. As in 1982, there was a positive correlation of 0.70 ($P = 0.01$) between the TRDS and SRDS (Table 4). In addition, there was a positive correlation of 0.96 ($P = 0.01$) between the ADS and TRDS of that plot. This agrees with the findings of Coyne et al (11) regarding individual plant infection ratings and overall disease reaction ratings on a row basis. This again supports the reliability of the rating system for measuring disease reaction differences.

Results of the 1982 and 1983 field tests involving single row plots are presented in Table 5 along with greenhouse test results. The 24 September 1982 scoring date was used for comparison to the 25 August 1983 date because analysis of variance between each of the five 1982

Table 5. White mold disease severity scores for all cultivars tested in 1982 and 1983 Oregon field and greenhouse trials.[z]

	1982		1983			Greenhouse	
Cultivar	Mean Score	Rank	Mean Score	Rank	Pooled[y] Rank	Mean Score	Rank
Astro	2.1	12	2.0	12	11	73.3	20
Aurora	1.6	4	3.4	27	17	77.4	25
B3749	1.9	11	2.4	18	13	19.7	1
B. Turtle S.	1.8	5	1.5	3	2	78.4	27
B. Valentine	2.9	27	2.6	22	27	63.3	13
Bountiful	2.1	13	1.9	8	9	62.9	12
BBL 53	2.3	15	3.3	26	21	69.9	19
Cape	1.9	9	1.9	6	7	53.4	5
Checkmate	2.5	21	2.0	10	16	69.4	17
Coloma	2.5	22	3.3	25	25	74.3	21
Contender	3.1	29	2.3	15	23	50.2	3
Ex Rico 23	2.3	16	2.0	11	12	54.9	7
Gabriella	1.0	1	1.5	4	1	78.3	26
Gallatin 50	2.3	14	2.5	20	19	69.7	18
Geneva 19-2	2.8	26	2.8	23	26	67.0	14
Green Crop	2.3	17	2.1	13	14	61.6	9
Harvester	1.4	2	2.3	16	8	77.3	24
Midnight	1.8	7	1.4	1	3	68.1	16
Orbit	1.9	10	1.4	2	4	76.3	23
Oregon 1604	4.1	30	3.8	29	30	81.4	29
Pinto UI III	2.8	24	4.2	30	28	80.6	28
Provider	3.0	28	3.4	28	29	67.7	15
Red Kidney	1.9	8	1.6	5	5	60.4	8
Roma	2.5	20	3.1	24	22	74.8	22
Taylor's Dwarf	2.4	19	2.1	14	18	54.3	6
Tendercrop	2.8	25	2.6	21	24	61.9	10
Tidal Wave	1.5	3	2.5	19	10	85.9	30
PI 169787	2.8	23	1.9	7	15	43.4	2
PI 204717	1.8	6	2.0	9	6	62.1	11
PI 415965	2.3	18	2.4	17	20	51.1	4
$\bar{x}$ =	2.3		2.4			65.6	
LSD (P=0.05) =	1.0		1.0			15.5	

[z] Field scores are means of 4 replications of single row plots. Greenhouse scores are percent of collapsed plants during 9 weeks of testing 15 plants/cultivar/week.

[y] Significant rank correlation coefficient (r=0.53, P=0.01) found between 1982 and 1983 field scores. Correlation between pooled field ranking and greenhouse ranking was non-significant.

dates and the single 1983 date produced a non-significant F-value (P = 0.05) only for this comparison. The 1983 scoring date was 80 days after planting while the 24 September 1982 date was 77 days from planting. The 1 month difference between these scoring dates corresponds to the 1 month difference in planting dates for the 2 years. Comparison of the mean disease score of 2.3 in 1982 and 2.4 in 1983 indicates the similarity of overall disease levels for the 2 years. A highly significant rank correlation coefficient of 0.53 (P = 0.01) was found between the 2 years' scores. This indicates a degree of repeatability of this method for evaluating cultivar disease reaction under these field conditions.

Rank correlation of the pooled field ranking and the greenhouse ranking failed to produce a significant value. Thus, there was poor agreement between the results of the field and greenhouse procedures. This was not totally unexpected because of the likelihood that in the field low disease levels in some cultivars are due to avoidance mechanisms. It is well documented that plant architecture (12,20,35) and maturity (14) can act as disease avoidance mechanisms. Placement of the inoculum directly on the stem of the plant as in the greenhouse screening procedure minimized the chance of disease avoidance. Results of the greenhouse trial should, therefore, give a better indication of disease resistance.

