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EPIDEMIOLOGY OF WHITE MOLD DISEASE (*Sclerotinia sclerotiorum*)= (*Whetzelinia sclerotiorum*) OF DRY EDIBLE BEANS (*Phaseolus vulgaris*) WITH EMPHASIS ON RESISTANCE AND HOST ARCHITECTURAL DISEASE AVOIDANCE MECHANISMS

by

Howard Frederick Schwartz

A DISSERTATION

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For the Degree of Doctor of Philosophy

Department of Botany (Plant Pathology)

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TITLE

Epidemiology of white mold disease (Sclerotinia sclerotiorum) = (Whetzelinia sclerotiorum) of dry edible beans (Phaseolus vulgaris) with emphasis on resistance and host architectural disease avoidance mechanisms.

BY

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GRADUATE COLLEGE **UNIVERSITY OF NEBRASKA**

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PREFACE

This dissertation deviates from the conventional thesis style by its division into a general introduction and literature review, two sections and six appendices. Section one and two are to be submitted to Phytopathology for publication, and some data presented in the appendices may be incorporated into future publications.

ACKNOWLEDGEMENTS

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GENERAL INTRODUCTION AND LITERATURE REVIEW

Sclerotinia sclerotiorum (Lib.) de Bary = Whetzelinia sclerotiorum (Lib.) Korf and Dumont (Korf and Dumont, 1972) is distributed world-wide. Although it is prevalent in the temperate zones of the northern hemisphere, S. sclerotiorum can be a problem in areas with tropical or arid climates such as India and the Middle East, especially during cool seasons or under favorable microclimatic conditions (Reichert and Palti, 1967). The fungus is established in dry bean and vegetable fields at higher elevations near Bogota, Colombia, and at locations near the Peruvian coastline in South America (G. Galvez, personal communication).

Sclerotinia sclerotiorum is pathogenic on a wide range of host plants. Dickson was reported (Partyka and Mai, 1962) to have claimed that 172 species of 118 genera in 37 plant families were susceptible hosts. A recent report (Adams et al., 1974) broadened this range to 45 families with 130 genera and 190 plant species. A thorough review of the world literature by this author revealed 399 host plants (unconfirmed reports in some instances) in 374 species of 237 genera in 65 families (Appendix I). The signs or symptoms of susceptibility include blossom rot of fruit trees, storage rot of vegetables and white mold of beans.

The white mold fungus was studied in Europe as early as 1837 and in the United States on lettuce by 1890 (Ramsey, 1925). It has caused a problem on dry beans in northeastern Colorado (Burke et al., 1957), Florida, Idaho, Montana, New York (Blodgett, 1946) and Wyoming (Starr et al., 1953) since the early 1940's. Undoubtedly S. sclerotiorum was

present in western Nebraska at this time also (Cook, 1973). White mold was not of great concern to Nebraska growers until the late 1960's. Evidently it took this long for the fungus to become established at significant levels within the majority of fields located throughout the North Platte Valley.

Taxonomy

Taxonomy of the fungus causing white mold of dry edible beans is in a confused state involving genus and species names. Purdy (1955) combined Sclerotinia sclerotiorum (Lib.) de Bary, S. trifoliorum Ericks., S. minor Jagger, and other closely related species into S. sclerotiorum. This grouping was based upon morphological criteria which showed as much variability between species as was inherent in single ascospore isolates within the individual species. Other workers also have observed variation in cultural characteristics, apothecial production and virulence between single ascospore isolates (Coe, 1944; Morgan, 1952; Morrall et al., 1972; Newton et al., 1973; Price and Colhoun, 1975; Young, 1936).

However, other comparative analyses have repeatedly demonstrated and confirmed the presence of three major species. S. sclerotiorum, S. trifoliorum and S. minor differ in ontogenetic, polyacrylamide gel electrophoretic, cytological and pathogenicity-host range studies (Held and Haenseler, 1953; Willetts and Wong, 1971 and 1974; Wong and Willetts, 1973, 1975a and 1975b). Isolates of S. sclerotiorum and S. trifoliorum are more closely related to each other than either is to S. minor.

The generic status of S. sclerotiorum is also controversial.

Since international botanical nomenclature rules were not adhered to in earlier mycological work by Professor Whetzel, the genus Sclerotinia was declared an invalid designation for S. sclerotiorum (Korf and Dumont, 1972). In honor of Whetzel's mycological accomplishments, Korf and Dumont (1972) proposed a new genus Whetzelinia for Sclerotinia sclerotiorum. They also alluded to potential species differentiation within Whetzelinia based upon observed differences in seasonal appearance of apothecia in nature and diverse requirements for growth.

