Chapter 7
White Mold

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Introduction

Sclerotinia sclerotiorum (Lib.) de Bary, similar to *Whetzelinia sclerotiorum* (Lib) Korf and Dumont (37), is distributed worldwide. Although most important in the temperate zones of the northern hemisphere, it also can be a problem in areas with tropical or arid climates, especially during cool seasons or under favorable microclimatic conditions (59). The fungus has been reported in dry bean and vegetable fields in Argentina (32), Brazil (20, 65), Mexico (24), Peru (17), Colombia and other areas in Latin America (27).

Sclerotinia sclerotiorum is pathogenic to a wide range of host plants. Adams *et al.* (5) found 190 species from 130 genera and 45 plant families susceptible to the fungus. Schwartz (60) listed 399 hosts (unconfirmed reports in some instances) and 374 species of 237 genera in 65 plant families mentioned in the world literature. Diseases include blossom rot of fruit trees and flowers, storage rot of vegetables, and white mold of beans.

Beans can be damaged severely by the fungus. Snap bean production has been reduced greatly in New York during growing seasons conducive to fungal development (1, 51). Zaumeyer and Thomas (81) report bean losses of 30% in Virginia during 1916. Yield losses averaged 30% in Nebraska during 1970-1973, while losses in individual fields were as high as 92% (36).

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. The fungus produces large (one to several mm

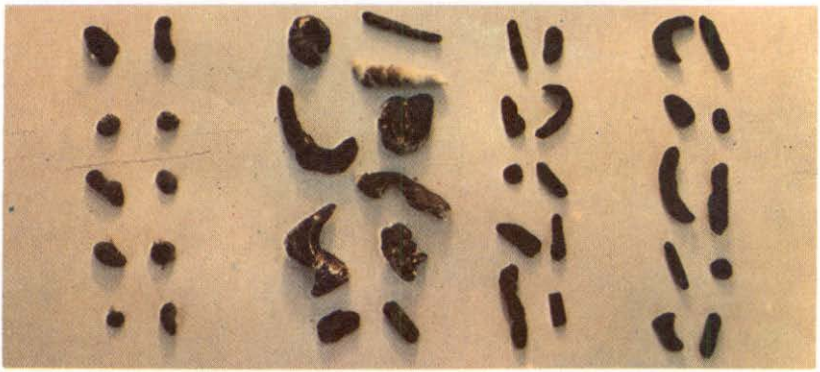

Fig. 1 - Sclerotial forms produced by the white mold fungus; left, culture produced; center, unconditioned and naturally produced; right, conditioned and naturally produced.

diameter), black and irregularly-shaped resting structures called sclerotia (Fig. 1) which germinate to form hyphae or mycelium. A sclerotium, after undergoing a conditioning period, also can germinate carpogenically to produce the sexual stage of one or more apothecia (Fig. 2). These may average 3 mm in diameter and protrude 3-6 mm above the soil surface (58).

Each apothecium contains thousands of cylindrically-shaped asci, each of which contains eight ascospores (78). The ascus measures 7-10 μ in diameter by 112-156 μ in length (18, 38, 58). Over a period of days, an apothecium may discharge more than 2 x 10^6 ascospores (62). The ascospores are ovoid and vary in width from 4-10 μ and in length from 9-16 μ (18, 38, 58, 78). *S. sclerotiorum* can produce microconidia (3-4 µ diameter) during any stage of its life cycle, but these have not been observed to function during sexual fertilization or host infection (38, 58).

Epidemiology

Fields used repeatedly for bean production, even in short crop rotations, often will contain many sclerotia. Sclerotia formed on or within diseased tissue may be dislodged onto the soil surface by wind or harvesting

Fig. 2 - Apothecia produced in field from germinated sclerotium.

operations. Subsequent land preparation redistributes them within the soil profile and over the field (19). Sclerotia also can be distributed by furrow irrigation within fields (62) and by reuse of irrigation runoff water between fields (14, 73). They can survive in sandy loam soils for at least three years (19) and are capable of producing secondary sclerotia (4, 19, 79).

The minimum quantity of soil-borne sclerotia needed to induce significant plant infection has not been intensively studied. Sclerotial populations of 0.2/30 cm^2 (1), 1-3/kg of soil (62) and 3/kg of soil (42) are known to exist in fields planted to snap, Great Northern and Pinto beans, respectively. Schwartz and Steadman (62) determined that 1 sclerotium/5 kg soil was sufficient to cause 46% plant infection in Nebraska. Suzui and Kobayashi (75) reported that 3.2 sclerotia/m^2 caused 60-95% plant infection in a kidney bean field in Japan.