The identification of cultivars with higher levels of white mold disease resistance is the ultimate goal of both the field and greenhouse test. To do this, disease avoidance mechanisms have to be separated from disease resistance. The following discussion will attempt to make this separation using information regarding cultivar growth habit, canopy characteristics, and days from planting to processing maturity (Table 6) along with greenhouse and field observations. In this study, as in Dickson and Hunter's work (18) greenhouse screening results will be used as a measure of true disease resistance. The processing maturity disease score (Table 6) is included to aid in identification of disease avoidance due to differing maturity dates of cultivars. It attempts to compare cultivars based on disease reaction at a given physiological stage rather than a given date. Two groups of cultivars will be considered as potential parents in a breeding program. One group is made up of the cultivars which performed best in both years of field testing. The other includes cultivars which performed well in the greenhouse test but were not among the best performing cultivars in field tests.

Based on LSD values there was no significant disease reaction difference among cultivars ranked from 1-11 in 1982 field trials (Table 5). In 1983 this was true of cultivars ranked 1-16. Eight cultivars: 'Gabriella',

Table 6. Processing maturity disease score,[z] days to maturity, growth habit, and canopy index rating of cultivars in 1982 field trial.

Cultivar	Mean Score	Rank	Days to Maturity	Growth Habit[y]	Canopy Index[x]
Astro	1.6	11	71	1	3
Aurora	3.0	29	90	3	3
B3749	1.9	15	77	4	3
Black Turtle Soup	1.8	12	77	3	2
Black Valentine	2.9	28	77	1	3
Bountiful	1.9	14	71	1	3
BBL 53	1.5	4	71	1	3
Cape	1.5	7	71	1	3
Checkmate	2.0	22	71	1	3
Coloma	2.3	23	71	1	3
Contender	2.5	26	71	1	2
Ex Rico 23	2.3	25	77	3	3
Gabriella	0.8	1	71	1	1
Gallatin 50	2.3	24	77	1	1
Geneva 19-2	2.0	21	71	1	3
Green Crop	2.0	20	71	1	3
Harvester	1.0	2	71	1	2
Midnight	1.9	19	90	2	2
Orbit	1.5	6	71	1	3
Oregon 1604	3.5	30	71	1	3
Pinto UI III	1.5	8	71	3	3
Provider	2.8	27	71	1	3
Red Kidney	1.9	18	77	1	3
Roma	1.9	17	71	1	3
Taylor's Dwarf	1.6	9	71	1	3
Tendercrop	1.9	16	71	1	1
Tidal Wave	1.5	5	77	1	3
PI 169787	1.8	13	71	1	3
PI 204717	1.1	3	71	1	2
PI 415965	1.6	10	71	1	3
LSD (P=0.05)	0.8				

[z] Mean disease score for each cultivar on day it reached green bean processing maturity.

[y] 1 = determinate; 2 = indeterminate, weakly vining; 3 = indeterminate, moderately vining; 4 = indeterminate, strongly vining.

[x] 1 = strongly erect plant, no pods contacting soil surface; 2 = semi-erect plant, some pods contacting soil surface; 3 = sprawling plant, much of plant in contact with soil surface.

'Harvester', 'Black Turtle Soup', PI 204717, 'Midnight', 'Red Kidney', 'Cape', and 'Orbit', were in both these groups and are ranked from 1-8 by the pooled ranking system. These were the best performers in the field trial.

The LSD value of 0.80 (P = 0.05) for the mean disease score at processing maturity indicates the cultivars ranked 1-8 showed no significant difference in performance 'Gabriella', 'Harvester', PI 204717, 'Cape' and 'Orbit' fall into this category. Each of these reached the processing maturity stage in 71 days. 'Black Turtle Soup', 'Midnight', and 'Red Kidney' are later maturing cultivars and do not fall into this category. Late maturity acted as an avoidance mechanism in these trials because fewer senescing flowers were available for colonization prior to the 24 September 1982 and 25 August 1983 scoring dates than for the earlier maturing cultivars. Although the season was not long enough for full possible expression of susceptibility, the 1982 processing maturity score may be a more accurate assessment of these three cultivars' field performance.