Other mycologists (Buchwald and Neergaard, 1972; Dennis, 1974) have argued for retention of the binomial S. sclerotiorum because of its notoriety. The name Sclerotinia sclerotiorum is used in this dissertation.

Sclerotium Formation

Sclerotinia sclerotiorum initiates sclerotial formation in 3-day-old cultures when liquid droplets or exudate appear on the aerial mycelium. On day 4 or 5, a sclerotium can be distinguished as a white mycelial clump covered by the liquid droplets. After seven days the sclerotium turns black, and the liquid droplets decrease in volume. The thin hyaline membrane surrounding the droplets persists during and after maturation. These liquid droplets decrease in size due to evaporation and may be partially absorbed by the sclerotium and metabolized (Colotello, 1973). The droplets contain potassium, sodium and magnesium ions, ammonia, amino acids, lipids, proteins and various enzymes (Colotello et al., 1971). The sclerotium itself also contains D-arabitol, D-mannitol, erythritol, fructose, glucose, glycerol and trehalose (Le Tourneau, 1966).

A mature sclerotium consists of a darkly pigmented rind 2-3 cells thick, a thin-walled hyphal cortex 2-4 cells thick and an inner medulla composed of loosely arranged filamentous hyphae (Kosasih and Willetts, 1975). The rind wall is constructed from β-glucose, chitin and melanin (Jones, 1970). A freshly produced sclerotium may contain 70% water. A sclerotium can still germinate myceliogenically on a nutrient medium if its water content is lowered to only 1% (Trevethick and Cooke, 1973).

A sclerotium forms on infected host tissue commonly in infection centers within a field where the moisture, temperature, plant canopy and fungus favorably interact (Coley-Smith and Cooke, 1971; Cook, 1973). A recently-formed mature sclerotium may be dislodged from host tissue onto the soil surface by the wind or during harvesting and threshing operations. Subsequent cultural practices - fall discing to incorporate bean plant residue, soil ridging to reduce wind erosion, spring discing and plowing to prepare the seedbed - redistribute sclerotia uniformly within the vertical soil profile throughout a field (Cook, 1973; Cook et al., 1975). Sclerotia can be distributed between fields by irrigation runoff water (Brown and Butler, 1936; Steadman et al., 1975). The fungus can also be distributed throughout a bean producing area within sclerotia- or mycelia-infested seed lots (Blodgett, 1946; Hungerford and Pitts, 1953; Steadman, 1975).

Sclerotium Survival

Integrity of the sclerotial rind is very critical to survival. S. sclerotiorum sclerotia produced on infected plants commonly have extensively fractured rind cells (Colotello, 1973; Merriman, 1976) which

leak nutrients and provide sites of entry for degradative and antagonistic microorganisms (Smith, 1972). A sclerotium, however, is capable of regenerating a rind if the original one is destroyed or removed (Jones, 1970; Saito, 1973). Other factors which affect sclerotial survival are soil texture and pH which in turn are affected by the frequency of soil wetting and drying (Merriman, 1976), soil temperature and moisture (Adams, 1975; Cook et al., 1975), the weakened structural condition of a sclerotium after apothecial production (Williams and Western, 1965b) and soil fumigation (Jones, 1974; Partyka and Mai, 1962).

Sclerotia are the primary survival structures of the white mold fungus. In western Nebraska 78% of the sclerotia buried at various depths under fallow conditions can survive for at least 3 years, and 72% of these sclerotia can then germinate to form stipes on water agar (Cook et al., 1975). However, Smith (1972) reported that sclerotia do not survive longer than 2 or 3 weeks when they have been dried and then remoistened. This decreased survival is apparently due to stimulation of myceliogenic germination and/or nutrient leakage, and the subsequent loss of sclerotial integrity caused by microbial degradation (Adams, 1975; Merriman, 1976; Smith, 1972).

Sclerotia of S. sclerotiorum can produce secondary sclerotia in field soil, and this can prolong survival (Adams, 1975; Cook et al., 1975; Williams and Western, 1965b). Furthermore, sclerotia can increase their weight by the absorption and metabolism of organic nutrients in the soil either directly through the sclerotial rind or through a mycelial intermediary originating from the sclerotium

(Williams and Western, 1965b).