Apothecia formation (carpogenic germination) is greatest at 15°-18°C with soil moisture at 50% of field capacity (Duniway, Abawi and Steadman, unpublished data). Carpogenic germination occurs in fields of dry beans, corn and sugar beet (61, 62), snap bean (1), cauliflower and tomato (40), lettuce (33,52) and table beet as well as in grassland (75). It also occurs in lemon, orange (66) and other fruit orchards (1). In a sandy loam soil studied by Schwartz and Steadman (62), numerous sclerotia germinated and formed apothecia in dry bean (11-14 apothecia/m^2) and sugar beet (7-11 apothecia/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 irrigated row.

While most ascospores discharged by a germinating sclerotium are deposited close to the release point (74), crop infection has been reported in fields as far as 0.8 kilometer away (9, 15). The fungus clearly survives periods of unfavorable microclimatic conditions. Ascospores on bean leaves remained viable for 12 days in the field and mycelium in dried, colonized bean blossoms remained viable for 25 days in the laboratory (1).

Sclerotinia sclerotiorum is a cosmopolitan fungus and can be expected to occur in regions where temperature and moisture conditions are favorable (59). Brooks (13) and Moore (50) report that white mold epidemics are favored by mean temperatures less than 21°C and high humidity or moisture levels. Secondary spread of the fungus is favored by 18°C and 100% relative humidity (67, 77). Abawi and Grogan (1) feel that a surface moisture film is necessary for the fungus to develop and spread on plant tissue.

The rate of spread also can be influenced by temperature (Weiss, Kerr and Steadman, unpublished data). Gupta (30) reported that Coriander plants infected with *S. sclerotiorum* were killed in four to 10 days at 19°-

24°C but were not killed at 29°C, apparently because the plants outgrew the fungus. Microclimatic conditions may be as important as macroclimatic conditions for infection and pathogen development. Hipps (34) showed that irrigation practices significantly altered microclimatic parameters present within the dry bean canopy and conducive to development of *S. sclerotiorum*. Frequent furrow irrigation reduced daytime air and leaf temperatures 3° - 4°C and soil temperatures 10°C, and increased soil moisture content by 10%.

Plant Infection and Symptomatology

S. sclerotiorum infects bean plants by colonization of senescent plant organs such as blossoms (Fig. 3), cotyledons, seeds, leaves or injured plant tissue (1, 2, 19, 47, 51, 56). Blodgett (12) observed cotyledonary rot on bean seedlings which developed from mycelia- or sclerotia-infested seed lots planted in the greenhouse. However, Steadman (68) showed that infected seeds were completely colonized by the fungus prior to germination and/or plant emergence, and that no plant infection was observed in healthy-appearing seed from infested seed lots. Colonization of senescent tissue usually is due to germinated ascospores, but mycelial colonization also has been observed (1, 19).

After colonization of a senescent plant organ, the fungus enters the host by mechanical disruption of the cuticle with a dome-shaped infection cushion developing from an appressorium. Large vesicles form between the cuticle and epidermal layers, and infection hyphae develop intercellularly.

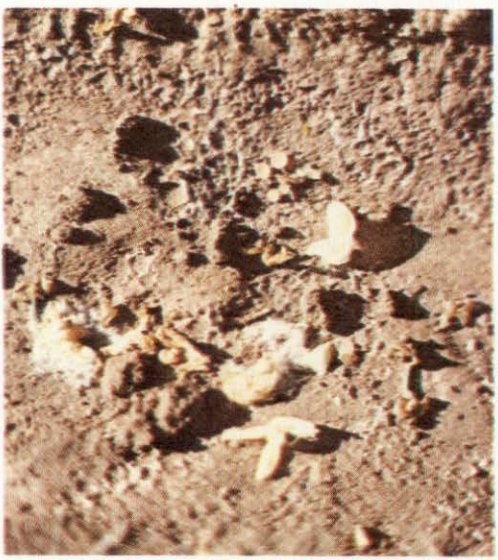

Fig. 3 - Bean blossoms colonized by ascospores of *Sclerotinia sclerotiorum*.

Hyphae branch from the infection hyphae and ramify inter-and intracellularly (44, 55), causing a watery soft rot. The fungus produces many enzymes and other products, including endo- and exopolygalacturonase, pectin methyl esterase (43) and oxalic acid (45) which are important to pathogenesis.