'Black Turtle Soup' is an indeterminate dry-edible bean. It is a moderately vining cultivar with a semi-erect plant habit. It was observed that the canopy of this cultivar is dense and intertwined but does not cling to the soil surface. Rather, a tunnel for air movement

seems to be formed by the plants of this cultivar. Fuller et al (20) reported that this type of plant architecture can contribute to disease avoidance by enhancing air circulation, thereby preventing prolonged periods of leaf wetness. Although Anderson et al (7) and Coyne et al (13) reported this cultivar to be highly resistant to white mold infection, Hunter et al (25) reported a similar lack of resistance in greenhouse testing to that indicated in this study by the rank of its greenhouse score in Table 5. Therefore, 'Black Turtle Soup' appears not to possess true disease resistance but rather its late maturity and plant architecture acted as disease avoidance mechanisms in these field trials.

'Midnight' is a dry-edible bean with a semi-erect plant habit. Although it is indeterminate, it has a weak vining tendency. Air circulation is probably not seriously impaired in the canopy. It was 1 of the 2 latest maturing cultivars in the trial. For both the 1982 and 1983 scoring dates it had not yet reached processing maturity. It gave intermediate results in the greenhouse but its performance in the field was apparently affected by very late maturity and a plant architecture favoring disease avoidance. Its processing maturity disease score supports this observation.

'Red Kidney' was a determinate dry-edible bean with a sprawling plant habit in this trial. In this respect it

is similar to most green beans grown for processing. However, it is later maturing by approximately 7 days. This fact appeared to contribute to its strong field performance. Yet it did display a high level of disease resistance in the greenhouse test. One other factor which should be considered in future trials and is applicable to evaluation of 'Red Kidney' is the effect of irrigation practices on plant habit and how this effect might alter disease reaction. For example, the dry-edible bean cultivar 'Aurora' was reported to have resistance to white mold (7,13). Studies by Blad et al (9) and Weiss et al (44) demonstrate the effect of irrigation on the disease reaction of 'Aurora'. More frequent irrigation changes the plant habit of 'Aurora' from an upright to a sprawling vine. This results in conditions more conducive to white mold infection. In their studies, 'Aurora' was very susceptible to infection when more frequent irrigation practices were used. Thus, it should be recognized that environmental conditions imposed to favor the development of white mold disease may also modify the growth characteristics of the bean plant resulting in increased susceptibility to infection. Although 'Red Kidney' normally has an upright plant habit, the frequent irrigation apparently changed its plant habit to a sprawling vine which is more susceptible to white mold infection. However, of the 3 later maturing cultivars under discussion 'Red Kidney'

appears to have the most potential for use in a breeding program for white mold disease resistance.

'Gabriella', 'Harvester', PI 204717, 'Cape', and 'Orbit' all reached processing maturity in 71 days. Each is determinate and each one performed very well in the field. However, 'Gabriella', 'Harvester', and 'Orbit' displayed little to no disease resistance in greenhouse testing. 'Gabriella' is a wax pod bean with a very erect plant habit. In the field trial it was a very small plant. 'Harvester' is a green bean with a semi-erect plant habit. It has a very sparse canopy. The plant architecture of both these cultivars appeared to act as disease avoidance mechanisms by enhancing air circulation thereby preventing prolonged periods of leaf wetness.

This type of reasoning does not explain the field performance of 'Orbit', which appears to have a poor plant architecture for disease avoidance. It is determinate and matures in 71 days. It was moderately susceptible in the greenhouse yet showed high levels of resistance in the field. However, it was observed in the field that its branches tend to fall to either side of the row leaving the center of the plant canopy open. Studies by Schwartz et al (35) indicate these canopy characteristics may also act as disease avoidance mechanisms by allowing air circulation and light penetration, thus inhibiting prolonged periods of leaf wetness.

'Cape' and PI 204717 appear to have potential as parents in a breeding program. Both showed resistance in both field and greenhouse tests. PI 204717 has the added advantage of having a more favorable plant habit for disease avoidance. Of the 8 cultivars which performed best in both years of field testing, greenhouse scores indicate 'Cape', PI 204717, and 'Red Kidney' appear to have done so due primarily to resistance to white mold rather than avoidance of the disease. This result supports use of the greenhouse screening procedure as an aid in identifying levels of resistance.