Sclerotial Germination

A sclerotium of S. minor may germinate by one of three methods (Adams and Tate, 1976): (1) Carpogenic germination, by which stipes and/or apothecia are produced; (2) Hyphal germination, by which individual hyphae emerge through the rind (observed only on artifical media); and (3) Myceliogenic germination, by which dense mycelium erupts from beneath the rind. This latter method of germination allows the fungus to grow to 2-3 mm, and directly infect actively growing plant tissue without the prior colonization of senescent tissue. A soil dormancy period of 8-15 weeks, depending upon the particular isolate, is required before the sclerotium can germinate myceliogenically.

A sclerotium of S. sclerotiorum only germinates by methods 1 and 3. However, there is no dormancy period associated with myceliogenic germination and the fungus must colonize senescent plant tissue before infecting a healthy host plant. Mycelium can extend 2 cm from the sclerotium before exhausting its potential for colonization (Newton and Sequeira, 1972).

Sclerotia of this homothallic fungus must undergo a conditioning process to remove their constitutive dormancy which can persist for 18-210 days (Cook et al., 1975), depending upon the isolate (Ramsey, 1925). The process is not completely understood. However, fluctuating temperatures of 7-21 C (Sproston and Pease, 1957), 10-15 C (Hawthorne, 1973), 14-20 C (Henson and Valleau, 1940) and 13-22 C (Ramsey, 1925) and constant temperatures of 11 C (Abawi and Grogan, 1975), 14 C

(Kosasih and Willetts, 1975), 15 C (Abawi and Grogan, 1975; Purdy, 1956), 18-20 C (Letham, 1976) and less than 21 C (Brooks, 1940) are reported to stimulate this conditioning process and subsequent carpogenic germination. An initial drying of sclerotia may delay or inhibit carpogenic germination even after a subsequent incubation under near-optimal conditions for 2-3 months (Abawi and Grogan, 1975). Cook et al., (1975) believe that moisture fluctuation may stimulate carpogenic germination by buried sclerotia in western Nebraska. Soil moisture levels of 15-50% reportedly favor carpogenic germination (Morrall, 1976). However, Abawi and Grogan (1975) claim that the soil moisture must reach near-saturation for an unspecified time before germination can occur.

Sclerotia produced on infected dry beans in western Nebraska undergo this conditioning process after exposure to unknown subsurface factors during fall to spring burial (Cook et al., 1975). Freshly-produced sclerotia can be conditioned by burial in 5-7 cm greenhouse soil in large metal flats (50 x 35 x 9 cm deep) with good drainage, which are then incubated at 13-18 C and lightly watered once a week for at least 12 weeks (Schwartz and Steadman, unpublished method).

Before carpogenic germination occurs there must be an initiation of meristematic cell clusters (at the circumference of the medulla) which develop into the stipe primordia (Saito, 1973). Histochemical studies by Kosasih and Willetts (1975) revealed that a reserve of soluble proteins and glycogen form at the base, and ribonucleic acid concentrates at the tip of the stipe primordium. The stipe primordium ruptures the rind, elongates and then upon exposure to light, differ-

entiates into the mature disc or apothecium. The apothecium now contains high quantities of glycogen and proteins, and paraphyses serve as storage centers of glycogen and other energy sources subsequently utilized for development of asci and ascospores.

Although rarely observed, S. sclerotiorum can produce microconidia at any stage of its life cycle under proper conditions. These microconidia, however, have not been observed to function during sexual fertilization or host infection (Kosasih and Willetts, 1975; Ramsey, 1925). Ramsey (1925) observed microconidial germination and obtained cultures originating from aggregates of germinating microconidia but never obtained a culture due to a single germinating microconidium.

Apothecial Production

When microclimatic conditions including soil moisture and temperature are adequate, carpogenic germination occurs in fields of dry bean, corn, sugar beet (Schwartz and Steadman, 1976), snap bean (Abawi and Grogan, 1975), cauliflower, tomato (Letham et al., 1976), lettuce (Newton and Sequeira, 1972) and table beet, as well as grassland (Suzui and Kobayashi, 1972b), and lemon, orange (Smith, 1916) and other fruit orchards (Abawi and Grogan, 1975).