Symptoms and signs of infection appear initially as a water-soaked lesion (Fig. 4) followed by a white moldy growth on the affected organ (Fig. 5). 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 (Fig. 6) (12, 81). Plant wilting also may be evident

Fig. 4- Watery soft rot and sclerotia production in bean pod infected by white mold fungus.

Fig. 5 - Mycelia and sclerotia production on infected bean pod.

Fig. 6 - White or bleached symptom of bean plant severely infected by the white mold fungus.

Fig. 7- Canopy wilt caused by white mold infection of bean vegetation.

within the plant canopy after infection of the plant stem and/or vines occurs (Fig. 7).

Control by Biological Organisms

Many soil microorganisms are associated with sclerotia of *S. sclerotiorum* and may cause sclerotia to degrade or not germinate. Such organisms include *Coniothyrium minitans, Trichoderma* sp., *Aspergillus* sp., *Penicillium* sp., *Fusarium* sp., and *Mucor* sp. (35, 49, 57, 76). *S. sclerotiorum* also is inhibited by various antibiotic substances produced by *Gibberella baccata* (29), *Streptomyces* sp. (39, 41) and other actinomycetes and bacteria (25). None of these biological agents has been used effectively in reducing *S. sclerotiorum* incidence under practical field conditions.

Control by Cultural Practices

Zaumeyer and Thomas (81) recommended cultural practices such as crop rotation, flooding, reduced seeding rates, fewer irrigations and destruction of bean cull screenings which contain sclerotia as methods of controlling the pathogen. Similar recommendations have been made for control in Brazil (20). Deep plowing also has been advocated (49) and disputed (13, 28, 54) as a control measure. Crop rotation is not likely to be effective since sclerotia survive in soil, and tillage operations will assure the presence of sclerotia at or near the soil surface (19).

Irrigation frequency can influence disease incidence on cultivars with indeterminate plant growth habits and dense plant canopies (11). Growers are advised not to irrigate if white mold infection is prevalent within their bean fields (70). Re-use of irrigation water should be eliminated, or the water treated to remove sclerotial and/or ascosporic contamination which can contribute to current or future disease epidemics (73).

A survey of bean fields in Canada revealed that infected and non-infected crops were grown on soils with a pH of 7.5 and 7.0, respectively. However, the authors did not determine the nature or applicability of this association (31). Heavy fertilizer rates are not recommended, since they are associated with increased disease incidence (7), presumably because of the stimulatory effect upon plant canopy density.

Control by Chemicals

Application of Benomyl, DCNA or Dicloran, Dichlone, PCNB or Thiabendazole around early- to mid-bloom controls *S. sclerotiorum* infection on snap beans under dryland conditions (10, 16, 20, 28, 42, 48, 51). However, Partyka and Mai (53) 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 dry bean cultivars grown under irrigation (69). Sporadic results also have occurred in California, Colorado, Montana, Washington and Wyoming. Timing of the chemical application and thoroughness of coverage are critical to successful control.

Control by Plant Resistance and Architecture

An association between plant canopy development and white mold disease incidence and severity has been observed in various crops including beans. Row spacing, growth habit and plant density can influence bean canopy development and disease incidence (12, 21, 22, 23, 31, 51, 64, 71, 81). An open plant canopy which will facilitate air circulation and light penetration within the canopy helps prevent infection. It results in more rapid drying of moisture-covered leaf and soil surfaces (21).

As an example of row spacing-cultivar interaction, the cultivar Aurora escapes infection when planted at a within-row spacing of 4-5 cm because of its upright, open growth habit (22). However, when planted 30.5 cm apart within the row, it sprawls and is more severely infected. Orientation of bean rows parallel to the prevailing wind direction also may reduce disease incidence by providing improved air circulation and better light penetration (31).

Resistance to *S. sclerotiorum* has been observed in *Phaseolus vulgaris* germplasm (12, 26, 46, 58, 80), but comparative differences between cultivars were not reported until recently (8). Resistant cultivars include Black Turtle Soup, Charlevoix and Valentine (8, 63).

Resistance also has been identified in *P. coccineus* (6, 72) and *P. coccineus* x *P. vulgaris* hybrids (3). Attempts are being made to develop stable resistance by using a plant structure which maximizes disease avoidance and also possesses physiological resistance to infection by *S. sclerotiorum* (22). Such cultivars would be conducive to an integrated control program which could include fungicides and cultural practices if a greater degree of plant protection still is required.

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