There were 6 entries, B3749, PI 169787, 'Contender', PI 415965, 'Taylor's Dwarf', and 'Ex Rico 23', which performed very well in greenhouse testing but not as well in field testing. Based on greenhouse performance each of these should be considered as having some potential for use in a breeding program for white mold resistance. With the exception of PI 415965, LSD values show each was among the top entries in 1 of the 2 years of field testing. PI 415965 was intermediate in performance both years. B3749, a *P. coccineus* selection, is an indeterminate, strongly vining runner bean with a sprawling plant habit. It displayed by far the highest level of resistance in greenhouse testing. 'Ex Rico 23' is a dry edible bean which Tu and Beversdorf (42) reported as resistant. It showed considerable resistance in the greenhouse test.

Although it also has an intermediate, sprawling plant habit, it does not vine as strongly as B3749. This characteristic appeared to result in considerable susceptibility when plants are exposed to the frequent irrigation practices used. Dickson and Hunter (16) report that under very severe disease pressure even lines which are thought to be resistant will succumb to the disease. PI 169787, PI 415965, and 'Taylor's Dwarf' are all determinate, sprawling plants which provide very conducive conditions for infection. Apparently the levels of resistance found in greenhouse tests were not strong enough to withstand the disease pressure encountered in the field. Like PI 204717, 'Contender' has the advantage of a semi-erect plant habit. Although it did poorly in the field the first year, it performed well in 1983.

Of the 30 cultivars studied, the following appear to possess useful levels of disease resistance: 'Cape', PI 204717, 'Red Kidney', B3749, PI 169787, 'Contender', PI 415965, 'Taylor's Dwarf', and 'Ex Rico 23'. Dickson et al (16,18,19) have tested PI 204717, B3749, PI 169787, and PI 415965 and report similar results. 'Ex Rico 23' was reported resistant by Tu and Beversdorf (42). Each of these 9 lines shows potential for use in a breeding program for white mold resistance.

FAMILY FIELD TRIAL

Table 7 presents results of field screening for F_3 families resulting from 8 different crosses. At the time these crosses were made, 'Aurora', and 'Black Turtle Soup' were thought to possess resistance while the other 4 lines were considered susceptible under the conditions of the field test. The range of scores which was found among the families of each respective cross indicates potential for selection. The range of scores in each cross exceeded both the high and low parental scores. This apparent transgressive segregation suggests that the diversity of combinations of growth habit and other characteristics which resulted from these crosses may have included some which were more, or less conducive to white mold infection. Families of each cross appear to approximate a normal distribution for disease score indicating quantitative inheritance (Table 8). This agrees with previous reports indicating white mold resistance is a quantitatively inherited trait (6,19,33). Because of the small differences in disease scores found in our tests between the parents used in these crosses, the variations which occurred in the large plot area required to grow the F_3 families, and the apparent interaction between plant habit and the extreme test environment (excess irrigation), it was not considered possible to obtain further information

Table 7. White mold disease reaction of F_3 families screened in 1982 field trials.

Parents[z]				F_3 Families				
P1		P2				Disease Score		
Cultivar	Score[y]	Cultivar	Score[y]	Number Families[x]	Mean Days to Maturity[w]	Mean[v]	Standard Deviation	Range of Scores[u]
Aurora	3.0	Oregon 1604	3.5	50	76	2.6	0.67	1.5-3.8
B. Turtle S.	1.8	Oregon 1604	3.5	45	76	2.3	0.68	1.5-3.8
Aurora	3.0	Gallatin 50	2.3	35	76	2.3	0.62	1.5-3.8
B. Turtle S.	1.8	Gallatin 50	2.3	50	76	2.0	0.51	1.0-3.0
Aurora	3.0	Midnight	1.9	54	91	2.8	0.74	1.5-4.0
B. Turtle S.	1.8	Midnight	1.9	56	91	2.5	0.69	1.5-4.3
Aurora	3.0	Red Kidney	1.9	18	76	2.3	0.54	1.5-2.8
B. Turtle S.	1.8	Red Kidney	1.9	43	76	1.9	0.53	1.0-2.5
Check (Oregon 1604)[t]					71	3.1	0.70	2.5-4.3
						4.4	0.84	3.5-5.0

[z] Parents were planted in the disease field plot area with F_3 families located in the 2 areas adjacent to each side.