Apothecial production is influenced by depth of the sclerotia in the soil, since more apothecia develop from the 1 cm level than from lower depths (Kruger, 1975; Williams and Western, 1965a) and no apothecia develop from sclerotia buried below 7.5 cm (Brooks, 1940). A cover crop such as clover can increase the initial development of stipes (Williams and Western, 1965a), while an amendment of 1000 ppm

alfalfa meal may inhibit carpogenic germination under laboratory conditions (Burke, 1973). Apothecial production has been inhibited in culture by an unknown volatile compound, possibly ethylene (Letham, 1976); however, this inhibition has not been reported to occur under field conditions. Letham (1975) also reported that light stimulated initiation and the rate of production of apothecial initials, as well as apothecium differentiation.

Chemicals such as benomyl [methyl-1-(butylcarbamoyl)-2-benzimidazolecarbamate], chloropicrin [trichloronitromethane], dazomet [tetrahydro-3,5-dimethyl-2H-thiadiazine-2-thione], DCNA [2,6-dichloro-4-nitroaniline], dichlozoline [3-(3,5-dichlorophenyl)-5,5-dimethyl oxazoline-dione-2,4], methyl bromide, nitrolime [calcium cyanamide], terrachlor [pentachloronitrobenzene], thiram [bis(dimethylthiocarbamoyl) disulfide], vapam [sodium N-methyldithiocarbamate] and numerous metal ions are reported to inhibit myceliogenic and/or carpogenic germination under laboratory and/or field conditions (Corbin et al., 1971; Hawthorne and Jarvis, 1973; Jones, 1974; Jones and Gray, 1973; McLean, 1958; Partyka and Mai, 1962; Steadman and Nickerson, 1975). Other reports indicate that terrachlor did not control apothecial production under field conditions (Partyka and Mai, 1962); and that dorlone [mixed dichloropropenes and 1,2-dibromoethane], D-D nematocide [mixture of 1,3-dichloropropene, 1,2-dichloropropane, 2,3-dichloropropene and related C_3 chlorinated hydrocarbons], nemagon [1,2-dibromo-3-chloropropane] and telone [1,3-dichloropropene] stimulated apothecial production (Partyka and Mai, 1958).

A sclerotium often produces two or more apothecia per germination cycle and can germinate more than once. A single sclerotium can produce 35 (Brown and Butler, 1936; Stevens and Hall, 1911) to more than 100 apothecia (Dickson and Fisher, 1923). An individual sclerotium can germinate carpogenically more than once (Hungerford and Pitts, 1953; Williams and Western, 1965b). As mentioned earlier, the structural condition of a sclerotium can be weakened after apothecial production (Williams and Western, 1965b) which therefore, reduces its length of survival in soil. Turner and Tribe (1975) state that Dr. Anna Snowdon used "a population of S. sclerotiorum sclerotia which formed apothecia on agar in 1968 and was subsequently kept dry in culture; about half produced a second crop of apothecia when buried in soil in 1970".

Ascospore Dispersal and Survival

Ascospores are forcibly discharged from a mature apothecium exposed to a sudden change in relative humidity, and may attain a height of 10-15 cm with a maximum of 65 cm (Suzui and Kobayashi, 1972a) or 50-100 cm (Stevens and Hall, 1911). The distance of ascospore dispersal is influenced by the discharge height, wind velocity and direction and specific gravity of the ascospores (Suzui and Kobayashi, 1972b). The majority of discharged ascospores are deposited within 2 m from the release point at a 1.2-4.5 m/sec wind velocity; however, wind currents have transported ascospores 230-280 m (Suzui and Kobayashi, 1972a). Other reports indicate that ascospore dispersal from adjacent (Burke et al., 1957) and nearby fields 0.4-0.8 kilometers away (Bardin, 1951) was responsible for infection of host crops.

Ascospore survival and successful plant infection are influenced by the number of viable ascospores present at colonization sites, spore resistance to desiccation and ultraviolet light (Suzui and Kobayashi, 1972b and 1972c) and the production of secondary survival structures such as appressoria (Grogan and Abawi, 1975). Ascospores which are collected in the laboratory and stored under dry conditions (50% relative humidity) at 22 C retain 50% viability for 3 months (Steadman and Cook, 1974). Field collected spores survived for 119 hours at 60% relative humidity (Partyka and Mai, 1962). Germinated ascospores can survive for 4 days in sterile water (Newton and Sequeira, 1972). The fungus can survive as viable ascospores on bean leaves in the field for 12 days and as mycelium in dried colonized bean blossoms in the laboratory for 25 days (Abawi and Grogan, 1975).

