Chapter 9

WHITE MOLD

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Introduction

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

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

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

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

Common names frequently used for white mold in Latin America include "moho blanco del tallo," "Sclerotinia," "esclerotiniosis," "salivazo," "podredumbre algodonosa," "mofo branco," and "murcha de Sclerotinia."

Etiology

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

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

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

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

Epidemiology

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

The minimal quantity of soil-borne sclerotia needed to induce significant plant infection has not been intensively studied. However, populations of 0.2 sclerotia per 30 cm^2 (Abawi and Grogan, 1975) and less than 1-10 sclerotia per kg of soil (Adams and Ayers, 1979; Lloyd, 1975; Schwartz and Steadman, 1978) are known to exist in fields planted to snap, Great Northern, and Pinto beans. Schwartz and Steadman (1978) determined that 1 sclerotium per 5 kg soil was sufficient to cause 46% disease severity in Nebraska. Suzui and Kobayashi (1972b) reported that 3.2 sclerotia per m^2 caused 60%-95% plant infection in a kidney bean field in Japan. Sclerotia are persistent and the availability of primary inoculum from outside bean fields apparently explains why there is no correlation between white-mold incidence and severity, and previous cropping history (Abawi and Grogan, 1979). Herbicide practices may also influence carpogenic germination in host and nonhost fields (Radke and Grau, 1986): some herbicides enhance, while others inhibit, germination.

Apothecia formation (carpogenic germination) is greatest after 10-14 days, at 15-18 ^0C, with soil moisture at 50% of field capacity (wet soil) (J. M. Duniway, G. S. Abawi, and J. R. Steadman,

unpublished data), or in a soil with a matrix potential of -80 to -240 mb (-8 to -24 kPa) (Abawi and Grogan, 1979). Carpogenic germination occurs in fields of common bean, maize, sugar beet (Schwartz and Steadman, 1978), snap bean (Abawi and Grogan, 1975), cauliflower, tomato (Letham et al., 1976), lettuce (Hawthorne, 1976; Newton and Sequeira, 1972), and table beet. It occurs in grassland (Suzui and Kobayashi, 1972b) and in lemon, orange (Smith, 1916), and other fruit orchards (Abawi and Grogan, 1975). In a sandy loam soil, studied by Schwartz and Steadman (1978), many sclerotia germinated and formed apothecia in common bean (11-14 apothecia per m^2) and sugar beet (7-11 apothecia per m^2) fields. An average of two apothecia were produced by each germinated sclerotium, regardless of the crop beneath which it germinated. The majority of apothecia were produced on the side of, or adjacent to, plant stems in the furrow of the irrigated row.

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

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

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

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

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

Symptomatology

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

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

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

Biological Control

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

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

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

Control by Cultural Practices

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

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

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

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

Chemical Control

Applying benomyl, DCNA or dicloran, dichlone, PCNB, or thiabendazole around early- to mid-bloom controls *S. sclerotiorum* infection on snap and common beans, particularly under dryland conditions (Beckman and Parsons, 1965; Campbell, 1956; Costa, 1972; Forster, 1980; Gabrielson et al., 1971; Lloyd, 1975; McMillan, 1973; Natti, 1971; Verdugo-G. and Fucikovsky-Zak, 1980). However, Partyka and Mai (1958) report that repeated soil fumigation with a dichloropropene-containing compound actually increased the incidence of white mold in lettuce. Satisfactory chemical control in western Nebraska has not been obtained on indeterminate common bean cultivars grown under irrigation (Steadman, 1979).

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

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

Control by Plant Resistance

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

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

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

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

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

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

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

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