[y] Disease scores of parents when each was at green bean processing maturity stage.

[x] Number of families derived from random single plants in the F_2.

[w] Average of all families from each cross.

[v] Mean disease score for families from each cross, derived from the average of 2 replications of each family.

[u] Range of mean scores over the 2 replications for families of each respective cross.

[t] Check was planted 9 times in each of the 2 blocks. Although it reached processing maturity in 71 days, scores reported are from dates corresponding to 76 and 91 days to maturity for comparison to families.

Table 8. Distribution of F_3 families by 1982 field disease score.

Pedigree	Disease score[z] 1.00	1.25	1.50	1.75	2.00	2.25	2.50	2.75	3.00	3.25	3.50	3.75	4.00	4.25
Aurora x Oregon 1604			2[y]	1	2	15	6	9	10	3		2		
B. Turtle Soup x Oregon 1604			3	4	14	8	6	7	1		1	1		
Aurora x Gallatin 50			3	1	10	8	6	5	1	1				
B. Turtle Soup x Gallatin 50	1		10	7	15	12	2	2	1					
Aurora x Midnight			1	1	2	11	9	3	11	7	5	3	1	
B. Turtle Soup x Midnight			3	2	6	8	15	11	5	3	2			1
Aurora x Red Kidney			2		4	3	7	2						
B. Turtle Soup x Red Kidney	2	2	8	6	12	7	6							

[z] 0 = no symptoms, 5 = completely diseased plot.

[y] Number of families in each disease score category.

on the inheritance of resistance. However, it is interesting to note that the 2 parents with the highest level of disease resistance based on greenhouse tests, 'Red Kidney' and 'Gallatin 50,' when crossed with the female with the best disease avoidance mechanisms, 'Black Turtle Soup', produced F_3 families with the best overall mean score and individual families exhibiting the best resistance in this field test. It would be interesting to evaluate these families for true disease resistance in the greenhouse test.

As selections are advanced in a breeding program the field and greenhouse screening procedure used in this study should be useful for identification of superior lines with white mold resistance. Selections which have disease resistance and a favorable plant architecture for disease avoidance should have the most potential for satisfactory performance under disease pressure.

SUMMARY AND CONCLUSIONS

The use of a windbreak to restrict air circulation and frequent sprinkler irrigation to prolong periods of leaf wetness proved to be effective promoters of white mold disease initiation and development in 1982 and 1983 field trials. Comparison of results from the high infection area and the low infection (control) area in the 1982 field tests indicate that relatively high levels of disease severity are required for differentiation of disease reaction among cultivars. Percent infected pods, number infected plants, average individual plant disease scores, single row disease scores, and triple row disease scores all proved effective at distinguishing disease reaction among cultivars. The disease score from triple row plots was positively correlated with each of these measurements of disease reaction thus providing support for the effectiveness of the disease scoring system. Plant weight and total number of pods were not effective indicators of yield loss at different disease severity levels. Since single row and triple row disease scores were positively correlated, there did not appear to be sufficient interaction between cultivars in adjacent single row plots to detectably affect disease reaction in this study.

Rank correlation of the field scores for each year

gave a highly significant positive correlation between these scores. However, no such correlation was found between the greenhouse results and the pooled field scores. This apparent inconsistency can possibly be resolved when data concerning plant architecture and days from planting to maturity are used in an attempt to identify disease avoidance mechanisms as opposed to true disease resistance. The greenhouse screening procedure appeared to be an effective aid in identification of cultivars with useful levels of resistance. Results of this study indicate that the field and greenhouse screening procedures utilized should be effective tools for identifying superior selections in a breeding program.

Nine of the 30 cultivars studied appear to possess useful levels of disease resistance for use as parents in a breeding program. Five of these lines have been tested by other researchers with similar results reported.

Results of screening of F_3 families of 8 crosses showed variability for disease reaction and apparent transgressive segregation among the families in each respective cross indicating potential for selection and further screening. Furthermore, this variability appeared to be normally distributed within each cross indicating white mold disease resistance is a quantitatively inherited trait.

LITERATURE CITED

1. Abawi, G.S., R. Providenti, D.C. Crosier, and J. E. Hunter. 1978. Inheritance of resistance to white mold disease in Phaseolus coccineus. J. Heredity 69:200-202.