Plant Infection

Infection of susceptible plants by S. sclerotiorum requires prior colonization of senescent plant parts such as blossoms, cotyledons, seeds, seed capsules, leaves, pollen or injured plant tissue (Abawi and Grogan, 1975; Abawi et al., 1975a; Cook et al., 1975; Hartill, 1975; Hartill and Campbell, 1974; McLean, 1958; Natti, 1971; Purdy and Bardin, 1953). Blodgett (1946) reported that a cotyledonary rot occurred on bean seedlings which developed from mycelia - or sclerotia-infested seed lots planted in the field. Steadman (1975) showed that infested seeds were completely colonized by the fungus prior to germination and/or plant emergence.

Brooks (1940) and Moore (1955) reported that white mold epidemics are favored by mean temperatures less than 21 C and high humidity or

moisture levels. Infection by ascospores requires continuous moisture contact between the plant part and the fungus for 48-72 hours (Abawi and Grogan, 1975). Free water is also required for 9 or 24 hours for lesion initiation and expansion in actively-growing host tissue at 25 or 20 C, respectively. Once colonized plant tissue or a lesion becomes dry, a subsequent 48-72 hour period of continuous moisture is required for further development by fungal hyphae or mycelium. Grogan and Abawi (1975) speculate that this moisture film acts as a medium through which fungal toxins and/or enzymes involved in pathogenesis are transported from the hyphae to infection sites.

Secondary spread by the fungus is favored by 18 C and 100% relative humidity (Starr et al., 1953). The rate of this spread may also be influenced by temperature, since Gupta (1963) reported that Coriander plants infected with S. sclerotiorum are killed in 4 to 10 days at 19-24 C but not killed at 29 C apparently because the plants outgrew the fungus.

An ascospore germinates to form one or more germ tubes which grow and differentiate into an appressorial initial 10-12 hours later. The initial develops into a compound appressorium within 48 hours after germination began (Abawi et al., 1975a). The fungus enters the host through the cuticle by mechanical disruption applied by a dome shaped infection cushion which develops from the appressorium. Large vesicles form between the cuticle and the epidermal layers, and infection hyphae develop intercellularly. Smaller hyphae branch from the infection hyphae and ramify inter- and intracellularly (Lumsden and Dow, 1973; Purdy, 1958).

Sclerotinia sclerotiorum produces a wide array of enzymes and other organic products some or all of which may be involved in fungal metabolism and/or pathogenesis. These metabolites include acid phosphatase, arabanase, arylesterase, cellulase, endopolygalacturonase, exopolygalacturonase, fumaric acid, galacturonic acid, glyceraldehyde-3-phosphate dehydrogenase, glycolic acid, laccase, malic acid, oxalic acid, pectin methyl esterase, phosphatidase, phosphogluconate dehydrogenase, succinate dehydrogenase, succinic acid, tyrosinase and xylanase (Echandi and Walker, 1957; Fuchs et al., 1965; Hancock, 1966 and 1967; Lumsden, 1969 and 1970; Lumsden and Dow, 1970; Maxwell, 1973; Vega et al., 1970; Wong and Willetts, 1974).

Control - Biological Agents

Many diverse soil inhabiting microorganisms are associated with sclerotia of S. sclerotiorum, and may cause sclerotial degradation or exert an antagonistic effect upon germination. Sclerotia and hyphae can be destroyed under laboratory and field conditions by Coniothyrium minitans Campbell (Campbell, 1947; Hoes and Huang, 1975; Huang and Hoes, 1976; Turner and Tribe, 1975 and 1976) and Trichoderma viride Pers. ex. Fr. (Jones and Watson, 1969). Watson and Miltimore (1975) inoculated sclerotia with Microsphaeropsis centaureae Morgan-Jones to demonstrate its pathogenicity under laboratory conditions and suggested that the same occurs under field conditions. They also suggested that M. centaureae is synonymous with C. minitans based upon similar culture characteristics. Rai and Saxena (1975) reported that the following fungi were antagonistic to S. sclerotiorum in culture and

some affected sclerotial survival under field conditions: Aspergillus niger Van Tiegh, A. flavus Link, A. ustus (Bain.) Thom et Church, Penicillium sp., P. citrinum Thom, P. funiculosum Thom, P. pallidum Smith, P. steckii Zaleski, Stachybotrys atra Corda var. microspora Mathur et Sankhla. Other organisms which were not antagonistic to S. sclerotiorum in culture but did degrade sclerotia in the field include: Rhizopus arrhizus, Cunninghamella echinulata Thaxter, Trichoderma lignorum (Tode) Harz., and Fusarium oxysporum Schlecht ex. Fr. Other fungi that have been isolated from field collected sclerotia include Fusarium culmorum, Mucor hiemalis, M. plumbeus, M. racemosus, Trichoderma hamatum, T. koningi, Alternaria sp., Epicoccum sp., Pencillium brevi-compactum, P. corymbiferum and P. cyclopium var. echinulatum (Merriman, 1976).

Antimycin (Leben and Keitt, 1948) and duramycin (Lindenfelser et al., 1958) are antibiotics produced by Streptomyces sp. which inhibit the growth of S. sclerotiorum, the latter antibiotic at a concentration of 100 µg/ml. The inhibition point of S. sclerotiorum to antimycin was not given. However, S. fructicola was sensitive to 0.4-1.6 µg/ml antimycin (Leben and Keitt, 1948). Baccatine A was isolated from Gibberella baccata (Wallr) Sacc. and is fungistatic to S. sclerotiorum (Guerillot-Vinet et al., 1950). Various antibiotics produced by strains of Actinomycetes and bacteria prevented white mold disease of sunflower (Darpoux and Faivre-Amiot, 1949). However, none of these biological agents were utilized to effectively reduce the incidence of S. sclerotiorum in a practical control program.

Control - Chemical Applications

Applications of benomyl, DCNA, dichlone [2,3-dichloro-1,4-naphthoquinone], terrachlor and thiabendazole [2-(4-thiazolyl)-benzimidazole] around mid to late bloom control S. sclerotiorum infection in some areas of bean production (Beckman and Parsons, 1965; Campbell, 1956; Gabrielson et al., 1971; Lloyd, 1975; McMillan, 1973; Natti, 1971). Effective chemical control of S. sclerotiorum is also reported for other crops such as cabbage (Gabrielson et al., 1973), celery (Potter, 1958), chicory (Staunton, 1971), gerbera (Vigodsky, 1969), ladino clover (Houston et al., 1954), lettuce (Skotland, 1961), muskmelon (Netzer and Dishon, 1970), peanut (Beute et al., 1975), tobacco (Hartill and Campbell, 1973) and tomato (Corbin et al., 1971).

However, Partyka and Mai (1958) report that repeated fumigation with a dichloropropene-containing compound actually increased the incidence of lettuce drop. In western Nebraska satisfactory chemical control on indeterminate dry bean cultivars has not been obtained (Steadman and Kerr, 1972).

Repeated application of specific chemicals to control a plant pathogen during the growing season or a succession of growing seasons can result in the selection of a tolerant strain or a mutated strain which is no longer sensitive to a specific chemical(s). There is no report of such a selection or mutation occurring in S. sclerotiorum yet. However, this has occurred in other fungal species. For example, isolates of S. homoecarpa (Goldenberg and Cole, 1973) and S. fructicola (Whan, 1976) are tolerant to 100 ppm benomyl. Mutants of S. fructicola can infect peach fruit dipped in 500 ppm (Whan, 1976) and 2000 µg/ml

benomyl (Koffman and Penrose, 1976). A single ascospore isolate of Botryotinia fuckeliana (de Bary) Whetz. (Botrytis cinerea Pers. ex. Fr.) was collected in the field and maintained in culture on PDA until conidial transfers were made to PDA amended with benomyl (Polach and Molin, 1975). A mutant resistant to 1000 µg/ml benomyl was found and the authors concluded that benomyl resistant mutants could easily occur in the field also.

Control - Cultural Practice Modification

For control of white mold of bean Zaumeyer and Thomas (1957) recommend the practices of crop rotation, flooding, reduced seeding rates, wider row spacing, fewer irrigations and destruction of bean cull screenings which contain sclerotia. Merriman (1976) states that deep plowing should reduce numbers of sclerotia which survive between crops. However, others claim that deep plowing is ineffective (Brooks, 1940; Gabrielson et al., 1971) and may bring more sclerotia closer to the soil surface (Partyka and Mai, 1962). In western Nebraska crop rotation is an ineffective control, and tillage operations assure the presence of sclerotia at or near the soil surface (Cook et al., 1975). A crop rotation with lowland rice (Stoner and Moore, 1953), continuous flooding for 23-45 days (Moore, 1949) or 42-56 days (Brooks, 1940) and cycles of alternate flooding and drying (Moore, 1949) control white mold in some fields in Florida.

Haas and Bolwyn (1972) found an association between reduced plant population and high disease incidence, and attributed this to high individual plant weights in response to reduced plant competition.