2. Abawi, G.S., and R.G. Grogan. 1975. Source of primary inoculum and effects of temperature and moisture on infection of beans by Whetzelinia sclerotiorum. Phytopathology 65:300-309.

3. Abawi, G.S., R. Providenti, R.G. Grogan, and J.E. Hunter. 1975 Predisposition of beans to infection by ascospores of Whetzelinia sclerotiorum prior to blossoming. (Abstr.) Proc. Am. Phytopathol. Soc. 2:61.

4. Adams, P.B. 1975. Factors affecting survival of Sclerotinia sclerotiorum in soil. Plant Dis. Reptr. 59:599-603.

5. Adams, P.B., C.J. Tate, R.D. Lumsdem, and J.P. Meiners. 1973. Resistance of Phaseolus species to Sclerotinia sclerotiorum. Annu. Rep. Bean Improv. Coop. 16:8-9.

6. Agbo, F.M.O., and D.R. Wood. 1979. Inheritance of resistance to white mold in beans (Phaseolus vulgaris, L.), Agronomy Abstracts p54.

7. Anderson, F.N., J.R. Steadman, D.P. Coyne, and H.F. Schwartz. 1974. Tolerance to white mold in Phaseolus vulgaris dry edible bean types. Plant Dis. Reptr. 58:782-784.

8. Baggett, J.R. and M. Hyer. 1982. Field tests of bean lines and cultivars for susceptibility to white mold. Annu. Rep. Bean Improv. Coop. 25:24-25.

9. Blad, B.L., J.R. Steadman, and A, Weiss. 1978. Canopy structure and irrigation influence white mold disease and microclimate of dry edible beans. Phytopathology 68:1431-1437.

10. Cline, M., and B.J. Jacobsen. 1983. Methods for evaluating soybean cultivars for resistance to Sclerotinia sclerotiorum. Plant Disease 67:784-786.

11. Coley-Smith, J.R., and R.C. Cooke. 1971. Survival and germination if fungal sclerotia. Annu. Rev. Phytopathol. 9:65-92.

12. Coyne, D.P., J.R. Steadman, and F.N. Anderson. 1974. Effect of modified plant architecture of great northern dry bean varieties (Phaseolus vulgaris) and white mold severity, and components of yield. Plant Dis. Reptr. 58:379-382.

13. Coyne, D.P., J.R. Steadman, and H.F. Schwartz. 1977. Reaction of Phaseolus dry bean germplasm to Sclerotinia sclerotiorum. Plant Dis. Reptr. 61:226-230.

14. Coyne, D.P., J.R. Steadman, and H.F. Schwartz. 1977. Inheritance and breeding strategy for white mold disease (Sclerotinia sclerotiorum) resistance and avoidance in beans (Phaseolus vulgaris L.). HortScience 12:397 (abstract).

15. Dickson, M.H. and J.E. Hunter. 1982. Progress in breeding for white mold resistance. Annu. Rep. Bean Improv. Coop. 25:100-101

16. Dickson, M.H. and J.E. Hunter. 1982 Sources of white mold resistance and breeding for resistance. Annu. Rep. Bean Improv. Coop. 25:104-105.

17. Dickson, M.H. and J.E. Hunter. 1983. Modifications of methods for screening for white mold resistance in the greenhouse. Annu. Rep. Bean Improv. Coop. 26:85-86.

18. Dickson, M.H., and J.E. Hunter, M.A. Boettger, and J.A. Cigna. 1982. Selection for resistance in Phaseolus vulgaris L. to white mold disease caused by Sclerotinia sclerotiorum (Lib.) de Bary. J. Amer. Soc. Hort. Sci. 107:231-234.

20. Fuller, P.A., J.R. Steadman, and D.P. Coyne. 1984. Enhancement of white mold avoidance and yield in dry beans by canopy elevation. HortScience 19:78-79

21. Fuller, P.A., D.P. Coyne, J.R. Steadman, and R.F. Mumm. 1984. Inter- and intra-row inter-genotypic competition influences selection avoidance of white mold disease in dry edible beans. J. Amer. Soc. Hort. Sci. 109(4):567-572.

22. Hagedorn, D.J. and R.E. Rand. 1983. Reaction of

selected bean lines to white mold (Sclerotinia sclerotiorum). Annu. Rep. Bean Improv. Coop. 26:67-68.

23. Halkilahti, A. -M. 1962. The survival of sclerotia of Sclerotinia trifoliorum Erikss. on the soil and the occurrence of clover rot in various years. Suomen Maataloustiet. Aikakausk. 34:154-161.

24. Hunter, J.E., G.S. Abawi, D.C. Crosier. 1978. Effects of timing, coverage, and spray oil on control of white mold of snap bean with benomyl. Plant Dis. Reptr. 62:633-637.

25. Hunter, J.E., M.H. Dickson, M.A. Boettger, and J.A. Cigna. 1982. Evaluation of plant introductions of Phaseolus spp. for resistance to white mold. Plant Disease 66:320-322.

26. Hunter, J.E., M.H. Dickson, and J.A. Cigna. 1981. Limited-term inoculation: a method to screen bean plants for partial resistance to white mold. Plant Disease 65:414-417.

27. Kerr, E.D., J.R. Steadman, and L.A. Nelson. 1978. Estimation of white mold disease reduction of yield and yield components of dry edible beans. Crop Science 18:275-279.

28. Kuehn, M. and J.R. Baggett. 1983. Field Evaluations for white mold resistance. Annu. Rep. Bean Improv. Coop. 26:22-23.

29. Mattusch, P., M. Gerlagh, A. Ester, and G. Spikman. 1982. Screening of snap bean cultivars for resistance to Sclerotinia sclerotiorum. Annu. Rep. Bean Improv. Coop. 25:48-50.

30. Ostle, Bernard. Statistics in Research. Iowa: Iowa State College Press, 1954.

31. Purdy, L.H. 1979. Sclerotinia sclerotiorum: history, diseases, and symptomatology, host range, geographic distribution, and impact. Phytopathology 69:875-880.

32. Purdy, L.H. 1958. Some factors affecting penetration and infection by Sclerotinia sclerotiorum. Phytopathology 48:605-609.

33. Roberts, M.E., M.H. Dickson, and J.E. Hunter. 1982. Heritability of white mold resistance. Annu. Rep. Bean Improv. Coop. 25:104

34. Schwartz, H.F. and J.R. Steadman. 1978. Factors affecting sclerotium populations of, and apothecium production by, Sclerotinia sclerotiorum. Phytopathology 68:383-388.

35. Schwartz, H.F., J.R. Steadman, and D.P. Coyne. 1978. Influence of Phaseolus vulgaris blossoming characteristics and canopy structure upon reaction to Sclerotinia sclerotiorum. Phytopathology 68:465-470.

36. Singh, S.P. 1982. A key for identification of different growth habits of Phaseolus vulgaris L. Annu. Rep. Bean Improv. Coop. 25:92-95.

37. Snedecor, George W., and William G. Cochran. Statistical Methods. Iowa:Iowa State University Press, 1980.

38. Steadman, J.R. 1979. Control of plant diseases caused by Sclerotinia species. Phytopathology 69:904-907.

39. Steadman, J.R. 1979. White mold - a serious yield limiting disease of beans. Plant Disease 67:346-350.

40. Steadman, J.R., D.P. Coyne, and G.C. Cook. 1973. Reduction of severity of white mold disease on great northern beans by wider row spacing and determinate growth habit. Plant Dis. Reptr. 57:1070-1071.

41. Suzui, T., and T. Kobayashi. 1972. Dispersal of ascospores of Sclerotinia sclerotiorum (Lib.) de Bary on kidney bean plants. Part 1. Dispersal of ascospores from a point source of apothecia. Pages 137-151 in Hokkaido Nat. Agric. Exp. Stn. Bull. 101 (English Summary).

42. Tu, J.C. and W.D. Beversdorf. 1982. Tolerance to white mold (Sclerotinia sclerotiorum (Lib.) de Bary in Ex Rico 23, a cultivar of white bean (Phaseolus vulgaris L.). Can. J. Plant Sc. 62:65-69.

43. U.S. Department of Agriculture Crop Reporting Board, Statistical Reporting Service. Vegetables. Vg. 1-2 (81) Annual Summary, Dec. 28, 1982.

44. Weiss, A., L.E. Hipps, B.L. Blad and J.R. Steadman. 1980. Comparison of within - canopy microclimate and white mold disease (Sclerotinia sclerotiorum) development in dry edible beans as influenced by canopy structure and irrigation. Ag. Meteorology 22:11-21.